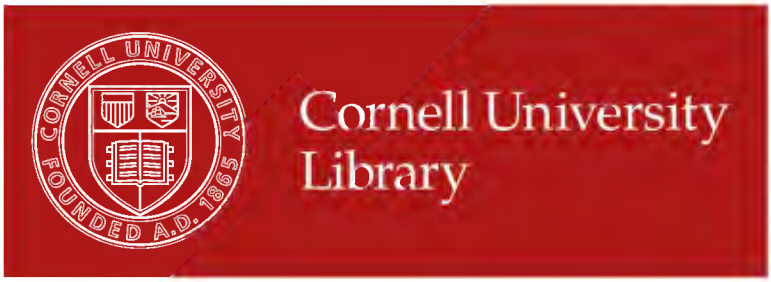

WHARTON AND STILLÉ'S

MEDICAL JURISPRUDENCE.

FOURTH EDITION.

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EDITORS' PREFACE TO THE FOURTH EDITION.

SINCE the publication of the third edition of this work in 1873, vast progress has been made in our knowledge of the action of poisons upon the animal economy, and the immense advances which have been made in the domain of organic chemistry have added much to our knowledge of the chemistry of poisons, and have resulted in improved methods of analysis, and, consequently, in the more ready and more certain detection of many of the organic poisons. Therefore it has been found necessary to add so much new material to that portion of the work treating of poisons as to require the assignment of an entire volume to this subject.

It will be seen that much more attention than formerly has been paid to the chemistry of the poisons, the most recent tests and the best methods for their detection in cases of poisoning having been added; the latest advances in our knowledge of the physiological action of the various poisons have also been incorporated, and great care has been taken to collect the more recent cases of poisoning, especially those illustrating points of medico-legal interest; for assistance in the collection of these cases the editors are much indebted to Charles Harrington, M.D., Assistant in Chemistry in the Harvard Medical School. Thanks are also due to A. Lawrence Mason, M.D., of Boston, for his assistance in the careful

revision of the proofs, which was rendered necessary by the unexpected absence in Europe of one of the editors.

An apology may be expected for the long delay in the appearance of this volume on Poisons, but, as will be seen, the vast amount of new material, especially of a technical character, required much patience and a great deal of labor, which could not be deputed to other hands, but must be personally prepared by one or both of the editors.

ROBERT AMORY, M.D.,
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Boston, March, 1884.

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ON POISONS.

CHAPTER I.

§ 1. *Definition of poisons.*—Physicians generally understand by the word *poison* a substance having an inherent deleterious property, which renders it, when taken into the system, capable of destroying life. It is difficult, however, to give a definition to the term which will meet the signification attached to it by different classes of persons; for while, in common language, poisons are understood to be those articles only which are deadly in small doses—as strychnine, prussic acid, arsenic, etc.—the lawyer and the physician will unite in affixing to it a general meaning, similar to that which we have given above. Some substances are habitually classed as poisons which, according to the popular signification, would have a doubtful claim to be so called, being fatal only in large doses; and every medical practitioner is aware that very many active remedial substances, and some which are not injurious when taken in ordinary amounts, may, in an overdose, produce serious and fatal effects. Moreover, questions may arise as to the applicability of the term to substances which destroy life by mechanical irritation, such as powdered glass, etc., although the administration of such substances to another, with intent to

injure, is made a criminal offence in the various penal codes. "Whoever wilfully administers to another, for the purpose of injuring his health, poison or *other substance*, which is capable of injuring the health, shall be punished," etc.[1]

"Whoever shall administer, or cause to be administered to or taken by any person, any poison, or *other destructive thing*, with intent to commit murder, shall be guilty of felony."[2] The French law interdicts the wilful administration of any substances "which, without being of a nature to produce death, are injurious to health."[3] In order to avoid the evil of giving too wide or too restricted a meaning to the word "poison," we adopt this definition, which makes no reference to the quantity required to produce a poisonous result, nor to the mode in which it is introduced into the system. Cases in which a really poisonous substance has been taken with impunity are more rare. In the majority, the immunity is only comparative, the person being affected merely in a less degree than is usual. This important consideration must not be overlooked, for there are many cases recorded, in which, for instance, among other deleterious medicines, opium, arsenic, belladonna, digitalis, chloral hydrate, and even strychnine, when the individual has become gradually accustomed to increasing doses, have been taken with impunity in amounts usually considered fatal to mankind; such cases attest the danger of assigning a deadly action to a poison, which has been administered to a person whose peculiar idiosyncrasy (so-called) may resist a dose which to the generality of mankind is considered dangerous or even fatal. Moreover, it has been repeatedly shown, that persons who have become habituated to a continuous use of certain drugs, as arsenic eaters, morphine consumers (whether by the mouth or by subcutaneous administration), and the like, require doses far beyond those which would prove fatal to persons in whom the habit does not prevail.

§ 2. *Conversion of harmless substances.*—The *idiosyncrasy* which converts a harmless substance into a poisonous agent, is very frequently observed in the case of articles of food. Thus mussels, fish, pork, and mutton have frequently given rise to all the symp-

[1] Translated from Das Strafgesetzbuch für das Deutsche Reich, nebst dem Einführungsgesetze vom 31. Mai, 1870.

[2] 24 and 25 Vict., c. 100, s. 11, Aug. 1861, and Taylor's Medical Jurisprudence, 1873, page 81.

[3] Tardieu, 1875, page 4.

toms of irritant poisoning. It should be remembered, however, that the symptoms in these cases may result as well from the mechanical irritation of the food—too large a meal having been taken, or from the fact of its being in a condition unfit for use; while it may be seen farther on in this treatise, that some meats become poisonous, because they are infested with animal parasites, or from commencing putrefaction.

§ 3. *Disease.*—Disease also has sometimes the effect of rendering the system tolerant of substances, which would be poisonous in the same doses in a healthy state of the system. In acute alcoholism or alcoholic intoxication, the blood and tissues may be so loaded with alcohol, as to interfere wholly or partially with the absorption of a poisonous agent. This fact is so well known to the medical profession, that the tolerance of chloral hydrate, belladonna, digitalis,[1] and opium, lead physicians to give larger and more frequent doses in delirium tremens than would seem to the inexperienced to be safe. In consequence of this knowledge fatal accidents have occasionally occurred[2] from the inconsiderate repetition of large doses to inebriate patients, who later may have eliminated the alcohol and be recovering from its intoxicating effects. This same observation and explanation may be true in certain diseases, where apparently the blood contains an organic poison due to functional or organic disease of the kidneys, and thus seems to offer a limited opposition to the absorption of a toxic agent.[3] During the active stage of severe inflammatory and febrile diseases, mercury may be given in large and repeated doses without producing salivation. On the other hand, certain diseases render the body more susceptible to the actions of poisons. Illustrations of this are presented by the aggravation or the ready production of cerebral symptoms, after the use of small doses of narcotics, in those who have a predisposition to cerebral congestion or apoplexy, and by the extreme facility with

[1] Medical Times and Gazette, Aug. 23d, 1873, p. 205.

[2] Irish Hospital Gazette, 1873; Year Book of Therapeutics and Pharmacy, 1872, p. 254; Deutches Archiv, Bd. VIII., Jan. 1871, quoted in the Practitioner for April, 1871, p. 243; Lancet, Feb. 1, 1879, p. 147; British Med. Journal, Aug. 29, 1868; Lancet, Aug. 27, 1870; De l'influence des liquides alcooliques sur l'action des substances toxiques, Dubois (Thèse de Paris, 1870).

[3] Lo Sperimentale, Fasc. 9, 1873, quoted in Practitioner, Feb. 1873, p. 132.

which salivation follows the administration of mercury in persons affected with certain forms of disorganization of the kidney. The use of iodide of potassium after mercurial preparations, is said to favor the development of the mercurial cachexia.

These facts tend to show that the activity of a poison depends largely upon the readiness with which the poison is absorbed in any individual case. It is unnecessary to state here all of the circumstances which govern the rapidity of absorption. This discussion should be left for each case as it arises, and be elucidated from the testimony of a competent medical expert, it being remembered that no general rule can be precisely laid down,[1] and that certain diseases or abnormal conditions of the system may facilitate, while others may retard, the action of a poison.

§ 4. *Evidence of poisoning.*—Medical evidence in cases of suspected poisoning is derived from several sources, to wit: the *symptoms*, *the post-mortem appearances*, *chemical analysis*, *and experiments on animals;* these are also called the *clinical*, *anatomical*, *chemical*, and *physiological* signs of poisoning. In addition, we have the moral evidence derived from non-medical facts, concerning which the physician must testify as well as other persons immediately connected with the case. In order to establish the fact of poisoning with certainty, everything, of however little importance it may seem to be at the time, which occurs during the life of the patient, must be observed and accurately noted. Especially should the physician in attendance upon any case, where there is the slightest suspicion of poisoning, take charge of everything which may by any possibility throw light upon the cause of the illness, such as all vomitus and excreta (urine and feces), bottles of medicine, powders, and any article of food or drink taken previous to the illness, if such can be obtained; all of these substances should be carefully preserved by being placed in clean glass vessels (ordinary glass preserve jars will answer the purpose) by the physician himself, sealed, and kept under lock and key until wanted. Of the greatest importance is the collection of the vomitus and urine during the life of the patient, if possible, for the detection of the nature of the poison by chemical analysis of these fluids.

§ 5. The *statistics of poisoning* show that the kind of poison

[1] *Vide* § 8.

most frequently used varies very greatly according to circumstances, such as the ease with which poisons can be obtained by the laity, the advancement of popular knowledge concerning the poisonous action of substances used in the arts and manufactures, etc. Hence the variation in frequency with which the different poisons are used in different countries and in different periods. Thus cases of criminal poisoning by phosphorus were unknown previous to the year 1850 on account of the ignorance of people generally as to its poisonous effects.

In Prussia during the years 1869 to 1873, of 32,613 deaths, 1454 were due to poisoning, as follows:—[1]

Poisonous gases	1007
Poisonous berries and fungi	62
Poison acids	40
Arsenic, phosphorus, and other poisons	57
Alcohol	288

Taylor gives the following statistics of poisoning in England for the years 1837–1838:—

Opium and its preparations	196
Arsenic	185
Sulphuric acid	32
Prussic acid	27
Oxalic acid	19
Corrosive sublimate and mercury	15
Mixed or compound poisoning	14
Oil of bitter almonds	4
Poisonous mushrooms	4
Colchicum, nux vomica (of each 3)	6
Nitric acid, caustic alkalies, tartar emetic, acetate of morphine, strychnine, deadly nightshade, aconite (of each 2)	14
Bichromate of potassium, nitrate of silver, Goulard's extract, sulphate of iron, muriate of tin, hellebore, castor oil seeds, savin, hemlock, cantharides, and cayenne pepper (of each 1)	11
Unknown	14
	541

[1] A. v. Fürcks, Rückblick auf die Bewegung der Bevölkerung im Preussischen Staate während des Zeitraumes vom Jahre 1816 bis zum Jahre 1874. From Maschka's Handbuch der gerichtliche Medecin, II., page 21.

In England and Wales, during the five years 1863–1867, the cases of poisoning were as follows:—[1]

Opium—laudanum, syrup of poppies, and Godfrey's cordial	482
Opium and its compounds	114
Morphine	32
Prussic acid and cyanide of potassium	151
Essential oil of almonds	31
Arsenic	83
Oxalic acid	66
Strychnine and vermin killer	61
Mercury (compounds of)	58
Sulphuric acid	53
Nitric acid	16
Hydrochloric acid	8
Carbolic acid	5
Alcohol	35
Phosphorus	15
Ammonia	11
Chloride of zinc	8
Mussels	8
Fungi	6
Aconite	6
Belladonna	6
Chlorodyne	4
Turpentine	3
Colchicum	3
Nitre	3
Sulphate of copper	3
Cantharides	2
	1270

Tardieu[2] gives the statistics in France for twenty-one years, 1851–1871, as follows:—

[1] Taylor on Poisons, 1875, page 179.

[2] Étude sur l'empoisonnement, 1875, page 164.

	1851.	1852.	1853.	1854.	1855.	1856.	1857.	1858.	1859.	1860.	1861.	1862.	1863.	1864.	1865	1866.	1867.	1868.	1869.	1870.	1871.	Total.
Arsenic . . .	35	25	33	25	42	14	18	9	9	3	14	5	8	7	6	4	4	8	14	2	2	287
Phosphorus .	13	3	4	12	21	14	23	20	16	15	13	16	5	25	12	6	18	11	11	4	3	267
Sulphate of copper	3	5	10	8	4	2	8	5	15	5	4	8	2	9	4	7	7	2	2	5	5	120
Verdigris . .	2	3	10	4	6	3	1	...	...	2	2	...	...	...	1	1	3	1	...	...	...	39
Sulphuric acid .	1	1	2	1	2	7	1	4	4	4	1	2	...	...	...	...	2	...	3	1	...	36
Cantharides .	4	1	3	3	...	6	1	2	2	...	1	...	...	3	...	1	2	...	1	...	5	35
Opium . . .	...	...	...	1	...	...	...	...	1	1	1	2	...	...	...	...	...	...	...	...	...	6
Hellebore . .	...	...	...	...	...	...	...	...	...	...	2	...	...	...	...	1	...	...	...	...	...	3
Tartar emetic .	1	...	...	...	...	...	1	1	...	...	...	1	...	...	...	...	...	...	...	...	...	4
Sulphate of iron	1	...	...	...	...	...	2	...	...	...	...	1	...	...	...	...	...	...	2	...	...	6
Nitric acid . .	1	...	1	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	3
Ammonia . .	...	...	1	...	...	...	...	...	1	...	1	...	1	...	...	...	...	...	...	...	...	4
Mercury . . .	...	...	1	...	1	...	...	...	...	1	...	...	1	...	...	3	...	1	...	...	...	8
Datura . . .	...	...	3	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	3
Nux vomica .	...	...	...	...	2	...	...	...	...	1	...	...	...	...	...	...	1	...	...	...	1	5
Hydrochloric acid . . .	...	...	...	1	...	...	...	...	1	...	...	...	1	2	...	3	...	...	...	...	...	8
Potash . . .	1	...	...	...	...	...	...	...	...	...	...	...	1	...	...	...	...	...	...	...	...	2
Acetate of lead .	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	0
Carbonic acid gas	...	...	...	...	...	...	...	...	...	...	...	1	...	...	...	...	...	...	...	...	...	1
Broom seeds .	...	...	1	...	...	...	...	1	...	...	...	...	...	...	...	...	...	...	...	...	...	2
Colchicum . .	...	...	...	...	...	...	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1
Mushrooms . .	...	...	1	...	...	...	...	...	...	...	...	1	...	...	...	...	...	...	...	...	...	2
Euphorbia . .	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1
Beaume Fioraventi . . .	...	...	...	...	...	...	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1
Aqua sedativa } Eau sedative }	...	...	...	...	...	...	...	...	...	...	...	1	...	...	...	...	...	...	...	...	...	1
Belladonna . .	...	...	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1
Powdered glass	...	1	...	...	...	1	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	3
Strychnine . .	...	...	...	...	...	...	...	...	...	...	...	...	...	3	1	1	1	...	1	...	...	7
Digitaline . .	...	...	...	...	...	...	...	...	...	...	...	...	...	2	...	...	...	...	...	...	...	2
Nicotine . . .	...	...	...	...	...	...	...	...	...	...	...	...	...	1	...	...	...	1	...	...	...	2
Prussic acid .	...	...	...	...	...	...	...	...	...	...	...	...	...	1	1	...	...	...	...	...	...	2
Laudanum . .	...	...	...	...	...	...	...	...	...	...	...	...	...	1	...	...	...	...	...	...	1	2
Antimony . .	...	...	...	...	...	...	...	...	...	...	...	...	...	1	1	...	...	...	...	...	...	2
Croton oil . .	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1	1	2
Sulphate of zinc	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1	...	...	...	...	...	1
Tincture of iodine . . .	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1	...	...	...	...	...	1
Eau de Javelle.	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1	...	...	...	...	...	1
Sulphuric ether	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	0
Cyanide of potassium . .	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	2	...	...	...	2

In Massachusetts[1] the State Medical Examiners reported, out of 2976 supposed deaths by violence viewed by them, that 45 were deaths from poisoning. These poisons may be tabulated as follows:—

[1] Transactions of the Massachusetts Medico-Legal Society for 1878–81, Cambridge, Riverside Press.

Opium and morphine	18
Arsenic	13
Oxalic acid	1
Oil of cedar	1
Oil of tansy	1
Lobelia	1
Chloral-hydrate	1
Chlorate of potassium	1
Cantharides	1
Prussic acid	2
Cyanide of potassium	1
Corrosive sublimate	1
Tartar emetic	2
Aqua ammonia	1
Total	45

§ 6. *Symptoms.*—It is but rarely that some knowledge of the symptoms preceding death is not obtained, even where the mode of their invasion has been unobserved. Occasionally, indeed, persons are found dead from the effects of poisoning, of the precise manner of whose death nothing can be learned, the suspicion of poisoning arising only in consequence of the finding of the phial from which the poison had been taken, or other circumstantial evidence of a similar character. Such are, in general, cases of self-destruction; the unfortunate victim of misfortune or excess having designedly withdrawn himself from observation, with the view of being undisturbed in his purpose.

A notable instance of such a case of deliberate suicidal poisoning in which the circumstances were so carefully planned as to avert suspicion of poisoning is personally known to the writer. The details of the tragedy may be briefly summarized. A man in perfect health, on a very hot day in summer, took a private room in a hotel, and, complaining of a violent headache, sent the messenger who answered his call to an apothecary shop for some camphor spirits, which he poured upon a handkerchief and bound on his forehead, at the same time dismissing the messenger. He was found dead several hours afterwards, and his death was attributed to a congestion of the brain brought on by the heat of the weather. Suspicions of suicide being aroused later, it was found that he had poisoned himself with prussic acid, and disguised its smell by the external application of camphor. No bottle or paper was found in

the room or about the person to point in the slightest degree to poisoning as the cause of death, yet chemical examination of the stomach showed a large amount of prussic acid. The *post-mortem* appearances were, of course, those of general venous congestion as is found in cases of prussic acid as well as some other forms of poisoning, especially cerebral congestion.

In most cases of accidental and homicidal poisoning, some knowledge is acquired, either directly or indirectly, of the nature and progress of the symptoms. With few exceptions however, medical aid is sought before death has occurred, and the direct testimony of the physician can thus be obtained. The history of the symptoms should in all cases be made in great detail, as these form a very important aid in the detection of criminal poisoning.

The first effect of a drug would, of course, be its local action, and this should be looked for upon any of the avenues through which poisons may be administered. A corrosive poison, if taken by the mouth, would show its local effects upon the face, lips, etc., but if the suspected poison be not a corrosive or a local irritant, the first condition of the intoxication (poisoning) is its absorption, and if this absorption does not occur, fortunately there are no poisonous effects to be noted; for example, the absorption of arsenious acid may be delayed or prevented by the ingestion of some albuminous substances, such as milk, thus locking up the poison in a mass, which cannot without subsequent digestion be received into the circulation.

There are several ways by which poisonous drugs may be introduced into the system. The principal of these are the mucous membranes, the lungs, the cellular tissue, and inflamed serous membranes. Whatever may be the way by which the agent is introduced, science possesses the means of recognizing deleterious substances, whether in the blood, the secreted fluids, or in the tissues themselves.

The careful researches of physiologists (Prof. Cl. Bernard and Prof. Gubler among others) of modern times show clearly that any mucous surface can absorb medicaments; briefly stated, these avenues for the administration may be enumerated in the order of rapidity as follows: the surface of the lungs, stomach, intestines, mouth, nose, eyes, tear-passages, rectum, vagina, uterus, bladder, as in case of atropine, and prepuce. The presence of food in the stomach and bowels interferes also with the rapid absorption of a drug or poison,

because the presence of alimentary substances separates the drug from contact with the absorbing surface. The lungs will absorb not only gases and volatile poisons, such as sulphuretted hydrogen, coal gas, ether, chloroform, prussic acid, etc., but also the watery vapors holding in suspension particles of drugs or poisons, and even dust which is suspended in the atmospheric air; for instance, it has been repeatedly observed at *post-mortem* examinations, that particles of cotton, lint, or wool, as well as coal dust, emery powder, and particles of steel, are inspired and incorporated in the lung substance, and several recent cases of chromate-of-lead poisoning have been recorded in which inhalation of the dust occurred while weaving yarn colored with this pigment. These cases will be referred to under the head of that poison.

The atomization of liquids, now so common a means of medication by inhalation, furnishes a very efficient avenue for the administration of drugs, though the chances of its use in criminal poisoning would not be probable. The uterus, especially when congested with blood or in a state of inflammation, furnishes also a very rapid means of absorption (as, for instance, in cases of carbolic-acid poisoning); and often, from carelessness or ignorance on the part of the practitioner, substances apparently non-poisonous may, by local contact with the outlet of the Fallopian tubes, originate an irritation which may finally effect a more extensive and even fatal and rapid peritonitis. It is undoubted that certain substances innocuous in themselves when introduced in the ordinary channels, may when introduced into extraordinary channels provoke an irritation which can and has secondarily caused fatal or serious inflammatory processes; for instance, fistulous openings into the chest or abdomen, or by the urethra into the bladder, etc. There are certain substances which may be introduced into the mouth and stomach, and are there so slowly absorbed and rapidly eliminated at one and the same time that poisoning may not occur, while, if the same drugs be directly introduced into the circulation, a very small amount acts as an active and often fatal poison; this is true also of similar substances introduced through the cellular tissue, as for instance, by subcutaneous injection or by a wound. The interior surfaces of the mouth, nose, and pharynx absorb actively many poisonous substances, even if they are not swallowed, and hence give rise to fatal poisoning;

among these substances may be mentioned cyanide of potassium and especially nicotine.

The inner folds of the prepuce contain a good absorbing surface. The inner surface of the bladder does not usually offer a good avenue to absorption. Atropine may be absorbed from the bladder even when it has been eliminated from the system by the kidneys, and hence arises the necessity for catheterization in the treatment of atropine poisoning. But a very important channel for the introduction of drugs and poisons in a state of solution is that of the cellular tissue. From the peculiar character of this tissue, medicines, especially those of a non-irritating kind, are readily introduced by means of a hollow needle through which the fluid is forced by pressure from a small syringe into the cellular or subcutaneous tissue, and very rapidly (from five to ten minutes) peculiar physiological or medicinal effects are produced. Morphine presents a fair illustration of this fact, because one-half or a third of the dose which would be required by the stomach, will in ten or twenty minutes exhibit the full action of the drug if administered by this means. Ipecacuanha, whose emetic properties are well known to follow the swallowing of a proper dose, will exhibit emetic properties, though not so violent, when injected under the skin. Apomorphia, an artificial drug, will produce more violent emesis when given subcutaneously than when swallowed. This fact is now so well known to the medical profession, that it is hardly worth while to discuss it in further detail here.

The skin in a healthy state absorbs but few medicines, and these only in small quantities, unless in a finely divided state, as when combined with some fat, as in the use of ointments. Mercury offers a good illustration of this method of drug administration by inunction. There are, however, some substances which are absorbed by the skin with considerable energy, as, for instance, a recent external remedy for skin diseases called chrysophanic acid. Gaseous, or the more volatile substances, are absorbable by the skin, while substances simply soluble in certain solvents do not readily pass through the cutaneous surface, and when dissolved in water, the natural action of the skin tends to exhalation rather than inhalation, and thus offers a difficulty to the absorption of the dissolved substance; though the palms of the hands and the soles of the feet present a slight exception to this statement, as well also as the armpits and groins. On the other hand, under certain conditions of

heat and bodily exercise, certain substances in contact with a large surface of the body are absorbed quite actively. There are recorded cases of soldiers,[1] who, having secreted next to their skin tobacco leaves, have, after walking sufficiently to excite an active perspiration, been overwhelmed by the absorption of nicotine and thus been fatally poisoned; there are also instances where warm cataplasms or poultices of tobacco applied to the skin of children by ignorant and careless persons have caused fatal poisoning by nicotine; poisoning has also been caused by the application of belladonna plaster to the skin.

Moreover, it should not be forgotten that, where there is a considerable abrasion or loss of cuticle, the absorption of many substances in solution can readily be effected, as by the endermic method of administering drugs; absorption of many substances may also be facilitated by a diseased condition of the skin, as, for instance, in eczema and in skin diseases peculiar to syphilis, many medicinal substances are readily absorbed which a healthy, unbroken skin will not absorb. The serous surfaces in chest or abdomen, when inflamed, also offer a very ready means of absorption.

§ 7. We come next to the consideration of what constitutes a case of poisoning. As facts in toxicology are becoming better known, it is somewhat difficult to make a general statement which would lead to suspicion of the administration of poison. In general terms it may be said, that, in cases where there are no marks of personal violence upon the body of the deceased, and the history of the symptoms can hardly, in the opinion of a competent medical practitioner, explain a sudden and suspicious death as attributable to natural causes, there is reason to suppose that the deceased came to his death either from some internal arrest of the vital functions by a naturally explained accident, whose pathological effects can be shown at the autopsy, or else by means of some poison which has been absorbed. In the more evident cases of poisoning, for instance, by an irritant or corrosive agent, or by a poisonous metallic agent, or poisonous alkaloid, etc., the history may be generally comprised in the following words:—

[1] Gallavardan, Empoissonnement par l'application des feuilles de tabac sur la peau, Gazette des hôpitaux, 20 Août, 1864, from Tardieu, op. cit., 1875, p. 929.

Whenever a man in apparently full health is suddenly overtaken with serious and increasingly alarming symptoms, accompanied with pains in the region of the stomach; or when, in the absence of vomiting and diarrhœa, a complete prostration of the vital forces, a cadaverous expression of countenance, and an abundant perspiration suddenly appear, and are soon followed by death, there is good reason to suspect poisoning, the attestation of which must be proved by anatomical and chemical examination of the body. In certain states, that of Massachusetts chiefly, the statutes, which provide for suspicious deaths, require that the official medical examiner, duly commissioned as such by the executive, shall proceed immediately to view the body, and examine the circumstances of death. This official, skilled in the science of medicine, unless satisfied that the death is due to natural causes, is required to take charge of the body, and to satisfy *himself*, not a jury, of the cause of death. This disposition of the investigation combines all the advantages of the Scotch method and that of the French system, because it allows a medical man to use his medical education and judgment in forming a professional opinion, which must immediately be reported in writing to the legal authority, judge, and district attorney, which is charged with the inquest as to who caused the death. If the district attorney is for any reason dissatisfied with the reported professional opinion of the cause of death, he may order an autopsy to be conducted. According to this process, which, in our opinion, is superior to that of any other, any sudden death may be a supposed case of violence, and upon this supposition the medical examiner is required to investigate and report upon the cause of death.

The object of the medico-jurist here, as in all other attempts upon the health or life of an individual, is to determine in a precise manner the cause of the disease or death; but, in the majority of crimes, the expert who is called upon to investigate these causes may not begin his examination for weeks or months after the commission of the deed, and until suspicions have led to judicial interference; hence, the natural difficulties the expert would experience are greatly enhanced by the unnatural delay.

The expert has three sources of information for his guide:—

Symptoms.—As to the nature of the symptoms which have pre-

ceded the death.[1] The great difficulties connected with this source of information are: 1st. The unwillingness and ignorance of those who witnessed the symptoms; and the fact that a great number of poisons cause symptoms that are almost identical in character, and similar to those of many diseases.

On the other hand, the symptoms following the absorption of some of the poisons are very similar, and it may be extremely difficult for an expert to determine by the history preceding death that a certain drug had been administered. It is hardly necessary to point out in this place in what way he may be misled or confused, as under the head of each poison the symptoms will be carefully given, and it will be seen that these alone can hardly elucidate this

[1] In regard to the value of symptoms as an indication of poisoning, Dr. Reese (Am. Journ. Med. Sci., April, 1872, p. 353) quotes from Christison: "It is now laid down by every esteemed author in medical jurisprudence, that the symptoms, however exquisitely developed, can never justify an opinion in favor of more than high probability." (Christison here refers to the work of Orfila, ii. 360.) "And again, p. 295, when treating of arsenic, 'the present doctrine of toxicologists and medical jurists seems universally to be, that symptoms alone can never supply decisive proof of its administration.' 'All these symptoms may be caused by natural disease,' . . . 'consequently, every sound medical jurist will join in condemning unreservedly the practice which prevailed last century, of deciding questions of poisoning in such circumstances, from symptoms alone.'"

The first of the above quotations made by Dr. Reese in his review of Mrs. Wharton's case, will be found on the 37th page of the second Edinburgh edition of Christison's "Treatise on Poisons," and immediately after these words occurs the following sentence: "In laying down this doctrine medical jurists appear to me to have injudiciously confounded together actual symptoms with their general characteristics. If the doctrine is to be held as applying to the evidence from symptoms only so far as they are viewed in questions of general poisoning, that is as applying to the general characteristics merely of the symptoms, it is deduced from accurate principles. But if it is likewise to be applied, as recent authors have done, to the actual symptoms produced by particular poisons, then it is a rule clearly liable to several important exceptions. These exceptions will be noticed under the heads of the mineral acids (p. 159), oxalic acid (p. 201), arsenic (p. 296), corrosive sublimate (p. 386)." Immediately following the second quotation will be found this sentence: "But modern authors appear to have overstepped the mark, when they hold that the rule against deciding from symptoms does not admit of any exceptions. For there are cases of poisoning with arsenic, not numerous certainly, yet not very uncommon either, which can hardly be confounded with natural disease; in which the power of deciding from symptoms alone is most required, because the chemical evidence is almost always wanting."

question. However, if there is found near the dead body, or in the room or place where it lies, any phial, cloth, or article of furniture which may be supposed to point to any suspicion of the substance used, it should be carefully taken in charge by the investigating officer and examined. Oftentimes a careful search of the premises and person of the dead body will reveal some suspicious article which must not be overlooked. Briefly stated, the action of narcotics is so often similar in symptoms, that a guess of the specific poisonous agent is not admissible. The same remark may also apply to many of the alkaloids whose symptoms are so rapidly followed by fatal issue that, unless a man skilled in medicine should himself have seen the symptoms preceding death, no reliable history of the symptoms can be obtained, since it is not safe to trust to the hearsay evidence of bystanders. The same may be said of certain natural accidents which cause sudden death and which may erroneously be attributed to a poisonous agent, such, for instance, as aneurism, embolism, or internal hemorrhage. Especially does the preliminary examination require tact on the part of the investigating officer.

§ 8. All poisons act by absorption[1] (though certain of them may cause local lesions by their irritant properties), and are carried by the blood through all the tissues. They are eliminated with the excretory fluids. On the ratio of elimination to absorption depends the activity of these poisons. If a drug is absorbed more rapidly than it is eliminated, we get symptoms of the action of this drug. Unless the inhalation of ether exceeds its exhalation, no etherization is produced. Unless prussic acid is absorbed more rapidly than it is eliminated, no poisonous symptom follows. In certain

[1] On the trial of Elizabeth McCraney, at Otsego, New York, in December, 1860, for the poisoning of Huldah McCraney, the evidence was that the deceased, during her illness, exhibited, in the opinion of several experts, symptoms common to arsenical poisoning, and that arsenic was found in the remains. The case of the prosecution was that the arsenic had been administered by injection in the rectum. There was some conflict among the experts in their opinion as to the cause of death, and the defendant was acquitted. (People *v.* McCraney, 6 Harris C. R. 49, where the medical testimony is reported in full.) That poison may be administered by injection, and be detected by the tests heretofore announced is well established. (Ibid., Taylor on Poisons, 2d Am. ed., pp. 111, 229, 373; Taylor's Med. Jur., 4th Am. ed., 68; Beck's Med. Jur., 11th ed., 447; London Lancet, Dec. 22, 1855; Med. Times and Gazette, Dec. 22, 1855; Edin. Med. Journ., Jan. and Feb. 1856, cited in 6 Parker's C. R. 110.)

animals the elimination of a drug is so rapid that it is extremely difficult to cause death by the administration of this drug. Belladonna is eliminated very rapidly by goats, and it is almost impossible to destroy their life by this drug. The same is true of prussic acid given to horses, etc. The fact must not be lost sight of, that some poisons even in non-poisonous doses may in certain conditions of disease or bodily weakness cause untoward results (*vide* § 3).

§ 9. *The mode of invasion of the symptoms.*—In most cases of acute poisoning, by which is meant those in which a single dose capable of destroying life is taken, the symptoms arise more or less *suddenly*. This, in a great measure, depends upon the rapidity of absorption, because the more rapidly a poison is absorbed the quicker will poisonous symptoms be manifested (*vide* § 8). In the chapters upon the individual poisons, the length of time elapsing before the accession of the symptoms will be given; it varies with each poison, and is influenced also by several circumstances, such as the fulness or emptiness or the healthy and diseased condition of the mucous membrane of the stomach, the state of health and the habits of the individual, as has been already stated. Although arising suddenly, the symptoms do not necessarily follow *immediately* the ingestion of the poison. If it have been swallowed in food or drink, the symptoms announcing the fact of poisoning may not come on for an hour or more afterwards. This fact has been frequently observed in arsenical poisoning, and is usual in poisoning by opium, belladonna, digitalis, and some other narcotics. But when the symptoms have begun to manifest themselves, there is a progressive development of them, and they present (like any disease) certain features, which, combined, form a portrait by which they may be referred to some one class of the poisons, or be known to depend upon a particular poison. It is not, indeed, meant that there may not be a remission of the symptoms in poisoning, due either to the influence of treatment or to the spontaneous struggles of nature; but this circumstance, which is more apt to take place when a dose insufficient for the destruction of life has been taken, can hardly affect the value of the sudden accession and development of characteristic symptoms. It should not be overlooked that poisons have sometimes been taken or administered otherwise than by the mouth, as has been already stated in a previous section.

§ 10. The *duration of the symptoms* is another consideration which has important bearings. Although sudden death is not produced by the majority of poisons, or at least by such as are usually swallowed, yet death from acute poisoning is an early result. A few minutes or hours may suffice; and, on the other hand, the patient may survive for days. No general rule upon this point can be laid down; arsenic usually destroys life within twenty-four hours, strychnine within an hour, opium within twelve hours, and prussic acid in a few minutes. Exceptions are seen to all of these general rules; and with no poison is there so wide a range in the duration of the symptoms as with arsenic, since it has been known to prove fatal in less than two hours, and after several days. The reader will find sufficient details upon this point hereafter.

All the importance of the evidence derived from the symptoms depends upon the possibility of showing a distinction between them and a disease suddenly developed. This distinction should be sufficient, not merely to satisfy the mind of the physician, but to afford convincing proof to the jury upon the subsequent trial. This is often the most difficult and annoying duty of the physician; for while his own mind may be perfectly satisfied of the correctness of his judgment, he can rarely, with perfect conscientiousness, assert that the symptoms might not be explained upon the supposition of disease. Hence, this portion of the medical evidence cannot stand alone, but must be supported either by the positive correspondence with it of the *post-mortem* appearances and chemical analysis, or by the absence of any evidence from the autopsy confirming the notion of disease, and, moreover, it will not answer to place reliance chiefly upon any prominent symptom, but all of the symptoms must be considered as a whole. Thus in poisoning by strychnine the tetanic convulsions caused by this alkaloid might be plausibly ascribed to some other cause, if the convulsions alone were taken into account without reference to their number, the rapidity of their appearance, the condition of the intellect during the interval between the paroxyms, the duration of the case, or any of the other symptoms which contribute to make up the picture of a case of strychnine poisoning. Yet in the case of many poisons it is impossible from the symptoms alone to base evidence sufficiently strong to procure a conviction. Thus, should a person die with the symptoms of irritant poisoning, the physician would find it difficult to defend the

position that similar phenomena might not be witnessed in an attack of cholera, or in gastro-enteritis, arising from some other cause. Dr. Lee says: "During the prevalence of malignant cholera in 1832, we mistook a case of poisoning by arsenic for an attack of this disease. A lady took more than a drachm of the arsenite of potash, as we afterwards ascertained, with the intention of destroying her life, which was followed by severe retching, vomiting, cramps, livid, cold, and clammy skin, and the other symptoms which usually attend a severe attack of cholera."[1]

It is evident that the symptoms observed after poisoning in some cases, as those, for instance, occurring in connection with the action of corrosive or irritant drugs, are caused by primary pathological lesions. Now these lesions may not immediately, that is, within two or three days or even months, result fatally, but their effects, which may cause destruction of tissue, perhaps of some important organ, as the stomach, air-passages, lungs, etc., may give rise to secondary symptoms of disease, which are the direct consequence of the action of the drug. In a similar way account should be made of the slower effects of poisons which cause a deterioration of vital organs, such, for instance, as the fatty degeneration of the tissue of the liver, kidneys, or other viscera by phosphorus, arsenic, and other substances. These alterations of healthy tissue in a vital organ may slowly accomplish the death of the victim, and without the history of the early symptoms the ends of justice might be defeated. In these latter cases especially the *post-mortem* examination may reveal a pathological condition which is not unfrequently observed in disease; but the extensive distribution of the diseased tissue in the case of chronic poisoning, when it is not usually confined to one or two organs, may awaken a suspicion which the experienced pathologist will recognize as unusual in death from natural causes. In such instances an experienced pathologist would lend material assistance in expert testimony, and it must here be observed that the ordinary practitioner of medicine is not by any means an expert pathologist. The knowledge of pathological anatomy requires a large personal experience in autopsies, and microscopical examination of healthy and diseased tissues, as well as a careful educational training to interpret the

[1] Copland's Dict., Am. ed., art. "Poisons."

results of his observation. It may also be further observed that the chemist may be an experienced pathologist and physiologist, and it should be the aim of the judicial investigation, as far as possible, to combine in one person the qualities of a trained pathologist as above stated, though it is evident that this aim cannot always be effective.

§ 11. *The examination of the viscera at the autopsy.*—The pathological condition of these is not alone sufficient to establish a conviction, and a nice discrimination must be made between the effects of disease and those of a poison. The condition of these viscera can only establish a presumption, especially if the autopsy is not made soon after death. And every precaution must be taken in making the autopsy to eliminate any natural cause of death; every organ should be carefully examined, and every abnormal appearance should be carefully noted. Every circumstance, however insignificant it may appear to be at the time, must be noted. The external appearance of the body, whether livid or pale, the expression of the face, whether or not there are any marks of violence or injury, such as contusions or abrasions, the amount of *post-mortem* rigidity, and the exact time after death that the examination is made, must be observed. Not only the appearance of the body, but its exact position in relation to other objects in the room, the temperature of the room, and all other circumstances and conditions, must be remembered by the physician making the examination; and it is best for him not to trust to his memory, but to make a note of everything in writing, together with a plan of the surroundings of the body in any case of suspected poisoning.

Important information can frequently be gained by careful observation of the external appearances of the body and its surroundings; thus the existence of stains upon the skin and mucous membranes about the mouth or upon the clothing, suggests a corrosive poison, and the color of such stains may show the nature of the corrosive; the odor of certain poisons can sometimes be perceived about the mouth and nostrils, as in the case of prussic acid and phosphorus; the mouth and nostrils should also be examined for phosphorescence, which can sometimes be perceived in phosphorus poisoning; the condition of the pupil should be noticed, as in poisoning by atropine it will be found dilated; the color of the *post-mortem* discolorations (suggillations), which are due to blood, will be affected by some

poisons, as, for instance, the bright rose-red in carbonic oxide poisoning, more rarely in prussic acid and cyanide of potassium poisoning, and the dark color in poisoning by the corrosive alkalies; the skin and conjunctivæ sometimes have a yellow color in phosphorus poisoning, and there may be found extensive ecchymoses. If putrefaction has commenced, its exact time should be noticed, since some poisons are apt to hasten while others retard it; as, for instance, in phosphorus poisoning, putrefaction is liable to begin early, while in arsenic poisoning, it is usually delayed, often for a very long time. The time at which the *post-mortem* rigidity appears may also prove an important question, since in some cases, as in strychnine poisoning, it is much hastened and lasts for a much longer time than usual.

In making the autopsy in a case of suspected poisoning, or wherever there is any liability of a legal investigation, special precautions must be adopted in order to guard against the possibility of a mistaken diagnosis. Everything done and seen should be noted at once. Every organ as it is removed from the body should be thoroughly and carefully examined, and, whenever necessary, a portion laid aside for later microscopical examination; for instance, for the detection of the fatty degeneration of the heart, liver, and kidneys so frequently caused by the action of arsenic, phosphorus, and other substances. *All* of the organs, including the brain and spinal cord, should be examined, so as to be sure that death was not due to natural causes. If any urine remains in the bladder, it should be removed and saved for chemical examination. After the organs have been removed from the body, care should be exercised in handling them; for instance, they should not be placed upon undertaker's boards or in wooden receptacles which have been cleaned with disinfecting solutions, lest they absorb minute amounts of arsenic, corrosive sublimate, or other poison, these substances being very common constituents of preserving fluids and disinfectants; the organs should only be placed in glass or porcelain-lined dishes, and the physician should see that these dishes were previously properly cleaned. The autopsy should always be performed in the presence of one or more medical witnesses.

If a chemical analysis for poison is to be made of any of the organs, these organs should be placed by the physician himself in perfectly clean glass jars; glass preserve jars with a glass or por-

celain-lined cover are suitable for the purpose, and can always be obtained in the country or city. Each organ should be placed in a jar by itself; for instance, the stomach in one jar, its contents in another, the intestines in another, contents of intestines in a fourth, the liver in a fifth, the kidneys in a sixth, the brain in a seventh, etc., etc. The organs which should be saved for chemical analysis are, in the order of their importance, as follows: stomach, contents of stomach, liver, intestines, contents of intestines, kidneys, brain, heart, spleen, and urine, if there be any; in some cases it is important to save portions of the muscular tissue, and in others a part of the lungs. In some cases it is wise for the physician making the examination, to divide each of these substances into approximately two equal parts, each part to be kept in separate jars, one to be given to one chemist for preliminary analysis, and the other to be retained by the physician himself, in case it may be necessary to have the analysis confirmed by another chemist, as is usually the case in trials for murder by poison. These jars containing the organs should be closed and sealed by the physician himself, the seal to be stamped with a private stamp. They should then be locked up until they are to be delivered to the chemist. It is better that the organs be placed under double lock, one key to be taken by one person and the other by another, so that neither one alone has access to the organs; this is, of course, not necessary if one person possesses the key, and another has possession of the stamp with which the seals have been stamped. When the jars are to be sent to a chemist, they should be sent by messenger, preferably two messengers, since in the event of the investigation resulting in a trial for murder, the identity of the organs cannot be lost by the death of the messenger. The organs should never be sent by express, since it is, in that case, impossible to preserve with absolute certainty the identity of the organs.

For precautions in regard to exhumations, reference is made to the closing chapter of this volume.

§ 12. It will be well to consider here in brief the question of the investigation of the cause of death by *post-mortem* examination. The theory that professional opinion of a competent and well educated medical man shall prejudge the case without dependence upon a jury of unprofessional men is fast gaining ground in the department of forensic medicine, and, as already stated, has given origin

to a new form of process in the State of Massachusetts. The question then of pathology becomes an important matter to be clearly understood in all judicial inquiries, and should demand more consideration in books on toxicology than has heretofore been given to it. Without, however, attempting to present a treatise on pathology, which would be entirely incomplete and out of place in this work, there are certain facts which should be clearly recited.

First. How should a *post-mortem* examination be conducted? To answer this question the following suggestions are incorporated from the Transactions of the Massachusetts Medico-Legal Society.[1]

"SUGGESTIONS FOR THE MEDICO-LEGAL EXAMINATION OF DEAD BODIES.

REPRINTED FROM THE REGULATIONS ADOPTED BY THE CROWN OFFICE AT EDINBURGH, SCOTLAND, TO BE OBSERVED IN CRIMINAL AND OTHER INVESTIGATIONS.[2]

"In the following suggestions we have not attempted to lay down full and minute instructions for the complete examination of dead bodies in all possible medico-legal cases; because such a plan would involve a statement of many long details, of which every medical man ought to acquire a competent knowledge in the course of ordinary study and practice, and especially in performing ordinary dissections for discovering the signs of disease. We have thought it of more importance to confine our attention to points which are of essential consequence in judicial investigations, or which are apt to be neglected in common dissections.

"It will be remarked that we propose to turn the attention of the medical inspector to some points which are often inquired into,

[1] Vol. i. page 139, Riverside Press, Cambridge, 1879.

[2] Through the courtesy of Professor Douglas Maclagan, of Edinburgh, the Massachusetts Medico-Legal Society has received a copy of the code of regulations governing the proceedings of legal and medico-legal officers in Scotland in their criminal and other investigations. This code includes an appendix containing, among other matters, a series of suggestions for the medico-legal examination of dead bodies; these "suggestions," prepared by three Scotch professors and medical jurists of distinguished and world-wide reputation, are so comprehensive in their character, and are so well adapted to aid Massachusetts medical examiners in the performance of their official duties, that the Committee on Publications takes pleasure in fulfilling the society's instructions to reprint them in this form.

not by him, but by magistrates or other official persons, not of the medical profession—such as the place where the body is found, its position when first seen, surrounding objects, the clothes, the history of cases of suspected poisoning, and the existence of poison in a house, etc. This we have been led to do, because we have had occasion to observe that on such points important articles of evidence have been overlooked, owing to the absence of a medical man, to whom alone their importance would have been apparent. On the whole, however, such matters would probably be best investigated by a law officer with the aid of a medical man.

I. General Duties.

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"(4) It is desirable that the medical inspectors shall have an opportunity of viewing the body before it is undressed, or moved from the spot where it was first found. If the body has been previously removed or meddled with, they ought to inform themselves accurately as to its original position. In many cases, it is material that they personally visit the place where it was first seen; and they should inquire minutely into all the particulars connected with the removal of it.

"(5) In cases where the body has been buried, and disinterment becomes necessary, it ought not to be removed from the coffin except in presence of the inspectors. In any case where subsequent investigations may involve a search for poison in an exhumed body, one or two pounds of the soil immediately above the coffin should be secured for analysis.

"(6) When a considerable period has elapsed between death and disinterment, the inspection must, in all cases, be proceeded with, although the body be found in a state of decay, unless the inspectors can positively say that the progress of decay is such as to render the examination nugatory in relation to its special objects. The degree of decay which will justify such an opinion will differ with a variety of circumstances which cannot be properly specified here. It may be observed, however, that where injuries of the bones are to be looked for, or the traces of certain poisons, it is scarcely possible to assign the limit at which an inspection must of necessity be fruitless. It is of moment to remember that the internal organs are often in a great measure entire, although the exter-

nal parts are much decayed. The dissection, where the body is putrid, will be rendered less annoying to those present by sprinkling the surface of the body with a solution of chloride of lime (one part in forty), or of carbolic acid (one part in thirty); but none of these disinfectants should be applied after the dissection is begun, especially in cases where there may afterwards be an examination for poisons.

"(7) No one should be allowed to be present at the examination out of mere curiosity; and we recommend that every one not engaged in the inspection, but who is in attendance to give information, or for any other purpose, and who may afterwards become a witness, should remain in an adjoining room. The medical inspector often furnishes good tests of the value of other evidence in the case; and it is therefore desirable that the general witnesses should not have an opportunity of knowing what is observed in the dissection of the body.

"(8) The examination and dissection of the body should not, if possible, be commenced without sufficient daylight in prospect to allow the whole inspection to be made without artificial light.

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"(13) When any portions of the body, or any substances found in or near it, are to be preserved for further examination, they ought never be out of the custody of the inspectors or of a special law officer. They must be locked up in the absence of the person who keeps them. When they are to be transmitted to a distance (which is to be done by the hands of an authorized individual, not by sending them by a public conveyance), the vessels containing them should be secured and sealed upon the spot. A label ought to be attached to each vessel, bearing a list of the articles therein contained, and dated and signed by the inspectors. Narrow-necked vessels and bottles may be secured by corks and wax impressed by a seal; wide-mouthed jars and other vessels, if large corks cannot be had, are best secured by tying them over with sheet gutta-percha, fortified, if requisite, with leather. It is important to observe that the covers are securely tied and sealed, so that the coverings cannot be removed and replaced without either cutting the strings or breaking the seals. . . . . .

III. External Aspect and Examination of the Body.

"(16) The importance of the external examination and the particulars to be chiefly attended to in performing it will vary in different cases with the probable cause of death. It comprehends an examination: 1. Of the position of the body when found, as well as of all external injuries or marks presented by it. 2. Of the vicinity of the body, with a view to discover the objects on which it rested, or from or upon which it may have fallen; marks of a struggle; signs of the presence of a second party about the time of death or after it; weapons or other objects, the property or not the property of the deceased; the remains of poisons; marks of vomiting; and where marks of blood are of importance and doubts may arise as to their really being blood, the articles presenting them must be preserved for further examination. 3. Of the dress, its nature and condition, stains on it of mud, sand, or the like, of blood, of vomiting, of acids, or other corrosive substances; marks of injuries, such as rents or incisions. Where injuries have been inflicted on the body, care should be taken to compare the relative position of those on the body and those on the clothes; and where stains, apparently from poison, are seen, the stained parts are to be preserved for analysis. 4. Of ligatures, their material and kind, as throwing light on the trade of the person who applied them, the possibility or impossibility of the deceased having applied them himself, their sufficiency for accomplishing their apparent purpose, etc.

"(17) Inspectors will commence the examination of the body itself by surveying the external surface and openings. Before cleaning it, they will examine it on all sides, not neglecting the back, as is often done, and look for marks of mud, blood, ligatures, injuries, stains from acids and the like, foreign bodies, or injuries within the natural openings of the body, namely, the mouth, nostrils, ears, anus, vagina, and urethra. If there are impressions of finger-marks, they will consider which hand produced them. If there be any doubt about stains being blood, the skin presenting them must be preserved for analysis. If there be acid stains, or other probable remains of poison, or any foreign matter, the nature of which may require to be determined by analysis, these must also be preserved. The ordinary places for the impressions of ligatures

are the neck, the wrists, the ankles, and the waist. The degree of warmth of the trunk and extremities, the presence or absence of cadaveric rigidity, and whether it exists equally in the upper or lower extremities, should be noted in this stage of the proceedings; in other cases, the progress of putrefaction, as indicated by the odor of the body, the looseness of the cuticle, the color of the skin and formation of dark vesicles on it, the evolution of air in the cellular tissue, the alteration of the features, the softness of the muscles, the shrivelling of the eyes, the looseness of the hair and nails.

"(18) In this part of the examination, it will sometimes be necessary to observe the particulars by which the body may be identified. These are numerous; but the most important are the stature, the degree of plumpness, the size and form of the nose and mouth, the color of the eyes and hair, the state of the teeth, warts, nævi, deformities, scars of old abscesses, ulcers, and wounds.

"(19) The body is next to be washed, if necessary, and the hair of the head shaved, or at least closely cut; and a thorough examination of the whole integuments is to be made. At this stage, the inspectors will look particularly for the appearance of lividity, noting its chief seat and its relation to the posture in which the body was found; for impressions on the skin of objects on which it had rested; for marks of injuries, more especially contusions, taking care to ascertain their real nature by making incisions through the skin; for marks of disease, such as eruptions, ulcers, and the like; for marks of burning; for marks of concealed punctures in the nostrils, mouth, external opening of the ears, the eyes, the nape of the neck, the armpits, the anus, the vagina, and beneath the mammæ or scrotum; in infants, also in the fontanelles and the whole course of the spine. At this stage, wounds and other injuries should be carefully examined according to the directions given in Division V. (*Infra.*) Where the injury may have caused loss of blood, the presence or absence of pallor in the skin, lining membrane of the mouth and the gums, ought to be noted.

IV. Dissection or Internal Examination of the Body.

"(20) In commencing the dissection of the body, it must be laid down as an invariable rule that all the great cavities should be examined, and also every important organ in each, however dis-

tinctly the cause of death may seem to be indicated in one of them. It is right to examine the cavity of the spine, and, at all events, its upper portion, in any case where an unequivocal cause of death has not been discovered elsewhere.

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"(22) The inspectors should begin with that cavity over which there is a wound, or other mark of injury. Or, if there be an injury on the extremities, the dissection ought to commence there. In the absence of any such guide, that cavity should be taken first where the circumstances of death, so far as they are ascertained, may lead the inspectors to expect unusual appearances. In other cases it is best to lay open the chest and abdomen, to take a general survey of the parts exposed without disturbing them materially; or to proceed to the head, which may be examined thoroughly in the first instance; afterwards to examine carefully the chest and belly; and the spine may be reserved till the conclusion. Wherever unusual appearances are discovered in the first cursory survey, the anatomical examination ought, in general, to be begun there.

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"(29) It is necessary to examine the pharynx and gullet, the larynx, trachea, and its greater ramification, the lungs, the heart, and the great vessels with particular care, because here are most frequently found the causes of sudden natural death. In examining the heart, each auricle and each ventricle ought to be laid open by an independent incision of its parietes, and this should not intersect any of the valvular openings of the septum cordis.

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"(31) In inspecting the organs in the chest, a cursory examination should be first made by turning them over, ascertaining the nature, and measuring the quantity of effused fluids, feeling for tumors or other diseases, and opening the pericardium to obtain a view of the heart. The most convenient course to pursue next is, without moving the heart from its place, to lay open its several cavities, in order to judge of the quantity and state of the blood in both sides of that organ, and then to remove the whole organs in the chest, namely, the lungs, heart, and gullet, together with the parts dissected downward from the throat, in one mass, and to examine them in detail on a table. But previously a ligature should

be applied on the gullet, just above the cardiac orifice of the stomach.

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V. Examination in Cases of Wounds and Contusions.

"(33) In a post-mortem examination, the most approved mode of examining these injuries is, if they be situated over great cavities, to expose the successive structures in the manner of an ordinary dissection, observing carefully what injuries have been sustained by the parts successively exposed before they are divided. Wounds ought not to be probed, especially if situated over any of the great cavities. The depth of a wound is best ascertained by careful dissection and exposure of the parts involved; but, after this is done, the thickness of the tissues penetrated may be measured by the probe.

"(34) The seat of the wounds must be described by actual measurement from known points, their figure and nature also carefully noted, and their direction ascertained with exactness.

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"(36) Apparent contusions must be examined by making incisions through them, and the inspectors will note whether there be swelling or puckering of the skin; whether the substance of the true skin be black through a part or the whole of its thickness; whether there be extravasation below the skin or in the deeper textures, and whether the blood be fluid or coagulated, generally or partially; whether the soft parts below be lacerated, or subjacent bones injured; and whether there be blood in contact with the lacerated surfaces. By these means the question may be settled whether the contusions were inflicted before or after death.

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"(39) In the course of the dissection of wounds, a careful search must be made for foreign bodies in them. When fire-arms have occasioned them, the examination should not be ended before discovering the bullet, wadding, or other article, if any, lodged in the body; and whatever is found must be preserved. When the article discharged from fire-arms, or when any other weapon, has passed through and through a part of the body, the two wounds must be carefully distinguished by their respective characters, especially as regards their comparative size, inversion or eversion, smoothness or

laceration of their edges, their roundness or angularity, and the comparative amount of bleeding from each. In gunshot injuries, the presence or absence of marks of gunpowder should be noted.

"(40) When wounds are situated over any of the great cavities, they ought not to be particularly examined till the cavity is laid open; and in laying open the cavity, the external incisions should be kept clear of the wounds.

"(41) When the discolored state of a portion of the skin is such as to render it doubtful whether it is due to injury or to changes after death, an incision should be made to ascertain whether there is blood effused into the textures, constituting true *ecchymosis*, or merely the gorging of the vessels of the skin, or discoloration from infiltration of the coloring matter of the blood, which takes place in depending parts of a dead body. The term *suggillation* should be avoided, as it has been used in opposite senses by Continental and British authors. The respective expressions 'discoloration from extravasated blood' and 'lividity after death' are preferable.

VI. Examination in Cases of Poisoning.

"(42) In examining a body in a case of suspected poisoning, the inspectors should begin with the alimentary canal: first, tying a ligature round the gullet, above the cardiac orifice of the stomach, and two round its pyloric end; then, removing the stomach and whole intestines; next, dissecting out the parts in the mouth, throat, neck, and chest in one mass; and, finally, dissecting the gullet, with the parts about the throat, from the other organs of the chest. The several portions of the alimentary canal may then be examined in succession.

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"(44) The medical inspectors may afford most important aid to the law officers in investigating the history of cases of supposed poisoning. For this purpose minute inquiry should be made into the symptoms during life,—their nature, their precise date, especially in relation to meals or the taking of any suspicious article, their progressive development, and the treatment pursued. It is impossible to be too cautious in collecting such information; and, in particular, great care must be taken to fix the precise date of the first invasion of the symptoms, and the hours of the previous meals. The same care is required in tracing the early history of the case, when the

inspector happens to visit the individual before death ; and, if suspicions should not arise till his attendance has been going on for some time, he ought, subsequently to such suspicions, to review and correct the information gathered at first, especially as to dates. All facts thus obtained should be committed to writing.

" (45) Besides inspecting the bodies and ascertaining the history of the case, the inspectors may afford valuable assistance to the law officers in searching for suspicious articles in the house of the deceased. These are: suspected articles of food, drink, or medicine ; the vessels in which they have been prepared or afterwards contained ; the family stores of the articles with which suspected food, etc., appears to have been made. All such articles must be secured, according to the rules in Section 13, for preserving their identity. In this examination the body, clothes, bed-clothes, floor, and hearth should not be neglected, as they may present traces of vomited matter, acids spurted out or spilled, and the like.

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" (48) The whole organs of the abdomen must be surveyed, but particularly the stomach and whole tract of the intestines, the liver, spleen, and kidneys, the bladder, and, in the female, the uterus and its appendages. The intestines should in general be slit up throughout their whole length ; and it should be remembered that the most frequent seat of natural disease of their mucous membrane is in the neighborhood of the ileo-cæcal valve, and that, next to the stomach, the parts most generally presenting appearances in cases of poisoning are the duodenum, upper part of the jejunum, lower part of the ileum, and rectum.

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" (51) The stomach and intestines should be taken out entire, and their contents emptied into separate bottles. If the stomach or part of the intestines present any remarkable appearance, examination may be reserved, if convenient, till a future opportunity ; but in every instance, it must be preserved and carried away, as it may itself be an important article for analysis. The throat and gullet may be examined at once, and preserved with their contents, which, if abundant, may be kept apart in a bottle. In addition to the alimentary canal and its various contents, portions of the solid organs of the body ought to be secured for analysis. The most important are the liver, spleen, and kidneys. A part of the liver, at least a

fourth part, should be secured in every case of supposed poisoning; and in cases where the fatal illness has been of long duration, and therefore only traces of the poison may remain in the body, the whole liver, the spleen, and both kidneys should be secured. A portion of the blood, especially when the odor of any volatile poison is perceived, should be at once put into a bottle, closed by a good cork or stopper.

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VII. Examination in Cases of Suffocation.

"(54) In cases of suspected drowning, the inspector will observe particularly whether grass, mud, or other objects be clutched by the hands, or contained under the nails; whether the tongue be protruded or not between the teeth; whether any fluid, froth, or foreign substances be contained in the mouth, nostrils, trachea, or bronchial ramifications; whether the stomach contain much water; whether the blood in the great vessels be fluid. When water with particles of vegetable matter or mud is found within the body, these must be compared with what may exist in the water in which the body was discovered, and should be preserved for further scientific investigation, if requisite. Marks of injuries must be compared diligently with objects both in the water and on the banks near it, and especial attention given to the question whether any observed injuries had been sustained by the body before or after death.

"(55) In cases of suspected death by hanging, strangling, or smothering, it is important to attend particularly to the state of the face as to lividity, compared with the rest of the body; the state of the conjunctiva of the eyes as to vascularity; of the tongue as to position; of the throat, chin, and lips, as to marks of nail-scratches, ruffling of the scarf-pin, or small contusions; the state of the blood as to color and fluidity; the state of the heart as regards the amount of blood in its several cavities; the state of the trunk and branches of the vena cava in the abdomen as regards distension with blood; and the state of the lungs as regards congestion, rupture of any of the air-cells, and small ecchymoses under the pleura or the pericardium. The mark of a cord or other ligature round the neck must be attentively examined; and here it requires to be mentioned that the mark is often not distinct till seven or eight hours after death, and that it is seldom a dark, livid mark, as is very commonly supposed, but a

pale, greenish-brown streak, presenting no ecchymosis, but the thinnest possible line of bright redness at either edge where it is conterminous with the sound skin. Nevertheless, effusions of blood and lacerations should be looked for under and around the mark, in the skin, cellular tissue, muscles, cartilages, and lining membrane of the larynx and trachea. Accessory injuries on other parts of the body, more especially on the chest, back, and arms, must also be looked for, as likewise the appearance of blood having flowed from the nostrils or ears, and the discharge of feces, urine, or semen. In cases where death may be due to the emanations from burning fuel or other poisonous vapors, a small vial should be filled with the fresh blood and securely corked, for further investigation, if requisite.

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IX. Examination in Cases of Criminal Abortion.

" (57) In suspected criminal abortion, when the woman survives, the chief points for inquiry are: the proofs of recent delivery, the ascertaining of facts tending to show that she has been subjected to manœuvres with instruments, and the occurrence of symptoms traceable to the action of any of the drugs reputed as capable of causing abortion.

" When the woman has died, the points requiring special attention at the dissection are: the state of the womb as regards its size, and the condition of its lining membrane in reference to the probable period of delivery; the condition of the intestinal canal in reference to the action of irritant drugs; of the mucous membrane of the bladder in reference to the action of cantharides; close inspection of the womb and vagina in reference to mechanical injuries, especially punctured wounds; and any appearances that the death may have been caused by inflammation in the organs of the pelvis, or by bleeding from the womb.

X. Examination in Cases of Infanticide.

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" (59) The inquiry in cases of infanticide should be conducted with reference to the five following distinct questions: 1. The probable degree of maturity of the child. 2. How long it has been dead. 3. Whether it died before, during, or after delivery, and how long after. 4. Whether death arose from natural causes,

neglect, or violence. And, 5. Whether a suspected female be the mother of the child.

"(60) The points to be attended to for ascertaining the probable degree of maturity of the child are: the general appearance and development; the state of the skin, its secretions, and its appendages—the hair and nails; the presence or absence of the pupillary membrane; the length and weight of the whole body; whether the navel corresponds or not with the middle of the length of the body; the situation of the meconium in the intestines; the site of the testicles in the case of males, and, in either sex, the size of the point of ossification in the lower epiphysis of the thigh-bone. This latter is easily observed by making an incision across the front of the knee into the joint, pushing the end of the thigh-bone through the cut, slicing off the cartilaginous texture carefully until a colored point is observed in the section, and then by successive very fine slices ascertaining the greatest diameter of the bony nucleus. This does not exist previous to the thirty-sixth week of gestation, and in a mature child is about one-fourth of an inch in diameter.

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"(62) The circumstances which indicate whether the child died before, during, or after parturition, and how long after it, are: the signs of its having undergone putrefaction within the womb; the marks on the crown, feet, buttocks, shoulders, etc., indicating presumptively the kind of labor, and whether it was likely to have proved fatal to the child; the state of the lungs, heart, and great vessels, showing whether or not it had breathed; the nature of the contents of the stomach and intestines; the presence of foreign matters in the windpipe; the state of the umbilical cord, or of the navel itself, if the cord be detached.

"(63) In order to ascertain properly the state of the lungs, heart, and great vessels, with a view to determine whether or not the child has breathed, the inspection should be made in the following order: Attend first to the situation of the lungs, how far they rise along the sides of the heart, to their color and texture, and whether they crepitate or not. Then secure a ligature around the great vessels at the root of the neck, and another around the vena cava above the diaphragm. Cut both sets of vessels beyond the ligatures, and remove the heart and lungs in one mass; this mass must be weighed, and put into water, to ascertain whether the

lungs, with the heart attached, sink or swim. In the next place, put a ligature around the pulmonary vessels, close to the lungs, and cut away the heart by an incision between it and the ligatures. Lastly, ascertain the weight of the lungs; whether they sink or swim in water; whether blood issues freely or sparingly when they are cut into; whether any fragments swim in the instances where the entire lungs sink; and, in every instance of buoyancy, whether fragments of them continue to swim after being well squeezed in a cloth.

"(64) The general question to be considered in relation to the cause of death is whether the appearances are such as to be traceable to the act of parturition, or whether they indicate some form of violent death. The directions given in Divisions V., VI., and VII. apply to infants as well as adults, but the following points are specially to be noticed in cases of supposed infanticide:—

"In relation to wounds and contusions, the possibility of minute punctured wounds of the brain or other vital organs; in reference to injuries of the head, the effusion of blood under the scalp not in the situation where it could have been produced during labor, or fracture of the bones not producible by compression of the head during labor, and not connected with defective ossification of the skull; in reference to the allegation that the head was injured by the child suddenly dropping from the mother when not recumbent, the presence of sand or other foreign matters on the contused scalp, and the existence of more than one injury of the head; in relation to suffocation, the external and internal signs of this form of death, marks of compressions of the mouth and nose or throat, and the presence of foreign matters in the mouth and throat, air-passages, gullet, or stomach, especially if the body be found in contact with similar substances; in reference to bleeding from the navel-string, a bloodless state of the body, without any wound to account for it; in reference to poisons, most commonly narcotics, the absence of any of the above appearances, with an otherwise healthy state of the body; in reference to starvation and exposure, emaciation of the body, absence of food from the stomach, and an empty contracted condition of the intestines. In reference to the possibility of the child having been suddenly expelled, and having fallen on the floor, or into privies, etc., the state of the navel-string is to be noted, whether long or short, whether remaining attached to the

child and connected with the after-birth, indicating rapid labor, or, if divided, whether it has been cut or torn across.

"(65) The circumstances noticed in §§ 60, 62, 63, and 64, compared with the signs of recent delivery in the female, will lead to the decision of the question whether the suspected female be the mother of the child. The circumstances may be shortly recapitulated as being the signs of the degree of maturity of the child; the signs on the body of the kind of labor; the signs which indicate the date of its death, and the interval which elapsed both between its birth and death, and between its death and the inspection.

R. Christison.
James Syme.
Douglas Maclagan."

§ 13. Second. *Rigor mortis or cadaveric rigidity* is due to a *post-mortem* irritability inherent in the muscles, the effect of which is to produce a firm and continuous contraction in their length; usually the contraction first commences in the muscles of the jaw and thence extends downwards. In the more robust individuals this cadaveric rigidity sets in earlier than in those who have died from a long and lingering illness.

It is not reasonable to attempt to lay down any specific rules which govern either the early appearance of this *post-mortem* sign, its duration, or its time of disappearance. It is still more unreasonable to attach any very great specific value to the time of its appearance, its duration, or time of disappearance after death as a means of determining the kind of poison used for destroying life. In spite, however, of this view of the question, the conditions of cadaveric rigidity have a certain circumstantial value in connection with symptoms which precede death by poison. In general terms this statement should be allowed, viz., that the appearance of *post-mortem* rigidity depends upon the inherent irritability of the muscular tissue. When death has been preceded by violent exercise of this inherent muscular irritability, the *post-mortem* rigidity sets in early and lasts but a short time, because the muscular irritability has been more or less exhausted. By way of illustration the following observation was made by the writer.[1] A brakeman who had been in muscular activity just before death, and was coupling together two railroad

[1] Reported in Boston Med. and Surg. Journal, Dec. 4, 1873.

coaches having platforms of unequal height, was suddenly compressed by the backward motion of the engine. Judging by the circumstances the platform of the forward car struck him on a level just below the diaphragm, whilst the platform of the other car struck him behind about four inches higher. Death was instantaneous. "I arrived about twenty minutes after the accident, and found the body of the man (which had been disengaged from the position of the accident at the time of the compression) lying upon its back in a state of commencing *post-mortem* rigidity." The muscular rigidity was only present in the head and trunk, not yet having reached the lower portions of the body, though six hours afterwards the whole muscular system was in a state of strong contraction. When first seen after the accident, the right arm was extended upwards in a state of semiflexion, and the left arm was stretched out from the body, slightly flexed at the elbow-joint, in fact, in just the position assumed for dropping the connecting pin into its socket with one hand, while the other was held up to signal the conductor when the car was connected. This single observation illustrates the question of the early appearance of *post-mortem* rigidity which is sufficiently common in military surgery,[1] especially after wounds apparently instantaneously fatal, as of the head and heart. Dr. Brinton remarks, "I have seen bodies rigid in their last attitude, twenty-four, forty-eight, and in one instance sixty hours after death. M. Armand, at Magenta, saw this rigor twenty-four hours, and M. Perier, at Alma, forty-eight hours after death. The fact of the prolonged continuance of this rigor would seem to militate against the idea of its tetanic nature, and of its being followed in turn by flexibility and by rigor mortis proper." On the other hand, probably from observations made upon persons and animal skilled only by poisons, M. Tardieu[2] is misled into making the supposition that convulsifying poisons induce a specific effect upon the time of appearance and permanence or duration of *post-mortem* rigidity, while those which relax the muscular system before death have a contrary effect.

The literature of the action of poisons on *rigor mortis* is volumi-

[1] See an interesting communication "On Instantaneous Rigor, as the Occasional Accompaniment of Sudden and Violent Death," by John H. Brinton, American Journal of Medical Sciences, Jan. 1870.

[2] Op. cit.

nous and contradictory,[1] and opinions based upon conclusions vouchsafed by French authorities should be taken with great caution, and only after careful study of the facts related by them in support of these conclusions.

M. Rondeau[2] draws from his own experiments, which seem well founded and carefully performed, the following conclusions:—

"1st. Contrary to the opinion of Hunter, muscular rigidity occurs after death by lightning; its appearance and duration are very variable.

"2d. Chloroform, whether by its introduction into the stomach or lungs, appears to prolong the duration of rigor mortis, though it has no apparent influence upon its production.

"3d. Phosphorus has no definite action upon the conditions of rigor mortis.[3]

"4th. Morphine and laudanum—no facts worthy of note.

"5th. Cyanide of potassium appears to have a definite effect upon the time of appearance and duration of *post-mortem* rigidity." It usually appears in a few hours, and persists for two or three days.[4]

"6th. Arsenic does not modify *post-mortem* rigidity.

"7th. Oxalic acid apparently hastens the appearance of rigidity, which is preserved for a relatively long time."

8th. . . . Does not concern the question of rigidity.

"9th. Salicylate of sodium apparently causes the early appearance and a very rapid disappearance of rigor mortis.

"10th. Sulphate of quinine, according to Niderkorn, causes an early and somewhat prolonged appearance of rigor mortis, during which decomposition rapidly goes on."

11th and 12th have no conclusive bearing upon this subject.

"13th. Death by drowning does not modify rigidity.

"14th. A fœtus born dead is in a state of muscular rigidity."[5]

[1] See a thesis, No. 396, 1880, Paris, by Pierre Rondeau, Etude Expérimentale sur la Rigidité Cadavérique au point de vue Medico-legal.

[2] Op. cit.

[3] Tardieu presents examples which seemed to him to suggest a law of cause and effect, but does not insist upon its terms.

[4] See also a paper by the writer published in the Boston Med. and Surg. Journ., Aug. 23, 1866.

[5] The experiments related by the above author were made under variable conditions of temperature from (6° C.) 42° F. to (23° C.) 64° F.

§ 14. *Sources of error arising from natural changes in the body after death.*—Having thus shown the chief means of distinction between the effects of poison and of some of the natural diseases to which the human frame is subject, it only remains for us to point out some sources of error which the natural changes taking place in the body *after* death may give rise to. This important subject is one which has received but little attention at the hands of medical jurists, but there can be no doubt that the *natural* appearances of those parts of the body usually inspected after death, where poisoning has been attempted or is alleged, are often mistaken for pathological changes induced by the administration of poison. Since the publication of the first edition of this work, the knowledge by pathologists of the marks of disease as distinct from those due to natural *post-mortem* appearances has become so well established, that the medical witness, whether he be a trained expert or a well-qualified practitioner, ought to be able to show clearly whether the *post-mortem* appearances in a given case are natural or abnormal, whether due to natural causes or the marks of disease: the *post-mortem* appearances due to natural causes would not be likely to mislead in an investigation of cases of suspected poisoning.

First. *Color of the skin.*—In order to obtain a definite knowledge of the complexion of the skin, the surface should be clean. The effects due to atmospheric exposure during life will, of course, be apparent in those parts not ordinarily protected by clothing. A peculiar bronze hue, even if it extends to the mucous surfaces, does not always indicate disease of the supra-renal capsules, commonly known under the name of "Addison's disease," and, therefore, no conclusion should be drawn from the color of the skin only; it may be yellow colored from the presence of the bile in the circulation during life, and "if the jaundice be of long duration and very intense, the discoloration may be dark-yellow or even nearly black."[1] The use of nitrate of silver, as a medicine during life, produces a grayish or slate-colored complexion. A waxy-yellow color of the skin indicates a state of anæmia during life, or may be the result of hemorrhage before death, or of blood poisoning from an abscess or other definite disease.

The changes of color resulting from *post-mortem* decomposition

[1] Orth, op. cit.

are: (*a*) a greenish hue which appears earliest on that portion of the surface adjacent to the viscera; (*b*) a livid-reddish hue of the underlying portions of the body, or this discoloration may be seen in the subjacent tissue in the form of *suggillations* ("maculæ emortuales"), or spots in which the blood, escaping from its vessels, lies in a non-coagulated form just beneath the external surface, or in a "*boggy*" tissue. Some of these spots may be due to the position of the body causing a natural gravitation of the blood, while others may be in consequence of the escape of the blood-coloring matter into the neighboring tissues. A discussion as to whether these spots are ecchymoses, or *ante-mortem*, will be more fully presented in the chapters on surgical injuries. It is sufficient here to note that a discoloration due to gravitation usually disappears on pressure, whilst that due to diffusion will not so disappear.[1] These *post-mortem* discolorations never give rise to elevation of surface as do those inflammatory spots occurring during life.

SECOND. *Visceral decomposition.*—The contents of the stomach may be acid from the fermentation of food, as for instance milk, which may soften the walls, and even digest them as has been before stated, and so mislead the observer to suppose that some corrosive poison had been administered previous to death; a chemical analysis would very readily solve the doubt, but if the digestion be due to a mineral acid, it would also corrode the stomachal contents and adjacent tissues. Besides these changes due to the quantity and character of the contents of the stomach, which changes are mostly confined to the larger curvature of the organ, from the fact that the food lodges there, it must be observed "that the least degree of acidity produces a gray opacity; when greater, a sort of digestion of the mucous membrane itself follows, so that it is converted into a soft, slimy, transparent mass, which is easily scraped off, and then the submucous tissue, or muscular layer, is laid bare. Finally, the softening may go still further, and involve the muscular and serous coats, when the already described softening of the stomach results. If this occurs in a stomach free from blood, it is termed *white softening;* if, on the other hand, the vessels are filled with blood, this is affected by the acid, so as to present a brown or brownish-black color, the neighboring parts are infiltrated with the coloring matter,

[1] Orth, op. cit.

and a soft, more or less brown, mass results,—*brown softening* of the stomach. A dirty-green color is due here, as in most other organs, to actual putrefaction."[1]

In contrast with these appearances, the results of inflammatory processes in the stomach are quite distinct, and are carefully laid down in the latest works on pathological anatomy; these are acute and chronic catarrh, parenchymatous inflammation, and that of the submucous tissue. The parenchymatous form also occurs in poisoning from phosphorus and arsenic, and, as has been already observed, is associated with similar appearances of the tissues of other viscera. Therefore, in this form of general degeneration of organs observed at the autopsy in the case of a suspicious death, it would be advisable to make a chemical examination for poison.

Still we cannot omit commenting upon a certain *post-mortem* appearance which has been referred to in works on forensic medicine, especially in France, and ascribed to sudden and violent death by asphyxia, as well as by certain poisons. This appearance has been called subpleural ecchymosis. Dr. Legroux has recently investigated the nature of these hemorrhagic spots, and in a report to the Socié·é de Mélecine de France,[2] and also to the Congrès International de Médecine Legale of 1878, has presented this matter so much in detail that its omission here would not be admissible. Their special location seems to be on the free borders of the lobes of the lungs, especially the lower lobes, where the pleural surface glides over the diaphragm, on the surface adjacent to the diaphragm, or that which is contiguous to the lobes, less often on the convex anterior surface, and more rarely still at the apex of the lungs. The dimension of these ecchymotic spots is variable, from an almost imperceptible point to that of a lentil (taches lenticulaires), or of a ten-cent or twenty-cent silver piece. There may be only one or two, or as many as four or five, or even twenty, upon the whole pulmonary surface. These bloody spots are disposed separately on the different localities above mentioned without any particular relation to each other, or may be grouped in small numbers by islets of four or five, some of which may fuse into others; occasionally they are so small and numerous, that the pleura has a marbled look, like the marbled wood which painters

[1] Orth, op. cit.

[2] Bull., tome v., 1879, p. 283.

form by spattering paint from a brush on a neutral tint. If the lung be carefully examined, after its surface has been washed and wiped, it will be seen that neither pressure, washing, nor scraping can obliterate these spots; yet in animals freshly killed, if the lungs are artificially inflated, when the heart is cut into they are dissipated; if, however, the *post-mortem* examination be not made until some time after death, no artificial means can make these hemorrhagic spots disappear, and if the blood has lost its fluidity, and decomposition has progressed, they are even more distinct. They can be distinguished from the false ecchymotic spots so often observed in autopsies made some time after death, by the disappearance of these latter simply by inflating the lungs.

When cut into, the true subpleural ecchymotic spot is found thin, and scarcely penetrates more than a quarter or half a millimeter into the lung tissue. Looked at under the microscope the pleura seems elevated or detached from the alveoli, and the blood-corpuscles in the tissue surrounding these alveoli seem squeezed against each other. The subpleural suffusions which accompany pulmonary apoplexy must not be confused with these subpleural spots. They are not supposed to follow death from bronchitis, asthma, or pleurisy, but are associated with "a rapid death which has taken the organism by surprise in a state of normal health or apparently so."

According to Tardieu and other observers, it seems well established that rapid death from phosphorus, arsenic, mercury, lead, and digitalis are likely to be followed by this *post-mortem* appearance. Dr. Legroux would arrange the cause of these ecchymoses in the same class with that which produces petechiæ of the skin in typhus and typhoid fevers, cholera, purpura, and hemorrhagic variola. These spots may be observed in any case of death which occurs suddenly, in ten or fifteen minutes; MM. Tardieu, Depaul, Tarnier, and other obstetricians have observed them in the fœtus which has been exposed by violence to a sudden death, as in pelvic malformations of the mother, or after certain obstetrical operations practised before birth on the fœtus, such as craniotomy.[1]

These subpleural spots, therefore, form certain positive signs of an abrupt and sudden death, and are not necessarily confined to any particular poison administered, but rather to the manner of

[1] See also a thesis of Dr. Dechondeau, Thèse de Paris, 1878, No. 420.

death which has occurred to a person previously in robust health, and unaffected with active disease of any organ of the body.

It would be a difficult and useless task in a work of this scope to enter into further detail in regard to the pathological anatomy of the body, and reference is here made to such works as Orth's, which has been frequently referred to, Virchow's, Rokitansky's, and Sieveking's. Where pathological appearances are due to a specific poison, these will be detailed under the head of that poison.

§ 15. In the examination of the *stomach* it will be found that it presents variable appearances, according as it is inspected during or after the process of digestion, or after long fasting; whether that organ be empty or full, distended or contracted; and whether the distension is due to liquid or to gas.

During and immediately after *digestion*, the stomach is filled with gas and the remains of the food, and is, therefore, moderately distended, and its mucous membrane appears thin and does not lie in folds. Its color is of a pale rose, uniformly spread over the surface, or, if the organ is unusually distended, it is gray or dirty-white. On the other hand, in the *fasting* condition it is strongly contracted, and the mucous membrane is corrugated and thick. Its color is of an ashen-gray when it is covered with mucus, but when this is not the case, it is of a reddish-brown. It may be partly contracted and partly distended, in which event, the differences referred to will be visible at the same time in the pyloric and cardiac portion. Moreover, the cadaveric hypostasis, or settling of blood, will be seen on the folds of the mucous membrane, or in those portions of the organ which are the least distended. After the process of digestion is entirely completed, the abdominal system of veins is loaded with blood, and the same engorgement occurs in certain diseases of the heart and lungs; should death take place at such a time, the mucous membrane of the stomach is found highly injected, and, in consequence of the transudation of liquids taking place in the dead body, ecchymoses are formed which often have the appearance of submucous extravasations; they frequently occupy the entire half of the stomach and both curvatures, and have a bluish-slate color. This injection may also occur in streaks, and thus give rise to an unfounded opinion that death was due to some irritant. This is especially the case where powdered substances, such as arsenic, are found near them, but it is a mere coincidence,

since the existence of folds or rugæ is a sufficient explanation of the adhesion of the powder to these places.[1]

The softening of the mucous coat after death is of course seen in lesser degrees, and most probably is purely a cadaveric change depending upon the solvent powers of the liquids contained in the organ. When the mucous coat is found apparently thickened, this condition is often due merely to the stomach being in a contracted state; and, on the other hand, it may appear to be very thin when the appearance is solely due to its distension. Similar sources of error to those we have thus cursorily noticed arise in the inspection of the brain, heart, and other organs. The physician should be upon his guard against them, and carefully distinguish the changes produced by disease from those which are brought about in the act of dying, or after death, by the position of the body and the transudation of liquids. If familiarity with the ordinary *post-mortem* appearances does not enable him to form a positive opinion as to the cause of death, it is far better that he should have the candor to say so, than, by giving an unwarranted opinion, incur the risk of causing the innocent to suffer. But in every case it is proper that precise and accurate language should be used in the description of *post-mortem* appearances, and that such expressions as inflammation, gangrene, etc., which imply the manner in which the morbid change has resulted, should not be used, but rather, instead, such terms as will simply express the *physical* condition of the part, in reference to its size, color, consistence, etc.

§ 16. From the foregoing remarks it is, we think, apparent that the most perfect evidence of poisoning is derived from the combined results of the investigation of the symptoms, *post-mortem* appearances, and chemical proof; should any portion of this evidence be wanting, the effect is thereby weakened, but not necessarily always to an equal degree; since a chemical analysis affording positive results, or a decided and characteristic *post-mortem* change, or a well-marked set of symptoms, may each in certain cases afford high probability, if not conclusive demonstration, of the fact of poisoning. On the other hand, the failure of the chemist to discover poison in the dead body does not always destroy the value of other evidence

[1] Darstellung der Leichenerscheinungen, etc., von Dr. Josef. Engel, Professor an der Universität zu Prag. Wien, 1854, 8vo.

sustaining the fact of its having been taken, since the whole of it may have been removed by vomiting and purging, or if the patient have lived long enough, been absorbed into the system, and then eliminated from it principally by the urine.

§ 17. *The discovery and the demonstration of the poisonous agent.* —The intervention of chemistry, and perhaps of physiology likewise, are necessary in this third source of information. If the poisonous agent can be isolated from the tissues of the body, and its poisonous nature be determined to the satisfaction of twelve capable men, then, indeed, have the suspicions assumed an important position in the field of investigation: "Tunc demum res certa erit, ubi venenum ipsum reperietur facile agnoscendum." Still this third source of information must not be relied upon to the exclusion of the other two.

In this third source of information, in addition to the chemical analysis, is included also the physiological, physical, and botanical examination. The chemical analysis is not always infallible. There are circumstances which render the detection of a poison by chemical analysis impossible; for instance, the examination may be unavoidably postponed so long as to permit the escape of a volatile poison, like prussic acid, from the body, although it may have been present immediately after death; or life may have been prolonged for a sufficient length of time to permit the elimination of most, if not all, of the poison which was taken, and which was the cause of death, as in the Dr. Alexander case, in which no arsenic could be found in the body, death having taken place in sixteen days after the ingestion of a large dose of the poison;[1] this is also the explanation of the non-detection of opium or morphine in many cases of fatal poisoning by these substances, and such non-detection of a poison does not necessarily invalidate the other evidences of poisoning.[2]

On the other hand, traces of poison may be found by chemical analysis when the poison has not been the cause of death; thus traces of compounds of copper, lead, arsenic, and mercury may be

[1] Medical Times and Gazette, April 18, 1857, page 389.

[2] This has been recognized in legal cases in Prussia. Vierteljahrsschrift f. gericht. med., 1857, xxxii. pages 177 and 193; 1858, xiv. page 185; 1860, xvii. page 177; 1862, xxi. page 1; 1864, N. F., i. page 96.

introduced into the body accidentally with the food, or in certain employments, or have been administered as medicine.

§ 18. The *chemical analysis* for the detection of a poison should be complete and exhaustive. The poison should be isolated and so identified as to admit of no possible doubt. Fortunately the progress of organic chemistry during the past few years has been so great that the chemist is now able to isolate and identify many of the organic poisons with as much certainty as the metallic poisons; thus he can isolate, so as to obtain in a pure crystalline form, weigh and test chemically, microscopically, and physiologically morphine, strychnine, and many of the other organic poisons as readily as he can arsenic, mercury, or any of the metals.

The chemist must, in legal analyses, take precautions similar in character to those recommended above for the physician in making the autopsy and preserving the identity of the organs. All of his work must be performed by himself; his apparatus must be perfectly clean, and must have been previously carefully tested; his chemicals must also be of known purity, by having been previously thoroughly tested; while an analysis is being performed, all of his materials, viscera, and other substances to be analyzed, chemicals, and apparatus must be kept under lock and key, so as to prevent the possibility of being tampered with. Since, in the analysis for many of the poisons, the material removed from the body is to be destroyed by chemical reagents beyond the possibility of restoration, this material should be first subjected to a thorough and systematic preliminary examination for the purpose of obtaining some hint as to the nature of the poison present, and to prevent the waste of material by being obliged to search unnecessarily for poisons not present; on this account also the chemist should be furnished with all the information which it is possible to gain in any given case from the symptoms, pathological appearances, and circumstances; since, if his suspicion be strongly directed to any particular poison, he may proceed at once to test for it without wasting any of his material (which is always very limited in amount) and time in useless work. This preliminary examination should include a careful and systematic examination of the mucous membrane and contents of the stomach and intestines with the naked eye, with an ordinary magnifying lens, and, if anything suspicious be seen, such as any white or yellow patches, it should

be examined with a microscope. The odor should be carefully noticed as well by the chemist as by the physician. The contents of the stomach should always be examined under the microscope, since frequently undissolved portions of the poison, such as arsenic, may be seen, or the character of the food, which was last eaten by the deceased, may thus be determined and the amount of digestion which it had undergone, thereby, perhaps, throwing light upon the time of death in uncertain cases, or giving other information, the importance of which may not be apparent at the time of the examination, but may prove of vital importance at the subsequent inquest or trial. The chemist, in addition to the material removed from the body, may be called upon to analyze and examine a large number of substances, such as food suspected to be mixed with the poison, stains upon clothing, bedding, carpets, or even portions of the floors where vomitus has fallen, any paper or bottle containing an unknown substance found in the neighborhood of deceased, or among the effects of any suspected person, etc. etc. If by such preliminary examinations he can obtain any hint as to the probable nature of the poison which was the cause of death, he should proceed at once by the most direct methods to test the viscera for that poison, but, if no such hints can be obtained, he must resort to the systematic methods of analysis for the isolation of poisons from organic substances. These methods are various, and need not be mentioned in detail here; they are given in special works upon toxicology, and are familiar to expert chemists.

When the nature of a poison has been determined by analysis, it is, of course, important to ascertain the *amount* of the poison present; this is not, however, of vital importance; that is, if a fatal dose cannot be isolated from the body, it does not show that a fatal dose had not been taken before death, because the whole of a body is never submitted to analysis, and a portion of the poison remains in those parts of the body not examined, and if a person lives sufficiently long after taking a fatal dose of poison, a portion or even the whole of the poison may be eliminated before death, and yet the death have been due to the direct action of the poison.

Finally, the nature of the poison must be determined with positive certainty. It must be isolated in a form so pure that it can be recognized by *all* of its properties, physical, physiological, and chemical. If a metallic compound, the metal itself should always

be obtained, and if an organic poison, the crystals, if it be crystalline, should be obtained. With regard to the possibility of poison being surreptitiously introduced into the stomach *after* death with a view of casting suspicion upon others, we may safely say that its consideration is not required until some authentic instance of the fact can be produced. This is one of the chimeras of medical jurisprudence, which the ingenuity of authors has evoked, but whose existence is fabulous, if not absurd.

§ 19. *Differential diagnosis of poisoning.*— The physician is often at a loss, upon the first view of a case of poisoning, to determine whether the symptoms presented by the patient may not be really due to disease. The aspect of a case of irritant poisoning presents a certain resemblance to cholera or to gastro-enteric disturbance, and most of the phenomena of narcotic poisoning are found in diseases affecting the brain or spinal cord. Should he content himself with remaining a silent spectator of the case, he may remain in doubt until its close; but if, on the contrary, he has witnessed or been made aware of the time and circumstances under which the symptoms came on, and their mode of invasion, he will be less embarrassed, and, if an autopsy is obtained, can seldom be at a loss to give a decided opinion. The diagnosis must remain incomplete without a *post-mortem* examination, whether its results be positive or negative, unless we except certain cases in which a chemical analysis of the excreta has clearly revealed the presence of a poisonous agent, in which case the combination of symptoms and chemical detection of the poison make an important link in the evidence; and hence, for practical ends, the careful analysis of symptoms, in those diseases which leave but few traces behind them, is of primary importance.

Reliance upon symptoms alone, no matter how suspicious the suddenness of the attack may be, must never to the mind of a professional expert be a presumption of criminal poisoning; because it is well known that many diseases are marked by the sudden appearance of symptoms which are almost directly followed by death. This prejudgment by symptoms alone may not only do a lifelong injury to an innocent person, but may even defeat the ends of justice, by preventing a cautious and quiet investigation of the cause of a suspicious death.

It must not be forgotten that pathological conditions preceded by

symptoms may cause confusion in simulating certain acute as well as chronic diseases, and sometimes even the *post-mortem* results of these diseases; for an instance of this, reference is made[1] to a case of poisoning by arsenic, resulting in a general fatty degeneration of the organs, which might have formerly passed under the head of a death from natural disease; this case, however, is one of the many which help to establish "the fact that arsenic, in common with phosphorus, antimony, and other poisons, will cause fatty degeneration of the liver, kidneys, gastric glands, heart, and other organs. . . . . Fatty degeneration is a frequent result of arsenical poisoning. Probably either it or the preceding condition of granular degeneration always exists."

On the other hand, "slow poisoning," or the result of the repeated administration of non-fatal doses of a poison, may by its symptoms simulate the appearance of a disease; in these cases, if suspicion has been directed towards an unnatural cause of death, chemical investigation and a careful *post-mortem* examination should form conclusive testimony. Again, the coincidence of disease may suggest to a criminal a means of diverting suspicion, and we may have a case presenting either a conflict of opposing symptoms due to the effects of a disease and a drug, or a confusion of similar symptoms of both. A case of this character is represented by the trial at Nashua, N. H., of E. W. Major for the murder of his wife by strychnine in Dec. 1874.[2] The strychnine was given to her while in labor, and the convulsions occurring were at first attributed to the effect of the patient's condition. The discovery of strychnine proved the fallacy of this opinion; and in fact it may safely be said that the convulsions occurring in puerperal eclampsia (which are most probably a symptom of functional or organic disease of the kidneys resulting in uræmic poisoning) may be numerous, whilst the convulsions due to strychnine poisoning are rarely more than five before the fatal result of poisoning occurs; and during the interval between the convulsions in strychnine poisoning the patient is conscious, while there is unconsciousness for a long time after a puerperal convulsion.

[1] Transaction of the Massachusetts Medico-Legal Society, vol. i. p. 43, Boston, 1878, and in Boston Med. and Surg. Journal, Sept. 19, 1878, p. 357.

[2] See Appendix.

§ 20. For convenience we may consider the various diseases which are liable to be confounded with the symptoms of poisoning, under the same heads in which the various poisons are classified.

1st. Irritant, viz., cholera, cholera-morbus, gastritis or inflammation of stomach, enteritis or inflammation of smaller bowel, colitis or colic, usually the result of a fecal accumulation in the larger bowel and rarely fatal when properly treated, peritonitis or inflammation of the serous membrane which lines the whole interior of the abdominal cavity and most of the abdominal organs, dysentery, and typhoid fever.

2d. Hyposthenians, viz., pneumonia, puerperal and uræmic convulsions, epilepsy, and tetanus.

3d. Stupefaciants, viz., certain diseases of the brain, apoplexy, septicæmia or blood-poisoning, embolism or obstruction of arteries by clots, typhoid fever, and dysentery.

4th. Narcotics, uræmia, certain diseases of the brain marked by the state of coma, pneumonia, and typhus or ship fever.

5th. Neurosthenic or neurotic poisoning, cerebro-spinal meningitis, certain acute diseases of the brain marked by delirium, and many other obscure diseases of the nerve-centres.

It would be impossible to enumerate all the forms of disease which might be mistaken for symptoms due to poisoning, for it is not that diseases are insufficiently marked by symptoms, but because these latter in the present system of medicine are referred to well-known pathological conditions or lesions of limited and definite organs concerned in performing the functions of life. Poisons, on the other hand, generally act upon more than one organ at the same time, and without necessarily causing the same conditions or lesions of organs which are associated with the symptoms due to these lesions; for instance, in pneumonia, life is destroyed by reason of a definite and well-known unhealthy condition of the lung-substance interfering with the functions of breathing, while hydrocyanic acid, which may also cause death by asphyxia, does not cause a similar lesion of the lung-substance.

§ 21. The diseases most apt to be mistaken for *irritant* poisoning are:—

Cholera.—This disease in its *malignant* form, as is well known, is often rapidly fatal; it may supervene shortly after a meal or a draught of liquid; its onset, marked by a lowered bodily

temperature, is sometimes sudden, although usually preceded by diarrhœa; there are great thirst, vomiting, and purging, without effort, of a thin and slightly turbid liquid; the surface is cold and shrivelled, the features collapsed, the voice almost extinct, the pulse feeble or imperceptible, and the intellect undisturbed; the lesions discovered after death are not sufficiently characteristic to be used in evidence; sometimes diphtheritic changes are found in the intestines, sometimes a deep injection of their submucous coat. In most of the symptoms enumerated, cholera may resemble very closely a case of poisoning by arsenic or other irritant, but there are, nevertheless, sufficient means of distinction. In poisoning by the irritants, a burning sensation in the throat and stomach, and pain and distress in the whole abdomen, but chiefly over the stomach, precede, or occur simultaneously with, the vomiting. These are the most prominent and constant symptoms during the continuance of the case. The matters passed from the stomach and bowels, after their previous contents have been evacuated, are mucous and bloody, and are not spouted forth as in cholera, but rejected with great distress and effort. The anus is often, indeed, excoriated by their irritating properties. Furthermore, it may be observed, that those who have once witnessed a case of malignant cholera will most probably have the peculiar but indescribable features of this disease so impressed upon their memory, that they will not readily mistake for it any case of irritant poisoning. Finally, the epidemic prevalence of the disease, or the fact that about the same time other cases resembling Asiatic cholera have occurred, will materially assist the physician in giving a positive opinion as to the nature of the attack.

§ 22. *Bilious cholera*, or *cholera morbus* as it is usually termed, is a disease which has more points of resemblance to the effects of poison than that which has just been mentioned. In it, both the extreme collapse and the peculiar rice-water discharges are not seen; but, on the other hand, the vomiting and purging are of a bilious character, and there is excessive pain in the abdomen. The progress of the case is, however, different. The pain in cholera morbus is remittent, coming on in paroxysms; and in proportion as the offending matters are discharged, the vomiting is less frequent and painful. In irritant poisoning, on the contrary, the pain is constant, and there is usually also tenderness upon pressure;

the vomiting is of mucus and blood, and the discharges from the bowels are of a similar character. The tendency in the latter is to death, in the former to recovery. Cholera morbus is seldom fatal, and when it is, death does not, in general, take place for several days. The contrary is the rule in poisoning by the irritants. Such are the distinctions usually advanced by authors; and while they are, as a general rule, undoubtedly correct, it should not be forgotten that distinctions valid in medicine may not be so in their application to criminal cases. While, in the science of medicine, diagnosis is founded upon a careful investigation of the prevailing and general characters of diseases, the most delicate questions in medical jurisprudence are, on the other hand, determined by exceptional cases. Now, as experience shows that persons may not die from the effect of the poisonous irritants until several days have elapsed, or, indeed, that they may not die at all, we are at a loss to perceive how, in such cases, it will be possible, in the absence of circumstantial and moral evidence, to decide that the symptoms were due to attempted poisoning, rather than to the disease in question; for, although there may be some points of distinction, as, for example, the early occurrence of a burning sensation in the throat, the unremitting character of the pain, and the sanguinolent discharges, yet these may be absent in mild cases of poisoning, and where life is not destroyed.

It may be observed, as a further aid in differential diagnosis, that diarrhœa in the earlier stage of cholera morbus will be usually absent, and sometimes does not occur at all, while in the case of irritant poisoning this is an early symptom.

The corrosive poisons leave traces behind them sufficiently distinct to prevent any likelihood of mistaking their effects for those of disease.

§ 23. *Rupture and perforation of the stomach.*—There are certain diseases or sudden accidents partially resembling in their symptoms those of the irritant poisons, which are so readily recognized on *post-mortem* examination, that it is needless to enlarge upon the modes of distinguishing them. These are rupture of the stomach, intestine, biliary ducts, and uterus, and no one will contend that such lesions can be produced by poisoning.

The *post-mortem* rupture of the stomach must be carefully distinguished from that occurring during life, and will be accompanied

by the escape of a varying amount of the contents of the stomach into the abdominal cavity, and an absence of inflammatory action. "The walls of the stomach, especially at the fundus (or lower portion) are much softened, and are frequently converted into a translucent, slimy mass."[1] If the digested condition of the stomachal walls which surround the opening be due to the action of a corrosive, as, for instance, sulphuric acid, there can hardly fail to be present certain inflammatory signs due to its more general action upon the internal surface. "The effects produced by perforations occurring during life range according to the relations of the surrounding parts; a communication may be established with the abdominal cavity, or with a closed sack, resulting from chronic inflammation, or the hole in the wall may become closed by the formation of adhesions betwcen the stomach and other organs. In the first instance, the result is a general peritonitis which is quickly fatal; in the second, a circumscribed purulent or ichorous inflammation, which is frequently of long standing; and in the last, as a rule, a chronic progressive ulceration. In such cases it is frequently necessary to vary from the usual method of performing the autopsy, and to remove the stomach in connection with the other organs, most frequently the pancreas and liver."[2]

The symptoms which precede death by perforation of the stomach or intcstines deserve but a passing notice. These may, however, give rise to embarrassment; not, indeed, so much from the symptoms of the disease when considered alone, but from their offering perhaps sufficient resemblance to those of poisoning to support the opinion that the lesion referred to may be due to this cause. While it is true that in this disease, the seizure is sudden and the pain in the abdomen acute, it is not preceded by the sensation of burning in the throat and stomach, nor is the vomiting urgent unless upon the ingestion of the liquids. There is again no diarrhœa; the main symptoms are acute diffused pain all over the abdomen, arising from peritonitis, and the patient is collapsed from the first. But in a case of this kind, which has not been closely observed, the discovery after death of the perforation in the stomach will natu-

[1] Orth, Diagnosis in Pathological Anatomy, translation, New York, p. 281.

[2] Idem.

rally awaken the suspicion of poisoning. If we now inquire under what circumstances this lesion is produced in cases of poisoning, we will find that with the exception of the corrosives it is seldom occasioned by any kind of poison. Perforation from arsenic is so rare an event that but three cases are said to be on record, and the fact of the perforation being so unusual in a form of poisoning so exceedingly common, renders it highly probable that in these instances it was due to an already diseased state of the coats of the stomach. But the corrosive poisons, which undoubtedly produce, in many instances, a perforation of the stomach, leave in addition such manifest traces of their action upon the throat, œsophagus, and stomach, not to mention the corrosion of the mouth and lips, that it seems to be inexplicable how the single fact of the perforation should leave any doubt in the mind of the examiner or of the jury. Moreover, the character of the perforation alone affords a sufficient ground of distinction. The stomach in such cases is blackened and extensively destroyed; the aperture is large, its edges rough and irregular, and the coats are easily lacerated. Further, the poison escapes into the cavity of the abdomen, where it may be easily discovered by chemical analysis. In perforation from disease, on the other hand, if the affection be of a cancerous nature, there will be no difficulty in distinguishing the cause, and, if it be simple ulceration, such as occurs sometimes in persons in the enjoyment of apparently good health, the opening is also characteristic in its nature. "The aperture is usually of an oval or rounded form, about half an inch in diameter, situated in or near the lesser curvature of the stomach, and the edges are smooth. Indeed, it has not unfrequently the appearance of having been 'punched out.' The outer margin of the aperture is often blackened, and the aperture itself is funnel-shaped from within outwards—*i. e.*, the mucous coat is the most removed, and the outer or peritoneal coat the least. The coats of the stomach, round the edge of the aperture, are usually thickened for some distance; and when cut they have almost a cartilaginous hardness." (Taylor.) Death takes place from peritonitis, the contents of the stomach escaping into the cavity of the abdomen.

Similar perforations may exist in the duodenum or upper portion of the small intestine, but they are comparatively rare. Where these perforations of disease occur, they are usually the result of the round or chronic ulcer, or more rarely still of cancerous disease.

All these perforations resulting from disease are quite small, the diameter rarely exceeding one-tenth of an inch.

§ 24. There is still another form of destruction of the walls of the stomach, which is due to a maceration of the stomach by its contents after death. It cannot give rise to a suspicion of poisoning, unless in the absence of any knowledge of the symptoms preceding death. It is purely a cadaveric phenomenon, and may occur in the stomach of persons dying from any cause, provided the peculiar digestive process necessary for its production exist.[1] It is formed only in the larger end of the stomach, the opening is large and irregular, with ragged and "moth-eaten" edges and no surrounding inflammation. The edges may be discolored and black, as the result of a chemical action of the intestinal gases upon the coloring matter of the blood. There is no peritoneal inflammation, but the spleen, diaphragm, or other subjacent viscus may be softened by the acid. The absence of any disorganization of the pharynx and œsophagus, and of peritoneal inflammation, is sufficient to distinguish this *post-mortem* perforation from that caused by corrosive poison, with which alone it is possible to confound it. Dr. Budd has found, naturally enough, that this *post-mortem* softening of the coats of the stomach is more common in hot weather. He says: "During the past summer, which was a very hot one, my attention was casually drawn to this subject, and from the middle of May to the middle of August, I carefully examined the stomach in all the bodies that were opened in the King's College Hospital. In several instances the mucous membrane of the stomach, in the greater curvature, was completely destroyed, and in a very large proportion it had been clearly acted upon more or less by the gastric juice. I renewed my observations in October, but the change, in a striking degree at least, was then much less frequent."[2]

Should the rules thus laid down for the discrimination of the

[1] See Med. Times and Gaz. (No. 246, p. 268), for a case in which the stomach of a child (which had been asphyxiated by its intoxicated mother hugging it too closely) presented the following appearances: Nearly the whole of the great *cul de sac* had disappeared; the edges of the aperture were thin, jagged, and flocculent; another similar but smaller opening existed lower down and abutted upon a corresponding aperture in the transverse colon. No unnatural adhesions or other morbid appearances existed, and a quantity of milk was found in the stomach and in the cavity of the abdomen.

[2] Lancet, 1847, p. 593.

source of these perforations not prove sufficient, a resort to chemical analysis will render the demonstration complete. If any poison has been taken in so large a dose, or is possessed of such violent properties as to cause the lesion thus referred to, it will readily be found by these means. It has been said that a person may die with the symptoms of irritant poisoning, and after death, perforation, the result of cadaveric change, be found, and that hence the knowledge of the true cause of the perforation does not exclude the idea of poisoning. It is true that such a rare coincidence may happen, but the want of connection between the poison and the perforation merely renders it necessary to support the charge upon other evidence. The case of Miss Burns, for the murder of whom, by poison, a Mr. Angus, of Liverpool, was tried in 1808, is one in which this doubt arose. The charge of poisoning was not sustained by chemical or pathological evidence, and the prisoner was acquitted.

§ 25. *Gastritis, gastro-enteritis, peritonitis.*—It is the natural effect of poisonous irritants to produce one or more of these diseases, but as they may arise from other causes, a distinction is in practice necessary. Gastritis has a more protracted course than is usual in irritant poisoning. Diarrhœa, so nearly universal a symptom of irritant poisoning, is not always present in these diseases. These names signify inflammation of stomach, or of intestines or of the peritoneal surface of the abdomen, which, like other inflammatory processes, is always accompanied with fever or an increase of bodily temperature ; hence, the natural effects of disease may be simulated, but if the medical attendant be a careful observer, the difference between the symptoms of an irritant poison, which may induce secondary inflammatory processes, and a natural disease will hardly escape notice. However satisfactory these distinctions may be to a physician, it is apparent that they may have little weight with others ; hence, practically, it is important to examine them closely, for the accusation in such cases will have to depend upon the results of the chemical investigation.

§ 26. *Strangulation of the intestines* has been enumerated among the diseases likely to awaken suspicion of poisoning, but with little justice, for if the symptoms are not sufficient to distinguish it, most certainly it cannot fail to be detected upon the *post-mortem* examination.

§ 27. The symptoms produced by *narcotic poisoning* may be closely imitated by those of natural diseases, such as apoplexy, epilepsy, congestion of the brain, and tetanus. Indeed, occasionally the similarity is so great that, upon the medical evidence alone, it may be impossible to acquire a certainty of the cause of death. Many distinctions have been drawn by writers upon toxicology between the effects of narcotic poisons and those of disease of the brain and spinal marrow, but they serve only to show the very close analogy between them. When, moreover, it is remembered that the most important of these poisonous agents leave no distinct traces of their action in the dead body, it will be perceived that the differential diagnosis must depend mainly upon the results of chemical investigation, and the moral or circumstantial evidence in the case.

The use of instruments, for instance, in determining by the stethoscope a condition of the lungs often associated with cerebral or brain diseases, the thermometer to indicate inflammatory processes due to disease of the nerve-centre, and other means now more familiar to the medical profession than formerly, may serve to differentiate disease from acute poisoning. An effusion upon the membranes covering the brain causes a very rapid appearance of lowered bodily temperature followed suddenly by increase of temperature; the action of opium tends to a depressed bodily temperature, yet narcotic symptoms are present in both cases. Unfortunately no carefully recorded observations of bodily temperature as the result of acute drug-poisoning have been presented in courts of law; yet, we believe it would prove of material value if a point of this kind were insisted upon in medical testimony in cases where reliance is placed upon symptoms. The thermometry of disease is pretty well understood by the educated physician, and the experience of every additional year tends to make our knowledge more exact.

§ 28. *Apoplexy*, it is said, may be distinguished from opium poisoning by the following considerations, viz: that it does not usually occur under the age of thirty, nor come on without warning symptoms, and that the time of seizure is irrespective of the taking of food or drink; but those distinctions are futile, even when, as is rarely the case, an accurate account of the whole history of the sickness can be obtained. More reliable are the facts that in poisoning by opium the symptoms are gradual in their accession, and that the more confirmed effects are preceded by

drowsiness, and that the patient, until an advanced period of the stupor, can be temporarily aroused. The pupils also, in general, are strongly contracted, and there is no contortion of the face or paralysis of the limbs. In the majority of fatal cases of apoplexy, the attack is sudden, although indistinct warning symptoms may have preceded it; the patient cannot be roused, the pupils are dilated and insensible, and the face is slightly contorted, indicating a paralytic condition of one side of the body. Yet there are numerous exceptions to these rules, a fact which is easily understood, when we reflect that opium in addition to its specific narcotic properties produces the very same pathological condition, with the exception of effusion of blood into the substance of the brain, from which it is our aim to distinguish it; this need not, however, cause confusion, for chemical science can easily determine the presence of opium or morphine in the brain substance in those cases in which its use has been followed by death, when it has not been destroyed or eliminated from the organism.

§ 29. *Embolism.*—A clot formed in the heart or larger bloodvessels may be sent into the arterial circulation and plug up an artery which feeds an important organ, the lungs for instance, and without manifest symptom, may cause sudden death. This is not so unusual a cause of sudden death as may be generally supposed, and, as the person may have been previously in robust health, this catastrophe, not unfrequently occurring in bed, may give rise to suspicion of foul play. This accident may also occur in any febrile attack or during convalescence of a lying-in patient. A careless autopsy may have caused the pathological proof of an embolism to be overlooked, because often these clots are quite small, or fall out by rough cutting or handling of the bloodvessels.

§ 30. *Pneumonia.*—Death from œdema (dropsy) of the brain or lung substance, from sudden shock, or from carbonic-acid poisoning, may supervene so suddenly that the individual may die before the arrival of a medical attendant. In fact, cases are recorded where the first sign of the attack was a sudden fall in the street, unconsciousness, coma, and death following almost immediately. Here, again, the *post-mortem* examination reveals the natural cause of death.

§ 31. *Cerebro-spinal meningitis.*—Symptoms of this disease were confounded with those of poisoning by tartar emetic in the trial of Mrs. E. G. Wharton, at Annapolis, Maryland, in January, 1872.

Without attempting either a review of that case,[1] or a thorough description of the above disease, it may be advisable to relate the character of the symptoms most usually noticed in this serious epidemic disease. Dr. Ed. Warren, in the case[2] alluded to, stated that the patients are in a "condition of semi-unconsciousness and of increased sensitiveness of the surface of the body, so that they shiver when touched; and have a rigidity of the muscles of the neck, back, and inferior extremities; pupils neither contracted nor dilated, but insensitive to light; suppression and retention of urine; lividity of countenance; trismus; opisthotonos; occasional jactitation and restlessness; incoherent articulation, and a speedy and violent death."

This disease occurs most generally as an epidemic, and is more frequent in winter and spring than in the summer months. The best authorities (Niemeyer, Aitken, Valleix, and in this country, J. B. Upham, A. Stillé, W. H. Draper, S. Ames, and others) describe the disease as commencing, often with little or no positive warning,[3] with a severe chill of variable duration, accompanied by a severe headache, and in most cases by vomiting. At the end of the first or second day, rarely later, the head is drawn backward. The severe headache continues, the pain extending down to the nape of the neck and along the spine. The third or fourth day the muscular contraction, especially in the vicinity of the neck and back, is quite remarkable, and to this succeeds opisthotonos (or an arching of the back in such a way that the body may rest only upon the heels and back of the head); consciousness is lost; constipation, rarely diarrhœa, continues throughout the whole progress of the disease; the pulse is not generally more rapid than in health, though it may be irregular and soft; towards the close of the disease, if fatal, the pulse is more rapid. The urine is suppressed or retained. The peculiarity of the respiration is a marked feature of the disease.

The *post-mortem* appearances of this disease are more marked if

[1] Amer. Journ. of the Med. Sciences, April, 1872, p. 329 *et seq.*

[2] Reported and published by the Baltimore Gazette, p. 97.

[3] A police officer at Dublin, Ireland, was stricken down suddenly while on his beat, became unconscious, then had violent convulsions, and was dead in less than ten hours, though he had the same morning left his house apparently in good health.

the disease has existed several days before death. If the patient dies during the first stage, viz. before exudation occurs, the appearance of the brain and spinal cord is not peculiar, and oftentimes no great change in any organ has been remarked.

Usually the appearance, as described by Niemeyer, in recent cases is as follows: "The subjects show no emaciation, protracted rigor mortis, or extensive hypostasis. The cranium contains much blood; the dura mater is more or less dense and occasionally covered with small hemorrhagic spots. There is usually no effusion between the outer membranes; in the subarachnoid space there is an exudation, which, both in extent and character, occupies about a medium position between the purulent exudation in meningitis of the convexity and the puro-serous exudation in meningitis of the base of the brain." The brain itself is more or less vascular. The dura mater of the spine is also more or less vascular, occasionally very tense, especially at the lower part. The arachnoid usually presents no peculiarity except a decided opacity. The spinal medulla itself is more or less vascular, occasionally infiltrated and relaxed. Except some accidental complications there are no particular anomalies of the other organs; we should especially note here that the spleen is almost always normal.

§ 32. *Tetanus.*—The same remark is applicable to the resemblance between the convulsions of *tetanus* and those produced by strychnine. It is needless, therefore, for us to draw a parallel between the diseases referred to and the symptoms so closely imitating them, produced by the poisonous narcotics. Where the success of either prosecution or defence comes to stand upon such vacillating ground as this, other sources of evidence failing, it would be better that the most approved works on pathology should be consulted and the descriptions of disease there given be compared with the symptoms enumerated under the narcotic poisons, than that the reader should draw an unwarranted conclusion from such an imperfect abstract as it would be proper for us to give in this place.

In Palmer's case, the distinction between the symptoms of poisoning by strychnine, and tetanus, was thoroughly investigated.[1]

§ 33. Finally, all cases of *sudden death* may awaken suspicion of poisoning. If, as may well be the case, the *post-mortem* altera-

[1] Lond. Lancet, May and June, 1856.

tions do not clearly indicate the seat and nature of the affection, a review of the manner of dying, and the absence of any positive result from chemical investigation, must at once negative the presumption of poison having been taken. Thus, certain diseases of the heart, over-distension of the stomach, fatal syncope, and some obscure diseases, may not be recognized at the autopsy, but the mode of death in them is entirely different from that in any form of poisoning, except, perhaps, by prussic acid, in which the odor or chemical tests will disclose the cause of death. The importance of a careful collection and comparison of *all* the medical evidence in every case, cannot be too strongly insisted upon; for upon this combination of proof it is that a correct knowledge of the true cause of death must depend.

§ 34. *Compound Poisoning.—Complication of symptoms due to the combined action of poisons.* The so-called antagonism of medicines is certain to play an important part in the subject of toxicology, and deserves mention here. By antagonism of medicines is meant the results of observation of symptoms of one medicine as opposed by the symptoms of another, or the modification of one set of symptoms due to a poison by those of an entirely antagonistic character; for instance, the dull heavy narcotism induced by an overdose of morphine may be antagonized by a moderate dose of atropine. In the former class of symptoms we have a slow respiration, say ten to fifteen inspiratory movements of the chest in a minute, and a slow pulse, forty or fifty beats per minute; whereas atropine quickens both respiration and pulse, and both of these drugs will produce a mental hebetude, and even coma; though atropine, in moderate doses, will induce an active delirium which is gay and lively, while opium produces a passive delirium which is dreamy and lascivious. Again, the quieting influences of chloral, which may obtain until death occurs, may be antagonized by the convulsifying action of strychnine. Antagonism of medicines does not necessarily imply that the poisonous dose of one drug may control the issues of life and death set in danger by another drug; for instance, a non-fatal dose of strychnine may antagonize the symptoms induced by a fatal dose of chloral, provided the strychnine be first given; while, again, a non-fatal dose of strychnine given to a patient whose life is already jeopardized by a poisonous dose of chloral may even accelerate the fatal issue. Still further, the

complication of symptoms may be masked by the use of a third or fourth drug. An illustration is offered in the following cases:—

Dr. Harrison reports[1] the case of an attempted suicide by the combination of strychnine and opium. About three-quarters of a grain of strychnine and ninety drops of laudanum were swallowed, and in three-quarters of an hour the strychnine convulsions appeared; during this time some urine, though only a portion of that passed was collected from the man, showed the presence of one-twentieth of a grain of strychnine. Chloral was used as an antidote to the strychnine; the man recovered. Another case is reported,[2] where a similar amount of strychnine was swallowed combined with two drachms of laudanum and some oxide of mercury. No convulsions occurred, but the man exhibited an intense narcotism which gradually disappeared. Dr. Marvin[3] reports the case of a patient who took, with suicidal intent, ten grains each of strychnine and morphine during an attack of melancholy, which a *post-mortem* examination proved to be due to a cerebral condition caused by uræmic poisoning from serious disease of the kidneys. The symptoms of morphine poisoning could hardly be recognized.

The second case reported by Dr. Marvin[4] was seen by him in consultation, and was remarkable from the fact that the antagonism was caused by disease; in this case serious coma and apparent narcotism supervened after the moderate ingestion of one and a quarter grains of morphine administered in fractional doses of one-quarter of a grain every three hours. Coma with stertorous breathing occurred four hours after the final dose, or fifteen hours after the first dose was taken; an hour afterwards one-sixtieth of a grain of atropine was administered subcutaneously, because the respiratory movements were slow and feeble and the pulse weak, with a bodily temperature of 103° and 106°. To this was added the stimulating effect of electrical treatment. The cerebral symptoms were so marked, and the patient so nearly dead, that Dr. Marvin left him as he supposed *in articulo mortis;* but he learned from the attending physician the next morning that the patient recovered from these severe symptoms. An examination of the urine revealed a low specific gravity, one-third amount of albu-

[1] Lancet, May 13, 1882, p. 780.
[2] Lancet, Dec. 23, 1871.
[3] Med. Herald, Louisville, 1879, No. 1.
[4] Idem.

men, and a number of waxy and granular casts. The high temperature would show that the cerebral lesions were not due to uræmia, but that this condition made the narcotic effects of morphine more pronounced than if the patient were free from organic disease.

A number of other cases have been observed by physicians in their daily practice to illustrate the more ready susceptibility to morphine narcosis of patients who are the subjects of advanced renal disease.[1]

The fact must not be overlooked too that the blood may be so surcharged with one poison that a second may not be absorbed until the first has passed out of the circulation; in this view of the matter the symptoms of two drugs may be masked; for instance, the writer has etherized a dog and administered subcutaneously a dose of prussic acid double that required to destroy its life; during three-quarters of an hour, while the ether was being inhaled, there was no evidence of poisoning by prussic acid, but, on allowing the dog to recover from the effects of the ether, the poisonous action of prussic acid became manifested, and the animal died with the usual effects of prussic acid intoxication. Though these latter effects of combination have not been extended to other drugs, yet it cannot be denied that the doubtful question of absorption of two remedies simultaneously in one individual will inevitably form an important factor in the matter of toxicological science. Prof. J. N. Langley,[2] after a very careful series of experiments on the antagonism of certain alkaloid drugs, puts the nature of the antagonism in the following words (p. 19): "Alkaloids act on the tissues by forming chemical compounds with them. When two poisons act on the same tissue, the result depends on their relative chemical affinity for the tissue and the mass of each present. Thus, then, within certain limits to be spoken of presently, if the one alkaloid is given, its effect can be antagonized by giving a sufficient quantity of the other. A limit is placed to this antagonism by the impossibility of giving very large quantities of any substance without injuring the tissue by physical processes consequent on alteration in the density of the fluids. The antagonistic action of alkaloids can only occur within the limits

[1] See The Hypodermic Injection of Morphia, its History, Advantages, and Dangers, by H. H. Kane, M.D. N. Y., C. L. Bermengham & Co. 1880.

[2] Journal of Physiology, August, 1880.

of doses which do not seriously alter the tissue by altering the normal rate of diffusion, etc. If, for instance, a nearly maximal dose of atropine be given, no other alkaloid we are acquainted with can antagonize its action on the salivary glands, for the chemical affinity of atropine for the tissue is so great that it would require, to compensate for this, a mass of the other alkaloid impossible to be given without direct injury to the tissue. . . . . The only case in which a return to normal seems to me possible, is when the poisons combine together, or when they act on some third tissue, as blood, which influences the gland or other structure in question." Now what is true in regard to physiology is true also in regard to toxicology, and in a given case of poisoning by more than one drug the expert should always bear in mind that the nature of symptoms must necessarily be influenced by the rules which govern the action of either or both. For a further discussion of this matter the reader is referred to an article by Rossbach;[1] also to the treatise of Fothergill.[2]

§ 35. *Suicide.*—It may be of importance to consider this question under a general head, since it is well known that the question of suicide plays an important part in forensic medicine on account of life insurance. There are well-known cases where a person has committed a self-murder in order to secure a premium effected upon the risk of his life for the benefit of his family. Provided he wished to effect this purpose fraudulently, and to conceal his act of suicide, the victim would seek a means of self-destruction which would escape detection, and to this end he will select a poison whose effects would resemble a natural disease. On this account, therefore, the question of determining a natural from an unnatural death assumes an important position, and therefore the physician who is called upon to state the cause of a sudden death should ascertain whether the deceased had taken a policy for life insurance.

§ 36. *Classification of poisons.*—Poisons have been variously classified by different authors, but none of the systems are entirely free from objections, either theoretical or practical. Thus they have been classified according to the kingdom from which they are

[1] Pflüger's Archiv, Bd. xxi. Hf. 1, p. 1, 1879. Rossbach's experiments are not so conclusive as those reported by Langley, and do not affect the question as above stated.

[2] The Antagonism of Therapeutic Agents, by J. Milner Fothergill, M.D. Edin. Phila., Henry C. Lea, 1878.

derived, into mineral, animal, and vegetable; also, according to their chemical properties, into organic and inorganic, acids, alkalies, and salts; but perhaps the most satisfactory methods of classification are those based upon their physiological action, the principal ones of which are those of Taylor[1] and Tardieu.[2]

We shall not in this treatise confine ourselves exclusively to any of the systems laid down in books upon poisons, but shall, in the main, as in our former editions, follow the old classification of Or-

- [1] Irritant
  - Mineral
    - Acid poisons.
    - Alkaline poisons.
    - Non-metallic.
    - Metallic.
  - Vegetable.
  - Animal.
- Nenrotics
  - Cerebral.
  - Spinal.
  - Cerebro-spinal.
  - Cerebro-cardiac.

Taylor on Poisons, 1875, p. 73.

[2] The following is the classification of M. Tardieu (Étude medico-legale et clinique snr l'Empoissonement, Paris, 1875, page 170): 1st. Irritants and corrosives; 2d. Hypostheniants; 3d. Stupefiants; 4th. Narcotics; 5th. Tetanics.

1st. Poisoning by irritant or corrosive poisons has the peculiarity attributed to the local irritant action, which may result in a violent inflammation, corrosion, and a disorganization of those tissues attacked by the poisonous substance, the effects of the substance which has been swallowed being almost exclusively due to lesions of the digestive organs.

2d. The hypostheniants have the peculiarity not merely of producing a local irritation, though this may actually occur, but also causing general disturbances resulting from their absorption; these latter may be disproportionate to the local effects, which oftentimes are entirely absent, and are very different from irritation or inflammation; these consist in fact of a rapid and profound depression of the vital forces, and are associated with a manifest alteration of the blood.

3d. Poisoning by stupefying poisons, of which the largest portion have been comprised under the improper denomination of narcotico-acrid, though producing neither narcotism nor acridity, has, as its essential character, a direct special action upon the nervous system, a depressant action which corresponds to what is called in *semiotics* (symptoms of disease) stupor, sometimes accompanied with a local irritation, oftentimes of a slight character.

4th. Poisoning by *narcotics* is characterized by an action quite special and distinct, that can only be defined by its own name, *narcotism*.

5th. Finally, poisoning by the tetanic poisons is characterized by a violent excitation of the nervous centres, the intensity of which may be so great as to produce instant death.

fila, viz., into IRRITANT, NARCOTIC, and NARCOTICO-IRRITANT, since it has become so familiar to American medical jurists, although it is by no means free from objections, nor is it strictly accurate. This division cannot be rigorously maintained except for the purpose of convenient reference; since there are poisons usually classed under irritants, which are likewise sometimes narcotic in their action, as, for example, arsenic and oxalic acid; and, on the other hand, some of the pure narcotics, especially opium, occasionally produce the symptoms of irritant poisoning. The classification is, however, sanctioned by the use of the most eminent toxicologists, and, in the present imperfect state of our knowledge of the mode of action of poisons, has fewer objections against it than can be urged against any other.

The symptoms produced by each class of poisons are sufficiently indicated by their name. Thus, the irritants produce vomiting and purging, intense abdominal pain, and fatal exhaustion. Septic irritants produce, in addition, symptoms which are known as typhoid; and certain metallic irritants in small and long-continued doses give rise to impaired digestion and nutrition, and death by a gradual exhaustion of the system. Their primary action is expended upon the intestinal tube, causing inflammation or corrosion, and the effect upon the nervous system is secondary. The narcotics produce fulness of the head, vertigo, dimness of vision, delirium, coma, paralysis, and sometimes tetanic convulsions. The narcotico-acrid poisons produce stupor or delirium and convulsions, and also irritate the stomach and bowels, causing vomiting and purging. Both these and the irritant poisons are commonly known by their taste, which is hot and acrid, metallic, nauseous, or bitter; arsenic is the chief exception to this rule, as its taste may be either unperceived or be only distinguished as rough or sweetish. The corrosive poisons, such as the mineral acids and caustics, have so violent an effect upon the mouth and throat, that the mere fact of their having been taken affords a good presumption, in this country at least (from the readiness with which any kind of poison can be obtained), against their having been voluntarily swallowed. The pure narcotics have only a slightly disagreeable taste. This class is, however, but a small one compared with the others, and, with the exception of opium, rarely gives rise to judicial investigations.

CHAPTER II.

IRRITANT POISONS.

I. *Sulphuric Acid.* (Oil of Vitriol.)

§ 37. CONCENTRATED sulphuric acid is a dense, oily, transparent and colorless liquid, with an energetic attraction for water. When diluted with water, heat is evolved by the mixture. It rapidly destroys and carbonizes organic matter, the extent of the destruction depending, of course, upon the degree of concentration of the acid, and the length of time it remains in contact with the organic structure. It is more frequently taken and given by mistake than by design, at least in our country, although cases are not wanting in which it has been criminally given to young children. An interesting case is related in Henke's Zeitschrift, where it was used with

the hope of inducing abortion. From the ease with which it can be procured, without awakening suspicion, on account of its extensive use in the arts, either pure or mixed with other substances, it has frequently been taken with suicidal intent, but on account of the excessive pain produced by this poison, and on account of the facility with which other less painful means of procuring death can be obtained, the frequency of sulphuric acid poisoning has much diminished of late years, especially in this country and France; most of the cases seen in recent years have been cases of accidental poisoning; it has, for instance, been taken by mistake for castor oil. Very rarely is sulphuric acid administered with criminal intent, its intense acidity and the immediate pain caused by it enabling the intended victim to detect it at once; it can only be employed for the purpose of murder in the case of young children or drunken persons, unless very great force be used, which would require the presence of several criminals. This acid has also proved fatal when used as an injection into the vagina for the purpose of procuring an abortion,[1] and when injected into the rectum either accidentally or designedly,[2] and of late years, in England, numerous cases have occurred where it has been employed for disfiguring the person, by being clandestinely thrown upon the face or neck.

§ 38. *Symptoms.*—Sulphuric acid may be taken as the type of the corrosive poisons. The symptoms, of course, vary very greatly according to the concentration and the quantity of the acid taken. When a large dose of the concentrated acid has been taken, the symptoms begin almost immediately, and are characterized by a very acute pain, which at first is that of burning, and extends from the mouth to the stomach. This abdominal pain is most excruciating, somewhat resembling that of cholera, and is located by the patient chiefly in the epigastrium. Violent and painful vomiting also occurs almost immediately; the vomitus consisting usually of black or dark brown material (so-called coffee-ground vomitus), of tarry consistency, and very acid in its reaction, so that if it happens to fall upon a marble slab or other calcareous material, effervescence will take place. The dark brown or black color of the vomitus is

[1] Journ. de Chim. Médic., tom. vii. 1831, Mai, No. 10, page 312.

[2] Preuss. Med. Vereins-Zeitung, 1848, No. 13, where the acid was used by mistake for oil; and Hofmann, Lehrbuch der gericht. Medicin, 1878, page 634, where the acid was used intentionally.

due to the action of the acid upon the blood. The microscopic examination of the vomitus shows the presence of gastric cells, and later shreds of tissue from the walls of the stomach, sometimes even tubular bodies consisting of the exfoliated membrane of the œsophagus are seen. This vomiting usually takes place immediately after the swallowing of the acid, but there are a few cases in which vomiting was delayed for some time; the condition of the stomach, whether full or empty, will explain most of these cases of delayed vomiting.

In some cases the concentrated acid comes in contact with the larynx or with the mucous membrane of the air-passages, when it may cause œdema of the glottis and death in a few minutes from suffocation, and the evidences of asphyxia will be seen at the *post-mortem* examination. This is especially liable to be the case when the sulphuric acid has been forcibly administered, as, for instance, to infants. Sometimes sudden death is due to perforation of the stomach or to extensive hemorrhage, due to the opening of one or more large bloodvessels by the corrosive action of the acid.

In ordinary cases where life is prolonged for several hours, the average duration being from eighteen to twenty-four hours, collapse takes place in a short time; there is almost suppression of urine due to irritation of the kidney caused by the acid, and absence of feces, although occasionally we see diarrhœa; the pulse is weak, the skin is cold and clammy, and the face is expressive of the most intense anguish; usually the intellect remains intact until death occurs. Sometimes convulsions are present, which are similar to those often seen in cholera. Rarely an intermission occurs in the symptoms, as in the case of many of the irritant poisons, shortly followed by an exacerbation and death.

Unless care be taken in administering or taking the acid, we can usually see evidences of its local action upon the skin of the face or neck, or upon the mucous membrane of the mouth or lips, and in many cases, stains are made by the acid upon the clothing. These eschars produced upon the skin and mucous membranes are at first white changing to a gray, and finally to a brown or black color.

The peculiar symptoms are the great suddenness of the attack, the extreme pain, the character of the stains upon the mucous membranes, skin, and clothing, the constipation, and the anguish which

precedes death. The localization of the pain in the epigastrium is also an important symptom from a diagnostic point of view, since in the case of many of the other irritants, the pain is more diffused over the entire abdominal region.

§ 39. When the dose of sulphuric acid has been small, or taken upon a full stomach, or when the acid has been quite dilute, the symptoms may be less severe, and produce the effect of *subacute* poisoning. In these cases life may be prolonged for months or even years, the patient finally dying of marasmus consequent upon the incurable dyspepsia due to the partial destruction of the mucous membrane of the stomach. At first in these cases the symptoms resemble those of the acute poisoning, but are much less severe; there may or may not be hemorrhage, as shown by bloody vomitus; the vomitus is usually glairy, containing much mucus; there is excessive pain in the mouth and throat, this pain extending to the stomach, and rendering the ingestion of food almost impossible. After the first eighteen or twenty-four hours in either acute or subacute poisoning, inflammatory symptoms appear, due to the inflammation of those tissues with which the acid has been in contact; these symptoms are those of febrile reaction accompanied with rapid pulse, rise of temperature, etc. Soon shreds of the disorganized membrane may be expelled by vomiting, and if the ulcerative process has affected the walls of the larger bloodvessels, there may be hemorrhage. If the patient's life be spared for a sufficiently long time for the ulcerations caused by the sulphuric acid to heal, the cicatrices resulting may cause stricture of the œsophagus, stomach, or intestines with the long train of symptoms due to the consequent obstruction, dyspepsia and indigestion, until finally, after perhaps months or years of suffering, the patient dies from inanition. The writer is familiar with a case in which an ounce of sulphuric acid (strength unknown) was taken by mistake for cider: the patient recovered from the acute symptoms, but died in a little more than two years, on account of the secondary troubles in the digestive organs, which have been alluded to above.

§ 40. The smallest *quantity* which has been taken with rapidly fatal results appears to be that which was given in a case quoted by Dr. Taylor. In it, the quantity is said not to have exceeded forty drops. But the patient was a child only a year old, and antidotes were administered about half an hour after it had taken the acid.

The symptoms, however, came on immediately, and the child died in twenty-four hours. Dr. Christison says, that one drachm proved fatal to a young man in seven days. It is always difficult, however, to ascertain the exact quantity which has been swallowed, since the immediate impression made upon the mouth by the contact of so corrosive a liquid naturally excites an instantaneous effort to get rid of it. The amount actually swallowed forms usually, therefore, but a small portion of what is taken into the mouth. It may, indeed, not reach the stomach at all, its action being expended upon the throat and œsophagus, and proving fatal by the inflammation and disorganization there produced.[1] Occasionally, also, it enters the air-passages. A case of recovery after an injection of sulphuric acid, given by mistake for castor oil, is reported.[2]

There are many recorded cases of sulphuric-acid poisoning; the shortest periods after swallowing the acid that death has occurred vary from half an hour to two hours and a half. The rapidity of the death will depend upon the character and extent of the primary lesion; for instance, if a perforation occurs in the stomach or intestines, death will rapidly follow from the consequent collapse; the tardy appearance of death will be influenced by the character of the tissue destroyed or organs impaired; for instance, if the secondary results from the destruction of tissue in the region of the throat cause inflammatory processes, and consequent contractions, by which the passage through the air-tubes or the gullet is obstructed, loss of life may occur from interference with the organs of respiration, or from starvation from an obstruction to the ingestion of food. Sometimes the limits of these inflammatory lesions have increased gradually in extent during weeks and months, and thus death has been indirectly caused at a period of time far remote from the moment when the acid was swallowed. There is, besides the case previously alluded to, one case on record referred to by Beck[3] in which death, the result of stricture of the œsophagus, did not occur until two years after the poison had been taken. It should be noted that the character of the contents of the stomach would have an important bearing upon the period at which death occurs, as well as

[1] This happened in a case reported by Dr. W. Hull, in the Lond. Med. Gaz., June, 1850.

[2] Ed. Month. Journ., April, 1850.

[3] See Taylor on Poisons.

upon the amount of destruction of tissue of that organ which is exposed to the action of the acid, except in those cases in which a large amount of acid is swallowed; for instance, if the contents were composed largely of milk and albuminous food, of fats and oleaginous substances, they might offer some protection to the walls of the stomach, which latter might escape serious injury. These foregoing remarks explain the wide range of time in which fatal results occur in persons who have taken about the same dose.

§ 41. *Post-mortem appearances.*—The following case will illustrate the effects of this poison when concentrated and swallowed. A man, thirty years of age, went to his closet in the dark, and drank a "good mouthful" of commercial sulphuric acid. He was bled shortly after, and the blood is described as being of a syrupy consistence. Milk and soapsuds were given to him, and brought on vomiting, but he died in two hours. The whole tongue was white and sphacelated, and the mucous membrane here and there detached. The fauces and œsophagus were of a grayish-black color; the stomach was perfectly black on both surfaces, and of a soft and pulpy consistence. The omentum majus was likewise partly carbonized, in consequence of the escape of the acid into the abdominal cavity. The upper portion of the small intestine was of a blackish color, and the mucous membrane swollen and indurated. The contents of the stomach yielded, on chemical examination, one drachm and seventeen and a quarter grains of free sulphuric acid.[1] In Mr. Traill's case, a washerwoman took a wineglassful of crude commercial acid of the specific gravity of 1.833, by mistake for ale. Although the proper antidotes were very soon administered, she died in one hour. A hole with ragged edges was found at the fundus of the stomach, and the adjacent tissue tore with the slightest touch. The rest of the mucous membrane was mottled with dark brown patches. There was also great inflammation of the peritoneum, from the escape of the acid.[2] When much acid has been swallowed, the stomach presents an appearance which is altogether peculiar and characteristic. It is as black as coal, and its tissue is softened to a jelly. The acid, softening the walls of the bloodvessels, allows their contents to escape, and then,

[1] Casper, Gericht. Leichenöff. 1 H. p. 117.

[2] Month. Journ., Aug. 1854.

acting upon the blood, gives it a dark color. That such is the source of the black color referred to is proved by the fact that it is not produced when sulphuric acid is introduced into a dead stomach.[1]

In general, the eroded and inflamed appearance of the mouth and throat is found to coexist with the blackened and disorganized condition of the stomach. But sometimes the poison does not reach the stomach, and, when this is the case with young children, death may take place from the local action on the fauces alone. In Dr. Hull's case, already referred to, it reached the lungs. The epiglottis was partially destroyed, the vocal cords and the mucous membrane of the trachea were softened, the left pleura was perforated, and a crust of sulphate of calcium formed upon the ribs. There was no trace of the acid, either in the œsophagus or stomach. The person was an adult female. It is probable, in such cases, that death takes place by suffocation on account of œdema of the glottis. Husemann reports two fatal cases in children of five years of age. The symptoms were chiefly those of inflammation of the throat and larynx, without any evidence of disorder of the stomach.[2] No section was made. In most cases of poisoning with sulphuric acid, there are also traces of its action left upon the skin, near the mouth, either from a portion escaping when swallowed, or from the corrosion of the vomited liquids. The marks thus left upon the skin are of a dark brown color, and of a leathery consistence. Where, however, the acid has been given in a spoon, the anterior portion of the mouth may escape entirely. In a case referred to by Dr. Taylor, the fauces, œsophagus, and stomach of an infant ten days old, were much corroded by sulphuric acid, but there was no appearance of injury to the mouth. This was probably owing to a spoon having been used, and the poison having been poured down the throat slowly, as the mucous membrane was extensively corroded at the back part. A case is reported, in which, although the acid was taken from a teacup to the amount of fifteen and a

[1] Orfila has established the fact that this acid, even when taken in a concentrated form, can be absorbed after it has been diluted in the stomach and intestinal canal, and united with liquids therein contained, or which had afterwards been swallowed; and that consequently in autopsies all the viscera and the urine should be examined for the presence of this acid, even though nothing was found either in the alimentary canal, vomited matters, or the feces.

[2] Journ. f. Pharmakodyn., ii. 166.

half drachms, there was not the slightest vestige of a stain on the outside of the lips, angles of the mouth, cheeks, neck, or hands, nor upon the clothing.[1]

There is a case on record[2] where a woman swallowed from a cup about a tablespoonful of strong sulphuric acid; the usual symptoms appeared, and she died in two days. The mucous membrane of the cheeks, gums, and tongue was not excoriated at any part.

We find a case of gelatiniform softening of the stomach, with perforation of the fundus, and effusion of a brownish liquid into the peritoneal cavity, reported by Dr. Casper. In this case there had existed suspicion of poisoning, which his opinion from the result of the examination set at rest. Were it admitted, in the absence of direct proof by chemical analysis of the contents of the stomach, that an inflammatory condition, a softening, or ulceration of the stomach could be regarded, apart from any corrosion of the mouth or fauces, as presumptive evidence of poisoning, we should be led undoubtedly into many grave mistakes. Spontaneous softening of the stomach in infants, and ulceration and perforation of this organ in adults, are not rare events, but their characters are far different from the charring and disorganization produced by sulphuric acid. We think that in no case is an opinion warranted that sulphuric acid has been swallowed, unless it can be clearly traced by its effects from the mouth or fauces to the stomach.

Dr. Norman Moore[3] reported the *post-mortem* appearances in a case of poisoning of a man, who lived only one and a half hours after swallowing half a pint of the oil of vitriol of commerce. As this report was carefully made, and because there was evidence that the poison corroded the tissues after death, as well as before, we present it in detail. The mucous membrane of tongue and fauces was grayish, not charred. The walls of the gullet were soaked with the acid, which had even diffused into the pericardium and posterior walls of the heart. Neither gullet nor diaphragm was perforated. One-eighth of an inch in thickness of the bottom of the lung substance was hardened by imbibition of the acid. The stomach and upper portions of the small intestine were quite rotten

[1] Dr. Walker, Ed. Month. Journ., June, 1850.

[2] Dr. Chowne, Lancet, July 10, 1847, p. 36.

[3] Br. Med. Journal, London, 1879, pt. i, p. 430.

from the action of the acid; the lower portions of the small intestine, and also of the large intestine as far as the rectum, were grayish and evidently had been attacked by the acid. The surface of the liver was also hardened, and all of the abdominal viscera were more or less acted upon by the corrosive, and the saturation had even affected the outside wall of the abdomen; the neighborhood of the navel being grayish also. Many of these changes were undoubtedly, in the opinion of Dr. Moore, due to the action of the acid upon the tissue after death.

It has already been mentioned that in a case of poisoning by this acid the blood had a syrupy consistence. This condition is stated by Casper to be the ordinary one. He adds that the blood is generally of a cherry-red color, and that it has an acid reaction. This acidity has also been noticed in a pericardial effusion, and in the amniotic liquid of a pregnant female poisoned by sulphuric acid. It would appear that after death by this poison bodies remain fresh and without smell for an unusually long time; a fact which Casper explains by stating that the acid continues, until exhausted, to neutralize the ammonia which is given off in decomposition.

We also sometimes see the inflammatory action of the acid upon the organs due to its having been absorbed and carried to them through the circulation; thus the kidneys are almost always irritated by the acid, sometimes to such an extent that bloody urine is secreted by them, and the urine is almost always albuminous and during life gives evidence of this irritation of the kidneys; Mannkopf in one of his cases[1] mentions having found a fatty liver, as in the case of many of the other irritant poisons.

In cases of subacute poisoning when life has been sufficiently prolonged, we always find the results of the previous destruction of tissue, in the form of cicatricial contractions or strictures of the œsophagus, stomach, intestines, or trachea, and hypertrophy of the walls of these organs, wherever they were attacked by the acid; also we frequently see ulcerations, which either have never healed or have been produced secondarily.

§ 42. *Poisoning by ink.*—A drunken soldier had given to him a large glass of ink, under the pretence that it was porter. He drank it, and, after sleeping for an hour, awoke in the most violent

[1] Wien. Med. Wochensch., 1862, No. 35, and 1863, No. 5.

pain. He suffered extreme weakness, headache, and painful cramps in the thighs. After four or five hours he commenced vomiting a pasty mass mixed with ink, which gave strong indications of sulphuric acid. Mucilaginous and saccharine beverages were given him, and after a short time he improved. He was convalescent on the third day, but still complained of weakness, trembling, and an oppressive pain in the back of the head.[1] The presence of sulphuric acid in the ink is explained by the decomposition occurring between the sulphate of iron and tannic acid, of which some inks are made.

§ 43. *Chemical examination.*—Concentrated sulphuric acid is known by its oleaginous appearance, great specific gravity, its property of setting free the carbon of organic substances, such as a splinter of wood or a granule of sugar (thus charring them), and by the evolution of heat on its being mixed with water. When *diluted*, the best evidence of its presence is obtained by the addition of any of the soluble salts of barium, the sulphate of barium being immediately precipitated in the form of a heavy white powder. The precipitate is insoluble in nitric and hydrochloric acids. Further proof of its presence may be obtained by igniting the precipitate with carbonate of sodium or with vegetable charcoal; by the latter the sulphate is reduced to a sulphide, which may be shown by its blackening a bright silver surface on being moistened with water. In testing a solution for sulphuric acid by means of a salt of barium, care should be taken that the liquid does not contain too much nitric or hydrochloric acid, as salts of barium, which readily dissolve in water, are almost insoluble in these acids. The liquid containing the precipitate may be diluted with distilled water, which will redissolve all the other salts but the sulphate. Solutions containing sulphuric acid or a sulphate are also precipitated by a solution of acetate of lead, the insoluble sulphate of lead being formed. In order to ascertain whether the sulphuric acid exists in a free state or combined with a base in the form of a sulphate, a portion of the liquid should be evaporated upon a water-bath until it no longer diminishes in bulk, or until it has become perfectly dry; if sulphuric acid be present in a free state, it can only be concentrated at the temperature of a water-bath, so that the residue in

[1] Am. Journ. Med. Sci., April, 1854, from Pharmaceutical Journal, Oct. 1853.

the evaporating dish will have the syrupy consistency of concentrated sulphuric acid, and any organic matter which may be present at the same time will be charred. The free acid may then be separated from any sulphate which is always present in animal fluids and tissues, by extracting the residue with absolute alcohol and filtering; the filtrate will contain the free sulphuric acid, while the sulphates will remain undissolved upon the filter-paper. The filtrate should then be evaporated upon a water-bath to drive off the alcohol, water added, and the solution tested for sulphuric acid, which can be recognized by its intense acid reaction to test-paper, and by the precipitate formed with a solution of chloride of barium or acetate of lead, and after concentration by its charring organic matter. During the evaporation of a solution of sulphuric acid in the presence of organic matter, fumes of sulphurous acid, having the odor of burning sulphur, are evolved.

§ 44. When the liquid to be examined is mixed with *organic matters*, it must be filtered; or, if turbid and thick, it should be first boiled with distilled water, and then filtered, before the above tests are applied. In many cases of poisoning by this agent, antidotes have been used, which may have completely neutralized the acid. Hence, although it may not be found in a free condition, the presence, in any considerable quantity, of sulphates, when corroborated by the corrosive effects peculiar to the acid, will leave but little doubt of its having been used. In such a case comparative analyses must be made to ascertain the amount of sulphuric acid in the suspected fluid or tissue, and in an equal amount of other fluid or tissue similar in kind to that suspected to have contained free sulphuric acid before it had been neutralized by antidotes. If the amount of sulphate found in the suspected substance very greatly exceeds that found in the material taken for comparison, it forms a presumption in favor of the previous existence of free acid in the suspected substance. It should be remembered, also, that, if life have been at all protracted, the poison may have been eliminated in various ways. A case has been reported by Mr. Letheby, in which, during the first four days after an ounce of concentrated sulphuric acid had been swallowed, a large quantity was passed in the urine. This same fact has been established by Orfila, in experiments upon dogs.

According to these experiments:—

1st. In poisoning by sulphuric acid, it is sometimes possible to determine after several months and even years the presence of the concentrated acid.

2d. When the acid is very weak, it is found after some months saturated by ammonia, which has been disengaged by the putrefying matters, and, in this case, the presence of sulphate of ammonium, which may be found, does not necessarily prove that there has been poisoning by sulphuric acid, though certain probabilities of such poisoning might be established.

§ 45. *Stains on clothing.*—The stains on blue and black cloth, made by dilute sulphuric acid, are at first red, and afterwards brown, and the stuff is corroded. Concentrated acid produces a brown stain at once, owing to the destruction of the fibre and the charring of the material. When a drop of dilute acid falls upon cloth, a portion of the water gradually evaporates, until the acid reaches a certain degree of concentration; hence the change in the color of the stain from red to brown. The color of black leather is not altered, but the substance is partially destroyed. The stains on all textile fabrics remain moist for a considerable time if the acid have been strong, and, owing to the attraction of the acid for water, they have an unctuous feel. The acid may be detected in these stains after the lapse of many years. Dr. Taylor has detected it in spots made upon a black cloth dress *twenty-seven* years previously. The stains should be cut out, digested in distilled water, and then the liquid, after filtration, be tested by the reagents already mentioned. An unstained portion of the cloth should be submitted to the same test, since many articles of clothing yield slight traces of sulphates when boiled in water.

§ 46. *Aromatic sulphuric acid* (elixir of vitriol).—Besides sulphuric acid, this preparation contains alcohol and aromatics. It has about twenty per cent. of officinal sulphuric acid by weight (U. S. P.). In an overdose, it produces the same effects as sulphuric acid.

§ 47. *Sulphate of indigo.*—This is a solution of indigo in Nordhausen, or fuming, sulphuric acid. In addition to the other symptoms of poisoning by a corrosive liquid, the deep blue and subsequently greenish color of the vomited matters will at once

betray the agent that has been used. In some cases the urine has had a bluish tinge.

II. *Nitric Acid.* (Aqua fortis.)

§ 48. Concentrated nitric acid, as met with in commerce, varies in color from a light yellow to a deep orange-red, owing to the presence of nitrous or hyponitrous acid, but the pure acid is colorless. Red fumes of nitrous acid gas are given off when a few copper-filings are treated with nitric acid. It produces a yellow stain upon the skin, mucous membranes, or cloth, which changes to an orange upon being neutralized with an alkali; this yellow color is due to the action of the nitric acid upon albuminous matters, with which it forms xanthoproteic acid. Poisoning by nitric acid is much rarer than by sulphuric acid, since the odor of the fumes evolved from the strong solutions permits its ready detection before any of the acid has been taken into the mouth; hence a case of accidental poisoning by this acid is exceedingly rare, although it is very extensively used in the arts. The fumes arising from concentrated nitric acid are also poisonous when inhaled, and fatal cases have been recorded from the inhalation of nitrous fumes in laboratories and factories.

§ 49. The *symptoms* produced by swallowing strong nitric acid do not differ essentially from those which have been already mentioned as caused by sulphuric acid. There is the same intense burning pain in mouth, throat, and stomach, the same violent efforts to vomit, and urgent constitutional symptoms. The lining membrane of the mouth is stained white, and then yellow or brown, and the enamel of the teeth is attacked. The soreness and swelling of the mouth and throat, the difficulty of swallowing and of respiration, the thirst and salivation, and the excessive pain and distress which are more especially referred by the sufferer from this poison to a diffused abdominal pain rather than at the pit of the stomach or epigastrium, as in the case of sulphuric-acid poisoning, are the most prominent symptoms. The air-tubes are also more likely to be affected by the volatile fumes of nitric acid rather than by the non-volatile sulphuric acid, which does not produce this effect unless by direct contact with the liquid. Sulphuric acid is also more liable to produce constipation, and nitric acid to cause diarrhœa. The eructation of nitrous vapors from the stomach

is a characteristic symptom. The vomitus is yellow rather than brown or black, as is the case in sulphuric-acid poisoning. The abdominal pain may be entirely absent, as in two recorded fatal cases. If the person survive long enough, large portions of the lining membrane of the fauces and œsophagus become detached and are thrown up, together with altered blood and shreds of mucus. Similar matters may be discharged by stool. The *diluted acid* occasions the same symptoms in a more moderate degree. Although the immediate corrosive effects of the acid may be recovered from, death may occur subsequently from exhaustion and the injury done to the digestive powers. Dr. Black referred, in some remarks before the Royal Medical and Chirurgical Society of London, to the case of a girl who, "in a fit of despondency, swallowed some strong nitric acid. She left the hospital, but died many months afterwards, but so altered in appearance, that she resembled a woman eighty years of age. She was kept alive for seven weeks entirely by spring water." The œsophagus was nearly closed by the strictures which had resulted from the healing of the ulcerations produced by the acid.[1] The period at which this poison proves fatal varies, therefore, according to its dilution, from a few hours to several months.

§ 50. Two drachms is the smallest quantity known to have destroyed life. This was nearly the quantity swallowed in a case reported by Dr. J. M. Warren. Death ensued on the fourteenth day. (It is stated that three drachms were taken into the mouth, but most of it was spit out.) Dr. Taylor refers to the case of a boy who died in thirty-six hours after taking *two* drachms of this acid. An instance, in which it was poured into the ear of a sleeping person, and caused death after some time, is related by Dr. Morrison.[2] Mr. James Haywood, a chemist, lost his life by inhaling the *fumes* of mixed nitric and sulphuric acids in consequence of the breaking of a carboy which held the mixture. The symptoms, which did not appear for more than three hours, consisted mainly of difficult respiration. Death took place in eleven hours. On examination, a considerable effusion of blood was found in the bronchial tubes, and their lining membrane and that of the trachea were congested. The larynx was not examined.[3] As in

[1] Lancet, 1850.

[2] Dublin Journal, vol. ix. p. 98.

[3] Lancet, April, 1854, p. 429.

the case of other corrosive poisons, death from nitric acid may occur within a few hours (from two to twenty-four hours) or after several months, according to the quantity and strength of the acid, and the vigor of the patient.

§ 51. *Post-mortem appearances.*—The stains made by nitric acid upon the mouth and lips are usually of a deep yellow color; as these consist of a sphacelation of the lining membrane, they are easily detached and the subjacent surface is found even and glistening. If the person have survived several days, they may have been already cast off. The same appearance is found in the fauces, and more or less of it in the œsophagus. The stomach is softened, sometimes perforated, its internal surface is of a greenish-yellow, but sometimes of a black color, owing to the erosion of vessels and the effusion of blood, and the mucous membrane is ulcerated or destroyed. In Dr. Warren's case, the patient was a negress, who swallowed the poison with the hope of destroying her child, supposing that she was pregnant. She died on the fourteenth day. On section, there was observed great rigidity; upon the middle of the tongue, a large, yellowish, smooth patch; some redness of the epiglottis; the œsophagus was healthy for the first two inches, but below this it was found exceedingly soft, of a greenish color internally, purple externally, and full of coagulated blood. The stomach was in a similar though much worse state; externally, it had the same purple color, and was universally adherent to the neighboring parts by recent lymph, except at the left extremity, where there were old and close adhesions to the spleen; internally, it was of a greenish-yellow color, emphysematous, and so perfectly softened and pliable, that it could not be separated from the surrounding parts without giving way in every direction; the anterior face became detached from the rest of the organ to a great extent when the abdominal parietes were raised; its cavity was filled with recent coagulated blood, and the open orifices of several vessels were distinctly seen on the inner surface. The intestine contained blood throughout the first two or three feet, but was otherwise uninjured. In a case observed at the Hôtel-Dieu, at Lyons, the stomach was distended with gas, and perforated in the *cul de sac;* the opening, however, was partially plugged by the spleen, which had become adherent over it, but which itself was much corroded. In the small intestine, there were numerous sloughs. In another case, where

the person survived the poisoning fifty-four days, the stomach was converted into a vast abscess, with fungous walls made by adhesions among the adjacent viscera. The natural shape and structure of the organ could no longer be distinguished.

In a case of poisoning by nitrous fumes,[1] the endocardium was found much inflamed; the lungs were nearly destroyed, so that the lung tissue was in many places soft like jelly. One portion of the left lung showed marked congestion and was stained yellow. The blood in the lung tissue had an acid reaction and the odor of nitrous fumes. Free nitric acid was detected in the lungs, but not in the other organs.

§ 52. *Chemical examination.*—Nitric acid may be readily known by its physical properties already mentioned, and by the red fumes which are given off when it is poured upon copper, tin, or mercury in contact with air. Fortunately, nitric acid, unlike sulphuric and hydrochloric acids, is not a normal constituent of the animal economy, so that its detection in toxicological cases is much easier than that of the other two acids, which are normally present in the animal fluids and tissues in the form of sulphates or chlorides, although there are no reagents which precipitate it. Hence it is not so important to distinguish between the free and the combined acid.

Nitric acid when free can easily be detected by the following tests: (1) A piece of copper introduced into the acid will be dissolved, forming a greenish-blue solution of nitrate of copper, and red fumes will be evolved. If the solution be very dilute, warming will facilitate the reaction. If the nitric acid be combined with a base, as nitrate of potassium for example, it can be freed from its combination by warming with sulphuric acid. (2) A crystal of ferrous sulphate forms a brown compound with nitric acid. If the nitric acid is free, the brown compound will form around the crystal. If it be combined, the best way to perform the test is to dissolve a small crystal of the sulphate of iron in the fluid and then pour concentrated sulphuric acid down the side of the vessel, so that it forms a separate layer of fluid at the bottom; the nitrate is decomposed at the junction of the sulphuric acid and fluid, and a brown zone forms at this point. (3) A solution of sulphate of aniline gives a purple or violet color, when treated in the same way as

[1] Annales d'Hygiène, 1875, p. 345.

ferrous sulphate with nitric acid or a nitrate. (4) A crystal of brucine or a solution of a salt of brucine gives an orange color with nitric acid. (5) Metallic gold is insoluble in pure nitric acid, but is readily soluble in a mixture of nitric and hydrochloric acids (*aqua regia*). Therefore, if to a solution of nitric acid or a nitrate, a little hydrochloric acid be added, and a piece of gold foil be introduced into the mixture, it will be dissolved, and we will obtain a solution of terchloride of gold, which can be tested by the appropriate tests.

§ 53: When organic mixtures, like vomitus or contents of stomach, are to be tested for nitric acid, the yellow stains, intensely acid reaction, and nitrous fumes and odor will usually leave no doubt as to its presence. In order to isolate it, however, the fluid to be tested should be treated with an excess of calcic carbonate (chalk), which forms with the nitric acid nitrate of calcium, which is a deliquescent compound and readily soluble in alcohol. This mixture should then be evaporated to dryness upon a water-bath, the residue extracted with strong alcohol and filtered; evaporate the filtrate upon a water-bath, when a residue of impure nitrate of calcium will remain; this can be dissolved in water and tested by the above tests. This solution can also be decomposed by carbonate of potassium or carbonate of sodium, and the characteristic crystals of the nitrate of potassium or nitrate of sodium obtained upon careful evaporation of the filtrate. These crystals should always be taken to court as the *corpora delicti* in any criminal case.

§ 54. *Stains on cloth.*—The spots made upon colored cloth by nitric acid are more or less yellow, but become brown after a time, the texture of the cloth is destroyed, and the spot, unlike that made by sulphuric acid, soon becomes dry. To determine the presence of nitric acid, the stain may be cut out and digested in distilled water. If the liquid have an acid reaction, it should be then neutralized with potash, and allowed to crystallize. The crystals may be examined as before, by heating with copper-turnings and moistening with sulphuric acid. An unstained portion of the cloth should be examined in the same manner. Stains made by nitric acid will not furnish evidence of its presence after a few weeks have elapsed, the acid being much less permanent than the sulphuric. Hence the necessity of proceeding at as early a period as possible to the examination of any suspicious stains upon a dress. Dr. Christison,

however, has obtained evidence of the presence of the acid in stains on cloth made seven weeks before, and Dr. O'Shaughnessy after an interval of some months.[1]

III. *Hydrochloric Acid—Muriatic Acid.* (Spirit of Salt.)

§ 55. *Symptoms.*—The reported cases of poisoning with this acid are few in number; but they present a strong analogy in their *symptoms and post-mortem appearances* with those of sulphuric acid poisoning. Immediately upon swallowing it there is a burning sensation from the mouth to the stomach, but especially in the throat, attended with a feeling of suffocation and followed by uncontrollable vomiting. Deglutition is almost impossible, all efforts to swallow bringing on vomiting; the voice is low and the respiration frequent and labored. The tongue and fauces are usually covered at first with a whitish pellicle, which afterwards becomes detached, exhibiting corroded spots underneath. In a case observed by M. Guérard,[2] a woman aged 24 years, who had swallowed about fifty-three grains of concentrated hydrochloric acid, presented the above symptoms. She however survived a considerable time. The matters vomited on the second night did not present any acid reaction, although no chemical antidote appears to have been administered. Death took place two months after the poison had been swallowed; and some time previous, portions of corroded mucous membrane had been discharged both by vomiting and by stool. Profuse salivation also was observed in this case, and in the beginning white vapors were exhaled from the mouth. The same symptoms and the same prolongation of life were noticed in a case which became the subject of judicial investigation in Belgium, and which is commented upon by Orfila.[3] In two cases referred to by Dr. Christison, and in another, of a child, by Orfila,[4] death took place within twenty-four hours. In this latter instance, the acid was poured down the child's throat by its stepmother, as confessed by her after her condemnation. A case of recovery after swallowing an ounce of strong hydrochloric acid, is reported in the *Lancet* for 1850. In this case the stomach-pump was used, contrary to the usual caution enjoined in poisoning

[1] Guy's For. Med.
[2] Ann. d'Hygiène, tome xlviii. p. 415.
[3] Ibid., tome xi. p. 35.
[4] Ibid.

by mineral acids. A Hindoo, while intoxicated, swallowed about two ounces of hydrochloric acid, but rejected a portion of it by vomiting. Twelve hours afterwards he presented the following symptoms: The head was thrown backwards; the respiration frequent and moaning; the gums were pale and the teeth unaltered; the tongue also was pale, and, near its centre, deprived of its epithelium; the skin was cold, the pulse small and frequent, the epigastrium tender, and the urine scanty. There was neither vomiting nor purging. In twenty-four hours death took place, the mind remaining clear until the last. The urine contained a large proportion of hydrochloric acid. In a case under the care of Dr. Budd, a woman 63 years of age died in eighteen hours from the effects of half a fluidounce of hydrochloric acid, taken with suicidal intent. Vomiting, collapse, whitening and abrasion of the lips, mouth, and fauces were observed; swelling of the throat and inability to swallow, with stridulous breathing and thick inarticulate voice, and intense epigastric pain were also noted. Death took place by exhaustion, but without loss of consciousness until near the last. In May, 1859, a woman sixty three years of age died at King's College Hospital, within eighteen hours after swallowing half an ounce of the strong acid.

§ 56. *Post-mortem appearances.*—These vary according to the length of time the person has survived, but bear a general resemblance to the effects produced by the other strong mineral acids. The digestive mucous membrane is covered with whitish superficial sloughs, which subsequently become of a darker color, and are found in all stages, lying loose or partly detached, and the mucous membrane inflamed, softened, or corroded. In some of the above cases, all the coats of the stomach were destroyed in spots, and perforations resulted. In Guérard's case, there was slight peritonitis. It is important, however, to note that the peritonitis, resulting from perforation of the stomach, only occurred in those cases which survived a long time. In Dr. Budd's case the force of the poison was spent upon the mouth, fauces, and larynx. The mucous membrane of these parts was whitened and softened, the soft palate and tonsils swollen, and a portion of the lining membrane of the larynx was entirely removed.

§ 57. *Chemical examination.*—If any of the liquid which has been taken remain, it will be easy, if it is hydrochloric acid, to

detect its nature, since the physical characters and chemical reactions of this agent are very striking.[1] It throws down, if sufficiently dilute, with nitrate of silver, a dense, white, curdy precipitate of the chloride of silver, which assumes a violet color when exposed to light, and is completely insoluble in nitric acid, but dissolves readily in ammonia. Its detection, when mixed with organic matters, is not easy, owing to its tendency to adhere closely to them; but in medico-legal researches we are further exposed to the error arising from the presence, *normally*, of free hydrochloric acid in the stomach. As the quantity of this natural constituent of the body is subject to many variations, and since as much as four or five grains of the pure acid have been obtained from a pint of the fluid of water-brash, it is evident not only that the detection of free hydrochloric acid in a case of supposed poisoning is no evidence that it has been swallowed, but that it is extremely uncertain what quantity it would be necessary to demonstrate before we could feel satisfied that it was not normally present. In the Belgian case, before referred to, it was supposed by the chemists that they had established the fact of poisoning by this acid, although the person had survived two months; a subsequent analysis, however, of the stomach of a person of the same age, who had died of phthisis pulmonalis, gave precisely the same result. Moreover, if the mixture be neutral, it becomes necessary to use sulphuric acid to decompose the chloride which has been formed. But as chloride of sodium (common salt) is almost invariably present in the stomach, and is indeed a natural constituent of most of the secretions, the detection of hydrochloric acid will afford no indication of the manner in which it was introduced.

[1] The following means of detecting hydrochloric acid in cases of suspected poisoning has been suggested. (J. Bouis, Journal of Applied Chemistry.) Filter the liquid through linen and paper previously moistened with acetic acid, and then introduce a few crystals of chlorate of potash and some gold foil. Heat the whole over a water-bath an hour or more, and the presence of the free acid is determined by the quantity of gold dissolved by the liquid. Chloride of tin may be used to detect even a few faint traces of gold which may have been dissolved. Diluted liquids should, before applying the above process, be concentrated by evaporation. A very few centigrammes of hydrochloric acid can be detected in a large quantity of liquid. The chlorides of sodium, potassium, and the like do not produce similar reactions.

§ 58. For these reasons, the chief evidence of poisoning must be obtained rather from the symptoms during life, and the *post-mortem* investigation, than from a chemical analysis. We merely subjoin, to complete the subject, the following process when the matter to be examined is acid, taken from Dr. Christison's work on Poisons: "Boil it with water; if necessary, filter, and distil it with a gentle heat till the residue acquire the consistence of a very thin syrup. Subject the distilled liquor to the tests for diluted hydrochloric acid. It will seldom be found there, however, because it is apt to be retained by the coexistence of organic matter. If it be not found, add to the thin extract in the retort a slight excess of a strong solution of tannin, filter, and distil the filtered liquid by means of a hot bath of solution of chloride of calcium (consisting of two parts of crystallized salt and one of water), taking care that the temperature of the bath never exceeds 240°, and stop the distillation just before the residuum becomes dry. Examine now the distilled liquor with the tests for diluted hydrochloric acid."

IV. *Oxalic Acid.*

§ 59. Oxalic acid is one of the more common poisons and is one of the most rapidly fatal and certain of ordinary poisons. Its intensely sour taste generally prevents its administration with homicidal intent, but very dilute solutions can be administered in the place of ordinary acid drinks or medicines. The sour taste does not always prevent its being taken accidentally. On account of its resemblance to Epsom salts, most of the cases of accidental poisoning have been due to its being mistaken for this compound. If oxalic acid and Epsom salts crystals be mixed together they cannot be distinguished from each other by their appearance, but they can easily be separated by alcohol, in which the oxalic acid is readily soluble, while the sulphate of magnesium is insoluble.

§ 60. *Symptoms.*—After the hot and sour taste in the mouth, vomiting is usually the first symptom, and is attended with burning pain and constriction in the throat and stomach, although it does not always occur, and in some cases the pain is absent. The vomited matters are dark-colored, and contain blood and sometimes membranous shreds, highly acid, greenish, dark-colored, or bloody mucus. Abdominal pains are also present, but oftentimes the symptoms of nervous prostration and collapse with stupor and lack

of sensation follow so rapidly the ingestion of the poison, that the gastric disturbance is not a very prominent symptom, and the pain is not always observed. If, however, the symptoms of collapse are not present early in the case, the sickness of the stomach and vomiting are very distressing, the abdominal pain is severe, the extremities are benumbed and drawn up, the surface is cold, and the pulse irregular or imperceptible. A degree of stupor or drowsiness is sometimes observed. The urgency of the symptoms depends upon the degree of dilution in which the salt is taken, for in the case of weak solutions the local irritation upon the mouth, throat, and stomach is less marked, and the symptoms of nervous prostration predominate, though they may come on more slowly.

In a case quoted by Dr. Christison, no vomiting occurred for seven hours, except when emetics were administered. The person had taken half an ounce dissolved in ten parts of water, and diluted immediately afterwards with copious draughts of water. In another case, a man swallowed an ounce, and immediately drank warm water; he was not seen until fourteen hours after he had taken the poison, and in the mean time had travelled a distance of ten miles.[1] When, on the other hand, the acid is given in a highly concentrated form, the irritant and corrosive action upon the mouth, throat, and stomach becomes the most prominent symptom, and may be immediately followed by collapse, stupor, complete paralysis, and death. In this latter case the symptoms are not dissimilar to those of any other irritant or corrosive.

§ 61. *The rapidity with which a fatal result ensues* varies a great deal. In some cases the action of the poison is extremely rapid. Mr. Iliff reports a case in which death appears to have been immediate. The wife of a chemist drank a saturated solution of oxalic acid in her husband's shop; she was found dead by the side of the counter, where she had probably fallen and died without a struggle.[2]

Dr. Christison quotes a case in which a young man survived hardly *ten minutes;* another, in which a woman, who swallowed two ounces, died in *twenty minutes;* and Dr. Taylor refers to a case where death ensued in *three minutes.* The quantity taken in the last case could not be ascertained. Pereira also mentions a

[1] Lancet, Sept. 1845, p. 293.

[2] Lancet, Oct. 1845.

case which he examined, in which a man died in twenty minutes after swallowing oxalic acid in mistake for Epsom salts. Although death usually occurs within a few hours, cases are mentioned in which it was postponed for several days. Dr. Jackson reports one in which the poison did not prove fatal until the tenth day;[1] and in a case described by Dr. Frazer, death occurred from its secondary effects upon the twenty-third day. Some instances of recovery are reported. An interesting one was observed by Dr. Didama, in which a woman dissolved two large tablespoonfuls of oxalic acid by mistake for Epsom salts, in a small quantity of water, and took it on an empty stomach. Some twenty minutes afterwards she vomited, at first the solution she had taken, and then a dark-colored bloody-looking fluid, in which were numerous white membranous patches. Ipecacuanha and afterwards prepared chalk were administered to her, and in about an hour she was found quiet and nearly free from the intense burning pain in her stomach and throat. She subsequently vomited again, and discharged from her bowels a large amount of matter resembling that which she had vomited. She soon recovered entirely, and returned to her work. A similar case, in which an ounce was taken, and the patient recovered in eighteen days, is reported in the *Lancet*;[2] and a few others are referred to by Dr. Taylor.

§ 62. The only manner in which the *quantity capable of destroying life* can be approximately ascertained, is by reference to such cases as have proved fatal in the absence of medical assistance. The smallest quantity which has proved fatal is recorded by Dr. Barker,[3] in which it was estimated roughly, that about a drachm, or sixty grains, were eaten "as a dry solid" by a boy of sixteen years, who was found an hour afterwards in an insensible pulseless condition with the jaws spasmodically closed. He had vomited some bloody mucus; his tongue and lips, though not injured by the local contact of the acid, were unusually pale. He died within nine hours from the time of taking the poison. Generally speaking, three drachms is a fatal dose, though it is very difficult to obtain a definite knowledge of how much has been taken, since this substance

[1] Bost. Med. and Surg. Journ., vol. xxxi. p. 17.

[2] July, 1846, p. 39.

[3] Lancet, Dec. 1, 1855, and Taylor's "On Poisons," Phila. 1875.

is usually, as before remarked, mistaken for Epsom salts or common salt, and, not being as agreeable to the taste as either of these salts, the whole quantity taken out is not generally swallowed, but thrown away. So again the concentration of the solution of the poison may increase the danger from the initial symptoms, and in the case of a diluted solution, medical skill may combat the more tardy appearance of the general constitutional symptoms and thus avert a fatal catastrophe from a comparatively large dose. It, however, by no means follows that a smaller quantity might not be attended with a fatal result, since some persons appear to have been far more seriously affected than others by equal amounts of this poison. This is proved by the above case of a lad, sixteen years of age, who ate about a drachm of dry oxalic acid, and died in eight hours.

§ 63. The *binoxalate of potassium*, salt of sorrel, or essential salt of lemons, produces the same symptoms as oxalic acid, and destroys life as readily. An instance is reported by Chevallier, in which death took place in *ten minutes*.[1] In another case, a third dose of a drachm and a half proved fatal in an hour.[2]

§ 64. *Post-mortem appearances.*—It is stated that death may ensue from oxalic acid, and yet no traces of its action on the alimentary canal be observable on section; this occurred in a case where an ounce had been taken,[3] and a similar case has been seen by the writer. This is certainly not the ordinary result. According to Dr. Geoghegan, who examined the stomachs of three persons poisoned with this acid, the mucous membrane of the stomach was softened, with various shades of brown discoloration, and erosion or gelatinization; there was a brownish-black, ramiform vascularity of the submucous tissue, and the contents of the stomach were of the color of coffee-grounds, owing to the action of the acid upon the mucus and coloring matter of the blood. In Dr. Jackson's case small ulcerations and thickening of the mucous membrane were observed, together with permanent redness. Hence the action of the acid may be chiefly expended upon the mucous coat of the stomach, no actual corrosion being observed. In an instance reported by Mr. Letheby, the coats of the stomach were so softened, that it could scarcely be handled without lacerating it, and in another, re-

[1] Ann. d'Hyg. 1850, vol. i.
[2] Ibid. 1842.
[3] Lond. Med. Repos., vol. iii. p. 380.

ferred to by Dr. Christison, it is said that this organ was perforated.[1] In some of these cases, however, it is evident that the conditions spoken of may have resulted from the long contact of the acid with the coats of the stomach after death, since even so feeble an acid as that naturally contained in the stomach is capable of producing similar results. It is certainly not the ordinary effect of oxalic acid. The œsophagus is also in many cases found altered, having a scalded or boiled appearance. It is pale, brittle, corrugated, and abraded in some places.

More recently a case has been reported by Dr. A. Wood, in which the stomach presented a large irregular aperture. As sufficient details of the section are not given, and the viscera do not appear to have been examined *in situ*, it is possible that this hole may have been artificially produced. The autopsy was made thirty-five hours after death.[2]

Kobert and Küssner[3] report that the urine contains during life a strong reducing agent of unknown nature, calcic oxalate crystals in abundance, and usually albumen and renal tube casts. A peculiar pathological appearance, according to these same writers, is the deposit of similar calcic oxalate crystals within the renal tubules.

There is a certain condition of the system called oxaluria, which is associated with certain diseases attended with impaired oxidation, in which traces of calcic oxalate may be recovered from the urine, but the insignificant amount, as compared with that in which a poisonous dose of oxalic acid has been taken, would settle any doubt, if such existed. This doubt, however, shows the importance of making an estimate of the amount of oxalic acid which should be attributed to a case of poisoning.

§ 65. A case of suspected poisoning by this acid was reported[4] in detail by Dr. J. C. White, of Boston, to the Society of Medical Sciences. The *post-mortem* appearances were as follows:—

Twenty-four hours after death the stomach, intestines, spleen, kidneys, liver, and part of the pancreas were removed from the body

[1] Med. Gaz., vol. xxxv. p. 49; Lond. Med. Repos., vol. xi. p. 20.

[2] Edinburgh Monthly Journal, March, 1852.

[3] Virchow's Archiv, Bd. lxxxviii. 109, and Wood's Materia Medica and Toxicology, p. 199. Phila. 1882.

[4] Boston Med. and Surg. Journ., Jan. 27, 1870, p. 57.

and brought to Dr. White for examination, and forty-eight hours after death appeared as follows:—

The *stomach* which, with six inches of œsophagus attached, was tied at both extremities, contained ten or twelve ounces of grayish fluid of the consistence of gruel, with an acid smell and reaction. It was marked externally by dark streaks corresponding to the position of the bloodvessels, and by large stains of a scorched appearance near the œsophagus. Within, its lining membrane, for an inch or more surrounding the œsophageal opening, was of the same black color as that tube, and the bloodvessels in the depending portions filled with black blood were visible through it. Elsewhere, and generally, the mucous membrane was of its natural color or paler than usual. The structure of the mucous membrane and other tissues was unchanged, except in the lower portions, where it was soft and thin (probably a cadaveric change). There was more or less mucus attached to its inner surface.

The *œsophagus* exteriorly was of a uniform deep slate color. Its interior surface was stained of a deep black, the same color penetrating in parts to and through the muscular tissue. The lining membrane was not materially softened nor easily separated from the tissues beneath.

In the *intestinal canal* there was nothing unnatural besides the usual cadaveric softening of the mucous membrane and the reddened patches.

All the *other organs* were apparently healthy; the blood contained within them being generally bright red, or vermilion.

Christison states that after concentrated doses the "stomach is found to contain black extravasated blood, exactly like blood acted upon by oxalic acid out of the body."

§ 66. *Chemical examination.*—The crystals of oxalic acid often resemble those of sulphate of magnesium (Epsom salts), and the former substance has hence often been taken by mistake for the latter. They are permanent, flattened, transparent, four-sided prisms, soluble in water and alcohol. If crystallized rapidly from concentrated solutions, it separates in the form of fine silky needles. The crystals of oxalic acid, when heated in a tube or upon a plate, at first appear to boil, and finally are entirely dissipated; the apparent boiling is due to the formation of carbonic acid, carbonic oxide, and water, by the decomposition of the oxalic acid. The

crystals do not char. [This is an important method of distinguishing oxalic acid from most other similar crystals.] If the test be performed in a test-tube, the carbonic acid gas formed can be conducted into lime-water, and a precipitate of calcic carbonate obtained. If a solution of oxalic acid be treated with lime-water or a solution of the acetate of calcium, a precipitate of calcic oxalate is formed; this precipitate is very characteristic, since it occurs in the form of octahedral crystals, which are very perfect if the precipitation has taken place slowly; it is insoluble in an excess of oxalic or acetic acids, but readily soluble in free mineral acids; usually calcic oxalate is seen in the urine in the form of octahedral crystals, but sometimes in the form of quadrilateral prisms terminated by pyramids, and sometimes in the form of dumb-bell, oval, and circular crystals. In a case of oxalic-acid poisoning, which the writer has seen, the crystals in the urine were chiefly of the prismatic form. Oxalic acid in urine is always combined with calcium in the form of calcic oxalate, but all of the calcic oxalate is not always found undissolved as a sediment; usually a portion of it is dissolved in the urine, it being held in solution by the acid phosphate of sodium, which is a normal constituent of the urine.

All of the calcic oxalate in urine can be isolated and estimated in the following manner: Add to the urine an excess of calcic chloride and render alkaline with ammonia, filter, wash the precipitate with distilled water, and treat it with acetic acid, which will dissolve the phosphates; the precipitate will then consist of calcic oxalate and mucus or other organic matter, from which the oxalate can be separated by treating with dilute hydrochloric acid, in which the oxalate is soluble, but the organic matter insoluble; filter and pour gently over the surface of the filtrate, so that it will form a separate layer of fluid, an excess of dilute ammonia-water; the calcic oxalate will gradually be precipitated in the crystalline form, and can be collected upon a filter paper, dried, and weighed. Calcic oxalate can be tested in the same way as free oxalic acid, except that it is necessary to first add sulphuric acid in order to decompose it; therefore, if treated with sulphuric acid, carbonic acid, carbonic oxide, and water are evolved as products of the decomposition and can be collected and further tested by appropriate methods. With nitrate of silver a copious white precipitate of oxalate of silver is obtained, soluble in nitric acid or in ammonia.

If the precipitate be dried, and heated on the point of a knife over the flame of a spirit-lamp, it becomes brown on the edge, detonates feebly, and is entirely dissipated in a white vapor. In this manner it is distinguished from other white salts of silver, which give off white fumes, but leave a residue.

§ 67. *Liquids containing organic matter*, as in matters vomited or in the contents of the stomach, should, if very acid, be diluted with water, if necessary, strained and filtered; the filtrate will contain all of the oxalic acid which existed in the free state in the liquid. This oxalic acid should then be precipitated by a solution of acetate of lead; the resulting oxalate of lead collected upon a filter paper, washed, then suspended in water and a current of sulphuretted hydrogen gas passed through the mixture, until there is no longer any oxalate of lead undecomposed; the fluid will then contain a black precipitate of sulphide of lead, and oxalic acid and sulphuretted hydrogen in solution; filter, and evaporate upon a water-bath; when a residue will be left, which is more or less pure oxalic acid, and which can then be examined by the appropriate tests. If, however, antidotes have been administered, such as chalk or magnesia, the supernatant liquid, after standing some time, may, if not acid, be thrown away, and the residue treated with dilute hydrochloric acid, which will dissolve the oxalate; filter, wash, and treat the filtrate with a solution of acetate of lead as above described; the precipitate will then contain chloride of lead mixed with the oxalate, but this will not interfere with the isolation of the oxalic acid by the above method, since the subsequent evaporation will volatilize the hydrochloric acid as well as the sulphuretted hydrogen.

§ 68. The tests for *binoxolate of potassium* in solution are the same as for oxalic acid. The crystals differ from those of oxalic acid in having a feathery appearance. They may be distinguished, also, by the action of heat, not being entirely dissipated like those of oxalic acid, but leaving an ash of carbonate of potassium. Both the quadroxalate and the binoxalate of potassium are kept in the shops under the names of salt of sorrel and essential salt of lemons, and are employed for removing iron rust and ink stains from linen. Free oxalic acid is also frequently sold for the same purpose, so that all of these poisonous compounds are frequently seen in households, and are often kept upon the same shelf as articles used for food or for cooking.

V. *Tartaric Acid.*

§ 69. The only case in which this substance incontestably proved poisonous is related in the *Lancet*, Jan. 2, 1845. A man took, by mistake for Epsom salts, an ounce of tartaric acid dissolved in half a pint of warm water. He immediately exclaimed that he was poisoned, and complained of a violent burning pain in his throat and stomach. Obstinate vomiting continued for nine days, when he died. Nearly the whole of the alimentary canal was found highly inflamed. Tartaric acid was detected in the dregs of the cup, and the druggist admitted that he had made a mistake. Another case is reported by Devergie, but the accuracy of his opinion and analysis is contested by Orfila.[1] Christison mentions an instance in which six drachms of tartaric acid were taken in twenty-four hours, without the least inconvenience, and both he and Coindet administered it to cats without observing any injurious effect. An instance is on record in which the *bitartrate of potasssium* proved fatal by excessive purging, but the quantity swallowed, or rather *eaten*, is said to have been more than a quarter of a pound.[2]

§ 70. The chemical detection of tartaric acid is very difficult, because it forms so large a constituent of ordinary food and cooking salts (cream of tartar); therefore, in any case of poisoning by tartaric acid it is necessary to isolate a large quantity from the stomach. Since tartaric acid is decomposed before being absorbed into the blood, it is, of course, useless to search for it in the blood or solid viscera. To isolate it, the contents of the stomach are diluted with distilled water if necessary, filtered and the filtrate divided into two equal parts; one portion is exactly neutralized with potassium hydrate or carbonate, then added to the other portion, the mixture shaken actively and allowed to stand for some time, when nearly all of the tartaric acid will be precipitated in the form of crystalline cream of tartar (acid tartrate of potassium).

VI. *Acetic Acid.*

§ 71. This acid in a concentrated form is highly irritant and corrosive. Only seven cases of poisoning by it are reported, one by

[1] See Ann. d'Hygiène, 1852, vol. i. pp. 199, 382, and vol. ii. p. 230.

[2] Lond. Med. Gaz., 1837–38, i. 177.

Orfila,[1] one by Taylor,[2] and two by Heine and Hergott.[3] In Orfila's case the liquid was swallowed by a young girl, and in a few hours afterward she died in great agony. The *post-mortem* appearances resembled somewhat those produced by sulphuric acid, the surface of the tongue being brown and leathery, the mucous membrane of the œsophagus of a blackish-brown color, and large black elevations marked the lining membrane of the stomach, the rest of the organs appearing congested. Eight ounces of a thick, blackish fluid were found in the stomach.

In Taylor's case a woman gave by mistake to her child of two years of age a dose of glacial acetic acid, which had been used for removing warts; the child suffered the most intense pain, and died in about thirty-six hours. In the two fatal cases of Heine and Hergott an injection of Villati's solution (sulphate of copper four parts, sulphate of zinc four parts, solution of subacetate of lead seven parts, acetic acid fifty-two parts) was used in a suppurating wound. The remaining three cases were attempted suicides, or where the acid had been taken by mistake for a medicine, only one of which ended fatally.[4] In Heine's case the injection was immediately followed by hemorrhage from the wound and violent local pains in it; these were followed by prostration with pallor of the skin, trembling of the entire body, accompanied with chill, and a rapid and weak pulse, the patient dying in a state of collapse in a few hours. In Hergott's case nausea and vomiting occurred, and death in a very short time.

Officinal acetic acid contains thirty-six per cent. of glacial acetic acid, and of course would be followed by similar symptoms as those accompanying the ingestion of the pure acid, viz., irritant and corrosive local action, with symptoms of collapse, muscular twitchings, and chills.

Dilute acetic acid can be taken in apparently large quantities without danger to life, though many standard books speak of a chronic poisoning from the daily use of vinegar or weak acid by hysterical women for the purpose of blanching the complexion. It is more probable that anæmic persons, who practise this injurious

[1] Toxicologie, vol. ii. p. 198.

[2] On Poisons, Phila. 1875, p. 241.

[3] Ziemssen's Cyclopædia of Medicine, New York, 1878, p. 349.

[4] Husemann, Toxicologie, Birkett, Lancet, 1867, Meliou, Frank's Magaz. in Ziemssen's Cyclopædia, op. cit.

habit, crave an acid to relieve their restlessness and vitiated appetite, and thus prevent the natural desire for good and wholesome food. This would result in increasing an already depraved process of nutrition, and impair still further the digestion. Hence the use of vinegar or weak diluted acetic acid is not the prime factor in inducing the gradual exhaustion, and the consequent deterioration of health.

§ 72. In case of an investigation to detect this acid in the contents of the stomach, it must be remembered, that it is contained in the natural secretions of this organ; hence, to be of any value, the chemical evidence must detect a considerable quantity of it. Also, as Christison suggests, vinegar is a common remedy with the vulgar for many diseases, and especially for poisoning.

§ 73. *Vinegar.*—Dr. David, of Montreal, met with an instance in which a woman endeavored to destroy herself by drinking a quantity of common vinegar. When seen, three hours afterwards, her countenance was wild and the pupils dilated, the body was covered with a cold perspiration, and the breathing was laborious and hurried. Her tongue was dry and cold, the abdomen distended, and she had acute pain in the epigastrium, which was increased by pressure. She was, moreover, delirous. She soon recovered after the administration of an emetic of sulphate of zinc.[1]

VII. *Carbolic Acid.* (Phenylic Acid, Phenylic Alcohol, Phenol.)

§ 74. Carbolic acid has of late years given rise to quite a number of cases of poisoning, chiefly suicidal and accidental. It is obtained from coal tar, and, when perfectly pure, is seen in the form of white colorless needle-shaped crystals, which, after standing for a long time, have a pinkish color when seen in bulk. It absorbs moisture readily and becomes converted into an oily liquid, having a reddish or brownish color. It has a penetrating, unpleasant odor like that of creasote. It is soluble in twenty parts of water, and is very soluble in alcohol, ether, and chloroform. The crystals melt at about 40° C., and boil at about 185° C. Upon the continued application of heat they are completely volatilized without leaving any residue. When applied to the skin carbolic acid produces a caustic and benumbing effect. The extensive use of this acid as a disinfectant and for other purposes renders it a common article of household use, and explains the numerous recent cases of poisoning

[1] Amer. Journ. Med. Sci., Oct. 1848, p. 302.

by it. Falck[1] has collected since 1868, from medical literature, eighty-five cases of poisoning by it, of which seven were suicidal, thirty-nine were due to its use as a surgical dressing or wash, and thirty-nine were cases of accidental poisoning, the carbolic acid having been taken by mistake for some medicine or drink. Most of the cases of acute poisoning by carbolic acid are due to the ingestion of crude carbolic acid, which is usually a brown, oily fluid having the odor and properties of carbolic acid, although it is impure.

§ 75. *Symptoms.*—The poisonous action of strong carbolic acid is that of a caustic and corrosive upon the tissues of the body; upon the skin it produces a white streak, upon the mucous membranes an œdematous, swollen, and inflamed condition and a bleached appearance, the mucous membrane often being readily peeled off from the subjacent tissues. The symptoms which follow the swallowing of a poisonous dose are twofold, depending very much upon the degree of concentration of the acid. Calvert's No. 1 acid is crystalline at a temperature of 60° F., or 15° C., and is a strong local caustic, benumbing sensibility to pain soon after the first moment of contact. The ingestion, therefore, of this form of the acid is followed immediately, or in a very few minutes, by narcotism and unconsciousness, relaxed or flaccid condition of the muscular system; the pupils are insensible to the stimulating action of light, sometimes of natural size, but more often very much contracted. The face is livid, and on the skin near the angles of the mouth and running down to the chin white streaks may be observed, showing the local caustic action of the drug. The breathing is labored and slow, pulse is small and slow, oftentimes regular, but most generally the pulse beats are irregular and intermittent in character; in about an hour after reception of the poison, the intermittence and irregularity of respiratory movements and pulse beats are very peculiar; for a few seconds they may be slow (about forty to the minute for the pulse with about nine respirations), then both may be rapid, the pulse one hundred and thirty, and the respirations forty-five to the minute. These peculiarities in pulse rate and respiratory movements are rather peculiar to the poisonous action of carbolic acid, and are also evidences of its marked narcotic action. The heart

[1] Lehrbuch der prakt. Toxicologie, 1880, p. 208.

sounds are pure, though the pulsations are usually tumultuous from weakness of the cardiac muscular fibres. These peculiarities of respiration and pulse become more marked as death approaches, and resemble the dying moments of a patient with fatal cerebral disease.

The other prominent symptoms which would usually be observed in persons who have taken a less concentrated form of the acid resemble those observed with any other corrosive; these are pain in the mouth, gullet, and stomach, which is soon followed by insensibility, with difficult or impossible deglutition, cold and clammy skin, decreased surface temperature (below 97° F., 37° C.), stertorous respiration, a small, frequent, and intermittent pulse, contracted pupils, insensibility to pain, and the voiding of a brownish, blackish, or greenish urine. The nervous system is profoundly impressed. The local action upon the intestinal canal is that of a corrosive or irritant poison, causing usually a white eschar, the skin and mucous membrane becoming cornified.

A case reported by Mr. Silk,[1] resident in the University College Hospital, London, is that of a person who had taken about a wineglassful of strong carbolic acid an hour before his reception into that hospital. Coma and stertorous breathing were quite well marked. Eyeballs were prominent and the pupils contracted to a pin's point and insensible to the action of light or touch. Lips were shrivelled, and the skin was white about the angles of the mouth, with excoriations. Pulse full and soft, occasionally intermittent, one hundred and twenty per minute. Temperature of the body was under 95° F., 35° C. Surface of the body cold, clammy, and apparently without sensation. Death occurred in about three and a half hours after swallowing the acid. A case of poisoning in a woman of 45 years of age was brought to the Charing Cross Hospital, London, of which Mr. Harrison presented the following notes.[2] He saw the patient half an hour after she had taken the acid. The friends reported that no pain or delirium had been observed; but that, immediately after the ingestion, insensibility appeared. On entrance to the hospital, breathing was stertorous, pupils were insensible to light or touch and were somewhat contracted. The

[1] Br. Med. Journal, April, 1881, p. 640.

[2] Med. Times and Gaz., Aug. 30, 1879.

smell of carbolic acid evolved from her person was very pungent, and the skin about the mouth was reddened; the pulse was small and irregular, and the surface of the body was cold. The stomach-pump removed about half a pint of dark grumous fluid, smelling strongly of the acid. The patient died in about three-quarters of an hour from the time of swallowing the poison.

Dr. Robert Kirk reports in great detail[1] the symptoms of a fatal poisoning of a woman; she took an unknown amount of Calvert's No. 1 acid (less than two ounces). Narcotic symptoms and unconsciousness came on in a very few minutes. She was seen by Dr. Kirk in less than half an hour after taking the poison. "Her whole body was flaccid, her pupils insensible to light, and were neither increased nor diminished in size. Her face was livid, and from the corner of the mouth a white streak passed downwards to the chin. Her breathing was slightly labored; the respirations eight per minute. Pulse 40, small, but regular. Her extremities were cold. At six o'clock, or fifty minutes after the poison had been taken, her respiration became irregular." From this time onward until her death, four hours after swallowing the drug, the respiratory movements and pulse beats showed alternations of increased and diminished rates of no particular regularity of intervals. At one time the pulse beat regularly for seventy seconds, about 40 per minute, with 9 inspirations per minute, and then the pulse movements were 100 per minute, respirations 30 per minute, shallow and gasping. Again, about one and a half hours after the acid was taken: "The pulse was beating about 50 per minute, and after fifty seconds it become rapidly quicker until it reached 130, at which rate it continued for half a minute, and then fell as quickly as it had risen. The respirations during the pause between the accelerations were not more than 8 per minute, deep and quiet. Almost simultaneously with the pulse, the rate of respiration increased in twenty to twenty-five seconds to 45 per minute, and then fell with the pulse." The behavior of both respiratory and heart movements was very peculiar and intermittent. "The heart sounds were quite pure, but the beating was tumultuous, and the contractions appeared to be only partial. The respirations now were very slow, about two to the minute, very deep, not stertorous."

[1] Glasgow Med. Journal, June, 1881, p. 433.

In two hours and a half from the time the poison was taken "the time between the acceleration of the pulse and respiratory rates increased, the pulse became slower, and the respiration ceased for a longer or shorter period" until three and a quarter hours had passed, when the pulse beats were only 20 or 30 per minute and very feeble, and then became so accelerated that the intervals between each pulsation could hardly be distinguished, the respiration partaking of the same general character as is often witnessed at the dying moments of a patient with fatal cerebral disease.

Dr. Reichert[1] reviews the symptoms of poisoning in fifty-four cases collected by him. The most rapidly fatal case in this synopsis occurred in three minutes after a dose of half an ounce of impure acid.[2] Of the other cases one was reported[3] where death occurred in ten or fifteen minutes, and another of a child[4] who had taken by mistake a tablespoonful of carbolic acid, followed by copious draughts of warm water, and who died in seventeen minutes. The longest duration of life after a fatal dose of poisoning by this acid was two days.[5] The smallest recorded fatal dose in this class of cases was one drachm taken by a man sixty years of age; but two children, who took a similar dose, recovered.

§ 76. Death from poisoning by this acid can also occur apparently from simple inhalation, for Dr. F. P. Blake[6] reports the case of a woman suffering from neuralgia, who sought for relief to her pain by inhaling the acid. She was found dead in her bed with a handkerchief enwrapping a sponge and applied close to her face, which was livid. There was a strong smell of carbolic acid in the room, and the sponge and handkerchief, though dry, were strongly impregnated with its odor; the appearance of the mouth, the skin of which was free from excoriation or caustic marks, did not indicate that she had taken any of the acid in her mouth, and the tip of the nose was blistered from contact with the cloth and sponge.

§ 77. Dr. Paul[7] reports a case where constitutional symptoms of poisoning occurred from the external application of carbolic acid

[1] Am. Jour. of Med. Sciences, Oct. 1880.

[2] See the Philadelphia Med. Times, 1873, vol. ii. p. 284.

[3] Hill, Richmond and Louisville Journal, and N. Y. Med. Rec., 1873, p. 383.

[4] Louisville Med. News, 1880, No. 10, p. 294.

[5] Med. Times and Gaz., 1870, p. 474.

[6] Phila. Med. Times, July, 1881.

[7] Idem, May 8th, 1880.

over a surface of about five square inches, of which only a small portion was denuded of cuticle. The symptoms began twenty minutes after the application. In this connection it must be said that susceptibility to carbolic acid intoxication absorbed by the lungs or from the skin is very variable, and in some persons occurs on very slight provocation. Proofs of intoxication are shown by the peculiar appearance of the urine, before alluded to. Danger of criminal poisoning by carbolic acid is not great, since its strong and pungent odor would make it difficult to administer without the knowledge of the victim; hence, when not the result of accident, a death from this agent might reasonably be assumed to be one of suicidal, not homicidal, intent. It must be said, however, that the smell and brown color of the strong solution of the acid might be mistaken for smoky whiskey by persons in whom the sense of smell is not delicate. In fact all the cases of poisoning by this agent, which have been recorded, are either those of carelessness or suicide.

§ 78. Cases of death from surgical dressings with carbolic acid have been reported. One[1] reported by Dr. Bradford is not conclusively a death from carbolic acid intoxication, thongh it is evident that a large amount of this agent was absorbed from the inflamed tissues in a cold abscess of the hip, which was washed out and distended by a two-and-a-half per cent. solution of carbolic acid, and dressed antiseptically with carbolized gauze. In this case the lesions of greatest importance were in the heart and kidneys, which were the seat of fatty degeneration, and they were analogous to the appearances of poisoning by phosphorus, arsenic, or ammonia. Dr. Bradford says, "the clinical history of the case directly suggests, that the lesions found may be attributed to the toxic action of carbolic acid." The urine passed within the first twenty-four hours after the carbolic acid was used "was tar color," and its chemical analysis showed a "color slightly smoky and the presence of a very large amount of a sulpho-acid (presumably sulpho-phenylic)." In an article by Mr. Macphail[2] the subject of poisoning by absorption of carbolic acid dressing is treated at length; this depends upon the fact that the solution of carbolic acid is in contact with tissues or

[1] Boston Med. and Surg. Journal, April 7, 1881, p. 324.

[2] Edinburgh Med. Jour., 1881, 2, xxvii. pp. 132–144.

serous membrane inflamed and abnormally active in absorbing deleterious substances. It is well known in surgery that decomposing fluids, pus, and fetid discharges may be readily absorbed by contact with diseased surfaces, and thus give rise to septicæmia or blood poisoning. It is reasonable, therefore, to explain the absorption of carbolic acid upon the same principle. In fact, Professor Lister, the earliest advocate for antiseptic dressings,[1] says, "I believe the secret of our immunity from these (carbolic acid) toxic effects is, that we avoid as much as possible all unnecessary action of the carbolic acid upon the tissues. Had I in this boy's case (a case of pleuritic abscess) injected carbolic acid lotion into the pleural cavity, and done this at every dressing . . . . I think it is in the highest degree probable that he might have suffered from carbolic acid poisoning. . . . . I cannot point to one single instance in which I can be sure that we have had carbolic acid poisoning of any moment whatever, either in my hospital or private practice during the two years I have been in London."

Nusbaum, of Munich, in a recent work on antiseptics[2] mentions a case of carbolic acid poisoning from the forcible and daily injection of carbolic acid into an abscess cavity. Dr. Whiteford, of Greenock, England,[3] observed symptoms of carbolic acid poisoning from the external application of a two-and-a-half per cent. solution to a ringworm of the scalp in two children, and here it should be observed that children are especially susceptible to the action of carbolic acid. Three other cases of carbolic acid intoxication are reported by Mr. Macphail[4] from the use of these dressings, but which finally recovered after their discontinuance. Mr. A. P. Gould[5] reports a case of rapid death after section of the tibia by the antiseptic method with carbolic acid. The presence of septicæmia was negatived by the absence of any sufficient source of septic absorption and of fever. The symptoms observed were similar to those reported by Dr. Bradford,[6] viz., vomiting, diarrhœa, nervous prostration, and lowered temperature. The patient died within thirty-seven hours after the operation.

[1] Lancet, Dec. 20, 1879.

[2] Leitfaden zur antiseptischen Wundbehandlung, p. 141.

[3] Related by Mr. Macphail, op. cit.

[4] Op. cit.

[5] Br. Med. Jour., May 28, 1881.

[6] Op. cit.

Küster[1] reports a case in which a puerperal uterus was being washed out with a two per cent. solution of carbolic acid on the fourth day after delivery, without any serious results, but on the next day the use of a five per cent. solution was followed by the severest symptoms of poisoning, and death on the ninth day.

This form of poisoning by the use of carbolic acid as a surgical dressing or wash, can always be prevented by careful examination of the urine. When the acid is absorbed, it pairs with the sulphuric acid radical to form phenylsulphuric acid, which has no deleterious action upon the economy, so that, as long as it can find any sulphuric acid to unite with, we see no toxic symptoms in the patient, and this event is characterized by the entire disappearance of sulphates from the urine. Ordinarily, if we add to urine acetic acid and a solution of chloride of barium, an abundant precipitate of sulphate of barium will be formed, but if all of the sulphates have been decomposed by carbolic acid to form the sulphophenylic acid, no such precipitate will occur; if, at this time, alkaline sulphates, such as the sulphate of sodium, which is found in many mineral waters, be given to the patient, it will neutralize the excess of carbolic acid and prevent toxic symptoms. Baumann[2] reports six cases in support of this action of the sulphate of sodium. In this connection it is important to remember that carbolic acid is entirely eliminated from the system in from twelve to sixteen hours.[3] The amount of carbolic acid which is daily eliminated with the urine of a healthy adult upon a mixed diet is, according to Engel,[4] about 0.015 grm. (about $\frac{1}{4}$ grain). The above cases and many others make it evident that carbolic acid in solution can readily be absorbed by inflamed tissues, especially the serous membranes as well as the mucous membranes. When the patient is constantly under observation by a medical attendant, as would naturally be the case after surgical operation, proofs of carbolic acid absorption would most generally be recognized before danger had arisen to the life of the patient. In cases of operations for criminal abortion, the use of carbolic acid might seriously by its absorption compromise the life of the patient, and, if performed by an irresponsible or un-

[1] Centralblatt f. Gynækologie, ii. 14, 1878.

[2] Archiv f. d. ges. Physiolog., xiii. p. 285, 1875.

[3] Binnendijk, Journ. de Pharm. et de Chim., 1880, tome 30, p. 515.

[4] Ann. de Chimie et de Physique, 1880, tome 20, p. 230.

educated person, might go forward to a dangerous, if not fatal issue without being recognized as a cause. Here, again, it must be observed, that the susceptibility to poisoning by carbolic acid in some individuals is much greater than in others, and this fact would explain apparent inconsistencies in the reported experience of various physicians and surgeons.

§ 79. *Post-mortem appearances.*—These are similar to the action of other corrosive and irritant poisons; the corrosive action usually extends from the mouth into and through the alimentary canal. The marks upon the lips, skin, and interior of the mouth have been previously described; the peculiar white streaks and shrivelled skin are very marked. The mucous membrane of the mouth and pharynx is reddened and often œdematous and swollen, sometimes it is whitened by the caustic action, but, as the local effects extend down the gullet and into the stomach, the mucous membrane becomes corrugated, bleached, and loosened from the underlying tissues. In one of the cases above cited,[1] the mucous membrane of the stomach was almost entirely eroded, and "lay in lines along the posterior surface, looking and feeling like whip-cord. The mucous membrane of the œsophagus was gathered into longitudinal folds and presented very much the appearance of cigarette paper." Dr. Cameron[2] exhibited the stomach of a patient, which showed this rough corrugated appearance of the mucous membrane of the gullet and stomach. In the Charing Cross Hospital case,[3] the root of the tongue and adjoining surface of the epiglottis presented a glazed appearance "of the color of recently cut lead," the surface of the gullet had a sticky feeling to the touch, and in its whole length had the same glazed, grayish look as the back of the tongue. This autopsy was made thirty-seven hours after death. The corrugated and shiny appearance of the gullet and stomach mucous membrane is commonly observed in this form of poisoning. In the lower part of the gullet and stomach, it may be detached in large pieces (as large as a silver dollar sometimes), and these fragments have a dirty red or greenish hue. The reddening of the mucous membrane ordinarily extends through the small intestines and into the large

[1] Br. Med. Jour., April, 1881.

[2] Transactions of the Liverpool Med. Inst., July, 1881, p. 208.

[3] See above.

intestine. The liver is also congested and smells of the carbolic acid, and the same may be said of the kidneys. The blood is usually fluid, and has a peculiar rich mulberry color, and also smells of the acid. The lung tissue is usually œdematous and injected. The brain is injected, and in one case,[1] there was some slight excess of fluid in the ventricles with a doubtful faint odor of the acid, but in two cases,[2] Dr. Allen reported that the brain was not much reddened, nor was the fluid between the membranes much increased.

§ 80. *Chemical examination.*—Carbolic acid can be recognized very easily by the following tests: (1) Its peculiar odor resembling that of creasote. (2) The coagulation of albumen. (3) A pine splinter soaked in an aqueous solution of carbolic acid becomes blue when moistened with concentrated hydrochloric acid. (4) An aqueous solution of carbolic acid when treated with a neutral solution of ferric chloride or sulphate, becomes of a violet or beautiful blue color. (5) Lex's test consists in adding to a dilute solution of carbolic acid, one-fourth of its volume of ammonia water, and then a little chloride of lime (calcic hypochlorite) solution, when the mixture, especially after gentle warming, will become a beautiful blue color. This test is delicate for a solution containing one part of acid in twenty thousand of water. (6) A solution of carbolic acid is precipitated by bromine water in the form of tribromphenol, a yellowish white, flocculent precipitate. This reaction is made use of in estimating the amount of carbolic acid, the precipitate being collected upon a filter paper, washed, dried, and weighed. One hundred parts of tribromphenol correspond to twenty-eight and four-tenths parts of carbolic acid. (7) Another and exceedingly delicate test is by means of Millon's reagent, which produces with dilute solutions of carbolic acid a beautiful red color, which is perceptible in solutions containing only one part of acid to eight hundred thousand parts of water.

§ 81. Carbolic acid is isolated from the urine or other fluid by distilling it in a retort, after acidulating it with sulphuric or phosphoric acid. The distillates can be tested for carbolic acid by the above tests, and an aliquot portion of it precipitated with bromine

[1] University College Hospital.

[2] Australian Med. Journ., March 15, 1880.

water for the quantitative estimation. To analyze tissues for carbolic acid, they must first be finely cut up and extracted with very dilute sulphuric acid; after sufficient concentration by evaporation over a water bath, the residual fluid can be introduced into a retort and subjected to distillation as above mentioned. Carbolic acid can be separated from water by shaking the mixture with ether, pouring off the ether after it has separated into a watch-glass or evaporating-dish, and allowing it to evaporate spontaneously, when the pure carbolic acid will be left as a residue.

CHAPTER III.

IRRITANT POISONS[1]—CONTINUED.

I. *Potash, Soda, etc.*

§ 82. THESE two alkalies and their carbonates may be mentioned under one head, since their poisonous effects are similar. Eau de Javelle (hypochlorite of sodium, or chlorinated soda, colored red with peroxide of manganese) is mentioned by Tardieu,[2] as having caused death by suicide and by mistake; this solution is used largely

[1] Nearly all of the substances belonging to this class produce very different effects when they operate for a long time in minute quantities, and when they are taken in large doses at once. It is only in the latter case that the term irritant is strictly applicable to them. In the former mode of action they gradually undermine the health, and may ultimately destroy life by interfering with the nutrition of the body. This effect may result either from their deleterious action upon the digestive organs, or upon the composition of the blood, or finally upon an impairment of that action of the tissues by which they appropriate for their own nutrition the organic elements contained in the blood. Arsenic and most of the salts of mercury are actively irritant, when applied to the tissues in a concentrated form; but in minute quantities, and largely diluted, they may slowly but surely sap the foundations of life, by wearing away old structures and preventing the substitution of sound materials for their repair.

[2] Sur l'Empoisonnement, 1875, p. 274.

in laundries for bleaching purposes, and consequently exposes many temptations to suicide or careless mistake from the readiness with which it may be handled. One hundred and fifty or two hundred grammes (five to seven fluid ounces) will produce fatal results. Caustic potash or soda (liquid) in the dose of half an ounce (two to five teaspoonfuls) is also fatal. Potassa dissolves in half its weight of water, soda in a little less than twice its weight; hence a solution of caustic potassa and soda may contain a large amount of the caustic alkalies. Our notice of them will be brief, as they seldom occasion poisoning, and, when they do, it is almost necessarily accidental, and its cause is readily known. They may prove fatal either by their immediate action or by their remote influence upon the system. When swallowed in large quantity and in a concentrated solution, the taste is exceedingly nauseous and acrid, and a sensation of burning heat and constriction in the throat, œsophagus, and stomach is excited. When a considerable portion of the solution has reached the stomach, there follow great pain and tenderness in the abdomen, vomiting of mucus and altered blood, inability to swallow, copious diarrhœa, and general collapse. If the patient survives a few days, the inside of the mouth is seen to be greatly inflamed, sloughs become detached from the throat, vomiting continues, there is a dysenteric condition of the bowels, and the patient dies from marasmus. When the dose is too small to induce an immediate fatal result from the acute irritant and corrosive action of the caustic, the patient will experience a very painful gastric catarrh, or active inflammation of the mucous membrane, resulting oftentimes in a constriction of the gullet, which may interfere with deglutition, and thus cause death by starvation.

Life may be, however, prolonged even for months and years, and the person finally die from the impairment of his digestive powers.

A case is related by Dr. Barham, in which a caustic solution of impure carbonate of sodium (soap lees) was swallowed by mistake, and the patient died two years and three months afterwards in consequence of stricture of the œsophagus caused by it.[1] Several instances of a similar kind are recorded, and Sir Charles Bell relates one in which death did not take place until twenty years after the accident. A more recent example is one furnished by Dr. Deutsch.[2]

[1] Lancet, 1850, vol. ii.

[2] Times and Gaz., May, 1858, p. 537.

The quantity drunk was a "portion of a glass," and was estimated to contain half an ounce of caustic potassa. The early symptoms were such as those described above, but partial recovery took place. Gradually, however, swallowing became difficult, and death took place in twenty-eight weeks after the accident. The lower part of the œsophagus was found enormously thickened, so that its canal was nearly obliterated near the stomach. The increased thickness was entirely confined to the mucous membrane, the muscular coat, on the other hand, having nearly disappeared.

§ 83. The *post-mortem* appearances which are found in the intestinal canal are more extensive than those which are observed in the case of the caustic acids, and the corrosions have not the same blackened or charred character, but are soft and moist; in acute poisoning these moist eschars and the diffused softening extend over the whole mucous surface. When death has been delayed, ulcerous or purulent inflammations may be observed, the partial healing of which may cause the contractions or constrictions of the gullet above referred to; in this case also the neighboring tissues may be softened, unless the repair has been completely effected.

§ 84. *Chemical examination.*—This depends upon the detection of large amounts of the alkalies in the vomitus, contents of stomach or other fluid examined, and, since salts of the alkalies such as common salt, cream of tartar, etc., are such common articles of food and medicine, and many are also normal constituents of the animal economy, it is, of course, necessary to isolate them in their original form, and separate them from their chlorides or other neutral salts. First, the reaction of the fluids to be examined should be ascertained; if any of the above substances are present, it will be strongly alkaline. The amount of alkali present can be determined by exactly neutralizing a portion of it after extracting with distilled water with a solution of some acid (preferably oxalic or sulphuric) of known strength. If the poison consisted of one of the alkaline hydrates, it can be separated from most of the salts by extracting with alcohol. (Potassium carbonate is also soluble in alcohol, but sodium carbonate is insoluble.) In any case of poisoning by an alkali, the substances to be submitted to chemical examination should at once be placed in a tightly stoppered bottle, so that no air can gain access to them, on account of the liability of the alkaline hydrates to absorb carbonic acid from the air and become con-

verted into carbonates. If potassium hydrate or carbonate were the poison, it may be detected by the following tests: (1) By the alkaline reaction. (2) By exactly neutralizing a portion with tartaric acid, and then adding as much more tartaric acid as was required to neutralize it in the first place. This will precipitate the potassium in the form of a white crystalline compound (cream of tartar). The reaction is facilitated by shaking or by adding a little alcohol. (3) Compounds of potassium impart a violet color to the colorless gas flame. (4) A drop of the concentrated solution acidulated with hydrochloric acid and treated with a drop of platinic chloride will yield a yellow crystalline precipitate, which may be seen under the microscope to consist of dodecahedral crystals. Ammonium compounds yield similar crystals, but can be expelled by previous evaporation and ignition. This platinum compound should always be produced as the *corpus delicti* in cases of poisoning by potassium compounds.

Sodium hydrate and carbonate are chiefly recognized by the strongly alkaline reaction, the intense yellow color imparted to the colorless flame, this flame giving the characteristic spectrum, which is different from the potassium spectrum, and by the absence of any potassium or ammonium compounds, both of which can be precipitated, while sodium only forms soluble compounds with the ordinary reagents, such as platinic chloride and tartaric acid.

§ 85. The detection of chlorinated soda and potash (sodium and potassium hypochlorites, *eau de Javelle*), which are bleaching compounds, depends upon the detection of the alkali and the chlorine compound together, the latter being detected by the evolution of chlorine, when a solution is treated with dilute hydrochloric or sulphuric acid.

II. *Nitrate of Potassium.* (Saltpetre, Nitre, Sal prunella.)

§ 86. "A German, who spoke English imperfectly, went into a store, and asked for 'bitter salt,' meaning sulphate of magnesium (*bitter Salz*). The attendant supposed he meant saltpetre, and gave him half a pound. The man took three ounces and a half at one dose. His bowels were opened three times within three or four hours. He complained of a slight sensation of heat in the epigastrium, and drank a good deal of water. About five hours after having taken the saltpetre, he suddenly fell out of his chair and died.

"The marked peculiarity, in this case, was the absence of the painful symptoms which usually follow the ingestion of irritant poisons; and the question arises, how was death produced? Certainly not by inflammation of the stomach, for he complained of nothing but a slight sense of heat in the stomach. The poison must have acted by destroying the vitality of the blood. There was no *post-mortem* examination. The rigor mortis was very imperfect, the lips of almost a natural pink hue, and the appearance of the countenance so life-like, that some persons who were present doubted the propriety of interment on the third day."[1]

This is the largest dose of nitre which we find recorded as having been taken, and its symptoms were, on that account probably, peculiar. There are numerous instances of death occasioned by an ounce or an ounce and a half of this salt, and in which the symptoms were those of a violent local irritant and a depressor, at the same time, of nervous power. Death may occur within two hours. On the other hand, recovery has taken place after doses of nitre varying from half an ounce to two ounces. The symptoms are vomiting, with extreme burning pain in the throat and abdomen, followed by coldness and collapse, and subsequently by bloody stools. In one case bloody vomiting is noted.[2] Nervous symptoms, such as tremor, spasm, and hallucinations, have been observed. Recovery is slow, and the digestive function remains feeble or deranged.[3]

§ 87. *Post-mortem appearances.*—The mucous membrane of the stomach will, in recent cases, be found more or less acted upon by the salt. Thus it may be seen in some parts reddened and in others covered with brown sloughs of partially detached membrane. Perforation has even been observed.

§ 88. *Chemical examination.*—Nitrate of potassium crystallizes in the form of colorless prismatic crystals belonging to the rhombic

[1] New Jersey Medical Reporter, Jan. 1855.

[2] Journ. f. Pharmakodynamik, ii. 178.

[3] *Sweet spirit of nitre* or *spirit of nitrous ether* is an alcoholic solution of ethyl nitrite, containing five per cent. of the crude ether. There are a few cases of poisoning by large doses, two or three ounces, of this medicine, but in ordinary doses and well diluted it can hardly be called a poison. When it is taken undiluted, it may set up an irritation of the stomach and bowels, but the same may be said of not a few of the regular medicines not ordinarily considered as poisonous. The few cases reported were among children or feeble persons (London Lancet, 1878, 2d pt., 766, and 1879, 1st part, p. 9).

system, and are free from water of crystallization. The crystals decrepitate upon being heated; they are readily soluble in water, but only slightly soluble in alcohol. Its solutions answer to the tests for both potassium and nitric acid (see above). In all cases of suspected poisoning by this substance, an analysis of the urine during life is very important, since it is rapidly eliminated by the kidneys. In examining an organic mixture for nitrate of potassium, it should be evaporated to dryness, extracted with as small a quantity as possible of boiling water, filtered, and the filtrate tested by the above tests; if necessary, the filtrate can be purified by boiling with pure animal charcoal and filtering again.

III. *Chlorate of Potassium.*

§ 89. Some cases of death from using this medicinal agent are recorded.[1] Dr. Jacobi in this communication, read before the Medical Society of New York, relates eight cases of poisoning by this agent used medicinally in disease, and states that the dose for an adult should not exceed two drachms, for a child two or three years of age half a drachm, and infants under one year one scruple in each twenty-four hours, and that this dose should be well diluted, to prevent gastro-intestinal irritation.

But these cases were the result of the treatment of disease under serious circumstances. There is no record of a criminal or suicidal death from this drug, and therefore its consideration in this treatise is not of much moment. When iodide of potassium and chlorate of potassium are given simultaneously on a fasting stomach, the double decomposition of these salts results in the formation of the iodate of potassium, which is a physiological poison, and has caused death.

Chlorate of potassium, according to Jacobi and other American authorities, produces an inflammation of the tubes of the kidneys, the so-called acute diffuse nephritis, which may result in death within three or four days, although in mild cases the irritation may extend only so far as to produce congestion of the kidney, and this author mentions a few cases in which the disease abated and the patient recovered. Hæmoglobinuria, one form of bloody urine, has also been seen in cases of poisoning by chlorate of potassium due to the

[1] Medical Record, N. Y., March 15th, 1879.

decomposition of the blood by this drug, as in Wegscheider's case.[1] He alludes to the case of Dr. Fountain, of Iowa, who experimented upon his own person with large doses of this drug, and died from the disease of the kidneys above mentioned. In the *American Journal of Pharmacy* for March, 1878, a druggist, Mr. Kennedy, reports a case of acute poisoning followed by death in a healthy child, who ate a large amount (about an ounce) of dry chlorate of potassium while playing the character of doctor to her brothers and sisters. Dr. J. K. Boudé, of Carthage, Ill.,[2] reports another case of acute fatal poisoning in a healthy child of two years and eight months of age, who ate three drachms of crystalline chlorate of potassium on a full stomach; she complained of pain at the epigastrium half an hour afterwards, but it was supposed to be caused by a slight indigestion, and consequently received but little attention, viz., a few drops of paregoric Two hours after, she vomited and had diarrhœa of greenish mucus. These symptoms were followed by prostration, an ashen pallor of countenance and a feeble pulse, but after the administration of spirit, her pulse improved in character, and she played quite brightly for a space of two hours, and then fell asleep. Eight hours after having swallowed the crystals, she again vomited and had a stool of greenish mucus; her extremities became cold and pulse imperceptible; the urinary secretion was increased rather than diminished; and there was certainly no suppression of urine. Prostration, hebetude, and exhaustion increased, and she died fifteen hours after taking the drug.

§ 90. The *post-mortem* appearances may indicate an irritant action upon the gullet and stomach and a congestion of the kidneys, and the urine should be albuminous and contain casts of the renal tubes. In the case of poisoning by iodate of potassium (decomposition of iodide of potassium by chlorate of potassium) the stomach shows a very marked condition of irritation and congestion.

§ 91. *Chemical examination.*—The chlorate of potassium crystallizes in colorless rhombic tables which have no water of crystallization. It is readily soluble in water (16.5 parts at 15° C.), and only slightly soluble in alcohol. Upon being heated it gives off oxygen gas, a residue of chloride of potassium remaining. Warmed

[1] Deutsche Med. Wochenschrift, 1880, No. 40, pages 533–535.

[2] N. Y. Med. Rec., Nov. 1874.

with hydrochloric acid, chlorine gas is evolved. Heated upon charcoal deflagration takes place. When rubbed up with many organic substances, such as sulphur or sugar, in a mortar, explosion takes place, which often results in injury to the manipulator. It can be separated from organic mixtures in the same manner as the nitrate of potassium.

IV. *Sulphate of Potassium.*

§ 92. This drug has given rise to quite a number of cases of poisoning on account of its use by the laity, especially in France and England, in many puerperal conditions, such as for diminishing the secretion of milk and for procuring abortion.[1] Some of the accidents caused by its use have been due to impurities which the commercial salt contained, such as the sulphates of zinc and copper, corrosive sublimate, and potassium oxalate and arseniate. Sonnenschein[2] records a case in which a woman took about ten drachms by order of her physician, and died in two hours.

§ 93. The *symptoms* produced by it are those of an irritant, severe pain, and burning sensation in the stomach and abdomen, and all of the symptoms of inflammation of the stomach and intestines, nausea, vomiting, purging, cramps, etc. Taylor[3] records a case of a woman who took a large quantity, presumably two ounces, for the purpose of procuring abortion, death taking place after symptoms of severe irritation of the stomach and bowels, and after death the *post-mortem* examination showed evidences of such irritation.

§ 94. *Chemical examination.*—This salt crystallizes in colorless, hard rhombic prisms which have a bitter saline taste, and are soluble in twelve parts of cold water, but are insoluble in alcohol. It can be isolated by extracting the suspected material with water and filtering; the filtrate should be evaporated to dryness, and the residue extracted with alcohol, which will remove many impurities, but will not remove any of the sulphate of potassium; this residue should be dissolved again in distilled water, and the solution tested for both sulphuric acid and potassium (see above).

[1] Mowbray, London Med. Gaz., 1843–44, vol. xxxiii. p. 54.

[2] Handbuch der gericht. Chemie, 1869, p. 62, and Ann. d'Hygiène, Avril, 1842.

[3] On Poisons, 1875, p. 265.

V. *Ammonia and Carbonate of Ammonium.*

§ 95. This volatile alkali has occasionally been used with homicidal purpose, but, in general, cases of poisoning by it are the result of accident. The vapor of strong ammonia has destroyed life, when respired too long a time by a person in a state of temporary insensibility. The effects produced by swallowing a strong solution of ammonia are somewhat similar to, though more intense than, those of the other alkalies. Plenck relates that a man who had been bitten by a mad dog had administered to him a glassful of ammonia. His lips, tongue, and gums turned black immediately, and he died within four minutes.[1] In another case, strong ammonia was taken with suicidal intent. The symptoms were collapse, serous and bloody diarrhœa, and bloody vomiting, excruciating pain in the abdomen, and death in six hours. The mind remained clear till the last.[2] Two cases are reported in the same paragraph of children who were killed by accidentally swallowing a mixture of hartshorn and sweet oil.[3] A little boy, two years of age, took about half an ounce of very pungent spirits of hartshorn from a bottle. He immediately screamed and was very sick, bringing up at first stringy mucus of a light color, and then some more which was dark. The lips were swollen, the breathing was harsh, hurried, and somewhat obstructed, and afterwards became somewhat croupy. There was no insensibility nor any diarrhœa. He recovered.[4] In another case, reported in the same journal, an ounce was taken in milk, by a man who supposed it to be castor oil, having poured it out in the dark. He took immediately copious draughts of warm water, and vomited a quantity of matter like soapsuds. The inside of the mouth, upper lip, tongue, and fauces were white, and other parts excoriated; there was great difficulty in swallowing. He said he felt as if he was on fire from his stomach to his mouth; his voice was husky, pulse small and frequent, and the surface cold. He was ordered dilute acetic acid and demulcent remedies, under which he soon recovered. There was no diarrhœa throughout the case.[5] A woman took a wineglassful of strong liquor of ammonia by mistake for the acetate, yet in a fortnight she was convalescent.[6] Dr.

[1] Mitscherlich, Lehrbuch, ii. 277.
[2] Annales de Therapeut., iii. 443.
[3] Times and Gaz., June, 1855, p. 353.
[4] Iliff, Lancet, 1850, vol. i. 337, Am. ed.
[5] Ibid., 1852, i. p. 374
[6] Wilkins, Lancet, 1846, i. 385.

W. Reed has reported the case of a man who swallowed by mistake three drachms of the strong solution of ammonia, and an equal amount of the sesquicarbonate dissolved in two ounces of oil. The symptoms resembled those above described, but they subsided and were removed under appropriate treatment, in about eight days.[1]

Chevallier relates an instance of an attempt to poison with ammonia. The mistress of an officer, he being desirous of breaking up the connection, at their last proposed interview attempted to make him swallow a quantity of ammonia. A physician was sent for immediately. He found the lips excoriated, with phlyctenæ, and the tongue swollen and deprived of its epithelium, and the mouth and palate abraded. The throat was so sore as to prevent swallowing, and pressure on this and the region of the œsophagus was very painful.[2]

As may be gathered from these cases, acute poisoning by ammonia is marked by the local caustic effects of a corrosive, and these local effects may be so violent as to cause death by shock. The following additional case reported by McGillam[3] is offered as a further illustration of acute poisoning by ammonia water. A man swallowed half a pint of liquor ammoniæ, and was brought into the Great Northern Hospital four and a half hours later. Mustard and oil were first given to the patient and afterwards followed by acidulated mucilage. There was great difficulty in swallowing. Ten hours from the time of taking the ammonia, the patient was hoarse and in a drowsy condition, with a dusky countenance; the skin was cold, the body temperature being 100.8°, pulse rate 130; the patient had freely vomited mucus and blood. Twenty hours after the ammonia had been swallowed, the lips were much swollen, tongue very dry, and mucous membrane of the mouth in parts eroded; the fauces were red and congested and so swollen that there was hardly any open space between its two sides, the mucous membrane of the throat being œdematous, softened, and inflamed. There was great pain in the abdomen and tenderness on pressure, with marked tympanites. The patient had several stools during the previous night but passed no blood, though he twice had vomited blood; he complained of burning pain in throat and at pit of the

[1] Times and Gaz., July, 1855, p. 59.

[2] Am. Journ. Med. Sci., April, 1854, from Gaz. des Hôpitaux.

[3] Med. Times and Gaz., London, Dec. 1878.

stomach. After thirty hours had passed, the patient became comatose and had great difficulty in breathing, the respiratory movements being sixty-six, and the pulse one hundred and eighty to the minute; diarrhœa had been present all day. He died in forty-eight hours after taking the dose.

Another case of fatal poisoning occurred in France, and was communicated in detail by M. Albert Robin to the Société de Biologie.[1] The patient, a woman, drank forty-five grammes, about an ounce and a half, of a solution of ammonia (strength not stated). Prof. Gubler remarked that in this case, besides the usual symptoms which are above described, she had an abundant flow of saliva, and albumen in the urine; she also suffered from quite severe muscular pains, especially in the thigh. Her death occurred after forty-five hours of suffering.

Dr. Francais[2] reports a case of suicide by ammonia. The details of his case are similar to those above mentioned. He concludes that ammonia acts not only as a local irritant and rubefacient, but also causes secondary corrosive action by imbibition and absorption into tissues in the vicinity of the alimentary canal, and deep-seated gangrene which may invade the spleen, liver, and kidneys. There was no corrosive action upon the lips or mouth. His patient lived seven days.

It seems from these cases, especially when explained by the *post-mortem* appearances, that even if the patient may survive the effects of the local irritant and caustic action upon the inlets of the body, the secondary inflammations may, as in the case of the acid corrosives, obstruct the air-passages or gullet, and that, moreover, like arsenic, phosphorus, carbolic acid, and other poisons, fatty degeneration may occur in the tissues of the vital organs, thus inducing a fatal result from impairment of their functions. This fatty degeneration may occur as early as the second twenty-four hours, and the rapidity of death will depend upon the extent of this degeneration.

§ 96. *Post-mortem appearances.*—In acute poisoning, that is, in those cases where death occurs within twenty-four hours, these are restricted to the inlets of the body. In a case related by Nysten, where a man died from the inhalation of the vapor while insensible, the nostrils were blocked up with an abnormal membrane. The

[1] Feb. 14th, 1874.

[2] Bull. de Société de médecine légale de France, vol. v., 1873, p. 32.

whole mucous coat of the air-passages was mottled with patches of lymph. There was a black eschar on the tongue, and another on the lower lip. In general, the liquid form of this poison produces marks of violent inflammation, sometimes of the pseudo-membranous form, in the fauces and œsophagus, redness, softening, or ulceration of the gastric mucous membrane, and to some extent of the small intestine. The blood is generally liquid.

§ 97. In the subacute or prolonged poisoning, that is, in those cases where death occurs at any time after the first twenty-four hours, the secondary effects are extensive gangrenous inflammations and ulcerations of the alimentary canal, which may penetrate below the mucous surfaces, and extend into tissues outside of the digestive tract. In McGillam's case the mouth was much swollen, its mucous membrane being generally softened and in portions destroyed; there were patches of hemorrhage into the submucous tissue. The stomach and intestines were filled with gas, and their tissues deeply injected with blood. The mucous membrane of the stomach was charred and in parts destroyed, and the contents had about ten ounces of dark altered blood. The gullet presented similar appearances of corrosive action, its mucous membrane being thickened, partly yellow and partly destroyed, and in places there seemed to be occlusions of the tube; the epiglottis was œdematous and swollen. The air-passages were nearly normal in appearance, though the larynx was reddened and congested. This patient died in about thirty hours. M. Robin's and Dr. Francais' cases may be taken as types of subacute or slow poisoning. In these cases the autopsy showed complete disappearance of the mucous membrane of the whole gullet, and extensive gangrene in the stomach and intestinal canal; in Robin's case the upper portion of the spleen was one great sloughing abscess, and, in its lower portion, nothing but a soupy mass of débris; the cæcum and rectum were also ulcerated; there was also abdominal peritonitis. In both of these last two cases there was fatty degeneration of the liver, and in Francais' case there was a double purulent pleuritic effusion or abscess, and in Robin's case there was fatty degeneration of the kidneys, and of the sheath of the sartorius muscle in the thigh.[1]

§ 98. *Chemical examination.*—In cases of poisoning by ammo-

[1] See, also, Journal de Thérapeutique, Paris, Mar. 10, 1874, p. 194.

nia or its compounds, the chemical analysis must be performed very soon after death on account of the liability to error from the formation of ammonium compounds by the putrefaction of the tissues. It can be recognized, when present in the form of ammonium hydrate (ammonia water) or ammonium carbonate, (1) by its odor, (2) by white fumes which are evolved when a glass rod moistened with hydrochloric acid is brought near the suspected fluid, and (3) by the alkaline reaction of the vapors emanating from the fluid, as shown by bringing a piece of moistened red litmus or turmeric paper near the surface of the fluid. (4) A drop of platinic chloride upon a watch-glass moistened with a little hydrochloric acid and exposed to the vapor will soon contain yellow crystals of ammonio-platinic chloride, which when examined under the microscope are seen to be dodecahedral crystals, like those of the corresponding potassium compound. This test can also be applied to a liquid as detailed under potassium (see § 84). (5) Tartaric acid gives the same result as with potassium compounds, except that, as in the case of the above test, it can be applied to the vapor as well as the liquid. If antidotes have been given, or if the ammonia has been neutralized in any other way, we can free it from its combinations by warming the compound with a little milk of lime, and test the vapors as above mentioned.

§ 99. Ammonia is separated from organic mixtures by distilling the mixture in a retort after rendering it alkaline with sodic hydrate, and preferably adding alcohol to the mixture in the retort; this facilitates the volatilization of the ammonia vapor, and also prevents the destruction of the organic matter (and consequent formation of ammonia) by the sodium hydrate. The distillate can then (after testing for ammonia by the reaction, odor, and fumes with hydrochloric acid) be neutralized, and the amount of ammonia in it be estimated, if desirable.

VI. *Barium.*

§ 100. The only preparations of barium which have proved fatal are the chloride, the carbonate, the nitrate, and the acetate.

Chloride of barium. — A student of medicine took three teaspoonfuls of this salt by mistake for sulphate of magnesium. He was seized with tormina and vomiting, his extremities became cold, pulse irregular and feeble, and his hands and feet paralyzed. He

recovered gradually in three days.[1] Two cases are referred to by Dr. Taylor, in which it proved fatal.[2] A healthy young woman took less than a teaspoonful of chloride of barium, mistaking it for Epsom salts. In half an hour she had burning pains in the stomach and bowels, with vomiting and purging, followed by the symptoms of collapse above described, and a scarcely perceptible pulse; there ensued, also, great impairment of muscular power, labored respiration with bronchial effusion, coma, convulsions, and death, nineteen hours after the poison had been taken. Sensibility did not seem to be impaired.[3] This salt seems to have a decided action in some cases upon the brain and heart, producing vertigo, headache, deafness, and convulsions.

§ 101. *Carbonate of barium.*—This salt is also said to have destroyed life in two cases.[4] In a case observed by Dr. Wilson, of London, the quantity taken was half a teacupful, but emetics were given and operated before any symptoms showed themselves. In two hours the patient complained of dimness of sight, double vision, headache, tinnitus, and cramps, with occasional vomiting and purging the next day. Recovery, however, took place.

§ 102. *Nitrate of barium.*—Tidy[5] reports a case of poisoning by this salt of barium, death taking place in six and a half hours.

§ 103. *Post-mortem appearances.*—In one of the cases of poisoning by chloride of barium before referred to, the stomach presented a uniform red appearance, with clots of blood and bloody mucus scattered over it; near the cardiac end was a perforation about half an inch in diameter within, and half as wide outside, the edges swollen, and the mucous coat thickened. The small intestines also exhibited signs of inflammation. Without doubt, as is remarked by Dr. Christison, the perforation was, in this instance, an accidental occurrence, not due to the chloride of barium. In a case of poisoning by the carbonate of barium reported by Dr. M. Seidel,[6] of Jena, in a healthy pregnant woman twenty-eight years of age, death took place thirty-four hours after the ingestion of the poison; the stomach mucous membrane contained numerous ecchymoses

[1] Am. Journ. Med. Sci., Jan. 1852. From Casper's Wochenschrift.

[2] Taylor on Poisons.

[3] J.Walsh, Lancet, Feb. 1859, p. 211.

[4] Parke's Chemical Essays, ii. 219.

[5] Medical Press and Circular, 1868, Nov. 25th.

[6] Eulenberg's Vierteljahrsschrift f. ger. Medicin, 1877, N. F., xxvii. page 213.

both in large patches and in spots, and between the ecchymoses it was pale and covered with mucus; ecchymoses were also seen beneath the outer serous covering of the stomach, and under the mucous membrane of the duodenum for a space of eight inches (21 centimeters) from the stomach. The liver was somewhat enlarged, free from blood, brittle, and of yellowish-red color. The kidneys were congested. The mixture taken was a mixture of sugar and carbonate of barium, which had been put up for a rat poison. The stomach was found to contain 0.124 grm. (about two grains) of carbonate of barium, and traces of barium were found in the liver.

§ 104. *Chemical examination.*—The soluble salts of barium, when introduced into a colorless alcohol or gas flame, impart a green color to it, and this flame when examined with a spectroscope gives the peculiar barium spectrum. They are also all precipitated from neutral or alkaline solutions by ammonium carbonate in the form of a white granular precipitate, carbonate of barium; this is readily soluble in acetic or hydrochloric acid with effervescence. They are also all precipitated by sulphuric acid, or a soluble sulphate, in the form of white sulphate of barium, which is insoluble in nitric or hydrochloric acids; the sulphate of barium thus formed can be decomposed by fusing it with sodium carbonate and charcoal, so as to form a sulphide of barium which is soluble. It is also precipitated from a solution acidulated with acetic acid by a solution of potassium chromate, in the form of a yellow barium chromate, care being taken that no free mineral acid be present.

§ 105. *Fallacies.*—In separating barium from organic mixtures we must bear in mind that the sulphates normally present in the animal fluids and tissues will partially, if not entirely, convert the soluble barium salt which may have been taken during life into the insoluble sulphate, and also that in the destruction of organic matter for the detection of the mineral poisons by hydrochloric acid and potassium chlorate, the method commonly used, the organic sulphur compounds, such as albumen, are oxidized and sulphuric acid is one of the products of the oxidation, so that the soluble barium compounds taken during life may be entirely converted into the insoluble sulphate, and cannot be detected in the filtrate, after treatment with hydrochloric acid and potassium chlorate, but must be sought for in the undissolved mass remaining upon the filter by drying it, igniting in a platinum evaporating dish, moistening with nitric acid, and

igniting again; the residue should then be mixed with four times its amount of mixed carbonates of sodium and potassium, and fused until the whole mass becomes liquid; after cooling, the fused mass should be extracted with water, filtered, and washed; the residue remaining will consist of carbonate of barium, which can be dissolved in hydrochloric acid and tested by the above tests. If, by chance, any barium is not converted into sulphate, but remains dissolved in the form of chloride after destroying the organic matter with hydrochloric acid and potassium chlorate, it can be detected and separated by the methods commonly used in qualitative analysis.

VII. *Alum.*

§ 106. *Dried or calcined alum.*—There are reported a few cases of poisoning by alum [about four only, one in England, three in France]. The action is that of a local irritant and corrosive, frothing at the mouth, vomiting, and depression (Taylor). As alum generally provokes immediate vomiting, when given in large amounts, it is not unfrequently combined with some other irritant emetic and prescribed by physicians who desire to promptly evacuate the contents of the stomach. Though it is not ordinarily considered by medical practitioners to have a poisonous character, yet it is considered as a poison by Mr. Taylor in his treatise, and even Orfila admitted that in animals a large dose operated fatally, destroying life in a few hours. We might make the general statement that, if a considerable dose of alum, say more than an ounce, is swallowed and retained in the stomach, it will act as a strong irritant and produce serious constitutional disturbance corresponding to the extent of this irritation. It is, on the authority of Orfila, undoubtedly absorbed into the blood of animals and may be recovered from the urine.[1] Tardieu, however, does not mention this drug in his treatise on Poisons. Higuet relates a case of poisoning by alum,[2] and at the autopsy found the lesions of an irritant poison.[3]

In all these cases the alum had been mistaken for salts, sulphate of magnesium.

[1] Ann. d'Hygiène, 1845, 2, p. 433.

[2] Bull. gén. de Thér., Aug. 1873, p. 137.

[3] See, also, an account of the same case in L'Union Médicale, No. 64, 1873, and Ann. de la Société méd. et chir. de Liège.

Some interest has been attached to this substance in England from its use as an adulterant in the making of bread by public bakers.[1]

§ 107. *Chemical examination.*—This depends upon the detection of both aluminium and sulphuric acid. Small amounts can be found in many articles of food, so that in cases of poisoning large amounts should be detected. Potash alum (sulphate of potassium and aluminium) crystallizes from its solutions in colorless, regular octahedra, with twenty-four molecules of water of crystallization. It is quite soluble in water, and gives the reactions of potassium, aluminium, and sulphuric acid; ammonia alum (sulphate of ammonium and aluminium) has the same properties, except that it gives the reactions of ammonium instead of potassium. It can be isolated from organic tissues and substances by dialysis, or by evaporation and ignition, the residue being tested for the several constituents of alum.

[1] See a small treatise, "The Injurious Effects of Alum upon the human system when used in bread or baking powders and as proven by experiments on man and animals," London, 1879, 8vo. 36 pp.

CHAPTER IV.

IRRITANT POISONS—Continued.

I. *Phosphorus.*

§ 108. *The different forms and modes of administration of phosphorus.*—Phosphorus as a poison was almost unknown previous to the year 1850, when its poisonous properties began to be understood by the laity, and it gave rise to many cases of poisoning, since it could be so easily procured. The cases of phosphorus poisoning in this country are chiefly accidental, due to the ingestion of the phosphorus ends of matches by children; some cases are caused by the taking of vermin pastes which contain phosphorus and which are usually mixtures of meal or flour with some coloring matter, like Prussian blue or Venetian red, and some flavoring matter suited to the taste of the animal for which they are intended. Phosphorus exists in two forms, the transparent or common form which is poisonous, and the amorphous or red phosphorus, which can be commonly ingested, when pure, with impunity. The form which is usually employed in the manufacture of matches and vermin or animal pastes is the transparent or poisonous variety. In the manufacture of matches but little phosphorus is required, a pound of phosphorus sufficing for nearly one-half million of matches. Matches are usually first dipped in an emulsion of sul-

phur and glue or some other glutinous material, and then the ends tipped with a mixture of phosphorus, coloring matter as vermilion, oxidizing material like potassium chlorate, and glue or mucilage. In the so-called safety-matches all of the phosphorus is placed upon the box or scratching surface. Matches are the most frequent source of phosphorus poisoning, and have been the cause of criminal, accidental, and suicidal poisoning; most of the cases are accidental, caused by the sucking of the phosphorus end of matches by children. In criminal cases matches are soaked in some article of drink like coffee or tea; but the mistake is often made of not soaking the ends sufficiently long, or of not stirring the fluid, in both of which cases all or nearly all of the phosphorus remains at the bottom of the cup or other vessel; or of soaking the ends too long, in which case the phosphorus is oxidized and transformed into a comparatively harmless compound, phosphorous or phosphoric acid.

§ 109. *Symptoms.* — Phosphorus is one of the most insidious poisons with which we have to deal, since the symptoms may be quite slow in making their appearance, and in different cases may assume an entirely different character, so that we may have three distinct kinds of symptoms in phosphorus poisoning according as they are chiefly irritant, as in the usual form of poisoning, or chiefly affect the nervous system or the blood, although in some cases we see symptoms belonging to all of these different varieties. In all cases there is usually an immediate eructation of gas which has the taste of phosphorus, and, if the mouth and throat be viewed at this time in the dark, luminosity will be perceived. The severe symptoms do not usually appear for several hours, generally in five or six hours, but sometimes earlier, and sometimes not for eighteen or twenty-four hours. Usually after the lapse of several hours there is pain in the throat and a sensation of heat in the epigastric region; the tongue is swollen, there is a general feeling of malaise, nausea, and vomiting; the vomitus may be bloody, but usually is not, and if it contain free phosphorus, as it generally does, it will be luminous in the dark, especially after being shaken with air; then colicky pains and diarrhœa come on, with tenderness of the abdomen. These symptoms last for one or two days, when they may cease entirely, although the pulse remains feeble, and pain still exists in the limbs and back, and, as the patient is apparently convalescing, death may take place suddenly in from two to four days. In

many cases between the second and fourth day the patient becomes jaundiced; there are headache, insomnia, retention of urine, which is frequently albuminous owing to the affection of the kidneys caused by the phosphorus, and contains the abnormal sediment characteristic of the affection; then vomiting takes place at intervals, the stools are frequent, painful, and sometimes involuntary; finally delirium comes on, followed by coma and death in from about six to twelve days. These symptoms, which are the most common, resemble somewhat those of so-called acute yellow atrophy of the liver, although some authors now doubt the existence of such a disease. In some cases in which the effects of the poison upon the nervous system predominate, the symptoms begin at about the same time; there is the same soreness of the throat and pain in the epigastrium with nausea, which may or may not be attended with vomiting; then there may appear sensations of numbness or tingling in the limbs with cramps and repeated fainting, and there is very great prostration; an erythematous eruption may appear, and, on or about the fifth or sixth day, delirium comes on, with the uttering of loud cries, gnashing of teeth, convulsive twitchings, coma, and death in from six to twelve days. In still rarer cases the phosphorus affects the blood, destroying the blood-globules and setting the pigment free, so that we see the evidences of this action. In these cases the progress of the case is generally slower, although the onset of the symptoms is the same, and the vomitus is more apt to be bloody. There are also bloody stools, with tenesmus; the liver becomes gradually enlarged, and there is very great weakness and prostration. After a few days amelioration takes place, although colic occurs occasionally, and in a few weeks hemorrhages begin to occur from all of the mucous membranes of the stomach, mouth, throat, nose, uterus, ears, etc., etc.; finally the blood becomes fluid, and extravasations take place beneath the skin and mucous membranes; dyspnœa, cardialgia, and general numbness occur from deterioration of the blood; death may take place after the lapse of perhaps several months from exhaustion caused by the numerous hemorrhages.

Cases of professional poisoning have occurred, but are prevented by exercising due care in the selection of employés. It has been noticed that those who have sound teeth are not affected with the disease, which consists of a necrosis of the jaw, so that it is now a

practice in factories of phosphorus matches to employ only those who have sound teeth.

§ 110. The *quantity* required to destroy life is very small. The earliest period at which death has taken place is four hours.[1] A young man died on the twelfth day from the effects of a grain and a half.[2] Martin-Solon relates the case of a patient who died in two days from less than a grain in the form of emulsion.[3] An apothecary named Doffenbach, in experimenting upon the effects of this substance, took one grain, the next day two, and the following day three grains; three days after the last dose he was seized with violent vomiting, and died on the seventh day.[4] A child two years and a half old died after swallowing the phosphorus on eight friction matches.[5] A child two months old is said to have died from the effects of two such matches.[6] If of the average quality, they would contain about one-fiftieth of a grain of phosphorus.

§ 111. According to the investigations of Dybkowsky,[7] it seems probable that the principal action of phosphorus is due to the formation of phosphoretted hydrogen in the alimentary canal; this can readily take place, since nascent hydrogen is produced during the natural processes of digestion. The local inflammatory effects can also be produced when the poison is taken in tolerably large pieces, but many cases are reported, especially those in which the phosphorus is taken in a finely divided state, where there is no evidence of any local inflammatory or ulcerative action.

§ 112. *Post-mortem appearances.*—It must be noted that the *post-mortem* appearances vary with the form of phosphorus used.

1st. When pure phosphorus has been used alone or in combination with fat or oil, the œsophagus and digestive canal are more often the seat of pathological alterations. "Fragments of solid phosphorus, recognizable by the odor or by the phosphorescence, have been found adherent to the mucous membrane of the intestines, and even in the large intestine."[8] In the œsophagus and digestive canal may be found here and there ecchymotic or gangrenous spots.

[1] Journ. de Chimie Médicale, 1845, p. 580.

[2] Worbe, Med.-Chir. Zeit. 1826. Bd. 4, p. 183.

[3] Christison, p. 151.

[4] Froriep. Notiz. Nr. 493.

[5] Schmidt's Jahrbücher, 1844. No. 6, Bd. xlii.

[6] Husemann, Journ. f. Pharm. ii. 169.

[7] Hoppe-Seyler's medecinisch-chemische Untersuchungen, 1866, i. p. 49.

[8] Tardieu, op. cit. p. 437.

Generally the mesenteric ganglia are engorged, softened, and friable.

2d. When this poison has been in the form of paste or the substance used on matches, it will often happen that no appreciable anatomical lesion can ever be detected, but ordinarily, if there be neither redness, trace of inflammation, nor ulceration, very many points of hemorrhage may be determined. On opening the abdomen, the mesentery and the visceral peritoneum appear spotted with black ecchymotic patches or points, analogous to the spots in purpura. The blood is liquid without any notable alteration of the blood-corpuscles. The bladder incloses urine mixed with blood, and presents oftentimes submucous ecchymoses.

The microscopical examination of the tissues is an important means of detecting poison by phosphorus. The liver, the kidneys, and the follicles of the stomach, the heart, and muscles are seen under the microscope in a condition of fatty degeneration. Still these peculiarities *per se* are not indicative of poisoning by phosphorus, as they are found after poisoning by many other agents, and also as the result of many diseases of the lymphatic system. These appearances must be associated with some of the important symptoms of phosphorus poisoning, as the *icteric* appearance of the body, muscular pains and weakness. Tardieu[1] gives a representation by plates of the microscopical appearances mentioned above.

In one case, the mucous membrane of the stomach was of a crimson color, softened in many places, and easily detached; near the pylorus was an ulceration of the size of a quarter-dollar, with brown everted edges, and the muscular coat under it bare. Another similar ulceration was found in the greater curvature. The whole of the small intestine exhibited signs of violent inflammation, but the large intestine was free from it, except in the rectum. In another case, the mucous coat of the stomach and duodenum was so softened that the handle of a knife, passed behind it, readily detached it in a softened condition. Similar conditions have been found in still other cases.[2] These morbid alterations, therefore, resemble those of gastro-enteritis, arising from other causes. The

[1] Op. cit., p. 441.

[2] In two cases, one of which was American, they were present in various degrees from light to dark injection, and thence to grayish and gangrenous ulceration. See Boston Med. and Surg. Journ., Nov. 1855, p. 323; and Nov. 1858, p. 343.

agency of phosphorus in producing such changes must be ascertained not only from the history of the case, but also from its detection by the senses, and by chemical examination. Krahmer states that phosphorus after absorption so affects the blood as to produce ecchymoses under the mucous membranes, skin, etc.[1] It should, however, be borne in mind, that fatal poisoning by phosphorus may take place without the stomach, or indeed any of the organs, displaying the slightest lesion. This is shown in Nitsche's[2] case, in which the dose of the poison was very large, in that quoted by Husemann,[3] in Lewinsky's case,[4] and others.

One of the most common alterations is a remarkable fluidity of the blood. Nitsche, Lewinsky, Krahmer, and Husemann describe it. Of this point, Casper remarks, that we must admit phosphorus to be sometimes fatal by destroying the vitality of the blood. In the cases reported by him, the blood-disks had become transparent by the loss of their coloring matter, which was diffused through the uncoagulated plasma, giving it the appearance of a cherry-red fluid of syrupy consistence. Such changes prove the blood to be deprived of vital and nutritive qualities.

The contents of the stomach or the matters vomited may give a white inflammable vapor, and be luminous in the dark. This was observed in Lewinsky's case, referred to above. They may also exhale a phosphoric odor. Flachsland reported a case in which the dejections obtained by enemata were luminous in the dark, and pieces of phosphorus were found in them.[5] It is said that the intestines, and even the flesh of animals poisoned by phosphorus, have the odor of garlic, and appear luminous in the dark. In a woman who died while taking phosphorus medicinally, it was remarked that the whole of the viscera of the body were luminous; thus indicating the extensive diffusion of the poison.[6] Brera observed also, in opening the body of a woman to whom he had administered phosphorus both by the mouth and rectum, that a white vapor, having an alliaceous odor, and taking fire at the approach of a flame, arose from the

[1] Handbuch, 2te Aufl., p. 462.

[2] Zeitschrift der k. k. Gesellschaft der Aertze zu Wien, and Am. Journ. Med. Sci., Jan. 1858, p. 290.

[3] Journ. f. Pharm., ii. 169.

[4] Brit. and For. Med.-Chir. Rev., Oct. 1859, p. 529.

[5] Med. Chir. Zeit. 1826, iv. p. 183.

[6] Taylor on Poisons, p. 244.

stomach.[1] In the report of a case by Dr. Bingley, in 1857, it is said, "on opening the stomach there was an escape of white smoke, accompanied by a strong smell of garlic."[2] Similar vapors, luminous in the dark, have been seen issuing from the rectum and even from the vagina.[3] Another case is interesting from the fact that, although the body had been buried *fourteen* days, phosphorus was discovered by means of its physical properties, in the stomach.[4] On the *tenth* day after it had been taken Mayer detected it in the contents of the intestine.

The above cases prove that sometimes a portion of the phosphorus may be absorbed as such into the blood and impart its luminosity to an internal organ; the same thing has been demonstrated by experiments upon animals, the breath exhaled being luminous, after the phosphorus had been introduced into the stomach through an external opening after the œsophagus had been tied.

§ 113. *Chemical examination.*—The appearance of phosphorus is familiar to every one. It is insoluble in water, but soluble in ether, alcohol, and the oils. It melts at 110°, and takes fire at a temperature a little above this. It has a peculiar odor, which may readily be perceived by rubbing the moistened finger over the phosphorus end of a match. In contact with air or oxygen it is luminous, if examined in a dark room. Sometimes it may be separated mechanically from the contents of the stomach, or from this organ itself. In a case of homicidal poisoning of a young actress in Berlin, the stomach was empty, there was no unusual smell, and only a few suspicious yellowish spots in the stomach, with no evidences of inflammation. In consequence of suspicions of the cause of death being aroused by the fact of the husband of the deceased having shortly before purchased phosphorus paste (under a special permit), the stomach was submitted to a chemical examination. Cut into pieces, and warmed in a dark place over a spirit-lamp, several shining points were observed in it, and afterwards, by directing a fine stream upon pieces of the stomach, removing fatty matters by boiling, and afterwards quickly cooling, the phosphorus was obtained in a globule of the size of the head of a pin.[5]

[1] Riflissioni Med. Pract. sul'uso interno del fosforo, etc. Pavia, 1778, p. 8.

[2] Lancet, June, 1857, p. 600.

[3] Casper, Gericht. Med., i. 401, 442.

[4] Schäffer, quoted in Henke, Zeitschrift, 1851. E. H. 43, p. 215.

[5] Schacht, Casper's Vierteljahrs., 1852, April.

In another case of attempted poisoning, a woman prepared some soup for her husband. After he had taken a few spoonfuls, he was seized with pain in the stomach. In the evening his wife again pressed him to eat some more of it, but his suspicions were awakened, when, on taking it out of the warm and dark oven in which it had been put away, he observed that it was luminous. The bowl was therefore sent to the magistrate. On uncovering it, white vapors, with a penetrating odor, proceeded from it. When the contents were poured out on an evaporating dish, a transparent, shining globule was observed at the bottom, and afterwards several more, which, when rubbed between the fingers, became luminous, and gave off white vapors. On boiling the soup over a spirit-lamp, bubbles rose to the surface, which inflamed spontaneously.[1]

The detection of phosphorus is, however, seldom so easy. Being mostly taken finely divided in the form of paste, and being not always rapidly fatal, it may have been either removed by vomiting, or exist in too small quantity to be recognized with certainty. Nevertheless, as we have before stated, it has been detected in the body fourteen days after death. In another case, it was found ten days after death.

Dr. V. Elvers[2] reports a case in which free phosphorus was detected in the body of a woman eight weeks and two days after death. In this case one and one-half grains of phosphorus were obtained from the intestines. This case is the longest on record of the detection of free phosphorus in the human body.

§ 114. Various processes have been recommended for the isolation of phosphorus from *organic mixtures*. The best method is that employed by Mitscherlich, which consists in distilling the suspected material previously diluted with water, if necessary, to the proper consistence, and acidulated with tartaric acid in a special apparatus called Mitscherlich's apparatus. In this apparatus the liquid as above prepared is contained in a flask, and on being heated, its vapors rise through a small glass tube, at first vertically, then horizontally, and finally downwards. The descending limb of the tube is enveloped to a certain height with a cold water condenser, penetrating which it passes into a closed vessel below. As the vapors, in coming over, reach the cool portion of the tube, they

[1] Henke's Zeitschrift, E. H., Bd. xxvi., p. 173.

[2] Vierteljahresschrift für gericht. Med., xxv. 1876, page 25.

are condensed, and become luminous in the dark. By this method, five ounces of a substance containing only one-fortieth of a grain of phosphorus, afforded three ounces of distillate in the course of half an hour, during all of which time the luminous zone was visible.

If the mixture subjected to distillation contain alcohol, ether, or oil of turpentine,[1] no luminosity would be observed so long as these substances distilled over. Alcohol and ether being very volatile would soon be separated, and the light would then appear; this would not be the case with oil of turpentine, which substance would rarely be present in a medico-legal examination, unless it had been used as an antidote. Neither tartar emetic, magnesia, iron oxide, musk, castor, opium, albumen, any of the metallic salts, volatile organic acids, nor free acids would interfere with the luminosity. Iodine, calomel, corrosive sublimate in large quantity, metallic sulphides, and particularly oil of wormseed will interfere with the luminosity.[2]

As a preliminary test it is well to make use of Scherer's test, which depends upon the fact that phosphorus, phosphoretted hydrogen, and phosphorous acid blacken nitrate of silver by reducing it to metallic silver. The best method of employing this reaction is that proposed by Hager,[3] which consists in treating a portion of the suspected substances, such as vomitus, contents of intestines, etc., with a little basic acetate of lead solution in order to neutralize any sulphuretted hydrogen which may be present, and placing the mixture in a tightly stoppered flask; a portion of it is placed in another flask, shaken vigorously with ether, and allowed to stand tightly stoppered for a few minutes; a piece of paper moistened with nitrate of silver solution should then be exposed to the action of the vapor in the upper portion of the flask by fastening it into a slit made in the under surface of the stopper; the flask is then placed in a cool dark place. If only a trace of free phosphorus be present, the silver paper will become blackened in from a few minutes to one-half an hour. If this preliminary test, which can be quickly performed, leads to a

[1] During the last few years many cases have been reported in the different medical journals, where the oil of turpentine has been successfully used as an antidote for phosphorus poisoning. One of these cases is reported in detail by *Andant* in the Bulletin Générale de Thérapeutique, tome lxxviii. p. 169.

[2] F. Hoffman, in London Chem. News, Jan. 1861.

[3] Pharmac. Centralhalle, 1879, No. 38.

suspicion of the presence of phosphorus, it can be confirmed by distilling in Mitscherlich's apparatus as mentioned above. The various other processes for the isolation of phosphorus, some of which are of great value in special cases, need not be mentioned in detail here; the most important are those of Dusart,[1] and Lipowitz.[2]

§ 115. *Chronic poisoning* by phosphorus is a disease which may be engendered by this substance in persons who are employed in its manufacture. Its principal organic lesions are caries and necrosis of the bones, with abscesses of the soft parts, added to which the digestion becomes impaired, and, after prolonged vomiting and diarrhœa, life is destroyed by hectic.[3] The caries and necrosis occur chiefly in the bones of the jaw, and are seen more frequently in those who have decayed teeth, so that, as before mentioned, in some match factories it is required that the workmen should have sound teeth.

II. *Bromine.*

§ 116. Pure bromine has given rise to very few cases of poisoning. When applied to the skin or mucous membranes in the liquid form, it acts as an escharotic of the severest type, and the symptoms produced, when it is taken internally, are those of a corrosive. It is a dark-red, very heavy liquid (sp. gr. = 2.97), readily volatile at ordinary temperatures; its vapor has a red or reddish-brown color, and can on account of its weight be easily poured from one vessel to another. The vapor like the liquid is exceedingly irritating to any membrane with which it comes in contact. If a piece of blue litmus paper is exposed to the action of the vapor of bromine, it is at first reddened and finally bleached by it.

§ 117. *Symptoms.*—According to Mr. Wurtz, it is highly destructive to organic matter. He placed a human stomach, with its contents, in a porcelain dish, covered it with water, and poured upon it an ounce of bromine. By the aid of a gentle heat, and occasional stirring with a glass rod, the stomach had entirely disap-

[1] Journal de Chimie Médicale, Sept. 1874.

[2] Poggendorff's Annal. der Physik u. Chem., xc. 1854, page 600.

[3] Accounts of this affection have been given by various authors, among whom may be mentioned Tardieu, Times and Gaz., Oct. 1856, p. 352, and Leudet, Arch. Gén. de Méd., Avr. 1857, p. 308; also Ann. d'Hygiène, 1856, ii. p. 5, and 1857, i. p. 431.

peared in *less than half an hour*.[1] Its corrosive properties have been heretofore observed only in animals.

The only case on record of fatal poisoning by bromine in man, has been reported by Dr. Sayre, of New York. A. H., aged twenty-four, of good health and temperate habits, a daguerreotypist by profession, residing at Williamsburg, near New York, swallowed one ounce, by weight, of bromine, for the purpose of destroying himself. The immediate symptoms, as reported by his medical attendants, were spasmodic action of the muscles of the pharynx and larynx, and great difficulty of respiration. This was soon followed by intense burning heat in the stomach, with great anxiety, restlessness, and trembling of the hands. The pulse was rapid, tense, and corded, and the respiration greatly hurried. The stomach was entirely empty at the time of the taking of the bromine. Various means were used, unsuccessfully, for his relief, the symptoms above described increased in intensity; the hands and feet became cold, with failure of the pulse, etc., until two P. M., when he died, *seven and a half hours* after taking the poison.

§ 118. The *post-mortem* examination was made seventeen hours after death. On opening the abdomen, the external surface of the stomach was found vividly injected, as was also the peritoneal coat of the duodenum, and of the mesentery. A portion of the latter nearest the stomach was stained of a deep yellow color, as were also other parts lying immediately beneath the stomach. A softened ecchymosed spot, an inch and a half in diameter, and several others of a smaller size were also found upon the peritoneal coat of the stomach. The stomach contained about four ounces of thick fluid, resembling port-wine dregs, and exhaling faintly the odor of bromine. Its whole internal surface was covered with a thick black layer, resembling coarse tanned leather. The mucous membrane was very thin, and there was intense submucous injection.[2]

§ 119. Bromine may be separated from *organic mixtures* by agitation with ether, chloroform, or bisulphide of carbon, which dissolve it. If a bromide has been formed, a few drops of a solution of chlorine should be added, to set the bromine free, when it can be detected by its color, odor, reaction, and bleaching properties. It

[1] Silliman's Journal, N. S., vol. vi. p. 405. [2] New York Journ. Med., Nov. 1850.

also forms a white precipitate with nitrate of silver solution, less soluble in ammonia water than the corresponding precipitate formed by chlorine.

III. *Iodine.*

§ 120. *Symptoms.*—This substance is capable of acting in a deleterious manner upon the system, under two circumstances, viz., by the long-continued use of small doses, or by the administration at once of a large quantity. Acute poisoning is caused usually either by taking into the stomach large quantities of tincture of iodine or of Lugol's solution (solution of iodine in iodide of potassium, "Liquor Iodi Compositus," U. S. P.), or by injecting solutions of the above preparations of iodine into internal cavities, such as ovarian or other cysts. But few of the former class of cases are recorded which are either suicidal or accidental. Dr. Bainbridge[1] records one case of accidental poisoning due to the ingestion of one ounce of tincture of iodine, representing one-half drachm of iodine, by mistake for the compound infusion of senna. Dr. Gairdner[2] records a fatal case in a child four years of age, caused by twenty grains of iodine in the form of tincture, and van Hasselt and Husemann report two suicidal cases by tincture of iodine. A large number of fatal cases due to the injection of tincture of iodine into ovarian cysts have been reported. Velpeau has collected records of thirty fatal cases out of one hundred and thirty operations.[3]

§ 121. The *symptoms* occasioned by taking an excessive dose of iodine into the stomach are those of irritation or inflammation of the stomach and intestines, such as are produced by other irritant poisons, pain in throat, gullet, stomach, and abdomen with vomiting, or perhaps only nausea. Vomitus is usually blue from the action of the iodine on the starchy matters, and has the peculiar odor of iodine. There is also usually diarrhœa, the stools being colored with iodine and often with blood. Urine is generally suppressed. In the fatal cases due to the injection of iodine solutions into ovarian cysts, the general symptoms are similar to those caused by the taking of such solutions into the stomach. The symptoms occasioned by its too prolonged use are incessant vomiting and purging, pain

[1] Medical Times and Gazette, Dec. 28, 1861, p. 669.

[2] Essay, 1824, p. 20.

[3] Husemann's Handbuch der Toxicologie, Supplementband, page 131.

in the abdomen, heat and dryness of the throat, headache, rapid emaciation, violent cramps, and a general febrile condition. A patient of Zink, a Swiss physician, after taking too large doses of iodine for about a month, became restless, had burning heat of skin, tremors, palpitation, very frequent pulse, violent priapism, copious diarrhœa, excessive thirst, emaciation, and occasional syncope. He died after an illness of six weeks. Salivation is also an occasional result of the prolonged use of iodine. A case is related in which one drachm of the tincture of iodine, in about an ounce of spirit, is said to have proved fatal.[1] Very often, however, large doses of this substance are productive of no evil effects, nor are unpleasant results generally experienced from its prolonged administration. Dr. Christison quotes a case, in which a child, three years old, took three drachms of the tincture, and suffered only from thirst and slight vomiting. Dr. Kennedy, of Glasgow, gave to a girl 953 grains of iodine, in the form of tincture, during eighty days, without any effects upon the health; and Mr. Delisser gave thirty grains a day to a patient, without any injury resulting.

§ 122. *Post-mortem appearances.*—In the case related by Zink, redness of the intestines, in some places approaching to gangrenous discoloration, was the chief morbid alteration observed. Interesting in connection with poisoning by injection into ovarian cysts is the observation of Edm. Rose,[2] that free iodine could be detected in the stomach and intestines.

§ 123. *Chemical examination.*—Iodine is usually met with in the form of soft micaceous scales of a grayish-black color, metallic lustre, acrid hot taste, and disagreeable odor. It is sparingly soluble in water, but is readily dissolved by alcohol, ether, solution of iodide of potassium, and carbon disulphide. The best test for it in a free state is starch, as a very minute proportion of this substance will give a blue color to a solution of iodine. This blue color is destroyed by heat; hence in testing, the liquids employed should be cold. If iodine is combined with a base, as in iodide of potassium, it must be first set free by nitric acid or chlorine water. It may be detected in the blood and secretions of a patient under its use.

It is slowly volatile at the ordinary temperature, the vapor having

[1] Prov. Journ., June, 1847, p. 356. [2] Virchow's Archiv, xxxv. 1866, p. 12.

a purple color. This vapor, like that of bromine and chlorine, is very heavy, so that it can be poured from one vessel to another, and is very irritating to the eyes and air-passages. It is precipitated by a solution of nitrate of silver, in the form of a yellowish iodide of silver, which differs from the corresponding compound with chlorine and bromine by being almost insoluble in ammonia water.

§ 124. Free iodine can be separated from organic mixtures by shaking the mixture with ether, chloroform, or bisulphide of carbon (preferably the last), when the solvent will separate, being colored deeply violet or purple by the iodine; this solution can be decolorized by shaking with a little potassium hydrate solution, and the whole evaporated, when the iodine will be left in the residue in the form of iodide of potassium. If the iodine is combined with a base, as in iodide of potassium, it can be separated from it by the addition of chlorine water or nitric acid, and then isolated by bisulphide of carbon as mentioned above.

§ 125. *Iodide of potassium*, although milder in its effects, is otherwise similar to iodine in its operation upon the system, and is usually preferred as a medicinal agent. It is well-known among physicians that some individuals have a peculiar susceptibility to the action of iodide of potassium; an extreme coryza, swollen nose, salivation, and gastric irritability sometimes follow the medicinal use of very small doses—one or two grains three or four times a day—and if the continuance of this medicine is persisted in, the disturbance is much increased, while with other patients, even doses of sixty grains have been taken for days together without producing any deleterious consequences. The only cases of death caused by iodide of potassium have been those in which a combination of iodide and chlorate of potassium has been administered, and as already noted, this combination produces an irritant and poisonous iodate of potassium.

IV. *Chlorine.*

§ 126. Orfila has shown, by experiments upon animals, that a saturated solution of chlorine in water produces effects similar to those of the mineral acids. No instances of its poisonous effects upon the human subject are recorded. The inhalation of chlorine vapor induces coryza, difficult breathing, and distress, which soon pass off when the irritating vapor is not inhaled.

CHAPTER V.

IRRITANT POISONS—Continued.

I. *Metallic Arsenic.*

§ 127. METALLIC arsenic is known under the name of mineral kobbolt (or "mineral devil," a name given by German miners), fly-poison, fly-powder, and fly-stone. It has a bluish-gray fracture and a metallic lustre, and by exposure to the air, becomes gradually black, and loses its lustre. It is easily pulverized, and has neither taste nor smell. Exposed to heat, it gives out an alliaceous odor, and in the atmospheric air its vapors are changed into the white vapor of arsenious acid. Accidental death is not uncommon from its use. A case is mentioned[1] in which a child, two years of age, died in consequence of taking it by mistake.

A highly interesting case of homicidal poisoning with it has been reported by Dr. Schütte.[2] It is, we believe, with one exception, the only one on record.[3] The wife of a barber, named Dombrowsky, was suddenly attacked, on the morning of the 11th of April, with violent vomiting and purging, with pain and heat in the epigastrium, and excessive thirst. A physician was called to visit her in the evening, but, being ignorant of the cause of the attack, prescribed simple remedies. She had no fever, her pulse was slow and soft, and the abdomen was not tender upon pressure. The vomiting, and especially the purging, still continued; and although they afterwards abated, her strength sank rapidly, and she died on the sixth day. On the third day after death an examination of the body was made. There was not found any natural cause of death. But, on opening the stomach, it was observed to have no putre-

[1] Boston Med. and Surg. Journ., vol. xxx. p. 17.

[2] Casper's Vierteljahrs., Oct. 1854.

[3] Briand, Méd. Lég. 452. (7th edition.)

factive smell; it was red in streaks, and the mucous membrane was softened. There were also several hemorrhagic erosions, especially in the neighborhood of the cardiac orifice. Some small black particles, having a metallic lustre, were seen upon it. These were carefully detached, washed with distilled water in a porcelain capsule, and then reduced on charcoal by means of the blowpipe. They gave out the well-known garlicky odor. The same odor was perceived when some were put in a reduction tube and exposed to heat, and both an arsenical crust and the small white octahedral crystals of arsenious acid were obtained. Evidence of the presence of arsenic was also obtained by the usual liquid tests. By Marsh's apparatus, arsenic was detected in the fluids contained in the stomach, and the amount obtained from this, and from the subsequent analysis of the stomach itself, was computed at nineteen grains. Additional circumstantial evidence, which fixed the crime upon the husband, was derived from the examination of a few particles of a shining black powder found in the extreme end of his pocket, and also from the analysis of the dried spilled contents of a cup of sago, which, intending to give to the deceased, he had placed in the stove to warm, but which had been cracked by the heat. It was also proved conclusively that he had himself purchased, at several times, portions of arsenic and of kobbolt, which were found in the house. This fact, it may be remarked, was clearly brought home to him by the commendable provision of the Prussian code, which requires that a person purchasing poison shall give a receipt therefor to the apothecary, containing his name, address, the date, and also the alleged purpose for which it is required. These receipts were produced upon the trial. The prisoner was convicted chiefly upon this and the admirable and minute chemical investigation, and sentenced to be beheaded.

Metallic arsenic when perfectly pure is not poisonous, but its poisonous effect in the above cases is explained by the fact that, when exposed to the action of a moist atmosphere, it becomes partially oxidized and changed to the form of white arsenic. This mixture was formerly much used as a fly poison, under the name of fly-powder. Metallic arsenic is also partly oxidized when placed in water, the white arsenic which is thus formed is dissolved in the water, and this solution has also been used as a fly poison under the name of fly-water. The principal cases of poisoning by metallic

arsenic (with the exception of the two cases mentioned above) have been due to the ingestion of these fly poisons by children.

§ 128. The principal compounds of arsenic used in poisoning are:—*Arsenious acid*, also called arsenious anhydride, arsenious oxide, white arsenic, and simply arsenic; in common and legal phraseology, when the word "arsenic" is used, it is this compound of arsenic and oxygen which is always referred to; pure arsenic is always spoken of as metallic arsenic:—*Arsenites* or compounds of arsenious acid with bases, the principal ones being the arsenites of sodium, potassium, and copper; the arsenite of copper is also called Scheele's green, Paris green, Schweinfurt green, emerald green, and by several other names:—*Arsenic acid*, which is also a compound of arsenic and oxygen, containing two more atoms of oxygen than arsenious acid:—*Arseniates* or compounds of arsenic acid with bases, the principal ones being the arseniates of sodium and potassium:—*Sulphides of arsenic*, of which the two principal ones are the red sulphide, or realgar, containing two atoms of sulphur with two of arsenic, and the yellow sulphide, or orpiment, containing three atoms of sulphur with two of arsenic; neither of these when pure is poisonous, but, when exposed to the action of moist air, they become partially converted into white arsenic:—*Arseniuretted hydrogen*, which is a gaseous compound of arsenic and hydrogen:—*Arsenical colors*, which include the above-mentioned copper and sulphur compounds, and a large number of aniline and other coloring matters.

II. *Arsenious Acid.* (Arsenic; White Arsenic; Arsenious Oxide.)

§ 129. The poison which is generally known under the name of arsenic, or white arsenic, is one of the oxides of the metal, and has a slight acid reaction. It is met with in one of two forms, either as a white, vitreous, crystalline substance, or as a white opaque and granular powder. It is sparingly soluble in cold water (about one in five hundred parts), but is more freely dissolved by boiling water, which takes up the acid in about the proportion of one of the acid to ten or twelve of water.[1] Dr. Taylor found, by numerous experiments, that a fluidounce of hot water took up, in cooling from the

[1] Will's Analysis.

boiling point, nearly one grain and a quarter of white arsenic—about one part in four hundred of water; but that, if boiled for an hour on the poison, and allowed to cool, the water held dissolved about twelve grains to the ounce—about one part in forty. In some experiments made by Schroff upon the solubility of arsenious acid, he found that one part of the acid in 480 of water, the liquid being frequently shaken, was not fully dissolved in fourteen days. He boiled one part of arsenic with 100 of water, and found that complete solution took place after fifteen minutes' boiling.[1] It is even less soluble in some liquids containing organic matter. The different statements with regard to its solubility possibly depend upon the difference in this respect between the crystalline and the opaque powder. Its *taste* is not, as was formerly represented, acid, but, on the contrary, is, when at all perceptible, rather sweetish. It is sometimes described as bitter, usually rough, etc. As a general rule, it may be stated that it is without taste, except when in solution, when the taste perceived may be faint and sweetish. The sparing solubility of this substance is the cause of its want of taste, and also explains the fact that, in the majority of cases of poisoning by it, it is found in larger or smaller quantity in the matters vomited, or adhering to the mucous coat of the stomach. Its solubility is increased by the presence of an alkali or an alkaline carbonate. It is also much more soluble in hydrochloric acid than in water.

§ 130. This poison gives rise to a very large number of cases of poisoning, criminal and suicidal as well as accidental. The number of cases met with in Europe has much diminished of late years, on account of the numerous restrictions placed upon its sale and purchase by law; but in this country arsenic is so largely used as a preservative for hides and other substances, for stuffing birds, and in many manufactures, that it is a very easy matter for any one who desires it, to obtain possession of a large amount of arsenic without any record being made of the purchase; this ease with which it may be obtained, and its freedom from taste, so that it cannot be detected immediately upon being taken into the mouth, render it a favorite poison for the criminal.

§ 131. *Symptoms.*—The symptoms occasioned by poisoning with arsenic do not always manifest themselves immediately upon its in-

[1] Canstatt's Jahresbericht for 1853, Bd. v. p. 52.

gestion, and this is particularly the case when the poison has been introduced into some article of food or drink, and taken at a meal. Still they may occur immediately. A child, three years old, drank from a saucer some arsenious acid mixed with milk. It was seized *immediately* with pain, vomiting, and diarrhœa.[1] In the case referred to by Dr. Taylor, the symptoms were proved to have attacked the deceased while he was in the act of eating the cake in which the poison was administered. In the case of Sager, tried in the State of Maine, in 1834, quoted by Beck,[2] extreme distress was *immediately* experienced after taking the poison. Generally the symptoms are not perceived until a later period, which is usually stated at from half an hour to an hour after the poison had been swallowed. On the other hand, they have been, in some cases of poisoning with arsenic, delayed in an unaccountable manner. In a case related by Dr. Ryan, where half an ounce of arsenic was taken in porter, the first symptom, which was vomiting, did not occur until nine hours afterwards. Mr. Clegg was called to see a girl who had taken a teaspoonful of arsenic, but who was supposed also to be addicted to the use of opium. Seven hours after she had taken it she appeared stupid, as if intoxicated, but no further symptoms of irritation occurred until near noon of the following day, when, although she had been cheerful all the morning, and was engaged in preparing dinner, she was suddenly seized with excruciating pain in the stomach, and died in half an hour, about twenty-four hours after taking the poison.[3]

A fatal case is reported[4] where no symptoms appeared for three days, though it is proper to add that iron preparations had been administered as antidotes; this patient died on the sixth day. In another case[5] the symptoms appeared in six hours after the dose had been taken; this patient also died. In another case of a man who took half an ounce of arsenic while intoxicated, the symptoms

[1] Henke's Zeitschrift, E. H. 43, p. 150.

[2] Vol. ii. p. 546.

[3] Lancet, vol. ix. p. 31. A case is also related by Belloc, in which *ten hours* elapsed after the taking of the poison before any symptoms showed themselves; the vomiting was then slight, as also the pain in the abdomen, and no mention is made of the occurrence of diarrhœa. She died as from the effects of a narcotic poison. Cours de Méd. Lég., p. 122.

[4] Medical Times and Gazette, July, 1843, p. 288.

[5] London Lancet, 1848.

of arsenical poisoning were delayed for nine days; this victim had been intoxicated with porter for a week before swallowing the fatal dose of arsenic. He lived three days after the symptoms of poisoning were manifested.[1] In still another case[2] a woman took a similar dose after a meal, and the poisonous symptoms appeared in eight days; she recovered.

§ 132. Authentic instances are on record in which there has been also an intermission in the regular progress of the symptoms. Thus, in the case of the girl Davidson, reported by Dr. Maclagan, the vomiting diminished on the fourth day, was trifling on the fifth, was absent on the sixth, but returned, accompanied by purging, on the night of the seventh. It is stated that there could not have been a repetition of the dose. So also in the case of McVey, by the same author, the man was taken ill with the symptoms of irritant poisoning about half an hour after eating an oat "bannock." Although he appeared to be very ill in the mean time, he was not again seized with vomiting until the morning of the fourth day, and died three days thereafter. "It did not appear that anything had been given him which would have contained a fresh dose of the poison."[3] Dr. Christison says: "A short remission, or even a total intermission, of all the distressing symptoms has been witnessed, particularly when death is retarded to the close of the second or third day. This remission, which is accompanied with dozing stupor, is most generally observed about the beginning of the second day. It is merely temporary, the symptoms speedily returning with equal or increased violence."

§ 133. The symptoms usually begin with a sensation of sickness, and a burning heat in the stomach. There is also a sense of constriction and heat in the throat, with great thirst, and violent efforts at vomiting soon take place. The substances vomited have no peculiar color, as this depends both upon the matters that happen to be present in the stomach, the antidotes administered, and the length of time the vomiting continues. When the stomach is empty, mucus streaked with blood, and yellow or greenish bile will communicate a color to the contents of the basin. If powdered arsenic have been swallowed, it may sometimes be recognized in the ejected

[1] This case is reported in the Medical Times and Gazette, 1851, April 21.

[2] Lancet, Oct. 19, 1844.

[3] Edinb. Month. Journ., Jan. 1853.

matters by its white and flaky appearance. The irritation of the poison being communicated to the lower bowels, diarrhœa usually supervenes, and is frequently thin and bloody and attended with much straining and distress, and cramps in the calves of the legs. When the latter symptoms are urgent, they are usually attended with an inability to pass the urine. These symptoms all increase in gravity till near the close of life. The general system also sympathizes with the disturbance of the digestive organs; the countenance is collapsed and anxious, the extremities and the surface generally ice-cold, the pulse almost imperceptible, the respiration accelerated, the voice oppressed; and convulsions, delirium, and stupor not unfrequently usher in the closing scene. Such is a picture of the ordinary train of symptoms in a case of poisoning by arsenic.

§ 134. It should be understood, however, that they are liable to many variations, and authentic cases are related in which the symptoms resembled, to a certain extent, those of narcotic poisoning. The system appears in such cases to be completely overpowered by the toxical effects of this substance, and extreme faintness or depression is the most prominent symptom. The pain in the abdomen and the vomiting are occasionally not urgent, except towards the close of life, and in rare cases have been absent. These variations, when occurring in persons addicted to the use of opium or of ardent spirits, may be partially accounted for, but often they must remain unexplained.[1] Maschka[2] records four cases of poisoning occurring together, and caused by drinking coffee containing arsenic, one of which was of this narcotic form in a child six years of age who slept for three hours after taking the arsenic, then waked, complained of abdominal pain and thirst, drank some sweetened water, and again fell asleep: a short time afterwards when the physician arrived she was dead.

[1] For cases illustrating these points, *vide* Christison on Poisons. Also, an interesting case by Dr. Ogston (Med. Gaz., 1851). In this there was headache, stupor, feeble pulse, cold extremities, nausea, and tonic and clonic spasms. Vomiting did not occur until several emetic doses of sulphate of zinc had been given, and then only two hours and forty minutes after the arsenic, amounting to three drachms, had been swallowed. The poison was discovered in the blood, liver, and contents of the stomach, the patient having lived six days. See, also, Appendix.

[2] Gerichtliche Medicin, ii. page 260.

§ 135. For convenience of reference, the following brief summary of the usual symptoms occurring in acute arsenical poisoning is given. It will be seen by the above cases and by the detailed reports given in the Appendix, that we may divide cases of acute poisoning by arsenic into two classes, according as death takes place rapidly within a few hours, or, as is most common, is delayed for twenty or more hours. In the first class of cases, which usually occur after the taking of a large amount of arsenic upon an empty stomach, the symptoms begin quickly with a burning sensation in the throat, gullet, and stomach, which is quickly followed by nausea and vomiting; there is great thirst, intense pain in the pit of the stomach, soon followed by diarrhœa and painful discharges which are usually watery, sometimes resembling the rice-water discharges of cholera, and sometimes they are bloody like the discharges of dysentery. There is usually intense headache, the limbs are cold, the pulse becomes nearly imperceptible, prostration is very great, there is repeated fainting, and no urine, or only a very small quantity, is passed; the expression of the face shows great anxiety, the lips, and often the skin, become blue and livid, convulsive twitchings of the arms and legs occur, and death takes place within twenty-four hours after taking the poison, often in from five to ten hours. It is this form of poisoning which may be mistaken for Asiatic cholera, especially during an epidemic of that disease; the main points of difference in the symptoms of the two diseases are the burning pain in the throat and gullet, and the fact that in arsenical poisoning the vomiting takes place before the purging; chemical analysis of the urine passed during life or of the organs after death must, of course, be resorted to for confirmation of the diagnosis. In very rare cases of this most acute form of poisoning, like that recorded by Maschka mentioned above, the symptoms assume the narcotic form, and pain, vomiting, and purging may be entirely or partially absent, death taking place within a few hours without pain.

§ 136. In by far the majority of the cases of arsenical poisoning, however, the progress of the case is much less rapid, the life of the patient being prolonged in many cases for several days after taking the poison, so that some authors have preferred to call this class of cases subacute, rather than acute. In these cases the vomiting which occurs at the beginning of the attack is abundant, frequent, and continues from twenty-four to thirty-six or forty-eight hours,

when it ceases for a short period, varying in different cases; during this remission there may be no stomach symptoms, although the sensation of heat in the throat and gullet, the great weakness, excessive thirst, irregular action of the heart and coldness of the limbs remain, and often also the burning pain through the entire length of the intestines with diarrhœa and painful discharges. In some cases there is so much pain in the throat and gullet that it is very difficult for the patient to swallow. In a longer or shorter time, according to circumstances, reaction occurs and the symptoms return often with increased violence without any fresh dose of the poison having been administered. The abdomen becomes swollen and very tender to the touch, and the general symptoms resemble those mentioned under the first class. In some cases an eruption appears upon the skin, this eruption varying in different cases, sometimes it consists of mere redness, at other times of pimples which may be filled with a watery fluid or with matter. In rare cases the patient has jaundice. In this form of poisoning death usually occurs in from two to eight or ten days.

§ 137. The shortest fatal period in acute poisoning by arsenic is recorded by Taylor in the case of a young man who died with tetanic convulsions in twenty minutes after the beginning of the symptoms. Recorded cases show no relationship between the amount of poison taken, and the rapidity of the symptoms and fatal period, these depending upon other conditions, such as the condition of the stomach, solubility of the preparation, etc.

§ 138. When, however, instead of one dose sufficient to destroy life, or several doses at short intervals, capable of producing this effect, the poison is given in small portions at comparatively distant intervals, the symptoms are not so marked in their succession, and are attended with phenomena different from those already described. The following may serve as an example: "A woman put daily into the soup of her fellow-servant a very small quantity of arsenious acid in powder. Shortly after dinner, this person was seized with vomiting which led to the rejection of the food and poison before the latter had caused any serious mischief. As this practice was continued for about six weeks, the stomach grew exceedingly irritable; there was pain in the bowels, and the woman became much emaciated. There was also spitting of blood, with such a degree of nervous irritability, that a current of air caused an attack of spasms

and convulsions. When the patient found that she could not bear anything on her stomach, she left the place and passed two months in the country. Her health became gradually restored there, and she returned to resume her usual occupations. The prisoner, however, renewed her attempts; and, to make sure of destroying her victim, gave her one morning, in coffee, a strong dose of arsenious acid in powder; violent vomiting ensued, and the poison was expelled with the breakfast. Arsenic was detected in the vomited matter, and the explanation of the cause of the long previous illness became clear. Under proper treatment, the patient recovered."[1] Christison relates a case somewhat similar, which, however, was not so protracted, and which terminated fatally.[2] It was by this means, probably, that the crime of secret poisoning was carried in the seventeenth century to such a fearful extent. The miserable woman who vended the liquid, called after her, *Aqua Tofana*, confessed at her death that she had destroyed by it no less than six hundred persons. It is generally supposed that its active ingredient was arsenious acid.

§ 139. Those who have partially recovered from the immediate effects of arsenical poisoning, are, moreover, liable to the secondary effects, above referred to: salivation, chronic intestinal disorder, palsy, dropsy, and an irritative fever soon prostrate the vital powers, and the fatal termination, although sometimes slowly attained, is, in the majority of cases, none the less certain. The period at which death supervenes cannot be definitely stated with reference either to these cases or to those of acute poisoning. In the latter it may take place within twenty-four hours; it may be postponed for several weeks or months. The average period in twenty-two cases reported by Dr. Geoghegan, was seventy-seven hours and a half, the shortest was five and a half hours.[3] Cases are, however, known, in which death has taken place within two hours;[4] and in the case above mentioned in twenty minutes.

[1] Taylor on Poisons, from Flandin, p. 257.

[2] Loc. cit., p. 250, Am. ed.

[3] Dublin Quarterly Journ., Feb. 1851.

[4] The following case will be found in the Lancet, Oct. 1845, by Mr. Iliff: "E. D——, the servant in a family, after placing the dinner on the table, retired to her chamber, and drank a glass of water, in which she had mingled as much arsenic as it would dissolve; *she fell directly and died instantly;* no struggling whatever took

§ 140. Arsenic is equally noxious when *inhaled* in the form of vapor, or applied *externally* to a denuded surface, or upon the mucous membrane of the *vagina* or *rectum*.[1] Its effects are extremely rapid when it is inhaled, but when it is absorbed from a wounded surface the symptoms usually do not occur so soon as when it is swallowed. Being an ingredient in most of the pastes used by cancer-curers in the extirpation of scirrhous breasts, it is by this means not unfrequently introduced into the system, and has produced death with all the symptoms of arsenical poisoning. Even the small proportion of arsenic which is contained in the stearine of some candles, has, when the latter has been used for the purpose of dressing a blistered surface, produced nausea, pain in the stomach, thirst, redness of the tongue, spasms of the muscles, weakness and irregularity of the pulse, and death within twenty-four hours.[2]

Several cases are quoted by Christison from Fodéré and others, where arsenic given by injection into the rectum proved fatal, and introduced into the vagina caused death in less than twenty-four hours. It is said, moreover, to have produced violent symptoms when applied to the unbroken skin, as when used as a depilatory.

§ 141. A case of death from the external application of arsenic to the head of a child two years of age, affected with porrigo favosa, is related by Dr. McCready, of New York. A woman obtained about half an ounce of arsenic, and, mixing it with gin, rubbed it well into the heads of several of her children affected with this disease. It was followed by redness and swelling of the face; in the child alluded to, however, it produced diarrhœa and tenesmus, with paralysis of the lower extremities, but no signs of local inflammation. The mother stated that she had on one previous occasion applied the arsenic, and, though the application was followed by some swelling, this soon subsided, and the head seemed much better.[3] Dr. Mitchell, of Liverpool, relates a case in which characteristic constitutional symptoms, as well as severe local inflammation, were produced by applying a mixture of arsenic and soft soap to the pubes and axilla for the purpose of destroying pediculæ.[4]

place. I saw these two cases (referring to another published at the same time) almost immediately after the poison was taken."

[1] *Vide* Christison on Poisons.

[2] Med. Gaz., 1842–43, p. 351.

[3] Am. Journ. Med. Sci., July, 1851, p. 259.

[4] Lancet, Aug. 1857, p. 127.

Binz and Schultz[1] state that an animal may be poisoned by dropping a solution of arsenic upon the extremely sensitive surface of the eye (conjunctival membrane), and without causing any serious local irritation, though a slight reddening may follow this local application. The destructive effects upon the internal organs of the body will follow this method of application, even when apparently no trace of the arsenic has come directly in contact with their structure; these lesions are observed principally in the stomach. According to these authors those organic structures or tissues of the body which receive and appropriate oxygen, among which are the glandular structures, are the special seats of destructive action. The neutral salts are just as poisonous as arsenious acid. These authors believe that both arsenic and phosphorus, as well as other reducing agents, act upon the cell growths by discharging their atoms of oxygen and then become changed into chlorides, and in this latter form exert a corrosive action which destroys the cells and blood. As further corroboration of their views they bring forward the fact that this rapid change by oxidation has been proved in the case of antimony, bismuth, and vanadium.

§ 142. The dose necessarily fatal (*vide* § 138) is not exactly known, though it might, in general terms, be stated as ranging from one-fifth of a grain (.012 of a gramme) to twenty-two grains (1.15 grammes); Tardieu mentions,[2] very truly, that this question loses its importance in practice, for in cases of criminal poisoning or of suicide it is very rare that the dose of arsenical poison swallowed does not exceed the quantity necessary to cause death, and that it does not attain five to ten or fifteen grammes (about seventy-five grains to half an ounce).

§ 143. In 1854 Dr. Tschudi published an account of the Toxicophagi of the lower countries of Austria and Styria. These persons were in the daily habit of taking large doses of arsenic, with the effect of improving the freshness of their complexion and becoming more active and free in respiration.[3] When this practice was suddenly discontinued, emaciation and gastric disturbance ensued.[4] It is well known that horse dealers give horses small quantities of

[1] Berichte d. d. Chem. Gesellschaft, xii., 2199; also in Archiv f. experiment. Path. u. Pharm., 200–230.

[2] Op. cit., p. 322.

[3] Wiener medizinische Wochenschrift.

[4] Med. Times and Gazette, London, July, 1854, p. 66.

arsenic, either mixing it with their oats or attaching a piece of linen inclosing a small fragment to their bits, and with the effect of improving the appearance of the animals. The accounts given by Tschudi and Vogt[1] have been opposed by Pereira, Christison, and others, especially Taylor,[2] who cites reasons why the fact of arsenicophagism, true or false, can be of no practical value to the medico-legist. Prof. La Rue[3] gives an interesting account of a man who was in the daily habit of eating and smoking arsenic without producing any but favorable symptoms. In the presence of the professor he swallowed three grains of pure arsenious acid, then a minor dose weighing a grain and a half. Another half grain he smoked, mingled with his tobacco, filling the laboratory with the odor of garlic. He the next day swallowed four grains of arsenious acid, and on the day following was perfectly well and free from any gastric disturbance. There can be but small doubt that the arsenic in these cases of arsenic eaters is absorbed; in fact Dr. Maclagan had some urine of toxicophagi collected and chemically analyzed, the arsenic being by this means recovered.[4] The medico-legal bearings, however, of such instances have a very definite aspect; because the habitual use of any drug, arsenic, opium, morphine, quinine, or any other, except hydrate of chloral, causes the consequence of acquiring tolerance by the gradual increase from a small, safe dose to that which, in a person unaccustomed to its use, might be a fatal dose. It results, therefore, to a simple question of evidence whether in any given case of doubtful poisoning, the victim has by habit become tolerant of what are ordinarily considered fatal doses.

§ 144. *Post-mortem appearances.*—The principal reliable and tolerably constant changes produced by arsenic in the healthy appearance of the viscera, are found in the stomach and intestines. The mucous membrane of the stomach is usually of an uniform deep brownish-red color, sometimes it is ecchymosed in patches, and at others there are spots or streaks of effused blood. These often have the appearance of crusts, and, being of a blackish color, are not unfrequently mistaken for gangrenous patches, and the slight

[1] Med. Jahrb. des öster. Statts, 1822, i. 99.

[2] On Poisons, London, 1859, p. 91; and Med. Jur., Phila., 1883, p. 185.

[3] Boston Med. and Surg. Journ., June 28, 1866, p. 439.

[4] Edinburgh Med. Journ., Sept. 1867.

depression under them for ulceration. But neither ulceration nor gangrene is an ordinary result of simple arsenical poisoning; if found, these are probably merely concomitant lesions, dependent upon other causes.[1] Perforation is exceedingly rare. Orfila says that he has never observed it.[2] The mucous membrane is also frequently swollen and thickened, possessing in some parts a fungoid appearance, and its structure is more frequently condensed than softened, owing possibly to a chemical union between the arsenic and the albumen. The powder, if the poison have been taken in this form, is often found imbedded between the folds of the mucous membrane, and closely adherent to it in brilliant points, or in white and flaky patches. The matters contained in the stomach are evidently too variable in character to be enumerated, since the ingestion of different liquids, and of the many so-called antidotes which have been given in most cases, naturally destroys the possibility of drawing any useful inference.

In addition to these inflammatory appearances in the stomach and intestines in cases of acute poisoning, evidences of the irritant action of the arsenic are found in other portions of the body, especially in the kidneys, which are irritated by the poison as it passes through them, and in some cases this irritation reaches such an extent that these organs are found much congested.

§ 145. Attention has already been directed to cases of acute and subacute fatal poisoning by arsenic (§ 135). In these cases the organs concerned in the maintenance of animal or organic life, especially the liver, kidneys, heart and glands of the stomach, undergo a serious anatomical change in which the proper healthy cells, which are essential to the functional activity of these organs, assume a retrograde change or degeneration of structure, whereby they cease to be active. Consequently the natural organic functions cease, and life is arrested. The *post-mortem appearances* in many of these cases can only be estimated or understood by a

[1] In the Lancet for Sept. 1843, it is reported that the body of a man poisoned by arsenic was disinterred one hundred and forty-one days after death. The stomach and intestines were in perfect preservation. About the middle of the small intestine was found a small ulcerated opening, through which some of the white powder was detected, similar to what was found in the stomach, and which proved to be arsenic.

[2] Méd. Lég., vol. iii. 330.

microscopical examination; this will show to an experienced eye that the cell growth has been replaced by degenerated or fatty tissue, the cells themselves being diminished in number or entirely absent. That this fatty degeneration may occur in an organ as the result of disease and not the consequence of arsenical or other poisoning is well known to the medical profession, and yet, as may be learned by reference to a previous paragraph (§ 144), an experienced pathologist can almost always by *post-mortem* observation determine whether the lesion be the consequence of disease or poisoning. Moreover the chemical examination can settle positively any doubt which may arise. These remarks apply with equal force to the subacute poisoning by phosphorus, ammonia, antimony, or other poison producing similar *post-mortem* appearances, which have been above described.[1]

§ 146. The introduction of arsenic into the system by external application (see § 141) is usually followed by the same alterations in the stomach and intestines, as when it has been brought into direct contact with the mucous membrane of these viscera.

In medicinal doses, the solution of the oxide of arsenic produces sometimes serious symptoms, and cannot be increased without at once causing symptoms of poisoning. The medicinal dose is from one-sixteenth to one-twelfth of a grain, and half a grain is sufficient to produce very alarming symptoms. Physicians are accustomed to watch the accession of conjunctivitis, swelling of the eyelids, gastrodynia, and general depression, as indications of the dose being too great or too frequently repeated.

§ 147. The facts relative to the effect of *arsenic upon the putrefactive process* are of a very contradictory character. A number of cases are quoted by Dr. Christison, which appear to prove a remarkable antiseptic property in arsenic, by which not only the digestive organs, but the whole body, have been preserved from the ordinary changes of putrefaction. There is no doubt of the preservative quality of an arsenical solution over organic textures placed and kept in it, and the experiments made by Klenck upon dogs seem to show that in cases of poisoning, also, this property may be witnessed. This physician poisoned dogs with arsenic, and left them for two months, sometimes buried in a damp cellar, and

[1] See Appendix.

sometimes unburied in the same place; the flesh and alimentary canal were red and fresh, as if pickled, at the end of this time. Dr. Kelch, of Königsberg, buried the internal organs of a man who had died of arsenic, and whose body had remained without burial till the external parts had begun to decay, and on examining the stomach and intestines five months after, he found that the hamper in which they were contained was very rotten, but that they had a peculiar smell, quite different from that of putrid bowels, were not yet acted on by putrefaction, but were as fresh as when first taken from the body, and might have served for the purpose of anatomical specimens. The body of Chapman, supposed to have been poisoned by Mina, was disinterred two months after death. The face was livid and putrid, but the odor of the corpse was not offensive. The abdomen was of a pale-white color, and Dr. Hopkinson, on cutting into it, was struck with its firmness and resistance. When the stomach was opened, a very peculiar smell was perceived, which he compares to that of pickled herring. The same remark was made by other medical witnesses.[1] The intestines were entirely empty, of a pale color and apparently rather disposed to dry than to putrefy. In a case communicated by Dr. Traill to Dr. Christison, the body of a captain of a vessel was disinterred five months after death. The face and neck were swollen, black, and decayed, but the rest of the body was quite free from the usual signs of putrefaction. The skin was white and firm, the muscles fresh, the lungs crepitating, the liver and spleen much shrivelled, the stomach and intestines entire throughout their whole extent, and capable of being handled freely without injury. In this instance the coffin contained water, owing to its having lain in a sandy soil resting on clay. The remarkable preservation of the body of a woman supposed to have been poisoned by arsenic, for nearly *fourteen* years after her death, led to its disinterment, the indictment and trial of her husband. Arsenic was found in the body.[2]

§ 148. On the other hand, Dr. Geoghegan has observed examples both of very tardy and of very rapid decomposition in cases of ar-

[1] In two cases of poisoning by arsenic, observed by Dr. Sanborn, of New Hampshire, the same peculiar odor was distinctly observed. Bost. Med. and Surg. Journ., vol. xxxvii.

[2] Webster, Bost. Med. and Surg. Journ., vol. xxxix. p. 489.

senical poisoning.[1] It would not be difficult also to find many cases in which, although death has resulted from other causes, the body has been as remarkably preserved as in those where arsenic was the cause of it. We have elsewhere enumerated the various causes which will retard putrefaction, as the dryness of the soil, and the depth at which the body has been interred, as well as individual peculiarities, which do not always admit of explanation. We may quote here an observation which will show, that, even under the most favorable circumstances for decomposition, this process may proceed very slowly. This is a case communicated by Dr. Routier, of Amiens, to Orfila, in which an old woman was destroyed by a blow upon the head with an axe. This was in the middle of summer. The body lay buried in the clayey soil of a cellar between eight and nine months. At the end of this time, a judicial inquest was held. The skin was perfect, the muscles firm, red, and distinct, the cerebrum was like that of a fresh corpse, and possessed its natural firmness and proper odor. The viscera of the chest and abdomen were perfectly well preserved; and in the stomach, which presented no signs of inflammation or other disease, a thick fluid was found in which the remains of articles of food were distinctly recognized.[2] The fact of the remarkable preservation of the brain in this case is also of some importance, for it is well known that usually it putrefies rapidly, and because also of a case lately reported by M. Dieu, where the body of a man poisoned by *arsenic* was disinterred after the lapse of two years and a half, the comparative integrity of the brain was attributed to the preservative powers of this substance.

In a summary furnished by Tidy,[3] it is asserted "that arsenic, antimony, chloride of zinc, also chloroform, phosphorus, and strychnine, when they are actually the cause of death, usually retard decomposition. In arsenical poisoning, putrefaction ordinarily commences as usual, but seems to stop after it has commenced, then a process very similar to mummification begins." And again,[4] "thus arsenic acts commonly (but by no means necessarily) as a preservative agent, so that in a case of arsenical poisoning, a fairly accurate opinion may sometimes be formed as to the

[1] Dub. Quart. Journ., Feb. 1851.

[2] Orfila, Traité de Méd. Lég. 4ème ed., vol. ii. p. 93.

[3] Legal Medicine, Philadelphia, 1882, p. 91.

[4] Idem, p. 106.

condition of the mucous membrane after the lapse of even three or four weeks.

Hence the medical witness cannot be authorized to assert that, because the body has resisted more or less completely the progress of putrefaction, this preservation is due to arsenic, since it may be really attributable to other causes. One cause of the discrepancy in the observations upon this point undoubtedly lies in the variable time occupied by the poison in producing its fatal effect, and the nature of some of the symptoms. It is reasonable to suppose, and observation also has shown, that, if death have resulted from the ingestion of the poison at repeated intervals, in small doses, or not until several days have elapsed, the arsenic has been disseminated through the system, and may thus exercise a more complete antiseptic influence in the dead body. If, again, the person has died within a short period after taking the poison, and after abundant and repeated vomiting and purging, we may be right in anticipating, that, although the violent action of the poison has been the cause of death, little or none will have remained in the body. Hence, in the latter case, putrefaction will probably pursue its ordinary course. Thus, in a case examined by Dr. Geoghegan, of a person who died sixteen days after taking a large dose of arsenic by mistake, no trace of the poison could be found in any part of the body.

§ 149. *The period of time* requisite to develop the inflammatory condition of the stomach is altogether a matter of conjecture, since the mucous coat of the stomach has been found inflamed when death has followed the poisoning at only the short interval of two or three hours; and, on the other hand, where the quantity swallowed and the duration of life have been such as to lead to the natural belief that inflammation would be discovered, the stomach has been found nearly or entirely free of any such morbid change. Indeed, in a few cases the arsenic has been observed in immediate contact with the gastric mucous membrane, without any signs of inflammation. Nevertheless, as a general rule, the inflammatory appearances will be found developed in proportion to the protraction of the case. In a case reported by Dr. Letheby, the stomach was of a pale color. Ettmüller reports the case of a girl in whose stomach arsenic was found, and yet neither in it nor in the intestines was there a trace

of inflammation.[1] Orfila, in his work upon legal medicine, says: "The existence or non-existence of cadaveric lesions, the extent and seat of these alterations, can never enable us to affirm that poisoning has taken place, but can only serve to corroborate the conclusions drawn from the symptoms and the chemical examination of the suspected matters." These remarks are confirmed by observations of Marc and Chaussier. The inflammatory appearances seldom extend further than the duodenum, although sometimes the small intestine and the rectum exhibit evidence of inflammation. In general, there are no other *post-mortem* changes at all characteristic of this mode of death. The blood is said to be often syrupy in consistence.

§ 150. The time required for the complete *elimination of arsenic* from the system has of late years become quite an important medico-legal question, especially in cases in which life has been prolonged for several days, so that the chemical analysis shows only small amounts of arsenic in the organs after death. Various authors give as the time required for the complete elimination about one month, but cases are recorded which show that it may completely disappear in a less time than this. Thus in the case of Dr. Alexander,[2] who took a large dose of arsenic by mistake, and died in sixteen days after taking the poison, no arsenic could be detected in the organs after death. In Dr. Maclagan's case,[3] arsenic was detected in the urine up to the twenty-first day after taking the poison. In another case of acute poisoning investigated by the writer, arsenic was detected in the urine on the nineteenth day, but was absent from the urine on the twenty-third day. In Dr. Gaillard's case, referred to by Dr. Ch. Roucher,[4] which was a case of chronic poisoning caused by repeated doses of Fowler's solution, arsenic was detected in the urine six and one-half weeks after the last dose was taken, but had completely disappeared in seven and one-half weeks.

§ 151. *Distribution of Arsenic in the Body.*—The relative proportion of arsenic found in the different tissues of the body after death has of late years become quite an important medico-legal

[1] Dr. Taylor records two cases, in one of which ulceration of the stomach was seen, when death occurred five hours after taking the poison, and in the other in ten hours.

[2] Medical Times and Gazette, April 18, 1857.

[3] Edin. Monthly Med. Journ., xiv. 1852, page 131.

[4] Ann. d'Hygiène, Oct. 1874.

question, for the following reasons: In the first place, the detection of the poison in the solid viscera, and especially in the brain, shows that the arsenic must have been absorbed from the stomach and intestines, and carried with the blood to the distant portions of the body; therefore, the arsenic must have been taken during life, while the circulation of the blood still existed, and could not have been introduced into the stomach after death. In the second place, after the arsenic has been absorbed and carried to the various organs and tissues with the blood, a larger or smaller amount is deposited in these tissues and, as it were, stored up in them temporarily; and experience has shown that the relative proportion of arsenic stored in the different tissues and organs varies, and also that the amount in the same organ varies at different periods after the ingestion of the arsenic. In the liver, for example, analyses made by Dr. Geoghegan[1] showed that this organ probably contains the maximum amount of the poison in about fifteen hours after it is swallowed. He tabulates the result of the analysis of seven cases as follows:—

"In	5½ to 7 hours	0.8 grains.
	8¾ "	1.2 "
	15 "	2.0 "
	17 to 20 "	1.3 "
	10½ days	1.5 "
	14 "	0.17 "
	17 "	nil."

Therefore, it is evident that, when a sufficient number of complete quantitative analyses of the different organs in cases of arsenical poisoning have been made, we shall have data which may show, within narrow limits, at what period before death the poison was taken.

For the purpose of comparison, and in order to stimulate further investigations in this direction, we give the following figures, recognizing the fact, however, that many more complete analyses are necessary before accurate inferences as to the time of ingestion, etc., can be drawn from a knowledge of the amount contained in the various organs. The amounts found in the stomach and intestines are, of course, of little value in connection with any of the questions regarding absorbed arsenic.

Case 1. In the case of an adult female, who lived about two days

[1] Taylor, On Poisons, 3d Amer. ed., 1875, p. 40.

after taking a fatal dose of arsenic, the following amounts were found:—[1]

	Total weight.	Weight of arsenic.
Stomach	179 grms.	0.0442 grm. = 0.68 grain.
Contents of stomach	6 "	0.0097 " 0.15 "
Intestines	490 "	0.0638 " 0.98 "
Contents of intestines	62 "	0.0205 " 0.32 "
Liver	1227 "	0.0497 " 0.76 "
Left kidney	149 "	0.0043 " 0.06 "
Right kidney	125 "	0.0026 " 0.04 "
Uterus[2]	318 "	0.0065 " 0.10 "
Brain	521 "	0.0088 " 0.069 "

In this case the organs were not examined until five months after death, so that the weights do not represent the weight of the fresh tissues.

Case 2. In the following case of an old gentleman who lived about a week after the symptoms of poisoning commenced, the organs were removed from the body about three months after death, and the analyses made in the same manner as in the foregoing case, and with the following results:—

	Total weight of organ.	Weight of arsenic.
Intestines	813 grms.	0.0203 grm.
Contents of intestines	198 "	0.0069 "
Liver	1555 "	0.0479 "
Both kidneys	309 "	0.0116 "
Brain (part of)	626 "	0.0108 "

Case 3. Adult male; duration of illness unknown; organs removed from the body and examined about eleven months after death:—

	Total weight of organ.	Weight of arsenic.
Stomach	98 grms.	0.009 grm.
Contents of stomach	5 "	0.0022 "
Intestines	347½ "	0.0103 "
Contents of intestines	40 "	0.0043 "
Liver	1028 "	0.0878 "
Spleen	46 "	0.0032 "
Heart	162 "	0.009 "
One kidney	67 "	0.0043 "
Brain	762 "	0.0052 "

[1] The analyses were made by Dr. E. S. Wood, Prof. of Chemistry in the Harvard Medical School, and by Dr. Wm. B. Hills, Instructor in Chemistry in the Harvard Medical School.

[2] The uterus was enlarged and contained a number of fibroid tumors.

Case 4. Reported in Appendix.

	Amount of arsenic.	Amount of copper.
Intestines	0.0005 per cent.	0.0003 per ct.
Liver	0.0133 grm.	0.009 grm.
Kidneys	0.0015 "	0.00076 "
Heart	trace	0.00051 "
Lungs	0.0007 per cent.	0.00047 per ct.
Brain	0.0024 grm.	0.0018 grm.
Muscles	0.00025 per cent.	0.0002 per ct.
Breasts	0.0003 "	0.0005 "
Hair	0.0011 "	none.

Case 5. Reported in Appendix.

Stomach and contents	5.78 grains.
Intestines and contents	3.86 "
Liver	2.77 "
Brain	traces.

Case 6. L. B., analysis made by Dr. Wm. B. Hills.

	Weight of organ.	Weight of arsenic.
Liver	955 grms.	0.575 grain.
Both kidneys	325 "	0.161 "
Spleen	120 "	0.126 "
Contents of stomach	80 "	0.084 "
Brain	1480 "	0.060 "

Case 7. Analysis reported by Profs. S. W. Johnson and R. H. Chittenden.[1] The organs were examined a year and a half after burial.

	Weight of organ.	Weight of arsenic.	Per cent.
Stomach and spleen	514 grms.	0.05359 grm.	0.0104
Kidneys	80 "	0.00660 "	0.00825
Liver	590 "	0.04788 "	0.00811
One lung and heart	441 "	0.01454 "	0.00329
Intestines and uterus	978 "	0.02582 "	0.00260
One lung and liquid from thorax	402 "	0.00583 "	0.00140
Bladder	73 "	trace.	
Brain	477 "	trace.	
Upper arm (left)	665 "	0.00542 "	0.00081
Forearm "	288 "	0.00158 "	0.00055
Hand "	150 "	0.00019 "	0.00012
Lower leg (right)	1323 "	0.00864 "	0.00065
Thigh	3160 "	0.01635 "	0.00051
Foot	468 "	0.00105 "	0.00022

[1] American Chemical Journal, ii. No. 5.

	Weight of organ.	Weight of arsenic.	Per cent.
Thigh-bone . . .	615 grms.	0.00040 grm.	0.00006
Transverse section of body above pelvis . .	1920 "	0.03011 "	0.00156
Muscle and ribs from left breast . . .	406 "	0.00371 "	0.00091
Abdominal muscle of right side	615 "	0.00358 "	0.00058

The above cases confirm the conclusions which have been arrived at by Ludwig,[1] that arsenic is found in the liver for a longer time than in any other organ; that the brain in cases of both acute and chronic poisoning contains only a relatively small amount of arsenic; that the kidneys may in acute poisoning contain a larger proportion than the liver. Ludwig also states that the bones may contain a small amount of arsenic in both acute and chronic poisoning, and retain the arsenic for a long time after the last dose has been taken, but not longer than the liver; he detected arsenic in the bones on the twenty-seventh day after the last dose of arsenic, but could not detect any on the fortieth day. The above experiments and analyses do not confirm the results obtained by MM. de Poncy and Livon,[2] nor those of Scolosoboff[3] and Gautier,[4] who report that the arsenic accumulates to the greatest extent in the brain and spinal cord.

§ 152. *Chronic poisoning* is caused by the absorption of small amounts of arsenic continued for a long period. This is sometimes due to taking some preparation of arsenic as a medicine, but far more frequently by its use as a pigment in ornamenting articles of household use, such as wall-paper, articles of clothing, toys, water-color paints, wrapping and other papers, and sometimes even it accidentally gets into articles of food and drink, such as confectionery and wines or syrups. The principal preparations of arsenic which are the sources of this form of poisoning are the arsenites of copper, the two sulphides of arsenic, and arsenical anilines. The persons most liable to this form of poisoning are workmen engaged in various manufactures, in which these pigments are used, especially wall-paper and artificial flowers, and those whose occupation is to place

[1] Chem. Centralblatt, 1879, No. 38, p. 602, and 1881, pp. 90, 110, and 121.

[2] Journ. de Pharm. et de Chim., Oct. 1879, p. 344.

[3] Bull. de la Soc. Chim. de Paris, xxiv. No. 3.

[4] Annales d'Hygiène, Jan., 1876, p. 136.

these papers on the wall. Two cases, one of which was fatal, are reported by Hoffmann and Ludwig.[1] A woman, 63 years of age, and her daughter, 22 years of age, were engaged in making crosses from artificial moss and flowers, a very large number being made during the few weeks before All-Souls Day. The first year both were poisoned, but the poisoning was only temporary; on the second, however, the symptoms were very severe, the daughter recovering after several months, but the mother died. After death extreme fatty degeneration of the liver, kidneys, heart, and muscles was found, and chemical analysis showed the presence of arsenic in all of the tissues except the bones, the largest amount being found in the brain and liver. The source of the poisoning was traced to the arsenical fuchsine used for coloring the artificial flowers; of six different kinds of fuchsine found in the shops only one was found free from arsenic.

It is not usual for a workman to be employed so continuously with arsenical pigments that a directly fatal result ensues; he usually suffers only with a more or less severe irritation of the skin and mucous membranes, with which the pigment comes in contact, such as inflammation of the nose (coryza) and eyes (conjunctivitis); if much of the dust gets into the mouth, there is usually a sore throat and perhaps some irritation of the stomach, which is quite certain to occur if the exposure be long continued; those portions of the skin which are habitually moist with perspiration, as in the folds of the groin, about the scrotum, and in the armpits, are especially apt to be inflamed; or if any portion of the skin be kept moistened or continually exposed to the action of the arsenical pigment for some time, it will become inflamed and covered with an eruption. If the exposure be continued sufficiently long, there will follow evidences of irritation of the stomach and intestines, as shown by more or less dyspepsia, loss of appetite, perhaps nausea and vomiting, and looseness of the bowels; still later various nervous symptoms appear, consisting chiefly of neuralgic pains in various portions of the body, and often more or less hysteria.

§ 153. The number of sources of this form of poisoning is very great, but the source of the greatest number of cases is the use of arsenical pigments in wall-paper; persons occupying rooms, the walls

[1] Wien. med. Jahrb., 1877, page 501.

of which are covered with such paper, being liable to symptoms very similar to those above described in the case of workmen; but often, especially in the case of women and children who are almost constantly exposed to the action of such paper, symptoms of poisoning are more apt to occur than in men, or in workmen engaged in the manufacture of such paper (unless the amount of arsenic is very large), both on account of the greater susceptibility of women and children to its influence, and on account of the greater constancy of the exposure, especially in chambers badly ventilated or used for sleeping apartments. Formerly only green papers were suspected to contain arsenic, it being in the form of one of the compounds of copper and arsenic, but of late years, probably owing largely to the popular suspicion in regard to bright green papers, the number of arsenical greens has diminished, and we find a very large number of arsenical reds, pinks, browns, and blues; in fact, nothing whatever in regard to the arsenical or non-arsenical nature of a wall-paper can be inferred from its color. The amount of arsenic which some wall-papers contain is very large, varying from a mere trace to thirty or forty grains to the square foot. In one case of red-colored wall-paper, the color contained 2.49 per cent. of arsenic. The number of authentic records of cases of chronic poisoning from this source, some of which have proved fatal,[1] is so great, that there can be no possible doubt about it.

In one case of illness traced to poisoning by arsenical wall-papers,[2] a careful analysis of excreta from the patient, and dust from the bed-room, which was decorated with arsenical paper, the following results were obtained:—

	White arsenic.	Scheele's green.
100 grains of dust from top of wardrobe contained	0.2	.36
100 " " " same room . . .	0.16	.30
Expectoration from patient	trace	trace
48 ounces of patient's urine	0.26	.5

The next most common sources of this form of poisoning are articles of dress, such as artificial flowers and cloth used for dresses; in artificial flowers, both the green leaves may be highly arsenical from the presence of Paris green, and the flowers from the presence of arsenical fuchsine, as in the cases mentioned above. A lady was prostrated with severe symptoms of arsenical poisoning by wearing

[1] See Med. Times and Gazette, May 3, 1862, and Appendix.

[2] Brit. Med. Journ., June 21, 1873.

a dress made from a fabric which contained about one-half a grain of arsenic to the square foot (0.291 grm. per square meter); this was a specimen of cloth known in the shops as "Foulard cambric," of dark and light-blue stripes; a specimen of garnet woollen brocade, which was intended for use as a bed-hanging for an infant's bed, was found highly arsenical. The most numerous cases of articles of dress being the source of chronic arsenical poisoning recorded are due to green tarlatan, which has produced severe symptoms in a large number of cases. The way in which arsenic may get into cloth, may be seen by a glance at the following recipes.[1] Mordant for madder purple:—

"*For light purple*—

1 gallon of acetate of iron at $1\frac{1}{2}°$ T.
3 gallons of purple assistant liquor.
3 gallons of water.
18 lbs. roasted farina.

This solution is ready for use after straining.

"*For dark purple*—

7 gallons acetate of iron at $2\frac{1}{2}°$ T.
$\frac{3}{4}$ gallon purple assistant liquor.
3 lbs. flour.

"*The purple assistant liquor* is made as follows:—

1*st.* 100 gallons of wood acid.
9 lbs. yellow prussiate of potash are dissolved in the wood acid.
2 gallons of sulphate of lead precipitate; made by dissolving acetate of lead in water, precipitating with sulphuric acid, and allowing the sulphate of lead to subside.

2*d.* 50 lbs. carbonate of soda dissolved in 8 gallons of water, to which is added 50 lbs. arsenious acid; when this is dissolved it is added to the first mixture, and thoroughly incorporated by stirring.

To the above complete mixture, add three gallons of hydrochloric acid and 200 lbs. of common salt. Mix well and store for use."

[1] Dyeing and Calico Printing, by the late Dr. F. Crace-Calvert, F.R.S., F.C.S., 1876, p. 73.

"Printing with madder extract[1]—

"*For dark purple*—

Boil together 1 litre of extract (Pernod's) in paste.
1 litre of acetic acid at 8° Baumé.
224 grams of olive oil.

The acid which is evaporated is replaced, and the mixture is thickened with 600 grams of powdered gum senegal. When required for use there is added—

224 grams of black mordant at 10° Baumé.
128 grams of arsenite of soda at 6° Baumé."

The following is an extract from the formula for making magenta:[2] "Into this apparatus, which is capable of holding about 500 gallons, a charge consisting of 2740 lbs. of a concentrated solution of arsenic acid, containing 72 per cent. of the anhydrous acid, is introduced, together with 1600 lbs. of commercial aniline."

§ 154. There are many other sources of chronic arsenical poisoning, as, for instance, anything which is habitually handled or eaten, if colored with an arsenical coloring matter; the most important of these are children's playthings, which are not only handled, but are often sucked by the child; toy paints, especially light green, the coloring matter of which is often pure Paris green, confectionery, both green and red, of which there has been a fatal case recorded.[3] Artificially colored syrups, vinegar, jellies, and wines, the use of such pigments in the manufacture of wrapping paper and glazed papers, so often seen on paper boxes, and as wrapping paper for lozenges; these papers are also largely used by children in kindergarten schools, where they may do a great deal of harm. Of six specimens of these papers examined for the writer by Dr. C. Harrington, one of the reds (unglazed) was found to contain about one grain of white arsenic to the square foot (0.690 grm. to the square meter), another red (glazed) about one-sixth of a grain to the square foot (0.096 grm. per square meter), one of the greens (glazed) about seven and one-fourth grains to the square foot (4.341 grm. per square meter), another green (glazed) about three grains to the square foot (1.76 grm. per square meter), an unglazed green about five

[1] Ibid., p. 97.
[2] Ibid., p. 366.
[3] Journ. de Chimie Médicale, 1875, page 131.

and one-fourth grains to the square foot (3.162 grm. per square meter), and a second unglazed green about three and one-fourth grains to the square foot (2.626 grm. per square meter). A child was given a green card to play with, and soon afterwards was seized with the symptoms of arsenical poisoning. It was found that the glazing of the card, which contained lead, was colored with Scheele's green.[1]

§ 155. Arsenic in wall-paper does not act merely by virtue of the dust which becomes detached from the paper, and diffused throughout the air of the room, so that it comes in contact with the mucous membranes, but it also acts by becoming partially converted into the exceedingly poisonous gas, arseniuretted hydrogen, which is formed when any of these arsenical pigments remain in contact with moist organic matter. It has been proved by Hamburg[2] and Bartlett,[3] that the air of rooms papered with arsenical wall-paper, contains arsenic in this gaseous form, so that it is inhaled and readily absorbed by the lungs.

§ 156. *Chemical examination.*—Arsenious acid, in its chemical relations, must be considered 1st, as a solid; 2d, in solution; 3d, mixed with organic matter.

As a solid.—When a portion of the solid is thrown upon the surface of water in a vessel, only a small part sinks to the bottom, most of it floating upon the surface, although much heavier than water; this serves as a ready preliminary test to distinguish arsenic from other heavy white crystalline powders, such as corrosive sublimate, calomel, lead, and bismuth salts, etc. It is entirely volatilized by heat, a temperature of only about 218° C. being required; this is also a ready preliminary test which will enable the physician, even at the bedside, to distinguish it from other mineral poisons, except corrosive sublimate and calomel, since it is only necessary to heat a little upon a knife blade over a common lamp or gas flame. This test is better performed by heating a little of the powder in a glass tube closed at one end; such as a small test-tube, when the powder will disappear from the heated end of the tube, but will be condensed upon the cool portion in the form of a white crystalline

[1] Bost. Med. and Surg. Journ., vol. xxxvii. p. 107.

[2] Journ. de Pharm. et de Chim., Aug. 1875, p. 143, and Zeitsch. für Biol., viii. p. 444.

[3] The Analyst, April, 1880, p. 81.

sublimate, the crystals having the octahedral form, as can be ascertained by examination with an ordinary pocket lens. Its solubility has been mentioned above (see § 129).

Thrown upon ignited charcoal, it gives off an aliaceous odor, which is due to the reduction of arsenious acid, which has in itself no odor when heated; the smell of garlic is only perceived when it is deoxidized. This odor, although striking, is not a positive proof of the presence of arsenic, as a similar one may be given off by several other substances. If, however, we heat arsenious acid with dry acetate of potassium, oxide of kakodyl is disengaged, by the peculiar insupportable smell of which compound even very minute traces of arsenious acid may be detected. This experiment may be conveniently performed by rubbing the substances together in a little mortar, and then heating them together in a test-tube, allowing the vapor, which is excessively poisonous, to be carried away by a current of air.

Heated in a narrow test-tube, or in the reduction-tube of Berzelius, with some freshly ignited (cold) charcoal, the same phenomena of deoxidation and evolution of odor occur as when it is placed on red-hot cinders in the open air; but in this case metallic arsenic is condensed by sublimation upon a cool portion of the tube, in the form of a metallic crust or ring, technically called an arsenical mirror, of an iron-gray color, brilliant and lustrous upon the outer surface, and crystalline upon the inner when seen under a low magnifying power. There are usually two crusts deposited, an upper and a lower, the latter of metallic arsenic, and the other of a browner color, which is a mixture of the metal and its oxide.

The arsenical nature of the ring may be further proved by volatilizing it by heat, in an open tube, by means of the flame of a spirit lamp; it is thus converted into arsenious acid, which sublimes upon the tube in the form of octahedral crystals, which may be dissolved in distilled water, and subjected to the liquid tests; or by dissolving them by means of nitric acid, and evaporating the solution to dryness, *arsenic* acid is formed, which is known by its giving a brownish-red precipitate, with nitrate of silver. When the quantity of arsenic is considerable, it is better to use a flux composed of the residue left by tartrate and acetate of sodium after incineration in a covered platinum crucible, black flux, as recommended by Dr. Taylor. The volatilization of the crust may be

accomplished either by applying heat directly to it, and chasing it up and down in the tube until it is all oxidized; or by carefully filing off that part of the tube in which it is contained, powdering it, and then introducing it into the end of another tube, which should in turn be subjected to heat; or, still better, by breaking off the sealed end, and, when heating the mirror, slightly inclining the tube so as to insure a current of air through it, when the white crystalline sublimate of arsenious oxide will be seen a short distance above the point at which the flame was applied.

The principal *precaution* to be taken in performing this test is to be sure that all of the substances used are perfectly dry. The metallic crust of arsenic may, moreover, be distinguished from the discoloration produced by *charcoal*, by the absence, in the dark stain of the latter, of any metallic appearance; the inner surface of the charcoal discoloration being powdery, black, and dull. This test is called the *reduction test* for arsenic, and is especially valuable, because it can be applied to any of the solid compounds of arsenic, whether soluble or insoluble, and also because, when properly and delicately performed, a satisfactory mirror can be obtained with an amount of arsenical compound corresponding to only one one-thousandth part of a grain of white arsenic,[1] and also especially because this test does not apply to compounds of antimony, which are so liable to interfere with many of the tests for arsenic.

The sublimate obtained by the reduction of the *compounds of mercury*, as calomel or corrosive sublimate, has indeed a metallic appearance, but may be distinguished, without using the liquid tests, by an inspection with a common lens, or even with the eye; the minute globules of metallic mercury can thus be readily seen, and by the point of a knife or a glass rod be made to run together.

The objections that have sometimes been made to the reduction process, on the grounds that *glass* contains arsenic or lead, are theoretical only. Glass does not contain arsenic, for although used in its manufacture, it is entirely volatilized by the heat required in the process, and when the glass does really contain lead (which ought not to be the case in suitable chemical implements), the mere loss of transparency caused thereby upon the application

[1] Wormley, Microchemistry of Poisons, 1869, page 257.

of heat, is evidently in the substance of the glass itself, and cannot, with the slightest attention, be mistaken for the arsenical crust. Moreover, the discoloration caused by the reduction of lead will be in the part of the tube to which the flame is applied. A crust weighing only a three-hundredth of a grain, a tenth of an inch broad, and four times as long, may show characteristically all the physical characters of an arsenical sublimate a hundred times larger. It may, therefore, be safely laid down that the appearances exhibited by a well-formed arsenical crust contained in the minute quantity of the three-hundredth of a grain are imitated by no substance in nature which can be sublimed by the process for the reduction of arsenic.[1]

If either white arsenic or metallic arsenic, as obtained by the reduction test, be treated with concentrated nitric acid, and the mixture evaporated to dryness, the residue consists of arsenic acid, which can be dissolved in water and the appropriate tests applied (see below). The same result can be obtained by treating the white arsenic or metallic arsenic with concentrated hydrochloric acid, adding a few crystals of potassium chlorate and evaporating to dryness. The sulphide of arsenic can also be oxidized by these last reagents to the form of arsenic acid.

§ 157. *In solution.*—The liquid tests for the detection of arsenic in solution are several in number. They are applied to clear solutions of arsenious acid free from organic matter, and are extremely useful in corroboration of the tests by which it is obtained in a metallic state ; the arsenical deposit having been first converted into arsenious acid by sublimation in an open tube, and then dissolved in water.

§ 158. *Hydrosulphuric acid.*—In the presence of free acid (hydrochloric), hydrosulphuric acid (sulphuretted hydrogen) throws down the tersulphide of arsenic, which is of a *lemon-yellow* color. This precipitate is soluble in ammonia, sulphide of ammonium and the fixed alkaline hydrates, by which means it can be separated from the sulphides of mercury, lead, copper, bismuth, and cadmium, if it happens to be mixed with these compounds. It can be converted into arsenic acid by hot nitric acid or hydrochloric acid and

[1] Christison.

potassium chlorate, and the tests for arsenic acid obtained with the resulting fluid. Dried, and heated with black flux in a reduction tube, a metallic sublimate of arsenic may be obtained (reduction test).

§ 159. *Ammonio-nitrate of silver.*—This reagent should be carefully prepared. (To a strong solution of nitrate of silver add a dilute solution of ammonia, until the brown hydrate of silver, which is thrown down, is nearly but not entirely redissolved. When properly prepared, there should be no free ammonia given off.) The *arsenite* of silver, which is precipitated by this reagent, is of a *lemon-yellow color*, which is soluble both in ammonia and nitric acid. It must be noticed that phosphate of sodium also produces, with nitrate of silver, a similar precipitate, which is equally soluble in nitric acid and ammonia.[1]

It frequently happens that common salt or hydrochloric acid is present in the solution together with the arsenic; the chlorine must first be removed before we can see the lemon-yellow color of the arsenite of silver; this is done by adding a drop or two of nitric acid to the solution, and then an excess of nitrate of silver solution and shaking; the chlorine will thus be entirely precipitated in the form of chloride of silver, while the arsenite of silver will remain dissolved by the nitric acid, and can be separated from the chloride by filtering; upon exactly neutralizing the filtrate with ammonia the yellow arsenite of silver will be precipitated.

§ 160. *Ammonio-sulphate of copper.*—(This reagent is prepared in the same manner as the foregoing. No more than is actually necessary for precipitation should be used, as its intense blue color is very apt to mask the proper color of the precipitate. If, however, this has occurred, filtration will separate the *green arsenite of copper* from the uncombined portion of the liquid.) The color of the precipitate is a *chrome green*. This precipitate is the familiar Paris green. It is readily soluble in an excess of ammonia to form an azure blue solution, and in free acids. When dried, and heated in a test-tube, arsenious acid is disengaged, and sublimes on the sides of the tube in the characteristic crystals, leaving a residue of the oxide of copper. Their nature may further be proved by dissolving them in distilled water, and submitting them to any other of the tests which may be desired. In relation to

[1] Bowman and Bloxam. London, 1871.

this test it must also be borne in mind that a similar precipitate is produced when the solution of copper is added to liquids containing some vegetable substances, though no arsenic may be present.[1]

§ 161. The *fallacies* to which these tests are exposed are the following: Phosphoric acid gives a yellow precipitate with ammonio-nitrate of silver, exactly like arsenious acid; several organic acids cause a green precipitate with the copper test, and the soluble salts of cadmium yield, with sulphuretted hydrogen or hydrosulphuric acid, a yellow precipitate, similar in appearance to the sulphide of arsenic. But doubts arising from these sources as to the true character of the precipitate may be corrected by a comparison of the tests and the production of a metallic or crystalline sublimate. Thus phosphoric acid gives only a pale-blue precipitate with the copper test, and is not affected by sulphuretted hydrogen; and the *pseudo*-arsenical precipitate obtained by cadmium with sulphuretted hydrogen, or by organic acids with the copper test, when dried and heated in a reduction-tube, gives neither the metallic deposit, such as is obtained from the sulphide of arsenic, nor the crystalline sublimate as obtained from the arsenite of copper. "No chemist in the present day would think of employing these liquid tests in solutions in which arsenic was mixed with *organic matter*. Almost all liquids used as articles of food are precipitated or colored by one or both of them, somewhat like a solution of arsenic, although none of this poison is present. An exclusive reliance upon them, as *color-tests*, has led to the rejection of chemical evidence on several trials."[2]

§ 162. If a solution of arsenic be rendered strongly alkaline with potassium or sodium hydrates, a few drops of a solution of sulphate of copper added, and the mixture boiled, a yellow precipitate of the suboxide of copper will be formed, owing to the removal of one-half of the oxygen from the oxide of copper by the boiling alkaline solution of arsenious acid, which is itself converted into arsenic acid. This reaction is also caused by other reducing agents, especially grape-sugar.

§ 163. *Marsh's test.*—This process for obtaining arsenic from simple or compound mixtures, by which it may be afterwards tried by any or all of the tests above mentioned, is exceedingly

[1] Ibid., p. 82.

[2] Taylor on Poisons, 3d Am. ed., 1875, 309.

delicate. According to Dr. Christison, a solution containing only the millionth part of white arsenic will part with it readily in the form of arseniuretted hydrogen, and the slighest trace of that gas in the hydrogen is indicated by this method. The process consists, essentially, in the disengagement of hydrogen gas by the action of sulphuric acid on zinc, and, in the presence of arsenious or arsenic acids, the consequent evolution of arseniuretted hydrogen, and the deposition of metallic arsenic upon a porcelain plate held in the flame, resulting from the combustion of the gas, or within the glass tube through which the gas is passing, the gas being decomposed by a flame applied to the tube.

The various modifications of the simple apparatus of Marsh require no particular description here. The simplest form consists of a wide-mouthed bottle with a closely-fitting cork perforated for two tubes, of which, the one furnished with a funnel dips beneath the liquid, and the other, bent nearly at right angles but sloping slightly towards the bottle, descends but a short distance into the vessel. This tube should be connected with some apparatus for drying the gas, such as a large tube filled with pieces of fused chloride of calcium, or a bottle or flask containing a little concentrated sulphuric acid. The gas after being thus dried is made to pass through a hard glass tube which is drawn out to a fine point. In this apparatus, hydrogen is generated by pure zinc and dilute sulphuric acid, and the action is continued until the atmospheric air is completely expelled before lighting the escaping gas, and all risk of an explosion is thereby avoided. The freedom from arsenic of the materials employed, should be ascertained by applying a flame to the exit tube for one-half an hour; if, at the expiration of that time, no mirror of metallic arsenic has been deposited upon the inner surface of the tube, we may be sure that the materials employed are absolutely free from arsenic. In this country there is usually no difficulty in obtaining zinc which is perfectly free from arsenic. When thus satisfied that the materials are pure, a portion of the suspected liquid may be poured into the funnelled tube, and the spirit lamp be immediately applied to the horizontal tube, in order to obtain a metallic ring or incrustation, which, if arsenious acid is present, will be deposited at the distance of about half an inch from the part to which the flame is applied. Having procured this, the gas, as it issues from the fine end of the tube, should be

ignited, and deposits obtained on porcelain or glass. The two processes may be continued until a metallic deposit is no longer obtained.

These rings or mirrors, as they are termed, should be tested to ascertain their arsenical nature as follows: That portion of the tube containing one of them should be filed off and gently heated, while the tube is slightly inclined so as to insure the passage of a slow current of air through it; the dark ring will gradually disappear, and a short distance above, on the cool portion of the tube, will appear a white crystalline sublimate of arsenious oxide, which can be tested by the appropriate tests. Another portion of the tube containing a mirror should be attached to a sulphuretted hydrogen apparatus, and the mirror gently heated while a very slow current of sulphuretted hydrogen is passing through the tube; the dark ring will disappear, as when heated in a current of air, and there will appear at a short distance from the flame a lemon-yellow sublimate of sulphide of arsenic, which will be found to be insoluble in hydrochloric acid, but soluble in sulphide of ammonium. The mirror in still another portion of the tube should be touched with a solution of sodium hypochlorite, which will immediately dissolve it (distinction from antimony mirror formed in the same way). The mirror in another portion of the tube should be touched with a solution of sulphide of ammonium; it should remain undissolved, whereas the corresponding antimony mirror would be quickly dissolved. Of these substances the three tubes containing the arsenical mirror, the sublimate of arsenious oxide, and the sublimate of the yellow sulphide of arsenic should be taken to court in legal cases as *corpora delicti*. The mirrors obtained upon the porcelain plates by depressing them into the ignited flame at the end of the tube should also be tested as to their arsenical nature. One should be touched with a solution of sodium hypochlorite in which, if arsenic, it will quickly dissolve. Another will be found to be insoluble in a solution of sulphide of ammonium. Another should be dissolved in a drop of nitric acid, the solution gently evaporated to dryness, and the residue, which will consist of arsenic acid, touched with a drop of a solution of ammonio-nitrate of silver, when the brick-red arseniate of silver will be seen.

If a cold beaker-glass or other glass vessel be held over the flame of arseniuretted hydrogen burning at the end of the tube, moisture will condense upon the inner surface of the glass, which, after dry-

ing, will contain a white residue consisting of white arsenic, the arseniuretted hydrogen on burning being converted into water and white arsenic. Marsh's test cannot be successfully performed, as a rule, with solutions which contain organic matter; this should, therefore, be first removed by the methods mentioned below.

§ 164. The *fallacies* to which Marsh's process may give rise proceed from the contamination of the zinc or sulphuric acid with arsenic, the presence of antimony in the suspected liquid, or of imperfectly charred organic matter. The mode of guarding against the first has already been noticed. Antimoniuretted hydrogen burns with a pale, bluish-green flame, and deposits a black stain upon a porcelain plate held in it.

The antimonial is distinguished from the arsenical crust by the following characters:—

First, the dark stain is less bright and metallic than the arsenical one, and, when viewed by transmitted light, is smoky-black, whereas that of arsenic is hair-brown.

Secondly, if the flame be allowed to play on a solution of ammonio-nitrate of silver, placed on the under surface of a plate of mica, no yellow arsenite of silver is obtained.

Thirdly, the greater volatility of arsenic, and its conversion into octahedral crystals of arsenious acid, may serve, where the crust is in an open tube, to distinguish it from antimony. This may be effected by a bath of olive oil; this liquid does not begin to boil until the heat rises above 600° F. Arsenic is completely sublimed under 500° F., and the process begins at a much lower temperature; but antimony is not at all affected by the heat required to boil olive oil. Hence, whether the stains of the two metals are mixed or not, their true nature can thus be readily ascertained. Dr. Maclagan says that in his hands the process has proved "so simple and easy of execution, so delicate in the results obtained by it, so advantageous in excluding the necessity of any chemical reagent whatever," and "also in affording, when tubes of equal size are used, so easy a method of determining approximately the proportion of arsenic in different articles examined, that in operating on small quantities of material, or where little arsenic is present, I have of late always, in practice, adopted it in preference to any other."[1]

[1] Month. Journ., Jan. 1853.

Fourthly, the tests applied to the metallic deposits inside of the tube mentioned above.

Fifthly, the metallic crust obtained by submitting a current of the gas to heat, presents some distinguishing characters; the arsenical crust is always deposited in the more distant or anterior part of the tube, whereas the antimonial one is first deposited on the *heated* part of the tube.[1]

Sixthly, arsenical spots on porcelain may also be readily distinguished from those of antimony by the more rapid solution of the former in hypochlorite of sodium. They are rapidly dissolved by it, and the porcelain becomes perfectly clean. If they are shining and thick, the process is somewhat longer, but does not occupy more than a few seconds. Antimonial spots, on the contrary, completely resist the action of the hypochlorite of sodium, unless they are quite faint and of a dull appearance. Furthermore, if any fluid containing *both* arsenic and antimony be introduced into the apparatus, the spots on the porcelain at first contain principally only arsenic, apparently in consequence of the antimony being less volatile; but if shining spots be produced upon the porcelain, which contain more antimony, these resist, more or less, the action of the hypochlorite of sodium, and are often eaten away only around the edges. While, therefore, by this reagent, a slight trace of antimony cannot be distinguished in spots of arsenical nature, arsenic, on the other hand, can be detected by it in antimonial stains.[2] The crusts resulting from the presence of imperfectly *charred organic matter* in the suspected liquid are not so readily soluble in nitric acid as are the arsenical crusts, and do not, like the latter when so dissolved, yield a brownish-red precipitate with nitrate of silver.

Seventhly, the following differential test is recommended by Taylor[3] as very reliable: "Receive the deposit from the burning gas on the interior of a small white porcelain capsule. Add a few drops of strong nitric acid. The deposit will be immediately dissolved. Evaporate gently to dryness. Moisten the dry residue with one or two drops of water, and then add a few drops of a *strong* solution of nitrate of silver. If the stain was owing to arsenic wholly, or

[1] Pereira.

[2] Wackenroder, Chem. Gaz., Aug. 2, 1852. This test was in use by Bunsen in 1844.

[3] On Poisons, Am. ed. 1875, p. 313.

in part, a brick-red colored precipitate will immediately appear. This will be more or less distinct, according to the quantity of arsenic present. The red precipitate (if owing to arsenic) is entirely soluble in ammonia. A deposit of antimony thus treated leaves a white residue (oxide of antimony) insoluble in water. Nitrate of silver added to it produces no colored precipitate; but if a little ammonia be brought near, either in vapor or liquid, and a solution of potash be added, a precipitate is formed, which becomes black by standing. Sulphide of ammonium dissolves the antimonial deposit immediately, and on evaporation leaves an orange-reddish colored film of sulphide of antimony, soluble in hydrochloric acid, and insoluble in ammonia. The sulphide of ammonium does not readily dissolve the arsenical deposit, but when gently evaporated, it leaves a bright-yellow film (sulphide of arsenic) not soluble in hydrochloric acid, but soluble in ammonia. Imponderable quantities of the two metals may be thus easily identified. In testing these minute films of arsenic, hydrochloric acid must not be used with the nitric, since, on evaporation, a portion or the whole of the arsenic may be volatilized, and lost as chloride of arsenic."

§ 165. Zinc, sulphuric and hydrochloric acids are often contaminated by arsenic. The best answer to all objections based on the presence of arsenic from accidental sources is, that the materials employed in analysis were tested repeatedly before the suspected liquids were introduced into the apparatus.

If, in any case, we have no other evidence to offer than that furnished by Marsh's process—a case in which the poison must be in infinitesimal quantity, and the metallic deposit proportionately minute—it would be better to abandon the evidence altogether, than to maintain the presence of poison from the results which admit of no sort of corroboration; for all who have experimented upon the subject, must have perceived the utter inefficacy of applying liquid tests to determine the chemical properties of imponderable and scarcely visible sublimates.[1]

§ 166. *Reinch's test.*—This method of separating arsenic is exceedingly simple and efficacious. A solution supposed to contain arsenic should be acidulated with hydrochloric acid and heated to the boiling point. A thin leaf of copper, or fine copper gauze or

[1] Taylor, op. cit., p. 316.

wire, bright and clean, should then be introduced, and if arsenic exists in the liquid, it will be deposited as an iron-gray film of the metal upon the copper. The copper, being removed after the deposit is formed, must be washed in distilled water, dried, and introduced into a reduction-tube. On the slow application of heat, arsenious acid will be sublimed and deposited on the sides of the tube in the form of minute octahedral crystals. If the solution contained a very large amount of arsenic, so much metallic arsenic may be deposited upon the copper that, when heated in the reduction tube, the lower portion of the sublimate may consist of an arsenical mirror and the upper portion of the arsenious acid crystals. These crystals may be examined by a lens, and then dissolved in water and subjected to the liquid tests. These supplementary tests are requisite, since solutions of various metals give a coating not unlike that of metallic arsenic, and, if the copper is put into the acidulated fluid before it is duly heated, a stain will almost always occur in the presence of organic matters. In proof of the delicacy of this test, Prof. Rainey, of Glasgow, says that "in repeated experiments" he has "found that one one-thousandth of a grain of arsenious acid in one million times its weight of fluid, could be separated as a distinct deposit on copper.[1] The copper thus coated, when heated gently in a small tube, yielded a slight but distinct sublimate, most obvious on a black ground, and which, with a magnifying power of ten to twenty diameters, was found to consist of crystals with triangular facets, and which when dissolved in water yielded orpiment and the red arseniate of silver when treated with the appropriate reagents." Dr. James St. Clair Gray read a paper to the chemical section of the Glasgow Philosophical Society, "On certain fallacies in the means of detecting some Poisons," in which he pointed out the fact that Reinsch's test for arsenic is liable to fail when the arsenic has undergone oxidation to arsenic acid, or when it exists in the state of sulphide. He advises the reduction of the arsenic acid by means of sulphite of an alkali; and in the case of the sulphide he would boil with caustic potash and dialyze.[2]

[1] $\frac{1}{60,000}$ of a grain in the presence of 5,000,000 parts of liquid, yields, after several minutes, a very distinct stain, the outer part of which has a dark, metallic appearance, and the inner, a brownish color. (Wormley, Microchemistry of Poisons, p. 287.)

[2] British Med. Journal, March 16, 1872.

The principal fallacy connected with this test exists in the fact that antimony may give precisely the same result, although usually the sublimate produced by heating the copper, upon which is a coating of metallic antimony, consists of prismatic crystals instead of octahedral, but the antimonial sublimate is very rarely in the form of octahedral crystals, so that this test is of little or no value in court as a means of distinguishing between arsenic and antimony. Reinsch's test is one of great value as a *preliminary* test in legal cases, because the presence of organic matter does not interfere with it.

§ 167. *Bloxam's test.*—Professor Bloxam has applied the process of electrolysis to the detection of arsenic. The apparatus proposed by him consists of a two- or three-ounce bottle, the bottom of which has been cut off, and replaced by a piece of vegetable parchment, bound on by platinum wire. To the mouth of the bottle is fitted a cork with a bent tube and a piece of platinum wire, which passes through the cork, and turns up beneath in the form of a hook. A slip of platinum then hooks into the end of the wire, and passes nearly to the bottom of the bottle; it forms the negative pole of the arrangement. The bottle stands in an ordinary test-glass, and the positive pole, also of platinum, stands in the glass. Dilute sulphuric acid is put into the bottle, and also in the glass, so as to stand at the same height in both vessels. The substance to be tested is introduced into the bottle, the cork adjusted, and the wires connected by five cells of Grove's battery; the heat of a spirit lamp is applied to the bent tube, and in the course of a quarter of an hour a distinct mirror is obtained, if arsenic is present. Standard solutions containing respectively a tenth, a hundredth, and a thousandth of a grain of arsenious acid, were prepared and examined by this process, and in every case a successful result was obtained.

The solutions were mixed with organic substances, such as the ordinary articles of food—meat, eggs, milk, etc.—and the resulting matter examined. This was then dissolved by means of chlorate of potassium and hydrochloric acid, and the resulting fluid evaporated down, by means of a water-bath, to a thick syrupy fluid. The arsenic was thus obtained in a state of arsenic acid, which does not give a certain result by the electrolytic process. Some sulphurous acid was therefore added, and the mixture introduced into the bottle, after expelling the excess of sulphurous acid by evaporation; a drachm of alcohol was then poured over the surface, and the process

put into operation. . . . . In all these experiments, of which a great number were made, the thousandth of a grain of arsenious acid was readily detected.

The other metals which may be detected by this process are mercury, antimony, copper, and bismuth; lead is precluded by the sulphuric acid which is present. These are all precipitated in the metallic form upon a slip of platinum, and even in the case of antimony a mere trace of antimoniuretted hydrogen is formed, the metal being deposited on the negative pole.[1]

§ 168. *Mixed with organic matter.*—It should be remembered that the liquid tests, with the exception of Reinsch's and Bloxam's tests, are not applicable directly to liquids containing *organic matter*. The same colors which have been mentioned as indicating with probability the presence of arsenic may be obtained in liquids used as articles of food, containing common salt or various colorless organic acids. Hence, unless the precipitate obtained can be made to yield arsenic by the other tests, there can be in an organic liquid no demonstration of its presence, except by the previous destruction of the organic matter and isolation of the arsenic by means of the methods mentioned below, or by Reinsch's or Bloxam's tests.

We have been favored, by Dr. Jackson, of Northumberland, with a reference to a case[2] in which chemical evidence of this kind would have procured the indictment of a faithful and exemplary wife for the murder of her husband by poisoning with arsenic, had it not been for his interposition; he showed not only that the man did not die with the symptoms of arsenical poisoning, but that the chemical investigation was both imperfect and fallacious.

The following is an extract from the minutes of one of the examiners: "The contents of the stomach—about sixteen ounces, and principally fluid—were thoroughly mixed by agitation and stirring, and successive portions submitted to the following tests: A small portion was put into a clean Florence flask, to which about four ounces of *common* water and a few grains of subcarbonate of potash were added; this was submitted to the heat of a spirit-lamp until boiling commenced. Portions of it were poured into two clean wine-glasses, to one of which a small quantity of sulphas cupri was

[1] Brit. and For. Med.-Chir. Rev., April, 1860, p. 527.

[2] Am. Journ. Med. Sci., Nov. 1829, p. 243.

added; this had the effect of changing the fluid, which had been of a light-hazel (owing to the color of the contents of the stomach), to a light-green color, resembling that of Scheele. To the surface of the other glass a stick of lunar caustic was applied; the effect was an immediate *white cloudy* appearance, which soon changed into a reddish-yellow or orange color, and, after standing a few hours, resolved itself into a reddish-brown. . . . The next day, the remaining contents of the stomach having been dried, half an ounce of the suspected matter was boiled with snow-water in a flask until it rose to the top of the vessel; the fluid was suffered to cool, when a stream of sulphuretted hydrogen gas was passed through it; this immediately changed the solution to a beautiful light golden-colored liquid; after which a solution of arsenious acid was submitted to the same, and the result was precisely similar," etc. Such rough and imperfect processes as these authorized, in the opinion of the examiners, the statement that the chemical analysis "clearly indicated the presence of arsenic." They were equally unfortunate in their deductions from the state of the stomach, which, from the description, appears to have presented that appearance not unusual in an habitually intemperate person, as was the subject of the examination, but which they looked upon also as "clearly indicating that the patient had died in consequence of poison from arsenic."

§ 169. *Isolation of arsenic from organic mixtures.*—Before the contents of the stomach, the liver, spleen, or the other organs containing arsenic by means of absorption during life, can be submitted to any of the tests for arsenic, it is necessary to obtain a solution as free as possible from organic matter. Various means have been recommended for this purpose, those which are the least open to objection are the following:—

If it be intended to separate metallic arsenic by means of Reinsch's process, all the soft solids should be cut into small fragments, distilled water, if necessary, added, and also hydrochloric acid in slight excess. This mixture should be boiled gently for an hour until all soft solids are either dissolved or broken down into fine flakes or grains. Filter through wet muslin, heat the filtered liquid again to the boiling point, and then introduce a slip of copper as before described.

§ 170. If, however, the apparatus of Marsh is to be used, the following process is recommended by MM. Danger and Flandin. Add to the organic matter contained in a porcelain capsule one-sixth of its weight of sulphuric acid, and heat until vapors of sulphurous acid appear. The matter is first dissolved, but during the concentration it is charred. The liquor is to be constantly stirred with a glass rod. The carbonization is effected without any swelling or frothing, and is to be continued until the charcoal is friable and almost dry. A small quantity of nitric or of nitro-muriatic acid is to be added by means of a pipette when the capsule is cold. This converts the arsenious acid into the more soluble arsenic acid. The mixture is then to be evaporated to dryness, treated with boiling water, and the limpid liquor introduced into Marsh's apparatus, in which it never froths.

§ 171. The best process, and the one most generally used in this country not only for arsenic but also for the other metallic poisons, is what is known as Flandin and Danger's process. It consists in finely dividing with scissors the solid parts, mixing them with portions of the liquids, adding a large excess of hydrochloric acid in an evaporating dish and about a teaspoonful of crystals of chlorate of potassium; this mixture should then be heated over a water-bath, when the solid portions will gradually become destroyed and the fluid will assume a light yellow or green color; distilled water should be added to the mixture from time to time as the fluid evaporates, and fresh crystals of chlorate of potassium, whenever the fluid becomes dark brown or black instead of remaining yellow. This should be repeated from time to time until the fluid remains light-colored after being heated at least one-half an hour after the last addition of potassium chlorate. By this means the arsenic, if present, is converted to the form of arsenic acid. The mixture may then be allowed to cool, so that the fatty matters will separate, and filtered, the solids remaining upon the filter paper being thoroughly washed. The filtrate and wash-water united must be again heated over the water-bath, until there is no more free chloride remaining in the fluid. It is then necessary to reconvert the arsenic acid into arsenious acid, which is done by adding a saturated solution of sulphurous acid or of acid sulphite of sodium to the fluid and continuing the heating over the water-bath, until the odor of sulphurous acid gas has disappeared. Filter the fluid, if necessary, into a

beaker or tall jar, and pass through it a slow current of sulphuretted hydrogen gas for twenty-four hours; this will precipitate whatever arsenic is present in the form of the yellow sulphide of arsenic, but it also precipitates some of the organic substances in the form of unknown organic sulphur compounds, which, unless arsenic is present in very large amounts, disguise the yellow color of the sulphide of arsenic; as a rule, the precipitate thus obtained has a dirty brown color. This precipitate should be separated from the fluid by filtration, and washed; if it contains sulphide of arsenic the only impurity consists of a certain amount of organic sulphides, and must, of course, be further purified; this is done by treating the precipitate, while on the filter paper, with a boiling mixture of ammonia and ammonium sulphide, which will dissolve the sulphide of arsenic together with the organic sulphides; it will also dissolve any sulphide of antimony or sulphide of tin which may be present, but it will leave upon the filter paper any sulphide of lead, mercury, bismuth, copper, or cadmium if present. The fluid passing through the filter should be collected in an evaporating dish and evaporated to dryness; usually the residue will have a dark-brown or blackish appearance; the organic sulphides can now be destroyed by moistening with concentrated nitric acid and evaporating, repeating this process three or four times, by which process also the sulphide of arsenic is converted into arsenic acid, or by moistening with hydrochloric acid, adding one or two crystals of potassium chlorate and evaporating to dryness. The arsenic is now left in the residue in the form of arsenic acid, which can be dissolved in hot distilled water and a portion of the solution subjected to all of the tests for arsenic acid (see below); another portion can be transformed to arsenious acid by acidulating with hydrochloric acid and boiling with a solution of the acid sulphite of sodium until the odor of sulphurous acid has disappeared; this solution can be used for all of the tests for arsenious acid (see above). All of the chemicals and pieces of apparatus used should have been tested and found to be absolutely free from arsenic by having been employed in a previous or parallel analysis of tissues which contained no arsenic.

§ 172. It was at one time supposed, upon the authority of Orfila, that arsenic was *a natural constituent of the human body;* but his own admission of his error, and repeated subsequent trials, have

proved that this is not the case,[1] that it exists neither in the bones nor in the soft parts. It has been found in the soil of cemeteries, but in an insoluble form, being separable only by concentrated sulphuric acid; the objection, therefore, that, if detected in the body, it may have been derived from this source, is not applicable, unless the coffin has been broken, and the soil become mingled with those portions of the body subjected to analysis. In this case a portion of the soil taken from the adjacent earth may be examined, for the purpose of ascertaining whether the arsenic is really derived from it. Where the coffin has been entirely disintegrated, and the earth is thus indistinguishable from the human remains contained in it, a chemical analysis can hardly be demanded. But when, on the contrary, the body is in a tolerable state of preservation, and the earth has gained access to it only through crevices in the coffin, without coming in contact with the stomach and other viscera, it is evidently an unwarranted and fanciful idea to attribute the origin of arsenic found therein to the minute trace which may possibly exist in the surrounding soil.[2]

Nevertheless, in the following case a competent chemist thought that there was good reason for supposing that arsenic must have been administered. A verdict of wilful murder having been returned by a coroner's jury against a woman named Rebecca Smith, for causing the death of her infant child by poison, the bodies of two of her other children, who had also died in infancy, were disinterred and sent to Mr. Herapath, of Bristol, for examination. The soft parts of the bodies were entirely gone, and the bones were all separated from each other. The coffins were decomposed and penetrated in all directions by the roots of a tree. The roots of trees as large as the little finger had passed through the head and skeleton, and had followed the bones in all directions. He found arsenic in the bones, in the black mould under the head, and a greater quantity in the black mould under the ribs. One of the bodies had been interred five and the other eight years. In his testimony before

[1] *Vide* Lehmann's Physiol. Chemistry, vol. i. p. 449. Translated by George R. Day.

[2] Walchner has discovered arsenic in many ferruginous earths, and in the deposits of certain mineral springs. Will and Scherer and others have made the same observations. Becker examined the soil of a churchyard, and found that every part of it contained arsenic. (Canstatt's Jahresbericht, 1846, 1847. Bd. v.)

the coroner, Mr. Herapath said: "I have never found arsenic in a body which was in its natural state." . . . "I have made experiments on hundreds of bodies of human beings and brutes, but have never discovered arsenic unless it had been administered medicinally or for a criminal purpose. I have also made experiments upon soils, and I believe the statement of Orfila to be a mistaken one. My opinion is, that arsenic was administered to both these children during life, and that it was the cause of death; it existed in too great quantity to have been administered for a medicinal purpose." The jury without hesitation returned a verdict, "That the deceased children died from the effects of arsenic, but how or by whom administered there is no evidence to show."[1] In the absence, however, of any statement of the process by which the arsenic was obtained from the bones and the mould, this case cannot be considered of great importance.

§ 173. The experiments of Orfila, in 1839, have since been confirmed by numerous observations, and the fact is well established that arsenic in the combination in which it exists in the soil is completely *insoluble*, and consequently cannot be carried by the percolation of rain into the organs of the body. Boiling water does not dissolve the slightest trace of it, and it is only by the prolonged action of boiling sulphuric acid that it can be separated from the earth containing it. In illustration of these remarks we append the following: In 1844, Nicolas Noble and a woman named Jerome, both of whom died with the symptoms of poisoning, were buried in the cemetery of Epinal, the earth of which contains arsenic. Their graves were two yards apart. The bodies were ordered to be disinterred; in the woman there was not found the slightest trace of arsenic, but it was discovered in the stomach and the intestines of Nicolas. Six months later the bodies were again exhumed; the *result was the same*, although the body of the woman had been, after the first examination, immediately replaced in the grave without any coffin, and covered with the soil which had been thoroughly soaked by an abundant rain. There were reunited here all the conditions of putrefaction and moisture supposed by some to be most favorable for the formation of an arsenite or arseniate of ammonia and the imbibition of the body by it; nevertheless the soil treated

[1] Lancet, 1849, p. 253, Am. ed.

with boiling water did not give up a trace of any arsenical salt, and no arsenic was found in the woman's body. In that of the man, on the contrary, it was found in the liver, after it had been carefully washed. The correctness of the inferences from the chemical analysis was soon fully established by the confession of the criminal.

Another case, occurring in the year 1851, in France, is not less remarkable. On that occasion, M. Barse, a distinguished chemist, gave the following opinion when called upon by the government. "Arsenic exists in the soil only in an insoluble state; hence it cannot be communicated by means of infiltration to the bodies contained in such soils; and, therefore, if arsenic is found in such bodies, it must have been derived from other sources." He examined other bodies contained in the same cemetery, and found that in none of them was there a trace of arsenic, although it existed in the soil.[1]

III. *Arsenic Acid.*

§ 174. *Arsenic acid* is seldom met with out of the chemical laboratory; it has been proved by experiments upon animals to be poisonous, and although it has given rise to no fatal cases of poisoning in man, yet its chemical study is of very great importance in legal cases, since in the separation of arsenic from organic tissues the white arsenic is always converted into arsenic acid, and some of the compounds of arsenic acid, which are formed by the tests used, should always be taken to court in legal cases as *corpora delicti*. The principal tests are the following:—

(*a*) *Ammonio-nitrate of silver.*—This test is performed in precisely the same manner as for arsenious acid. The resulting arseniate of silver has a reddish-brown color, and is soluble both in an excess of ammonia and in free acids.

(*b*) *Ammonio-sulphate of copper.*—This test is also performed in the same manner as for arsenious acid, the resulting precipitate, arseniate of copper, having a bluish-green color instead of the light green, and is soluble in an excess of ammonia and free acids.

(*c*) *Sulphate of magnesium.*—If to a solution of arsenic acid be added a solution of chloride of ammonium, a little ammonia, and a solution of sulphate of magnesium, and the mixture be shaken violently or stirred with a glass rod, a white crystalline precipitate of ammonio-magnesian arseniate will be formed. This precipitate occurs in the

[1] *Vide* Briand, Méd. Lég., p. 520.

form of prismatic crystals if slowly formed, but in the form of fern-leaf crystals if rapidly formed; these crystals are exactly like those of the ammonio-magnesian phosphate, but can readily be distinguished from the latter by the fact that they yield metallic arsenic when treated by the reduction test, Reinsch's test, or Marsh's test, and also yield a yellow precipitate when dissolved in hydrochloric acid and treated with sulphuretted hydrogen. This precipitate is also soluble in acetic acid.

(*d*) *Nitrate of uranium.*—A solution of arsenic acid is precipitated by a solution of nitrate or acetate of uranium in the form of a pale-yellow flocculent or granular precipitate, which is readily soluble in free mineral acids, therefore it is always best to add a little acetate of sodium before adding the uranium solution in order to neutralize any free mineral acid which may be present. A similar precipitate is formed by phosphoric acid, but can be distinguished in the same way as the ammonio-magnesian compounds, and, moreover, in the regular process of analysis in legal cases, the absence of phosphoric acid would be insured by the previous precipitation by sulphuretted hydrogen and thorough washing of the precipitated sulphide. This test is especially valuable, because it can be used as a means for estimating the amount of arsenic quantitatively, as it is used for estimating the amount of phosphoric acid quantitatively.

§ 175. Arsenic acid is also, like arsenious acid, precipitated by sulphuretted hydrogen, although more slowly, because the sulphuretted hydrogen must first convert the arsenic acid to the form of arsenious acid, which conversion is attended with the separation of sulphur, so that the precipitate formed consists of a mixture of sulphide of arsenic and sulphur. Arsenic acid also responds to Marsh's, Reinsch's, and the reduction tests like arsenious acid.

IV. *Arseniate of Potassium.*

§ 176. Dr. Christison is the only author who refers to cases of poisoning by this substance. He quotes two cases of accidental poisoning by it.

V. *Arseniate of Sodium.*

§ 177. The only instances reported of poisoning by this preparation are quite recent. Two young men sent to a chemist for

doses of tartrate of sodium, in place of which the arseniate of sodium was sent by mistake and taken. In about five minutes they were attacked with violent cramps in the stomach, which speedily became very intense. One died in consequence, and the other remained in a dangerous state.[1]

VI. *Sulphides of Arsenic.*

§ 178. There are several of these compounds known in commerce as *realgar* of an orange-red color, orpiment which is yellow, and (it is said) another preparation, bearing the same name, which is a compound of pure sulphide and arsenious acid. The pigment known as King's yellow contains a sulphide of arsenic and a considerable proportion of lime and sulphides. Cases of intentional and accidental poisoning with orpiment are known. A female was poisoned with it in England in 1835, and the poison found in considerable quantity in the stomach of the deceased fourteen months after death. Its character was satisfactorily proved by chemical analysis, and led to the apprehension and conviction of the murderess, who was afterwards executed.[2] Another case, in which it was mixed in porridge, by mistake for turmeric, is related by Dr. Jochner. An old man and his nephew both partook of the food without immediately discovering the mistake. The prominent symptoms were continual vomiting, burning pain in the stomach, and gradual collapse. The old man died in twenty-two hours; the boy escaped. Evidence of violent inflammation was found in the œsophagus and stomach, the mucous coat of the latter being soft and thickened. There was a sphacelated spot, one inch in diameter, in the œsophagus, and another in the stomach of three inches in extent.[3]

§ 179. The sulphides of arsenic when pure are not poisonous, but when exposed to a moist atmosphere they become partially oxidized, and thereby mixed with arsenious acid, and all of the cases of poisoning reported are in reality cases of poisoning by arsenious acid.

These sulphides when pure are insoluble in water or hydrochloric acid, but are soluble in hot concentrated nitric acid, being converted

[1] Am. Journ. Med. Sci., Oct. 1852, p. 553, from the Journ. de Méd. and Chirurgie, June, 1852.

[2] See the case quoted in Beck, vol. ii. p. 560.

[3] Henke's Zeitsch., Erg. H. 43, 162.

into arsenic acid, and are also oxidized into arsenic acid by hydrochloric acid and potassium chlorate. They yield metallic arsenic when subjected to the reduction test. They are soluble in the alkalies. When impure, that is, when they contain, as they usually do, more or less arsenious acid, they of course respond to all of the tests for arsenious acid.

VII. *Arseniuretted Hydrogen.*

§ 180. This gas is colorless, has the smell of garlic, and is exceedingly poisonous. Several cases are related in which chemists, in experimenting with it, have perished in consequence of accidentally inhaling it; and recently cases have been recorded caused by inhaling the gas which was evolved in certain processes of manufacture or mining (see Appendix). The symptoms are sometimes delayed one or two days after the beginning of exposure, and consist chiefly of giddiness, nausea and perhaps vomiting, pain in the limbs, jaundice, deep sleep, bloody urine, and bloody stools; later, high pulse and temperature, intense headache, delirium, stupor, and death in from one to eight or ten days. The bloody urine is of the form called hæmoglobinuria, in which only the blood-coloring matter gets into the urine, the blood-globules being broken up by the arseniuretted hydrogen, while the blood is circulating through the body; this is the cause also of the jaundice and bloody stools. In the case related by Dr. O'Reilly, death ensued on the sixth day, and Gehlen, the German chemist, died in nine days.[1] The gas inhaled was, in the first of these cases, supposed to be pure hydrogen, but was contaminated with arsenic, owing to the impurity of the sulphuric acid used in generating it. The mode of obtaining it and of testing its properties has been already described.

VIII. *Arsenite of Potassium.*

§ 181. These and other compounds of arsenious acid with alkaline bases are poisonous. The solution of arsenite of potassium is of much use in medicine, especially in the treatment of chronic skin diseases and intermittent fever, under the name of Fowler's solution or liquor potassii arsenitis (U. S. P.). It receives its taste and color from the spirit of lavender, as officinally prepared, and con-

[1] Dublin Journ., vol. xx. p. 422; Buchner's Toxicologie, p. 476.

tains four grains of arsenious acid to the fluidounce, the *usual* dose of which is, for an adult, five to fifteen minims three times a day. From the occasional adulteration of arsenic with the sulphate of lime, this preparation is no doubt sometimes weaker than the officinal strength. Two cases of fatal poisoning by it have been before referred to. In one of them the dose did not amount to more than two grains in five days. In the other the amount swallowed was not ascertained. The symptoms are the same as those produced by arsenious acid. The dose, however, which will produce poisonous effects is very uncertain. This liquid may be tested, after acidulation with hydrochloric acid, by means of any of the tests for arsenious acid in solution.

There are a number of solutions of arsenious acid in alkalies used for various purposes; thus, solutions of arsenious acid in soft soap are sometimes used for washing sheep, and have given rise to cases of poisoning. Taylor records cases of poisoning due to arsenite of sodium, which was used as a cleansing preparation; there is a soap (called *savon de Bécœur*) employed by taxidermists for the preparation of skins, which contains thirty-two per cent. of arsenious acid.

IX. *Arsenite of Copper.* (Scheele's Green.)

§ 182. As this article owes its poisonous properties rather to the arsenic contained in it than to the oxide of copper, we have ranged it with the arsenical compounds. It has frequently occasioned accidents in England and on the continent from its use as a coloring ingredient in confectionery. In 1850, Dr. Letheby reported three cases of poisoning by Sheele's green, which came under his notice; and he states that between thirty and forty children were poisoned at the same time by sweetmeats sold to them by a confectioner in Petticoat Lane. He stated, moreover, that as many as seventy cases of poisoning had been traced to a similar source within three years.[1]

Within the past few years in this country, there have occurred a very large number of cases of acute poisoning, mostly suicidal, from this compound, which can always be bought by the pound at almost every country store for the purpose of killing potato bugs; it is sold under the name of Paris green. The writer has seen four or

[1] Brit. and For. Med.-Chir. Rev., July, 1851.

five cases within the last two years. The *symptoms* caused by large doses of Paris green are exactly the same as those caused by arsenious acid, with the exception of the coppery taste and the green color of the vomitus and stools. The symptoms caused by this substance when slowly absorbed, as in cases where it is used for coloring wall paper, have been already mentioned.

§ 183. The arsenite of copper when pure can be very easily recognized, since it responds to the tests for both arsenic and copper. Its bright green color will usually lead at once to a suspicion as to its nature. A small portion of the powder treated with a drop of ammonia upon a watch-glass immediately imparts to the latter an azure-blue color, and if a drop of a solution of nitrate of silver be allowed to flow down the side of the watch-glass, so as to form a separate layer of fluid under the ammoniacal solution, a lemon-yellow precipitate of arsenite of silver will be seen at the junction of the two fluids; this test will serve to detect the pigment in wall paper, when no other pigments are present. If a little of the powder be heated in a reduction tube, it will be decomposed, arsenious acid will be volatilized and condensed on the cool portion of the tube, and oxide of copper will remain at the bottom. These tests will serve to detect the nature of Paris green quickly, but in legal cases they must be confirmed by applying all of the tests both for arsenic and for copper.

In order to isolate this compound from organic mixtures, the same process should be resorted to, which has already been described in detail under arsenious acid (see § 171).

X. *Antimony.*

§ 184. Compounds of antimony belong to the class of hyposthenian poisons, but they give rise to very few cases of poisoning compared with arsenic. The only compounds of antimony which have any toxicological importance are the tartrate of antimony and potassium (tartar emetic), and the terchloride of antimony. Metallic antimony has been thought to be poisonous, since it has caused injurious symptoms when inhaled in the form of vapor, but, as Christison suggests, these symptoms may have been due to arsenic with which antimony is almost always contaminated.

XI. *Tartrate of Antimony and Potassium.* (Tartar Emetic.)

§ 185. This compound, which is so largely used in medicine, has given rise to most of the cases of antimony poisoning. It is a white crystalline solid readily soluble in water, and contains forty-four per cent. of antimonyl, which is a compound of one atom of antimony with one of oxygen. It may cause both acute and chronic poisoning, some of the cases of criminal poisoning recorded being of the latter variety. Of the preparations used in medicine are the tartar emetic itself, the wine of antimony, which contains four parts of tartar emetic in one thousand of the wine (U. S. Ph., 1880), and the compound syrup of squill, which contains three parts of tartar emetic in two thousand parts of the syrup (U. S. Ph., 1880).

§ 186. *Symptoms.*—The salt is capable of producing violent and alarming symptoms, and occasionally also fatal effects. Its immediate action upon the stomach appears to be irritant, since it produces a burning pain in the stomach, excessive vomiting, and diarrhœa. The large doses which have been tolerated in some febrile affections, such as pneumonia, rheumatism, and mania-à-potu, have thrown some doubt upon its irritant properties, and its speedy rejection from the stomach in other cases defeats, in a measure, both its local and constitutional poisonous effects.

§ 187. The phenomena of acute poisoning by this agent may be thus described: The patient is attacked, within fifteen minutes or half an hour after ingestion, with incessant retching, præcordial cramps, and burning heat, distension of the epigastrium, watery and frequent stools, dryness of the throat, difficult deglutition, an unpleasant metallic taste in the mouth, and sometimes a copious discharge of saliva. The mucous membrane of the mouth becomes coated with a white aphthous crust like that of canker, which finally becomes brown or black in color; when this condition is seen in the mouth, we may also expect to find it, if the case ends fatally, in the stomach and intestines. Soon after, the patient complains of distress at the pit of the stomach, and becomes faint or falls into a state of syncope, of agitation, of vertigo, or of a sort of drunkenness. The region of the stomach continues painful, and there is heat in the throat and a difficulty in swallowing; vomiting reappears with extreme facility, and the vomited matters are sometimes mixed with blood; the urine is scanty; the extremities become cold, and

the skin is covered with a clammy perspiration; the depression is very great, and often there is loss of muscular power; agitation continues, and sleep is broken; the pulse, moreover, is a little quickened, but regular and soft. About the fourth or fifth day there appears upon the extremities and on different parts of the body a vesiculo-pustular eruption like that caused by antimonial ointment. In aggravated cases, the vomitings give place to a persistent hiccough, the stools become involuntary, the urine is suppressed, the coldness at the extremities extends and becomes general, the extremities become cyanotic, delirium and convulsions ensue, and death approaches in a period of time, varying from three to six days. In children, death may occur much earlier, sometimes after a few hours.

In a few rare cases the ingestion of an excessive dose of tartar emetic does not cause vomiting, but may be followed by alvine evacuations, complete prostration, collapse, a few convulsive movements and death in a few hours.

Most often a single excessive dose does not cause death in a person poisoned by this agent. The abundance and suddenness of the vomitings oppose the absorption of the poison, and the reaction, which may occur very early, limits the symptoms of poisoning to their first period; after having vomited for several hours, accompanied by alvine evacuations, the symptoms abate, the tongue becomes red, the pulse full, hard, and frequent, and the surface of the body warm. There remains for a short time a headache, epigastric distress, and a feeling of lassitude. The patient recovers his usual health in ten days or a fortnight.

§ 188. These symptoms, as will be seen, much resemble those of arsenic poisoning, and from the symptoms alone it is impossible to distinguish between the two forms of poisoning. Important points to keep in mind, however, are, that there are no remissions in antimony poisoning as are very liable to occur with arsenic, the case progressing steadily to death or recovery; that the urine is not so liable to be suppressed as in arsenic poisoning, the antimony being chiefly eliminated by the kidneys, so that in chronic poisoning the urine is rather increased than diminished; that the depression of the vital forces is very great, more noticeable, as a rule, than with arsenic; and that the antimony can always be detected in the urine.

§ 189. *Fatal dose.*—The amount requisite to endanger life is not accurately known, although a case is related by Dr. Beck, in which fifteen grains of tartar emetic killed a child a few weeks old, and in a case reported by Mr. Hartley, ten grains killed a child in a few hours. In several of the fatal cases collected by Dr. Beck, the dose did not exceed a quarter of a grain, but the patients were already weakened by disease. Two grains have proved fatal to an adult.[1] Dr. Pereira refers to a case in which death ensued in four days after forty grains had been swallowed. A case is related in which four grains nearly proved fatal. Violent pain in the abdomen, vomiting, and purging took place, and were followed by convulsions: the man became speechless, no pulse could be perceived, and the skin was quite cold; in short, it was supposed he was dead. Stimulating frictions and cataplasms were employed, and he slowly recovered in about fourteen days.[2] An Italian courier died in eleven hours after swallowing, by mistake, *one* drachm of tartar emetic.[3] In a case related by Dr. McCreery, U. S. N., a physician took, through a mistake of the apothecary, half an ounce of tartar emetic. In little more than half an hour he experienced nausea, which was followed by distressing vomiting and purging, with most violent cramps of the legs, and slighter ones of the wrists. Copious draughts of green tea, large doses of tannin, and other appropriate remedies were used, which did not, however, immediately mitigate the symptoms. He remained very much prostrated, but recovered in a few days.[4] In a case observed by Dr. J. T. Gleves, of Tennessee, where a tablespoonful was taken, these symptoms ensued, but reaction was brought about in seven hours. On the third day the fauces were covered with pustules, and on the following day the skin also. The patient recovered.[5] A case is related by Deutsch, in which a woman, who took, by mistake, a scruple of tartar emetic, was brought exceedingly low by its violent action, and died in the course of a year in consequence of its irritant effects upon the intestinal canal.[6] From a tabular view of thirty-seven

[1] Archives Gén., xxvi. 262.

[2] Taylor on Poisons, p. 389. See, also, two cases in the Union Médicale, 1852, No. 61, p. 245.

[3] Monthly Journ., May, 1850.

[4] Am. Journ. Med. Sci., Jan. 1853, p. 131.

[5] Am. Journ. Med. Sci., vol. xv., from West Journ. of Med. and Surgery, Jan. 1848.

[6] Canstatt's Jahresbericht für 1851, Bd. iv. p. 270.

cases of acute poisoning by tartar emetic, collected by Dr. Taylor,[1] it appears that sixteen proved fatal. Boudet observed local irritant effects upon the fauces twenty-six times out of one hundred and forty-four cases of pneumonia treated with tartar emetic.[2] Difficulty of swallowing and copious perspiration has also been frequently observed. Applied to the skin in the form of ointment, it produces a crop of painful pustules, which in weakly subjects may occasionally give rise to ulceration. It may cause nausea and vomiting even when thus used.

§ 190. *Post-mortem appearances.*—These have been observed in but few cases. In those cases before referred to as having been seen by Mr. Hartley, in which two children, of the ages respectively of five and three years, swallowed each ten grains of tartar emetic, the following appearances were noted. The bodies were examined between four and five days after death. "In that of the boy, there was effusion of serum in the right pleura; the lower lobe of the right lung, posteriorly, was redder than natural, and the peritoneum was injected from recent inflammation. The mucous membrane of the duodenum was inflamed and covered with a whitish-yellow viscid secretion; this was observed throughout the intestinal canal, although the color was deeper in the colon and rectum; there was no ulceration. The peritoneal coat of the stomach was congested. The mucous membrane of this organ was much inflamed, especially about the larger curvature and at the cardiac orifice; there was no ulceration. The contents (about two ounces and a half of a dark grumous fluid, having a slightly acid reaction) were very adherent to it; and in one place there was a patch of lymph. The tests used did not indicate the presence of antimony. With regard to other appearances, the tongue was covered with a white fur and appeared soddened; the fauces were not inflamed; the trachea and œsophagus had a natural appearance. On opening the cranium, the dura mater was found very vascular; the longitudinal sinus contained a coagulum of lymph, and but very little blood. The vessels of the surface of the brain were very much injected with dark blood, the whole surface having a deep purple color. Every portion of the brain, when cut, presented many bloody points. The

[1] Guy's Hospital Reports, 3d ser., iii. 409.

[2] Canstatt, 1853, Bd. v. p. 148.

cerebellum and medulla oblongata were also extremely vascular; there was no effusion in the ventricles or at the base of the brain. In the body of the girl, the morbid appearances were similar; there were also patches resembling the eruption of scarlatina on the arms, legs, and neck. The arachnoid membrane was more opaque than usual; and on the mucous membrane of the stomach, where the inflammation was greatest, were two or three white spots, each about the size of a split pea, which appeared to be the commencement of ulceration."[1] The body of a woman, who died in seven hours after taking an unknown quantity of tartar emetic, and was examined thirty-nine hours afterwards, presented no lesions whatever in the stomach or elsewhere.[2] But, when life is protracted after the ingestion of the poison, the changes will usually be found like those in the two cases above described. In other words, an aphthous condition of the mouth, fauces, and œsophagus; softening, inflammation, or ulceration of the stomach, and to some extent, also, of the intestines; and generally a dark color and a liquid state of the blood.

§ 191. M. G. Felizet, in an interesting article,[3] insists that the principal point of interest in this trial,[4] from a scientific point of view, is that of furnishing an example of poisoning by tartar emetic without the appearance of anatomical lesion. Drs. Douglas Maclagan and Littlejohn reported that the body of Mrs. Pritchard presented no appearance of morbid action capable of accounting for death.

§ 192. Most ordinarily, and especially when a single excessive dose has been taken, tartar emetic produces extended and multiform lesions. The œsophagus appears red and presents some ulcerated spots. The stomach and intestines are the seat of a violent inflammation, characterized by a vivid redness with softening of the mucous membrane, upon which are distributed blackened or brownish-red patches formed by the infiltration of blood and by hypertrophy of the follicles. The internal surface of the stomach and small intestine is covered with a blackish, thick, and viscid secretion, sometimes streaked with blood. Occasionally may be found in the *primæ viæ*

[1] Taylor, from Lancet, April 25, 1846, p. 460. See, also, a case by Mr. Beal, Lancet, Jan. 21, 1854.

[2] C. Ellis, Bost. Journ., Dec. 1856, p. 400.

[3] *De l'Action toxique du Tartre Stibié*, procès du docteur Pritchard. Archives Gén. de Méd., Sept. 1865.

[4] *Vide* also Bost. Med. and Surg. Journ., Aug. 10, 1865.

and even in the commencement of the small intestine, true pustules filled with pus. This inflammation of the intestines is especially liable to be found located in the very upper and lowest portions, the duodenum, cœcum, and rectum.

§ 193. Antimony produces fatty degeneration[1] of the liver, and this fact is so well known in the Duchy of Brunswick that the peasants of that district feed their geese upon white oxide of antimony to make them fat and well flavored. The pathological action of this poison upon the lungs is in no way different from that of other poisons of the hyposthenic group, and consequently not much importance should be attached to the pulmonary lesions occurring in a suspected case of poisoning by this agent.

This fatty degeneration may also extend to other organs, especially the kidneys and heart, and also to the muscular tissue generally, as in arsenic poisoning. The liver may become so fatty that it resembles the condition known as acute yellow atrophy of the liver, so that some authorities are, at the present time, inclined to believe that all reported cases of acute yellow atrophy of the liver were really cases of phosphorus, arsenic, or antimony poisoning. The writer met with a case a few years ago, which was diagnosticated at the Boston City Hospital from the autopsy as a case of acute yellow atrophy of the liver; analysis of this organ showed the presence of antimony in it, although no tartar emetic or other antimonial compound had been given while the patient was in the hospital; no previous history of his case could be obtained.

§ 194. *Chronic poisoning.*—The poisonous effects of tartar emetic, when used in small doses for a long time, have been carefully studied by Mayerhoffer[2] and by Dr. Taylor. The latter toxicologist considers it probable that the secret poisons which from time to time have produced so many victims, and have rendered so many names infamous in history, were antimonial. The following description of chronic poisoning by tartar emetic is furnished by Dr. Taylor.[3]

If tartarized antimony be given in small but increasing doses for a long period, there is uneasiness, nausea, and retching, followed by vomiting; the stools are pasty, and diarrhœa, attended with thin bilious and mucous discharges, gradually sets in; the abdomen at

[1] *De la steatose*, Thèse du concours d' aggregation de la Faculté de Médecine de Paris. Blachez, 1866, p. 38.

[2] Beiträge zur Heilkunde, ii. 372.

[3] Guy's Hosp. Reports, 3d ser. iii. 388.

the same time is distended and tense. The voidance of the urine becomes frequent and violent. The region of the stomach is tender and painful; that of the liver appears fuller, and is sensitive to the touch. There are griping pains in the bowels, with stiffness and pains in the lower limbs. The warmth of the skin is at first increased; there is itching or irritation with alternation of heat and cold. The appetite is suppressed, and when any substance is eaten there is nausea with an immediate disposition to vomit. A roughness or rawness is perceived in the throat with painful swallowing; the tongue is covered with a dirty mucus, and the mouth is clammy. The head feels full and heavy. At a still later period the feces contain much mucus, and are frequently strongly colored with bile. The blood gradually loses its fibrin, and becomes almost liquid, and dark-colored. It contains an increased proportion of fluid and saline matters with traces of antimony. If the use of the substance be discontinued, the action of the heart and pulse becomes slow with a loss of power; the breathing is difficult, the complexion dusky; there is complete depression of the vital powers with great debility and emaciation; the legs become heavy and stiff, as if paralyzed, and death may follow as a result of the noxious impression produced on the more important organs of the body. Small doses act more powerfully when dissolved, than when administered in the form of powder. In addition to the above symptoms, the vomiting, from which the patient suffers, is either attended or followed by the distension of the abdomen, and flatulence; by liquid bilious motions, with colicky pains, paleness and sunken appearance of the countenance; and by cold perspiration, giddiness, great prostration of strength, incapacity on the part of the patient of raising himself to the erect position, disposition to rest and sleep, loss of strength, fulness and frequency of the pulse, faintings, in many cases a feeling of coldness, accompanied with a heat or flushing of some parts of the body, *e. g.*, the face. If there is a recovery from this condition, pain in the stomach is felt for a long time afterwards, and inflammation of the stomach to a greater or less degree is set up. After the vomiting, there remains, for a longer or shorter period, an unwillingness to take food, and nausea in partaking of it. Among other effects, the perspiration and the urinary secretion are observed to be greatly increased.

If the use of tartarized antimony be continued in increasing doses

when the poison has been already carried into the blood, the secretory organs are more strongly stimulated, and absorption as well as secretion, especially of the serous liquids, is greatly augmented.

If vomiting does not take place after large doses, the following symptoms of poisoning are observed: metallic taste, nausea, retching, and bilious vomiting; burning pain in the throat, gullet, and stomach; spasms of the jaw and neck; pain and flatulent distension of the abdomen, with frequent watery motions; coldness, pallor, and clamminess of the skin, sometimes great heat of skin; difficult breathing, painful sobbing, giddiness, stupefaction, loss of consciousness, delirium, spasms of the arms and legs, with complete prostration of strength. Death appears to result from the impression produced on the nerves of motion, as well as on the nerves of the lungs and heart; leading either to asphyxia or paralysis.

§ 195. Attention has been already called[1] in this work to the liability of confusing the symptoms of poisoning by tartar emetic with those of a disease called cerebro-spinal meningitis, and in the trial of Mrs. Wharton for poisoning General Ketchum, it has been shown that this is a difficult question to decide. It must, however, be remembered that some of the medical men summoned as experts in that case were not competent to give technical testimony, and may have been prejudiced, either from a spirit of rivalry, or by a premature conviction that the prisoner was guilty of the charge of poisoning. The testimony of some of the witnesses was to the effect that the symptoms preceding the death of General Ketchum could not be attributed to any known disease, and yet they seemed to resemble those of the above-named malady, as has been clearly exposed in an article by Dr. Reese, in the *Amer. Journ. of Med. Sciences*, before referred to.

§ 196. *Chemical examination.*—It is proper to bear in mind, as Dr. Taylor remarks, that antimony given in a large dose, or repeatedly in small doses, is rapidly absorbed and eliminated chiefly by the urine. It is at the same time deposited in a greater or less quantity in the tissues and organs. Under recent administration, if in sufficient quantity, it may be found in the stomach and bowels, and little or none may be present in the liver. After a variable time it disappears from the stomach and bowels, although it may be

[1] *Vide* § 31 (*g*), p. 57.

present in the feces, while the liver, kidneys, and spleen may contain it in large, and the other organs in small quantity. In certain diseased states of the system, the complete elimination of the metal may require a period of twenty-five or thirty days or longer; but in a healthy subject, to whom only ordinary medicinal doses have been given, the antimony is quickly expelled.

The double tartrate of antimony and potassium (tartar emetic) is readily soluble in water, even when it contains cream of tartar. It is insoluble in alcohol. It crystallizes in the form of small white crystals belonging to the rhombic system.

If a little of the crystalline powder be heated upon a knife blade or porcelain plate it blackens, and there is evolved an odor resembling that of burnt sugar, due to the tartaric acid. A black residue having an alkaline reaction to test-paper is left.

If a drop of a solution of sulphuretted hydrogen or sulphide of ammonium be placed upon a few of the crystals, they will assume an orange color owing to the formation of tersulphide of antimony. A solution of tartar emetic, if strongly acidulated with hydrochloric acid and subjected to the action of sulphuretted hydrogen, will be precipitated, the precipitate having an orange color, and consisting of tersulphide of antimony, which is readily soluble in sulphide of ammonium, or in a solution of potassium or sodium hydrates; it is insoluble in a solution of sesquicarbonate of ammonium, and can by means of this reagent be separated from the tersulphide of arsenic; it is soluble in hot concentrated hydrochloric acid, which does not dissolve the sulphide of arsenic. If not first acidulated with hydrochloric acid, the above reagents will only produce an orange-colored solution.

Exposed to the reducing flame of the blowpipe with carbonate of sodium, white incrustations and globules of antimony are obtained, known by the needle-shaped crystals with which they are beset, and by their metallic brilliancy and brittleness.

Dilute nitric or hydrochloric acid throws down from a solution of tartar emetic a white precipitate, which is soluble in an excess of the reagent, and also in tartaric acid, which enables us to distinguish this compound from a similar one produced by the same reagents in solutions of bismuth salts. If just enough acid has been added to redissolve the precipitate first formed, and the resulting solution be thrown into a large amount of water, the white oxychloride or nitrate

will be again precipitated. The same is true of the solution formed by the action of strong hydrochloric acid upon the tersulphide of antimony.

A solution of tannic or gallic acid causes a precipitate in solutions of tartar emetic; hence the value of astringent infusions containing either of these acids as an antidote in cases of tartar emetic poisoning.

Ferrocyanide of potassium causes no precipitate, as it does in solutions of salts of most of the metals.

Galvanic test.—If a solution of tartar emetic be acidulated with hydrochloric acid, and a piece of platinum foil around which is bent a piece of metallic zinc be introduced into the fluid, galvanic action will take place, hydrogen gas will be evolved, and the antimonial compound will be decomposed, metallic antimony being deposited upon the platinum in the form of a black sooty coating, which can be dissolved in tartaric or hydrochloric acids, and subjected to the other tests for antimony.

Reinsch's test.—This is performed in the same way as for arsenic. The coating formed upon the copper has, as a rule, a more sooty appearance than with arsenic, and when the copper is heated in a tube closed at one end, the white sublimate is formed much nearer the flame than the arsenical sublimate, and it is usually in the form of prismatic crystals, although rarely it may assume the form of octahedral crystals, as mentioned above in speaking of arsenic. If the copper containing the coating of antimony be boiled with a solution of potassium hydrate, the antimony will be dissolved and can be tested by adding a solution of sulphuretted hydrogen, filtering from the sulphide of copper thus formed, and acidulating with hydrochloric acid, when the orange sulphide of antimony will be precipitated.

Marsh's test.—This is performed in the same way as the test for arsenic. The antimonial mirrors obtained, both in the tube and upon the porcelain plate, are usually blacker and less brilliant than those of arsenic.

The following table[1] may be convenient to determine at a glance the different properties of arsenic and antimony:—

[1] Médecine légale, par Briand et Claudé, Paris, 1869, p. 654.

Arsenic.	Antimony.
Volatile, easily displaced in a current of hydrogen.	Less volatile, melting into minute globules.
Disappears in presence of nitric acid. The solution, evaporated to dryness, deposits by a neutral solution of nitrate of silver a brick-red precipitate of arseniate of silver.	Gives no reaction with nitrate of silver, when treated in the same way.
Is dissolved by the addition of an alkaline hypochlorite.	Preserves its metallic lustre, if the layer is rather thick.
The metallic ring heated in a current of sulphuretted hydrogen gives a yellow sulphide of arsenic, soluble in ammonia, unaltered by hydrochloric acid gas.	The ring gives an orange sulphide, which is transformed into a volatile chloride by hydrochloric acid gas.
The gas set free in Marsh's apparatus reduces the nitrate of silver and gives a soluble arsenious acid, metallic silver being precipitated.	The gas reduces nitrate of silver, and the antimony is wholly precipitated with the silver.
Is not dissolved by a solution of ammonium sulphide.	Is dissolved by ammonium sulphide.

§ 197. By this test arsenic can be separated from antimony, when compounds of the two metals occur together, as may be the case either from the combined administration of the two drugs, or from the presence of arsenic as an impurity in tartar emetic which has been administered. M. Bouis states that the best method for separating these two poisons is the following, based on the action of arseniuretted hydrogen, and of antimoniuretted hydrogen upon a solution of nitrate of silver :—

Instead of forming a metallic ring in Marsh's apparatus, the gas is passed into a test-tube containing a solution of nitrate of silver; the arseniuretted hydrogen is transformed into arsenious acid; the antimoniuretted hydrogen gives the insoluble antimonide of silver; the two gases produce at the same time reduced silver. When all the suspected matter has been introduced into the apparatus, and the gas has been disengaged long enough, the black precipitate of silver and the antimonide of silver are collected on a filter and thoroughly washed. In the filtered liquid the excess of silver is precipitated by hydrochloric acid in the form of white chloride of silver; which is again filtered and the arsenic is precipitated from the clear solution by sulphuretted hydrogen. As for the black residue which remains upon the filter, that can be digested by aqua

regia, and then diluted with a little water; chloride of silver is deposited, and in the filtered liquid the chloride of antimony is left. It would be better, instead of treating the residue with aqua regia, to boil it with a concentrated solution of tartaric acid, which will dissolve the antimony without attacking the silver.

§ 198. *Detection of antimony in organic liquids.*—If an examination of the *tissues* is required for the purpose of detecting absorbed antimony, the liver should be selected as the organ most likely to contain it.

The method employed for the destruction of the organic matters is the same as for arsenic, the sulphuretted hydrogen precipitating the antimony in the form of the orange-colored tersulphide of antimony; this color is, however, always disguised by the presence of organic sulphur compounds, so that no inference whatever can be drawn from the color of the precipitate produced by the sulphuretted hydrogen. This precipitate should be purified in the same way as described under arsenic, by dissolving it in a hot mixture of ammonia water and ammonium sulphide, evaporating to dryness, oxidizing the residue with either concentrated nitric acid, or hydrochloric acid and chlorate of potassium, and evaporating to dryness again. This residue can be dissolved in water to which a little hydrochloric acid has been added, and subjected to all of the tests mentioned above. The spleen, kidneys, and urine should likewise be examined for antimony.

§ 199. It is evident that the presence of antimony, either in the stomach or absorbed in the other organs, may be due to the proper medicinal administration of the salt. The forms under which it is given, and the occasions on which it is prescribed, are numerous, and it is not unfrequently administered in cases of poisoning with other substances, without a thought of the complications it may, in case of a death, place in the way of the chemist. Hence, unless the possibility of its introduction into the system under any of these circumstances be fully excluded, the object of the medico-legal inquiry may be entirely frustrated.

XII. *Terchloride of Antimony.* (Butter of Antimony.)

§ 200. This substance is highly corrosive in its action. The following case will be sufficient to illustrate its effects: "An army surgeon swallowed, for the purpose of suicide, from two to three

ounces, by measure, of chloride of antimony. About an hour afterwards he was seen by Mr. Mann. There was entire prostration of strength, with coldness of the skin and incessant attempts to vomit. The most excruciating griping pains were felt in the abdomen, and there was a frequent desire to evacuate the bowels, but nothing was passed. In the course of a few hours reaction took place, the pain subsided, and the pulse rose to 120. There was now a strong disposition to sleep, so that he appeared as if laboring under the effects of a narcotic poison. In this state he continued until he died, ten hours and a half after he had swallowed the poison. On inspection, the interior of the alimentary canal, from the mouth downwards to the jejunum, presented a black appearance, as if the parts had been charred. In general, there was no mucous membrane remaining either in the stomach or elsewhere, only a flocculent substance, which could be easily scraped off with the back of a scalpel, leaving the submucous tissues and the peritoneal coat. All these parts were so soft that they could be easily torn with the fingers."[1] The symptoms have been similar in some cases which recovered. Poisoning with this substance is, however, very rare, only four cases being recorded, and mostly happens from mistake, or when it is taken with suicidal intentions. The tests are the same as for tartar emetic.

XIII. *Mercury.*

§ 201. All forms of mercury are poisonous. Even metallic mercury itself when in a finely divided form, as it is in mercurial ointment and in blue pill, can readily be absorbed from any surface, mucous membrane or skin, and give rise to symptoms of poisoning. Metallic mercury also frequently causes chronic poisoning when inhaled in the form of vapor, since it is slowly volatile at the ordinary temperature of the atmosphere (above 40° F.). There are a large number of employments in which metallic mercury is used, and in which the workmen are exposed to the danger of inhaling its fumes, such as separating gold from its ores, and mirror, thermometer, and barometer manufacture. All of the compounds of mercury, many of which are used in medicine, also give rise to cases of poisoning. The most important of these are corrosive sublimate (mercuric chloride, bichloride of mercury) and calomel (mercurous chloride, pro-

[1] Taylor on Poisons, Am. ed., 1875, p. 457.

tochloride of mercury), which latter is most frequently used in medicine, and has caused poisoning by being contaminated with corrosive sublimate, which is much more active, since it is quite soluble, and also by being converted in the stomach by digestion into corrosive sublimate. Of the other sources of mercurial poisoning we have the two iodides, mercuric and mercurous (red and yellow) iodides, both of which are quite largely used in medicine; the writer has recently met with a case of suicidal poisoning by the bisulphate of mercury. The two chlorides are, however, the most common sources of poisoning, since they can, as a rule, be easily obtained by the laity, corrosive sublimate being a common bed-bug poison, and calomel being largely used in children's intestinal diseases. Corrosive sublimate is exceedingly active as a poison on account of its ready solubility in water, alcohol, and ether, while calomel being insoluble in these fluids may often be given in very large amounts without producing serious symptoms. Most of the cases of poisoning are either accidental, owing to the ingestion of some preparation which was intended for external use, or owing to the use of impure (corrosive sublimate containing) calomel, or are suicidal. The exceedingly acrid, metallic, and styptic taste of the corrosive sublimate renders it unpopular with the criminal. Experiment has shown also that calomel may, when taken during digestion, be partially converted into corrosive sublimate owing to the presence of free hydrochloric acid and the digestive ferment in the gastric juice at that time.

§ 202. *Symptoms.*—We may consider corrosive sublimate as the type of mercurial compounds. Like many other poisons, corrosive sublimate or other mercurial preparations may cause death in a few hours, or the fatal issue may not occur for several days or weeks. Where death occurs within two or three days the poisoning is called *acute*, and when it occurs later or within a few weeks the poisoning is called *subacute* or *chronic*.

§ 203. *Acute poisoning.*—The *symptoms* caused by acute poisoning are of the most urgent and alarming character, and generally supervene very soon after the corrosive sublimate is swallowed. At the moment of being swallowed there is usually an involuntary constriction of the throat, which has in many cases prevented the whole of the draught from being taken, and a metallic taste in the mouth. Vomiting of a bloody and frothy liquid soon occurs, and continues

throughout the case, attended with violent purging, but the latter symptom is sometimes absent. There is excessive pain in the abdomen, not always increased by pressure, and also burning and smarting in the throat. The general symptoms are at first those of febrile excitement, with great thirst, and are followed by exhaustion and collapse, a cold and clammy skin, small and frequent pulse, and difficult respiration. In most cases the urine is either entirely suppressed, or very scanty and difficult to void.

The duration of the case is very variable, sometimes terminating in a few hours, but sometimes death may not occur until the third or fourth day. If life be prolonged sufficiently, the saliva should be carefully collected and examined chemically for the presence of the poison. The urine should also be subjected to chemical analysis, because, if it is found therein, it shows that it has been absorbed by the general system, and is undergoing a natural process of elimination; the urine should also be examined in the ordinary way for the detection of disease of the kidney, since, in all cases of acute poisoning by corrosive sublimate, when the poison is being eliminated by the kidneys it irritates or inflames these organs, so that we are sure to find the urine albuminous and more or less bloody according to the amount of irritation caused; in some cases the kidneys are acutely inflamed, and we find the condition known by physicians as acute nephritis or acute Bright's disease. When there is diarrhœa, as is usually the case, the dejections are very painful, frequent, and bloody.

The symptoms of poisoning by corrosive sublimate more closely resemble those of arsenic than of any other poison, but may be distinguished from the fact that the local action of the mercuric chloride is corrosive, while that of arsenic is irritant; in the former the animal tissues brought in contact with the corrosive show a whitish or grayish eschar with swelling, not unlike the effects produced by nitrate of silver. Occasionally, however, the corrosive action on the lips and inside of the mouth may be absent. Occasionally, also, the symptom of suppression of urine may be absent.[1] Another distinguishing feature, though, as will be seen farther, one not to be relied upon, is that corrosive sublimate, as well as all mercurials, usually

[1] Boston Medical and Surgical Journal, 1879, Pt. 2, pp. 781-783, reported by Drs. Schenk and Post.

cause salivation or ptyalism as an early symptom. Generally salivation occurs within the first three or four days, but in one case on record[1] it occurred within the first four hours, and in another within one and a half hours.[2] Occasionally, this symptom is very much delayed, and in a case reported by Dr. Venables,[3] where two drachms caused death in eight days, no salivation occurred. The amount of salivation also varies greatly in different cases where similar doses were taken but it should be noted that salivation may occur without mercury, since in one case reported by Dr. A. Farre,[4] in a pregnant woman who had taken no mercury, saliva was secreted to the amount of three pints a day, but in this case there was no swelling of the salivary glands nor fetid breath. In cases of salivation caused by mercury these glands are usually swollen, and there is associated with the salivation an intense fetor and ulcerated condition of the inside of the mouth, and, as a consequence of this condition, the teeth may loosen and fall out. According to Mr. Taylor,[5] salivation may occur after the use of gold, copper, bismuth, lead, antimony, iodine and iodide of potassium, croton oil, opium, prussic acid, carbolic acid,[6] sulphuric acid, arsenic, colchicum, foxglove or digitalis, and cantharides. Ulcerated gums and salivation may occasionally coexist in the salivation caused by arsenic and bismuth, but a chemical analysis of the saliva will reveal the nature of the poison. In salivation caused by mercury, the presence of this poison may be chemically detected in the salivary secretion, because it is eliminated from the system with this secretion, as well as by the intestines and by the kidneys. Salivation by mercurial preparations is not so common in the young as in the adult.[7] Dr. Craigie and Sir R. Christison state[8] that in the granular disease of the kidneys a small amount of mercury will induce salivation very readily. Several instances are detailed by Mr. Taylor, in which death was apparently caused by profuse salivation even after the use of medicinal doses of calomel. It may not be improbable that in these cases the death was due to the fact that the unhealthiness of the kidneys may have prevented a sufficient elimination of the poison; at any rate, it is

[1] Guy's Hospital Reports, 1843.
[2] Med. Gaz., 1843.
[3] Taylor, op. cit., p. 356.
[4] Trans. Obstet. Soc., vol. xv., 1874, p. 222.
[5] Op. cit.
[6] Guy's Hosp. Rep., 1870, p. 533.
[7] Dub. Med. Press, May 12, 1847.
[8] Taylor, op. cit.

important in deaths caused by salivation from small doses of mercury, that the *post-mortem* appearances of the kidneys should be presented in evidence.

§ 204. *Chronic poisoning.*—The above account of symptoms is mostly concerned with cases of acute poisoning by corrosive sublimate with the exception of the account of salivation, which is more common with subacute and chronic poisoning than with acute. In the case of slow or subacute or chronic poisoning by mercurial preparations, the symptoms of local corrosive action on the *primæ viæ* may be absent, and the effects of slow poisoning only may be observed. In cases of slow poisoning the symptoms of continuous use of small non-corrosive doses will be those of salivation as above described with the appearance of a blue line on the margin of the gums not unlike that caused by lead-poisoning. Usually the first symptom of chronic poisoning caused by the repeated or continuous absorption of small amounts of mercury, is a tenderness of the teeth, which is especially noticeable on bringing the jaws forcibly together, so that physicians when prescribing a mercurial in cases of syphilis should always direct their patients to do this, and if they notice any such tenderness to omit the medicine for a short time; sometimes a little looseness of the bowels will occur before the tenderness of the teeth. Of the other premonitory symptoms of chronic poisoning we have great pallor and excessive weakness; this weakness progresses steadily, affecting first the muscles of the upper limbs, after a longer period extending to the lower limbs, and finally, if the patient lives long enough, it ends in general palsy or trembling of all of the muscles. Colicky pains in the abdominal region associated with nausea, vomiting, general restlessness, and a feeling of malaise may also be present. The salivary glands become tender and swollen, and the mouth inflamed and ulcerated. The patient may also be troubled with difficult breathing and deglutition. The serious salivation or ptyalism is the most prominent symptom, and may threaten or cause death. Dr. Johnson[1] reports a case where three grains of gray powder (mercury with chalk, hydrargyrum cum cretâ), which contains 50 to 60 per cent. of mercury, caused salivation, and Sir R. Christison[2] reports another case in which two grains of calomel (mild chloride of mercury) destroyed life by

[1] Sanitary Record, July, 1874. [2] Taylor, op. cit.

salivation. Taylor reports a case[1] in which profuse salivation and sloughing ulcerations caused fatal poisoning in a child of eight years, death following four days after taking three doses in one week, which amounted to a total only of sixteen grains.[2] In still another case six doses in three days, amounting to a total of eighteen grains, caused in a girl of nineteen years severe salivation, loosening of the teeth, and death in twelve days. It is also sufficiently well shown that salivation may appear, disappear for several months (three to six), and then reappear in an aggravated form without any mercurial compound having been taken in the mean time. In all these cases it is very important, as has been before observed, to examine the condition of the kidneys. There is one curious fact to be observed in regard to mercurial treatment, which may be of medico-legal importance, that the system can easier and more rapidly eliminate one large dose of mercury than the continuous administration of small doses frequently repeated, and that habitual constipation associated with continuous mercurial treatment seriously obstructs the elimination of the drug; this fact is not unreasonable, since the mucous glands in the intestinal canal lend material assistance in the elimination of mercury, and large doses of mercury generally induce diarrhœa.

It should be observed here that mercurial inunctions (where mercury is rubbed up with fat and thus becomes subdivided into very many minute globules) cause a very ready absorption of mercury through the skin, and the broken skin of the hands in contact with the poison, will easily allow its absorption. It is also well known that the presence of a large body of a mercurial preparation in badly ventilated apartments will readily allow absorption of the poison from the distribution of mercurial vapor in the air, and its inhalation by the lungs. Solutions of corrosive sublimate were formerly used by surgeons to purify their hands; Mr. Taylor[3] gives instances where mercurial poisoning was thus induced. This writer also enumerates several instances where ointments or lotions sold by quacks as secret remedies for diseases of the skin have induced fatal poisoning. One case of especial value, that reported by Mr. Annan, illustrates this danger. A shepherd used a lotion of two drachms

[1] Op. cit., p. 357.
[2] Lancet, Dec. 20, 1851, p. 579.
[3] Op. cit.

of corrosive sublimate dissolved in twenty ounces of water, to wash sheep affected with some skin disease, and died from the secondary effects of mercurial poisoning; and of ten sheep thus treated two died shortly after the application of the lotion.

As before observed, death may occur in a few days in some cases, and in others be prolonged beyond a week. In a case reported by Dr. Coale, death took place on the eleventh day; and in another, by Dr. Jackson, on the thirteenth day.[1] Death may not occur until later from the consecutive effects. Such was the case in an instance reported by Dr. Ware, where the patient died with profuse discharge of mucus and bloody matters from the bowels on the fifteenth day.[2]

§ 205. A few other cases of death from the *external application* of corrosive sublimate are on record. In one the subject was a child, and the severest constitutional effects were produced. It died in about a week.[3] In two others, also children, of seven and eleven years respectively, an ointment, composed of two drachms of corrosive sublimate to an ounce of tallow, was rubbed into the scalp. The children were affected with *porrigo favosa*. Excessive suffering was the almost immediate consequence, and in forty minutes they were completely delirious. They vomited continually a green-colored matter, had great pain in the bowels, with diarrhœa and bloody stools. In the youngest there was complete suppression of urine. Death occurred in one on the seventh, in the other on the ninth day. There was no *ptyalism*.[4] In a case in which from fifteen to twenty grains of corrosive sublimate were injected into the vagina, there occurred, besides local symptoms, vomiting, bloody purging, coldness of the extremities, spasms of the fingers and toes, and salivation. Recovery took place.[5]

§ 206. "The symptoms of mercurial poisoning are of incontestable value as evidence, and more so, perhaps, than for any other poison of the hyposthenians; the clinical record has here a certain signification, and allows of an almost certain diagnosis.[6] There is no disease which, in the acute form, has such a fatal result, consequent upon the specific buccal lesions; and for the chronic form

[1] Am. Journ. Med. Sci., Jan. 1851.
[2] Ibid.
[3] Ibid., July, 1844, p. 259.
[4] De Ricci, Dub. Quart. Journal, Aug. 1854.
[5] Butcher, Dub. Quart. Journal, Feb. 1856, p. 242.
[6] Tardieu, op. cit., p. 571.

the cachexia, which constitutes the common ground of so many diseases, imprints in this case a particular character to such nervous phenomena as the tremblings, partial paralyses, and pains in the bones. Syphilis alone can be confounded with chronic mercurial poisoning."[1]

§ 207. As has been remarked before, there may be some reason for confounding mercurial poisoning with that of arsenic. But the taste of these two poisons is different; that of mercurial preparations is much more pronounced, and has not the sweetish, followed by the bitter taste which has been mentioned as occurring occasionally in arsenic. The appearance of the first effects of the corrosive sublimate is instantaneous, whilst that of arsenical poisoning may be delayed an hour or more. The lesions of the mouth and œsophagus are most generally wanting in arsenical poisoning, whereas they are almost specific signs of mercurial poisoning, as are the fetor of the breath and the loosening of the teeth.

§ 208. Poisoning by copper is, in some respects, analogous to that of mercury, but a close attention to the signs of copper poisoning, which will be discussed further on, as well as the comparatively less violent action of the former, will serve to distinguish these poisonings.

§ 209. The subject of chronic poisoning by mercurial preparations, and the discussion of questions arising out of the specific effects of them upon the system, involves too many considerations to be profitably introduced here. We, therefore, refer the reader, for information on these points, to Dr. Christison's treatise, and to the standard works on Pathology and the Practice of Medicine.

We cannot forbear, however, to call the reader's attention more particularly to that form of disease known under the name of *cancrum oris*, *gangrœnopsis*, and *mercurial sore mouth*, especially in children.

§ 210. That death may occasionally result from the action of mercury upon the mouth, there can be no doubt. It is extremely important, however, to know, if this can be distinguished from those forms of inflammation and gangrene of some portion of the buccal cavity which are the result of certain depressed and diseased conditions of the system, independent of the action of mercury; and

[1] Ibid.

also, whether mercury given to a patient whose vital force is thus reduced, and whose blood is already depraved, may not be the *exciting* cause by which the tendency to gangrenous ulceration becomes developed. It is much to be feared that the inappropriate administration of this drug, has, in some cases, been followed by serious, and even fatal, disorganization of the mouth; while, on the other hand, physicians may be unjustly blamed for consequences which were really not the result of their imprudence, but of other causes which they were unable to control.

We propose first, by a few cases, to contrast the appearances presented by the effects of mercury on the mouth, with those which are due to disease.

A boy about ten years old, supposed to be suffering with bilious colic, was given twenty grains of calomel, which purged him in four hours; he took, in twenty-four hours, ten grains more with the same effect, but without much relief. On the morning of the fourth day (medical treatment having been continued, but no calomel or any of the acids used) all the symptoms of the early stage of ptyalism set in; the inflammation and swelling of the salivary glands rapidly increased, so that by the day following, there was a general swelling of all the soft parts of the face usually affected by severe ptyalism. On the next morning, a small, gangrenous spot, of a dark-brown color, was discovered on the middle and inner surface of the lower lip, which rapidly spread until the seventh day; at this time, the entire lower lip, the inside of both cheeks, and the surface of the tongue, were completely grangrenous, the lower lip and the tip of the tongue were wanting, having been destroyed by ulceration. The ptyalism increased, a stream of viscid saliva was constantly running out of the mouth, and the patient presented a most piteous spectacle. The breath was very fetid and offensive. Death occurred on the twelfth day. In another case, a little girl, ten years old, who received a fracture of the head from a fall, was given some calomel for the purpose of opening the bowels, but it did not operate. Her mouth became sore, and got rapidly worse, notwithstanding every effort was made to relieve it. The throat and face became immensely swollen, the teeth became loose and several came out, and the whole inside of the mouth, tongue and all, had a very black appearance, emitting a constant flow of a dark, putrid saliva of intolerable fetor. The greater part of the mouth

and tongue mortified, and part of the tongue, the under lip, and part of one side of the face sloughed off, presenting a most horrible spectacle, and one exquisitely distressing to the parents and friends of the little patient—the more so as the child continued to live some days after these parts had become detached.[1]

§ 211. That form of disease due to the action of mercury upon a depraved constitution may be illustrated by the following cases.[2] A boy, aged thirteen years, after suffering from influenza and partially recovering, was attacked with gastro-enteritis, from over-indulgence in animal food. The bowels were moved daily with about two grains of calomel, followed by a teaspoonful of castor oil when necessary. He amended under this treatment; but, about three weeks after the commencement of his sickness, it was discovered that mortification had commenced under the tongue, near the third molar tooth, on the left side; it extended around all the molar teeth of that side, embracing the gum and a portion of the cheek. The cheek was slightly swollen, and the left eye was opened with some difficulty. The mortification spread rapidly, notwithstanding the use of caustics, a dark spot appeared on the outside of the cheek, and the patient died on the third day. It is stated that the boy had dug out a piece of a tooth with a knife, a few days before the mortification began, at the place where it commenced. About twenty grains of calomel were taken during the first week of the disease, and none afterwards. The glands were not affected; and the mouth, when the mortification commenced, presented a healthy appearance.

Dr. S. Jackson (late of Northumberland) says: "I applied mercurial ointment to the face of a child, about three years old, to prevent the pitting of confluent smallpox; in a few days the gums were swelled and the teeth loosened, but only on the side upon which the patient continually lay. The gums soon mortified, the gangrene spread to the cheek, bringing on a fair case of gangrænopsis, and she died of her twofold disease in a very few days. This," he says, "was not a case of salivation, for the other side of the jaw remained sound, and the teeth on that side firm in their sockets."[3] In the same manner were fatal ulceration and gangrene

[1] Boston Med. and Surg. Journ., vol. xxxii. pp. 459 and 517.

[2] Ibid., p. 342.

[3] Trans. Coll. of Phys. Philad., U. S., vol. ii. No. 3.

developed, in a case reported by Dr. Marshall Hall. A child four years of age, with whooping-cough, took, according to a prescription furnished from a dispensary, three grains of calomel, on the 29th of October, and the same dose four times thereafter until the 7th of November. About this time the right cheek became much swollen, and there was great difficulty in opening the mouth, with very offensive breath. The gums and inside of the cheek became ulcerated, and on the 16th a sphacelus appeared on the right cheek, of about the size of a shilling, which rapidly extended to the size of a crown. The child continued to get worse, and died on the 23d. On *post-mortem* examination, there was found pleuro-pneumonia of the lower lobe of the right lung; there was an extensive eschar in the right cheek; its size, externally, was two and a half inches in length by one and a half in breadth. It penetrated through the entire cheek, and occupied an equally extensive space on its internal surface; the contiguous gum was in a similar state of sloughing, the alveolar processes were denuded, one or two teeth had disappeared, and several adjacent ones were loose. On the left side there was incipient gangrene of the cheek internally, and also of the contiguous gum, and the teeth were loose. The rest of the mouth was not affected.

§ 212. Cases of true gangrene of the face, however, have a different origin and course. A single example will suffice. It is taken also from Dr. M. Hall's Observations in Medicine. A little girl, aged three years and a half, had been affected with *fever* about fourteen days, and was apparently convalescent, when the left side of the face and lips was observed to be swollen, and to have a red and glistening appearance. About the same period, three spots were observed, one on the gum of the lower jaw, and the other two on the left cheek. These spots became dark-colored, and gradually spread. A slough separated from the cheek, and exposed the inside of the mouth. The contiguous teeth fell out. The breath and the exhalation from the ulcer were extremely offensive. The child lingered about fourteen days, and sank gradually.

Having thus seen the various forms of disease of the mouth which may give rise to a suspicion of poisoning by mercurial preparations, it only remains for us to point out the means by which the symptoms really caused by such preparations, may be distinguished from others which are spontaneous in their origin.

§ 213. All authors agree that mercury does not produce salivation in children as readily as in adults. Dr. Clarke says, that, although he has prescribed mercury in very large quantities in a great number of cases, he never produced salivation, except in three instances, in any child under three years of age. Dr. Warren, of Boston, observes: "That he has never known an infant to be salivated, notwithstanding he has given, in some cases, large quantities with this view." Mr. Colles, of Dublin, says: "No man in the present day requires to be told that mercury never does produce ptyalism or swelling and ulceration of the gums in infants." Drs. Evanson and Maunsell say: "Mercury does not seem capable of salivating an infant; we have never seen it do so, nor are we aware of any such case being on record."[1] Dr. West, of London, says: "In infants under five years of age, the gums hardly ever become affected by mercury, though most energetically employed; and it has never yet occurred to me to meet with an instance of profuse salivation or dangerous ulceration of the gums. Such accidents, however, do now and then occur, and have been known to terminate in fatal gangrene of the cheek or necrosis of the jaw." Yet, when salivation does occur, there is quite sufficient testimony, which it is unnecessary to quote, that the most disastrous consequences may follow. In this fact, however, appears to lie the great distinction between the disease resulting exclusively from the use of mercury, and that which is spontaneous or merely called into action by it. Dr. Hall says: "It is well known to every observer that the effect of calomel, when it does take place, is *uniformly diffused* over the *gums*, *tongue*, and internal parts of the cheek." Further, "it is *diffused* and is totally different in many respects from the *circumscribed* form of the gangræna oris." In other words, the mercurial disease commences in the gums and tongue; they swell, ulcerate, and slough, and the disease may then extend itself to the lips and cheek. The disease is, therefore, different in its early manifestations, is attended with salivation, is slower in its progress, and at first confined to parts which in true gangrænopsis are only secondarily affected.

§ 214. We copy the following description of *gangrœnopsis* from

[1] Beck's Essays on Infant Therapeutics, p. 48.

the monograph of Dr. Jackson, which we have already referred to:—

"I. The gangrænopsis attacks the cheek, the lip, or the nose, sometimes the fauces; most frequently in children, but sometimes in adults.

"II. It begins in those soft parts, and never in the maxilla, often where no mercury has been used, in a debilitated and febrile state of the system, as in idiopathic fevers and dysentery. Van Swieten saw it in scurvy, and Huxham in measles. Dr. Marshall Hall says (p. 178): 'In all the cases which came to my knowledge, this affection had been preceded by fever, acute disorder of the digestive organs, inflammation of the lungs, variola, rubeola, or scarlatina.' An exhausted state of the vitality, with cachectic fever, is, therefore, the predisposing cause.

"III. The exciting cause is any injury done to the parts. I saw it evidently started in two cases by the child's lying continually on one side, with a hand under the cheek, thus pressing the mucous membrane against the molar teeth; a protuberance of this membrane being caught between the teeth, was continually bruised, and a point of gangrene was thus established in an exanimate state of the whole system.

"IV. It is sometimes the result of severe cases of cancrum oris, the irritation spreading from the gums to the cheek."

It is well known that cancrum oris and the gangrene which attacks the cheek often occur in cases where no mercury has been given. We think that there is between these two little pathological difference; the most tangible distinction being, as it appears to us, that the canker sore-mouth of children (as it is called) sometimes prevails endemically in low, unhealthy situations, and among the poorer classes, being frequently seen in the hospitals for children, and occurring without being necessarily preceded by disease; whereas the gangrene of the cheek is commonly a sequel of exanthematous or other prostrating diseases such as measles, scarlet fever, and diphtheria. Both are allied closely to the gangrene of the genitals in female children, elsewhere referred to. (*Vide* RAPE.)

§ 215. We have carefully given the various opinions of physicians upon the subject of mercurial sore-mouth in children, and the well-known fact that sore-mouth unprovoked by mercury may exist among infants. Though the question appears from the above histo-

rical *résumé* to be somewhat complicated, it need produce no serious embarrassment in any given case, since all doubts of the cause in a suspected case of poisoning may be set at rest by a chemical analysis of the child's saliva or urine, when the presence of mercury will be declared, if that is the cause. On the other hand, the fact cannot be denied that many medical practitioners use mercury in cases even of sore-mouth, yet this matter can be settled by evidence adduced to show by the physician's testimony that the sore-mouth existed prior to the institution of mercurial treatment. It is even now (1883) a mooted question among physicians whether mercurial treatment in serious ulcerations and gangrenous affections of the mouth and throat is a wise measure; for, at a recent discussion in a medical society in the presence of the writer, he was astonished to learn that practitioners of good reputation advised the use of mercury in putrid sore-throat and diphtheritic inflammations. The symptoms and course of gangrene of the mouth are described by Niemeyer[1] on the authority of Barthez and Rilliet.[2] The gangrene commences, usually without pain, on the inner surface of the oral mucous membrane; a soft, regular, circumscribed œdema occurs in the affected cheek and lips, and gradually spreads. A hard, round nucleus forms in the centre, over which the skin appears shining, pale, or mottled violet. Even when the inside of the cheeks and a great portion of the gums have become gangrenous, the child often sits quietly in bed. A sanguineous, or even black, saliva runs out of its mouth; but it plays, demands food, takes it eagerly, and with the food swallows the sloughs that fall off from the gangrenous patches. At the same time the skin is pale and cool, the pulse small and moderately frequent, and there is delirium at night. Occasionally, mostly at the fifth or sixth day of the disease, a circumscribed, dry, black slough forms on the cheeks or lower lip; this may increase daily, till it affects half the face. Occasionally, even at this stage, the child is tolerably strong, will ask for food while pulling gangrenous patches from his mouth. The child's appearance is repulsive, as the slough separates from the underlying tissue, and the jawbone and loosened teeth show frightfully. The odor in the exhaled breath is offensive, the patient grows weaker and

[1] Text-book of Practical Medicine, New York translation, 1869.

[2] Maladies des Enfants, Paris, pp. 359 *et seq.*

weaker, and diarrhœa supervenes. The patient may die from exhaustion without any febrile disturbance, or may even recover from the extremity of the disease, a healthy suppuration taking the place of the gangrenous sloughs. We have taken great pains to give a detailed description of this gangrænopsis, or canker, or sore-mouth, since unjustly many children have been supposed to have been poisoned by mercury when this spontaneous disease is present.

§ 216. The description of *mercurial stomatitis* is presented in an article by Chauffard,[1] from which we take the following abstract: "The commencement of mercurial stomatitis or sore-mouth is marked by a dryness of the mouth, attended with heat and pain, and is accompanied by a true inflammation of the mucous glands. The breath has a peculiarly offensive odor difficult to describe, but easily recognized by one who has once perceived it, and simultaneously with this fetor the subject complains of a disagreeable taste, nauseating and metallic. The teeth feel as if they were 'on edge,' and become painful, and seem lengthened on account of the swelling and inflammation of the underlying periosteum of the alveolar process. Very soon the movements of the mouth in efforts of mastication become painful, but this is soon replaced by a pain which is restricted to the teeth and cheeks, though sometimes it is likewise felt at the angle of the jaw, and extends to the side of the face, simulating facial neuralgia. The pain soon becomes so intense that only liquids are swallowed, the patient having some difficulty in separating his jaws on account of a sort of reflective tetanus. The initial dryness of the mouth is of short duration, and soon is replaced by the opposite phenomenon of salivation; in light cases this salivation is distressing and painful, but may not be excessive; the patient may spit during the day, and drivel (droole) during the night, and this may be the whole extent of salivation. In severe cases, however, the ptyalism may assume remarkable proportions, there being a continuous dribble, or excessive flow of a clear or grayish saliva, having an insipid and fetid odor (Grisolle). Several quarts may flow during twenty-four hours, and the most simple chemical test will show the presence of mercury. This excessive secretion exhausts the patient, troubles his sleep, and may even, as before remarked, cause his death. During the presence of the above

[1] Nouveau Dict. de Méd. et Chirurgie, tome xxxiii., Paris, 1882.

symptoms, a physical examination will reveal an intense inflammation in the mouth; in light cases, the gums are swollen and tumefied, and of a livid, violet shade; where the gums naturally should join the teeth, they are separated, and show a lively red or whitish line, ulcerated, and bleeding at the slightest touch. The teeth thus exposed seem lengthened, loose, painful on pressure, and are covered with a grayish envelope. Simultaneously with these physical appearances the inflammation will extend over the whole cavity of the mouth—on the margin of the tongue, the internal aspect of the cheeks, the posterior surface of the lips, the mucous membrane assumes a vivid red color, is infiltrated, covered with grayish or opalescent epithelial scales, and shows the imprint of the teeth and the raised impress of their furrows. Even in light cases the tongue participates in the inflammatory action of the mouth, the evidence of its swollen, inflamed condition corresponding with the stomatitis, which is a very certain evidence of its mercurial cause."

§ 217. In severe cases of mercurial stomatitis or sore-mouth a copious and even bloody salivation is accompanied by very active pain in the submaxillary and other salivary glands. The teeth loosen and fall out in a few days' time, sometimes making the patient actually toothless. Along with these phenomena the grayish patches of the inflammation of the mouth invade the inner aspect of the cheeks, the margins and upper surface of the tongue, and below it may be found fissures and more or less deep ulcerations of the mucous membrane. The swelling of the tongue becomes so extensive that it may fill up the whole cavity of the mouth, and may even cause it to project. Respiration becomes difficult on account of the swollen and infiltrated tissues, and may cause a true mechanical asphyxia. The inflammation of the tongue may extend backward into the pharynx and throat, and may cause deafness as well as pain in the ears from extension of the trouble through the Eustachian tubes; its extension into the throat may cause an attack of suffocation from œdema of the glottis.

In the most severe cases, but rather exceptional, gangrenous patches of the surface of the mouth may be observed, necroses of the jawbones, a general restlessness accompanied by fever, and headache. The patient can neither eat nor sleep, and may pass into a state of cachexia which may become fatal. The duration of mercurial sore-mouth is entirely dependent upon its intensity, and

light cases may recover in eight or ten days. In cases of medium intensity, on the contrary, the salivation may resist the most rational treatment, and persist obstinately during weeks and even months. Convalescence is always slow, and it is seldom that patients can chew solid food for a long time without provoking pain and swelling of the gums. Finally, the extreme cases when cured are left in a pitiable condition, feeble, cachectic, and often disfigured by frightful scars caused by the loss of the teeth, or decay (necrosis) of the jawbones.

§ 218. The *differential* diagnosis between true gangrene, stomatitis or sore-mouth, and mercurial stomatitis is easy of recognition, even without the aid of a chemical test for the presence of mercury in the saliva. A comparison of the symptoms above given in either case shows a marked difference between the affections. In natural gangrene the disease begins by ulceration without inflammatory action of the mucous glands and without pain, while in mercurial stomatitis both are present. The other points of difference are presented in such detail that it would be wearisome to repeat them. It is evident, we think, from what has been said, that the diagnosis of the cause of these various forms of disease is not difficult. It depends chiefly upon the possibility of ascertaining the manner in which the disease first manifested itself—whether by swelling and ulceration of the gums generally, with an increased flow of saliva, or whether it commenced in the mouth or cheek with a hard red swelling, rapidly running into gangrene. The character of the disease under which the child was suffering, and its hygienic conditions, must also be known.

If an opinion is required only after death, or at an advanced period of the disease, it may be impossible to know whether it can be ascribed to mercury, or whether, in case it is known that mercury has been exhibited, it can be fairly attributed to it. Dr. Taylor does not admit the validity of the criterion that mercurial poisoning can be known by the *uniform diffusion* of the disease over the gums, tongue, and internal parts of the cheek, as advocated by Dr. Hall; and Dr. Christison, he says, has recorded a case in which, although the gangrene resulted from mercury, it was observed to occur on the skin near the mouth, on each side, whence it spread over the whole cheek, and destroyed life in eight days.[1]

[1] Op. cit., p. 319.

He also gives a case in which a charge was made against a medical practitioner, of having caused the death of a child aged four years by administering an overdose of some mercurial preparation. The child was laboring under whooping-cough, and some medicine was prescribed; on the fourth day the child complained of soreness of the mouth, the teeth became loose and fell out, the tongue and cheek were very much swollen, and the child died in the course of a few days from gangrene in the left cheek. The answer to the charge was that not a particle of mercury had been exhibited; a fact clearly proved by the production of the prescription-book of the medical attendant.

Chlorate of potassium is an almost certain remedy against *cancrum oris*, and also is the best treatment for the mercurial sore-mouth.

§ 219. The *smallest quantity* capable of destroying life is not ascertained with precision; children have been killed by three grains; and Dr. Taylor considers that the average fatal dose may not differ widely from that of arsenic, *i. e.*, two or three grains, but like doses of arsenic, a fatal dose in one case has proved innocuous in others, and in cases of disease like syphilis, enormous doses have been given with impunity for one and two years continuously; in this the syphilitic poison seems to offset the dangers from the metallic poison. Corrosive sublimate is not, however, the most common form of prescribing mercury in syphilis, practitioners preferring the biniodide, or protochloride of mercury, or by inunction.

In Dr. Coale's case less than ten grains were swallowed. Dr. Frisselle reports a case which is remarkable for the indifference to the symptoms upon the part of the patient. A woman took a drachm of corrosive sublimate in solution. She was immediately seized with a burning sensation in the throat, and copious vomiting of a dark, frothy substance, which was followed in about an hour by purging, which continued till the next day. She still, however, attended to her domestic duties, and no remedies were given internally until thirty-six hours afterwards. She died on the sixth day, with vomiting of a dark grumous matter.[1]

"Persons have been known to recover who have taken very large doses when remedies have been timely administered, or when there was early vomiting. . . . A case of recovery after forty grains

[1] Boston Med. and Surg. Journal, 1850, p. 279.

had been taken in whiskey, under circumstances favorable to its fatal operation, *i. e.*, on an empty stomach, is recorded by Dr. Andrews (in Cormack's *Journal*, Feb. 1845, 102)."[1] Poisoning by this agent is oftentimes extremely slow, and sometimes is not manifested for several months after exposure to mercurial vapors. Long before this period the face becomes swollen, the complexion livid, all the functions languish, ending in hemorrhages from the mucous surfaces, and from time to time the patients are afflicted with diarrhœa. The theory of the action of this poison has been explained by Dr. G. Sée.[2] As soon as the mercury is received into the blood, it is combined directly with the albumen of the *plasma* and the proteine of the corpuscles; this is the cause of mercurial anæmia. The white corpuscles, however, maintain their normal condition.

The antidote to mercurial poisoning is albumen in a large amount of water (too much of the antidote causes a *re*-solution of mercury). The albumen is precipitated in a solid form by the mercury, and this can then be ejected from the stomach, but if retained in an excess of albumen, its *re*-solution may cause subsequent and sub-acute poisoning.[3]

§ 220. *Post-mortem appearances.*—In the case mentioned by Dr. Frisselle, no appreciable lesion was discovered. In one reported by Dr. Williams,[4] the stomach, which was contracted in the shape of a dumb-bell, presented patches of dotted injection, of a bright crimson tint. There was no ulceration nor ecchymosis, but the mucous membrane was a little softened in the neighborhood of the most vivid red patches. Similar patches were seen throughout the small intestine. The bladder was contracted, and contained about a drachm of turbid urine. The other organs were healthy. The dose in this case was thirty grains, and the patient lived two days. In an instance reported by Dr. Herepath, the stomach seemed to have escaped the action of the poison, but the cæcum was of a deep black-red color, and portions of it were in a sphacelated condition.[5] The mucous membrane of the mouth and fauces usually exhibits

[1] Taylor, Am. ed., 1875, p. 367.

[2] Leçon de Pathologie Experimentale, 1ér fascicule, Paris, 1866, p. 275.

[3] See the case referred to in Bost. Med. and Surg. Journal, by Drs. Schenck and Post, p. 447.

[4] Boston Med. and Surg. Journal, 1850, p. 279.

[5] Lancet, Dec. 1845.

traces of the action of this corrosive poison, being changed to an ashy blue color; but in the first two cases here noted, and the following one, in which a drachm was taken, the mucous membrane of the mouth and œsophagus was perfectly healthy. The principal effects of the poison were observed in the stomach, its mucous and muscular tissues (beginning at the cardiac portion, three inches and a half in extent by about the same in breadth) were converted into a gangrenous mass, having a corroded, ragged appearance, of a dusky-brown color, approaching to black. The mucous coat, to some extent, around this gangrenous portion, was of a brownish-red, but the lining membrane of the pyloric half of the stomach, except a few slightly red patches, was quite healthy. The bladder contained only half an ounce of urine, although none had been passed for twenty-four hours before death. The lungs were extremely congested.[1]

Other cases are mentioned, similar to those of Drs. Coale and Williams, in which, although the quantity taken has been large, and the signs of suffering in the stomach and the general symptoms presented the most violent character, the traces of a corrosive poison have been comparatively insignificant.[2] The period of survivance seems to make little difference in the aspect of the alterations found *post-mortem*. Dr. Coale's patient lived eleven days, and the two reported by Taylor between four and five days.

Ordinarily the change in the intestinal canal occurs as ecchymotic spots, accompanied by sanguineous effusions. The kidneys present a lively injection of the renal parenchyma, especially near the Malpighian bodies; the epithelial cells are deformed, granular, and partially destroyed, obstructing the *canaliculi*. The blood is ordinarily black and fluid. The alteration in the kidneys above described would harmonize with the albuminuria, which occurs in poisoning by mercury. It is rather remarkable that in cases of poisoning from the external application of mercury precisely the same traces of inflammation, ulceration, and gangrenous patches are observed as in cases from its internal administration. There is a case reported by Vidal[3] of the poisoning of a woman by the

[1] Wade, Lancet, June, 1848, p. 498.

[2] *Vide* Taylor on Poisons, and a case by Dr. Hodges, Am. Journ., Jan. 1855.

[3] Gazette des Hôpitaux, Juillet, 1864.

application of the acid nitrate of mercury as a lotion, instead of a liniment, to the cutaneous surface of the whole body. The internal surface of the stomach was red, the vessels were injected, and there were ecchymotic spots. The same ecchymoses were observed under the mucous surface of the bladder, and in the whole length of the intestines. The blood was black and fluid. In the microscopical examination the renal parenchyma was found very much injected, especially in the vicinity of the Malpighian bodies; the epithelial cells were deformed, granular, and partially destroyed. M. Flandin, by chemical analysis, had recovered a sensible quantity of mercury from the tissue of the liver; the other organs showed no traces of the poison. It would seem, therefore, that like other poisons corrosive sublimate may cause a fatty degeneration of vital organs, thus destroying cell-life and functional activity of the organs, unless death shall supervene too early (within forty-eight hours); and that similar *post-mortem* appearances occur after absorption through the skin, as after its administration by the mouth, rectum, or vagina.

§ 221. It must be admitted that in these cases of renal disease, whether acute or chronic, the effects of mercurialization upon the system will be greatly increased, and may even cause serious, if not fatal consequences. The evidence, therefore, of albuminuria and casts of the renal tubes being present in the urine during life, as well as the *post-mortem* changes of the kidney tissue, will throw light upon the consequences of the administration even of small doses of mercury; and this evidence should not be omitted in a medico-legal investigation.

§ 222. Mr. T. W. Bogg[1] reports a case of poisoning by corrosive sublimate in a very puny infant, in which the *post-mortem* appearances were similar to those above described. He speaks, however, of the readiness with which this poison can be obtained in England, because the sheep farmers keep large amounts of corrosive sublimate on hand for use in lotions for sheep washing.

A case reported by M. Barthélemy[2] to the Société de Médecine Legale de France, of poisoning by bichloride of mercury (corrosive sublimate), shows not only that a single dose of less than eleven grains (0.70 grm.) may cause a fatal subacute poisoning in the space

[1] Lancet, Dec. 21, 1878.

[2] Annales d'Hygiène, July, 1880, p. 337.

of nine days, but that the most serious lesions may occur in the kidneys. The immediate symptoms in this case following the ingestion of the poisoning were vomiting, stomatitis, salivation (expectoration of saliva and clots), deep-seated eschars, and ulceration of the mucous membranes, abundant alvine discharges, and collapse, but no suppression of urine, though there was albuminuria on the fifth day. The autopsy showed fluid blood, absence of lesions in the liver, and a granular and fatty degeneration of the kidneys, the tubes of which were choked up with fat. A second case was reported at the same meeting of the society, where the usual *post-mortem* lesions were supplemented by the same congestion and fatty degeneration of the kidneys, though the structure of the liver was normal in appearance.

§ 223. *Chemical Examination.*—We take the following lucid exposition of the chemical analysis for corrosive sublimate from Dr. Guy's work on *Medical Jurisprudence.*

"*Tests.*—We may have to examine the poison in the solid form, in solution, and in organic liquids.

"*Corrosive sublimate in the solid form.*—On the supposition that we are ignorant of the nature of the substance submitted to analysis, we first heat a small quantity on platinum foil. It is completely volatilized. It may, therefore, be arsenic, corrosive sublimate, or calomel. The great solubility of corrosive sublimate in water distinguishes it at once from the other two substances. The addition of a few drops of liquor potassæ places the nature of the substance beyond a doubt. Corrosive sublimate is changed to a yellow color, while arsenic undergoes no change, and calomel is turned black. We may obtain still further assurance by the following tests: 1. Hydrosulphuret of ammonia changes the powder to a black color. 2. A solution of iodide of potassium turns it to a bright scarlet. 3. Moisten a clean rag with dilute muriatic acid (one part of the acid to two of water), sprinkle upon it a small quantity of the powder, and rub it on a clean plate of copper. A silvery stain is formed, which is readily volatilized by heat. 4. Mix one part of the poison with three or four parts of calcined carbonate of sodium; place the mixture in a reduction tube, and apply the heat of a spirit lamp, having previously dried the upper part of the tube. A ring of globules will be formed on the cool sides of the tube.

§ 224. "*Corrosive sublimate in solution in water. Sulphuretted hydrogen.*—On the supposition that we are ignorant of the contents of a liquid submitted to analysis, we test for a base by sulphuretted hydrogen. Corrosive sublimate is one of those which yields a black precipitate, first giving a milk-white appearance to the liquid. With liquor ammoniæ it gives, in common with lead and bismuth, a white precipitate, but with liquor potassii, a yellow (the hydrated peroxide). By this we recognize a per-salt of mercury. The supernatant liquor contains chloride of potassium, and, if we add to it nitrate of silver, we obtain the white chloride of silver, which proves that the salt of mercury is a chloride. This precipitate, being collected, washed, and dried, and heated in a reduction tube, with carbonate of sodium, gives a well-defined ring of mercury."

This precipitate is also insoluble in hot dilute nitric acid, which enables us to readily distinguish it from the other black precipitates produced by the same reagent, such as lead, bismuth, copper, and silver; it is, however, readily soluble in nitro-muriatic acid (*aqua regia*), which reconverts it into the form of corrosive sublimate.

The following are additional tests:—

"*Protochloride of tin.*—A solution of this substance throws down a white precipitate, turning rapidly to gray, and from gray to black. This consists of minutely divided mercury, from which the supernatant liquor may be decanted, or separated by filtration. On introducing into the tube containing this precipitate a plug of blotting-paper, and pressing it firmly against the bottom of the tube, the globules are made to coalesce, so as to form a drop of mercury.

"*Metallic test (Reinch's test).*—Acidulate the liquid with a few drops of muriatic acid, and introduce a narrow slip of clean copper. A gray film will be formed on the surface of the metal. This, being carefully dried, may be introduced into a reduction-tube, and heated with the flame of a spirit-lamp. A ring of metallic globules will be deposited on the upper part of the tube. Pure tin, zinc, or silver, may be substituted for copper. The latter is to be preferred to any other metal.

"*Galvanic test.*—Take a narrow strip of sheet zinc of a size convenient for introduction into a reduction tube; moisten it, and take up as much gold leaf as will adhere to it. Introduce this into the solution, slightly acidulated with muriatic acid; the gold will soon be covered with a gray film. Remove it from the solution, and dry

it carefully in the heated air, above the flame of a spirit-lamp. Introduce the dried metal into a reduction-tube, and apply the flame of a spirit-lamp. A ring of metallic globules will be formed. This test is one of extreme delicacy, and will give a characteristic result, when all other tests fail. It is that which should be preferred for the discovery of very minute quantities of the poison. The metallic deposit may be readily obtained by placing a drop of the acidulated solution on a surface of clean copper or gold, and touching the moistened metal with a fragment of zinc or iron. Dr. Wollaston once employed a key and a sovereign for this purpose. The acid in combination with the mercury may be shown to be the hydrochloric, by testing the fluid from which the mercury has, by any of the foregoing methods, been precipitated; on the addition of the nitrate of silver, we obtain a white precipitate, the chloride of silver, which is insoluble in nitric acid.

"*Corrosive sublimate in organic liquids.*—As the poison is very soluble, it is rare to meet with it in a solid form. But when it has been taken in mass, it may sometimes be separated, by merely stirring the liquid, at the same time adding, if it be very viscid, distilled water. The corrosive sublimate, from its great weight, will subside, and may be collected. As the poison is decomposed by the secretions of the body, by the mucous membrane, and by several articles of food, it might not be found in solution in the stomach, even though no antidote had been given. We must, therefore, expect to find it in one of two states; in solution, or in combination with the solid contents of the stomach. In the former case we procure a clear liquid by diluting with distilled water, boiling if necessary, and filtering. In the latter case, one of two processes may be adopted. We may boil the solid matters in distilled water, and in this way bring the soluble salt of mercury into solution; or, if the solid matters treated in this way yield no trace of mercury, in consequence of the soluble salt having been decomposed, evaporate to dryness, and digest the dried residue in warm nitromuriatic acid. The insoluble compound of mercury is thus reconverted into the soluble bichloride. This acid liquor must be evaporated to dryness, and the residue be dissolved in distilled water, and filtered." The corrosive sublimate may now either be dissolved out by ether, or at once tested by the protochloride of tin, or by the galvanic test.

§ 225. A *new* test has been proposed. If a strong solution of

iodide of potassium be added to a minute portion of any of the salts of mercury, placed on a clean bright plate of copper, the mercury is immediately deposited in the metallic state, appearing as a silvery stain on the copper, which cannot be mistaken, as no other metal is deposited by the same means. By this method, it is said, corrosive sublimate may be detected in a drop of solution, unaffected either by caustic potash or iodide of potassium. In a mixture of calomel and sugar, in the proportion of one grain to two hundred, a distinct metallic stain will be obtained with one grain, which contains $\frac{1}{200}$th of a grain of calomel; in like manner $\frac{1}{400}$th of a grain of peroxide of mercury may be detected, although the mixture of sugar is not in the least colored by it.

With the preparations of mercury in the undiluted state, this process acts with remarkable accuracy; the smallest possible quantity of calomel or peroxide of mercury, such as would almost require a magnifying glass to perceive, placed on copper and treated with iodide of potassium, will give a distinct metallic stain. This single precaution must be observed: the liquid to be examined should be concentrated by evaporation.[1]

§ 226. Corrosive sublimate can be distinguished from calomel by its solubility in water, alcohol, and ether, by its reactions with sodium or potassium hydrates (yellow precipitate), with potassium iodide (salmon-colored or scarlet precipitate), and with ammonia water (a precipitate white with corrosive sublimate and black with calomel).

§ 227. *Isolation from organic mixtures.*—Corrosive sublimate and all of the other compounds of mercury, as well as metallic mercury itself, can be detected in organic liquids and tissues, as far as their mercurial nature is concerned, by the same process as was recommended in the case of arsenic (see § 171). After removing the organic sulphur compounds and any sulphide of arsenic or antimony which may be present by dissolving them with the mixture of ammonia water and ammonium sulphide, the sulphide of mercury will be left as a solid black precipitate upon the filter, since it is insoluble in the ammonia solutions; this black precipitate should then be treated with hot dilute nitric acid, which will dissolve any sulphide of lead, copper, silver, or bismuth if

[1] Pharm. Jour., Feb. 1852.

present, but leave the sulphide of mercury which may then be dissolved by nitromuriatic acid (*aqua regia*), the solution evaporated to dryness, the residue dissolved in water, and this solution, which will be one of corrosive sublimate, may be tested by any or all of the above tests.

In legal cases a globule of mercury obtained by the protochloride of tin test, by Reinsch's test, or by heating in a tube with sodium carbonate, should always be taken into court as one of the *corpora delicti*, since its fluid and metallic nature can always be recognized by the jury.

§ 228. Where corrosive sublimate has undoubtedly been the cause of death, it has not always been found in the body of the deceased. Thus, in a case reported by Dr. Wegeler, of a young man who poisoned himself with three drachms of this substance, and died on the sixth day thereafter, none of the poison could be detected in the stomach or intestines.[1] In another, by Dr. Taylor, where two drachms were swallowed, and the man died in four days, no mercury was detected in the stomach or tissues.[2]

Orfila (the nephew of the distinguished toxicologist) undertook numerous experiments for the purpose of ascertaining what length of time was required for the disappearance of certain poisons from the system. With respect to corrosive sublimate, he states, that when it has been administered for some time, it will generally disappear from the organs in eight or ten days, and he found it but once on the eighteenth day after its discontinuance. An individual had undergone a course of treatment with corrosive sublimate, and died four months after ceasing the course. He was poisoned with a mercurial preparation. On analysis, mercury was found in his organs. Hence, according to this author, the mercury could not have been derived from the preparations taken four months before death. He also says, that if a man survives fifteen days after being poisoned with corrosive sublimate, it is quite probable that the experts will find no trace of mercury. They will, however, commit a gross error, if they conclude from this, that there has been no poisoning.[3]

§ 229. Any mercurial preparation may induce analogous symp-

[1] Canstatt's Jahresbericht für 1846, Bd. v. p. 81.

[2] Op. cit., p. 377.

[3] Am. Journ. Med. Sci., from Comptes Rendus, Jan. 15, 1852.

toms to those which have been described under the head of corrosive sublimate, as acute, subacute, or chronic poisoning. The corrosive effects upon the mouth and gullet may, it is true, be absent, but the ulceration, salivation, and inflammatory action upon the *primæ viæ* are usually present. By some physiologists, principally Prof. Cl. Bernard, it has been assumed that the mercury is changed by the secretions into corrosive sublimate, but the truth of this theory has not yet been substantially proved.

The principal mercurial preparations are—

Calomel, or mild chloride of mercury (Hydrargyri Chloridum mite, U. S. P.). Cyanide of Mercury (Hydrargyri Cyanidum, U. S. P.). Red Iodide, Biniodide of Mercury, or Mercuric Iodide (Hydrargyri Iodidum Rubrum, U. S. P.). Green Iodide of Mercury, Protiodide of Mercury, Mercurous Iodide (Hydrargyri Iodidum Viride, U. S. P.). Yellow Oxide of Mercury, Yellow Mercuric Oxide (Hydrargyri Oxidum Flavum, U. S. P.). Red Oxide of Mercury, Red Precipitate, Red Mercuric Oxide (Hydrargyri Oxidum Rubrum, U. S. P.). Yellow Subsulphate of Mercury, Basic Mercuric Sulphate, Turpeth Mineral (Hydrargyri Sulphas Flava, U. S. P., 1870; Hydrargyri Subsulphas Flavus, U. S. P., 1880). Mercuric Sulphate (Hydrargyri Sulphas, Br.). Red Sulphide of Mercury, Red Mercuric Sulphide, Cinnabar, Vermilion (Hydrargyri Sulphuretum Rubrum, U. S. P., 1870; Hydrargyri Sulphidum Rubrum, U. S. P., 1880). Mercury, Quicksilver (Hydrargyrum, U. S. P.). White Precipitate, Mercurammonium Chloride, Ammoniated Mercury (Hydrargyrum Ammoniatum, U. S. P.). Gray Powder, Mercury with Chalk (Hydrargyrum cum Cretâ, U. S. P.). Sulpho-cyanide of Mercury (Hydrargyri Sulpho-cyanidum). Nitrate of Mercury (Hydrargyri Nitras). Mercuric Methide.

XIV. *Nitrate of Mercury.*

§ 230. A case of homicidal poisoning, attributed to the administration of a portion of this salt in a pudding, is related in Henke's *Zeitschrift* for 1849. The symptoms were very similar to those of poisoning by corrosive sublimate; the man survived five days in great agony, and the *post-mortem* inspection revealed softening and congestion of the mucous coat of the intestines and stomach. The most curious feature of this case was, that mercury was found

in the metallic state in the stomach and intestinal canal, and had been voided also with the discharges during life. This circumstance was attributed by the examiners to the readiness with which the acid, in this combination, is separable from the base, especially under an elevated temperature. A case of suicide by the acid pernitrate is reported by Mr. Bigsley, in the *London Medical Gazette*.[1]

A case is also reported by M. Vidal,[2] of accidental poisoning by the application of this mercurial salt to the skin.

The concentrated salt is used as a caustic in surgery, and is exceedingly active. It is stated that symptoms of mercurial poisoning have arisen from its use in this form.

§ 231. Other salts of mercury, such as the *white* and *red precipitates*, *cinnabar*, the *cyanide*, and *turpeth mineral* are all poisonous, but it is not necessary to enlarge upon them in this place. They act as irritants or corrosives, and the *post-mortem* appearances in the cases reported are not unlike those found in poisoning by corrosive sublimate.

§ 232. The *sulphocyanide* of mercury, which has been used as a toy called "Pharaoh's Serpents," when ignited swells up to an enormous size and gives off poisonous mercurial fumes. The action of this salt is especially described by Prof. Cl. Bernard[3] as destroying by its contact with the muscular tissue the inherent muscular irritability, and directly attacking the nervous system. Thus it arrests the heart's contraction, and causes death by this paralysis of cardiac pulsations. Hence after death by this poison, there should be absence of rigor mortis. According to this physiologist Treviranus, Gmelin, and Tiedeman, as well as himself, have proved the presence of this salt in normal saliva, but in very small amount.[4]

§ 233. *Chemical examination.*—This depends chiefly upon the detection of the mercury, which may be accomplished in the same manner and by the same tests as described above in connection with corrosive sublimate. Mercuric nitrate also responds to the tests for nitric acid or nitrates (see § 52), and the sulphates respond to the tests for sulphuric acid or sulphates (see § 43). The oxides, sul-

[1] Vol. vi. 329.

[2] Vide § 205.

[3] Substances Toxiques, Paris, 1857, p. 356 *et seq.*

[4] Op. cit., p. 350.

phides, white precipitate, and calomel, are all insoluble in water or alcohol. The sulphocyanide of mercury also responds to the tests for sulphocyanic acid, the principal one of which is the formation of the blood-red solution, when treated with a solution of sesquichloride of iron, similar to that caused by the same reagent with a solution of an acetate or meconic acid, a constituent of opium, but readily distinguished from the former by the fact that it is not bleached by a slight excess of hydrochloric acid, and from the latter, by the fact that it is bleached by corrosive sublimate and nascent hydrogen, while the meconate of iron is not bleached by either of these.

The separation of all of these compounds from organic tissues and fluids, so far as the mercury is concerned, is by the same method as described above under corrosive sublimate (see § 224[1]).

XV. *The Salts of Lead.*

§ 234. *Sources.*—The principal sources of acute lead poisoning are the two acetates of lead, sugar of lead, and the basic acetate of lead (Goulard's extract), and the carbonate of lead or white lead. The normal acetate, or sugar of lead, is largely used in the arts and often in the household, and has given rise to numerous cases of accidental poisoning. It has a sweet taste, whence its popular name, is readily soluble in water and alcohol, and has a white crystalline appearance. It has through ignorance been used for sweetening wines, and the writer is familiar with several cases of acute poisoning due to its having been used by mistake for sweetening cider. Sugar of lead is made by dissolving oxide of lead in acetic acid and crystallizing. The basic acetate, or Goulard's extract, is made by boiling a solution of sugar of lead with an excess of the oxide; it is soluble in water, and its solution forms a very valuable reagent in the chemical laboratory. The carbonate of lead, or white lead, is very largely used for painting, and, although insoluble in water, may, if swallowed, give rise to acute poisoning on account of its solubility in the gastric juice. Chromate of lead (chrome yellow) has also caused acute poisoning, but will be referred to below under a separate heading, since the chromic acid of the compound appears to be an important factor in producing the symptoms. The numerous

[1] Page 226.

sources of chronic lead poisoning will be enumerated in speaking of chronic lead poisoning (see § 240).

§ 235. *Symptoms.*—In general the poisonous effects of acetate of lead arise gradually and become slowly developed after its long-continued use. When taken in a large quantity at once, it is capable of producing symptoms analogous to those caused by other irritant poisons. It is eliminated from the system with the urine, the perspiration, and the milk. The constitutional effects of lead have been thus described by Tanquerel des Planches:—[1]

1st. Saturnine coloration of the gums, of the buccal mucous membrane, and of the teeth. A narrow, leaden-blue, or slate-blue line, from one-twentieth to one-sixth of an inch in breadth, is formed on the margins of the gums nearest to two or more teeth (usually the incisors) of either jaw. The inner part of the lips and cheeks is sometimes stained blue. The blue discoloration is supposed to depend upon the formation of the sulphide of lead.

2d. Saturnine taste and breath.

3d. Saturnine jaundice.

4th. Emaciation, most evident in the face.

5th. Slowness, smallness, and irregularity of the pulse.

The true saturnine diseases which follow may either exist alone or be complicated with each other. They are: 1st. Lead or painter's colic. 2d. Lead rheumatism or arthralgia. 3d. Lead paralysis, often accompanied with loss of sensation in the part affected. 4th. Disease of the brain—*encephalopathia saturnina*—manifested by delirium, coma, or convulsions, and the loss of one or more senses.

Acute poisoning by lead has a different character from the chronic form. The metallic lead is poisonous to animal life, no matter in what form it may be swallowed; the rapidity in the appearance of the symptoms, however, depends upon the form in which it may be administered, because some of the salts of lead are more easily absorbed into the system than others. All the compounds of lead may, therefore, cause acute poisoning, though Dupasquier at one time denied this fact; more recent experience shows that even the insoluble salts and metallic lead itself can, after ingestion, cause poisoning, the rapidity and intensity being in proportion to the

[1] Traité des Malad. de Plomb, Paris, 1839.

degree of solubility in the secretions of the body, for instance, chloride of lead combined with common salt causing the most rapid poisonous consequences.

§ 236. It is difficult to specify the minimum fatal dose of lead, because the size of the dose required to produce poisoning varies with the individual, probably agreeing, as in other poisons, with the rapidity with which this toxic agent is absorbed and eliminated. Its elimination is by way of the intestines and kidneys, and in case of a diarrhœa coexisting with the administration, the drug passes out too quickly to produce its peculiar physiological effects upon the tissues of the body. Lead salts are often used by physicians in cases of diarrhœa, and, though sometimes evil consequences may follow this use, the occasions are rare. A case is recorded[1] in which a young man experienced serious consequences from taking a little more than two grains (15 centigrammes) of acetate of lead in three days, and an adult[2] died after drinking half a litre (a little over one pint) of wine out of a bottle which had been cleansed with a few grains of metallic lead. Of course the gravity of the danger and the severity of the poisoning depend upon the amount of poison absorbed, and not upon the amount swallowed. So, also, the form in which the lead is administered, as above remarked, affects the rate of absorption, as a mixture of lead and common salt is more readily absorbed than the lead itself; the subacetate of lead is more readily absorbed than the acetate. The degree of concentration also affects the size of the necessary fatal dose, since the greater the concentration, the greater will be the astringent action of the drug, and the less will be the amount absorbed at any one time. According to the experiments of Orfila, de Gaspart, and of R. Moreau,[3] acetate of lead in the dose of half a gramme (8 grains) injected into the veins of a dog will destroy its life in twenty-four hours, and a single dose of ten or twenty centigrammes (one and a half to three grains) administered in the same way will kill it in eight days.

The cases in which this drug has been used with criminal intent are very rare, and in each case the acetate of lead was used mixed

[1] Nouveau Dict. de Méd. et de Chirurgie, tome xxviii. p. 307.

[2] Ibid.

[3] Recherches experimentales et cliniques sur l'empoisonnement aigue par le plomb, Thèse de doctorat, Paris, 1875.

with food or drink. One case[1] occurred in France, and two[2] in England. It is more often by accident or for suicidal purposes that mixtures containing lead are the cause of death. The officinal dilute solution of subacetate of lead, containing three per cent. of the subacetate, commonly called Goulard's lead-water, has thus accidentally been mistaken for water, or, in one recorded case, when used as a wash, been sucked off of sore nipples by a child, and with fatal consequences in eleven days.[3] Death has been caused by drinking a solution of lead salt from mistaking a bottle of lead-wash for a cathartic. It has also caused serious consequences where used to color children's toys. Finally, death has occasionally followed the medicinal use of lead, when used to counteract diarrhœa or the spitting of blood in phthisis, but this always in case of enfeebled persons.[4]

§ 237. After swallowing the lead poison, there is, on the part of the victim, perceived a sweetish taste, then an astringent or metallic after-taste; soon after there is often a sensation of burning, distress, more or less distinct, in the throat, and extending down the gullet even to the stomach; at other times there is simply a sensation of weight at the pit of the stomach. The tongue, often slightly swollen, is generally white, and is covered with small, prominent points. Nausea and vomiting may then occur, at first of a limpid and clear liquid which contains lead. Later on the vomited matter contains mucus and little white patches due to the salts of lead. The chromate of lead alone colors the vomited matter yellow. Then very acute gastro-intestinal colicky pains occur, accompanied by a contraction of the intestinal tube, and sometimes by meteorism, a tympanitic resonance of the abdomen. These pains are often associated with constipation of the bowels, though sometimes there is a diarrhœa, in which the stools appear black from the combination of lead with the sulphuretted hydrogen gas of the intestines producing sulphide of lead. Along with these active symptoms the limbs feel heavy, and there is a pallor of the countenance with livid lips.

The bluish-black line along the margin of the gums may not be

[1] Ponchou, in 1842.

[2] Central Criminal Court, 1844, and Chelus Ford, Summer Assizes, 1847.

[3] Taylor, op. cit.

[4] Fouquier, Léridon, and Ruva.

present in acute poisoning, and may be absent in chronic lead poisoning, but this subject will be more thoroughly discussed when we treat of chronic saturnism. If the poisoning is severe, the respiration becomes stertorous and asphyxia is imminent, accompanied with a painful hiccough. Vertigo then comes on, trismus (local lockjaw), and epileptiform convulsions may supervene, preceding a comatose condition (due to a true inflammation of the brain), which is followed by death in two or three days. The intellectual faculties may occasionally remain intact up to the last moment of life.

In cases of acute lead poisoning terminating in recovery, a febrile condition supervenes after the symptoms of irritability and its succeeding constitutional depression. Blackish spots of sulphide of lead are developed upon the skin; the epigastric pain and distress gradually diminish, and convalescence proceeds slowly, while the heaviness, digestive disturbances, feebleness, and anæmia from the saturnine cachexia may persist for a long time.

§ 238. *Post-mortem appearances.*—In acute poisoning these are neither constant nor characteristic: there is usually a slight and superficial inflammation of the mucous membrane of the stomach (which in the latter may be somewhat thickened) of a grayish color, and this membrane may be even softened and eroded. Dr. Taylor considers that the corrosive action belongs to a neutral salt, and not to the poison combined with an acid. The white shreds adhering to the mucous membrane, described by Orfila to be peculiar to acute poisoning by lead, are not always present, and are difficult of recognition. In two cases where serious cerebral symptoms were observed, Bergeron and Tardieu reported lesions due to true inflammation of the encephalon by the presence of lead. These appearances were a dead white discoloration of the brain with firm consistency, and a flatness and effacement of the cerebral convolutions. Dr. Taylor[1] does not consider the acetate, or sugar of lead as an active poison, and states that it has been given in medical practice in considerable doses without any serious results. Sir R. Christison states that he has given it medicinally, in divided doses, to the amount of eighteen grains daily for eight or ten days without observing any unpleasant symptoms, except once or twice a slight colic. Dr. Taylor, however, admits that in large doses, one or two

[1] Op. cit., p. 392.

ounces, symptoms which we have above described in detail will appear. English toxicologists and physicians have been slow to admit that metallic lead is a poison, and twenty-three years ago (1860) many of their prominent teachers would not believe in a disease called lead poison; many cases attributed by French and American writers to acute poisoning by lead were explained by English authorities as representing organic lesions of the nerve centres without being caused by the presence of lead in the animal tissues. Christison[1] states: "It is probable that all the preparations of lead are poisonous except the metal, and perhaps also the sulphuret. . . . . Every preparation of lead will excite colic and palsy when long and frequently introduced into the body. The metal, if it was not liable to tarnish, would indeed be an exception. But, as it is constantly covered with more or less carbonate of lead, it acts as well as the rest—which is proved by tradesmen being liable to suffer, although they have merely to handle the metal." At the present time all the standard English medical authorities admit that acute poisoning may be caused by salts of lead, and most of the clinical writers describe symptoms of acute poisoning by the metal, in spite of the contrary statement of Christison and that of Taylor[2] "that lead appears to exert no directly poisonous action when swallowed in the metallic state. Under exposure to air, water, and carbonic acid, it is easily converted into a poisonous white salt—carbonate of lead. In the interior of the body, however, we have no reason to believe that this metal produces any noxious effects," etc. etc. The cases reported by him where lead was swallowed, and passed from the bowels with loss of substance, in one case ten grains in about a week, does not prove the fact that metallic lead is not poisonous, since the bowels would be able for a short time to slowly eliminate the metal almost as rapidly as it dissolved, and the solid particles swallowed may pass out of the intestines within a short time. This fact would be more readily believed if the bowels were not constipated by the presence of the lead. The clinical experience in hospitals and in private practice shows that metallic lead can be absorbed when in the stomach or tissues of the body, and give rise to poisoning of either subacute or chronic form.

[1] Treatise on Poisons, Ediuburgh, 1832, p. 488.

[2] Op. cit., p. 390.

§ 239. An instance of recovery from an ounce and a half of sugar of lead, swallowed by mistake, is related by Dr. Taylor.[1] The woman fell ill almost directly, had a nauseous metallic taste in her mouth, with a burning heat in it, the throat, and the stomach. On taking some water to wash away the taste, vomiting was brought on. The mouth became very dry; she had great pain at the pit of the stomach, and excessive vomiting. Two hours afterwards, she felt sleepy and stupid—alternately perspiring and shivering; she complained of violent colic, which was relieved by pressure. She had also cramps in the thighs, great languor, and numbness over the whole body with giddiness. The gums were tender, and had, apparently, a blue line on their edge; there was some salivation, and the breath was foul. There were other symptoms such as have been before detailed. She was relieved by treatment in a few days. Several other cases of the same kind, which it is needless to describe, are reported. They all recovered; as undoubtedly it must be admitted many cases may recover, provided a sufficient amount of the poison pass through the alimentary canal without being absorbed into the tissues of the body. An eminent toxicologist presents in brief the symptoms of acute saturnism. It results from the ingestion of a soluble salt of lead or of some drink with which it has been mixed accidentally in considerable quantity, such as cider, wine, or vinegar. Very soon after, nausea, not always accompanied with vomiting, supervenes, followed by very acute pains in the abdomen; sometimes there is diarrhœa, but more often obstinate constipation. The countenance becomes pale and, oftentimes from the moment of attack, there may be noticed a bluish line upon the borders of the gums. The voice becomes indistinct, a painful hiccough supervenes; syncope and terrible convulsions precede a state of comatose stupor, which may endure for two or three days and terminate in death. (Tardieu.)

The only diseases likely to be confounded with the acute poisoning are simple enteric or gastric fever. The lead poisoning can be distinguished from these by the much more acute pains in the abdomen, the state of the mouth, the line on the gums, though it may frequently be absent, the fetor of the breath, the serious nervous disturbances, especially their rapid progress, the tendency to constipa-

[1] Op. cit., p. 348.

tion, and the relief of the pain by free catharsis, and often by firm pressure applied to the abdomen.

Subacute and Chronic Poisoning.

§ 240. *Sources.*—When it is remembered how varied are the means for the employment of lead in the industrial pursuits, it may not be surprising to find that chronic lead poisoning is very common. The number of employments which may be the source of the slow absorption of lead, and consequently give rise to chronic lead poisoning, is very great. The following extract from an article contributed by the writer[1] will show how universal is the danger of the absorption of small amounts of lead, and how difficult it is sometimes to trace the poisoning to its source.

"The subject of chronic lead poisoning is becoming more and more important every year on account of the gradually increasing use of lead in the arts, which augments the number of sources of poisoning and renders the detection of the source in some cases exceedingly difficult. . . . . A lawsuit has recently occurred, in which a white-lead company has been sued by a workman, who had contracted the disease while in the company's employ.

"Of the numerous sources of chronic lead poisoning, by far the most important, and those giving rise to the greatest number of cases, are the use of lead carbonate (white-lead) in painting, and the use of lead, or lead-containing, pipe for the conduction of drinking water.

"With regard to the use of white lead as a pigment, the greatest danger arises during the process of its manufacture, and the utmost care should be taken by the workmen to prevent the inhalation or ingestion of the lead-dust, those portions of the process, in which the dust is disseminated throughout the air of the work-room, such as the grinding and packing, being the most dangerous. With constant care and watchfulness on the part of the workman little fear need be apprehended of poisoning, and a new workman should always be informed of the danger attending the employment, and instructed with regard to the precautions to be taken to prevent poisoning. These precautions are chiefly the following:—

"1. The use of respirators in all work-rooms which are liable to

[1] The Sanitary Engineer, New York, Aug. 3, 1882, page 185.

be dusty. Any lead-containing dust coming in contact with the mucous membrane of the mouth, nose, or throat is naturally swallowed, dissolved in the gastric juice and absorbed. Swallowing is not, however, necessary to absorption, since the lead compound can be absorbed from any surface, even through the unbroken skin, although it is extremely slow in this way and may be prevented by strict attention to cleanliness.

"2. No article of food or drink should be brought into a work-room. All food and drink should be taken where there is no possibility of its being contaminated with lead dust. Before partaking of any drink the workman should thoroughly rinse out his mouth and throat, and should wash his moustache, if he have one.

"3. The workman should not eat any food until he has thoroughly washed himself and changed his clothing. In some European manufactories the workmen are obliged to change all of their clothing, before either entering or leaving the work-rooms, suitable ante-rooms being provided for the purpose, and to thoroughly wash themselves before putting on their clean clothing.

"4. Strict attention to cleanliness. All lead-containing dust should be removed from the skin upon leaving the work-rooms so as to prevent absorption through the skin which takes place upon long contact. Special attention should be paid to the hands, face, and head, no dust being allowed to remain in the hair or beard.

"In some factories sulphuric acid lemonade is provided for the workmen to drink. This may help to a certain extent in preventing or delaying the absorption of those lead compounds which are swallowed by forming with them lead sulphate, which is insoluble in water and acids, but it does not entirely prevent it, because lead sulphate is slightly soluble in the intestinal fluids. In one factory I have known of milk being provided for the workmen to drink, and, it was said, with excellent results in preventing lead poisoning.

"With regard to painters, many of whom are affected with lead disease (to such an extent that the lead colic has received the name of painter's colic), the principal precaution necessary is cleanliness, since they are exposed to danger arising from white-lead dust only while mixing the paint; but the same care should be taken as to cleanliness as in the case of white-lead manufacturers.

"The use of lead pipe for conducting drinking water is very extensive, and yet comparatively few cases of poisoning result from it.

This is due chiefly to the fact that certain hard waters cause a deposit of an insoluble coating consisting of lead sulphate and carbonate upon the inner surface of the pipe which prevents any further action of water upon it, and that the people generally have learned the necessity of thoroughly emptying from the pipe the water which has stood in it for several hours. All waters do not act upon lead pipe with equal readiness. As a rule, the purer the water, the more readily does it attack metallic lead. Therefore soft waters take up the most lead and hard waters the least. Hard waters, however, vary in this respect. If a hard water contains only lime and magnesian sulphates and carbonates without any alkaline sulphates or carbonates, it has no action upon the pipe except to deposit the insoluble coating upon its inner surface; if, however, alkaline salts are also present, the alkaline carbonate takes up a little of the lead salt, however hard the water may be.

"Lead cisterns should never be used for containing drinking water, nor, as a rule, should lead pipe be allowed to project beneath the surface of the water in a well, although in the case of most hard waters no serious consequences would result. It is better to use pure block tin pipe beneath the surface of the water, soldering it on to the lead pipe above the highest level of the water in the well. Then only the small amount of water, which is contained within the pipe, has an opportunity to attack the lead, and if the pipe is thoroughly emptied of the water which has stood in it for several hours, no serious consequences will result. Tin-lined lead pipe and tinned lead pipe are, in my opinion, more dangerous than pure lead pipe, since the galvanic action set up by contact of the two metals increases very much the action of the water on the lead.

"Although the above-mentioned are the two principal sources of chronic lead poisoning, they are by no means the only ones; these sources are so numerous that in certain cases the physician finds it very difficult, or even impossible, to trace the poison to its source. Any employment in which metallic lead or any compound of lead is used, is liable to cause chronic lead poisoning. Among the more common employments may be mentioned working in lead mines, or any mines in which plumbiferous ore is handled; working with metallic lead, as in the case of plumbers and type-setters; the handling of lead compounds, as in the case of glazers of porcelain and pottery ware, manufacturers of enamel, etc. The use of lead

chromate (chrome yellow) and red lead (minium) as pigments; the use of lead or any of its compounds in any cooking utensil, or article used for preserving food, especially acid fruits and vegetables, which are often put up in soldered tin cans. The acid contained in some of these—as tomatoes—may easily remove a dangerous amount of lead from the solder, if eaten frequently during a considerable period. The use of lead-containing enamel for lining iron-ware to be used for cooking, is very dangerous. I have personally known of several cases of acute and subacute lead poisoning resulting from cooking tomatoes in such enamelled iron-ware, and one fatal case in an infant, due to drinking milk which had been warmed in one of these vessels. This particular vessel is in my possession, and yields to water, to which a few drops of acetic acid have been added, when allowed to stand a few hours at the ordinary temperature, an amount of lead which might produce symptoms of poisoning in an adult. The addition of lead compounds to articles of food or drink —thus many yellow candies are colored with lead chromate, and many olive-green ones with a mixture of lead chromate and Prussian blue. Snuff has been known to be colored with lead chromate and with red lead. A not uncommon source of chronic lead poisoning is the cleaning of glass bottles with shot; the greatest danger arises in the case of champagne bottles, one or more shot sticking in the angle between the side and bottom of the bottle, owing to the carelessness of the bottle-washer. The carbonic or other acid of the wine, malt liquor, or cider readily removes a dangerous amount of lead from the shot. I have in mind a recent case of lead colic due to drinking bottled ale, the lead being derived from shot left in the bottles. Wine is sometimes sweetened by treating it with oxide of lead (litharge) or sugar of lead. The use of lead pigments for coloring children's playthings is very dangerous, the child being liable to put the toy in its mouth and suck off some of the poison. The use of spurious (lead-containing) tinfoil is another source of chronic lead poisoning; some of this foil consists chiefly of lead (one specimen examined contained about 98 per cent. of lead) and is used for wrapping tobacco, pressed meat, and other substances. There is one case of poisoning recorded of a clergyman who was in the habit of using *cachous* for a sore throat, between one and two boxes being consumed daily; finally, after diligent search for the cause of the poisoning, it was found that several milligrammes of

lead were contained in the foil surrounding the *cachous* in a single box. The habitual use of some hair-dyes is also a frequent source of lead poisoning, many of these compounds consisting of sulphur, sugar of lead, and some fluid, as glycerine, water, etc. Some cosmetics also contain a lead compound.

"The above enumeration shows how varied are the sources of chronic lead disease, and how difficult it often is to detect them. A case illustrating this is that of a newspaper editor, who was in the habit of sticking his clippings upon paper with red wafers which were colored with red lead, the wafers being moistened with the tongue. A limited epidemic of lead poisoning in Paris, was finally traced to a baker's oven, which was heated by burning painted wood, the lead oxide volatilized by the high temperature was deposited upon the bottom of the oven where it came in contact with the bread."

§ 241. In the slow form of lead poisoning, several days sometimes may elapse before the symptoms are noticed, but more generally it is after two or three months of sojourn in an atmosphere vitiated by the saturnine emanations, or of contact with lead, that the first symptoms of poisoning become apparent, and these cases furnish instances of what is known under the name of chronic poisoning. These are preceded by characteristic signs of saturnine affections, such as gradual pallor and emaciation, the skin (of the face especially) of a pale yellow (*subictérique*), in which the sclerotic coat of the eye participates. The serious symptoms commence with lead colic, followed by epileptic convulsions and a comatose or delirious condition. It is a matter of observation that from the commencement there exists a certain absence of the sensation of pain, sometimes of the whole surface of the body, but more especially of the arms and forearms. (Tardieu.)

When lead has been found in a suspected case of poisoning, it is of course important to show that the peculiar occupation or profession of the individual would not explain the presence of this metal in his organs. The experiments of the younger Orfila show that eight months is the shortest time of the sojourn of the lead in the liver, the intestines, and bones.

The symptoms which follow the introduction of the *carbonate* or other slightly soluble salts of lead into the body, are precisely similar to those already mentioned; occurring soon when the dose is large, and gradually when entering the system by water, wine,

cider, or other liquids which are apt to be impregnated with them, and also when inhaled by the lungs. The subject of chronic poisoning by lead in these ways is one which has indeed its interest for the physician, in its relation to medical police, and the facts relating to it are fully detailed in the comprehensive works on poisons and the treatises upon the practice of medicine.[1] The character of the poisoning differs so completely from that of the irritant poisons, that no mistake on this point can ever be made; the only embarassment which ever presents itself being the discovery of the particular way in which the lead has been introduced into the system.

According to Dr. Taylor,[2] if the patient recovers from the first symptoms of lead poisoning, the secondary effects often last a considerable time. The acute poisoning, unless recovered from, usually ends fatally within a week or a fortnight; if the symptoms are prolonged beyond this period, the form of poisoning assumes that of a subacute character. In the subacute form of poisoning, when the lead has been absorbed into the tissues, its presence seems to deteriorate their nutrition and functions; hence we see disturbances in the nervous sensations, muscular motions, and in the abdomen severe colicky pains and deficient alvine evacuations. The patient becomes pale, emaciated, and feeble in body, with a general feeling of lassitude, heaviness of limbs, but without mental hebetude. The presence of lead in the systemic circulation appears to destroy the red blood corpuscles, causing the sallow pallor of the countenance, and paleness of the mucous membranes; later on the metallic lead is deposited in the skin and external covering of the eyes and in the mucous membranes, causing a dusky countenance, dulness of the eyeballs, and pallor of the mucous surfaces generally. This appearance is illustrated in the case of those whose occupation necessitates

[1] Besides the chief authorities which may be consulted on this subject, are the following: Tanquerel des Planches, Traité des Maladies du Plomb (also translated by Dr. Dana, of Boston); Dr. Burton, Med.-Chir. Trans., vol. xx.; Gueneau de Mussy, Dub. Quart. Journ., vol. vii.p. 405; Dalton, Am. Journ. Med. Sci., Oct. 1849; Alb. Smith, Month. Journ., March, 1853; Bois de Loury, Rev. Méd., Juillet, 1852; Alderson, Lancet, July, August, Sept. and Oct. 1852. For some cases of poisoning by visiting cards (glazed with lead), *vide* Med. News, 1854; or Med. Times and Gaz., July, 1854. Eichmann. Reynold's System of Med., London, 1876. Practice of Medicine, Tanner, Phila., 1866. Cyclopedia of Medicine, von Ziemssen, American Edition, New York, 1881, vols. vii.,viii., xi., xii., xiii., xiv., xv., xvii., xix. Nouveau Dict. de Méd. et Chir., Paris, 1880, vol. xxviii.

[2] Op. cit.

the handling of the metal, or respiring an atmosphere in which various forms of lead are used in manufactures or in the arts, such as painting, plumbing, type-setting and the like; in plumbing the heating of the lead evolves fumes of the oxide, often in illy ventilated apartments, and thus exposes the workman to the dangers of acute, subacute, or chronic lead poisoning. That the same individual may practise his art or labor for a long time without showing symptoms of poisoning, and finally at an unexpected time be overpowered by the lead is not strange, since either he may eliminate the poison so rapidly by proper attention to the action of the bowels, or by being fortunate enough to escape the risk of the absorption of a poisonous amount by working in the open air, and by cleanliness of person. One instance is known to the writer where a plumber of several years' experience in his trade was found dead in a water tank he had been lining with lead, and with sufficient evidence that his death was caused by lead absorption. His health had previously been excellent. We cite among cases reported the following[1] instance of poisoning from the use of lead in the arts. There is one part of the process of pottery manufacture, where the articles are briskly rubbed together, causing the presence of lead particles in the air of the factory. A workman of sixteen years of age working in one of these factories was admitted to the Royal Infirmary at Glasgow with symptoms of acute lead poisoning, marked by emaciation, anæmia, and an exhausted appearance, wrist drop, and wasting of the muscles of both arms, the contractile power of the extensor muscles of both arms being absent; his gums presented the blue line peculiar to lead poisoning, the abdomen was quite natural in appearance, soft and elastic, and pressure caused no pain. His pupils were dilated, though the sight was not much disturbed. The pulse was rather weak and 70 to the minute. His urine had a specific gravity of 1012, neutral, of pale color, without sediment and without albumen; the phosphates appeared on heating, but otherwise the physical examination yielded normal results.

The patient was given the usual treatment of iodide of potassium, which was, however, discontinued after three days, the bromide of potassium being substituted. On the morning of the ninth day after admission he had coma, and in a few moments exhibited

[1] Lancet, Feb. 26, 1881.

clonic convulsions extending over the whole body; he was unconscious with dilated pupils, foamed at the mouth, had retraction of the abdominal walls, involuntary micturition, with a pale, clammy face, and rapid and forcible action of the heart. The convulsions soon ceased, but he remained unconscious in a comatose state. The next day his temperature became 102°, and on the tenth day his urine became albuminous, but no lead could be found in it.

A *post-mortem* examination showed nothing particularly abnormal about the head, but the muscles of the forearm were examined microscopically, and the extensors were found to have undergone atrophy of the muscular fibres, the transverse striæ being very much less marked in these than in the flexor muscles. The paralyzed muscles had also more nuclei than in the normal extensor muscles, the fibres of the former being also very much narrower.

§ 242. We may have in a case of subacute poisoning by lead either a predominance of the symptoms referable to the nervous centres, or of those referable to a disease of the kidneys, since, like many other poisons which are eliminated by the kidneys, the effort of elimination may cause structural decay of these organs. It should be observed here that where there is suspicion of lead poisoning, cathartic treatment, not by salines except Epsom salts, since these favor lead absorption, helps to hurry the discharge of the metal and its salts through the intestinal canal; and that when the salts of lead are used as astringents in diarrhœa, unless this diarrhœa be checked, absorption in the intestinal canal is prevented owing to the rapid escape of the lead with the fecal discharge. Dr. Headland[1] speaks of the danger of using in medicinal preparations a combination either of opium and acetate of lead on account of the chemical decomposition, or of alum and lead on account of the increased astringency and risk of lead absorption from its retardation within the intestinal tract. Either of these agents combined with lead would naturally tend to detain the metallic salt in the intestinal canal.

The differential diagnosis between subacute and chronic lead poisoning is not an easy matter, since liability to both forms may occur to the same individual, and the difference depends upon the amount of the poison absorbed within a given time. This will be evident

[1] On the Action of Medicines, Phila. 1868, p. 299.

from the very nature of the case, since it may take a longer exposure in one case than in another to receive a given dose. So also a person in active health and of a full vigorous habit of body, by which we mean a normal blood supply and an active nutrition of tissues, may resist the absorption for a long time, the emunctory organs discharging a large amount of lead. This reasoning will explain why, as is stated farther on in this article, the presence of lead in the urine is absent in the last stages of saturnine intoxication, the kidneys in this condition being unable to perform their function of elimination not only of lead, but of other deleterious substances from the blood and tissues.

§ 243. *Chronic* poisoning by lead, or *saturnine intoxication* or *saturnism*, as it is variously called, may follow exposure to the ingestion of any salt of lead, or exposure to the action of the metal itself by ingestion, by handling, or by means of inhalation, as has been before mentioned. E. Gaucher,[1] in an elaborate article on lead poisoning, sums up the results of his experience in the following conclusions: 1st. Nutrition in lead intoxication is considerably retarded as shown by a diminution in the solids of the urine, its specific gravity being diminished, the excretion of urea (which is the measure of changes caused by the metamorphosis of tissue), and that of the chlorides and phosphates being below the normal standard. 2d. During the active period of this intoxication, or, in other words, of the presence of lead in the system, a large number of the red blood globules or corpuscles are destroyed, which results in a saturnine icterus, or the presence of bile in the blood (ictère hémaphéique), and by the abundant discharge of the coloring matters of the blood with the urine. This deglobulation of blood and the retardation of nutrition of the tissues explains the early appearance of anæmia in those poisoned by lead. [The saturnine icterus is not due to the presence of bile in the blood and tissues.—EDS.] 3d. The urinary secretion presents two distinct phases, whose characteristics are opposed to each other; in the commencement of the lead poisoning, the urine is deficient in quantity, is concentrated and has a high color, while at the end of a certain time after poisoning, it becomes more abundant and of a lighter shade. 4th. Most generally permanent albuminuria (presence of albumen in the

[1] Revue de Médecine, 1881, p. 900.

urine from the organic changes of the kidneys) is not so common as a transitory albuminuria. This is considered by M. Gaucher as symptomatic of lead poisoning. This toxical albuminuria is the result of a deteriorated nutrition, or destruction of anatomical elements, whilst the albuminuria from organic disease of the kidneys (nephritis) derives its albumen from the blood plasma. 5th. The elimination of substances not natural to organic life, and which are usually and readily absorbed, such as medicaments, is retarded. This elimination goes on in an irregular way, at one time more active than at another (*saccadé*).

§ 244. With this introductory, though perhaps a rather more physiological synopsis of the theory of lead poisoning than most of our readers would relish, we pass directly to the history of symptoms which may be more easily comprehended. We have spoken both in this article, as well as in that on mercury, of a peculiar but rather constant appearance of a blue line along the gums. This is a very important evidence of lead poisoning, more especially so in the subacute or chronic form, since at one time it was considered pathognomonic of this metal; yet it may not always be present, and especially in that class of persons who are of cleanly habits and brush their teeth often, though the line may not be easily gotten rid of even by this means. The presence of the line is most generally a positive physical symptom of poisoning by lead or mercury. Its absence is merely a negative evidence of small value when taken in connection with the other evidences, especially the chemical evidence. The presence of this line once ascertained, the cause of it, whether mercurial or leaden, can perhaps be determined by analysis of the saliva and urine.

This slate-colored line, sometimes black when intense, is of about the width of two or three millimeters (about $\frac{1}{10}$ of an inch), and runs along at the junction of the gums with the teeth, particularly of the incisors and lower canines. It is due to the deposition of the sulphide of lead, which is formed by the contact of lead with the vapor of sulphuretted hydrogen, which is so often present in the cavity of the mouth, whether caused by the decomposition of particles of food lodging between the teeth, as suggested by Tanquerel,[1] or of the alkaline sulphates contained in the saliva (Schebach), or

[1] Op. cit.

by the blood.[1] It makes no difference whether the lead is absorbed by the mouth or stomach, or by the skin or lungs; this line is formed no matter how the poison is introduced into the system, and in fact it can often be made to appear when absent in a case of lead poisoning, by giving the patient a course of treatment by iodide of potassium, and will then persist for several weeks. Frank Smith[2] has proved the existence of this *secondary* or *indirect* line under the use of iodide of potassium, and Hilton Fagge[3] has since confirmed this statement. In the case of Schroenbröd, in 1873,[4] this line was not present until after the patient had ceased for four weeks from using a vinegar containing lead which had brought on lead colic.

In addition to this blue line on the gums there may be observed in cases of lead poisoning anatomical lesions in the mouth, which are quite constant and of a peculiar character, such as lead discolorations of the mucous membrane, and patches on the inside of the lips and cheeks before mentioned. The gums, oftentimes swollen and bloody, generally become thin and retracted, leaving the teeth prominent and encrusted with a deposit of *tartar*, and these often undergo decay.

These peculiar appearances of lead poisoning need not be mistaken for somewhat similar ones which have been observed with those persons who work on copper, or with those who use medicinally nitrate of silver or iron, or who brush their teeth with a dentifrice containing charcoal, which, according to Gubler,[5] sometimes produces analogous appearances, or to those having a scorbutic exhalation. It will always be easy to distinguish the leaden line from any of the above, or even from that caused by mercury. Oxygenated water brought in contact with the line gives rise to a whitish trace due to the conversion of the sulphide into the white sulphate of lead, and sulphuretted hydrogen water will change the sulphate back into the black sulphide of lead,[6] and even make this line darker than it was originally. Pauvert suggests another crucial test of scraping the incrustation off of the teeth, treating it with nitric acid and distilled water, thus transforming the lead salt into a soluble nitrate; if, after evaporation to dryness and dissolving the

[1] Gubler, Dict. Encyc. de Sci. Méd., art. *Bouche*, 1869.

[2] Lead Poisoning, Lancet, 1869, i. p. 746.

[3] Lancet, 1876, i. p. 709.

[4] Nouveau Dict. de Méd. et de Chir.

[5] Op. cit.

[6] D'Arcet, Tanquerel, Grêhant.

residue in water, the liquid be shaken with a solution of iodide of potassium (1 part in 20), it will become yellow from the presence of iodide of lead.

Functional disturbances of the secretions of the mouth may accompany the presence of the above phenomena, causing salivation, inflammation of the gums, and fetid breath; the reaction of the saliva, instead of being alkaline, becomes strongly acid. These functional troubles in the mouth precede those of indigestion with furred tongue, capricious appetite, and sometimes even nausea and distressing vomiting.

As the diseased condition advances, the tongue becomes white and dry, there is a disgust for food, and a feeling of heaviness and tenderness at the pit of the stomach. To this succeeds constipation of the bowels, hardness and morbid sensibility of the whole abdomen with pain in the back, and frequently headache. These symptoms are nothing more than what is often met with in aggravated dyspepsia or in gastric catarrh, but in lead poisoning they culminate in a sudden attack of colicky abdominal pain preceded by albuminuria, which it is needless to say, may often be overlooked. These colicky pains, feeble at first, gradually increase and become almost intolerable, being especially marked by increasing and decreasing paroxysms of pain; pressure gently exerted over the whole abdomen may diminish these pains, which become intolerable when the hand is suddenly removed after pressure. These attacks are in no ways different from the ordinary intestinal colic caused by impacted feces, and it is not improbable that their cause may be the same. The only means of distinguishing lead from other colics consists in the persistency of the former and its obstinacy to the ordinary means of treatment; these pains may last in an interrupted manner for days or even weeks, and are usually succeeded, sometimes even accompanied by an inflammation of the brain and an obstinate paralysis. These colics, upon the authority of Tanquerel, Kussmaul, and Maier, are attributed to lesions in the intestinal glands and muscular fibres, which may cause atrophy or destruction of both. According to Kussmaul and Maier, in the whole length of the alimentary canal, from the mucous membrane of the stomach to that of the lower portion of the colon, there is an atrophy or a fatty degeneration of the structure and anatomical elements of the glands and muscles, especially in the intestines, by

which their calibre is narrowed, this being more apparent near the pyloric orifice of the stomach and along the small intestine. Alcoholic excesses increase the tendency to colic, and so do also the drinking of acid wines and vinegar. More recent investigations render it probable that this abdominal pain is due to a neurosis of the intestinal plexus of nerves.

It may be generally said, without going too much into detail, that lead may be deposited throughout the whole body, and wherever it is so accumulated, it will prevent the functional activity of tissue, whether it be the blood corpuscles concerned in nutrition, in the glandular cells in pouring out secretions essential to the chemistry of life, in nerve centres, which excite and control the sensations and motions of the body, in the brain which presides over the whole organic life, or in the kidneys which remove deleterious substances from the circulation and the various tissues.

§ 245. Dr. Wm. Norris, of Stourbridge, gives an account of the poisoning of a vast number of persons, by acetate of lead accidentally mixed with flour. About thirty pounds of this salt were mixed with sixty or eighty sacks of flour, which was retailed to a great many persons in the neighboring villages. Nearly a thousand persons suffered from the poisonous effects of lead. The persons who ate the bread, after a few weeks complained of a peculiar taste; some compared it to soda, others to rusty needles or copper. The tongue was covered with a darkish cream-colored mucus, and was soft and flabby; the gums were swollen, with a blue line on the margin, and in many cases the blue tinge extended nearly over the gums, and occasionally on the inner side of the lower lip, and in a faint degree over the mucous membrane of the mouth and towards the fauces; the tonsils were in some cases enlarged, and in other cases there was salivation. These symptoms were accompanied by loss of appetite, nausea, vomiting, flatulency, and obstinate constipation, with a sense of constriction in the throat and epigastrium, and a violent spasmodic pain and twisting around the navel, which was retracted; the pain was sometimes increased by pressure, and when the paroxysms were violent, the muscles of the abdomen were contracted spasmodically, and a most frequent symptom was pain in the loins about the situation of the lumbar fascia, and in the deltoid muscles. The patients were chilly, with great languor and lassitude; the skin dry; the intellect was clear,

but there was general depression, and the pulse was low and feeble; the features were sallow and shrunken, and the muscles flabby; the fluid vomited was often mixed with bile and, occasionally, a coffee-ground secretion; the feces were dark and highly offensive, with scybala; the urine scanty and of a dark-red color almost like porter.[1]

§ 246. An interesting case of imputed poisoning by acetate of lead may be found in Dr. Maclagan's "Contributions to Toxicology," in the *Ed. Month. Journ.* for Dec. 1848. Although the falsity of the charge was shown by many circumstances, which it is not here necessary to relate, there was one which in itself would have had great weight in its refutation. The acetate of lead was said by the prosecutor to have been given to him in coffee. Now acetate of lead is the very agent employed to decompose and decolorize coffee in isolating its characteristic constituent caffeine. The precipitate which the lead salt forms in its infusion, if it is allowed to rest, subsides, and leaves a pale-colored fluid, in no respect resembling that which people are accustomed to drink as coffee. One ounce of ordinary coffee was boiled for ten minutes in the coffee-pot, which had been used, with six cupfuls of water. It was allowed to settle for five minutes, and then poured off. It had the ordinary appearance of unclarified coffee, dark-brown, and slightly turbid, and depositing some coffee-grounds. "Half an ounce of sugar of lead, being the same proportion to this bulk of fluid which was found in the coffee got from the prosecutor, was now added; the coffee was boiled again and allowed to settle for five minutes after removal from the fire. Another similar portion, being decanted, was now found to be a clear transparent liquid, with hardly any color, except a faint shade of green, and more resembling a weak infusion of green tea than coffee. It was obvious, therefore, that if, during the breakfast, the coffee-pot remained at any time at rest for five minutes, the next cupful poured off must have been so different in appearance from ordinary coffee as at once to attract attention." This decolorizing property of the sugar of lead should, not, therefore, be lost sight of in any future case of alleged poisoning by its mixture with coffee.

§ 247. *Post-mortem appearances.*—There are few poisons productive of so much suffering, and, when fatal, of such violent

[1] Prov. Med. and Surg. Journal, June 27, 1849.

symptoms towards the close of life, and yet leave in the body such indistinct traces of their action as these. In a case which terminated with the symptoms of saturnine *encephalopathia*, viz., delirium, insensibility, and tetanic convulsions, Empis and Robinet found no anatomical alterations of any importance. Lead was discovered, by incineration, in the brain and liver. Likewise, in another case examined by Dr. Hopfgartner, of Vienna, lead was found in the same organs, but no pathological alterations, except that one of the lateral columns of the spinal marrow appeared to be wasted. In Dr. Letheby's case, lead was freely detected in the contents of the stomach, in the brain, muscles, liver, intestines, blood, and in the serum of the cerebral ventricles. The stomach and intestines were pale and nearly empty, and the latter contracted, and in some places invaginated. Most generally a microscopical examination of the muscles will reveal a deterioration of the muscular fibres and an enlargement of their nucleoli.

§ 248. *Chemical examination.*—The detection of lead and compounds of lead is very easy, since the tests for lead are so characteristic. The principal ones are the following: (1) All solid compounds of lead, if mixed with sodium bicarbonate and heated upon charcoal with a blowpipe, are reduced and a globule of metallic lead is left upon the charcoal; this globule can be removed from the charcoal and examined in respect to its color, malleability, hardness, etc. (2) Solutions of lead compounds, if acidulated, and sulphuretted hydrogen passed through the solution, will yield a black precipitate of sulphide of lead, which precipitate is readily soluble in nitric acid, but is insoluble in hydrochloric or acetic acids. (3) If a solution of a lead salt be treated with a solution of sodium carbonate, a white precipitate (white lead) is formed, which is soluble in nitric or acetic acid with effervescence. (4) If a solution of a lead salt be treated with dilute sulphuric acid, or with a solution of a sulphate, such as sodium or magnesium sulphates, a white precipitate of sulphate of lead will be formed, insoluble in nitric or hydrochloric acids, but decomposed by hot alkalies or alkaline carbonates; hence, the value of sulphates as antidotes in cases of poisoning by lead compounds, the sulphate of magnesium being the best one for this purpose on account of its cathartic effect, which removes the sulphates of lead from the intestines rapidly, otherwise it would be slowly decomposed by the

intestinal fluids and absorbed; this precipitate of sulphate of lead is also soluble in a solution of ammonium acetate. (5) If a solution of chromate of potassium be added to one of a lead salt, a yellow precipitate, chromate of lead, chrome yellow, will be formed which is decomposed by caustic alkalies, but is insoluble in nitric acid; this enables us to distinguish it from a similar precipitate formed by the same reagent with soluble salts of bismuth. (6) If hydrochloric acid or a chloride be added to a solution of a lead compound, a white precipitate, chloride of lead, is formed, which is quite soluble in boiling water, but separates again on cooling in the form of long prismatic crystals. (7) In the same way if hydriodic acid or an iodide, like iodide of potassium, be added, a yellow precipitate, iodide of lead, is formed, which is soluble in boiling water, and separates from it on cooling in the form of golden yellow hexagonal scales; free nitric acid will prevent this reaction. (8) If a piece of zinc be introduced into a solution of a salt of lead, metallic lead will be deposited upon the zinc in the form of a black granular powder, which can be melted into a metallic globule.

§ 249. *Separation from organic matter.*—The method usually adopted is that used for the separation of arsenic from organic tissues, as already described in speaking of arsenic. The sulphide of lead formed by the sulphuretted hydrogen in that process is freed from organic sulphur compounds by the mixture of ammonia water and sulphide of ammonium; the black sulphide, which is left upon the filter paper, is dissolved by nitric acid, which separates it from sulphide of mercury if present, the solution of nitrate of lead thus formed should be evaporated to dryness to expel all excess of nitric acid, and the residue dissolved in water to which a few drops of acetic acid may be added if necessary, and this solution used for performing the above tests. If bismuth or copper compounds were present at the same time, the lead must be separated from them by precipitating it with dilute sulphuric acid, washing the sulphate of lead thus formed, decomposing it with boiling sodium carbonate, washing the lead carbonate which results, dissolving it in acetic acid and performing the tests with this solution.

Since lead when eliminated passes out with the urine, although only in small amount, the analysis of this fluid for lead is very frequently necessary for the purpose of diagnosis. About a quart of the fluid should be evaporated to dryness, the residue burned in a

crucible, until only a white mass remains ; this complete combustion of the organic matters of the urine is facilitated by the addition of pure nitre before ignition. This white mass should then be dissolved in hot dilute hydrochloric acid, filtered while hot, the filtrate treated with ammonia water and then a solution of ammonium sulphide, which precipitates the phosphates and the sulphides of iron and lead ; wash this precipitate with boiling water several times by decantation, dissolve the phosphates and sulphide of iron by acidulating with hydrochloric acid, and allow this fluid to stand until the next day for the sulphide of lead to settle ; then filter through a pure Swedish filter paper, when the sulphide of lead will be left as a black coating upon the filter paper ; after washing with water, dissolve this by pouring over it drop by drop concentrated *pure* nitric acid, which is collected in a watch glass and evaporated to dryness over a water bath ; a residue of basic nitrate of lead remains, which is best tested by moistening it with a drop of water and moving over it a very small crystal of iodide of potassium, when there will be formed the yellow iodide of lead wherever the crystal of iodide of potassium touches the lead compound. This complicated process is necessary in analyzing urine, because even when the patient is subjected to the appropriate treatment for removing lead from the body, only a few milligrammes of lead pass out with the urine in twenty-four hours. The principal precautions necessary are to be sure that the ammonia water is free from lead, and the nitric acid and the Swedish filter paper are free from iron, which would give an orange color upon performing the final test with iodide of potassium. Drinking water suspected to contain lead should be analyzed in the same way, except that it is not necessary to evaporate it to complete dryness and ignite the residue ; it is only necessary to concentrate one or two quarts to the volume of two or three ounces.

§ 250. The method recommended by Tardieu for the separation of lead from organic tissues is the following : " Divide the solid portions into small pieces, which can then be immediately mixed in a mortar with half their weight of pure and dry carbonate of sodium. The pulp that results is first dried in a water-bath as completely as possible, and then placed in a covered porcelain crucible ; the mixture should occupy but half the capacity of the crucible. This is then gradually heated either in a little furnace or over a Berzelius

lamp, in such a way as to prevent overboiling, and then increasing the heat until the carbonate of sodium becomes fused. As soon as the fusion is complete, the fire is gradually withdrawn; care must be taken not to allow a continuance of the heat much beyond the fusion, as some of the reduced metal will become volatile. After the crucible has become quite cold, it is withdrawn from the furnace and carefully wiped, and it may then be placed in a large porcelain capsule containing boiling distilled water. The ebullition is continued until all the vitreous mass in the crucible is completely dissolved (this can easily be ascertained by removing the crucible and by examining the substance in its interior). By means of a washing bottle the exterior and interior of the crucible are thoroughly washed, the latter being kept in the capsule. The crucible is then allowed to dry. The liquid in the capsule, after being allowed to stand for some time, is cautiously decanted, so as to remove the charred portions and the soluble salts, and to preserve the heavier metallic portions which sink to the bottom. At the end of several washings and decantations, the metal, if present, is found in little bright particles which may easily be collected and dried upon bibulous paper. The crucible had better be examined, as some of the lead globules may have adhered to its sides. If the metallic particles are too small to be easily handled and observed, it is a good plan to reunite them with a little pure and dry carbonate of sodium in a cavity of a bit of charcoal, and by the blowpipe to collect them in one or two large globules. Thus obtained they can easily be produced in a court of investigation."

XVI. *The Salts of Copper.*

§ 251. The most common of these salts, met with in cases of poisoning, are the sulphate (blue vitriol), the subcarbonate, and the subacetate of copper (verdigris). Arsenite of copper (Scheele's green), and the mixture of this with the acetate of copper (Schweinfürt's green) have been mentioned in connection with arsenical poisoning. Verdegris is formed in alloys of copper by the combined action of air and dampness, or by the maceration of copper in acetic acid. It has been made by allowing copper weights to soak in vinegar. Verdegris has been used in cooking to give to vegetables a more brilliant green color. M. Derheims has recorded a case of poisoning by *liqueur d'absinthe* colored with blue vitriol. Bakers some-

times add sulphate of copper to dough, to whiten the bread and increase its weight; especially is this the case, where an inferior or damaged flour is used, since the sulphate of copper improves the apparent quality of the bread and helps the flour to absorb more water. Hence its more frequent use as an admixture to flour made from grain harvested after it has become too mature. In some countries, also, it is the custom to steep the seed wheat in a solution of sulphate of copper in order to destroy parasitic insects, or to prevent the germination of the eggs of those which injure the grain after it has been sown; if the grain is allowed to steep too long in the copper solution, the seed absorbs a notable amount of copper. This risk could be avoided by using, instead of the sulphate of copper solution, one which should contain sulphate of sodium, which is equally effectual in preventing the damage threatened to the wheat, and is not injurious to animal life; for it has sometimes happened that instead of being planted, the seed has been sold and manufactured into flour, and in consequence serious illness has occurred to those who have eaten the bread. As above mentioned, the mixture of sulphate of copper with dough makes a light bread out of an inferior grade of flour. This is a reprehensible act, since it introduces an irritant drug along with natural food, and may give rise to serious disturbances of the digestive functions. Kuhlmann has shown that a mixture of one part of sulphate of copper with seventy thousand parts of damaged flour will improve its bread-making qualities. This amount of the copper salt cannot cause any material disturbance of health to those eating the bread; but unfortunately it has been shown that bakers use a much larger proportion than is necessary, and in consequence have done harm to their customers. One baker, in Germany, was shown to have used a proportion of one five-thousandth part of sulphate of copper by weight, and in the bread of another baker the blue vitriol crystals were easily distinguished in the bread sold to the public.

§ 252. It is seldom that these poisons are designedly administered with homicidal intentions, since their detection, both by the color and taste, is too easy to permit it. A husband attempted to poison his wife by adding verdigris to a dish of beans. The bad taste prevented her from eating them. He buried the cooked mess in his garden, from which it was disinterred, and then examined by chemists. They proved the certain presence of the metal. He

was condemned to hard labor for life.[1] Cases of poisoning from these salts may then be divided into those in which a large dose is swallowed, either by accident, or with a view to suicide, and those which proceed from the contamination of food by *unclean* copper vessels, or by the salts of copper used as coloring matters for confectionery, etc., the injurious qualities of which will depend upon the amount of contamination.[2] If metallic copper is subjected to the action of water, acetic acid or vinegar, or hydrochloric acid, it is not affected by it unless the fluid is in contact with air or oxygen, so that copper vessels can be used for cooking, if the fluid be removed from the copper vessel while it is hot, in which case the vinegar, fatty acids, or water will have no effect upon the copper, but if the fluids be allowed to stand in the copper vessel for several hours when cold, air is absorbed, the copper is in part oxidized, dissolved by the acid, and impregnates the food. If metallic copper in a very finely divided state comes in contact with the mucous membranes, it becomes oxidized and absorbed, and gives rise to certain disagreeable symptoms; this is the so-called professional poisoning, which rarely occurs in workers in bronze, where very finely divided copper in the form of dust is inhaled.

§ 253. The *symptoms* of acute poisoning from copper salts come on very soon after the ingestion of the poison. There are violent headache, vomiting and purging, severe colicky pains, eructations, salivation, cramps in the limbs, and finally convulsions and insensibility. The vomitus after large doses has a green color, and is colored azure-blue by ammonia water. The diarrhœa is usually like that of dysentery. Sometimes jaundice is observed. In a case related by Dr. Percival, two drachms of sulphate of copper produced fatal convulsions. In another, where the same salt was swallowed, there were no convulsions. The child, which was sixteen months old, died in four hours.[3] Those cases which have terminated fatally have lasted a variable period. Thus in one reported by Pyl, a woman, who swallowed two ounces of verdigris, died in *three* days; in another, by Neumann, half an ounce destroyed life in *sixty* hours;

[1] Journ. de Chimie, Chevalier, 1854.

[2] Metallic copper is not poisonous, according to the best authorities, unless organic decomposition or the addition of an acid has formed it into an irritating salt of copper.

[3] Med. Gaz., vol. xviii. p. 742.

and in another, in which an ounce of blue vitriol was taken, death ensued within *twelve* hours.[1] In most cases, however, of poisoning with these salts, the patient has recovered, when timely and efficient means have been used.

In those cases in which the poison has been conveyed accidentally, through articles of food, into the system, the symptoms have been the same as those mentioned, although they have usually not come on until a few hours afterwards. They are thus described by Orfila: "An acrid, styptic, coppery taste in the mouth; parched and dry tongue; a sense of strangulation in the throat; coppery eructations; continual spitting; nausea; copious vomiting, or vain efforts to vomit; shooting pains in the stomach, which are often very severe; horrible gripes; very frequent alvine evacuations, sometimes bloody and blackish, with tenesmus and debility; the abdomen inflated and painful; the pulse small, irregular, tense, and frequent; syncope, heat of skin, ardent thirst, difficulty of breathing, anxiety in region of the præcordia, cold sweats, scanty urine, violent headache, vertigo, faintness, weakness of the limbs, cramps of the legs, and convulsions." Such are the symptoms which, it is said, are produced by the ingestion of articles of food contaminated with copper salts. How far they are *really* due to this cause we shall presently inquire.

In a very instructive article by Dr. Hönerkopf,[2] it is maintained that sulphate of copper cannot in a strict sense be considered a poison. He refers to numerous cases in which this medicine was taken in large or repeated doses without harm, and usually with benefit, and shows that the greater number of symptoms ascribed to it are not really observed.

Salts of copper even in moderate doses produce, after they are received into the stomach, and sometimes immediately after their ingestion, disturbances of digestion, such as are above enumerated: pain at the pit of the stomach, nausea, vomiting, and abdominal colicky pains, which are followed by diarrhœa. These effects occur principally after the use of the sulphate, carbonate, and acetate of copper.

It should be remarked also that the irritating effect upon the mucous membrane does not hinder the absorption of the salts of

[1] Quoted by Beck.

[2] Casper's Vierteljahr., viii. 212.

copper, for these are found in the urine and the secretions from the lungs.[1] According to Miahle,[2] the salts of copper with the organic acids are more readily absorbed than those formed with the stronger or inorganic acids, since the precipitate produced by a combination of the latter with the proteine substances in the intestines dissolves quite slowly. It should be observed that, as in the case of the lead salts, compounds of copper are not so easily absorbed into the system when taken in large as in small doses, since the large doses cause more local irritation, and hence are more rapidly eliminated or expelled by the natural outlets of discharge; the small doses, *per contra*, being longer retained in contact with the internal surfaces, are slowly but more surely absorbed into the circulation. Thus, as medicines, these salts are prescribed in small unirritating doses when the practitioner wishes to effect their absorption, and in large doses, where he wishes to evacuate offending materials from the *primæ viæ;* one quarter or a half grain is used in the former, and two to ten grains in the latter case, or for an emetic action.

§ 254. Pecholier and Saint-Pierre[3], in their observations of persons exposed to the absorption of verdigris and, in corroboration of these observations, by experiments on animals, believe that its slow absorption favors the development of fat, and noticed that the women in these factories were free from anæmia and chlorosis. This opinion is at variance with the older writers on toxicology, who have taught that the slow absorption of the copper salts causes colicky pains similar to those in lead poisoning, as well as emaciation and constitutional disturbances. It may be said, however, that many cases of so-called poisoning by copper salts have also been simultaneously subjected to the absorption of metallic lead, or of arsenic in the form of Scheele's green (vert de Schweinfurt, vert mitis, vert de Vienne, etc.) which contains arsenite of copper. It is highly probable, therefore, that the ill effect of this exposure may be due to another agent than copper, and one upon which there is an agreement as to a definite poisonous action.

[1] Dangers and Flandin.

[2] Chimie appliquée à la Physiologie et à la Thèrapeutique, Paris, 1856.

[3] Etude sur l'Hygiène des ouvriers employés à la fabrication de verdet, Montpellier Medical, t. xii., 1864, p. 97.

Desayure[1] relates that the workmen exposed to the manufacture of copper after a certain time show symptoms of nausea, vomiting, stomachal disturbance, diarrhœa, oppression, and fever, as well as intestinal distress, meteorism, and habitual constipation. Perron[2] adds, in addition to the above effects, that these successive morbid conditions predispose to the development of pulmonary consumption, especially in those whose temperament and constitutional tendencies are towards this disease. As a rule, however, in cases of copper poisoning, no lesion of the lungs can be detected to account for the cough and night sweats from which these patients suffer. In our opinion these symptoms and dangers are more likely to be caused by the presence of fine metallic dust in the respired air, than to any poison caused by the absorption of metallic copper into the circulation. A similar example is offered of the fine particles of diamond powder, corundum, iron, and other similar substances, which irritate the pulmonary and gastric mucous membrane. A consultation and study of conflicting authorities leads Maisonneuve[3] to express the following conclusions in reference to the so-called "copper-colics":—

"1. Work and manipulation of cold metallic copper are innocuous, but in workshops, where the metallic particles of oxide or salts of copper abound in the air, the inhabitants may receive this irritating dust into the air-passages, and cause oppression and very intense dyspnœa (asthma?) with bronchial and laryngeal spasms.

"2. Copper-colic exists, though it is denied by some authorities; it is, however, of short duration and of very slight consequence."

Maisonneuve describes this colic as characterized by a painful sensation having its location at the pit of the stomach or at the navel, and extending over the region of the transverse colon, being increased by pressure; it causes nausea and vomiting, and rarely diarrhœa; these symptoms have a very short duration, and the workmen never seek for medical relief, but treat themselves by drinking milk in large amount. From this last-named successful

[1] Etude sur la maladie des ouvriers de la manufacture d'armes de châtellerault. Ann. d'Hygiène et de Médecine, Légale, 1861.

[2] Des malades des ouvriers produites par le cuivre et l'absorption des molecules cuivreuses. Idem, 1861.

[3] Hygiene et pathologie professionelles des ouvriers des arsenaux maritimes, ouvriers en cuivre. Arch. de Med. Navale, 1865, p. 25.

means of treatment one is led to the conviction that the so-called colics are caused by local irritation from the presence of a mechanical irritant, because the effects of this irritation are so readily allayed by the reception of an unirritating substance, such as milk would seem to be.

It will be seen by comparison with the description of lead colic, that the colic caused by copper is very different. A purple line also appears at the junction of the teeth and gums, at the same place as the blue line of lead poisoning, and due to the same cause, viz., the formation of the sulphide of copper.

As has been previously mentioned, when death is caused by the poisonous, or rather irritating, action of sulphate or other salt of copper, it follows the ingestion of a large amount of the agent, and in this case the evidence of irritation or inflammation, as well as the amount of copper in the *primæ viæ*, must be large in extent or amount. The fact must not be overlooked that sulphate of copper is often used as an emetic in cases of suspected poisoning.

§ 255. *Post-mortem appearances.*—The mucous membrane of the stomach and intestines is inflamed and thickened, in some places eroded, and, in a case quoted by Orfila, the small intestine was perforated. If the patient has not survived long, the mucus of the intestines will be found tinged of a green color. The digestive tube is generally distended by an enormous quantity of gas, and the mucous membrane of the alimentary canal is often red and inflamed, sometimes throughout its whole extent; at other times some ecchymoses distributed in the submucous cellular tissue are noticed, and also ulcerations and gangrenous patches. But the most important and remarkable appearance is the predominance of inflammatory lesions over blood extravasations, in fact a true catarrhal inflammation such as might be caused by any mechanical irritant; these latter rarely occur, and are met with in exceptional cases under the serous coat of the intestines, lungs, and heart. (Tardieu.)

No other changes worthy of note have been observed. A fatal case is reported by Mr. Cockburn,[1] in a woman who swallowed two or three drachms of sulphate of iron with seven of sulphate of

[1] Lancet, Aug. 1856, p. 248.

copper. The symptoms were such as are described above, and death took place in about twenty-four hours. Yet there was no disorganization whatever of the mucous coat of the stomach and intestine.

§ 256. The use of copper utensils in the preparation of food has occasionally given rise to serious consequences, on account of the impregnation of the food by some salt of this metal. If the vessels are bright and clean, very little harm can possibly result from this cause, when ordinary articles of food are boiled in them and not allowed to remain in them after they become cool. Saline, acid, or oily matters act, however, upon copper vessels, and if these are not clean, having been already exposed to moist air and become covered with the carbonate, the food may be impregnated with this poisonous salt in sufficient quantity to produce alarming symptoms. Such will be especially the case, if articles of the kind have been allowed to remain in the vessels to cool. It need hardly be stated that tinning the vessels is the only certain mode of preventing such effects, though here we have a further risk from the fact that copper is frequently not properly tinned, but is coated with an alloy containing metallic lead. In the latter case, the colic and poisoning due to lead may mislead the mind of the medical witness into the supposition that the symptoms were due to copper, when the true explanation would be that of lead poisoning.

There can be little doubt, we think, that the frequency of accidents from this cause is much exaggerated, and that in many cases the sudden illness which is mistaken for the symptoms of copper poisoning is really due to the unwholesome nature of the food eaten, or to other causes. We are led to this belief not only from a consideration of the extremely small quantity of copper that in most cases can be dissolved, but also, as before stated, from the reflection that unwholesome food is capable of given rise to a set of symptoms very similar to those produced by copper, and, finally, from the fact that in several cases of suspected poisoning by copper, this metal could not be detected by chemical analysis in portions of the food used.

Prof. Langenbeck, of Göttingen, reported an instance of the poisoning of thirty-one persons, who had eaten a portion of beef sausage. This sausage meat had been fried in lard, which had

stood for two days in a badly tinned copper vessel, and was said to have become green in consequence. The poisoning was, therefore, attributed to copper. Dr. Paasch, in order to estimate the amount which each person in this instance must have taken, makes the following calculation. He assumes as barely possible that one scruple of metallic copper could have been dissolved by means of the fatty acids existing in the lard. This amount would correspond to twenty-five grains of oxide of copper, fifty-seven and a half grains of anhydrous, or sixty-three grains of crystallized acetate of copper. Supposing that the whole of this had been taken up by the food and entirely consumed, each person would have swallowed *two* grains of verdigris.[1] That so small an amount should be capable of producing alarming symptoms of poisoning is hardly possible, when we reflect how much larger doses of this, and other equally poisonous salts of copper, have been given without harm in medical practice. Gerbier is said to have given the subacetate in doses amounting to twenty and even thirty grains in the twenty-four hours, and Solier de la Romillais ten to twelve grains a day.[2] The sulphate of copper is frequently given in doses of fifteen grains at a time, for the purpose of procuring emesis, in narcotic poisoning. Richmond gave as much as a scruple of carbonate of copper daily to patients suffering with obstinate neuralgic affections, and Key, for the same purpose, administered as much as half an ounce daily, divided into three doses, continuing the treatment for a fortnight, with no other result than the cure of the disease.[3] Pereira says that he administered six grains of the sulphate of copper thrice a day for several weeks in an old dysentery, without any other obvious effect than slight nausea and amelioration of the disease for which it was given. If the symptoms arising from the use of unwholesome food, such as sausages, old cheese, and the like, be now compared with those which are ascribed to poisoning by copper, a very great similarity will be found between them.[4] Dr. Paasch relates instances in which the conviction was so strong that the symptoms of poisoning must have been due to a salt of copper, that a chemical investigation of the food was undertaken,

[1] Casper's Vierteljahrschrift, Jan. 1852.

[2] Guersent, Dict. des Sci. Méd., art. Cancer.

[3] Dict. de Méd., art. Cuivre.

[4] *Vide* Poisonous Food.

which resulted, however, in the fact that not a trace of copper could be discovered.[1] Dr. Taylor, in his work on poisons, states that he was required to examine the following case: "In an extensive poor-law union, a number of the paupers had been seized with diarrhœa and dysentery, and several of them died. There was no apparent cause for this sickness and mortality; and it was suspected that the soup, which was daily prepared in large copper boilers, might have become impregnated with the metal, and have given rise to the symptoms, although these were scarcely indicative of irritant poisoning. I ascertained that the copper vessels were cleaned out daily, that the soup was made with salt and other vegetables, but was poured into other vessels to become cool." The soup, moreover, gave no trace of copper by the iron test, was unaffected by a current of sulphuretted hydrogen gas, and the incinerated residue, after evaporation and calcining, gave no sign of the existence of copper with any of the tests.

While these considerations throw doubts upon the frequency of poisoning by food impregnated with copper from the use of cooking utensils of this material, they do not, of course, destroy the well-attested fact of its occasional occurrence. It appears evident, however, that the slightest attention to cleanliness in the keeping of such articles is all that is necessary to secure immunity from danger.

The use of verdigris or other salts of copper for the coloring of confectionery or of other edible articles is manifestly a very pernicious practice. A highly interesting case, in which a whole family was poisoned, and two of its members died, from the use of vegetables thus colored, is reported by Kramer.[2] Dr. Percival found a strong impregnation of copper in pickled samphire, of which a young lady ate one morning a considerable quantity, and which proved fatal in nine days. Dr. Falconer once detected so large a quantity in some pickled cucumbers, bought at a great London grocer's, that it was deposited on a plate of iron, and imparted its peculiar taste and smell to the pickles. It seems, indeed, to have been at one time the custom to make a point of adulterating pickles with copper; for in many old cookery-books the cook is told to make

[1] Loc. cit.

[2] Canstatt's Jahresbericht für 1851, Bd. iv. S. 269.

her pickles in a copper pan, or to put some half-pence among the pickles, to give them a fine green color.[1] Many of the cases of poisoning by confectionery are due to the arsenite of copper, or Scheele's green, which we have elsewhere treated of.[2]

§ 257. Numerous cases are related in which *copper coins* have been swallowed with the symptoms of copper poisoning resulting. On the other hand, a case is mentioned by Dr. Jackson, of Boston, in which a half-cent swallowed by a child produced nausea and vomiting, another by Dr. Budd and others,[3] and another by Dr. Hartshorne, in which a boy five years old died with all the symptoms of poisoning by copper, just two years after having swallowed a brass button.[4] A curious case is related by Deutsch, of a boy six years old, who swallowed a number of small copper coins. His medical attendant prescribed vinegar and other organic acids! In consequence of this singular treatment, he was seized with alarming symptoms, violent colic, and vomiting and purging of greenish-colored mucus. Finally he was enabled to throw up the coins by means of an emetic of ipecacuanha, but recovered very slowly from the effects of the poison.[5] A curious question might well arise in such a case as this. Metallic copper is usually acknowledged to be not poisonous; the poisonous salt, viz., verdigris, was here formed in the stomach by the administration of vinegar. *Query:* Was the poison *administered?*

§ 258. There have been occasional periods of excitement among toxicologist and physiological chemists as to whether copper is a natural constituent of the body. Frey[6] states that "Copper has been noticed in the blood, bile and biliary calculi of man. It is excreted by the liver." Tardieu also discusses the question. Messrs. Bergeron and L'Hôte[7] record an examination to show that in all probability no copper is present in the tissues of the body unless accidentally introduced. They were induced to conduct their experiments on account of the poisoning of two women at St. Denis by Moreau by means of copper, the presence of which was not discovered until revealed by chemical analysis, corrosive subli-

[1] Christison, p. 352.
[2] *Vide* Arsenite of Copper.
[3] *Vide* Beck.
[4] Taylor, Am. ed., p. 112.
[5] Canstatt, 1851, Bd. iv. 269.
[6] Histology and Histochemistry of Man, Heinrich Frey, translated by A. E. J. Barker, New York, 1875, p. 62.
[7] From New York Med. Record, April 17, 1875, p. 272.

mate having been the suspected poison. It is probably a fallacy to suppose that copper is a normal constituent of the body, and with this explanation it may be stated that Orfila has detected traces of copper in the bodies of animals not poisoned by any of its preparations, and Wackenroder has obtained it from human blood. It has also been found in coffee, wheat, and flour by M. Sarzeau and others.[1]

§ 259. According to the calculations of Sarzeau, a man would introduce into his system by ordinary aliments six and nine-tenths grains of metallic copper in the course of fifty years, not allowing that any of the metal is eliminated by the emunctories. "Though the results announced by chemists and the narration even of their experiments were of such a character as not to be accepted without discussion, they were still allowed entrance upon the rolls of science, and for several years it was found easier to accept rather than to verify them. This tendency soon became general. Along with copper were ranged *normal arsenic*, *normal lead*, *normal manganese*, etc. The reactionary movement, however, was not long in asserting, through the results of several chemists who carefully experimented, putting out of their way every possible source of error, that the animal system did not naturally contain these poisonous products. M. Roussin records an experiment upon the body of a soldier killed suddenly by a fall from the barracks in Algeria, where he endeavored to determine the presence of normal copper in the organs and other tissue with negative results.[2]

MM. Bergeron and L'Hôte[3] examined the liver and kidneys of fourteen bodies, and found traces of copper in all of them; in these analyses every precaution was taken to prevent the accidental introduction of copper during the analyses. These authors conclude that copper is always present in the liver and kidneys, and that the amount never exceeds two and a half or three milligrammes in the liver, unless copper compounds have been taken as medicine, and rarely reaches the amount of two milligrammes. MM. Bôurneville and Yvon found in the liver of an epileptic,[4] who had taken during life forty-three grammes of ammonio-sulphate of copper as a medicine,

[1] *Vide* Bley's Archiv für Pharmacie, Oct. 1853; also Christison on Poisons, Am. ed., p. 356.

[2] Tardieu, op. cit.

[3] Journ. de Chimie Médicale, 1875, page 115.

[4] Ibid., page 236.

0.295 gramme of copper, which corresponds to 1.166 grammes of sulphate of copper, although the patient had taken none of the copper medicine for three months before death. These gentlemen made the analyses of the organs of the two females in the "Moreau" poisoning case, and found in the liver an amount of copper corresponding to 0.119 gramme of sulphate of copper in one case, and to 0.084 grm. in the other. They stated, therefore, that since so large an amount of copper was found, and because the symptoms were not inconsistent with acute copper poisoning, and a careful *post-mortem* examination failed to reveal a natural cause for death, a compound of copper must have been the cause of death. L. M. V. Gallippe[1] takes exception to the above conclusions of Bergeron and L'Hôte, and considers that an expert has no right to state that death has been produced by copper poisoning when such small amounts as the above are found, since much larger quantities have been detected in cases in which death has been due to natural causes. Yvon found in the liver of another person who had been treated during life with copper compounds 0.236 grm. of metallic copper; Rabuteau found 0.239 grm.; and Galippe found 0.310 grm., 0.220 grm., and 0.120 grm. of metallic copper in different cases. From experiments upon animals and men, Galippe concludes that (1) except in cases of suicide, acute poisoning by compounds of copper ought not to be possible, both on account of the horrible taste which these compounds have, and on account of their emetic property, which causes the expulsion of the poison; and (2) poisoning of a mild grade is impossible, since, according to his experiments upon animals, when small doses are given, tolerance of the system for the copper compound is established without any injurious influence upon the health.

Dr. Taylor[2] described chronic poisoning by copper, in which sometimes the patients die from fever and exhaustion.[3]

§ 260. *Fatal dose.*—As cases of acute poisoning are rare, it is difficult to determine how small a dose will cause death. The

[1] Etude toxicologique sur le cuivre et ses composés, Paris, 1875.

[2] On Poisons, p. 520.

[3] *Vide*, also Böcker, Vergiftungen, 1857, p. 42; Orfila, Toxicologie, i. 912; Casper's Vierteljahrschrift, 1852, i. p. 79; 1855, ii. p. 22; 1856, ii. p. 41; 1857, ii. p. 228; Annales d'Hygiène, 1847, i. 392; and Avril, 1858, p. 328; Dublin Hospital Gazette for Sept. 1854; Lancet, Jan. 1855.

effect of swallowing a single large dose is emetic, and consequently we may suppose that a poisonous dose is often rejected by the stomach. Five drachms of the sulphate have been taken without causing death. (Taylor.) Böcker assigns one or two ounces of verdigris as a fatal dose; but seven drachms have caused the death of an adult. (Taylor.) The greatest danger to life lies in the continued administration of the poison. Sulphate of copper is frequently prescribed by physicians to cause emesis in the dose of five to fifteen grains, and this dose is often repeated till vomiting is effected.

§ 261. *Chemical examination.*—The tests for copper are quite delicate, and there is usually no difficulty in detecting it when present in very small amounts. The principal tests for copper compounds are the following: 1. The color of copper salts is either green or blue, and metallic copper has a peculiar odor, which can be perceived by rubbing the moistened finger upon a copper coin. 2. If nitric acid be added to metallic copper, it readily dissolves it, and reddish vapors are evolved, which can be easily perceived by looking down into the vessel in which the test is performed. 3. A solution of potassium hydrate produces a bluish-green precipitate of the hydrate of copper, which is soluble in ammonia water, and in some organic fluids, such as glycerine and tartaric acid. 4. Ammonia water, if added to a solution of a salt of copper, gives at first a precipitate of the hydrate of copper, but if an excess of the ammonia is added, the hydrate is redissolved, and an azure-blue solution results. 5. A current of sulphuretted hydrogen passed through a solution of a salt of copper produces a brownish-black precipitate of the sulphide of copper, which is readily soluble in nitric acid. 6. A solution of ferrocyanide of potassium added to a solution of a salt of copper produces a reddish-brown precipitate of the ferrocyanide of copper, which is insoluble in hydrochloric acid. 7. A piece of metallic iron introduced into a solution of a salt of copper which has been acidulated with hydrochloric acid, becomes coated with a deposit of metallic copper, which can readily be recognized by its color and physical properties; a steel needle or a knife blade is usually the most convenient form of polished iron to use, and this deposit of copper upon the steel should always in legal cases be taken to court as one of the *corpora delicti*, since the jury can easily recognize metallic copper. 8. The galvanic

test, like the foregoing test, results in the separation of metallic copper from its solutions. It is performed by placing the solution in a platinum vessel, acidulating with hydrochloric acid, and introducing a piece of metallic zinc so that it will come in contact with the platinum; galvanic action will thus be set up, and all of the copper in the solution will be deposited upon the platinum in the form of the reddish metallic coating, which can be recognized by its physical properties, color, etc., or by dissolving it in nitric acid, evaporating this solution to dryness upon a water bath, dissolving the residue of nitrate of copper in water, and testing this solution by the above liquid tests.

§ 262. *Separation from organic mixtures.*—The organic matter must first be destroyed, either by the method described for the isolation of arsenic, or by burning up the organic matter in a crucible after the addition of pure saltpetre. After precipitation of the sulphide of copper by sulphuretted hydrogen, the precipitate should be dissolved in nitric acid, the resulting solution evaporated to dryness upon a water-bath, the residue of nitrate of copper dissolved in water after the addition of a drop or two of hydrochloric acid if necessary, and this solution tested by the special tests given above.

XVII. *Salts of Zinc.*

§ 263. *Oxide of zinc* has been of late years used as a substitute for white lead, with the view of avoiding the dangerous effects of the latter on the workmen. It has been supposed to be innocuous, and this idea appeared to be confirmed by some experiments made by M. Flandin. He rubbed animals over with ointments of oxide of zinc, of carbonate of lead, and sulphate of lead; the last two were found always to produce poisonous effects, but the animals rubbed with the ointment of oxide of zinc continued to enjoy their usual health. A case has, however, been recorded by Dr. Bouvier, of the Hôpital Beaujon, at Paris, in which a laborer who had been employed for fifteen days in barrelling oxide of zinc, and who in other ways had handled this substance, and breathed an atmosphere loaded with its powder, was attacked with vomiting, colic, and constipation. These symptoms persisted, and increased in intensity so much that he rolled on the floor in agony. The vomited matters were bilious, he rejected his food almost immediately after swallowing it, and he had been constipated for five days. From the whole

history of the case, it was considered to be one of genuine zinc colic. He was cured by the remedies usually employed for painter's colic. The particles adhering to his body were examined, and found to consist of oxide of zinc.[1] Ladouzy and Maumené have seen workmen, who were obliged to inhale an atmosphere loaded with particles of oxide of zinc, affected with inflammation of the mouth and throat, salivation, general distress, colic, and diarrhœa, or obstinate constipation.[2]

It must not be forgotten that oxide of zinc is very likely to be contaminated with salts of lead and arsenic, and as many of the above symptoms are those of poisoning by either of these metals, it is highly probable that the suspected case was not due to the zinc oxide. It should, at least, be shown in evidence that no such contamination was possible in a given case of suspicion.

Cases of zinc-poisoning, arising from the inhalation of the oxide of zinc, have also been observed among the workmen engaged in twisting and beating the iron wires galvanized with zinc used for securing champagne corks. Four had symptoms of general depression, with sore throat, swelling and ulceration of the tonsils, salivation, fetid breath, colic, and diarrhœa. In one case there was colic and obstinate constipation. These symptoms subsided readily on abandoning the occupation, and did not return when the work-people resumed their work with wires better prepared, and free from loose oxide or carbonate of zinc.[3] M. Blandet has described, as effects of breathing the vapors of zinc fused at the temperature required to melt copper, chilliness, trembling, headache, fainting, vomiting, buzzing in the ears, contusive muscular pains, etc.[4] In these cases it is more probable that the galvanized iron contained impurities of lead, than that zinc oxide was the cause of the poisonous symptoms.

§ 264. With regard to zinc as a poison, the best authorities are in disagreement. Briand and Chaudé, in their excellent work, declare that metallic zinc is not poisonous.[5] If accidents have occurred to workmen who work with this metal, they affirm that these must be due to the presence of arsenic in the zinc. Dr. Taylor[6] mentions

[1] Am. Journ. Med. Sci., Oct. 1850, from the Comptes Rendus.

[2] Briand, Méd. lég., 6ème éd., p. 433.

[3] Am. Journ. Med Sci., Oct. 1850, from Monthly Journ., Aug. 1850.

[4] Journ. de Méd., 1845, p. 76.

[5] P. 672.

[6] Medical Jurisprudence, p. 255.

the case of an epileptic, who took in the course of seven months one pound, having taken in one day seventy grains. Tardieu does not mention zinc as a poison in his work on toxicology. Neither the oxide nor the sulphate of zinc can be regarded as powerful irritants. There is no case, we believe, on record where this agent has been proved incontestably to have caused death. Dr. Schockow[1] gives symptoms of poisoning by those who work in zinc mines, but as these symptoms are similar to those of arsenic and lead, and as these metals are present in the metallic ore used in the smelting furnaces, his evidence is of small account, as compared with the statements of toxicologists of higher authority, who have been concerned with the history of persons who have taken large medicinal doses without accident. These remarks apply equally to the so-called cases of chronic poisoning by zinc oxide.

§ 265. *Sulphate of zinc.*—The prompt emetic action of sulphate of zinc (white vitriol) is the cause of its seldom producing serious effects. The dose usually administered with a view to its emetic operation is from fifteen grains to half a drachm, and unpleasant results have seldom been witnessed from this amount. Dr. Babington once gave thirty-six grains three times a day, for several weeks, without any sickness or other untoward effect being produced; but cases in which the stomach would tolerate such doses as these must be very rare.[2] It should be remembered that the sulphate of zinc is very commonly used as an emetic in cases where the physician wishes to get rid of poisons from the stomach, and that it is never in his mind that he is giving another poison when he administers this drug. The records and experience of the medical profession should outweigh all other doubtful evidence.

§ 266. Christison mentions several cases in which the salts of zinc were supposed to have been the cause of death; yet some of these seemed to him doubtful. "Even in large doses it can hardly be accounted poisonous, as it merely gives rise to vomiting and slight diarrhœa; and that an adulteration to such an amount would always betray itself by its strong disagreeable taste."[3] The best-marked cases are, however, those which have been reported as

[1] Deutsches med. Wochensch., 1879, 208, from H. C. Wood's Mat. Med. and Toxicology, Phila. 1882.

[2] Guy's Hosp. Rep., vol. xii. p. 17.

[3] Christison, op. cit., p. 450; and Orfila's Toxicologie, i. 567.

occurring at Pavia. The first case occurred in the person of a strong woman, who took, by mistake for Epsom salts, a solution of an ounce and a half of sulphate of zinc. She instantly vomited, and then became affected with almost incessant retching and purging for half an hour, which continued afterwards, at short intervals, for three hours, and then gradually diminished. The pulse was frequent and small, and extreme prostration existed, accompanied with distressing restlessness and anxiety; the temperature of the skin was diminished; great pain in the abdomen, limbs, etc., existed, as well as a sense of burning in the throat and stomach. She died thirteen and a half hours after taking the poison, retaining her intellectual faculties to the last. On examination, forty hours after death, the following were the chief appearances observed: great lividity of the skin, congestion of the brain and its membraues, congestion of the lungs, flaccidity of the heart, the inner surface of the stomach covered with a yellowish pultaceous matter, on the removal of which an uniform yellow ochrous color was observed, except towards the great curvature, where it became reddish; a gelatiniform softening (*ramollissement*) of the mucous membrane prevailed, exposing in some parts the submucous cellular tissue. The small intestines were somewhat injected, and contained yellowish matters. In the *second* case a similar dose was taken, followed by nearly the same symptoms, but the patient recovered. In the *third* case a quarter of an ounce was taken, which produced the same symptoms of irritant poisoning, ending in recovery. In the *fourth* case, of which little account is furnished, but which proved fatal, it is stated that "two drachms of sulphate of zinc were detected in the liver and blood, the fluids of the alimentary canal furnishing but little."[1]

One case, in which the sulphate of zinc was supposed to have been given with criminal intentions, became the subject of judicial inquiry in France. An old man died somewhat suddenly, having suffered from severe pain and great heat in the chest and abdomen with violent vomiting and purging. He was not seen by a physician. On inspection, the stomach and bowels were found highly inflamed, and sulphate of zinc was found in the contents of the stomach, and detected in the tissues. The body of a woman who had died

[1] Brit. and For. Med.-Chir. Rev., April, 1849.

two months previously was also disinterred, and sulphate of zinc found in the viscera.[1] Violent enteritis was also observed in a case reported by Krauss.[2] Dr. Gibb has reported the case of a lady who took by mistake about sixty-seven grains of sulphate of zinc in solution. She recovered, and her more serious symptoms were probably owing to two grains of tartar emetic ignorantly administered to her.[3] A case has been recorded by Dr. Ogle, of a drunkard who attempted to commit suicide by cutting his throat. It was believed, but upon no direct evidence, that he had been in the habit of swallowing a strong lotion which he was using for inflamed eyes. The immediate cause of death was not determined, but the reporter states that an examination of the body revealed the presence of sulphate of zinc in the stomach, a white and shrivelled appearance of the mouth and fauces, a condensed, indurated, and tripe-like appearance of the lining membrane of the stomach, and to some degree of the small intestine, and an unusually contracted state of the colon and rectum.[4] Two more recent cases are recorded by Dr. Niemann.[5] In the first, a sickly man died with violent gastric pains and vomiting after a dose administered to him by his wife, and sulphate of zinc was found upon chemical analysis in the contents of the stomach. The second case was one of suicide. In neither was the quantity of the poison determined.

It is extremely probable that most of the recorded cases of poisoning by zinc salts could be explained by the feeble condition of the person reputed to have been poisoned, or by the known impurities of the commercial salts sold under their name, as the best toxicologists, even Christison and Orfila, appear to have had doubts of their poisonous nature.

§ 267. *Chemical examination.*—Sulphate of zinc is a white, crystalline substance, bearing considerable resemblance to sulphate of magnesium, readily soluble in water, and having a disagreeable styptic taste. From its solution, if pure, the oxide is thrown down by the *caustic alkalies*, in the form of a white hydrate, which is easily soluble in an excess of the precipitant. The *sulphide of ammonium* gives a white precipitate, and also sulphuretted hydrogen, provided there is no free acid in the solution. *Carbonate of ammo-*

[1] Journ. de Chimie Méd., 1845, p. 529.

[2] Canstatt's Jahresbericht, 1853.

[3] Lancet, May, 1850, p. 540.

[4] Ibid., Aug. 1859, p. 210.

[5] Henke's Zeitschrift, lxxviii. 219.

nium precipitates carbonate of zinc, also white, which is readily dissolved in an excess of the precipitant. *Ferrocyanide of potassium* also causes a white precipitate. If mixed with carbonate of sodium and exposed to the action of the reducing or inner flame of the blowpipe upon charcoal, the charcoal becomes covered with a coating of oxide of zinc, which is yellow while hot but becomes white upon cooling. If a compound of zinc be moistened with a solution of nitrate of cobalt and heated by the blowpipe, a mass is left which has a beautiful green color. The *sulphide of ammonium* is the most characteristic and unobjectionable of these tests, for zinc is the only metal, with the exception of aluminium, the salts of which are thrown down white by it, but can easily be distinguished because the aluminium salts are also thrown down white by ammonia water, and the precipitate is not soluble in an excess of the reagent. Having discovered the base, the presence of sulphuric acid in the combination may be easily detected by testing with chloride of barium.

§ 268. *Separation from organic mixtures.*—The same process for the destruction of the organic matter may be resorted to that has already been mentioned under the head of arsenic and the other metallic poisons. After the precipitation of the arsenic, antimony, copper, lead, bismuth or mercury, if present, by sulphuretted hydrogen, and the removal of these sulphides together with organic sulphur compounds by filtration, the filtrate, which contains the zinc, should be concentrated and boiled with nitric acid, in order to oxidize any compound of iron which may be present to the form of a ferric salt; the fluid should then be filtered, if it has been rendered turbid by the concentration, and treated with an excess of ammonia water, which will precipitate any iron or aluminium com pounds present in the tissues; then filter from the precipitate produced, and add to the filtrate while hot, or preferably when boiling, a solution of sulphide of ammonium, which will throw down all of the zinc in the form of the white sulphide of zinc, which, after being washed with water, can be dissolved in dilute hydrochloric acid, and this solution, after expelling the sulphuretted hydrogen formed by dissolving the sulphide of zinc in the acid, can be used for performing the above tests for zinc.

§ 269. *Chloride of zinc.*—Several cases of poisoning by a solution of this salt, known by the name of "Sir Wm. Burnett's disin-

fecting fluid," have occurred in England and Canada. This is probably due to the fact that it contains so much hydrochloric acid that it produces a highly caustic action upon the mucous surfaces.

The symptoms observed have been violent epigastric distress, followed by vomiting, and attended with burning heat in the mouth and throat. In a case reported by Mr. Letheby, where a child fifteen months old was poisoned by it, prostration was extreme, and the child died comatose in ten hours. The body was examined twenty-two hours after death. The lips, mucous membrane of the mouth, fauces, and œsophagus, were white and opaque. The stomach felt hard and leathery, and contained a liquid like curds and whey. Its inner surface was corrugated, opaque, and tinged of a dark leaden hue; this appearance ceased abruptly at the pylorus. On digesting the stomach in an ounce of distilled water, the liquid obtained gave white precipitates with ferrocyanide of potassium, carbonate of sodium, sulphuretted hydrogen and acid nitrate of silver, no precipitate being obtained on the addition of a soluble salt of barium. The author concludes, from some experiments, that the chloride of zinc is distinguished from the other salts of the metal by its quick and firm coagulating action on liquid albumen and on the delicate tissues of the body, and that its toxicological action is twofold: first as an irritant and caustic; and, second, by a specific constitutional impression upon the nerves.[1] In the cases reported by Dr. Stratton, although death appeared imminent, the patients were saved by timely medical aid.[2]

Here also we have an instance of a preparation which contains commercial and cheap forms of zinc compounds, as well as other substances of uncertain character and presumed poisonous materials of a corrosive nature, although there can be no doubt as to the corrosive effect of pure chloride of zinc.

Since the first edition of this work several fatal cases have occurred, some of which are referred to by Dr. Webb, in the account published by him of a case in which recovery took place after an ounce of the liquid had been swallowed.[3] A like quantity in another case, and in still another a pint of the solution, were fatal. The symptoms observed in nearly all of the cases were intense burning

[1] Lancet, July 6, 1850.
[2] Med. Exam., Feb. 1849.
[3] Times and Gaz., July, 1856, p. 59.

pain in the epigastrium, extending afterwards to the rest of the abdomen; persistent vomiting first of the contents of the stomach and then of blood with urgent thirst; a cold and pale surface, a failing and gradually extinct pulse, dilated pupils, cramps in the extremities, a husky and whispering voice, and death without a struggle or previous loss of consciousness. After death the body has been found unusually livid; the stomach extremely vascular and purplish, lined with tenacious mucus, and its mucous membrane more or less softened, corroded, and disintegrated in the cardiac half of the organ. The pyloric orifice was constricted, and corrugated, and its lining membrane may be dense and tough, and in color and consistence resemble wash-leather. The duodenum partakes of the discoloration and softening observed in the stomach, and the same changes exist in some degree in the jejunum. The œsophagus is lined with tenacious mucus, or with patches of false membrane. It is remarkable that the mouth is seldom acted upon by the poison.[1]

Death may result after a considerable period from alterations produced in the stomach by the poison. A woman who had taken an ounce of Burnett's fluid, recovered from its immediate effects, but died in ten weeks from the inability of the stomach to retain food. On examination, the stomach near the pylorus was found so contracted as scarcely to admit a probe of the size of a crow's quill.[2]

XVIII. *Tin.*

§ 270. *Chloride of tin.*—This preparation needs little notice: it is an irritant poison, but has seldom given rise to accidents. An old man dried some wet cooking salt in a tin dish upon a stove, and then ate some meat and bread with which he had wiped the dish. He was seized with chilliness, violent pain in the stomach, and the abdomen became swollen and tender upon pressure. A febrile condition was soon set up; but the most striking symptom was salivation, with extreme fetor of the breath, and a grayish discoloration of the gums. They, as well as the tongue and inside of the cheeks,

[1] For cases, see Lancet, Sept. 1857, p. 271; Beale's Archives, 1858, No. iii. p. 194.

[2] Markham, Times and Gaz., June, 1858, p. 595.

became covered with ulcers. By an appropriate treatment, and gargles of chloride of lime, he was restored in a few days.[1]

It is highly improbable that tin and its salts are poisonous *per se*, but on account of other metallic contaminations, and the case above cited is evidently illustrative of this theory, since the tin dish so called could hardly be other than an iron dish plated with tin. The plating of tin either on copper or iron is well known not to be pure tin-plate, the tin of which is insoluble or without irritant or corrosive properties. Among the contaminations of tinned metal is principally lead, which on account of its cheaper price is largely used in place of tin. The proportion of lead to the amalgam used to coat copper and iron, varies from ten to fifty per cent., and when the amalgam coating contains the latter amount, it is easily dissolved in water or the weak organic acids, and hence gives rise to symptoms of lead poisoning in those who may be exposed to its absorption. The large amount of lead used in the tin-lead amalgam causes a bluish white appearance, and hence artisans combine a certain amount of zinc to whiten this coating and to give it a bright white lustre; the amount of zinc added to the amalgam varies from fifteen to twenty-five per cent. Consequently who would dare to impute the well-known symptoms above related as peculiar to tin poisoning, when the amount of tin used in tin-plate is reduced to twenty-five per cent. for the least or seventy-five for the heaviest plated ware? Is it not more reasonable, unless chemical research shall disprove the fact, to attribute these symptoms to poisoning by lead, and in the case above quoted to chloride of lead (from decomposition of the cooking salt) which is the most active and dangerous of lead salts? To those of our readers who desire to know more of this dangerous, because so extensive, use of lead, reference is made to the article Étain,[2] and to the bibliographical references therein contained. The fact is so evident that further discussion here is unnecessary.

XIX. *Silver. Gold. Platinum.*

§ 271. *Nitrate of silver.* (Lunar caustic.)—The appearance of this caustic is well known. Poisoning by it is not, however, of

[1] Memel. Deutsche. Klinik., No. xli. 1851.

[2] Nouveau Dict. de Méd. et de Chir., t. xiv. p. 278 *et seq.*

frequent occurrence. A patient at the Hôpital St. Louis, in Paris, recovered, after having swallowed an ounce in solution. The nitrate of silver was neutralized by the administration of common salt.[1] In a case reported by Krahmer a like quantity produced insensibility to tactile impressions, loss of consciousness, and convulsions. By the sixth day the patient had recovered.[2] The nature of the poison can be readily detected by the grayish color which it communicates to organic matter.

The *terchloride of gold* is also a highly irritant poison, acting very much like corrosive sublimate. Cullerier, the nephew, has seen one-fifteenth of a grain excite, at the second dose, gastric irritation, dryness of the tongue, redness of the throat, colic, and diarrhœa.[3]

The *bichloride of platinum* is a powerful caustic poison. It is rarely used, in medical practice, for the treatment of secondary syphilis.

§ 272. *Chemical examination.*—In the case of all of these metallic compounds, tin, silver, gold, and platinum, the organic matter can be destroyed in the usual way, as recommended in the case of arsenic. Sulphuretted hydrogen precipitates all of these metals in the form of sulphide of the metal from acid solutions of their salts. The sulphides of tin, gold, and platinum are, like the sulphide of arsenic, soluble in sulphide of ammonium, also soluble in aqua regia, or chlorate of potassium and hydrochloric acid, becoming, thereby, changed into chlorides of the metals which can be tested by the appropriate tests. These tests need not be detailed here, since poisoning by compounds of these metals is exceedingly rare, and there is no case of criminal poisoning upon record to the writer's knowledge, so that circumstances would almost always reveal the cause of the poisoning in any given case.

XX. *Iron.*

§ 273. To show in what way iron may sometimes produce serious and perhaps fatal effects, we make no apology in presenting the result as derived from professional experience, and from the most

[1] Am. Journ., 1840, p. 239.

[2] Canstatt's Jahresbericht, 1846, p. 247.

[3] Pereira, Mat. Med.

recent therapeutical work.[1] "Small doses of the insoluble and less irritating preparations of iron produce at first no marked symptoms, except some improvement of appetite and a tendency to constipation. If the treatment is long continued, or larger quantities, and the more irritating preparations are used, there appears, according to the susceptibility of the patient, decided gastric irritation, as shown by a furred tongue, a sensation of weight in the stomach after eating, or gastralgia and pyrosis. Constipation and headache are the more common symptoms." This is the therapeutical effect of iron used as a medicine. When larger doses are used, or doses which produce the last-named effects are continued in spite of the constitutional disturbances, the poisonous properties become pronounced, or, in other words, the exaggerated therapeutical effects are observed, and the patient or victim will suffer from an intense inflammatory action of the whole alimentary tract, and will suffer from the symptoms of a gastro-intestinal irritation, marked by pains similar to those which have been described under the head of irritants, pain, nausea, vomiting, diarrhœa, and serious constitutional disturbance. These symptoms will follow inordinate doses of any preparation of iron, the most irritant of them causing the symptoms from smaller doses, the less irritant from larger doses. The cases in which medicinal iron preparations would be used with suicidal or criminal intent are naturally rare.

§ 274. *Sulphate of iron.* (Copperas; Green vitriol.) *Symptoms.*—A case of supposed criminal poisoning with this substance is related by Dr. Christison. A girl, four years of age, and previously in good health, was attacked with violent vomiting and purging immediately after breakfasting on porridge, and died in the course of the afternoon of the same day. The porridge had a blue color, and it was proved that a woman in the house had purchased both this salt and the sulphate of copper. The body being disinterred four months after death, the stomach was found soft, gelatinous, and of an uniform intense black color through the whole thickness of its parietes, and the entire alimentary canal lined with a thick layer of jet black mucus "from the pharynx down to the very anus." There was no evidence found of the presence of

[1] Edes's Therapeutic Handbook of the United States Pharmacopœia, New York, p. 126.

copper, but abundant proof was obtained of the presence of iron, both in the textures of the stomach and the black mucus which lined it.[1] More recently, a case somewhat similar has been observed by Orfila. It was that of a child, aged fifteen months, who died after purging and vomiting a black fluid. On opening the body ten days after burial, the stomach was found to be filled with a greenish fluid, and the vessels of the lungs and brain were gorged with black blood. M. Orfila detected sulphate of iron, in notable quantities, in the portions of the abdominal contents forwarded to him.

§ 275. *Chemical examination.*—It should not be forgotten, in making a chemical examination of the viscera, in cases of supposed poisoning with the salts of iron, that iron is a normal constituent of the body and food. The saliva contains traces, according to Prof. Bernard, Besanez, Wright and Enderlin, Humbert and Lassaigne. The intestinal secretions, as well as the gastric juice and the bile, also contain a considerable amount of iron, which comes mainly from the food. A man of average weight secretes about eighteen grains a day with the gastric juice, and about twenty grains with the bile during the same period. In fact all the secretions and excretions from the body contain iron varying in amount from a trace to several grains, so that two and a half per cent. of the ashes of the burned body are oxide of iron. Moreover, that all the iron of the body does not come from food is shown by a well-known physiological experiment of feeding dogs on food almost deprived of iron, where it is shown that the elimination of iron far outweighs its absorption. A dog, which was fed for twenty-seven days on food containing a total amount of 39.5 milligrammes, eliminated 89.5 milligrammes, and afterwards received 116 milligrammes, and excreted 144.5 milligrammes. The blood in man contains by estimation a little more than one-half of one per cent. of iron by weight, and a man of average weight would have in his blood about three grammes or forty to fifty grains of iron. On account, therefore, of the large amount of normal iron in the animal economy, it is necessary in legal analyses to be able to isolate a very large amount, much more than the normal. Several criminal cases have been investigated in France,[2] so that such analyses are

[1] Pereira, Mat. Med., page 393.

[2] Journal de Chimie Médicale, 1874, page 149.

sometimes necessary, but usually such large doses have been taken in these cases that large amounts remain in the stomach or intestines adherent to the lining membrane, either changed into the form of sulphide, which imparts a black color to the membrane, or unchanged, if death has taken place very rapidly. If the iron exists in the contents of the stomach or intestines in the form of sulphide or sulphate of iron, it can be easily separated by treating with hydrochloric acid, straining and filtering, when the filtrate will contain the iron which can be separated by boiling with nitric acid, to convert it into the form of ferric chloride, and precipitating by the addition of ammonia water in the form of ferric hydrate, a reddish-brown precipitate. This, after being washed, can be dissolved in hydrochloric acid and the solution of chloride of iron tested; (1) by adding a solution of sulphocyanide of potassium, which gives a blood-red solution of sulphocyanide of iron, which is not bleached by the addition of free mineral acids nor by a solution of corrosive sublimate; (2) by neutralizing or rendering slightly alkaline with sodium hydrate, adding a solution of ferrocyanide of potassium and then acidulating with hydrochloric acid, when a dark-blue precipitate will be formed (Prussian blue); and (3) by rendering alkaline with ammonia water, which will precipitate the hydrate of iron, and then adding sulphide of ammonium, which will change the hydrate of iron to the form of the black sulphide of iron.

§ 276. *Chloride (Muriate) of iron.*—The medicinal tincture of this salt of iron has frequently given rise to serious and fatal accidents. The symptoms produced by it are very much like those of the corrosive acids, viz., heat, dryness, and swelling of the throat, with a burning pain in the stomach and in the course of the œsophagus, vomiting of blood, and inky evacuations. Its corrosive properties seem to be due to the presence in it of free hydrochloric acid. Dr. Christison relates a case in which death occurred in about six weeks after an ounce and a half of the tincture had been swallowed. A case of recovery after three ounces of the concentrated tincture had been swallowed is reported by Sir William Murray.[1]

A gentleman, aged seventy-two, swallowed three ounces of it by mistake. He was found "tossing about in the utmost consternation

[1] Am. Journ. Med. Sci., July, 1849.

and agony ; his tongue was swelled, and protruded from the mouth ; its skin was parched and peeling off, while ropy mucus flowed from the mouth and nose ; the eyes seemed starting from their sockets ; the respiration was noisy and laborious, and suffocation seemed to be impending. During this time his hand was riveted to the region of the stomach, as the principal seat of pain ; the palate and the interior of the mouth were burned, and presented a parboiled appearance." The acid was first neutralized by an alkaline mixture, and this treatment was followed by demulcents and laxatives. The gentleman rapidly recovered.[1] Several other cases of recovery from large doses are recorded, which it is not necessary to particularize.

The tincture of the chloride of iron, as it is now called (tincture of the muriate of iron), contains a notable amount of free hydrochloric acid, especially when it has been recently prepared, and hence poisoning by this substance is similar to that of weak hydrochloric acid (§ 55), and also intensified by the irritant action of the salt of iron called chloride.

XXI. *Bismuth.*

§ 277. *Subnitrate of bismuth.*—A man subject to water-brash took two drachms of this preparation by mistake. He was immediately attacked with burning in the throat, vomiting, purging, cramps, and coldness of the limbs, his pulse became intermittent, and he had a constant metallic and nauseous taste. On the third day he had hiccough, labored breathing, and swelling of the hands and face ; suppression of urine was then discovered to have existed from the first. On the fourth day, swelling and tension of the abdomen were added to the pre-existing symptoms ; on the fifth day, salivation ; on the sixth, delirium ; on the seventh, swelling of the tongue, and enormous enlargement of the abdomen ; and on the ninth, he expired. The tonsils, uvula, pharynx, and epiglottis were gangrenous, and inflammatory redness with spots of gangrene, existed throughout the whole intestinal canal.[2] Sobernheim explains the poisonous effects in the above case and in one observed by himself, by supposing that the patients' stomachs contained bi-

[1] Sir Wm. Murray, Dub. Med. Press, Feb. 1849.

[2] Christison, Treatise on Poisons, 2d ed., Edinburgh, p. 444.

tartrate of potassium enough to convert the subnitrate into an acid nitrate of bismuth, which is stated to be an active irritant.[1] Certain it is that pure subnitrate of bismuth is constantly given in unmeasured doses without any toxical effect whatever. It has been found that this preparation of bismuth is often carelessly made, so that the arsenic which is commonly found in bismuth ores has not been previously excluded. M. Cornut recommends that it should be tested before being dispensed for medicinal purposes. Moisten half a drachm of the subnitrate of bismuth with a sufficient quantity of *pure* sulphuric acid, evaporate to dryness in a small porcelain capsule, wash the residue with a little distilled water, filter, and put the filtrate into a Marsh's apparatus.[2]

The above and one other[3] are the only recorded cases of poisoning by this drug, and it is exceedingly doubtful whether the effects may not be attributed to impurities in the agent or to some other cause. The first case was reported in 1831, and since then there has been but one death attributed to the use of this drug. Many years ago arsenic was not an infrequent combination with bismuth in the form of arseniate of bismuth owing to its incomplete preparation, and even at the present time a trace of arsenic may be found in almost all of the preparations of subnitrate or subcarbonate of bismuth, although the process recommended by the U. S. Pharmacopœa for the manufacture of these compounds is intended to eliminate all but the merest trace of arsenic from them. The largest amount of arsenic found by analysis of various preparations of subnitrate and subcarbonate of bismuth recorded is, so far as the writer has been able to learn, one-sixth of one per cent. The presence of the arsenic as a contamination in these bismuth preparations is due to the fact that the bismuth ores, from which they are manufactured, are arsenical, and it is very difficult to separate the arsenic from the bismuth in working upon a large scale.

§ 278. *Chemical examination.* — Bismuth is readily separated from organic mixtures by the same process recommended for the separation of arsenic and the other metals. The sulphide of bismuth is separated from the other sulphur compounds in the same

[1] Arzneimittellehre, 6te Aufl. p. 268.

[2] Association Med. Journ., June 17, 1853.

[3] Arzneimittellehre, 6te Aufl. p. 268.

way as sulphide of lead; it can then be dissolved in nitric acid, the excess of acid expelled by evaporation, and the solution of nitrate of bismuth employed for the tests for bismuth, the principal ones of which are the following: (1) Sulphuretted hydrogen precipitates bismuth in the form of the black sulphide, which is insoluble in hydrochloric acid but soluble in nitric acid. (2) Ammonia water precipitates the white hydrate of bismuth. (3) A solution of the nitrate or chloride of bismuth, when treated with a large excess of water, becomes turbid on account of the decomposition of the salt, the subnitrate or subchloride, which is insoluble, being precipitated. This precipitate is insoluble in tartaric acid. (4) Carbonate of sodium precipitates the basic carbonate of bismuth, a bulky white precipitate. (5) Chromate of potassium precipitates the yellow chromate of bismuth, which is soluble in dilute nitric acid but insoluble in potassium hydrate, thereby differing from the chromate of lead. (6) A solid compound of bismuth mixed with carbonate of sodium and heated on charcoal with the blowpipe, gives brittle globules of metallic bismuth, the charcoal at the same time becoming coated with oxide of bismuth, which is orange while hot, but becomes yellow when cold.

§ 279. As the subnitrate and subcarbonate of bismuth are quite freely used in the treatment of diarrhœa and other intestinal irritations, in the dose of from two to six drachms, without any apparent danger to life, they can hardly be called corrosive poisons. In fact, Headland[1] asserts that the subcarbonate of bismuth is not more easily absorbed in the alimentary canal than charcoal. On account of this frequent use of these bismuth preparations in the treatment of intestinal irritations they are often given for the irritation caused by irritant poisons, so that their detection becomes a matter of very great importance in toxicological analyses.

XXII. *Chromic Acid and its Salts.*

§ 280. *Bichromate of potassium.* — This salt, being extensively used in dyeing, has given rise, in several instances, to accidental poisoning. Locally applied, its action is irritant, causing, in the workmen who use it, troublesome sores and ulcerations upon the hands. Taken in poisonous doses internally, its action is highly

[1] On the Action of Medicines, p. 91.

irritant also, and death has been caused by it with the symptoms usually attending the action of irritant poisons. Mr. Wilson, however, relates a case in which death was produced by it without any vomiting or purging having occurred.[1] Several fatal cases have occurred in Baltimore. The following was communicated by Dr. Baer to Professor Ducatel: A laborer, aged 35, received a small quantity of the solution into his mouth on attempting to draw it off from a refiner by exhausting the siphon by suction. His first impression was, that he had spit it out; but only a few minutes elapsed before he was seized with great heat in the throat and stomach, and violent vomiting of blood and mucus. The vomiting continued until just before his death, which occurred in five hours. On section, the mucous tissue of the stomach, duodenum, and about one-fifth of the jejunum, was found destroyed in patches. The remaining parts of it could be easily removed by the handle of the scalpel.[2] A boy, who swallowed about two ounces of bichromate of potassium, was seized in half an hour with vomiting, and became almost totally insensible. He was pale and collapsed, the pupils were dilated and fixed, the pulse feeble, and he had also cramps in the legs. An emetic of sulphate of zinc was given, and the stomach-pump used, until the pinkish color of the washings obtained by it had ceased. He had an attack of gastro-intestinal inflammation, from which he did not recover for four months.[3] Dr. A. McCrorie[4] reports the following case of poisoning by bichromate of potassium. A child twenty months old swallowed a piece of "red chrome," about five or ten grains in weight, and was found semi-comatose with a livid complexion. The eyes were half closed and pupils were dilated; the respiration was difficult and wheezy, the pulse almost imperceptible. There was tenderness on pressure over the abdomen and very marked at the pit of the stomach causing him to cry and draw up his legs. He also had vomiting and purging. He improved somewhat, but two hours later he again became comatose, fell into a state of collapse with coldness of the surface, and was bathed in a cold and clammy sweat. He died in nine and a half hours after swallowing the poison. A man[5] sixty years

[1] Med. Gaz., vol. xxxiii. p. 734.
[2] Beck, vol. ii. p. 666.
[3] Guy's Hosp. Rep., 1850, p. 214.
[4] Glasgow Med. Journ., May, 1881.
[5] Ibid., July, 1881, reported by Wm. A. McLachlan.

old swallowed three drachms (180 grains) of bichromate of potassium dissolved in cold water. In quarter of an hour afterwards he showed the signs of irritant poisoning, such as vomiting and purging accompanied by violent abdominal pain. When first seen by Dr. McLachlan, two hours after swallowing the poison, he was reclining in a chair, moaning and complaining of these cramps; surface of body cold with shrivelled hands and feet, the skin wrinkled and dusky, like the collapse observed in cholera. The pulse was weak and barely perceptible, respiration was hurried, and urine was suppressed. His mental faculties were unimpaired. After a stimulating treatment and application of heat he gradually improved, and two days later was well, except that his urine and skin were yellow. He fully recovered. The suppression of the urine is probably due to inflammation of the kidneys produced by the chromic acid. Experiments upon animals have shown[1] that after the subcutaneous injection of chromic acid animals suffer from vomiting, diarrhœa, albuminuria, and finally die in a few days; after death inflammation of the kidneys is found. The same results were produced in rabbits by the injection of a neutral chromate (yellow chromate of potassium).

§ 281. *Chromate of lead.* (Chrome yellow.)—Although this substance is insoluble in water, and under many circumstances in the stomach and intestinal fluids, sometimes it gives rise to acute poisoning owing to its decomposition after it enters the body. That such a decomposition of the chrome yellow does take place is shown by the fact that in one case of poisoning reported by R. C. Smith,[2] chromic acid was detected in the urine; this was a case of professional poisoning, the patient being employed in weaving yarn colored with chrome yellow.

The frequency with which this poison is used as a pigment for coloring articles in common use, such as children's playthings, and even confectionery, shows the importance of a consideration of this substance, and is the apology for the introduction of the following extracts of several cases of poisoning by it made by the writer for the Boston Medical and Surgical Journal.[3]

"Two fatal cases of acute poisoning by chrome yellow are reported

[1] Gergens, Arch. f. exp. Path., vi. p. 148.

[2] Brit. Med. Journ., 1882, p. 8.

[3] July 9, 1874, and Feb'ry, 7, 1878.

by Dr. von Linstow.[1] These cases occurred in children, aged, respectively, $1\frac{3}{4}$ and $3\frac{1}{2}$ years, and the poisoning was caused by sucking an unknown number of small, yellow substances, which had been used for ornamenting pastry, and which consisted of gum tragacanth and chrome yellow. A piece measuring 13×5 millimeters yielded, on analysis, 0.278 grm. of gum tragacanth, and 0.0042 grm. of chromate of lead.

"Chromate of lead, on account of its insolubility, has never been considered an active poison, and the fact that it is used so largely as a pigment for coloring not only ordinary substances, but also children's playthings, and even articles intended for food, such as confectionery, etc., renders these cases of more than ordinary interest. The extent of its use in confectionery can be seen by an examination of the report of analyses of confectionery, by H. B. Hill.[2] Thus 77 samples, both white and colored, were analyzed; 21 were colored yellow, and in 17 of these the pigment consisted entirely of chrome yellow, in 2 partially; of 12 specimens which were of an orange color, the pigment in 9 consisted entirely of the chromate of lead, and in 2 partially; 7 specimens of green were examined, 6 of which contained chrome yellow mixed with Prussian blue in five specimens, and with Scheele's green in the other. Of the 77 specimens examined, 36, therefore, contained the chromate of lead.

"The symptoms of poisoning did not commence until several hours after the ingestion of the chrome yellow, which took place between 9 and 11 A. M. Both children were taken sick at the same time (between 2 and 3 P. M., of the same day) with vomiting, which lasted for several hours. The vomitus was yellow in color. There was great prostration and extreme thirst, but no diarrhœa and no pain. On the second day, both had a hot and red countenance, and were stupid. The younger, about twenty-four hours after the commencement of the symptoms, had a slight diarrhœa and convulsions, which continued until death, which took place in forty-eight hours. On the third day, an erythematous eruption appeared on the chest and abdomen of the elder. He was dull and stupid, and the temperature in the axilla was 39.5° C. On the fourth day, the pulse and respiration became irregular, the breath extremely fetid, stupor

[1] Vierteljahressch. für gericht. Medicin, Jan. 1874.

[2] Mass. State Board of Health Report, 1873, p. 390.

and unconsciousness came on, and the patient died five days after the ingestion of the poison.

"After death, the mucous membrane of the stomach and duodenum was found swollen and loose, so that it could easily be raised from the submucous tissue; it was inflamed, as was also that of the œsophagus, throat, and larynx. In some places, the mucous membrane of the stomach and duodenum was entirely destroyed, and in one spot perforation had taken place, showing that the chrome yellow had a corrosive action. These appearances were probably not caused by the chromate of lead, as such, but by soluble compounds formed after the pigment had lain in the stomach some time, and had been decomposed.

"Besides the above appearances, there were found also hyperæmia of the brain and its membranes, beginning fatty degeneration of the liver, commencing icterus, hyperæmia of the kidneys, suppurative pyelitis, and a softened spleen; conditions which are often seen after death from poisoning by other corrosive poisons.

"The number of these yellow ornaments ingested by the children could not have been more than six, since only seven were given them to play with, and one was afterwards recovered. If each child had eaten three of these, the fatal dose was less than 0.01 grm., or between $\frac{1}{5}$ and $\frac{1}{6}$ of a grain, of the chromate of lead."

"Leopold[1] reports five cases of this form of poisoning, one of which proved fatal. The patients were employed in weaving cloth colored with chrome yellow (chromate of lead), which was quite loosely applied to the thread, so that a portion of the pigment was easily detached and became diffused throughout the air of the room. The patients were affected with a yellow-coated tongue, yellow sputa, loss of appetite, malaise, in some cases vomiting, pain in the region of the stomach and umbilicus, obstinate constipation and debility. The feces were yellow. These symptoms disappeared in a few weeks after the removal of the cause, except in the case of an infant nine weeks old, who died in six or eight days after the beginning of the symptoms, which, however, did not appear until about three weeks after exposure to the infected atmosphere. The symptoms in this case were fever, restlessness, shrieking, several yellow-fluid

[1] Vierteljahresschrift für gerichtliche Medicin, xxvii. page 29.

stools daily, redness of the skin over the chest and abdomen, parched lips, and, just before death, short respiration.

"After death there was found inflammation and perforation of the stomach, the same appearances which were seen in the two cases previously reported by Dr. von Linstow,[1] caused by ingesting the chrome yellow. None of the poison could be detected in any of the organs except the lungs, in which 3.6 milligrammes were found.

"This is the third fatal case of chrome-yellow poisoning reported within a few years."

§ 282. *Chemical examination.*—(1) Chrome yellow when heated with carbonate of sodium upon charcoal with the blowpipe, yields a globule of metallic lead. (2) It is insoluble in dilute nitric acid, but soluble in potassic hydrate, thereby differing from the yellow chromate of bismuth. (3) When fused in a crucible with sodic carbonate and nitre, chrome yellow is decomposed, chromate of the alkali and carbonate of lead being the result; these can be separated by treating the fused mass with water, which will dissolve the chromate of the alkali, and will leave undissolved the carbonate of lead; the filtrate may then be tested for chromic acid, and the undissolved portion for lead as given under the head of lead.

§ 283. The tests for chromic acid are: (1) Chromic acid and chromates are either red or yellow, and when soluble in water give red or yellow solutions. (2) Sulphuretted hydrogen changes the color of such solutions, if previously acidified with hydrochloric acid, from yellow or red to dark green, owing to the formation of the sesquioxide of chromium, which change may also be caused by other reducing agents. (3) A solution of chloride of barium causes in aqueous solutions of chromates a yellow precipitate of chromate of barium, which is insoluble in acetic acid but soluble in dilute hydrochloric or nitric acids. (4) A solution of sugar of lead produces a yellow precipitate of chromate of lead (chrome yellow), which is, as mentioned above, insoluble in nitric acid but soluble in potassic hydrate. (5) Nitrate of silver produces in aqueous solutions of a chromate an orange-red precipitate of chromate of silver, which is soluble both in nitric acid and ammonia water, and which is not produced in the presence of chlorides until all of the chlorine has first been precipitated as chloride of silver. (6) Solutions of bis-

[1] See above p. 288. (See also Appendix.)

muth salts produce a yellow precipitate of chromate of bismuth soluble in nitric acid but insoluble in potassic hydrate.

§ 284. *Separation from organic mixtures.*—The same method may be used for the destruction of the organic matter as for lead and arsenic. By that process the chromic acid will be converted by the sulphuretted hydrogen into the sesquioxide of chromium, which will remain in solution and must be precipitated from the filtrate, after boiling off the excess of sulphuretted hydrogen, by ammonia water in the form of the hydrate of the sesquioxide of chromium. This can be collected upon a filter paper, dried, fused in a crucible with carbonate of sodium and nitre, the fused mass extracted with water, and the tests for chromic acid performed with the filtrate, as mentioned above. The lead would be separated as described in speaking of the salts of lead.

In the case of small amounts of colored substances, such as colored spices, confectionery, etc., the material can be at once fused with carbonate of sodium and nitre, which will destroy the organic matter and leave the chromic acid and the lead as mentioned above [§ 282, (3)].

CHAPTER VI.

IRRITANT POISONS—Continued.

I. *Colchicum Autumnale.* (Colchicum; Meadow Saffron.)

§ 285. *Symptoms.*—The seed and cormus of this plant, and it is said the leaves[1] and flowers[2] also, are capable of producing violent poisonous effects. The symptoms are an acute, gnawing pain in the stomach, vomiting and purging, tenesmus, reduced pulse, and great debility. They are said to resemble occasionally those observed in Asiatic cholera, from being sometimes attended with cramps in the various parts of the body, ice-cold surface, purging of rice-water stools, suppression of urine, and general collapse. The purging is first of a serous discharge, then composed of a small amount of mucus, and sometimes it is bloody. Abdominal pain does not always exist. Convulsions are sometimes present, and may just precede death. Not unfrequently there may be a heaviness or actual pain in the muscles followed by paralysis of motion and general collapse. The pulse partakes of the above-mentioned characteristics, becoming weak and thready. The action of colchicum upon the intestines is not that of a local irritation, like some of the irritant poisons previously described, but it is first absorbed into the

[1] Bleifus, Repertor für die Pharmacie, lxix.

[2] Magazin für Pharmacie, xxx.

blood and the resulting diarrhœa, prostration, etc., are the direct effects of intoxication, as has been proved by administering the drug by subcutaneous injection, nor is the rapidity of death in proportion to the size of the dose administered. Colchicum owes its action to the presence of an active principle, called colchicia, colchicin, colchiceina, or colchiceia (as it is variously written), and which is crystallizable, neutral, and soluble in water, and about eighty to one hundred times stronger than the cormus from which it is derived; a dose of one-third to one-fifth of a grain of colchicin produces poisonous symptoms in man,[1] though Husemann has reported a case of recovery after two-thirds of a grain. Prof. Wood[2] states that death has followed a dose of two and a half drachms of the wine of colchicum root.

Of *colchicin* Casper remarks that it is one of the most deadly poisons, and, in its power, hardly surpassed by phosphorus. He relates four cases of fatal poisoning in males between the ages of fifteen and forty. In each of these the dose was between two-fifths and one-half of a grain of colchicin; but it occasioned death rapidly, with violent vomiting, purging, and collapse.[3]

§ 286. The seeds and cormus are poisonous, but the dose of the medicinal tincture, or the quantity of the crude seeds or bulb, that is requisite to produce the fatal effects described, is not precisely known. The officinal dose of the dried bulb or of the seeds is six or eight grains, and of the wine of the seeds from one-half to one and a half fluidrachms, and of the wine of the bulb ten drops to half a fluidrachm. A case has been reported in which a person swallowed a wineglassful of the tincture by mistake; he was soon seized with violent pain in the stomach, and vomiting, and died on the next day of exhaustion.[4] In another case, reported by Mr. Feraday, the same quantity was taken. The symptoms did not appear for an hour and a half; there was then urgent pain and vomiting, followed by great exhaustion, purging, and tenesmus. In this case the intellect was unaffected. The patient died in forty-eight hours. (The uniformity with which, in the reported cases of poisoning by colchicum, either no mention is made of any cerebral disturbance, or, on the

[1] Jour. für Pharmacodynamik, ii. p. 561.

[2] U. S. Dispensatory.

[3] Gericht. Med., i. 402.

[4] Med. Times and Gaz., 1853, 1.

other hand, an express statement is given that the intellect was not at all impaired, justifies the position which we have given to this substance, viz., among the irritants, instead of its customary place among the narcotico-acrid poisons.) Ollivier met with two cases of death within twenty-four hours, in consequence of a tincture being taken which contained the active part of forty-eight grains of the dry bulb, and the period mentioned is the shortest in which death is recorded as the direct effect of colchicum. Dr. Christison states that he has known very violent effects produced by half an ounce taken by mistake, although most of it was brought away by emetics in an hour; and that, in medical practice, he has seldom seen the dose of a sound preparation gradually raised to a drachm thrice a day without such severe purging and sickness ensuing as rendered it prudent to diminish or discontinue the remedy.[1]

The preparations of colchicum made from the root and seeds are of more constant strength, since the seeds mature at a given season and contain a pretty uniform amount of the active principle, colchicin; the cormus, on the other hand, is present during nearly the entire year on the plant, and the amount of active principle in it varies according to the season at which the fresh cormus is gathered; on this account the United States Pharmacopœia (1880) directs that only the root and seeds should be used in official preparations.

Dr. Geo. W. Major[2] reports seventeen cases of accidental poisoning from one bottle of wine of colchicum seeds, which would hold sixty-four fluidounces. The constitutional disturbance was proportionate to the amount of poison taken, with due consideration for age, vigor of health, strength, and previous habits. In the seven fatal cases the dose varied from four to eighteen ounces, except in one feeble woman who died after a dose of two ounces. The fatal cases, with the exception of a young lad and the feeble woman above referred to, were persons of dissipated habits. The children seemed to be more capable of throwing off the effects of doses, which, to them, ought to have been quite as fatal as very much larger ones to adults. When small doses, as half an ounce to one and a half ounces, were taken, convalescence progressed

[1] On poisons, p. 667. For a collection of curious cases, *vide* Ed. Month. Journ. of Med. Sci., 1852, by J. McGrigor Maclagan.

[2] Can. Med. and Surgical Journal, Dec. 1873.

quite rapidly; though for two or three days there were signs of irritability of the stomach and bowels.

In the vast majority of cases the symptoms were similar. A feeling of malaise and uneasiness was experienced in less than an hour, soon followed by vomiting and purging, which lasted until death took place in the fatal cases, that is, from nineteen to twenty-nine hours, and, in the cases of recovery, for several days. The vomiting was first of the contents of the stomach, then of bile and mucus, and finally of a pea-soup character. The pain over the stomach was quite severe, but not so agonizing as usually stated by authorities; they complained but little. Pressure over the region of the stomach and bowels caused pain which was relieved when the hand was removed. The purging was preceded by griping pains, and the intestinal evacuations were at first of the natural contents, then bilious stools and rice-water discharges (compared by one of the patients to clear soapsuds). The stools became involuntary, and were voided unconsciously. Heat and pain were felt in the arms, cramps in the legs, toes, and fingers. In one case very great pain was felt in the left shoulder. There was no headache, but great pain was felt in the throat with hoarseness and great thirst. The nose was pinched and blue, and the lobes of the ears and nose were blue and cold. Countenance anxious and livid, and the face appeared like one shivering with cold. The pulse was 135 to 145, and in bad cases weak and thready. In the convalescence the heart's impulse and radial pulse grew stronger. Mind and intellect were notably clear, and there were no nervous symptoms. When the purging was not excessive, a considerable amount of thick, dark-colored urine was voided. None slept; all sat up before dying. Breathing was very rapid, but not embarrassed. One case was slightly convulsed before death. Collapse was very marked, and resembled that of Asiatic cholera. The features for twenty hours after swallowing the wine were pinched and drawn. There was no *post-mortem* examination in any of the fatal cases.

§ 287. The *post-mortem appearances* in *fatal* cases may be those of inflammation. In a case in which a decoction made with a tablespoonful of the seeds had been taken, and the inspection was made twenty-three hours after death, a remarkable rigidity, especially of the abdomen was noted. The muscles were of a deep-blue color, as if they had been dried in the air. The heart was covered with spots

of a black, violet, and brown color; the stomach was of a light-violet color, and the veins of it and of the intestines were much engorged with blood. The other organs had a healthy appearance.[1]

The following is an analysis of the lesions found in seven cases of fatal poisoning by colchicum. The skin upon the back and sides was usually purple, livid, violet, or greenish, and decomposition was unusually rapid. The lungs and brain were gorged with dark, imperfectly coagulated, and pitchy blood, and so were the veins of the trunk. In several cases the gastro-intestinal mucous membrane was intensely congested, and in some places softened; in about one-fourth of the number its color was normal. Sometimes ecchymoses were found under the mucous coat, and sometimes also the discoloration extended to the peritoneal membrane. Whether the peculiar difficult coagulability of the blood is due to the poison or to the death caused by pulmonary congestion and asphyxia is not easily determined. Recent investigations show that the chief pathological appearances are an œdematous, congested, and ecchymotic condition of the intestinal mucous membrane with extravasations of blood into the liver and intestinal canal, and sometimes also in the stomach.

§ 288. *Chemical examination.*—The possibility of demonstrating the fact of poisoning by colchicum, by means of the extraction of its active principle, *colchicin*, from the *contents of the stomach*, etc., has been shown by Schacht and Wittstock, of Berlin.[2] Four persons were poisoned by drinking tincture of colchicum by mistake for "schnapps;" but the fact being somewhat uncertain, a chemical examination of the contents of the stomach was instituted with the view of detecting, if possible, the presence of colchicin. Previous to the experiment upon the viscera, however, what remained of the suspected liquid was examined, and also a tincture known to be the officinal preparation of colchicum. In both cases colchicin was obtained, and it was found that half an ounce of the officinal tincture of the drug yielded about four and a half per cent. of pure colchicin.

The contents of the stomach were mixed with a large quantity of alcohol, to which a few drops of hydrochloric acid had been added, then shaken, the liquid portion filtered off and evaporated to the consistence of a thin syrup. This residue was dissolved in distilled

[1] Neubrandt, in Ed. Med. and Surg. Journ., July, 1840.

[2] Casper's Vierteljahrschrift, Jan. 1855.

water, by which much fat was separated, then filtered, carefully evaporated, alcohol added again as long as any foreign material appeared, and then filtered and evaporated as before. To the mass now obtained, after having been reduced by evaporation to about eight ounces, half a drachm of calcined magnesia was used to free the colchicin, and, after a time, three ounces of ether added. This was allowed to evaporate spontaneously after being filtered. The residue was taken up again by water, and evaporated in a watch-glass. The residue gave, with tincture of galls, chloride of platinum, and tincture of iodine, all the reactions of *colchicin*.

As mentioned above, colchicin is readily soluble in water; it is also soluble in alcohol, chloroform, benzol, and amyl alcohol; soluble with difficulty in ether, and almost insoluble in naphtha. It is usually obtained in the form of an amorphous or resinous body, but has been obtained in the crystalline form. It has a very persistent bitter taste. It is soluble in dilute acids and alkalies, forming a yellow solution. By long-continued action of these reagents, or when warmed, it is converted into an isomeric body called colchicein, which is also very bitter, and is slightly soluble in water. The principal tests for colchicin are the following: (1) It gives a gamboge-yellow color when treated with concentrated sulphuric acid, even when only a small fraction of a grain is tested. (2) If a trace of nitric acid or a small crystal of nitre be introduced into the above yellow-colored solution (*vide* 1), a blue-violet color is at once produced, which rapidly changes to a brown and finally to a yellow again. (3) Concentrated nitric acid (sp. gr. = 1.4) gives a violet color which changes quickly to a dirty brown; if a little water is now added, the solution becomes light yellow, which is colored blood-red by a solution of sodium or potassium hydrates. (4) It is precipitated from dilute solutions by tannic acid, terchloride of gold, solution of iodine in iodide of potassium, potassio-bismuth iodide solution, phosphomolybdic acid solution, and from concentrated solutions by platinic chloride. It is not precipitated from its solutions by potassio-cadmic iodide, potassio-mercuric iodide (Mayer's reagent), corrosive sublimate, picric acid, or ferrocyanide of potassium, thereby differing from many of the other organic poisons.

§ 289. The *isolation of colchicin from organic mixtures* depends upon the fact that, if a solution of colchicin in water is acidulated with sulphuric acid, and this solution be shaken with chloroform or

benzol, the colchicin will pass over from the acidulated water into the chloroform or benzol, which separate from the water on standing for a few minutes, and can be removed either by pouring off the upper fluid or sucking up the lower fluid into a pipette. The process, as recommended by Dragendorff,[1] is as follows: Treat the finely divided tissues, or the fluids which are to be analyzed with water, to which enough sulphuric acid has been added to give the fluid a strong acid reaction with blue litmus paper, warm gently for a while, filter, and wash once or twice with more acidulated water; mix these filtrates together and evaporate them over a water-bath to a small bulk, transfer this residue to a flask and add to it four or five times its volume of strong alcohol, which will precipitate albuminous substances and some inorganic salts; filter, wash once or twice with alcohol, evaporate off most of the alcohol, and shake the remaining fluid, which will be an impure aqueous acid solution of the colchicin, in a flask first with naphtha, which will remove certain impurities and which can be poured off from the aqueous solution after standing awhile, and then add chloroform or benzol, preferably the former, and shake again; the chloroform will, as mentioned above, remove the colchicin from the water; remove the chloroform after it has separated from the water with a pipette, allow it to evaporate upon a watch-glass, when the colchicin will be left as a yellowish amorphous mass upon the watch-glass, and can be subjected to the above tests. The best substances to subject to chemical analysis in cases of fatal poisoning are the kidneys, large intestines, and feces, since usually the poison has gotten out of the stomach and liver before death takes place.

II. *Drastic Purgatives.*

§ 290. The chief articles enumerated under this name are *jalap*, *scammony*, *gamboge*, *colocynth*, *croton oil*, and *elaterium*. With the exception of the last two, they are seldom given singly in medical practice, but generally combined with each other and with milder purgatives, or with mercurials, and always in small doses. That they may give rise to fatal consequences from over-purging is not only possible, but is demonstrated by cases upon record. Being the most usual ingredients of quack cathartic medicines, especially

[1] Die Ermittelung von Giften, St. Petersburgh, 1876.

in the pilular form, they have often been taken in large doses, and have thus caused death by the exhaustion arising from over-purging. One element in the consideration of cases in which death is attributed to the use of any of these drugs should not be forgotten, viz., that the very young, or, on the other hand, the aged, cannot bear the operation of violent purgative medicine with the same impunity as those of other periods of life; and also that those who are already enfeebled by disease may readily perish from the effects of a comparatively small dose. In general, where the quantity of a drastic purgative taken has been very large, there will be found evidence of inflammatory action in the intestines.

§ 291. *White and black hellebore*, besides being violent irritants of the stomach and bowels, produce nervous symptoms, such as cramps, convulsions, and delirium. *American or green hellebore* has occasioned similar phenomena in a slight degree, but its poisonous action upon the nervous system is rather shown by vertigo, somnolency or coma, dimness of sight, dilatation of the pupil, and impaired muscular action.[1]

§ 292. The roots of the *Helleborus viridis*, *Helleborus fœtidus*, and *Helleborus niger* owe their action to two active principles, helleborin and helleboreïn, which belong to the class of organic substances termed glucosides. Helleborin is easily soluble in alcohol and chloroform, but with difficulty in water and ether; concentrated sulphuric acid added to it gradually produces a violet color. Helleboreïn is soluble in water, from which it can be precipitated by tannic acid and by the phosphomolybdate of sodium; it is with difficulty soluble in absolute alcohol and almost insoluble in ether; concentrated sulphuric acid produces with it almost immediately a beautiful, bright red color. Helleboreïn is the more important principle from a chemico-legal point of view, since it can be isolated from organic mixtures in precisely the same way as colchicin, and can be detected in the chloroform residue by the above tests, and by the physiological effect which it produces when given to animals, since it diminishes very much the frequency of the pulse beats.

[1] *Vide* action of alkaloids, veratrine.

III. *Castor Seeds or Beans.*

§ 293. It is stated (Wood and Bache) that two or three of these seeds are sufficient to purge, and that seven or eight act with great violence. The property depends upon an acrid principle which exists, as is now satisfactorily ascertained, in the embryo. Dr. Hartshorne states that he has known them to be eaten freely with impunity at times, and in other cases to produce the most violent and even fatal emeto-catharsis. Dr. Taylor gives an instance of poisoning by them. A young lady ate about twenty. About five hours after they were eaten, she felt faint and sick; vomiting and purging came on, and continued through the night. On the following morning she appeared like one affected with malignant cholera. The skin was cold and dark colored, the features contracted, and the breath cold; the pulse was small and wiry; there was restlessness, thirst, pain in the abdomen, and she lay in a sort of drowsy, half-conscious state. Whatever liquid was taken was immediately rejected, and the matters passed by stool consisted chiefly of a serous fluid, with blood. She died in five days. On inspection, a very large portion of the mucous membrane of the stomach was found abraded, and softened in the course of the greater curvature. There was general vascularity of the organ, and the abraded portion presented the appearance of a granulating surface of a pale rose-color; it was covered with slimy mucus. The small intestines were inflamed and the inner coat abraded.[1]

IV. *Euphorbiaceæ.*

§ 294. There are many species of this plant (*corollata*, *ipecacuanhæ*, *lathyris*, *resinifera*) vulgarly called the large flowering spurge, which are violent emeto-cathartics, as well as irritating vesicants to the skin, and in large doses become acrid poisons. Their medicinal virtues reside chiefly in the resin of the plant. Medicinally they are used for the emeto-cathartic characteristics. Occasionally fatal accidents, caused by the irritating action of the resin upon the primæ viæ, have been recorded, but no case is known where poisoning was criminally effected by these plants. Their medicinal dose in powder is stated to be twenty or twenty-five grains.

[1] Med. Jur., p. 165.

V. *Fungi.* (Mushrooms.)

§ 295. There is a vast number of cryptogamous plants thus denominated, some of which are generally wholesome as food, while others are exceedingly poisonous. The rules laid down for their selection by M. Richard, in the Dict. des Drogues to persons who eat mushrooms, are that those should be rejected that have a narcotic or fetid odor, or an acrid, bitter, or very acid taste; that occasion a sense of constriction in the throat when swallowed; that are very soft, liquefying, changing color, and assuming a bluish tint when bruised; that exude a milky, acrid, and styptic juice; or that grow in very moist places and upon putrefying substances; in fine, all such as have a coriaceous, ligneous, or corky consistence. The last, however, are injurious in consequence rather of their indigestible than of their poisonous nature. Yet it is now known that fungi rarely have an acid taste, and the beefsteak fungus, which does have a decidedly acid taste, is considered very nutritious. Even mushrooms which are usually edible may prove poisonous if collected too late, or in places which are too moist. It should also be observed that unless they are collected when young, or, if mature, before there is any sign of decay, they are injurious as food; so also if not cooked soon after gathering—with comparatively few exceptions—they may cause indigestion. It is said, moreover, that the poisonous species become innocent when they grow under favorable circumstances; and that the most noxious may be rendered edible by boiling them in water acidulated with vinegar;[1] but the water should be poured off, and the mushrooms thoroughly washed in clean water before being cooked or served as food. In many portions of Europe, but especially in Poland and Russia, they form the most important part of the food of the common people; and in the latter country whole tribes are mainly supported by them, scarcely any species, except the dung and the fly *agarics*, being rejected. Even those kinds which are elsewhere refused, by common consent, as poisonous, on account of their extreme acridity, are taken with impunity, being extensively dried, or pickled in salt and vinegar, for winter's use.[2]

[1] On the Medicinal and Toxicological Properties of the Cryptogamic Plants of the United States, by F. Peyre Porcher, M.D., of Charleston, S. C., in the Trans. of the Am. Assoc., vol. vii.

[2] Berkeley, ibid.

§ 296. It appears very certain that the poisonous properties of mushrooms may be removed by boiling, and especially with acidulated water or with vinegar, provided the above cautions be taken.

Dr. Pouchet, of Rouen, gave a quart of the water, in which five poisonous mushrooms (*Amanita muscaria* and *Avenenata*) had been boiled, to a dog, causing death in eight hours; but the boiled fungi themselves had no effect upon other dogs. Another, which was fed for two months on little else than boiled amanitas, not only sustained no harm, but actually got fat on his fare.[1] M. Gérard exhibited, before a committee of the Paris Council of Health, the complete innocuousness of these two most poisonous varieties after having been macerated in water. He directs that some two or three spoonfuls of vinegar or some coarse salt should be added to the water, and the fungi macerated for two hours, after which they should be washed, and then put into cold water and boiled for half an hour. They may then be taken out, washed, dried, and used as food.[2] In a later number of the same journal, Dr. Gondot relates the cases of seven persons poisoned by eating mushrooms, three of whom died. The mushrooms had been fried in butter. One person ate, the next day, at least half in quantity of those that had served the family the day previous. These, however, had lain in water for an hour, and were then drained and pressed. In this condition they were fried in butter and eaten. Diarrhœa followed for several hours, but without any other dangerous effect. For a full account of the facts so far ascertained, regarding the distinction between the poisonous and edible mushrooms, we beg leave to refer the reader to Dr. Porcher's admirable essay above quoted, and to Christison and Orfila's treatises on Poisons.

§ 297. The *symptoms* of poisoning by mushrooms or other fungi, are both of an irritant and narcotic character. In Dr. Gondot's cases the symptoms did not come on until several hours after the meal; in the fatal cases, not until twenty hours, the patients dying in sixteen hours afterwards. They were all affected with vomiting, purging, and cramps. In three cases related by Dr. Peddie, the symptoms began in half an hour with giddiness and stupor; there were no abdominal symptoms, and the patients recovered. The principal symptoms in sixty-eight cases referred to by Ballardini

[1] *Vide* Christison on Poisons.

[2] Union Méd., 1851, No. 148.

were nausea, uneasiness in the abdomen, vertigo, a state resembling intoxication, vomiting and diarrhœa, and loss of power of locomotion with convulsions. The following case exhibits a singular form of the narcotic effects. A boy of fourteen, who had eaten the *Agaricus pantherinus*,[1] near Bologna, was in the course of two hours seized with delirium, a maniacal disposition to rove, and some convulsive movements. "Ere long, these symptoms were succeeded by a state resembling coma in every way, except that he looked as if he understood what was going on, and, in point of fact, did so."[2]

Intestinal irritation followed the eating of raw fungi in one instance in two hours.[3] Both the lethargy and the symptoms of irritation may continue for a considerable length of time, and both may occur simultaneously.

§ 298. The *post-mortem appearances* in the few cases recorded have been the following: An unusual fluidity of the blood, turgescence of the vessels of the brain, inflammation, and even gangrene of the stomach. In one of Dr. Gondot's cases (the only one examined) there was a decomposition of the tissues, the abdominal viscera were softened, and the odor from them was extremely fetid. Dr. Maschka, of Prague, has reported seven fatal cases of poisoning by mushrooms, in which, after death, there was an entire absence of cadaveric rigidity, dilated pupils, blood of a dark-brown color mixed with dirty yellow and soft fibrinous clots, numerous ecchymoses and sanguineous effusions in the serous membranes and the parenchymatous organs, and remarkable distension of the bladder with urine.[4]

The evidence in cases demanding a legal investigation will most probably be derived from circumstances and the symptoms. In cases of doubt a microscopical examination of the contents of the stomach would reveal the presence of fungi. The structure of fungi is composed of threads, densely entangled, and nearly colorless, which, frequently branching, end in ovoid swellings where they reach to the outer surface of the spore-bearing portion of the fungus. The presence of spores alone in the intestinal canal or its contents is hardly characteristic of the presence of mushrooms, because

[1] The Agaricus campestris is not poisonous in this country.

[2] Christison on Poisons.

[3] Chicago Medical Examiner, 1874.

[4] Prager Vierteljahrs., 1855, ii. 137

spores of many species of mushrooms cannot readily be distinguished from those of moulds and other swollen fungi, which are frequently introduced into the alimentary canal with ordinary food. In only a very few cases is the shape of the spores of such a character that the species to which they belong can be recognized. The presence of masses of fungus-threads, however, especially when taken in connection with the gastro-intestinal irritation and cerebral disturbance, is an important circumstantial evidence.

§ 299. As muscarin is found in several of the fungi which are likely to be eaten by mistake for the common edible mushrooms, it would be well to examine the contents of the alimentary canal for that substance. The only case which can present difficulty is where an irritant poison has been designedly introduced into the preparation of mushrooms. An instance of this kind occurred: a woman died from the effects of arsenic mixed by her servant with mushrooms. The girl afterwards confessed the fact.[1] Of course, the only means of distinction in such cases is a chemical examination for the suspected poison.

§ 300. *Chemical examination.*—Muscarin is a colorless, syrupy body, without odor or taste, and having an alkaline reaction. It is very soluble in water and alcohol, slightly soluble in chloroform, and insoluble in ether. It can be obtained in crystalline form by drying in a desiccator over sulphuric acid, but these crystals readily absorb water on exposure to the air. There are no characteristic chemical tests for it, and we must rely upon the physiological test when the microscope fails to detect the fungus in the stomach or intestines. From two to four milligrammes (0.03 to 0.05 grain) is a sufficient amount to kill a cat in from two to twelve hours, the symptoms being excessive flow of saliva, vomiting and diarrhœa, contraction of the pupil, at first increase in frequency of breathing and difficult breathing, and later, a diminution in the frequency of respiration, convulsions, and finally cessation of respiration and death. The urine in cases of mushroom poisoning should always be examined for muscarin. It should be concentrated by evaporation to a small volume, the resulting fluid extracted with alcohol, the alcohol evaporated, and this residue extracted with water; this aqueous solution can be used for performing the physiological test upon frogs, and its peculiar action upon the heart seen.

[1] Christison on Poisons.

CHAPTER VII.

IRRITANT POISONS—Concluded.

I. *Cantharides.*[1]

§ 301. *Properties.*—The cantharis, or Spanish fly, much used in medicine as a counter-irritant, and also occasionally given internally, is capable of producing fatal results, and should be classed as a definite poison to animal life.

A description of this insect, called *Cantharis vesicatoria*, or *Lytta vesicatoria*, is out of place in this treatise, but it should be known that its active principle is most abundant in large, full-grown insects, while in very young specimens no cantharidin is found, and that the dried beetles are liable to be eaten by mites and various other minute insects, so that a given weight of unselected specimens may vary in the amount of irritation on the body or in the amount of cantharidin. Its vesicating and its irritant property results from the presence of the same principle, viz., *cantharidin*, which has been believed to exist chiefly, if not entirely, in the wing-cases of the insect. There is a variety of insects which contain cantharidin, and these vary in appearance according to the locality in which they may be indigenous. One variety is found in Southern Europe

[1] Poisons of this class Casper denominates *septic*, because the greater number of them, resulting from animal decomposition, corrupt the blood when taken into the system.

and Western Asia, another, *Mylabris cichorii*, in Eastern and Southern Asia, another, *cantharis*, or *Lytta vittata*, in Colorado and Kansas, called the potato-bug or fly. Powdered cantharides is liable to be adulterated with Euphorbium. For some experiments of Dr. Leidy, the vesicating principle of *Lytta vittata* appears to belong to the blood, the peculiar fatty substance of certain accessory glands of the generative apparatus, and to the eggs.[1] Cantharidin, however, is too active for internal use, and is only employed for the purpose of vesication. Cantharides are usually taken in powder or in tincture. In a case reported by Dr. Homans, the powder was taken by mistake for *hiera picra*.[2]

§ 302. Cantharidin has been obtained from different samples in about one-fourth to rather more than one-and-a-half per cent., and it has been ascertained that the hard parts of the insect contain about six times more of the active principle than the soft parts. Cantharidin, which may be obtained from any of the above-named varieties of the fly crystallizes in colorless four-sided prisms.

Fahnestock[3] recovered from the *Mylabris cichorii* of an old dried up character a yield of 1.25 per cent. of cantharidin, and from the *cantharis vittata* (potato-bug) 1.3 per cent. of cantharidin. In solution or mixed with lard it may produce very powerful irritation of the skin and blistering or vesication, and taken internally it is a violent irritant poison. It being commonly known to cause irritation in the genital organs, which is a secondary effect of its use, it has frequently, in ignorance of its dangerous properties, been employed for the purpose of exciting the sexual propensities, and it is occasionally taken also with the hope of procuring abortion. It may produce serious internal symptoms even by external use. Taken internally, generally in the form of tincture, it has been used medicinally for dropsical effusions and for paralysis of the bladder, cystitis or inflammation of the bladder, and in gonorrhœa. It is also sometimes wrongly used in leprosy, hydrophobia, and other nervous diseases.

§ 303. The *symptoms* occasioned by an overdose of either of these preparations commence with nausea, vertigo, and a burning

[1] Am. Journ. of Med. Sci., Jan. 1860, p. 60.

[2] Bost. Med. and Surg. Journ., March, 1855, p. 153.

[3] Am. Journ. of Phar., June, 1879.

sensation in the mouth and throat. This sensation presently extends to the œsophagus and stomach, is succeeded by violent pain and extreme tenderness in the abdomen, thirst, difficulty of swallowing, and vomiting of blood, mucus, and shreds of membrane, and frequently green particles of the insect, which are easily recognized with the aid of a microscope. There is also violent pain in the loins, strangury, and priapism; occasionally there is satyriasis with seminal emissions, and sometimes a diarrhœa accompanied by discharges of blood. There is often a frequent desire to urinate, and the urine on standing is liable to be clouded with a deposit of mucus; it has a lemon-yellow color, and usually contains blood. There may be also false membranes and albumen passed with the urine; the false membranes are due to an inflammation of the mucous coat of the bladder; the albumen comes from congestion of the kidneys; when albumen is found in the filtered urine, it is probable that renal disturbance, active congestion or parenchymatous nephritis, exists. In the female, swelling and heat of the organs of generation have been observed, and during pregnancy abortion is apt to be produced. It has repeatedly happened that the genital organs were attacked with gangrene, even in cases in which no sexual excitement was manifested. The secretion of urine is, in some cases, suspended; for in a case in which the catheter was introduced, no urine could be obtained. Occasionally profuse salivation occurs, and, in fatal cases, violent cerebral symptoms are observed. A young girl at Windsor was killed by the external application of a blistering ointment, which was rubbed over her whole body by mistake for sulphur ointment, which had been prescribed for the cure of the itch. Although the ointment was washed off, the cuticle came with it, and the girl died in five days with the symptoms above described.[1] Guibourt has reported the case of a young man, suffering from acute pleurisy, who, having had a large blister applied to his side, became affected with symptoms of irritation in the urinary passages, and died after falling into a state of complete collapse.[2]

Cantharides may be absorbed into the system when applied externally, and especially when applied to mucous surfaces. It is eliminated by the kidneys and skin, and produces, as may be gathered from the above symptoms, an irritation in the urinary passages; it

[1] Taylor on Poisons.

[2] Abeille Méd., xv. 153.

may also, when eliminated by the skin, cause signs of irritation of that surface.

§ 304. The accidents caused by cantharides poisoning depend upon the quantity of the poison absorbed and the rapidity of its elimination from the body; hence, if there be a disease of the kidneys, a smaller dose may cause more serious consequences than where these organs are in a healthy functional activity. There is a form of subacute or slow poisoning, the symptoms of which may succeed those above enumerated. Instead of the skin remaining hot and feverish with injection and suffusion of the eyes, the surface of the body may be cold and covered with a clammy sweat, the eyeballs sunken, the eyes without expression and almost motionless; a few convulsive jerks in the muscles may be observed followed by rigidity, and the victim becomes comatose and dies soon after. These symptoms may last only a few hours after the poison is swallowed.

In another form of slower poisoning, the symptoms are restricted to the genito-urinary and intestinal passages, there being an almost constant erection of the penis, which is painful, and without erotic sensations or seminal emissions. There is a feeling of anxiety and great agitation with craving for water, vomiting incessantly, and with an abundant flow of saliva. These symptoms are followed by muscular twitching and convulsive movements, which are succeeded by great prostration and exhaustion, with slow and feeble pulse accompanied by fainting; the victim may die within two or three days.

A boy,[1] suffering from a suppuration in the neighborhood of the ear and with evidence of meningitis, was ordered a cantharides blister four inches square, which he put into his mouth and chewed, though there was no means of ascertaining how much he swallowed. As soon as it was known what he had done, a large dose of ipecacuanha and castor oil was given. No evil effects followed for twelve hours, at which time there appeared tenderness over the region of the stomach and bowels, great restlessness, increased stupor gradually deepening to complete coma, convulsions and death in twelve hours after the first appearance of symptoms. The boy had been improving, and before chewing the blister had shown every evidence of convalescence from the disease.

[1] Brit. Med. Journ., June 25, 1881.

§ 305. The *quantity required to destroy life* is not accurately ascertained. Drs. Wood and Bache state the medicinal dose as from one to two grains of the powder, and from twenty drops to a fluidrachm of the tincture. It is evident, however, from the frequently deteriorated condition of the powder, that the active principle may, in any given quantity, be found in less than the average amount, and that the strength of the tincture may be often thus impaired. This fact will serve to explain the large quantities which have been sometimes taken without dangerous symptoms. The smallest quantity of the powder which has been known to destroy life, was in the case of a young female, mentioned by Orfila, who took *twenty-four* grains in two doses. She took it to procure abortion, and, as this followed, it is uncertain whether it may not have hastened the fatal result. Much larger doses of the powder have been taken, followed by the most dangerous symptoms, but early vomiting, no doubt, removed a great deal of the poison.

In a case observed by Dr. Ives, of New Haven, a boy of seventeen died from the effects of an *ounce* of the tincture. Death occurred seventeen days after he had taken it. A curious case occurred in France, in 1846, where the ointment of cantharides, consisting of a fourth part of the powder, and three parts of resin, wax, and lard, was administered to a man in his soup, with the intention of poisoning him. The criminal was condemned to death, although his intended victim recovered from the dangerous symptoms from which he suffered. The exact dose in this case is not mentioned.

Cantharidin in the dose of from six-tenths to one-and-a-half grains is followed by serious symptoms, and a dose over eight grains may cause death.

§ 306. The *post-mortem appearances* are those of inflammation. If a quantity sufficient alone to destroy life has been taken, the œsophagus, stomach, and small intestine will most probably be found highly inflamed, and if the person have lived for several days, the kidneys, ureter, and the neck of the bladder also. Such has been the case in the few fatal cases of poisoning by this substance which have been examined. Sometimes the lining membrane of the mouth and throat is destroyed, and in Dr. Ives's case that of the stomach was pulpy and easily detached. A characteristic pathological effect is the presence of false membranes upon the mucous surfaces, especially at the back of the throat and on the internal surface of the

bladder. The latter is found empty, softened and with thickened membranes (cystitis). The genital organs are congested and sometimes gangrenous.

In Guibourt's case the kidneys were softened and filled with hemorrhagic spots; the same appearance was presented by the ureters and bladder, the mucous membrane of which was partially disorganized. A child nearly three years old is said to have rejected by vomiting the entire mucous membrane of the œsophagus, after taking about a drachm of tincture of cantharides.[1]

§ 307. *Chemical examination.*—The presence of the greenish, gold, or copper-colored scales, derived from the wing-cases of the insect, is the best evidence of the nature of the poison. They adhere very closely to the mucous membrane of the intestines, and may be easily recognized by a common lens. Although there are many other insects which have wings of the same color and are not poisonous, it is hardly possible that these should find their way into the stomach, and much less that they should have been given with any evil intent. M. Poumet recommends also that the suspected liquids which have been vomited should be mixed with alcohol and allowed to evaporate on sheets of glass, by which means the brilliant colored particles of the fly will be visible after evaporation. Or, the stomach and intestine may be inflated and dried, after which, upon cutting them open and examining them upon a flat surface, the particles above mentioned, if present, will be seen sticking closely to the mucous membrane. They are not affected by putrefaction, and, according to Orfila, may be recognized as long as nine months after interment. If, however, the tincture has been taken, this method will not, of course, be available. An effort may be made to detect the cantharidin in the suspected liquid, by digesting in ether what remains after evaporation, and then testing the vesicating properties of the product, but it is evident that the evidence derivable from such a method is very imperfect, if the experiment should not succeed, it being very possible that cantharides may have been used and yet not be detected by these means. One principle of the cantharides insects has been shown to be identical with the green coloring matter of plants, chlorophyll, and, if present, can be easily detected by the spectroscope, though, if the amount be small,

[1] Am. Journ. of Am. Sci., Oct. 1857, p. 560.

the microspectroscope would be the proper instrument for its detection. The spectroscopic test for chlorophyll is thoroughly positive and reliable, but the cantharidin itself is colorless, and, therefore, cannot be recognized by the spectroscope. So many other substances, however, contain chlorophyll that the detection of this coloring matter can only be considered as a confirmatory test. The detection of the green particles of the insect, and the isolation of the active principle, cantharidin, and its recognition by its chemical and physiological tests, constitute the chemical proofs of poisoning by cantharides or any of its pharmaceutical preparations.

Cantharidin crystallizes in the form of quadrilateral right-angled prisms, or frequently in plates. It is slightly soluble in alcohol, chloroform, ether, benzol, and bisulphide of carbon. It is readily soluble in water containing a little alkali, since it is an acid and combines readily with the alkali to form salts which are soluble in water, but soluble with difficulty in alcohol. Solutions of these salts of the alkalies, when not too dilute, give a white precipitate with chlorides of barium and calcium, a green precipitate with the sulphates of copper and nickel, a red one with the sulphate of cobalt, and a white crystalline one with the acetate of lead, corrosive sublimate, and nitrate of silver. (Dragendorff.)

§ 308. To isolate cantharidin from organic mixtures the best method is that recommended by Dragendorff,[1] viz: The substance, finely divided, if necessary, is boiled with a solution of potassic hydrate (one part of the hydrate to twelve or fifteen of water) until the mass becomes of homogeneous consistency. This mass is then diluted with water if necessary, so that it shall not be too thick to shake with chloroform; it is then shaken vigorously with chloroform which will remove many impurities; the chloroform is then removed after it has separated, the fluid rendered strongly acid with sulphuric acid, mixed immediately with four or five times its volume of alcohol of 90 to 95 per cent., the mixture boiled for a while, filtered while hot, and the alcohol expelled from the filtrate by evaporation; the aqueous acid fluid remaining should then be shaken with chloroform which will remove all of the cantharidin; wash the chloroform thoroughly by shaking it with water, and allow it to evaporate spontaneously in a watch glass. The residue can be

[1] Ermittelung von Giften, 1876, p. 291.

mixed with a little oil, and the physiological test applied, after examining with a microscope for cantharidin crystals, which are usually found when large amounts were present.

The physiological test consists in applying the residue to the skin or mucous membranes, when the usual blistering effect of cantharides will be obtained. Even 0.00014 gramme (=0.002 grain) of cantharidin is a sufficient amount to raise a blister upon the skin, and a much smaller amount will produce redness and inflammation when applied to a mucous membrane. (Dragendorff.) When quite large amounts are present, the crystals and crystalline salts can be obtained and taken to court as the *corpora delicti*.

Cantharidin, like some of the other organic poisons, resists decomposition of the animal tissues for a very long time. Dragendorff has detected it in the body eighty-four days after death.

II. *Sausage Poisoning and Trichinosis.*

§ 309. *Nature of the poison.*—These and analogous articles of food have so frequently given rise to poisoning that we cannot pass them over entirely unnoticed. According to the statistics of Prof. Schlossberger, there have occurred in the kingdom of Wurtemberg alone, since 1800, no less than 400 cases of poisoning by sausages. Blood and liver sausage (*blut-* and *leber-würste*) constitutes one of the most ordinary articles of the diet of the Germans, and other smoked and fatty preparations, obtained chiefly from pork, are much used. The nature of the sausage poison has not been definitely ascertained. Liebig considered it a kind of ferment, Buchner believed it to be due to a peculiar acid which he termed botulinic acid, while Schlossberger considered it to be an organic base. He supported this theory by referring to the now numerous sources of the ammoniacal bases, the transformation of protein combinations by putrefaction, and the very poisonous nature of many of these alkaloids, among which conicine and nicotine are already well known.[1] The study of these alkaloidal products of putrefaction is still in its infancy, so that we may confidently hope for further light in a short time upon the active principles in poisonous cheese and sausages. This writer, in a more recent essay, states that the uncertainty regarding the source of the poison continues. He also adds an im-

[1] Canstatt's Jahresbericht für 1850. V. Band, p. 136.

portant fact, that poisonous sausages are eaten by dogs and cats with impunity.[1]

§ 310. In December, 1841, over forty cases of cholera morbus occurred in New York, which, according to Dr. Lee, were traced to some smoked beef, sold from a particular grocery, and of which the individuals attacked had freely eaten. The symptoms did not generally make their appearance until several hours after the beef had been eaten. They commenced with pain and uneasiness in the præcordial region, which extended to the back and loins, and were only temporarily relieved by the dejections which followed. Vomiting soon came on, attended with great thirst and a burning sensation at the pit of the stomach, and the irritability of this organ became so great, that no substance, either as food or medicine, could be retained for an instant. Extreme prostration followed; the functions of the nervous, muscular, and the digestive system were much impaired, and convalescence was very slow and protracted. In one case, that of a girl six years of age, the disease proved fatal on the fifteenth day; on dissection the blood was found fluid, the mucous coat of the ileum deeply injected, and inflamed; the other organs were healthy.[2] The cases of poisoning occur chiefly in the winter and spring months. The unwholesome sausages are described by the last-mentioned author as showing, especially in the interior, little masses of soft consistence like curd; they have a repulsive odor, and a sour, bitter, and rancid taste.

§ 311. The opinion among physicians of the present day is, that most of the cases of poisoning by sausages is due to a disease called *trichinosis*, which is supposed to be caused by the presence of a flesh-worm (*trichina spiralis*) which infests the muscular tissue of pigs. Meat containing this insect has a number of small white dots interspersed among the muscles; and the appearance of this insect under the microscope is so well known among physicians and naturalists as to require but a passing notice in this work. Cases of persons afflicted with *trichinosis* in the western part of our country have been reported. For a thorough description and a very full account of the natural history and appearance of the trichinæ, reference is made to a lecture by Prof. John Dalton, M.D., reported

[1] Virchow's Archiv, xi. 569.

[2] Copland's Dict., Am. ed., art. Cholera.

in the *N. Y. Medical Record*, April 1, 1869, as well as in the *Boston Med. and Surg. Journal*, May 6, 1869. Dr. Dalton states that one-fiftieth of all the pigs brought into the market at Chicago in 1866 were infested with trichina. To kill the worms, the ham must not only be salted and smoked, but must be subjected to a temperature of 212° F., and continued for a considerable length of time, so that each portion of the meat (central as well as outside) should attain nearly that temperature.

§ 312. In some cases suspicion is wrongly thrown upon the food. Thus in the narrative (communicated by Prof. Röse to Casper's Journal, 1852) of the poisoning of a family by the smoked breasts of geese (*spickgans*), it was found upon chemical examination that a considerable quantity of *sulphate of zinc* was contained in the food. It had been used instead of saltpetre in its preparation. The symptoms were of a choleraic nature, and nothing like the narcotism produced by sausage poison was observed. Consequently a chemical search should be made in all such cases for mineral or other poisons. When there is no evidence that these are present, the sausages or meat should be examined by the sense of smell and taste, as well as by the microscope.

§ 313. The *symptoms* are well seen in the following narration. The family of Ehrmann at Limmetshausen, with a number of guests, partook of a supper of pork sausages, in consequence of which all were more or less affected with symptoms of poisoning, eight with severe symptoms, and three died. The sausages were made of the liver of a healthy pig prepared eight days previously, slightly boiled, then smoked and hung up. There must have been something peculiar in the taste of the sausages, as one of the guests remarked that they were not wholesome, and did not partake of them, in consequence of which he escaped. The symptoms were similar in all, differing merely in degree. *Shortly after partaking* of the sausages, pain in the bowels, vomiting, giddiness, dryness of the mouth and throat, and difficulty of swallowing came on. The pupils soon became dilated and fixed, the headache and vertigo increased, and the power of vision was lost. Great prostration of strength followed, the power of speech was destroyed, the abdomen was painful to the touch, the pulse small, weak, and frequent, and at last intermittent. The respiration became difficult, and deglutition impossible, lividity of the countenance came on, spasms of the muscles

of the extremities ensued, and rapid death. Death occurred within thirty-six hours after eating the sausages.

These cases show that the symptoms are not always so slow in appearing as is generally stated. Many other cases might be referred to in which the effects were precisely similar to those described. Consequently the sickness and fatal result should not always be attributed to the disease called trichinosis. Should there be doubt from the symptoms, it will be easily cleared up by an examination of the tissues of the dead body by the aid of a microscope. The disease of trichinosis being due to the presence of the trichinæ in the mucous membrane and deeper tissues of the *primæ viæ*, they would be seen by observing the inflamed and swollen parts with a microscope, or, if the patient has survived the first few hours of the attack of gastro-intestinal irritation, these entozoa will be found in the muscles.

§ 314. Besides the effects of this parasite, the meat of the sausages may have become putrid and cause intestinal irritation and violent constitutional typhoidal symptoms, and in these cases obviously a microscopical examination will fail to find the presence of entozoa. Several instances of death being caused by tainted or putrid meat are recorded in medical literature. We give two instances:—

A family at Greenock,[1] one member of which, the grandmother, died after a night of suffering from abdominal pains, was poisoned by eating old putrid sausages. The sausages were collected from an ash heap. *Post-mortem* examination showed that there was no poison, but that death was caused by putrid meat. The other members of the family, though quite ill, recovered.

Dr. Tripe read a paper before the Society of Medical Officers of Health, at its meeting in November,[2] in London, giving an account of the poisoning of sixty-four persons from eating a batch of sausages; sixty-six persons had eaten of this same batch. One of the victims died. The symptoms were those of a narcotic-irritant, somewhat resembling those of cholera, but with a greater amount of cerebral disturbance. The early symptoms were sickness, purging, and giddiness; great feeling of weakness, with a sense of acridity in the throat, the stools smelling very badly, and looking like dirty soap-water, or the washings of putrid meat. Many had severe cramps

[1] Med. Press and Circ., Dec. 2, 1879. [2] Med. Times and Gaz., Nov. 29, 1879.

in the legs and pains in the stomach, but others were not affected in this manner. In the majority of cases the vomiting and diarrhœa continued from thirty-six to forty-eight hours. In the fatal case a *post-mortem* examination showed patches of redness in the intestines. The sausages were examined, but, beyond the tainted and putrid odor, showed nothing abnormal by the microscope or chemical analysis.

§ 315. *Symptoms of trichinosis.*—The disease called trichinosis is now so well recognized by the medical profession that its extensive discussion is not needed here.[1] The absence of all intestinal symptoms, as well as severe attacks of vomiting and purging, is only exceptionally observed after the use of trichinous flesh. The greater number complain, usually not till a few days after the poisoning, when the young brood has been hatched, of dryness of the throat accompanied with great thirst, præcordial distress, pain in the abdomen and stomach, and of eructation and nausea combined with a feeling of great muscular heaviness and depression. There is almost always diarrhœa, the discharges being brownish, afterwards yellow, thin, and accompanied by more or less severe colicky pain. These gastric symptoms are followed, sometimes also accompanied, by vague muscular pains, stiffness, swelling, and œdema of the eyelid, and sometimes a peculiar staring expression to the eyeball (Kittell) due to the infection of the muscles which control the movements of the eye. The muscular pains become more severe on motion, and soon the different muscles swell considerably and become tense and hard. Cohnheim describes the patient's position: "He lies on the back with the shoulder and elbow-joints sharply bent, and the hands slightly flexed; on the other hand, the knee and hip-joints are but slightly bent, or nearly straight; so that he cannot lift the arm, extend the forearm, sit up, or bend the knees."[2] There is also an insidious form of the disease, the gastric disturbance not being very great; but often these apparently mild cases assume the violent and dangerous form. Death in this disease is not uncommon during the first two weeks; children have a certain immunity to trichinosis, perhaps because they do not digest a part of the meat.[3] The attesta-

[1] Reference is made to Ziemssen's Cyclopædia of the Practice of Medicine, New York, vol. iii. pp. 613, 630; vol. xviii. p. 201.

[2] Niemeyer, vol. ii.

[3] Ibid., op. cit.

tion of poisoning by the trichina can always be proved by the microscopic appearance of the muscular tissues.[1]

§ 316. It may be gathered from what has been said of sickness caused by sausages, that damaged meat itself will cause serious illness and disease, which may in some cases result in death, even when there are no entozoa nor injurious animalculæ present.

The symptoms produced by the use of unsound meat are similar to those arising from irritant poisoning, but in addition there are typhoid symptoms which indicate the contamination of the whole body by the products of decomposition. In some cases related by Dr. Christison, the patients were soporose or delirious, and one died comatose in six hours after eating a portion of a putrefied calf. The rest, being freely purged and made to vomit, eventually got well; but for some time they required the most powerful stimulants to counteract the exhaustion and collapse which followed the attack.[2] Game which has been long kept may be the source of the symptoms of irritant poisoning. In this country the flesh of the pheasant (*Tetrao umbellus*), when snow is upon the ground, is apt to prove unwholesome to some, in consequence, as is supposed, of the bird feeding upon the leaves and berries of the laurel. It is doubtful, however, whether this explanation is correct. That meat in winter may be in the first stages of decomposition, which is arrested by chilling in frosty or ice-cold air, and does not appear to be tainted, is a well-known fact, especially in the case of fowl which are killed in cold weather and kept for sale for the Thanksgiving or Christmas festivals. Eating of meat in this arrested state of putrefaction may not cause a distaste, since, either by being chilled or cooked, it may have no tainted smell or taste; yet it is not unfrequently followed by indigestion in stomach or intestines, especially in those of feeble health, and by consequent diarrhœa and signs of irritant poisoning.

The whole subject, made familiar by popular stories of persons showing serious signs of irritant poisoning after rising from a

[1] For further details see an exhaustive paper on this subject of trichinosis in Mass. State Board of Health Report, Jan. 1880, by Dr. Billings.

[2] Articles on diseased meat as affecting the health may be found in Brit. and For. Med.-Chir. Rev., Jan. 1858, p. 87, and in North Amer. Med.-Chir. Rev., May, 1858, p. 483. Duchesne has written on the unwholesomeness of poultry fed on diseased meat, Ann. d'Hygiène, 2ème sér. xi. 63, and Roeser on that of over-driven cattle, Prager Vierteljahrs., xl., anal., p. 86.

sumptuous meal of game, is still an uncertainty. Cases of illness caused on shipboard or in cheap boarding-houses, where meat is furnished for food which was in the arrested putrefactive process and salted in brine, or preserved as canned meat, are quite frequent; there is no doubt that more than temporary illness may be thus caused, because in endeavoring to eliminate the poison resulting from putrescing meat, the kidneys frequently may become diseased.

Drs. E. Ballard and E. Klein present a group of cases[1] of poisoning of persons who had eaten, at a sale on the Duke of Portland's estate, Wellbeck, Nottinghamshire, of cooked ham prepared in England from pork imported in a salted state from America. Four persons attacked with the disease thus induced died after a few days' illness. The period of incubation after eating the meat was twenty-four to thirty-six hours. The illness commenced sometimes with chilliness and rigors, sometimes with faintness, giddiness, or pain in the chest, back, or abdomen; but sooner or later there was pain in the abdomen, with fetid diarrhœa and usually vomiting. Muscular weakness was an early symptom, and usually there was headache and thirst. Various nervous phenomena were observed, such as cramps, convulsive flexions of the hand or fingers, muscular twitchings, pricking sensations, or numbness of the hands, drowsiness, hallucinations, etc. The *post-mortem* examinations were very imperfect, but microscopic examination of the kidneys showed that the arterioles and capillaries of the Malpighian bodies were choked up with masses of *bacilli*. This bacillus was also in the intermuscular connective tissue. Experiments of feeding with these bacilli, or by their inoculation into animals, was followed by the death of the animals, and the result of their *post-mortem* examination was pneumonia or pulmonary hemorrhage, hemorrhage into the liver, enlargement of the spleen, pleuritis, peritonitis, etc.

A second group of fifteen cases of illness, with one fatal result, was traced to the eating of hot baked pork, purchased also at Nottingham. Bacilli of similar character to those observed in the first group, accompanied with similar *post-mortem* appearances, were observed, but the meat which caused the poisoning could not be obtained for examination.

[1] Read before the Section on State Medicine at the International Med. Congress (7th Session) in 1881, London.

It may be seen from these cases that there is a form of poisoning caused by putrescent meat, which is not infested with the results of decomposition, such as fungoid or germ growth and the accompanying animalculæ which feed upon these growths. Unfortunately many cases of chronic illness from disturbance of the eliminating organs, the intestines, kidneys, and lungs, may occur at a distance from the residence of an experienced pathologist and microscopist, and the individuals may there succumb to the disease, or fall into a chronic form of illness difficult to trace to its actual cause. These diseased persons may show the results of serious disease of the kidneys, lungs, or intestines, which are familiar to the pathologist though not known to the general practitioner. It would be out of place in this treatise to present the scientific studies of Koch, Pasteur, Bastian, and other experimenters and teachers of minute animalcular life in the living and dead tissues of our bodies, for their results have little or no bearing upon the subject of toxicology.

III. *Poisonous Cheese.*

§ 317. The nature of the poisonous quality occasionally acquired by cheese, is not so precisely known as that of sausages; it was supposed by Hünefeld and Sertürner to depend upon two animal acids, analogous to, if not identical with, the caseïc and sebacic acids. According to the researches of Proust, the sharp, peculiar taste of old cheese is owing to the gradual conversion of the curd of caseïne into the caseate of ammonium, which in sound cheese is always united with an excess of alkali. But if the fermentation has been too much hastened, or allowed to go too far, a considerable excess of caseïc acid is formed, as well as some sebacic acid. According to Hünefeld, the deleterious cheeses are yellowish-red, soft, and tough, with harder and darker lumps interspersed; they have a disagreeable taste, redden litmus, and become flesh-red instead of lemon-yellow under the action of nitric acid.[1] Instances of poisoning by cheese have been hitherto observed chiefly in Germany, some few in England, and within a few years in this country also.

Dr. Parrish has given an account of several cases which fell under

[1] Christison on Poisons, p. 495.

his observation in Philadelphia.[1] A poor family, consisting of a laboring man and his six children, after a meal composed of tea, bread and cheese, were seized with severe vomiting and purging, with dizziness of the head and great prostration of strength; the liquids discharged from the stomach and bowels were thin and watery, and not very dissimilar to the rice-water discharges of cholera. The attacks were frequent and distressing, and the cases exhibited, at first view, very much the appearance of poisoning from some metallic irritant. These symptoms occurred within an hour after partaking of the meal, and the mother of the family, who was alone unaffected, had been absent from dinner on that day. On the following day all of Dr. P.'s patients had recovered. Similar symptoms having occurred in numerous instances in the same neighborhood, after eating cheese obtained from the same grocer, inquiry was made of him, and it was found that the cheese, from which the deleterious slices had been cut, was one of a large lot from a celebrated dairy in New York, all of which up to that time had produced no unpleasant results, but on the contrary had been considered remarkably good. There was nothing in the taste or external appearance of the remnant to indicate any poisonous properties. It was moreover tested by a competent chemist, and no mineral poison was detected in it. Dr. P. was unable to explain the sudden development of poisonous properties in any other way than by reference to the peculiar state of the atmosphere existing at the time. "It was in the month of January, during a spell of remarkably damp, foggy, and mild weather, succeeding to a cold atmosphere. In the two days during which these cases occurred, the air was loaded with moisture, and the fog on the Delaware was so heavy that the boats were very much impeded in crossing. Might not the softening of a mass of cheese, after being hardened by freezing, develop deleterious properties?" He also considers that the fact of the cheese being mild and newly made, would favor the changes referred to.

Here we are brought face to face with the same teaching illustrated in the matter of damaged sausages or arrested putrescence of meats; it will be wise to look upon this matter in the same light. Nothing is more liable than cheese to undergo putrefaction, and

[1] Trans. Col. Phys., Jan. 1854.

the limit, beyond which the healthy intestinal tract can accept the burden of expelling what is deleterious in our food, is not yet known. Undoubtedly it will be admitted by any one experienced in sumptuous dinner festivals, that it is more difficult to digest cheese in the early stages of putrefaction than when it has gone through the final processes and become what the epicure calls "ripe;" it is also true that a fresh cheese is more digestible than either, that a poor, thin skim-milk cheese is less indigestible than a rich creamy one, and that the latter is more liable to decay. It should be added, moreover, that a cheese may be placed in such circumstances that it becomes more rapidly mouldy (filled with unwholesome vegetable moulds) and corrupt. This subject, not only of cheese, but also of decomposing milk, has not yet been subjected to such careful scientific study among biologists, that the medical practitioner can satisfy himself under what physical or chemical condition this organic substance, cheese, is wholesome or deleterious. The next few years of scientific and microscopical study will undoubtedly determine this point, as has been already mentioned in speaking of sausages.

IV. *Poisonous Fish.*

§ 318. There can be no doubt, as Dr. Christison observes, "that the subject of fish poison is one of the most singular in the whole range of toxicology, and none is at present veiled in so great obscurity." In many cases it is possible that the symptoms of poisoning may be due to the fish having been kept too long, in others to its mode of preparation, as by pickling or smoking; but nevertheless some few cases will still remain in which the freshest and usually most wholesome fishes have caused symptoms of irritant poisoning. These cases are, however, far more common in tropical countries, their occurrence in this latitude being so rare that it is reasonable to suspect either idiosyncrasy on the part of the persons eating the fish, or some deleterious quality acquired by it after its removal from the water.[1] Here again we refer our readers to what has been written above on the subject of unsound food, since the same course of

[1] Consult Moreau de Jonnés, Recherches sur les poissons toxicophores des Indes Occidentales, Annales de Thérapeutique, i. 461; Guillon, Abeille Méd., xii. 67. See also an account by Dr. Hamilton, in the London Pharmaceutical Journal, Jan. 1853, p. 344.

reasoning applies equally well to fish, because these are subject to the same natural laws of decomposition and deteriorated conditions under which food may become an irritant poison, acting upon the *primæ viæ* and the emunctory outlets.

§ 319. *Oysters* have not unfrequently proved dangerous. On several occasions in France, they appear to have become suddenly unwholesome.[1] In the autumn of 1854, numerous deaths in our principal cities were ascribed to their use, and it was generally conceded that, for a period of a few weeks in the month of October, they frequently gave rise to choleraic symptoms. No clue to the nature of the poison was in this instance, or in any of the previous epidemics in France, obtained by chemical examination. We doubt very much whether *crabs* and *lobsters*, if not eaten too long a time after their capture, acquire any peculiar poisonous quality apart from their general unwholesomeness as articles of food.

§ 320. *Mussels*, however, by general consent seem to have a specific poisonous property accorded to them. The idea that it is due to an impregnation from copper is wholly untenable, since not a trace of this metal has been discovered in those taken from the stomachs of persons who have been killed by them. Unquestionably, in many instances, idiosyncrasy is the cause of the mussel proving unwholesome to some individuals. This, also, is the conclusion at which M. Lunel arrives after a careful investigation of the subject.[2] But it need hardly be remarked, that it, after all, amounts to a confession of our ignorance of the efficient cause of poisoning by these shell-fish.

In the cases described by Dr. Combe, of Leith, not only were the mussels perfectly fresh, but every person who ate those from a particular spot was more or less severely affected, and even animals were poisoned by them, a cat and a dog having been killed by the suspected article. Dr. Christison was unable to detect in them any principle which did not exist in the wholesome mussel. Many cases descriptive of the symptoms have been reported. The following is a recent one: A boy, aged ten years, ate the thick part of two mussels; forty-five minutes afterwards he complained of uneasiness

[1] Mém. sur les Empoissonnements par les Huitres, les Moules, les Crabes et par certains Poissons de Mer et de Rivière, par A. Chevallier et E. A. Duchesne. Ann. d'Hyg., vol. li.

[2] Abeille Méd., Juin, 1857, p. 173.

in his stomach; he had a sensation of heat, giddiness, and a desire to vomit; there was an eruption of nettle-rash over the whole body as far as the knee, attended with swelling of the face and intolerable itching; after an emetic, the symptoms disappeared completely.[1] Very much the same symptoms were exhibited by the persons seen by Dr. Combe.[2] In the fatal case of an adult of intemperate habits, reported by Dr. Lee, who, with his whole family, seven in number, were poisoned by eating mussels, the attack commenced with severe distress in the stomach, followed by vomiting and purging, painful muscular spasms with great anxiety and prostration; the pulse was frequent and feeble, the skin of a deep crimson or livid color, and covered with a cold, clammy sweat; sleeplessness, subsultus tendinum and delirious agitation, great heat at the epigastrium, the rest of the surface being cold, pupils contracted, and face sunken; voice and intellect unaffected until four hours before death, about which time vomiting of matters resembling coffee-grounds came on; death occurred about forty-eight hours after the time of the attack.[3] Chevallier and Duchesne report a number of cases of poisoning by the *roes* of a fish called the barbel (barbillon), and several cases of a similar origin are reported by Dr. Trapenard.[4] The symptoms were such as would be produced by a violent emeto-cathartic; copious evacuations, and constant efforts to vomit, headache, frequent pulse, great pain, and an insupportable sensation of heat.

A girl, seven years of age,[5] whose parents reside in Sinclair's Close, Port Glasgow, gathered some mussels from the quay, had them boiled, and ate them. On the following day she suffered from severe vomiting. Medical aid was sent for in the evening, but the doctor arrived just in time to see the child die.

It is not improbable that in the above case, perhaps in some others, these shell-fish, from their habits as scavengers, may have received and given to the victims eating them some decomposing organic matter, which produced what is called septic poisoning, or a peculiar form of organic poisoning from putrefying materials received into the blood and tissues.

[1] Gny's Hosp. Rep., 1850, 213.

[2] Ed. Med. and Surg. Journ., xxix. 86.

[3] Am. ed. of Copland's Dictionary. Art. Cholera.

[4] Ann. d'Hyg. 1850, and Journ. de Chimie, 1851, p. 584. For a recent case, see Edinb. Med. Journ., April, 1860, p. 958.

[5] Lancet, June 5, 1880.

V. *Poisonous Puddings and Vegetable Matters.*

§ 321. While meats, cheese, fish, etc., produce deleterious action upon the human organism, so bread and other cereals or vegetables may be exposed to certain atmospheric changes, under which they may produce deleterious effects, and even act as poisons. One well-known instance is that of the "smut of rye," called *secale cornutum* or ergot, which is undoubtedly a poison, and should be classed as such. There are other vegetable moulds which are likewise injurious to health. The long-continued use of ergoted rye or other grain has given rise, in certain countries in Europe, to epidemics of ergotism, which are called gangrenous and spasmodic, the disease being much favored by cold and hunger in persons not in robust health. When the summer is wet and cold, rye is more apt to become "smutted" or ergoted. Gangrenous ergotism begins with a peculiar itching sensation in the feet, backache, muscular spasms, apathy, dizziness, and nausea, followed by pains in the legs; coldness of surface, and chilliness is experienced; afterwards a gangrenous spot appears, with loss of sensation in the neighboring tissues, or in an extremity, if this be the locality of the gangrene, and in the centre of this spot a large, watery blister appears; general malaise and exhaustion follow, which may terminate in death. In the spasmodic ergotism, the symptoms of muscular tremblings and contractions preponderate with nervous phenomena, such as peculiarities of vision and dizziness, with catalepsy and epilepsy, delirium, and idiocy. Gastro-intestinal irritation is very marked, and the skin assumes a yellow or earthy pallor, accompanied with eruptions or boils. Death may occur from exhaustion, or earlier from convulsions or cerebral symptoms. While it is true that this peculiar disease may result, under favoring circumstances, from eating ergoted grain, it is rarely, if ever, caused by the use of the medicinal preparations.

It has been sometimes supposed that mouldy bread, made of rye or flour, may act somewhat in the same way, but the proof of this is not yet recorded. A few instances are reported where bread-puddings have caused death, and in these cases the symptoms have been those of a gastro-intestinal irritant.

A remarkable case of fatal poisoning[1] of two persons from eating

[1] Sanitary Record, London, Oct. 25, 1878.

a bread-pudding is reported by Prof. A. H. Allen, of Sheffield, England. Two bread-puddings were made from scraps of bread and sandwiches, which had been accumulating for some weeks, together with the addition of milk, eggs, sugar, currants, and nutmegs; these scraps were very mouldy, and were mixed with ham, butter, etc. The two puddings were baked in separate ovens, one, a very large pudding, being imperfectly cooked in a notoriously slow oven, and was subsequently eaten by the cook who made it, the proprietor of the eating house, the waiter, a child of three years old, and a customer. Every one who ate of this pudding was violently ill with symptoms of irritant poisoning. One person only ate of the second pudding, and was not made ill. The pulse of the poisoned persons was greatly accelerated, but none had bloody stools. The child and the waiter died, the latter lingering a week. A *post-mortem* examination of the viscera of the child leading the medical men to suspect its death from poison, the organs, together with the remains of both puddings, were sent to Prof. Allen for a thorough chemical analysis. He made a most exhaustive research, with wholly negative results, and fed a puppy on the suspected pudding for two whole days without the least appearance of any poisonous effect. The evidence that this suspected pudding had poisoned the persons who ate it was so overwhelming that, at the suggestion of Dr. Tidy, a careful examination for ergot was made by Prof. Allen, as well as a microscopical research for fungi; both of which were found. The flour used was examined unsuccessfully for ergot, and this was the case with another pudding made from the same materials used in making the bread. The poisonous pudding showed a large amount of the mouldy fungus, but the latter did not give the lake-red reaction with soda, which is characteristic of ergot. The poisonous pudding had no sign of crust on it, but was "sloppy," and of the consistency of bread and milk. The lumps of bread in it were distinguishable, and the starch granules were, in many cases, unaltered.

The verdict of the jury was that the diseased persons "died from eating unwholesome pudding, but how and by what means such pudding became unwholesome, there is no sufficient evidence to show."

According to Dr. Taylor,[1] ergot in large doses causes symptoms

[1] Op. cit.

of an irritant poison with diarrhœa, and the appearances after death are chiefly patches of inflammatory redness in the mucous membrane of the stomach and small intestines; but, as it is not probable that sufficient evidence of the presence of the active principle of ergot can be obtained from the body by chemical analysis, reliance must be placed on the detection by the microscope of ergot fungus in the matters suspected of furnishing the poison.

Dogs are not readily poisoned by ergot, so that the negative proof offered in the case of Prof. Allen, where the puppy was fed on the poisoned pudding, should have little weight if the poison were the mouldy fungus of ergot. Mr. S. A. Wright[1] gave to dogs an amount of ergot equal to one hundred and twenty grains to each pound of their weight without injury or poisonous symptom.[2]

§ 322. *Chemical examination.*—Ergot contains the volatile alkaloid trimethylamin, which is easily recognized by its peculiar odor which resembles that of pickled herring. This odor is set free in ergot, or in any mixture containing ergot to the extent of more than one and one-fourth per cent., by adding a solution of potassic hydrate of 1.33 specific gravity. If less than this amount of ergot is contained in any meal, flour, or bread, the odor of trimethylamin cannot be detected simply by adding potassic hydrate to it, but we must resort to a longer process recommended by Jacoby,[3] which depends upon the detection of a constituent of ergot, which is soluble in alcohol acidulated with sulphuric acid, this solution having a red color.

Ten grammes of the meal or flour is extracted twice with thirty grammes of boiling alcohol of ninety per cent., and strained through cloth; this does not remove the desired coloring matter, but does get rid of a number of impurities. The residue is then transferred to a test-tube, covered with ten grammes of alcohol, and vigorously shaken; the supernatant fluid should be colorless; if not, the purification with alcohol must be continued until it is colorless; when this point is reached, from ten to twenty drops of dilute sulphuric acid (1 : 5) are added, the mixture shaken vigorously and allowed to settle. If the original substance were pure, the supernatant fluid is colorless or only pale yellow, but if it contained ergot, the fluid

[1] Edinburgh Med. Journ., 1839.

[2] See Husemann's Handbuch der Toxicologie.

[3] Pharm. Zeitsch. f. Russland., Jahrg. III. page 25; and Dragendorff's Ermittelung von Giften, page 304.

is more or less red according to the amount present. Ergot when present in meal to the extent of only one-fourth per cent. can be detected in this way. Bread containing ergot is spotted violet. The red coloring matter above mentioned cannot be extracted from bread, and it is doubtful whether it can be extracted from the contents of the stomach and intestines.

VI. *Mechanical Irritants.*

§ 323. There is a vast number of indigestible substances which, when introduced into the stomach, may give rise to fatal consequences. Pins, needles, and powdered glass are those which, in case of death, are most likely to give rise to a suspicion of their having been criminally given. Naturally, such questions can be reasonably raised only in the cases of children or imbecile persons; although, indeed, at one time, glass in powder was considered as extremely poisonous, and was occasionally criminally administered. Thus, in France, in 1808, a man was tried for poisoning his wife with this substance. It was found in the stomach, but there were other causes which might have produced death. A negro woman, in Jamaica, attempted to poison a whole family with pounded glass, put into a dish of curried fish. The fact was discovered towards the end of the meal, and purgatives were given, which brought away large quantities of coarsely powdered bottle-glass. The persons did not suffer any inconvenience. Dr. Bowling, of Kentucky, obtained as much as eighty grains of powdered glass from the discharges of a child. It had not suffered at all.[1] Nevertheless, Dr. Christison reports a case in which a child, eleven months old, was evidently killed by it.[2]

Pins and *needles*, when swallowed, rarely cause death; they frequently emerge from various parts of the body or are found after death in the viscera. Thus a case is related in which a needle was found in the kidney, in another in the liver, in another in the heart, and in another across the œsophagus; the point of it had, in the last case, penetrated the common carotid artery, and produced fatal hemorrhage. Sometimes, no doubt, they are thrust under the skin by hysterical patients, animated by a morbid desire of attract-

[1] West. Journ. of Med. and Surg., Nov. 1848.

[2] Op. cit., p. 504.

ing attention and curiosity. They may be the accidental cause of death in many ways, but the most usual is that of disturbance of the digestive functions. A girl, twelve years of age, to avoid work, swallowed pins and needles, of which nineteen of the former and five of the latter were passed from the bowels. She suffered occasional colicky pain, and had much swelling and hardness of the abdomen.[1] A case is related,[2] in which a woman eventually died from the continued irritation produced by a quantity of needles she had swallowed. The stomach, which was enormously enlarged, contained *nine* ounces of pins of a purple-black color, and the duodenum contained a *pound* of the same. In the body of a lunatic, who died suddenly of peritonitis in the Peekham House Asylum, the following articles were found in the stomach: thirty-one entire spoon-handles about five inches long, four half-handles, nine nails varying in size from a garden-wall nail to a spike nail, half of the iron heel of a shoe, a screw two and a quarter inches long, four pebbles, and one metal button, weighing in all two pounds eight ounces. The whole of them were stained of a black color, and the angular articles rounded off and blunted. In the duodenum an entire spoon-handle was found, and here the perforation occurred which caused the peritonitis.[3]

A case in which a mass of hair and string, weighing from eight to ten pounds, was taken from the body of a girl aged eighteen, who had been in the habit of swallowing these substances, is related in the same journal.[4] A remarkable instance of the passage of a tinned iron fork through the whole alimentary canal was communicated to the French Academy of Medicine by Velpeau.[5] Another in which death was caused by eating raw rice.[6] M. Gosselin related, to the Surgical Society of Paris, the case of a man who swallowed a clay pipe three inches and a half in length. It occupied two months in passing through the bowels, and at the end of this time it was discharged unbroken. But the man's health had suffered severely from the irritation it had caused, and he died five days after its discharge.[7] Cases enumerated in this section are evidently rather subjects of curiosity than of any importance in legal medicine.

[1] Boston Med. and Surg. Journ., Oct. 1859, p. 227.
[2] Lancet, Am. ed., 1852, p. 224.
[3] Lancet, 1852, vol. ii. p. 296.
[4] Vol. i., 1852, p. 224.
[5] Ibid., 1849, p. 246.
[6] Ibid., April, 1847.
[7] Ibid., 1851, vol. ii. p. 462.

CHAPTER VIII.

NARCOTIC POISONS.

§ 324. The name *narcotism* is applied to a certain condition of the nervous system produced by the action of a class of poisons called narcotic.

The effects of poisoning by the narcotics are: heaviness of the head, vertigo, exaltation of the senses, increase of temperature and of the impulse of the arteries (caused by a congestion of the smaller bloodvessels), dryness of the throat and skin, nausea, vomiting, diminution and suspension of all the secretions, and very lively *pruritus* (extreme itching of the skin), often accompanied by papular or vesicular eruptions; a drowsiness sometimes profound from the commencement, relaxation of the extremities, suffusion of the face, a fixity of expression of the eyes, and sometimes dilatation of the pupil, generally a stertorous respiration increasing until death, which generally takes place from seven to twelve hours after the ingestion of the poison, but may be hastened by the contraction of the thoracic muscle or diaphragm, or by convulsions. Recovery is marked by an abundant perspiration, and by the successive return of sensation and intelligence.

While the above description may be considered generally true as a *résumé* of the action of a number of narcotics, the action of no particular narcotic should be comprised in this general description, which should be still further subdivided into: First, a class whose primary quieting action is exerted upon the brain centre, and suppresses the intellectual faculty, and induces sleep; second, a class whose primary effect may also be exerted upon the brain centre, but manifests its action by an excitement of the intellectual faculty, causing dreams of an exciting or exhilarating type, and secondarily causes a stupefying effect resulting in sleep; and third, a class whose primary action is not upon the brain centre, and which may not produce soporific or exciting action of the intellectual faculty, but whose action is sedative and calms nervous excitement or cerebral agitation, moderates spasmodic movements or pains; some of this class are feeble narcotics, such as hemlock, and others are more energetic in their action, such as hydrocyanic acid.

These narcotics might be still further subdivided into subordinate classes, but it will be more convenient to consider in a treatise on poisons each drug by itself, in order to prevent confusion or repetition of symptoms.

The first class of narcotics includes opium, chloral, ether, carbolic acid (previously mentioned, § 74), and chloroform, together with others to be presently described.

The second class, called also mydriatics because they cause dilatation rather than contraction of the pupil of the eye, includes, among others, belladonna, hyoscyamus, and datura stramonium.

The third class, which has no action upon the pupil, includes conium or hemlock, and hydrocyanic or prussic acid.

We shall defer a description of the symptoms of each narcotic to the special paragraph devoted to each individual poison.

§ 325. The pathological or *post-mortem* appearances resulting from narcotic poisoning are principally a very intense congestion of the principal organs, especially of the lungs and brain; the blood is black and fluid, and the right side of the heart filled with fluid, dark colored blood. They resemble in this respect the action of certain deleterious gases, coal gas, etc. These latter effects are probably due to the imperfect aëration of the blood from an

embarrassed respiration occasioned by the spasms of the muscles concerned in the act of inspiration and expiration.

Cerebral or pulmonary apoplexy and congestion of the brain may be mistaken for poisoning by the narcotics; but the first can generally be distinguished by a contraction of some few of the muscles of one side of the body, especially of the face and mouth, and by peculiarities in the reflex phenomena of sensation and muscular motion, as well as imperfections of the faculty of speaking or naming correctly material or abstract ideas; the second, if no vomiting or expectoration of blood occurs, could be distinguished at the autopsy and by chemical examination.

I. *Opium and its Preparations.*

§ 326. The *symptoms* produced by a poisonous dose of opium or its preparations, differ from those which are occasioned by moderate and remedial doses of the drug. While in the latter the purely narcotic effects do not occur without a certain degree of previous exhilaration and stimulation, in the former dizziness and stupor are the first symptoms, or the excitement is so temporary as to pass unnoticed.

§ 327. The crude drug, opium, contains various active principles, such as morphine, 12 to 16 per cent.; codeine, 0.03 per cent.; narceine, 0.02 to 1 per cent.; narcotine, 1.30 to 11 per cent.; thebaine, 1 per cent.; papaverine, 1 per cent.; meconic acid, 3 to 4.3 per cent.; and traces of other less important constituents, such as cryptopine, hydrocotarnine, pseudo-morphine, laudanine, protopine, meconidine, laudanosine, lanthopine, and rhœadine. Of these alkaloids, morphine, codeine, narcotine, thebaine, narceine, and papaverine are poisonous to the lower animals, though there are no recorded instances of death being caused in man by any other than the morphine. As the latter represents the poisonous principle of opium in man, we shall discuss the action of this alkaloid rather than the crude substance imported under the name of opium. By authority of the United States Government, opium is admitted to our country in a strength based upon its morphine richness, which must be regulated according to the prescribed assay and standard of the Pharmacopœia of the United States of America. The last edition of this official book, Sixth Decennial Revision (1880), prescribes for the minimum standard of opium that it shall contain

12 per cent. of morphine, and publishes a process under which all opium shall be assayed. The maximum strength of opium, according to the same authority, shall contain 16 per cent. of morphine. Therefore the strength of opium preparations of the present date will probably be found to contain about one-third more of morphine than during the last decade, since the Pharmacopœia of a previous edition required in imported opium only 9 per cent. richness in morphine. The denarcotized tincture of opium causes in proportionate doses nearly the same symptoms upon man as may follow the use of morphine, though it is less liable to cause the troublesome itching or pruritus of the skin observed after morphine has been absorbed into the system.

§ 328. Opium itself is absorbed into the system slowly or rapidly according to the method by which it may be administered, and the form in which it is given; for instance, an old opium pill may be swallowed and so slowly acted upon by the digestive fluids in the stomach, that its peculiar effects follow very slowly. Opium may be administered combined with oil of theobroma as a suppository in the rectum and be slowly absorbed. Liquid preparations of opium are absorbed more slowly in the rectum than when swallowed into the stomach. Opium administered to a fasting stomach is more rapidly absorbed than when taken with food. Upon these and similar circumstances depend the absorption of opium and its active principle (alkaloid), morphine; hence it follows that when it is more slowly absorbed, it may be gradually undergoing elimination from the body, and the symptoms of its action may be delayed or so much retarded and modified that what might in one case be a poisonous dose becomes under other circumstances a non-poisonous dose. In regard to its action in diseased states of the eliminating organs, reference is made to a previous paragraph (§ 3), where this part of our subject has been fully discussed.

§ 329. Opium smoking is another method of its administration, and though not precisely like the effects produced upon the body by other avenues of introduction, its stupefying and intoxicating effects are pretty generally known from the popular works of De Quincy and other writers; in the case of smoking, however, some of the alkaloids are destroyed or weakened by the action of heat or by combustion, and other injurious alkaloids formed, such as picoline.

§ 330. The hypodermic or subcutaneous method of introducing opium into the system is unfortunately well known to others than medical practitioners, and undoubtedly might be used in cases of criminal intent, though no recorded cases are known to the writer where a criminal has resorted to this means of destroying the life of his victim. The pain involved in the method is an obvious disadvantage which would occur to the mind of an unprofessional person desiring to escape the consequences of his criminal action.

Morphine itself is rarely used, as it is not so soluble in water as its salts. By an easy computation one part of the sulphate and the hydrochlorate of morphine will be found to contain about three quarters of their weight of morphine, and the acetate about two-thirds. The most commonly used of these salts of morphine are the sulphate and acetate. The subcutaneous method of introducing morphine is followed in about fifteen minutes with the symptoms of its action, and in several recorded instances where this method has been employed upon persons whose great susceptibility to the poisonous action was unknown, a small dose (one-fourth of a grain) has caused death, and one instance is recorded where even one-eighth grain was followed by profound and fatal coma, or a state of hebetude from which it was impossible to arouse the patient. Occasionally, too, a careless introduction of the drug directly into a vein has deluged the central circulatory system with a dose which would not, if slowly absorbed, have done any material harm.

§ 331. *Symptoms.*—No matter in what way morphine may be given to a person in good health, the absorption of an amount sufficient to produce poisonous effects is followed by a train of symptoms easily recognized under the head of opium narcosis: A sensation of fulness or dizziness of the head accompanied by a feeling of mental and physical ease, an exhilaration of intellectual activity, and a rather rapid pulse; often, however, there is an unpleasant disquieting sensation of an intolerable itching of the skin (*pruritus*), especially noticed about the nose and forehead, which the patient makes slow and feeble efforts to relieve by scratching; and which may extend all over the body, and may even interfere with the sleep-producing effects of the drug. Nausea and vomiting may be an early symptom, and sometimes, when the dose has been too large, this symptom is so severe that it may cause rejection of a large amount of the poison, and thus prevent the absorption of an

amount sufficient to cause death; it is in this way that occasionally a person intent on self-destruction has awakened to a consciousness of his object having been defeated. Faintness is sometimes an early symptom, but instances of this are not common. A sufficiently large dose, or the continued use of several small doses, causes a very marked contraction of the pupil of the eye, and generally a loss of sensitiveness of the surface of the eyeball. The experienced medical practitioner will recognize this condition of the eye as peculiar to opium narcosis. After these primary symptoms the individual will fall into a condition of sleep or drowsiness accompanied with quiet and pleasant dreams, not unfrequently with a cold and clammy skin bathed in perspiration with suppression or retention of urine, and sometimes with increase of surface temperature and a general blush. If the dose be repeated after this train of symptoms has appeared, or if the original dose has been large enough, these symptoms grow more intense, and the pulse and respiratory movements become very much slower, the latter being reduced from the normal number of eighteen per minute to three or four per minute. With poisonous doses, the period of intellectual excitement and exhilaration is almost always so short as to escape observation, and the whole train of symptoms above described in detail follow each other so rapidly that the victim may fall into a deep sleep from which he may at first be aroused, but this is soon succeeded by coma, and stertorous and slow breathing, the pulse is sluggish and scarcely perceptible, the face dusky and cyanotic, the extremities cold and clammy to the touch, the pupils contracted to a pin's point; but just before death takes place, signs of asphyxia are present, the pupils dilate and the pulse becomes quick and scarcely perceptible. It should be noted here that the urine may be albuminous and contain casts from the tubules of the kidneys, which may lead to the suspicion that the victim is the subject of Bright's disease. We have already seen in fatal poisoning by other violent poisons (mercury, ammonia, arsenic, phosphorus, etc.), that this eliminating organ may be subjected to such efforts to get rid of the fatal drug, that it becomes congested, and may show signs of disturbance of its functions by secreting albuminous urine containing casts; this is undoubtedly a common occurrence in poisoning, and should not be overlooked. If the kidneys have been the subject of disease, the *post-mortem* appearances will solve the

doubt whether the disease or the drug is responsible for the fatal result.

The apparent inconsistency in the presence of two opposing conditions of dryness and excessive moisture of the surface of the body needs some comment. Generally speaking the dryness and warmth of the surface of the body is the most common symptom, but there are a number of undoubted cases of morphine poisoning where copious perspiration is a singular and infrequent symptom. It is mentioned by Christison, who says that in one case "the sheets were completely soaked to a considerable distance around the body;" and Dr. Morland observed it, in an equal degree, in a case he has reported.[1] Delirium is very rare, and, when it occurs, is of a passive character. In the adult, *convulsions* have been seldom observed, although one or two curious cases have been reported in which they were witnessed. In children, however, they are not uncommon. Occasionally spontaneous vomiting takes place, especially after the ingestion of the drug in large quantities, and some instances have occurred in which this early rejection of the poison from the stomach has saved the life of the individual. A child nine years of age, mentioned by Dr. Coale, recovered in this way after having swallowed four grains of opium and four of extract of belladonna. The pulse also varies in character, being usually feeble and irregular, but sometimes full and slow; in a case reported by Dr. J. B. S. Jackson, it is described as "rapid, full, and throbbing." Such differences depend often, probably, upon the variable periods at which the observation is made; thus, the skin is warm and perspiring, and the pulse rapid and feeble, or perhaps even strong, early in the case; while later, if the patient gets worse, the surface becomes cold and pale, and the pulse slow, feeble, and irregular. Much also may depend upon the constitutional irritability of the system, but in persons who have accustomed their system to the use of morphine, enormous doses may be tolerated without producing opium narcosis or other dangerous effects; though it should be remarked that the pernicious habit of continued use of morphine most usually entails disease and an early death from chronic poisoning, though there are exceptional cases.

The differences between poisoning by opium and by other narco-

[1] Am. Journ. Med. Sci., Oct. 1854.

tics are briefly these: aconite, digitalis, and tobacco do not produce stupor, nor does conium, except in very large doses, and even then not uniformly; hyoscyamus, stramonium, and belladonna excite violent delirium, and extreme dilatation of the pupil. Inebriation by alcohol bears a very close resemblance to opium narcotism in many cases, but the former is preceded by confusion of ideas or complete delirium, and the breath is strongly tainted with the alcoholic odor.

§ 332. The symptoms usually commence, in the adult, within an hour after the poison has been taken, but sometimes the confirmed narcotic effects do not come on until a later period. In a case quoted by Dr. Taylor, the patient was found totally insensible in fifteen minutes. In Dr. Lyman's case,[1] a female, after taking an ounce of laudanum with suicidal intent, began very suddenly, in thirty-five minutes, to lose her pulse and muscular power, and had slight spasms; the lips became livid, there was spasmodic dropping of the lower jaw, the extremities were cold, and in ten minutes more she was unmistakably dead. Thus, three-quarters of an hour only elapsed from the ingestion of the laudanum to her death. Dr. Coale[2] states that he met with a case fatal in the same time, and Dr. Taylor quotes a similar one. Dr. Beck reports a case which proved fatal in two hours. Another is given which terminated in two hours and a half.[3]

§ 333. The *average duration* of cases of poisoning by opium is stated by Christison to be from eleven to twelve hours. The rapidity, or indeed the certainty, of death does not always correspond with or depend upon the amount taken, when this is beyond the limits of safety. Among the cases above referred to is one by Dr. Jackson, which recovered, although ninety grains of opium were taken, and no relief was afforded for three hours afterwards. In the case mentioned by Dr. Taylor, of a man who had taken from twenty-eight to thirty grains of opium, the symptoms were so little characteristic of poisoning by this drug, that no suspicion was entertained of its having been used, and death took place rather suddenly in ten hours after the fatal dose had been

[1] Am. Journ. Med. Sci., Oct. 1854.
[2] Ibid., Jan. 1850, p. 73.
[3] Bost. Med. and Sug. Journ., vol. xi. p. 285.

swallowed.[1] On the other hand, the most rapidly fatal case yet recorded was the one above referred to, in which only an ounce of laudanum was swallowed. The fact should, however, be borne in mind that laudanum, or any *solution* of opium, is more prompt in its effects, because it is more readily absorbed than solid opium.

§ 334. *Amount.*—Owing to the varying susceptibility of individuals to the poisonous action of opium, it is not possible to state the *amount* which will be uniformly fatal. The dose which may cause poisonous symptoms varies even among those unaccustomed to the use of morphine, but when the habit of taking morphine is an old one, it is impossible to attach any limit to the tolerated dose. Cases have been reported where a dose of from twenty to fifty grains have not caused a fatal result. It should not be gathered from what is here stated that no dose of morphine can cause death even in those who are habituated to its use, since this inference is not true; the only doubt exists as to stating in advance what number of grains is sufficient to cause death in any given case of a morphine eater, who takes a definite amount per diem of the same drug. There are two factors which interfere with a correct answer to this question; first, the difficulty of ascertaining accurately what amount of morphine is being taken each day; and second, the insufficient number of attested cases upon which to base an opinion. The former of these conditions is obscure, from the fact that the opium or morphine eater is especially anxious to conceal his habit, as well as the amount which he takes daily, and the vitiated intellectual condition caused by his habit has so obtunded his moral sense, that no intelligent and experienced medical practitioner would accept his statement on oath as to the dose he habitually takes.

The personal experience, not only of the editors, but of other physicians who have had under observation cases of this character, tends to show that it is only by dogging the steps of the individual and those about his person that any correct surmise can be made. But, leaving entirely out of our consideration of this question the morphine and opium eater, it is difficult to form an opinion of the precise size of the fatal dose of morphine required in individuals not accustomed to its use.

A case is referred to by Dr. Taylor in which four grains proved

[1] Med. Jurisp., 6th ed., p. 162.

fatal, and another in which death was supposed to have resulted from a dose of two drachms of laudanum, but it was uncertain whether as much as half an ounce had not been swallowed. A gentleman affected with acute rheumatism died comatose after taking, at intervals of an hour, four pills, each of which contained one-third of a grain of morphine.[1] In Dr. Lyman's case, one ounce of laudanum was the cause of speedy death. The smallest dose which has proved fatal to a child is the one-twentieth of a grain of opium.[2] The child was six days old. Dr. J. B. S. Jackson met with a case in 1845, in which five drops of laudanum, injected into the rectum of a child eighteen months old, caused death in six hours. Instances are quoted by all writers on toxicology of death in children from extremely small doses, such as the one-tenth or the one-fifth of a grain, and most practitioners have witnessed alarming symptoms from a few drops of laudanum, or fractional doses of Dover's powder given to children. In many instances these have been dissipated only by active medical interference, such as cold affusion, galvanism, etc.[3]

Trousseau states that he has seen narcotic effects in children from a dose of the wine of opium equivalent to less than the one-hundredth of a grain of this drug. It is well known that a child may be narcotized by the milk of a nurse who has taken opium. Bouchardat relates that nine new-born children were narcotized by the decoction of a single poppyhead. In London an infant four days old was destroyed by one-twentieth of a grain of opium, or about one drop of laudanum;[4] and in Edinburgh the same effect was produced by two drops of laudanum in an infant also four days old.[5]

The following cases prove the possibility of recovery after excessive doses of this drug. A gentleman, seventy-two years of age, recovered from the effects of twelve drachms of laudanum;[6] another,

[1] Med. Times and Gazette, June, 1860, p. 254.

[2] Dr. E. Smith, Assoc. Med. Journ., April, 1854.

[3] *Vide* I. Young's case, and the references given by Dr. Hays, Am. Journ. Med. Sci., April, 1852, p. 426; also a very instructive case by Dr. Herapath, where respiration was artificially maintained by means of the galvanic battery until the narcotism passed off, and the child was saved. Lancet, Am. ed., 1852, p. 450.

[4] Times and Gazette, April, 1854, p. 386.

[5] Edinb. Med. Journ., ii. 146.

[6] Lancet, July, 1857, p. 80.

aged thirty-five years, after half an ounce of this preparation had been taken ;[1] in the third case above an ounce was swallowed, and, although the symptoms were intense, the patient recovered, temporarily at least, with paralysis of the right side.[2] Another case is reported, in which ninety grains of opium were taken by a female, who, however, recovered.[3] An infant of twelve months has recovered from the effects of seventy-two drops of laudanum ;[4] another, six days old, after two grains of powdered opium ;[5] and a child, not quite six years old, from a dose of seven and a half grains of opium, which were, however, mixed with an equal quantity of prepared chalk.[6]

According to Dr. Taylor,[7] cases are recorded where one grain of the hydrochlorate of morphine has proved fatal to adults, one of whom took the medicine in solution, and died in about seven hours ; a second in pill, and died in thirteen hours ; a third in a powder, and died in about ten hours ; and a fourth by subcutaneous administration, and died in about eighteen or twenty hours. A fifth case[8] is reported in which a woman died in twelve hours after taking only one grain of morphine in a pill. "Dr. Anstie met with a case in which three grains of morphine given as an injection, *per rectum*, caused death in sixteen hours."[9] On the other hand, the Lancet[10] records a case of a child only two years of age, who recovered after a dose of one grain of the acetate. A number of similar cases which are reported in medical journals and other literature, show that the susceptibility to the poisonous action of morphine is very great in some cases, and experience has taught the danger of giving a dose of this narcotic larger than one-quarter of a grain, unless the medical attendant is aware of the peculiarity of his patient in this respect. It is obvious that too short an interval between the repetition of small doses may cause poisonous symptoms, as well as if the commencing dose be large. Dr. Taylor[11]

[1] Bost. Med. and Surg. Journ., Aug. 1855, p. 21.

[2] Brit. and For. Med.-Chir. Rev. xxii. 523.

[3] Am. Journ. Med. Sci., Oct. 1854, p. 385.

[4] Edinb. Med. Journ., iii. 716.

[5] Boston Med. and Surg. Journ., Dec. 1857, p. 357.

[6] Am. Journ. Med. Sci., April, 1859, p. 367.

[7] Treatise on Poisons, Philadelphia, 1875, p. 549.

[8] Lancet, 1872, ii. p. 24.

[9] Ibid.

[10] 1863, ii. p. 8.

[11] Op. cit., p. 550.

shows that in England homœopathic physicians give doses of morphine—and without a written prescription—of size equal to those of physicians in regular practice, who write their prescriptions to be made up by the apothecary. He cites a case in his own experience, where a woman's life was imperilled in this way from alternate powders of morphine to the amount of half a grain, and of sugar of milk without medicament. He suggests that if the woman had died and left only one powder uneaten, it would have been found by analysis to contain only the innocent sugar of milk, and thus, upon the theory of the practice of a homœopathic practitioner, no suspicion would have attached to the poisonous character of the powder, because it would be supposed to contain only an infinitesimal amount of any medicine. We do not say that homœopathists in America practise in this way, but the writer has personal knowledge of a case occurring in his vicinity of a similar character to that quoted by Dr. Taylor. Dr. Eastes[1] has met with some homœopathic globules which contained one grain of morphine instead of $\frac{1}{1000}$ grain.

§ 335. Attention is directed to what has been mentioned in a previous paragraph (§ 327) in regard to the morphine strength of opium. Since a variation is allowed of from nine to sixteen per cent. in the opium imported into this country, it may be seen that the amount of poisonous material in a sample of opium taken from a drug store may vary nearly fifty per cent. in strength. In previous years, when the adulteration of drugs was not so seriously scrutinized as now, this variation of morphine strength may have been very much wider, and hence the above uncertainty in the results of drugs compounded from crude opium is not remarkable. Therefore, slight dependence should be placed upon the history of cases reported as fatal from preparations of which the morphine strength has not been assayed. Nor should much reliance be placed upon the measure of drops, the size of which depends not only upon the physical character of the liquid, but also upon its temperature and the form of the vessel from which the drop is allowed to fall.

The tincture of opium is liable to considerable variations in strength; and although in adults the difference of effect will hardly, within certain limits, be perceptible, it may certainly be so in children,

[1] Lancet, Dec. 13, 1873.

on whom this drug has, moreover, always a disproportionately speedy action. The tincture, properly prepared, should contain one grain of opium in 12.8 minims, or about twenty-five drops. Taking for our guide the United States Pharmacopœia (of 1880)—

100 parts of tincture of opium (laudanum) contains from 1.2 to 1.6 per cent. morphine.
" " " opium (deodorized) contains from 1.2 to 1.6 per cent. morphine.
" " " ipecacuanha and opium contains from 1.2 to 1.6 per cent. morphine.
" " " wine of opium contains from 1.2 to 1.6 per cent. morphine.
" " " vinegar of opium contains from 1.2 to 1.6 per cent. morphine.
" " " paregoric (camphorated tincture of opium) contains from 0.048 to 0.064 per cent. morphine.

Godfrey's cordial contains a little more than one grain of opium in the fluidounce.[1] Dalby's carminative mixture has five minims or ten drops of laudanum in two ounces.

The relative strength of the preparations made according to the directions of the Pharmacopœia of the United States in the edition of 1870, is as follows:—

	Contains	
	of opium.	of morphine.
Tincture of opium (laudanum) . . .	9 per cent.	0.9 per cent.
" " (deodorized) . . .	9 "	0.9 "
Wine of opium	13 "	1.3 "
Vinegar of opium	16.3 "	1.6 "
Camphorated tincture of opium (paregoric)	0.2 "	0.02 "

These figures are calculated from the minimum strength allowed by the United States laws.

§ 336. The fatal consequences arising from the constant and ignorant use of nostrums containing opium or its alkaloids in domestic practice are undoubtedly very frequent. There is a certain chronic poisoning induced by the use of this drug and its derivatives (especially morphine), which has only recently been brought to notice. Paregoric and certain "soothing syrups and

[1] Wood and Bache's Dispensatory.

powders" given to children by ignorant persons in order to produce quiet and sleep, have been proved to be very injurious to health, and in a few instances death has been caused.[1] The children, however, become tolerant of the poison, and, though their death may not be immediately attributed to the continued use of the narcotic, yet their constitution becomes undermined. "In the late Mr. Granger's report on the Children's Employment Commission, it is stated that laudanum and other preparations of opium are given to young children in gradually increased doses, until the child will bear from fifteen to twenty drops of laudanum at a time. The majority of these children die by the time they are two years old."[2]

The following is from the report of the Superintendent of Health and City Registrar of Providence, R. I., on the deaths in that city for March last.[3]

"The decedent from poisoning in March was a child killed by a dose of Mrs. Winslow's soothing syrup. It has long been known to physicians that the soothing properties of this popular medicine are due to opium in some form, and that the quantity of opium is so large as to make it a decidedly dangerous nostrum. There is no doubt that a considerable number of deaths every year should be recorded: 'Poisoned by Mrs. Winslow's soothing syrup.'"

§ 337. The influence of *idiosyncrasy* in modifying the usual effects of opium are often seen, and may be of importance in legal medicine. Thus Grisolle states, that he saw narcotism induced in a lady by half a grain of opium. Dr. Christison mentions the case of a gentleman who was always narcotized by as little as seven drops of laudanum, and Taylor observed alarming symptoms from the injection in a clyster of one grain of opium. Some diseases render the system extremely susceptible to its poisonous action, as has been observed in a previous chapter (§ 3), where this portion of our subject, in relation to forensic medicine, has been fully described. On the other hand, painful diseases enable the person to use very large doses, not only without injury, but with

[1] California Medical Gazette, Nov. 1869; Am. Journ. of Pharmacy, May, 1872, p. 221, from Pacific Med. and Surg. Journ.

[2] Taylor, Med. Jurisprudence, 1865, p. 1154.

[3] Boston Med. and Surg. Journ., April, 1873, p. 432.

positive advantage, but it has been found by experience, that it is a popular medical fallacy to suppose that morphine or opium may be freely given so long as pain is experienced. There are many cases recorded where this fallacy has led to such free use of the anodyne, that the patient suddenly becomes comatose and dies.

§ 338. The *habit* of taking opium diminishes the influence of this drug upon the system, and doses, which in other cases would be absolutely poisonous, are taken with entire impunity by persons who indulge in this habit. When this habit has been acquired in later adult life, its effect upon the duration of life is not so well marked as in younger persons, yet recent knowledge shows, as a general rule, that opium eaters die from defects of interstitial nutrition or from cerebral diseases.[1] The picture of the opium eaters and smokers of the East, as drawn by travellers, is indeed a melancholy one, and their general testimony is, that *there* it undoubtedly has the tendency to shorten life. This is the conclusion arrived at by Mr. Little, of Singapore, as the result of careful and extensive inquiries at that place of the owners of opium shops, of the smokers who frequented them, the prisoners in the house of correction, and the paupers of a poor-house. The following picture of the effects of the habitual use of opium is drawn by this gentleman: "As the habit grows upon its unhappy victim, the first evils experienced are disturbed sleep, watchfulness, giddiness, sometimes headache, capricious appetite, a white tongue, frequently costiveness, indescribable oppression in the chest, and haziness of the eyes. Afterwards a copious secretion of mucus takes place from the eyes and often from the nose also; digestion becomes much impaired and micturition difficult; a mucous discharge begins to flow from the organs of generation; the sexual organs, at first preternaturally excitable,

[1] This subject received particular attention from Dr. Christison in consequence of its importance in a remarkable civil trial. The Earl of Mar effected insurances on his life to a large amount while addicted to the vice of opium eating, and died two years afterwards of dropsy. He had used laudanum for thirty years, at times to the amount of two or three ounces daily, and died at the age of fifty-seven. He suffered greatly from rheumatism. The insurance company, having been unaware of this habit, refused payment on the ground of its having a tendency to shorten life. The persons holding the policy, therefore, instituted an action against the company, which was decided in favor of the former, but on other grounds.

gradually lose their tone; the body wastes, the muscles lose their tonicity, and the bones are affected with dull, gnawing pains for some hours in the morning. By and by the figure stoops, and a peculiar shuffling gait is acquired, by which alone a practised eye may recognize an old opium debauchee. At the same time, the eyebrow droops, the lower eyelid becomes dark, the eye itself seems to sink and grow dim, and the whole expression is that of premature old age. In both sexes the procreative power is greatly lessened, and in those women who, nevertheless, do bear children, the secretion of milk is defective. The influence of the habit on the generative functions is indeed so decided, that, were it not for fresh arrivals from China and other parts of the East, the population of Singapore would very soon be seriously diminished."[1] Finally, according to this author, structural derangement is induced, the digestive and assimilative functions become very much impaired, strumous affections are readily developed, and the opium smoker succumbs without resistance to any violent disease.

Some Eastern travellers, however, assert that the habit has no tendency to shorten life. Thus Dr. Macpherson says, that, although the habit of smoking opium is, in China, universal among rich and poor, we find them to be a powerful, muscular, and athletic people; and Dr. Burnes, who resided several years at the court of Scinde, says, that "it will be found in general that the natives do not suffer much from the use of opium.[2] A celebrated Cutchee chief, who had taken opium largely all his life, was alive at the age of 80, paralyzed by years but his mind unimpaired."

Dr. Christison, moreover, found upon an examination of twenty-five cases, the particulars of which he obtained from various quarters, that instances of longevity among opium eaters in Great Britain were not uncommon. In most of these cases it is expressly stated that no injurious effect upon the general health was observed; in some instances, indeed, the persons being ruddy and robust in appearance. In a few, unpleasant symptoms were experienced only upon the intermission of the habit. The only inference that can at present be drawn from the testimony of travellers, and from these observations reported by Dr. Christison, is adverse to the

[1] Edinb. Month. Journ., June, 1850. [2] Christison on Poisons.

general belief that opium, like intemperance in strong drink, has a tendency to shorten life when habitually used. Future observation must decide whether this reasonable belief is really the correct one. The possibility of abruptly discontinuing the habit without injury to the constitution has been shown by Dr. Christison.[1]

The most recent therapeutical authority on this subject of morphinism,[2] writes in regard to this subject as follows:—

"A statement of the extreme amounts of morphine taken by persons habituated to its use would have but little value, and could perhaps be paralleled from the experience of a large proportion of the readers of this book. A somewhat extended inquiry among druggists in Massachusetts, by Dr. F. E. Oliver, revealed the fact that a drachm, four grammes, per week is by no means an uncommon allowance for a regular consumer. The dangers of a confirmed morphine habit have probably been somewhat exaggerated so far as the bodily health is concerned. There is no doubt that a person may be a habitual user of morphine for years and retain a reasonable degree of health, sustaining himself nearly as usual in society and business, and keeping his stimulus from being suspected. The effects most obvious to himself will be digestive disturbances, in the form of loss of appetite, dyspepsia, constipation, and emaciation; and on the part of the nervous system, loss of mental energy, clearness, and decision, and extreme and indescribable distress as the effect of each dose passes off, and lasting until its repetition. The most good and least bad effects may be attained by always using the smallest possible dose which will give the desired effect, and increasing as slowly as possible. The usual tendency among habitual users of morphine for purposes of stimulation, is to increase the dose rapidly. The habit is difficult, but by no means impossible, to escape from. Unfortunately it is far from rare, and it is practised in both ways, the ordinary method by the mouth and the subcutaneous injection, the latter causing apparently less disturbance of the digestion. The physician should never put a hypodermic syringe into the hands of a patient for habitual use, except under extreme necessity; and if obliged to do so occasionally, should withhold all instructions which would enable the patient to continue

[1] Edinb. Month. Journ., June, 1850. United States Pharmacopœia, Robert
[2] Therapeutic Handbook of the T. Edes, M.D., New York, 1883.

the administration without medical advice. Many so-called opium antidotes are solutions of morphine."

§ 339. There is no doubt in the minds of medical men of the present time that the habit of opium eating is injurious to health, and therefore calculated to shorten life. In any proposal for life insurance, the insurers ought to be informed of this habit, where it exists, and no medical man should sanction its concealment merely because many persons addicted to the habit have lived for years in tolerable health. The practice often is, and may be, concealed from a medical attendant; then the insured, if not candid in avowing its existence, must expose his representatives to the risk of losing all benefit under the policy.[1]

§ 340. *Post-mortem appearances.*—It is quite unnecessary to particularize the morbid alterations which have been seen in persons dying from poisonous doses of opium, since there are none which are sufficiently constant or distinctive to be attributed to this cause. As a general rule, the vessels of the brain and spinal marrow will be found turgid, and the lungs and other vascular organs congested, while the blood remains fluid The multitude of diseases and of accidental modes of death which may occasion these conditions preclude us from attaching any importance to them as indicative of death from opium. Sometimes opium in substance or laudanum may be found in the stomach, or the latter recognized in this organ by its smell, but in the vast majority of cases the poison is rapidly absorbed or eliminated from the system, so that at the *post-mortem* inspection no trace of it will be found.

Though the lesions which have been noticed in the organs of those poisoned by opium have no very specific character, still they are almost always constant. The stomach and intestines are sometimes colored on their interior aspect by the saffron-yellow tint of opium. The mucous membrane shows varied and striking tints, formed by the mixture of the coloring matter of the medicament with the arboriform injections of the bloodvessels, thus adding a vivid red to the yellow color (Tourdes).

M. Tardieu in his excellent treatise on Poisons,[2] quotes the detailed account of a *post-mortem* examination on the body of an old

[1] Taylor, Med. Jurisprudence, 1865, p. 1155.

[2] Op. cit., second edition, p. 1052.

man poisoned by about an ounce of laudanum, as given by Prof. Tourdes.[1] The autopsy was made sixty-two hours after death.

Rigor mortis was general and well pronounced; there was no sign of putrefaction; the abdomen had no bluish discoloration, though somewhat tympanitic. There was nothing remarkable about the expression of the face; the face was not discolored; the eyelids had a bluish tinge. The pupil was moderately dilated, its diameter being four millimeters. The scalp was somewhat injected. The whole surface of the body was pale. There was no sign of papular eruption, but the skin of the chest and neck presented in an unmistakable degree the condition of papillary elevations known as "gooseflesh." There were several spots of laudanum on both hands. The skin of the scrotum was strongly injected; there was no erection of the penis; at the meatus urinarius a whitish milky fluid was found, which, on microscopical examination, was seen to contain spermatozoa. The tongue, which lay behind the dental arch, was yellowish in color, and slightly injected at the base. The soft palate and the pharynx were of a reddish tinge. The upper part of the œsophagus was of a pale pink color; the lower portion was bright red.

The stomach was remarkable on account of the yellowish, greenish, and reddish appearance of its mucous membrane; the yellowish, greenish, and saffron color predominated in the right half; a strong reddish injection was particularly noticeable in the left half. Some red spots with a tinge of purple were observed in the yellowish discoloration extending over a great part of the organ. These spots were formed either by lively very limited injections, or by bloody effusions. The mucous membrane was of the usual consistence, and showed no trace of erosion or ulceration. The stomach contained but a small amount of yellowish matter colored evidently by laudanum. There was a sour smell. The duodenum presented the same yellowish discoloration with red spots and arborizations; its mucous membrane was intact. It contained some yellowish matter like that found in the stomach, but in greater abundance. The yellowish discoloration extended into the small intestine to a point two meters distant from the pylorus; beyond this point the mucous membrane was of normal appearance. The liver was

[1] Gazette Médicale de Strasbourg, 1858, p. 102.

normal in color and size. There was a small amount of bile in the gall-bladder. The spleen weighed 410 grammes; its external covering was covered with old false membranes of a thickness of one to two centimeters. The right kidney was enormous; it contained a calculus as large as an elongated hazel-nut, filling the pelvis, and taking its shape. The calculus was formed of uric acid and urate of ammonium.

The bladder contained a small amount of urine having an acid reaction. The pericardium contained a small quantity of serous fluid of alkaline reaction. The heart was very large, weighing 620 grammes; the left ventricle was much hypertrophied, the thickness of the walls varying from $2\frac{1}{2}$ to $3\frac{1}{2}$ centimeters. The arch of the aorta was dilated. The heart contained some fibrinous, dense, colorless clots, which had prolonged themselves like polypi into the aorta and pulmonary artery. A small amount of reddish and grumous blood flowed from the pulmonary veins. Blood, which was more fluid, flowed from the superior vena cava. The lungs were rose-colored and crepitant. There was a well-marked pulmonary congestion. The dura mater was much congested. The sub-arachnoid fluid was extremely abundant, and gave the brain the appearance of a gelatiniform mass; the reaction of the fluid was alkaline. The arachnoid presented some old milky spots. The vessels of the pia mater were much injected. The cerebral ventricles contained a small amount of serous fluid. The cerebral parenchyma was markedly injected, which injection was equally pronounced front and back. The chemical analysis performed by M. Hepp, chief pharmacist of the civil hospital, demonstrated the presence of morphine in the substance extracted from the stomach and duodenum.

At first sight it would seem reasonable to attribute the *post-mortem* effects of morphine to poisoning by nitric acid, were it not that the absence of injury to the tissue removes the doubt. Generally the mucous membrane of the alimentary canal is free from alteration. The surface of the body is completely discolored, and animal heat persists for a long time, even after the invasion of cadaveric rigidity.

§ 341. *Poisoning by morphine* or its salts requires no further consideration than has been given above, since opium owes its poisonous nature principally to this alkaloid, and the intensity of the effects of poisoning by any preparation of opium is directly pro-

portionate to the amount of morphine present in the opium. The symptoms are rather more prompt in their appearance, but are otherwise entirely similar to those produced by opium or laudanum. For an adult male, the medicinal dose of morphine, or its acetate, hydrochlorate, or sulphate, is *one-sixth* of a grain. Death has been caused by *one* grain of the hydrochlorate taken in divided doses over a period of six hours,[1] and by one and one-third grains within four hours.[2] The former dose was equal probably to six grains of opium. For an adult female the medicinal dose is one-eighth of a grain. For a child the proportionate dose should not be more than that fraction of the dose prescribed for adults, found by dividing the child's age, in years completed, by the number twenty-four; for instance, for a female child under one year, one ninety-sixth of a grain of morphine. It was for poisoning with the acetate of morphine, that Castaign, who had formerly been a pupil of Orfila, was, in the year 1823, tried and executed in Paris. He was convicted less upon the medical than the circumstantial evidence offered, since, with the most ingenious refinement of cruelty, he had administered tartar emetic to his victim for the purpose of getting rid of any of the poison which might have remained in the stomach. The medical testimony could only show that the symptoms and *post-mortem* appearances were not opposed to the supposition that morphine was the cause of death.

§ 342. *Compound poisoning with opium or morphine.*—It would be impossible to describe in this treatise the symptoms of compound poisoning in such a manner as to prevent a confused idea. A medical expert should be competent from a knowledge of the symptoms presented in any given case, to explain the modifications of opium poisoning by the control of another poison (see § 34), from a knowledge acquired of the symptoms caused when either poison was administered alone; but the predominance of these symptoms will depend upon the more active of the two or three drugs used together, or the interval of time elapsing between the period at which each was administered, or the relative size of the dose taken. After the general remarks upon this subject in our opening chapter further comment is unnecessary.

[1] Ed. Month. Journ., Sept. 1846. [2] *Vide* § 334.

§ 343 *Chemical examination.*—The recognition of opium depends upon the isolation and detection of one or more of its constituents, the two of especial importance being meconic acid and morphine, since they exist in opium to a larger extent than any of the other constituents; it is probable, however, that the detection of narcotine, which is present in opium to the extent of six or eight per cent., will in the near future constitute an important factor in the chemistry of opium poisoning. The narcotine is, however, mostly removed from some of the pharmaceutical preparations of opium, such as the "denarcotized opium" (U. S. Ph., 1880), and the "deodorized tincture of opium" (U. S. Ph., 1880). If in a chemico-legal analysis we find both meconic acid and morphine in the contents of the stomach or vomitus, it shows that the form of the poison taken was opium itself or one of its preparations, whereas, if morphine alone is present without meconic acid, the form of poison used must have been morphine or one of its salts. If a notable amount of narcotine were detected, it would prove that the form of poison used was one of the preparations of opium from which the narcotine had not been removed, such as opium itself and laudanum.

§ 344. *Morphine.*—Pure morphine crystallizes in the form of prisms belonging to the rhombic system. It is soluble in about one thousand parts of cold water, and in five hundred parts of boiling water; also in about one hundred parts of cold alcohol and in thirty-six parts of boiling alcohol; it is almost insoluble in ether, and but slightly soluble in chloroform; it is very soluble in amyl alcohol; also very soluble in dilute acids, acid salts of morphine, which are readily soluble in both water and alcohol, being thus formed.

The following are some of the chief characteristics of morphine:—

1. *Nitric acid* reddens morphine or its salts (the chlorate excepted, according to Dumas), and forms with them an orange-red solution, which is much darkened by an excess of ammonia, and which becomes yellow after a little time. *Fallacies.*—Nitric acid produces a red color with several other bodies, as brucine, commercial strychnine, several volatile oils (as oil of pimento and oil of cloves), some resinous substances, etc.

2. *Iodic acid* is deoxidized by morphine, iodine being set free. Hence, when this alkaloid is added to a solution of iodic acid, the

liquid becomes reddish-brown, and forms a blue compound with starch (*iodide of starch*). *Fallacies.*—Sulphuretted hydrogen, sulphurous acid, phosphorous acid, sulphocyanide of potassium, sulphosinapisin, and all other reducing agents have a similar effect on iodic acid. Of course if the morphine be pure, these fallacies have no application.

3. Neutral sesquichloride of iron, dropped on crystals of morphine, renders them blue, or added to an aqueous solution of morphine, gives a blue color, unless an excess of the iron compound be added, in which case a green color is produced; therefore, the precaution should be taken to add at first only the smallest possible amount of the iron solution. It is usually stated that this green color is due to an excess of acid in the iron solution, but the ordinary laboratory reagent, which always contains a slight excess of acid, will give the blue color if not added in excess, as the writer has repeatedly observed. This is an exceedingly delicate test when the morphine is pure, but the presence of strychnine, or other organic matter of various kinds diminishes its delicacy; also the solution of morphine should not be more dilute than 1 : 5000. This test is also valuable, because the mixture of iron and morphine solutions does not prevent the reaction with nitric acid (see 1). *Fallacies.*—Tannic and gallic acids with a little water, and infusion of cloves or pimento also form blue compounds with sesquichloride of iron.

4. Fröhde's reagent, which is concentrated sulphuric acid containing in solution molybdate of sodium or ammonium in the proportion of one milligramme of molybdate in one cubic centimeter of sulphuric acid, forms one of the most delicate tests for morphine which we have. A drop of this reagent brought in contact with morphine or one of its salts dissolves it, producing immediately a beautiful violet colored solution, which changes soon to a green, then to a brown-green, later to a yellow, and after twenty-four hours to a bluish-violet again. This is an exceedingly delicate test, reacting with morphine when only five one-hundreths of a milligramme of the latter is tested (Dragendorff). This same reagent gives characteristic color reactions with many of the other alkaloids.

5. Husemann's test[1] is also an exceedingly delicate one for mor-

[1] Fresenius's Zeitschrift, 1876, p. 103.

phine. It is performed as follows: Allow a crystal of morphine to dissolve in a drop of concentrated sulphuric acid and remain for twelve or fifteen hours at the ordinary temperature, or for one-half an hour at the temperature of boiling water, or for an instant at 150° C. (302° F.), and then introduce a small particle of some oxidizing agent, such as nitric acid, nitre, chlorate of potassium, chlorine water, sodium hypochlorite, or sesquichloride of iron, when a beautiful blue or reddish-violet color is produced, which changes quickly to a blood-red and gradually becomes colorless. This test is delicate for one one-hundreth of a milligramme of morphine. Other organic substances do not usually interfere with it, if one of the chlorine compounds is used as the oxidizing agent.

6. Morphine is precipitated from solutions of its salts by phosphomolybdic acid. This reagent being very sensitive, the process of preparing it is here given: A yellow precipitate from molybdate of ammonium, to which nitric acid has been added, is obtained by phosphate of sodium, and well washed; this precipitate is then suspended in water and heated with carbonate of sodium till perfectly dissolved; then the solution is evaporated to dryness and calcined to drive off the ammonia. If after the calcination only a partial reduction has been accomplished, the mass is again calcined, after having been sprinkled with nitric acid. Afterwards heat the calcined mass in distilled water, add enough nitric acid to make the solution strongly acid, and a sufficient quantity of water to make ten parts of the liquid to one of the saline substance. In this way a golden-yellow liquid is obtained, which should be kept away from ammoniacal vapors. This reagent with two-tenths of a cubic centimeter of a solution of the sulphate of morphine, of the strength of one part in five thousand of water gives gradually a turbidity. It also precipitates many of the other alkaloids from solutions of their salts.

7. If morphine be mixed with a little cane sugar, and this mixture treated with concentrated sulphuric acid, a wine-red color is produced (Weppen). This test will show the color with one one-hundredth of a milligramme.

8. Among other tests for morphine the following may be mentioned as the more important: (1) Morphine is precipitated from solutions of its salts, when not too dilute, by ammonia water, and the crystals of morphine thus obtained may be examined with the

microscope. (2) A crystalline precipitate is also produced by Mayer's reagent, which contains in one liter of water, 13.546 grammes of corrosive sublimate, and 49.8 grammes of iodide of potassium. (3) A solution of iodide of potassium produces in a solution of a salt of morphine a precipitate consisting of groups of white silky needles. (4) A brown precipitate is produced by a solution of iodine in iodide of potassium similar to those given by this reagent with many of the other alkaloids. (5) A solution of bichromate of potassium gives with solutions of morphine an amorphous precipitate, which gradually becomes crystalline. All of these precipitates may be formed in a drop of the solution of morphine in a watch glass or upon a glass slide by the addition of a drop of the reagent, and the precipitate can then be easily examined with the microscope.

The principal salts of morphine (the sulphate, acetate, and the hydrochlorate) are crystalline, soluble in water and alcohol, insoluble in ether, and possess a disagreeable bitter taste. One hundred parts of the last correspond to eighty parts of morphine (crystalline). The other derivatives of opium are meconic acid, codeine, narcotine, narceine, thebaine (para-morphine), papaverine. Thebaine is supposed by M. Cl. Bernard to possess the strongest convulsive action of all the alkaloids of opium and to be the most poisonous. The most notable poisoning by the acetate of morphine is that by Castaign, in June, 1823. He was, however, convicted by circumstantial evidence.

§ 345. *Meconic acid.*—The characteristics of this acid are as follows: 1. It reddens the neutral sesqui-salts of iron; the red color is destroyed by alkalies, protochloride of tin, and nitric acid, assisted by heat. This, which is the most reliable test for meconic acid, is still open to objection. Thus sulphocyanic acid produces a similar red color with the persalts of iron. The force of this objection is derived from the fact that sulphocyanic acid is naturally sometimes present in the saliva. Christison, indeed, states that "it is seldom possible to procure a distinct blood-red coloration from the saliva, except by evaporating a large quantity to dryness, and redissolving the residue in a small quantity of water;" but Pereira dissents from this statement, and says, that in a large majority of cases, he has found saliva distinctly and unequivocally reddened by the persalts of iron. He says, moreover, that he has several times obtained from the stomach of sub-

jects in the dissecting-room, a liquor which reddens the salts of iron. We believe that the opinion of chemists now is, in general, in accordance with the statements of the last-named author. The means of distinguishing the sulphocyanide from the meconate is to be found in the action of chloride of gold, or of corrosive sublimate, since, if a few drops of a solution of either of these reagents be added to the red liquid, the color, if due to sulphocyanic acid, will be immediately destroyed. Further, the liquid may be diluted and a few drops of a solution of the acetate of lead added; a precipitate falls, which is either meconate or sulphocyanide of lead. The former is insoluble, while the latter is quite soluble in acetic acid.

Meconic acid is also precipitated from its solutions by chloride of calcium or chloride of barium in the form of crystals of meconate of calcium or barium. The acetate of lead is the best reagent to use in separating meconic acid from organic mixtures, since the precipitated meconate of lead can be collected upon a filter paper, suspended in water and decomposed by sulphuretted hydrogen, meconic acid remaining in solution in the water and the black sulphide of lead being precipitated; this latter can be separated by filtration and the excess of sulphuretted hydrogen expelled from the filtrate by warming, when we have left a tolerably pure solution of meconic acid, which can be tested as above mentioned.

§ 346. *Narcotine.*—This alkaloid usually crystallizes in the form of rhombic prisms. The crystals are soluble in 25,000 parts of cold and in 7000 parts of boiling water, but when amorphous, as when separated from its salts by ammonia water, it is soluble in 1500 parts of cold and in 600 parts of boiling water. It is soluble in 120 parts of cold and in 20 to 24 parts of boiling alcohol, in 126 parts of cold and in 48 parts of boiling ether; it is very soluble in chloroform (2.69 parts), in 60 parts of acetic ether, and in about 22 parts of benzol.[1] It is quite soluble in dilute acids, except acetic, and is easily precipitated from these solutions by ammonia water in crystalline form. The best test for narcotine is that recommended by Husemann; this consists in treating a little narcotine (one-half of a milligramme is sufficient) with 0.2 of a cubic centimeter of concentrated sulphuric acid, or dissolve it in dilute sulphuric acid (1 : 5) and evaporate this solution slowly, when a

[1] Dragendorff, loc. cit.

red residue will be left, which, when touched with a trace of nitric acid, becomes violet; if the sulphuric acid residue is heated to 200° C. (392° F.), it becomes deep violet red in color, and the fact that this color is not produced below 100° C. (212° F.), enables us to distinguish narcotine from curarine, which latter gives the same color with sulphuric acid at 100° C. Fröhde's reagent (see above) gives a green color with narcotine.

§ 347. *Separation of opium or its constituents from organic mixtures.*—Various processes have been recommended for this purpose. The principal ones are those recommended by Stas and by Dragendorff. We give them both in some detail, since they are applicable to the isolation of very many of the vegetable and organic poisons as well as that of opium and morphine.

The method of Professor Stas, of Brussels, justly distinguished for his admirable reports in the Bocarmé case, is detailed by him as follows: The method I now propose for detecting the alkaloids in suspected matters, is nearly the same as that employed for extracting those bodies from the vegetables which contain them. The only difference consists in the manner of setting them free, and of presenting them to the action of solvents. We know that the alkaloids form acid salts, which are equally soluble in water and alcohol; we know also that a solution of these acid salts can be decomposed, so that the base set at liberty remains either momentarily or permanently in solution in the liquid. I have observed that all the solid and fixed alkaloids above enumerated, when maintained in a free state and in solution, in a liquid, can be taken up by ether when this solvent is in sufficient quantity. Thus, to extract an alkaloid from a suspected substance, the only problem to resolve consists in separating, by the aid of simple means, the foreign matters, and then to find a base, which, in rendering the alkaloid free, retains it in solution, in order that the ether may extract it from the liquid. Successive treatment by water and alcohol, of different degrees of concentration, suffices for separating the foreign matters, and obtaining in a small bulk a solution in which the alkaloid can be found. The bicarbonates of potash or soda, or these alkalies in a caustic state, are convenient bases for setting the alkaloids at liberty, at the same time keeping them wholly in solution, especially if the alkaloids have been combined with an excess of tartaric or of oxalic acid." To put in practice

the principles thus explained, the following method is proposed. "I suppose that we wish to look for an alkaloid in the contents of the stomach or intestines; we commence by adding to these matters twice their weight of pure and very strong alcohol; we add afterwards, according to the quantity and nature of the suspected matter, from thirty to forty-five grains of tartaric or oxalic acid—in preference, tartaric; we introduce the mixture into a flask, and heat it to 160° or 170° Fahrenheit. After it has completely cooled it is to be filtered, the insoluble residue washed with strong alcohol, and strained, and the filtered liquid evaporated *in vacuo*, or it may be exposed to a strong current of air at a temperature of not more than 90° Fahrenheit. If, after the volatilization of the alcohol, the residue contains fatty or other insoluble matters, the liquid is to be filtered a second time, and then the filtrate and washings of the filter evaporated in the air-pump till nearly dry. If we have no air-pump, it is to be placed under a bell-jar, over a vessel containing concentrated sulphuric acid or quicklime. We are then to treat the residue with cold anhydrous alcohol, taking care to exhaust the substance thoroughly; we evaporate the alcohol at a low temperature, or better still *in vacuo*, spontaneously. We now dissolve the acid residue in the smallest possible quantity of water, introduce the solution into a small test-tube, and add little by little pure powdered bicarbonate of soda or potash, till a fresh quantity produces no further effervescence of carbonic acid. We then agitate the whole with four or five times its bulk of pure ether, and leave it to settle. When the ether swimming on the top is perfectly clear, then decant carefully about two cubic centimeters of it into a small glass capsule, and leave it in a *very dry place* to spontaneous evaporation." If the suspected alkaloid is solid and fixed, there may or may not be a residue containing it. If there is, a solution of caustic potash or soda should be added to the liquid, and agitated briskly with ether. "This dissolves the vegetable alkaloid, now free, and remaining in the solution of potash or soda. In either case, we exhaust the matter with ether. Whatever be the agent which has set the alkaloid free—whether it be the bicarbonate of soda or potash, or caustic soda or potash—it remains, by the evaporation of the ether on the side of the capsule, as a solid body, but more commonly a colorless milky liquid, holding solid matters in

suspension. The odor of the substance is animal, disagreeable, but not pungent. It turns litmus paper permanently blue."

In order now to obtain the solid alkaloid in a crystalline state, the foreign matters, with which it is generally associated, must be first removed. Prof. Stas, to accomplish this purpose, adds a few drops of water feebly acidulated with sulphuric acid, to the contents of the capsule, and thus forms an acid sulphate, which should be carefully decanted, evaporated *in vacuo* or over sulphuric acid, the residue treated with pure carbonate of potassium, and the alkaloid dissolved out by absolute alcohol. The evaporation of the alcohol gives the alkaloid in crystals. By this process, Prof. Stas has isolated all the important fixed alkaloids previously mixed with foreign matters.[1]

§ 348. The following is the detailed process of Dragendorff,[2] which the writer much prefers to any other method for the isolation of organic principles, poisonous or otherwise, from ordinary organic mixtures, such as animal tissues and fluids, articles of food, etc. The chemical principles upon which the process depends are the same as in Stas's process. The detailed scheme is given below, in order to avoid the necessity of repetition in speaking of the organic poisons to be considered hereafter.

I. Extract with water acidulated with sulphuric acid two or three times at 40°–50° C. for several hours. Strain and filter the united extracts.

II. Evaporate to beginning syrupy consistence, mix the residue with three or four times its volume of alcohol, macerate twenty-four hours at about 30° C., cool, and filter. Wash the solid upon the filter with 70 per cent. alcohol.

III. Evaporate off the alcohol, transfer the residue to a flask, cool, and, if necessary, dilute and filter. Shake this aqueous acid fluid with freshly rectified naphtha ("petroleum ether") at the ordinary temperature frequently and vigorously. After the two fluids have separated decant the naphtha and allow it to evaporate in several watch glasses. The shaking with naphtha should be repeated as long as anything is removed by it. The naphtha

[1] Am. Journ. of Pharmacy, Jan. 1853. [2] Ermittelung von Giften, 1876, page 141.

removes from the fluid certain impurities (coloring matters, etc.) in addition to the following substances:—

Naphtha Residue from the Acid Fluid.

1. Crystalline.	2. Amorphous.	3. Fluid, and with a strong odor.
a. Yellowish and volatilized with difficulty.	***a.*** Non-volatile.	**Ethereal Oils, Carbolic Acid, etc.**
(α) The crystals dissolve in concentrated sulphuric acid with a pale yellow and later brown and greenish-brown color.	(α) Concentrated sulphuric acid dissolves it immediately with a violet and later a greenish-blue color.	
Piperine.	**Constituent of the Black Hellebore.**	
(β) The solution in sulphuric acid remains yellow. Potassium cyanide and potassium hydrate color it blood-red on warming.	(β) Concentrated sulphuric acid dissolves it with a yellow color which gradually changes to a violet-red and then to a roe-brown.	
Picric Acid.	**Constituent of the Aconite and a decomposition product of Aconitine.**	
b. Colorless, easily volatile and with a strong odor.	***b.*** Pale, with a sharp taste and reddens the skin.	
Camphor and similar substances.	**Capsicin.**	

IV. The aqueous fluid is next shaken in the same way with benzol. The benzol is decanted into several watch glasses and allowed to evaporate.

Benzol Residue from the Acid Fluid.

1. Crystalline.	2. Amorphous.
a. Well formed colorless crystals.	***a.*** Colorless or pale yellow residue.
(α) Sulphuric acid dissolves the hair-like crystals colorless. Evaporation with chlorine water leaves a residue which gives the murexide test with ammonia.	(α) Sulphuric acid gives at first a yellow solution, which becomes red later. Fröhde's reagent does not give a violet color.
Caffeine.	**Elaterin.**
(β) Sulphuric acid dissolves the rhombic crystals colorless. Mixed with oil and applied to the skin the substance blisters.	(β) Sulphuric acid gives a red solution. Fröhde's reagent a violet red. Tannin gives no precipitate.
Cantharidin.	**Populin.**

(γ) Sulphuric acid dissolves the scaly crystals at first colorless, slowly changing to a red. It does not blister. Warm alcoholic potassium hydrate gives a temporary red color.

Santonin.

(δ) Sulphuric acid dissolves the racemose crystals with a yellowish-orange color, and this solution gives a transient violet color with nitric acid.

Caryophyllin.

(ε) Sulphuric acid colors the crystals nearly black, and becomes itself colored a beautiful red.

Cubebin.

b. Crystals pale or clear yellow.

(α) **Piperin** (III. 1 ***a***, α)

(β) **Picric Acid** (III. 1 ***a***, β).

(γ) Potassium hydrate = purple color.

Aloëtin.

c. Usually only indistinct colorless crystals.

(α) Sulphuric acid=greenish-brown. Bromine colors this solution red, which becomes green again on diluting with water. The substance slows the action of a frog's heart.

Digitalin.

(γ) Sulphuric acid gives a deep red color. Fröhde's reagent = beautiful cherry red. Tannin=yellowish-white precipitate.

Colocynthin.

(δ) Sulphuric acid = a beautiful red, often gradual. Tannin=no precipitate.

Constituents of the Pimento.

b. Clear yellow residue.

(α) Sulphuric acid = yellow. This solution becomes green, then quickly blue and violet upon the addition of nitric acid.

Colchicine.

(β) Sulphuric acid dissolves it with the separation of a violet powder. Potassium hydrate = red. Sulphide of ammonium = violet, and, on heating, indigo blue.

Chrysammic Acid.

c. Greenish bitter residue. Sulphuric acid = brown. Fröhde's reagent = at first brown, then green, blue-violet, and finally violet, the change of color beginning at the edge.

Constituents of the Wormwood, Absinthin.

V. The aqueous acid fluid is now shaken with chloroform, which is removed with a pipette and allowed to evaporate in watch glasses.

Chloroform Residue from the Acid Solution.

1. More or Less Distinctly Crystalline.

a. The solution of the sulphate reacts like an alkaloid with a solution of iodine in iodide of potassium.

2. Amorphous.

a. The solution in acetic acid produces slowing of a frog's heart, or local anæsthesia.

(α) Sulphuric acid = colorless solution. Chlorine and ammonia water do not give murexide reaction.

Cinchonine.

(ζ) Sulphuric acid = colorless solution. Chlorine and ammonia water give the murexide test like caffeine.

Theobromine.

(γ) Sulphuric acid = colorless solution in the cold, but on warming=blue violet.

Papaverine.

(δ) Sulphuric acid = blue in the cold.

Unknown Impurities in Commercial Papaverine.

(ε) Sulphuric acid = at first gray-brown, and in about twenty-four hours blood-red. Iodine water = blue.

Narceine.

b. It does not react like an alkaloid.

(α) Sulphuric acid = beautiful yellow. Mixed with nitre, then moistened with sulphuric acid, and finally treated with concentrated sodium hydrate it assumes a brick-red color.

Picrotoxin.

(β) Sulphuric acid = beautiful red. It produces slowing of a frog's heart.

Helleborein.

aa. Does not produce local anæsthesia.

(α) Sulphuric acid = reddish-brown. Bromine colors this solution beautiful purple, which changes again to green on diluting. Hydrochloric acid=greenish-brown.

Digitalein.

bb. Produces local anæsthesia.

(α) Sulphuric acid = brown. This solution becomes violet with the absorption of water.

Saponin.

cc. Sulphuric acid = dirty red. Hydrochloric acid = red-brown in the cold, but on boiling brown.

Constituents of the Hellebore, principally Jervine.

VI. The acid fluid is again shaken with naphtha in order to remove the last traces of chloroform. The naphtha is removed and the fluid is made alkaline with ammonic hydrate.

VII. Shake the ammoniacal fluid with naphtha at the ordinary temperature.

After the separation of the two fluids decant a part of the naphtha into two watch glasses, one of which has been moistened with concentrated hydrochloric acid.

Naphtha Residue from Ammoniacal Fluid.

1. Crystalline.

a. Crystals volatilize with difficulty.

aa. Sulphuric acid = colorless.

(*α*) Potassium bichromate = blue then red in sulphuric acid solution.

Strychnine.

(*β*) Potassium bichromate does not give blue color. Chlorine water and ammonia water give dalleiochin reaction.

Quinine.

b. Sulphuric acid = yellow, gradually changing to a beautiful deep-red.

Sabadilline.

c. The crystals are easily volatile.

Conhydrine.

2. Amorphous.

(*α*) Purest sulphuric acid = almost colorless solution. Sulphuric acid which contains nitric acid = red color which changes to orange.

Brucine.

(*β*) Sulphuric acid = yellow color which changes to a deep-red.

Veratrine.

(*γ*) Sulphuric acid = brown-green. Fröhde's reagent = red color which soon becomes green.

Emetine.

3. Volatile and with a Strong Odor.

a. Crystalline residue on watch glass moistened with hydrochloric acid.

aa. Bichloride of platinum = no precipitate in its solution.

(*α*) The crystals of the hydrochloric acid compound act upon polarized light, and are mostly needle-shaped and prismatic.

Coniine and Methylconiine.

(*β*) Crystals are cubes or tetrahedra.

Alkaloid of the Capsicum.

b. The hydrochloric acid compound is amorphous, or only crystalline after decomposition.

aa. Bichloride of platinum = precipitate in its dilute aqueous solution.

(*α*) The hydrochloric acid salt treated as quickly as possible with Fröhde's reagent gives, in about 2 minutes, a deep violet solution which gradually fades.

Lobeline.

(*β*) The hydrochloric acid salt smells like nicotine. Fröhde's reagent = yellowish color, which becomes pale-red after 24 hours.

Nicotine.

bb. Bichloride of platinum = no precipitate in its dilute solution.

(*a*) Its naphtha solution gives no turbidity with a solution of picric acid in naphtha, but the mixture leaves a crystalline residue after evaporation (mostly triangular plates).

Trimethylamine.

(*β*) Its naphtha solution treated in the same way gives moss-like crystals. It is colored blue by a solution of calcic hypochlorite, and also by dilute sulphuric acid and potassium bichromate.

Aniline.

VIII. Shake the ammoniacal fluid with benzol.

BENZOL RESIDUE FROM THE AMMONIACAL FLUID.

1. MOSTLY CRYSTALLINE.

a. Sulphuric acid=colorless solution which does not become colored either by standing or by the addition of nitric acid.

aa. Dilates the pupil of a cat's eye.

(*a*) Bichloride of platinum = no precipitate in aqueous solution. The sulphuric acid solution has a peculiar odor on warming.

Atropine.

(*β*) Bichloride of platinum = precipitate when used in exactly the right amount.

Hyoscyamine.

bb. Does not dilate the pupil.

(*a*) Sulphuric acid solution colored blue by potassium bichromate.

(*aa*) Tetanizes a frog.

Strychnine.

(*ββ*) Slows the respiration in a frog.

Ethel- and Methylstrychnine.

(*β*) Sulphuric acid and potassium bichromate do not give blue color.

2. MOSTLY AMORPHOUS.

a. Purest sulphuric acid = colorless or pale-red or yellow.

(*a*) The sulphuric acid solution is immediately colored red by nitric acid, then orange.

Brucine.

(*β*) The solution gradually becomes brownish-red. Calcic hypochlorite colors it red. It contracts the pupil.

Physostigmine.

(αα) The dilute sulphuric acid solution fluoresces and gives the dalleiochin reaction.

Quinine.

(ββ) The solution does not fluoresce.

Cinchonine.

b. Sulphuric acid = at first colorless solution, which becomes on standing rose-red or bluish-violet, and blood-red or brownish on the addition of nitric acid.

(α) A solution in dilute sulphuric acid on heating gradually becomes deep blood-red, violet when cooled and treated with nitric acid. The dilute sulphuric acid solution gives a precipitate with ammonia water.

Narcotine.

(β) The dilute sulphuric acid solution usually becomes blue on heating. An excess of ammonia water=no precipitate in dilute solutions.

Codeine.

c. Sulphuric acid = yellow.

(α) The solution remains yellow on standing.

Acolyctine.

(β) It becomes beautiful red on standing.

Sabadilline.

d. Sulphuric acid = deep reddish-brown immediately.

Thebaine.

b. Pure sulphuric acid = yellow solution, which later becomes red. (In the case of delphinin more quickly and more of a dark cherry-red.)

(α) The hydrochloric acid solution becomes red on heating.

(αα) It causes retching in frogs, and in large doses tetanus.

Veratrine.

(ββ) Without action on frogs.

Sabatrine.

(β) The hydrochloric acid solution does not become red.

Delphinine.

c. Pure sulphuric acid=yellow, then reddish-brown, and gradually violet-red.

(α) In small doses paralyzes frogs. Dilates pupil of cat's eye. Difficultly soluble in ether.

Nepaline.

(β) Much more feeble physiol. action. It usually does not dilate the pupil. Very soluble in ether.

Aconitine.

(γ) Very feeble physiol. action. Does not dilate pupil. Difficultly soluble in ether.

Napelline.

d. Sulphuric acid = dark grayish-brown, changing in a few seconds to blood-red.

Alkaloid in the Aconitum Lycoctonum.

e. Sulphuric acid = brown-green. Fröhde's reagent=red changing quickly to a green.

Emetine.

IX. Shake the ammoniacal solution with chloroform.

CHLOROFORM RESIDUE FROM AMMONIACAL FLUID.

a. Sulphuric acid dissolves it colorless in the cold.

aa. It becomes slightly colored on warming.

(*a*) After cooling, nitric acid = blue-violet. Sesquichloride of iron = blue. Fröhde's reagent = violet. **Morphine.**

(*β*) Not colored by nitric acid or sesquichloride of iron. **Cinchonine.**

bb. The solution becomes blue-violet on warming. **Papaverine.**

b. Sulphuric acid = grayish-brown, and on standing blood-red. **Narceine.**

c. Sulphuric acid = blue-violet. **Alkaloid of the Celandine.**

X. Shake the ammoniacal fluid with amyl alcohol.

AMYL ALCOHOL RESIDUE FROM AMMONIACAL FLUID.

a. Sulphuric acid = colorless solution in the cold. **Morphine.**

b. Sulphuric acid = bright yellowish-red color, which becomes brownish. Iodine water = deep brown color. The alcoholic solution gelatinizes. **Solanine.**

XI. Evaporate the aqueous fluid with powdered glass, and extract the powdered residue with chloroform.

The residue from the first chloroform extract slows the respiration in a frog, that of the second and third gives a blue color, which changes to a permanent red when treated with sulphuric acid and potassium bichromate. Another part of this residue becomes red on heating with dilute sulphuric acid. **Curarine.**

The principles, as thus obtained by the above process, must then be subjected to the various tests mentioned under the head of each individual poison.

II. *Alcohol.*

§ 349. The pernicious effects upon the system, of the abuse of alcoholic liquors, are too well known to need any mention here. We propose, therefore, to refer only to their immediate poisonous action when taken in large quantity into the stomach.

Death, from this rapid saturation of the system with alcohol, is by no means rare. Orfila mentions an instance in which a man died immediately from the effects of a large dose of brandy.[1] Dr. Rösch relates three cases in which adults died from the immediate effects of excessive drinking in a few hours.[2] Taylor says that a

[1] Op. cit., ii. 528.

[2] Henke's Zeitschrift, 1850, 4 H.

man died in half an hour after swallowing a bottle of gin for a wager. Rösch also relates the cases of two children in which quite a small quantity proved fatal. The one was a boy aged two years, who drank some brandy and soon after became comatose, had convulsions, and died in a few hours. The other was a little girl of four years, to whom her uncle had given about two tablespoonfuls of spirits. The child soon sank down insensible, was seized with convulsions, and, in spite of all remedies that were used, died within twenty-four hours. In another case, the same quantity of brandy was given to a child six months old to keep it quiet during the night. In less than a minute it was attacked with convulsions; its face was purple, the eyes staring, the pupils dilated and insensible, the mouth open, the head extremely hot, while the rest of the body was cool, the breathing stertorous, and the pulse hardly perceptible. It had also bloody evacuations. It died, in a state of coma, in nine hours.[1]

In general, the state of stupor is preceded by a short period of great excitement, but in some cases this preliminary stage is either very short or entirely absent. The difference probably depends upon the strength and quantity of the spirit and the age of the person.

§ 350. *Symptoms.*—The general characteristics of the comatose stage in the adult are the following: The face may be either pallid or flushed; the pupil at first contracted, and afterwards dilated and insensible to light; respiration slow and sometimes stertorous; the pulse quick and jerking, and the limbs cool and relaxed. In general, the appearance is very much the same as in poisoning by opium, or as in the apoplectic condition. In the absence of any knowledge of the mode of accession of the symptoms, the diagnosis of the case will often be incomplete. The odor of alcohol in the breath, is, of course, an uncertain sign, since ardent spirits may have been swallowed without being the cause of the symptoms. The ability to arouse the patient temporarily is also no means of distinction, as this may be possible in the stupor from intoxication, and opium poisoning.

In fact, should the individual die, the physician will often be left in doubt as to the cause of the symptoms until some evident explanation

[1] Deutsch. Canstatt's Jahresbericht für 1851, Bd. 10, p. 286.

for them is found in the *post-mortem* examination. Even then nothing may be found to throw light upon the case, since a person presenting the above symptoms may have died of concussion of the brain, which leaves no ascertainable morbid change. If the symptoms have been due to opium or other narcotics, these may not be discovered, and, as will presently be seen, the evidences of death from alcohol may also be deceptive. It will only be from a careful analysis of the history of the case, and comparison of it with the *post-mortem* signs, and by isolating a considerable amount of alcohol from the tissues and viscera, that the physician can hope to come to a probable conclusion.

§ 351. Mention should be made here of the foolish and criminal bets, that have caused sudden and frightful deaths from drinking a large quantity of some alcoholic beverage. This has been observed not only among those who have by long experience acquired a certain immunity from the frightful use of excessive doses of alcohol, but also among persons little used to alcoholic excesses, and who have allowed themselves to drink a quart of alcohol, hardly stopping a moment to take breath. These unfortunates fall, as if struck down, into a profound coma, accompanied by little convulsive muscular shocks. Respiration, at first stertorous, becomes more and more difficult, and a bloody froth appears on their lips. Involuntary evacuations occur, and they die sometimes in an hour or half an hour, and sometimes as soon as fifteen minutes after the fatal drink.

§ 352. The following extract, in fact the whole paper on acute alcohol poisoning, was read before the Massachusetts Medico-Legal Society at its meeting in June, 1880,[1] and since it covers the ground so completely, from a medico-legal point of view, no apology is required for its introduction here.

I. ACUTE ALCOHOLIC POISONING.

"The pathological anatomist meets with two classes of cases of alcohol poisoning, the acute and the chronic. The former is not unfrequently the immediate cause of death, the latter rarely so. That fatal cases from acute poisoning are not so rare as might be supposed is shown by the fact mentioned by Taylor, that in four

[1] Medico-Legal Relations of Alcoholism, Its Pathological Aspects, by G. K. Sabine, M.D., Medical Examiner for Norfolk County, Mass.

years (1863–1867) thirty-five deaths from this source occurred in England and Wales alone.[1]

"In describing the *post-mortem* appearances it will be necessary to describe those belonging to the two classes separately. The statements of different observers vary greatly in regard to these appearances, and in many respects are entirely contradictory. This is due, no doubt, to the want of making a proper distinction between the acute and the chronic forms, many symptoms thus being considered as due to acute poisoning which are in reality met with in chronic cases only.

"Within a short time the effects of the different kinds of alcohol in poisonous doses upon dogs has been thoroughly investigated by Dujardin-Beaumetz and Audigé. These investigations were carried on by means of more than two hundred and fifty experiments upon as many different animals, and the general conclusions drawn from them were as follows:—

"'In animals which succumb to acute alcoholic poisoning anatomical lesions are constantly found, which vary in intensity with the strength of the alcohol. These lesions occur especially in (1) the digestive organs; (2) the circulatory and respiratory organs; (3) the nervous system; and (4) the kidneys.

"'(1) *Lesions of the digestive organs.*—In regard to the stomach the changes are less marked when the alcohol has been administered subcutaneously, and then only a slight redness towards the pyloric extremity is perceptible. But when the alcohol is administered by the œsophagus the lesion is more marked, and, in certain cases, the mucous membrane is more or less softened; the effects depend on the strength of the alcohol, being much more marked when the alcohol is concentrated, and therefore more caustic.

"'The alteration in the small intestine is more constant and more marked when the alcohol has been administered hypodermically; it exists more especially in the upper part of the intestine, but when death is protracted it occurs throughout its whole length. The intestinal mucous membrane is then softened, its surface of a dark-red color, and it presents, in the majority of cases, more or less abundant hemorrhages. These appearances may be accounted for by the elimination of the alcohol by the intestinal glands.

[1] H. C. Wood, Therapeutics, Materia Medica, and Toxicology.

"'In the large intestine a hemorrhagic appearance is met with, especially towards its inferior extremity and on the longitudinal bands.

"'The liver is the gland most seriously affected in acute alcoholic poisoning; this organ, always very quickly congested, is soft throughout and friable; it can be torn between the fingers, and the hepatic cells may be destroyed in a great measure. The spleen is also engorged with blood and its tissue softened. Finally, in a certain number of cases, the head of the pancreas is found to share in the congestion presented by the duodenum.

"'(2) *Lesions of the circulatory and respiratory organs.*—In acute alcoholic poisoning, the blood is essentially altered; it is blackish and forms more or less abundant clots in the heart. The pulmonary lesions are characterized by a distension of the vascular system. This congestion is frequently more marked when the alcohol has been introduced by the stomach, and in these cases there are ecchymoses at the base of the lungs.

"'(3) *Lesions of the nervous system.*—Lesions of the cerebro-spinal axis are characterized by considerable venous congestion of the meninges. In the brain, the veins and sinuses are engorged with blood and the gray matter is also somewhat congested. These cerebral lesions are more pronounced the longer the comatose period continues.

"'(4) *Lesions of the kidneys.*—These lesions are but slightly marked when caused by fermented alcohol, but when due to œnanthylic and caprylic alcohol are more pronounced. But it is above all in acute glycerine poisoning that they attain their greatest intensity; there exists then not only a congestion of the organs, but a certain quantity of blood is found in the bladder.'

"Lallemand, Perrin, and Duroy (Alcohol et des Anesthésiques) state that 'marked anatomical changes are found in animals which have died from the administration of alcohol. The mucous membrane of the stomach and of the upper part of the small intestine is inflamed, sometimes quite violently. The liver is much congested. The lungs show no signs of asphyxia or even real congestion. The right side of the heart and the large veins are filled with very liquid, dark blood. The meninges are engorged with blood. The alcohol is diffused through all the tissues and secretions, but the brain,

cord, and liver contain a much larger proportion than any of the other tissues.'

"So much for poisoning in the lower animals.

II. ALCOHOL POISONING IN MAN.

"On inspection, the dependent portions of the body are found discolored by hypostatic congestion. It has been claimed by some that decomposition sets in very slowly, but this is denied by others.

"'The general appearances resemble more or less closely those of asphyxia; the right side of the heart, the pulmonary arteries, and the systemic veins being loaded with blood, while the left cavities and the arterial system are comparatively empty, the blood which they do contain being dark.[1] The sinuses and the whole venous system of the brain are turgid with dark blood.'[2]

"The blood is very fluid or imperfectly coagulated and of a dark color.[3]

"The mucous membrane of the stomach is usually found very much injected, as indicated by either a bright[4] or deep red[5] color, covered with coagulated mucus (albumen),[6] and sometimes ecchymosed.[7] The congestion usually extends both into the œsophagus and into the small intestine.[8]

"The action of strong alcoholic liquid on the mucous membrane of the stomach so closely resembles the effects produced by arsenic and other irritants as easily to give rise to the suspicion of mineral irritant poisoning.[9]

"The amount of alteration in the gastric mucous membrane will vary, of course, with the quantity of alcohol taken, its degree of concentration, and the amount of food, etc., in the stomach.

[1] Carpenter on Alcohol.

[2] Woodman and Tidy, Forensic Medicine and Toxicology; Carpenter on Alcohol.

[3] Casper, Forensic Medicine, vol. ii.

[4] Taylor on Poisons; Christison on Poisons; Birch-Hirschfeld, Lehrbuch der Pathologischen Anatomie.

[5] Wood, Therapeutics, Materia Medica, and Toxicology; John J. Reese, Manual of Toxicology; Oesterlen, Heilmittellehre; Taylor on Poisons; Woodman and Tidy, Forensic Medicine; Birch-Hirschfeld, Lehrbuch der Pathologischen Anatomie.

[6] Oesterlen, Heilmittellehre.

[7] Wood, Therapeutics, Materia Medica, and Toxicology; Oesterlen, Heilmittellehre; Birch-Hirschfeld, Lehrbuch der Pathologischen Anatomie.

[8] Woodman and Tidy, Forensic Medicine.

[9] Taylor on Poisons.

"A very few cases have been reported in which the mucous coat has been found in a sloughing condition. According to Baer frequently no lesions are perceptible. In such cases the fluid has been taken very much diluted.

"Alcohol is found to be present in the contents of the stomach in variable quantity or entirely absent[1] from the same, according to the quantity ingested, the rapidity of absorption, and the time which has elapsed between the last potations and the death of the individual.

"The length of time that portions of it may remain in the stomach is consequently very uncertain. Taylor mentions a case in which a pint of spirits was taken and a fatal effect produced in eight hours, but no traces of it could be detected in the stomach. That it may entirely disappear from the stomach long before it is eliminated from the other organs is proven by a number of cases of more or less chronic poisoning related by Magnan. In one of these three days and six hours had been passed without excess in drink; the liver and brain contained alcohol, but none was found in the blood or other organs. In another patient who had died fifty hours after his last potation, alcohol was found in the liver, brain, and blood in very appreciable quantity; the lung also contained traces.

"When present, as it probably almost always is,[2] at the time of death in cases of acute poisoning, the contents of the stomach will of course possess the odor of the liquor ingested, unless masked by some other substance present.

"The mucous membrane of the respiratory tract exhibits a widely spread and intense injection of the bloodvessels;[3] the bronchi are filled with frothy mucus;[4] more or less hyperæmia of the lungs may always be expected;[5] they are very often found in a state of œdema;[6] still more frequently hypostases and hepatization are discovered in their posterior and inferior portions.[6]

"The liver, kidneys, and spleen are usually found loaded with venous blood.[7]

[1] Oesterlen, Heilmittellehre.

[2] Christison on Poisons.

[3] Boehm, Ziemssen,

[4] Carpenter on Alcohol.

[5] John J. Reese, Manual of Toxicology; Casper's Forensic Medicine; Woodman and Tidy, Forensic Medicine and Toxicology; Wharton and Stillé's Medical Jurisprudence; Lallemand, Perrin, et Duroy, Alcohol et des Anesthésiques.

[6] Boehm, Ziemssen.

[7] Carpenter on Alcohol.

"The presence of alcohol may be detected in the liver even after it has entirely disappeared from the stomach and all the other organs with the exception of the brain, for it is these two organs which show the greatest affinity for it.[1]

"Nearly all authorities state that the veins of the cerebrum and cerebellum together with their membranes are engorged with blood.[2] According to Birch-Hirschfeld, however, the substance of the brain itself is frequently found anæmic and œdematous.

"According to some an effusion of serum[3] is frequently found within the ventricles or beneath the arachnoid, and according to many others an effusion of blood[4] is very apt to take place, especially in the latter locality. It is probably somewhat doubtful how frequently these effusions take place in the most acute cases. The statement that hemorrhagic apoplexy occurs quite constantly seems to be founded on a very limited number of cases.

"Through the kindness of Dr. Harris and Dr. Amory, of this society, I have been enabled to obtain the autopsy records of two cases of undoubted acute alcohol poisoning, which fell into their hands for examination, and Dr. Wood, of the Medical School, has kindly furnished me with an account of the result of the chemical analyses in the same cases. Dr. Draper has also published an account of the examination of a more or less chronic case in the Transactions of this society, volume i., No. 1.

"*Dr. Harris's case.*—Two women of bad character were induced in the evening by sailors to go on board a vessel lying at one of the wharves, where they drank a large quantity of Spanish rum, known as aguadiente. At two in the morning one of the women was found dead.

"*Autopsy* fourteen hours after death. "Rigor mortis marked.

[1] John J. Reese, Manual of Toxicology.

[2] Oesterlen, Heilmittellehrè; John J Reese, Manual of Toxicology; Wharton and Stillé's Medical Jurisprudence, Part I.; Casper's Forensic Medicine; Taylor, Treatise on Poisons; Birch-Hirschfeld, Lehrbuch der Pathologischen Anatomie; Lallemand, Perrin, et Duroy, Alcohol et des Anesthésiques.

[3] Oesterlen, Heilmittellehre; Carpenter on Alcohol; Taylor on Poisons; Manual of Toxicology, John J. Reese.

[4] Wharton and Stillé's Medical Jurisprudence; Tardieu, Étude sur l'Empoisonnement; Taylor on Poisons; Woodman and Tidy, Forensic Medicine and Toxicology; Christison, Treatise on Poisons.

Lividity of dependent portions of the body. An abrasion of the mucous membrane in the lower part of the vagina, one by one and a half inches was discovered. No marks of violence.

"*Section.*—The lungs were bound to chest walls, especially posteriorly, by not very firm adhesions. The heart presented no very unusual appearances except that the muscle was a little lighter colored than usual. The abdomen contained about one ounce of dark-colored fluid. The lungs were slightly congested, and the bronchi contained a little frothy mucus.

"The kidneys were firm, quite dark in color, with the pyramids very strongly marked. The internal surface of the stomach was intensely congested, the contents (about two ounces) smelling strongly of rum. The brain was of normal consistence, the vessels of the pia mater not overloaded; the puncta cruenta were, however, strongly marked. The uterus contained free, dark blood, and in the right ovary was a corpus luteum of menstruation. The intestines and bladder presented nothing unusual. The spleen presented appearances of old inflammation. The liver, brain, stomach and contents were reserved for chemical analysis, the kidneys, part of heart and uterus for microscopical examination.

"Dr. Wood isolated from the contents of the stomach one half cc. of alcohol, 0.825 specific gravity, which contains eighty-nine per cent. of absolute alcohol, and from one half of the brain two cc. of alcohol of the same strength. This corresponds to four cc. in the whole brain, and since eighty-nine per cent. is about double the strength of ordinary spirituous liquors, the amount found in the brain corresponds to about eight cc. of ordinary liquor.

"*Dr. Amory's case.*—A man about forty-two years old left home in the company of a friend. Two hours later, after visiting a number of places where liquor was sold, and drinking freely, he was brought home in a comatose condition, and died shortly after.

"*Autopsy* seven hours after death. Body well developed. Face livid. A superficial cut just beneath left orbital ridge, from which a small quantity of blood was slowly oozing. Thick layer of fat under the skin. The deceased wore a truss for double inguinal hernia. Left inguinal canal open, the right closed. Diaphragm on the right side opposite the fourth rib and opposite the fifth on the left. Pigmentary deposits in both lungs. Old pleuritic adhesions

on posterior surface of right lung, chiefly confined to the lower lobe. Pericardium contained about one half ounce of serum. Left ventricle contracted, right flaccid. Mitral valve admitted two fingers with difficulty, tricuspid three readily. No clots in heart or large vessels. The posterior portion of the lower lobe of right lung engorged with blood, but pressure caused crepitation. A small cyst about the size of a pea on the periphery of right kidney. The external surface of stomach considerably congested, the internal intensely reddened, showing the action of some irritant substance. The rugæ quite prominent. The contents consisted of fragments of meat, potatoes, and carrots, and about one and a half pints (by estimate) of fluid smelling of alcohol. No clot found in any part of body or bloodvessels. Vessels of pia mater moderately filled with blood.

"Dr. Wood isolated from the contents of the stomach 33⅓ cc. of eighty-nine per cent. alcohol, and about one half cc. of the same strength from one half of the brain.

"The medical examiner meets with another class of cases much more frequently than with the preceding. These are cases in which the subjects have been addicted for a long time, during life, to alcoholic excess and have died during a debauch, the immediate cause of death being either some exposure to cold, some chronic disease caused by the long-continued use of alcohol, or the continued dose of alcohol in a system already very much weakened by its excessive use.

"In these cases one is apt to meet with certain appearances which belong to cases of acute poisoning, if alcohol has been recently taken, such as redness of the gastric mucous membrane, fluidity of the blood, etc., and either few or many of the signs of chronic poisoning may also be present, but no one of them is constant or pathognomonic."

§ 353. *Chronic alcoholism.*—The symptoms of chronic alcoholism are more for the medical practitioner than for the toxicologist, but inasmuch as the *post-mortem* appearances may enshroud a doubtful case of suspected poisoning, it would seem advisable to present a careful *résumé* of the results of recent study, which is offered in the paper herewith published by permission of its author, G. K. Sabine, M.D., Medical Examiner for Norfolk County, Mass., upon—

THE MEDICO-LEGAL RELATIONS OF CHRONIC ALCOHOLISM, AND ITS PATHOLOGICAL ASPECTS.[1]

"Although the acute form of alcoholic poisoning is not unfrequently the immediate cause of death, and the chronic alone rarely so, the two are so often combined that the signs of the latter should be familiar to the medical examiner.

"Of all the various conditions that are attributed to the habitual use of alcohol but very few, if any, are pathognomonic. The statements of different observers vary greatly in regard to the post-mortem appearances of the chronic as well as of the acute form, and the question to what degree certain diseases may be attributed to this form of poisoning remains an open one. In fact, almost every known chronic affection has been attributed some time or other to the intemperate use of alcohol. It is probably the fact that the remote is frequently looked upon as the immediate cause. That the habitual and long-continued use of alcohol so alters the tissues and impairs their functions that they are more prone to become diseased there can be no doubt.

"Perhaps there is no question which the medical examiner is more frequently called upon to decide than whether or not he has to deal with a subject who has been an habitual drunkard.

"Although no single pathological condition is sufficient to determine this positively, yet there are certain ones which point strongly in this direction, and the same inference can be drawn from others taken collectively which are almost equally conclusive.

"Among the various pathological conditions resulting from chronic alcoholism are the following:—

"*Changes in the skin.*[2]—In the earlier stages of this affection the skin is remarkably smooth and soft, owing to an increase in the fatty tissue. According to Frerichs[3] the secretion contains a larger amount of oil than normal, a condition similar to that which exists when cod-liver oil is taken in large doses for some length of time. Later on the skin becomes dry, and on the extremities hard and inelastic.

[1] Transactions of the Mass. Medico-Legal Society, vol. i. No. 5, 1882.

[2] The following statements are largely taken from Baer..

[3] Klinik der Leberkrankheiten, l. c., S. 319.

" Acne rosacea, consisting of an inflammation and even suppuration of the sebaceous glands, is among the characteristic symptoms of the intemperate use of alcohol. The nose and face are its favorite seat. Besides the nodules the skin is reddened, owing to the dilated capillaries and consequent blood stasis, and is also infiltrated to a greater or less degree.

" *The blood.*—The most striking change in the blood is an increase in the watery elements and diminution in the fibrine. It contains much serum, forms no coagula, or only very small ones, and is of a very dark color; hence the term " venous plethora," used by some of the older authors. The dark color is explained by an increase in the hydrogen and carbon (Steinheim); or by a destruction of the red blood corpuscles (C. H. Schulz), so that less oxygen is taken up and less carbonic acid is given off. Another peculiarity presented by the blood is the increase of fat contained in it. It has been stated that not unfrequently the serum of blood drawn from a person suffering from delirium tremens is more or less opaque or even milky (Morgagni, Nasse), this appearance being due to an excessive amount of fat.

" *Fatty tissue.*.—There is a marked increase in the subcutaneous fat, in the fat between the muscles, about the different organs, especially the heart, kidneys, intestine, in the greater and lesser omenta, in the mesentery, etc. In the later stages of alcoholism, when the digestion becomes impaired and the blood deteriorated, this accumulation of fat disappears. According to Rokitansky there is an increase of fat in the marrow of the bones, the bony tissue at the same time being atrophied.

" In the earlier stages of the affection the increase of fat is due to an infiltration rather than to a degeneration. The fatty liver, for instance, is of essentially a different nature at this time from that met with later on in the disease.

" *The stomach and intestine.*—A chronic catarrhal condition of the stomach is quite constant, and appears early in the disease. This is indicated by abundant soft gray mucus, projections of the mucous membrane, and by the slaty color that occurs, especially near the pylorus. Another form is very apt to be met with, which is characterized by circumscribed hypertrophies of the whole mucous membrane (*gastritis prolifera*), and produces little warty projec-

tions (*gastritis verrucosa*) ; later, larger polypoid growths result (*gastritis polyposa*).

" Owing to the disturbance of circulation which takes place later in other organs, the return of blood from the stomach is interfered with, so that a varicose condition of some of the veins is produced.

" The hypertrophy is very apt to be accompanied by dilatation of the glands, due to compression at their outlet, so that small cysts, which are filled with a clear fluid and project from the surface, result. According to Klebs[2] an inflammation of the submucous tissue may be produced by the excessive use of alcohol, and this go on to suppuration, or it may result in the formation of large masses of connective tissue without destruction of the mucous membrane. In this manner it occurs at the pyloric extremity, producing stenosis.[3]

" The continued irritation of the diseased mucous membrane is productive of a variety of ulcerations, from the small hemorrhagic erosion, characterized by a superficial loss of substance, to the so-called round or perforating ulcer.[4]

" According to Erbstein.[5] after administering large quantities of dilute alcohol to dogs for three or four days, the peptic and ordinary gland cells of the stomach are found cloudy and granular. The lumen of the pyloric glands is plugged by a finely granular, yellowish or yellowish-brown mass. In extreme cases, fat drops appear in the altered cells. In most cases chronic alcoholism produces no marked effect on the intestine, although in many a chronic catarrh exists.

" *The liver.*—The liver is the first and the most severely affected by the abuse of alcohol of all organs in the body. The alcohol being taken up by the portal system is carried directly to this organ, and there, by its irritating effect, produces various disorders according to the individual's condition, and more especially the character of the alcohol. The more concentrated the alcohol, the sooner and the more severely is the liver affected.[6] Beer and wine seldom affect the liver, and are almost never productive of severe

[1] Orth, Diagnosis in Pathological Anatomy, page 285.

[2] Handbuch der Pathologischen Anatomie, S. 179.

[3] Birch-Hirschfeld, Lehrbuch der Pathologischen Anatomie, S. 831.

[4] Klebs, Handbuch der Pathologischen Anatomie, S. 186.

[5] Virchow's Archiv, Bd. 55, S. 469.

[6] Frerichs, A Clinical Treatise on Diseases of the Liver, vol. ii. p. 33. Sydenham Trans.

forms of degeneration.[1] The most frequent affections of the liver produced by chronic alcoholism are simple fatty infiltration,[2] inflammation of the parenchyma, and fatty degeneration of the same,[3] and lastly inflammation and hyperplasia of the interstitial connective tissue.

"Among the causes of fatty liver the abuse of alcohol is one of the most prominent. According to Frerichs it ranks only second, chronic disease of the lungs standing at the head of the list. He says: 'Of thirteen individuals who died of delirium tremens, in six the liver was very fatty, in three the organ contained little fat, and in two none at all; lastly, two died of cirrhosis of the liver.' It is probable, but not absolutely certain, that the alcohol acts by retarding the metamorphosis of tissue[4] and the blood being overcharged with fat, deposits it in this organ.[5]

"In higher grades the liver is enlarged, but usually appears flattened, the edges are generally thickened and rounded off. The peritoneal covering of the liver is transparent, smooth, and shining. According to the grade of fatty infiltration the surface of the liver is yellowish-red or distinctly yellow. The consistence of the organ is diminished; it feels doughy, and pits on pressure with the finger. On incision we meet little resistance; a coating of fat remains on the warm knife blade. The cells in the periphery of the acini first become infiltrated, and later on those nearer the centre.[6] The quantity of blood in the capillaries is diminished in proportion to the amount of infiltration.[7] On microscopic examination, according to the grade of the disease, the enlarged or usually rounded liver-cells appear filled with fine fat globules, or those have united to form single larger drops, or, lastly, individual liver-cells are entirely or mostly filled by one large drop of fat.[8]

"Fatty or granular degeneration of the liver is attributed to the

[1] Ibid.

[2] Frerichs, A Clinical Treatise on Diseases of the Liver, vol. i. p. 299. Klebs, Handbuch der Pathologischen Anatomie, S. 382. Orth, Diagnosis in Pathological Anatomy, p. 318. Niemeyer, Text-book of Practical Medicine, vol. i. p. 656.

[3] Klebs, Handbuch der Pathologischen Anatomie, S. 406.

[4] Niemeyer, Text-book of Practical Medicine.

[5] Baer, Alcohol.

[6] Niemeyer, Text-book of Practical Medicine.

[7] Baer, Alcohol.

[8] Niemeyer, Text-book of Practical Medicine.

abuse of alcohol by Baer, who quotes Klebs in so doing. The reason why the latter assigns this as a cause is perhaps owing to the fact, that he makes a somewhat different classification of the diseases of the liver, considering certain forms as degeneration which are looked upon by other authorities as simply infiltration. According to him the direct action of other toxic substances upon the liver, such as hydrocyanic acid, carbonic oxide, phosphorus, arsenic, and antimony, produce this degeneration. Among the organic substances alcohol, ether, and chloroform hold an important position.

"*Interstitial hepatitis. Cirrhosis of the liver.*—The most common cause of this form of interstitial hepatitis, which extends uniformly over the whole organ, is usually considered to be the intemperate use of alcohol, still this is not necessary; most drunkards do not have a cirrhotic, but a fatty liver, and many persons with cirrhosis are not in the habit of dram drinking.[1] Certainly cirrhosis is so commonly the result of the abuse of alcohol that when met with the cause may fairly be suspected.[2] Frerichs speaks of it as 'the chief cause.'

"Birch-Hirschfeld says that it is doubtful if cirrhosis is ever due to any other cause. The volume of the liver is increased or diminished according to the stage of the process. It is only accidentally met with in the early stages. The principal change produced in the liver consists in an increase of the interlobular tissue, and the appearance of small grayish masses at the periphery of the lobules. The consistency of the liver is increased. The cause of this change consists of a growth of granulation tissue from Glisson's capsule, from which small projections extend into the acini. In the later stages of chronic interstitial inflammation the liver is more or less diminished in size, in rare cases fully one half; its surface is uneven and covered with prominences, which vary from a millet grain in size to that of a pea, and are usually of a yellow, icteric color. At the edge of the liver, especially in front where it is sharp, single nodules are frequently found, completely isolated, as the capsule belonging to the two surfaces comes in contact here. Upon section a similar condition of things is seen in the interior of the organ."

"*Organs of respiration.*—Drunkards are very subject to

[1] Orth, Diagnosis in Pathological Anatomy. Frerichs, A Clinical Treatise on Diseases of the Liver. Niemeyer, Text-book of Practical Medicine.

[2] Baer, Alcohol.

catarrh of the larynx,[1] which is often accompanied by a similar condition of the pharynx.[2] This catarrhal inflammation of the larynx not unfrequently extends into the bronchi.

"A very important question is whether the habitual use of alcohol predisposes to disease of the lungs. Upon this point authorities differ so widely that it is quite impossible to draw any conclusion.

"*The heart.*—In habitual drunkards the heart is almost always found hypertrophied. This hypertrophy may be brought about in various ways. As is well known, the effect of alcohol is to increase the frequency and force of the pulse. Whenever a muscle is called upon to do an extra amount of work the effect is to increase the size of that muscle. This hypertrophy of the ventricular walls, which is simply the result of an increased amount of work, is also produced by various obstructions to the circulation which the heart has to overcome. Owing to the deposit of fat in and about the different organs, to the fatty infiltration of the cardiac muscle itself, the work of the heart is increased. This will also be caused by the disturbance of the pulmonary circulation, owing to bronchial catarrh, emphysema, etc., and also to disturbance of the portal circulation from fatty liver, cirrhosis, etc. Another very important factor in the cause of hypertrophy of the cardiac muscle, and especially of the left ventricle, is, according to some authorities, the condition of the kidneys frequently met with in chronic alcoholism. Here one may find fatty degeneration of the parenchyma, accompanied by an increase of interstitial connective tissue which has become more or less shrunken. According to Traube this contraction cuts off a large number of small vessels, and results in an increase of pressure in the aortic system, producing dilatation and hypertrophy of the left ventricle. This explanation is objected to by others, for instance Bamberger. Still another obstruction to the circulation is owing to the lumen of the vessels being increased, for when this occurs the blood-stream is rendered slower, and has to be overcome by increased heart's action. Finally, an atheromatous condition of the arteries is to be mentioned, as this causes a decided obstruction.

"In the later stages of alcoholism a fatty degeneration of the cardiac muscle occurs, and in the very last stages, owing to the

[1] Baer, Alcohol. Niemeyer, Text-book of Practical Medicine.

[2] Niemeyer, Text-book of Practical Medicine.

general inanition at that time, the muscle becomes atrophied and diminished in weight. The organ is pale and flabby, diminished in size in all directions.

"*The vessels.*—The change in the capillaries consists in an increase in their lumen, that of the smaller and larger arteries in the so-called atheromatous degeneration. The dilatation of the small vessels and passive hyperæmia of all the organs has been explained on the ground that the alcohol has a paralyzing effect upon the vaso-motor system; also that the alcohol, by its irritating effect upon the walls of the vessels, causes a fatty degeneration of the same, and as a consequence a loss of tonicity.

"Aside from the fatty condition a sclerosis of the walls takes place, owing to hyperplasia; this may result in the so-called ossification, an infiltration of lime salts into the newly formed tissue. As a result of these conditions the vessels lose their elasticity, become hard and stiff, and thus are more resistant to the flow of blood. This chronic inflammation of the walls of the vessels which underlies this process may be brought about by the continued use of alcohol. The irritation which alcohol produces in all the tissues may be sufficient to produce inflammation of the walls of the vessels. A still more important element in the causation is the constant stretching which the walls undergo and which predisposes them to the atheromatous change. Traube attributes this degeneration in alcoholism to this, and also to the diminishing of the rapidity of the blood-stream, which occurs at the same time. He says: 'The worst of these cases occur in drunkards. It is not unlikely that the increased tension of the aortic system which is observed in drunkards, not only while they are under the immediate influence of liquor, is owing to a contraction of the smaller arteries, which results in an increased tension by interfering with the flow of blood from the aortic system. This being the case, the increased tension observed in the larger branches would be accompanied by a slowing of the blood-stream.' The primary inflammatory condition may also be brought about by the overdistension of the smaller vessels, caused by the increased heart's action. When they have once lost their tonicity this increased action ceases. It is a well-established fact that as the quality of the blood becomes deteriorated, a condition which is a constant accompaniment of the general cachexia, the nutrition of the walls of the vessels is interfered with, and a fatty degeneration results.

"*Affections of the urinary organs.*—After each ingestion of alcohol, the secretion of urine is increased, as a larger quantity of water is excreted with it.

"The diseases of the kidneys which most frequently occur in drunkards, and especially in the latter stages of alcoholism, are the parenchymatous and interstitial or granular nephritis. This latter is divided into two stages, that of infiltration of cellular elements, and the other of connective-tissue formation. At first the inflammatory process produces an active hyperæmia, with an exudation of fluid and white blood-corpuscles into the interstitial connective tissue. This in turn is productive of anæmia, impaired nutrition of the renal epithelium, and granular degeneration of the same.

"If this process advances to another stage, there occurs either a hyperplasia of the interstitial connective tissue, or, what is more frequent, a granular condition with atrophy. The cellular elements lying between the urinary tubules become converted into masses of connective tissue, which serve to obstruct the glomeruli and tubules. The increased blood pressure in the aorta induces hypertrophy of the left ventricle, and albumen appears in large quantity in the urine, which is increased in quantity, and of low specific gravity. According to all authorities, the abuse of alcohol is one of the most common causes of the granular kidney. According to Christison, three-fourths or four-fifths of all cases of granular atrophy are induced by it.

"*The nervous system.*—The affections of the nervous system in drunkards are both numerous and important. No organ, with the exception, perhaps, of the liver, suffers so constantly and from such a variety of lesions as the central nervous system. Many alterations in the functions are recognizable after death by a change in the tissues, but there are various affections, on the other hand, which point to a marked change in the cerebro-spinal system that cannot be detected. The very delicate and complicated structure of the nerves and ganglion cells requires not only that their anatomical but also their chemical relations shall be preserved for the performance of their functions. Ever so slight a deviation in the nutritive processes produces a disproportionate disturbance in their functions; much greater than in any other tissues of the body.

"*The brain.*—The calvarium is altered. It is increased in weight by hyperostosis and sclerosis, both the outer and inner table

being thickened. The cancellated structure is more dense, owing to a concentric formation of bone about the Haversian canals. Upon the inner surface, the channels of the vessels are deeper than normal as well as the depressions for the Pacchionian bodies.

"There is an increase in the amount of blood in the brain owing to the abnormal action of the heart and fatty or atheromatous degeneration of the walls of the small vessels, or diminished nutrition of the same, which paralyzes them so that their lumen becomes increased, and hyperæmia results. In the earlier stages of alcoholism, where alcoholic excess is relatively frequent, this hyperæmia is more of an active process, which, in the later stages, assumes a passive character when obstruction to the circulation exists in other organs, as the liver, kidneys, lungs, etc.

"*Cerebral apoplexy.*—An effusion of blood into the brain substance frequently occurs in drunkards. All conditions brought about by the intemperate use of alcohol which tend to produce cerebral hyperæmia favor, in a marked degree, the occurrence of either large or capillary effusions.

"*Serous apoplexy.*—An acute or chronic serous effusion into the cavity of the skull, into the brain substance, or into the membranes of the brain, and into the cavity of the arachnoid, may result from the abuse of alcohol. This transudation occurs as a complication in other cerebral diseases, and in those troubles which tend to produce hyperæmia of this organ by mechanical stasis, as in diseases of the lungs and heart. It may also result from a very watery condition of the blood, such as occurs in Bright's disease. In alcoholism the blood is poor in plastic material, and as a consequence the transudation is favored. Either an acute or chronic collection of fluid in the ventricles of the brain is not an infrequent result of drunkenness.[1]

"*Pachymeningitis interna chronica.*—This inflammation of the inner surface of the dura mater consists at first of a very slight layer of fibrin on the surface of the dura, from which a thin layer of connective tissue is afterwards developed, which adheres to the surface of the membrane. A second and a third layer of inflammatory exudation is then formed, and so on until there are many layers. The dura mater thus becomes materially thickened. Each one of these layers is vascular, and occasionally one of these ves-

[1] Hasse, Krankheiten der Nervensystems. Handbuch der Spec. Pathol. und Therap., von R. Virchow, Band 4, S. 365.

sels ruptures, resulting in a hemorrhage between two of the layers. That this affection is more liable to occur in the intemperate there can be but little doubt, although it has been but seldom produced in the lower animals even after long and continued administration of alcohol."

§ 354. *Chemical examination.*—Alcohol when pure and concentrated is quite easily recognized by its odor and its inflammability; also when mixed with a large amount of water its odor can usually be detected, but it cannot always be perceived when mixed with other substances which have a powerful odor. Even when much diluted, it can be detected by adding to its solution a few drops of a solution of potassium bichromate, and then a few drops of concentrated sulphuric acid, when a green solution will be formed from the conversion by the alcohol of the chromic acid into the form of sesquioxide of chromium, the salts of which are green; a large number of other volatile organic substances will produce the same effect. An exceedingly delicate test for alcohol is the iodoform test.[1] To the solution is added a little potassium hydrate solution and enough iodine to give the fluid a yellowish-brown color; a yellow crystalline precipitate, which can be seen by the microscope to consist of hexagonal tables, will soon form; these crystals can also be recognized by their peculiar odor.

§ 355. *Separation from organic mixtures.*—All solid tissues should be finely divided, placed in a retort, and distilled over a water or chloride of calcium bath. Fluids if at all acid must be rendered neutral by adding a sufficient amount of sodium carbonate, before being subjected to distillation. The fluid which distils over will contain the alcohol, more or less impure and dilute, so that it should be mixed with solid chloride of calcium or potassium carbonate and distilled again. This second distillate will usually have the odor of alcohol distinctly, and the amount of alcohol which it contains can be determined by placing it in a graduated vessel, and adding to it as much solid carbonate of potassium as it will take up; the alcohol will separate from the saturated carbonate of potassium solution, and form a separate layer at the top of the fluid. This upper layer of fluid consists of alcohol of 0.825 specific gravity, and contains 89 per cent. of absolute alcohol. It can then be removed and subjected to the above-mentioned tests.

[1] Annal. der Chemie und Pharm., Suppl. Bd., vii. pages 218 and 377.

CHAPTER IX.

NARCOTIC POISONS—Concluded.

III. *Chloroform.*

§ 356. *When injurious.*—Anæsthetic agents, now so much used in surgical, dental, and obstetrical practice, in the form of vapor,

are, as is well known, capable of producing fatal effects. Occasionally death has been due to their mal-administration, the patient being either unfitted to respire them, or having been required to inhale them unmixed with atmospheric air, or for too long a time. Such accidents have been extremely few in the case of ether, and, since the proper mode of its administration has been understood, that is, since care has been taken to admit a sufficient proportion of atmospheric air along with the ether into the patient's lungs, there is not a single authenticated example of its having destroyed life. Chloroform, being more energetic and rapid in its action, has so frequently been the evident cause of death, that the operator cannot be too careful to ascertain its purity, and the probability of his patient being able to bear it, and to see that he does not inhale it to the exclusion of the atmospheric air, or for too long a time.

§ 357. *Symptoms.*—Notwithstanding every precaution, however, occasionally death will suddenly occur in the most unexpected manner from its inhalation. This has occurred in the practice and under the supervision of the most eminent surgeons. Many cases have been published, which it would, however, be tedious to enumerate.[1] We append, however, a few by way of illustration. Patrick Coyle, chloroformed for fistula; he inhaled for about a minute and almost instantly expired.[2] Abbey Pennock inhaled about three drachms in two applications, to relieve the pain of toothache, and died almost immediately after the second application.[3] John Griffiths had chancres and hæmorrhoids; inhaled about three drachms, and died in about ten minutes, during the incision of the hæmorrhoids.[4] In the case of Madame Labrunne, related by M. de Confevron, the fatal effects were manifested in eight seconds, and the operator remarked constant winking of the eyelids. The patient repulsed the dentist's hand, making signs that the effect was not complete. She then made four or five fuller inspirations. At that instant, M. de Confevron removed the handkerchief, and only took his eyes off her for the instant occupied by placing it on the table;

[1] Dr. Crisp laid before the Medical Society of London, a table which he had made, of the recorded deaths from chloroform, up to June, 1853. They amounted to *forty-two*. At the end of 1859 they had already exceeded sixty in number.

[2] Dr. Warren, Effects of Chloroform, etc., Boston, 1849.

[3] Ibid.

[4] Ibid.

but in this brief instant he found the patient's face turned pale, the lips discolored, the features altered, the eyes turned upwards, the pupils horribly dilated, the jaw closed, the head drawn backwards; the pulse could not be felt, the limbs were all relaxed and a few inspirations at long intervals were the only indications of life.[1]

§ 358. Up to the beginning of the year 1867, the total number of recorded deaths by chloroform inhalation were one hundred and thirty-three, in thirty-three (twenty-five per cent.) of which no possible explanation of death, in the state of our present knowledge, could be given, other than that of poison.[2] "The rate of mortality is as 1 in 3500 administrations of chloroform (I really think it is as 1 in 2000 to 3500); we have no other remedial agent which approaches chloroform in point of danger."[3] Since that time the number recorded in the various medical journals of death which could be imputed only to the use of chloroform inhalation is very considerable.[4] Up to the time of the fourth edition of this work (1883) the number of unavoidable deaths during, or immediately after, the chloroform administration has been largely increased. Unlike ether and certain other anæsthetic agents death by chloroform may occur without asphyxia, and without any apparent warning, by syncope either from arrest of the heart's action or from interference with the movement of the blood through capillaries in the nerve centres, chiefly in the brain; this explanation is shown to be correct in a large number of cases, from the fact that recovery from syncope often occurs by starting up this arrested capillary circulation by electrical stimulation, or by lifting the patient up by the

[1] Med. Gaz., vol. ix., 1849, p. 295; *vide* also Med. Times and Gaz., Oct. 1852, p. 361; April, 1853, p. 369; Aug. 1853, p. 173; Oct. 1853, pp. 407, 422, 461; Nov. 1853, p. 562; Dec. 24, 1853, p. 665; Lancet, June and Oct., pp. 523, and 410; Edinb. Monthly Journ., April, 1850, p. 377; 1855–6, i. p. 524; Med. Gaz., vol. ix. 1849, p. 295.

[2] Am. Journ. of Med. Sci., Oct. 1867, p. 324.

[3] B. W. Richardson, M.D., F.R.S., London Med. Times and Gaz., 1870.

[4] *Vide* also Medical News and Library, Philad., vol. xxvii. pp. 89, 103, 118; Medical Times and Gazette, London, June, 1869, p. 695; Nov. 1869, p. 579; New York Medical Journal, vol. x. p. 194; British Med. Journ., 1878, ii. 699; 1879, i. 307; ii. 94; 1880, i. 613; Australian Med. Journ., 1878, 170; Med. Times and Gaz., London, 1878, ii. 465; 1880, i. 613; Lancet, 1878, ii. 571, 862; 1880, i. 268, 346, 825, 973; 1880, ii. 376, and 1882, i. 378; Edin. Med. Journ. 1879-80, 1007, 1014; Canada Lancet, Toronto, 1879–80, 349; Buffalo Med. and Surgical Journal, 1879–80. 531; Philadelphia Med. News, 1882, 295; Medical Press and Circular, 1882, 124

heels and allowing the head to hang down, thus by gravitation inviting a flow of blood in the depending portions of the body, and thereby restoring its suspended functions. A careful examination of such cases as these can leave no doubt upon the mind that death was attributable solely to the inhalation of chloroform, and that it may occur with a celerity unparalleled by any other poisonous agent whatever.

From Jan. 1881, to Feb. 1882, there occurred in the British Isles nine deaths from chloroform, five of which occurred in five weeks, four from ether, one from a mixture of ether and chloroform, and one from ethidene. In two of the deaths from ether the kidneys were diseased. Of the chloroform deaths two only occurred after severe operations.

A case of death from chloroform occurred at the Royal Infirmary, Edinburgh, under Geo. B. Silke, House Surgeon, Jan. 3, 1880, during an operation for removing a portion of bone from a finger which had been chopped off. Before the operation the patient had eaten a hearty meal as was shown by the autopsy, which also revealed extensive pleuritic adhesions of left lung, liver much enlarged and fatty, though the heart was not excessively distended because the external jugular vein had been opened in hopes of relieving the patient; the blood was fluid, having an odor of chloroform. Out of one hundred and twenty-one deaths under chloroform, the period at which death occurred was as follows:[1]—

For intended operations, or immediately before	54
During operations	42
After, immediately, or soon	25
	121

§ 359. In cases of ordinary surgical practice, when chloroform is administered by a competent person, and with those precautions which experience has shown to be necessary, the surgeon is probably not culpable in the eye of the law; but increasing familiarity with its soothing effects, and ignorance of its toxical properties, may be the source of fatal results in the hands of unqualified persons. A case bearing upon the point has been reported.[2]

[1] British Med. Journal, Feb. 18, 1882, from an article on chloroform, by E. H. Jacobs, M.D.

[2] Med. Times and Gaz., April, 1855.

Here the chloroform was procured and administered by a nurse to a woman in labor, contrary to the injunction and without the knowledge of the physician. The woman's death could be attributed to no other cause than the inhalation of chloroform.

§ 360. The external phenomena of etherization, whether produced by chloroform or ether, are very nearly alike. There is usually at first a little cough, with expectoration of mucus and a flow of saliva, and some laboring of the respiration; then the inspirations become strong and deep and take place without difficulty; the pulse becomes quickened, and the eyes injected. With these early symptoms there are often irregular movements of the limbs, and expressions of various kinds are uttered; sometimes a patient will try to put away the sponge or instrument used, but more generally he is anxious to retain it. If the inhalation goes on, the face generally becomes flushed, the eyes are brilliant, and turn in different directions, often upwards; soon the eyelids droop; very often laughter and incoherent expressions are heard; the pulse begins to be slower, and a general insensibility with muscular relaxation follows. This is the true surgical period of anæsthesia. If this period be passed and etherization be pushed to its utmost limits, the respiration becomes stertorous, the face livid, the pulse slow and weak, and death may take place with symptoms of asphyxia. To sum up, temporary excitement, then stupefaction or disorder of the intellectual powers, insensibility, complete muscular relaxation, and death, are the great and observable stages of etherization pushed to its utmost limits.[1] We have already described the psychical effects of the inhalation of the vapors of ether and chloroform.[2]

Sometimes death may take place, as before remarked, without any warning symptom, the pulse and respiration suddenly ceasing, while the complexion and expression of the patient's face is perfectly natural; these cases are more apt to occur when the chloroform is administered to a person erect or in a sitting posture.

§ 361. Serious and alarming symptoms of chloroform poisoning may arise from taking it *internally*. A lady, weakened by miscarriage, was affected, within five minutes after taking half an ounce of chloroform, with convulsions, insensibility, dilated pupils, *trismus*,

[1] Brit. and For. Med. Chir. Rev., Jan. 1852.

[2] *Vide* RAPE.

flushed face, a full and oppressed pulse, and foaming at the mouth.[1] Another person, also a female, swallowed two ounces of chloroform; there was a deep stupor without congestion of the face, and the pupils were contracted.[2] In neither case was the pulse reduced. In a third case the symptoms were the same as in the second, and were also produced by two ounces of the liquid.[3] When death has been produced by the internal use of chloroform, its local irritant action has evidently been the chief cause of the fatal result. Three cases, at least, of this description are recorded.[4] In all of them the quantity taken was from one to two ounces, and the local symptoms were those of an active irritant of the stomach. In one case the air passages shared in the irritation, and their congestion was the immediate cause of death. In all, examination of the bodies showed softening of the mucous membrane of the stomach, and in one case ulceration also.

§ 362. Poisoning by the internal administration of chloroform is totally distinct from the effects produced by its inhalation.[5] It is not impossible that a portion of the chloroform taken into the mouth may be inhaled before it is swallowed, and we may then have the symptoms of inhalation, as well as those due to the ingestion. The quantity of chloroform necessary to occasion serious symptoms is variable. One drachm of the liquid chloroform has brought on narcosis, followed by death in the case of a boy four years old.[6] In another case of a private[7] in a cavalry regiment, two ounces were swallowed, causing serious narcotic symptoms. The stomach was washed out by means of a stomach-pump, twenty minutes after taking the drug, and then ammonia was injected. This man recovered.

The effects of chloroform taken internally appear in from ten to twenty minutes, and are very similar to alcoholic intoxication; but these only precede the more serious symptoms of coma, complete insensibility, stertorous respiration, and convulsions; and finally death in a few hours. Most generally, however, the patient

[1] Med. Times and Gazette, Dec. 1857, p. 615.

[2] Annuaire de Thérap., xviii. 55.

[3] Am. Journ of Med. Sci., Oct. 1857, p. 367.

[4] Month. Journ. of Med. Sci., v, 77; Phila. Med. Exam., Nov. 1856, p. 659; London Lancet, April, 1859, p. 400.

[5] Tourdes, Gaz. Hebd. de Médecine et Chirurg., Paris, 1866.

[6] Taylor on Poisons, p. 741.

[7] Amer. Journ. of Med. Sciences.

slowly recovers his consciousness, but is left with a pain in the throat and stomach, and often has jaundice, cough, and a slight catarrhal bronchitis. These symptoms are those related by Tardieu and Taylor, each of whom give illustrative cases.

§ 363. The only peculiar *post-mortem* irregularities to be recorded are the slow putrefaction and persistent rigor mortis, and the special odor of chloroform; but as this latter is much less diffusible than ether, it is not so readily recognized. Consequently all the organs and tissues reserved for chemical examination should be kept as much as possible from the air.

§ 364. In regard to the mode in which the inhalation of chloroform occasions death, a review of the fatal cases furnishes a very uniform result, and in general shows but very few symptoms as precursors or concomitants of death. In a very small number more or less twitching of the muscles of the face and extremities, and in two or three instances spasmodic contractions of the muscles of the lower portion of the body have been noticed; but, in general, the breathing grows feeble and infrequent, the pulse small and faint, and the face pale and cold. In a smaller proportion of cases, where asphyxia is caused by exclusion of air from the lungs, the features are congested instead of pallid, and in these the respiration is more or less stertorous, and there is sometimes foam upon the lips. In the former group the mode of death indicates exclusively a direct poisonous action of the chloroform, producing an arrest of the heart's action, or syncope, while in the latter are *superadded* the effects of an exclusion of atmospheric air, in other words, the signs of asphyxia.

§ 365. Casper treats of a *chronic poisoning by chloroform*,[1] maintaining that the vapor may prove fatal after the lapse of hours, days, or even weeks, the patient meanwhile suffering more or less from its effects.

§ 366. The *post-mortem appearances* found in those who have perished by chloroform are remarkably uniform when the death is due to asphyxia, but when death is due to syncope there may be no visible *post-mortem* peculiarities. In the former cause of death, like other narcotics, we find great congestion of the lungs and bronchial tubes, whilst the blood may be dark and fluid. Exceptions have, indeed, been observed to this rule, but in a very large

[1] Gericht. Med., i. 621.

majority these appearances are constant. This condition is not, however, significant of any peculiar action of chloroform upon the blood. As Dr. Snow has remarked, it generally remains fluid after death by chloroform, only because it remains fluid in every kind of sudden death. It is found equally so in death from any asphyxiating cause, and in cases of narcotic poisoning. It may also be due to disease. Dr. Faure is of opinion that the congestion of the lungs met with in cases of death from chloroform is, to some extent at least, a hepatization produced by the combination of the vapor with the blood in the vessels.

§ 367. *Medico-legal bearings.*—Dr. Charles Kidd[1] says, that the following five points are deducible from an increasing experience with chloroform: 1st. Dilatation, or rather engorgement of the right side of the heart may be calculated on, if the *post-mortem* be made correctly, and the patient may not have died of simple syncope rather than this apnœa. 2d. Chloroform may be recovered by distillation from the blood and lung tissues. 3d. Chloroform acts slightly upon the blood corpuscles, they being jagged or crenated at the edges, while ether breaks them up; the lungs, however, are usually anæmic or pale under chloroform. 4th. Chloroform is more fatal in the struggling stage, or if there be signs of delirium tremens present. 5th. Great caution should be observed in believing the confused ideas of women menstruating at the time of taking chloroform.

§ 368. Dr. Stephen Rogers, president of the Medico-legal Society of New York, has contributed to the *Journal of Psychological Medicine* for October, 1871, a very interesting essay on the question "whether chloroform can be used to facilitate robbery." He maintains the negative in an argument from which we extract the following:—

"Referring to the case of the jeweller's shopman, who alleges that the wife covered his face with a handkerchief, while the husband held his hands; it must be obvious to any one at all acquainted with the use of chloroform, that the theory of his allegation is preposterous. Would a strong man, determined to save himself from impending suffocation, stand passively and allow a man and a woman to practise the administration of chloroform on him? He could at least have fallen down and turned his head away enough to have

[1] Edinburgh Medical Journal, September, 1870.

enabled him to scream for help, like the man Kendal, or the young woman in the stable-yard. From all the light which practical experience and the investigation of crimes throw upon this case, there seems little doubt that the shopman was party to the robbery.

"It is at least a very suggestive case, as showing that there is great liability to err on the part of courts in accepting this kind of allegation, unless, upon detailed inquiry into the circumstances and the manner of the alleged giving of chloroform, they be found consistent with the thoroughly well-known facts and phenomena uniformly attending the administration and action of this agent.

"Any inconsistency such an inquiry might develop should be accepted as indicating honest delusion, studied deception, or complicity in the crime.

"I feel convinced that such a test would exclude, at least nineteen in twenty of all these cases of the alleged felonious use of chloroform and similar agents, as mere fictions.

"But as there may be, as in times past there has been, a popular disposition to accept the statements made by the alleged victims of the felonious use of chloroform, and by their friends, as true, notwithstanding what I may say, or other authors may say to the contrary, with the single motive of bringing before the public and the legal profession all that may be true and well authenticated in this matter, I have challenged, and now repeat it, the production of any proof of the successful use of chloroform on the human subject to facilitate robbery in a single instance.

"As I have before had occasion to say, when any such proof is furnished me, that robbery has ever been committed by means of the use of chloroform received unconsciously by the person robbed, or given forcibly against the resistance of the person robbed, I will be ready to admit it, and this Society will promulgate the fact to the world.

"And I cannot conclude these remarks in a more truthful and forcible manner than by adopting the language of Dr. Snow, who so long ago said: 'The public have been greatly and unnecessarily alarmed about the employment of chloroform by thieves; what they really have to dread is, that robbers will still resort to the old means of the bludgeon, the pistol, and the knife, and not to one, which, like chloroform, allows the victim so good an opportunity of escape, and themselves so great a chance of detection.'"

Dr. Snow says: "The sensation of pungency in the nostrils and throat that is caused by this agent when its vapor is in sufficient quantity to produce any effect on the sensorium, is so strong and peculiar, that no person can take a single respiration without being aware that he is inhaling something very unusual. Chloroform, in fact, can never be administered without the consent of the party taking it, unless by main force, which has to be used in the case of children who are not old enough to be reasoned into taking it. If a child be asleep when the process of inhalation is commenced, it nearly always awakes before being made insensible, however gently the vapor may be insinuated. As breathing is perfectly under the control of the will, a person would, on finding such a strange attempt being made upon him in the public street, instantly hold his breath, and use all his powers of resistance to repel the assault," etc.[1] Lord Campbell, in his speech in the House of Lords advocating the adoption of the bill making unlawful administration or application of chloroform and other stupefying agents felonious, made the following remarks: "A most respectable physician had done him (Lord Campbell) the honor to write him a letter, which he had printed, and there he stated the fear arising from the use of chloroform in this way was altogether imaginary, that no strong man who made resistance could possibly be chloroformed. He believed that was true; but in the case of those who were not strong, and unable to resist, it might happen to many of that class that the chloroform would be employed most effectively for facilitating robbery. The gentleman to whose letter he had referred, stated that a person thus attacked might refuse to breathe, and that he might turn away his head. But, suppose a wet handkerchief was put to his nostrils, and held there, the man must breathe, and thus inhale the particular gas that came from the chloroform. It stood, indeed, on record, that since the discovery of chloroform, persons had been convicted before the competent courts of using that article for the purpose of robbery. He hoped, therefore, their lordships would be of opinion that those who made such an attempt should not be guilty of a misdemeanor only, as was at present the case; but that any person who tried to commit a robbery by means of chloroform or such like substances, *though he did not succeed*, if convicted, be

[1] Lond. Med. Gaz., 1850.

held guilty of felony, and be liable to be transported beyond the seas."

We have not seen the evidence brought forward in the cases thus referred to, but we do not doubt that it was fully sufficient to establish the fact of chloroform having been used for the purpose alleged; the only recorded instance which we have met with is the following; its employment was, however, as will be seen, unsuccessful. A gentleman named Mackintosh had retired to bed at a hotel in Kendal. He was awakened about twelve by a man attempting to suffocate him by means of a rag steeped in chloroform. Mr. Mackintosh, who is an elderly man, struggled desperately with his assailant; but, whether from the fumes of the chloroform, or the disadvantage at which he was taken by his midnight assailant, he felt himself fast fainting, when his cries of "Help! murder!" roused the house. When the landlord made his way into the room, Mr. Mackintosh was almost powerless, and his assassin or robber was lying upon the bedding, which had fallen upon the floor in the scuffle, apparently sound asleep. On being roughly shaken, the latter professed that he had long been a sleep-walker, and appeared to be astonished to find himself where he was. A policeman was sent for and the man taken into custody. A strong smell of chloroform was perceived by the parties who entered the room upon the alarm being given, and a bottle containing chloroform was found under Mr. Mackintosh's bed, and a similar bottle in the carpet-bag of the prisoner, who had been at the hotel several days. The probability was that the ruffian was secreted under the bed when Mr. M. retired, as the latter had placed a chair previously against the door to prevent intrusion, there being no lock upon the door.[1] This criminal escaped with eighteen months' imprisonment; the offence not being a felony at that time, since there was no intent to commit *murder* shown.[2]

[1] Med. Gaz., Nov. 1850.

[2] The following testimony is taken from the records of a recent trial (New Bloomfield, Perry county, Pa., Jan. 18, 1871) for an attempt at robbery by the use of chloroform:—

F. F. Maury, M.D., recalled: Chloroform *very, very* often produces resistance. It sometimes produces irritation and sometimes a depressing feeling. It produces vomiting. If the stomach is full, nausea and vomiting almost always follow. Sometimes it does not. I experimented with chloroform in six sleeping persons. Out of that number all resisted, more or less. Two *men* woke up immediately, and one remarked, "You are trying to give me

Several remarkable instances of robbery of persons designedly rendered insensible by chloroform have lately been reported in the

something." Unquestionably, it requires more chloroform to produce death in a recumbent position than in an upright posture. One man cannot administer chloroform to another.

Chloroform will not mix with water; will mix with alcohol. Thrown into water, it breaks into small pieces, as it were. I administered it to a child *four days* old. It offered resistance. With my experience I could not administer chloroform to *four persons*.

B. Howard Rand, M.D., sworn: Am Professor of Chemistry and Lecturer on Medical Jurisprudence in Jefferson Medical College. Cases are numerous where persons have been overcome by smoke and noxious gases, and have perished without attempting to escape. They have been found near lime-kilns, brick-kilns, cement-kilns, and in houses on fire. The gases formed when wood or coal is burned are chiefly carbonic oxide and carbonic acid. They are both capable of causing death by suffocation. Carbonic oxide is by far the more poisonous; a single inhalation of the pure gas will produce insensibility; it acts like chloroform to produce insensibility, but is vastly more powerful; death is the rule and recovery the exception after the inhalation of carbonic oxide, while the reverse is the case with chloroform. When a house is burning, insensibility may result in part from the diminution of the oxygen within it. When the proportion of oxygen falls below about 18 per cent. insensibility will follow. The vapor of chloroform is 4.2 times heavier than an equal bulk of air. Gases and vapors diffuse or mingle more slowly as they are heavier. Chloroform vapor formed from the liquid poured on the floor would form a layer near the floor, from the upper surface of which, supposing the air of the room to be quiet, diffusion would slowly take place, so that the smell of chloroform would be perceptible in the room, but not enough of the vapor would arise to stupefy; the greater part of it would make its way under the door and through cracks, if any existed, in the floor. This vapor is so heavy that it can be poured like a liquid from one vessel into another. It is not inflammable, and puts out a light. The experiment detailed by Dr. Maury was made at my suggestion, and as a participant I confirm his statements. We concluded the experiment at the end of fifty minutes because we were satisfied that no different result would be arrived at by prolonging it. The only effects noticed were quickening of the pulse of those in the close room, with a pound of chloroform on the floor, except that I was nauseated by the smell of the chloroform vapor, but did not vomit. Mr. Boman remained the whole time lying on the floor with his head resting on his hand, at an average distance of seventeen inches from the floor, and was unaffected, except as before stated.

Cross-examined.—Chloroform vapor, as ordinarily administered, when it kills, does so not by deprivation of oxygen, but from its own noxious qualities. In lime-kilns a large amount of the noxious gases arise from the decomposition of the limestone, which consists of lime combined with carbonic acid; in the case of brick and cement-kilns, the noxious gases arise from the fuel. The proportion of carbonic oxide to carbonic acid in any case will vary; in a smouldering combustion, one where the supply of air is limited, carbonic oxide would predominate; where the

newspapers of this country; although they may be authentic, we do not feel warranted in further alluding to them while unable to attribute them to responsible sources. It is obvious that a person may allege that he has been robbed or maltreated after being rendered insensible by chloroform, and also that the allegation may be false, and be put forward so as to divert suspicion or awaken sympathy.

§ 369. *Chemical examination.*—This is a very easy matter if not delayed long after death, but unless the chemical research is undertaken very soon the volatile agent will not be detected.

The method to be employed[1] is that proposed by Duroy, and consists in forcing over from a retort, warmed slightly (about 107° F.), and containing blood, liver and brain tissue finely divided, the vapor of chloroform through a hard-glass or porcelain tube heated red hot into a potash-bulb containing a solution of nitrate of silver slightly acidulated with nitric acid; if chloroform be present, a flocculent, white precipitate of chloride of silver will be seen in the silver solution, and white fumes will be seen issuing from the orifice of the potash-bulb; these fumes are those of chlorine gas, which can easily be recognized by inserting a piece of moistened blue litmus paper into the exit tube, when the chlorine fumes will first turn the the litmus paper red and finally bleach it. The vapor may be forced out of the retort by a pair of bellows attached to a tube resting just above the organic matter, or else by an aspirator placed at an outlet of the potash-bulb, or still better, by attaching an ordinary gas holder filled with air to a tube which just dips beneath the surface of the fluid in the retort, and allows the air to be forced slowly through the fluid. Care should be taken to force the vapor through the combustion-tube quite slowly. By this process the chloroform is decomposed into chlorine and hydrochloric acid, which forms chloride of silver with the silver solution. This latter may

supply of air is abundant, carbonic acid would be formed in larger quantity. Anthracite would weight for weight yield more carbonic oxide than charcoal, and charcoal than wood. Ordinary smoke contains, beside the noxious gases mentioned, many other bodies, as steam, tar, wood spirit, wood vinegar, etc., the nature and proportions of which will vary with the dryness of the wood and the temperature or degree of heat to which it has been subjected.

[1] Du Rôle de l'Alcool et des Anesthetiques dans l'Organisme, Lallemand, Perrin, et Duroy, Paris, 1860.

be recognized in the following ways: It is insoluble in water and boiling nitric acid,[1] but is easily soluble in a slight excess of ammonia; and if the solution containing the precipitate be boiled, black metallic silver will be formed. Provided no white flocculent precipitate has been produced in the silver solution by the above process in fifteen minutes after the combustion-tube has been heated to redness, the experimenter may fairly conclude that no chloroform is present.[2]

§ 370. *Fallacy.*—Any free hydrochloric acid in the tissue must be tested for first of all by litmus paper, and if it is present, a little soda or potash should be added. If chloral had been taken by the patient just previous to death, and the soda and potash be added to the tissues, the chloral would become decomposed into chloroform. These three substances hydrochloric acid, chloroform, and chloral hydrate can all be detected by the same process and apparatus, and at the same time; for the detection of the hydrochloric acid the air should be forced through the mixture in the retort before the combustion-tube is heated, when, if hydrochloric acid in the free state is present, a precipitate of the chloride of silver will appear in the silver solution; if none appears in a few minutes then the tube should be heated, when, if chloroform is present, the precipitate and fumes mentioned above will be seen; if this result does not take place, then soda or potash should be added to the contents of the retort when, if chloral is present, a precipitate of chloride of silver will be formed in the potash-bulb, and the fumes of chlorine will be seen.

IV. *Hydrate of Chloral.*

§ 371. This is a chemical compound discovered by Liebig, in 1831, and introduced in the year 1869 by Prof. Oscar Liebreich, as a therapeutic agent. It is not proposed to discuss here its physiological or therapeutical action.[3] This agent has in many

[1] Cyanide of silver is soluble in the latter, and can thus be distinguished from the chloride.

[2] *Vide* some experiments concerning chloral; by the editor of third ed., in the New York Med. Journ. for June, 1872. Also, Sansom on Chloroform, 1866. Lindsay & Blakiston, p. 45.

[3] Reference may be made to Das Chloral-hydrat; also Action du Chloral sur l'Economie, Revue Thérapeutique, 1st Oct. 1869; and Allg. Med. Central Zeitung, Jan. 1870, by O. Liebreich; and Demarquay, Comptes Rendus de l'Académie des Sciences, t. lxix. p. 968; translated in Boston Med. and Surg.

instances been unfortunately the cause of death when taken in an overdose. There have also been cases reported, where it was used with suicidal but none with criminal intent. Our notice will, therefore, be confined to a review of some of the cases which have been noticed in the various medical journals.

Dr. Smith, of Baltimore,[1] reports four cases which came under his personal observation; in some of these fatal cases of poisoning, chloral-hydrate had been used in moderate continued doses, and in the others in an excessive single dose. Dr. Smith calls especial attention to a rash and cutaneous desquamation that occurred in these cases.

§ 372. Dr. Richardson, of London,[2] considers two drachms of chloral a dangerous, and three drachms a fatal dose. He thinks also that we cannot safely increase the daily dose (in fact it is in this way that many of the accidents have occurred) of chloral as we can that of opium, and that the system does not become habituated by its continued use, nor able to eliminate larger doses than at first.[3]

A clergyman in England[4] had been in the habit of using chloral in small doses to induce sleep. Though the precise dose that preceded his death could not be ascertained, yet during ten days he had consumed from fourteen to fifteen drachms (nearly two ounces). At the autopsy the peculiarity noticed was a congestion of the cerebral membranes, while the brain mass was pale and friable; there was no very great increase of blood in the smaller vessels except in the choroid plexus.

In the first half of the volume for 1871 of the *Lond. Med. Times and Gazette* are recorded three more cases of death occurring after the use of this drug.

Journ. Nov. 1869, p, 253. Jacquemet, le Choral et ses Vicissitudes Experimentales, Montpellier Médical, 1869, t. xxiii. pp. 450 et 554. Laborde, Dangers de l'Administration du Chloral, Comptes Rendus de l'Académie des Sciences, t. lxix. p. 987. Personne, Sur la Preparation et les Propriétes de l'Hydrate de Chloral, Repertoire de Pharmacie, 1870, p. 241. Dr. B. W. Richardson, Report on Hydrate of Chloral, Med. Times and Gazette, London, Sept. 1869, p. 290. A Review of Liebreich's Pamphlet, in London Practitioner, 1869, p. 239. Gazette Hebdomadaire, Liebreich, 1871, p. 715; also some experiments reported in N. Y. Med. Journ. for June, 1872, by editor.

[1] Boston Med. and Surg. Journ., July, 1871, p. 33.

[2] Med. Press and Circular, Feb. 25, 1871.

[3] See Appendix.

[4] Brit. Med. Journ., Feb. 25, 1871.

Dr. H. W. Fuller,[1] reports a case in which thirty grains (half a drachm) of chloral produced alarming symptoms, and another in which the same dose produced death.

Dr. Needham[2] reports the case of a woman, aged fifty years, who took thirty grains of chloral to relieve a mental unquietness, amounting to insanity. The first day she took this dose at half-past five in the evening and repeated it at eleven o'clock. The second day at ten in the morning she took fifteen grains, and at three in the afternoon she repeated this dose. The third day she took thirty grains at one o'clock in the morning, again a few moments after eight, and finally at half past one in the afternoon. On the evening of the second day she was out of bed and moving about. On the third day she was sleeping and had a somewhat rapid pulse, and at six in the evening she was still sleeping. The next morning her pulse was 108, and attempts to arouse her were of no avail. The pupils were a little contracted. After several ineffectual attempts to arouse her during the day, one-tenth of a grain of strychnine (an antidote recommended by Prof. Liebreich) was subcutaneously injected in three separate doses; but she passed from her lethargic sleep into a state of coma, and died the next day at four in the afternoon.

At the autopsy an examination was made only of the brain, which was found everywhere deeply congested, and in the meshes of the pia mater was a sero-gelatinous exudation.

Dr. Dabb reports in minute detail[3] a case where serious apprehension was felt for the life of his patient.

Another case has been reported in the service of Dr. Ludlow[4] at the Philadelphia Hospital, where a nurse, supposed to have swallowed four hundred and sixty grains (an ounce and a half), was found in an unconscious condition, from which she was with great difficulty rescued by vigorous flagellation of the surface of the body and by the application of electricity.

In acute alcoholism, or mania à potu, the absorption of chloral hydrate is slow or is retarded; this sometimes leads to fatal accidents from the incautious use of large doses. As the elimination of alcohol is accomplished, the accumulated large doses of chloral in

[1] London Lancet, March 27, 1871.
[2] The Medical Times, Feb. 15, 1871.
[3] Med. Times and Gaz., Oct. 8, 1870
[4] The Medical Times, Oct. 15, 1870

the stomach may be all at once absorbed and bring about a fatal poisoning.

From what is known with regard to the use of this drug it is probable that not more than twenty to thirty grains of chloral should be administered in a single dose, and that it is somewhat dangerous to repeat this at shorter intervals than six to eight hours. (The absorption of chloral by a fasting stomach is quite rapid.)

§ 373. The first symptom of danger, after the continued *abuse* of this drug, is a peculiar rash resembling in some particulars that of scarlatina or roseola. This may be followed by desquamation of the cuticle, and is generally, perhaps invariably, accompanied by albuminuria.

The dangerous symptom that follows a single overdose of chloral is profound sleep, accompanied with stertorous breathing, not very unlike that produced by opium; but still the patient can be partially aroused though unable to talk coherently; this profound sleep may pass into coma, muscular relaxation, and finally death.

Strychnine is not to be relied upon[1] as an antidote to chloral poisoning. The most theoretical and practical method of relief in cases of poisoning would be external stimulation, rectal injections of some stimulating liquid, as a mixture of castor oil or broth with turpentine, and the use of electricity, either in the form of galvanization or faradization; and also the placing of the patient in a warm atmosphere, and causing him to inhale warm air.

"The maximum quantity of chloral-hydrate that can be borne, at one dose, bears some proportion to the weight of the animal subjected to its influence. The rule, however, does not extend equally to animals of any and every class. The proportion is practically the same in the same classes, but there is no actual universality of rule. . . . The human subject, weighing from one hundred and twenty to one hundred and forty pounds, will be made by ninety grains to pass into a deep sleep, and by one hundred and forty grains into fatal sleep. . . . Evidence has been brought before me which leads me to think that, although eighty grains would in most instances prove fatal, it could, under very favorable circumstances, be recovered from.

[1] *Vide* Gaz. Hebdomadaire de Méd. et de Chirurgie, June 21, 1872, p. 409; and July, 1872, p. 457.

"Dr. Hills, of the Thorpe Asylum, Norwich, has, for example, favored me with the facts of an instance in which a suicidal woman took no less than *four hundred and seventy-two grains* of the hydrate of chloral dissolved in sixteen ounces of water, and actually did not die for thirty-three hours. Such a fact, ably observed as it was, is startling; but it does not, I think, militate against the rule that one hundred and forty grains is the maximum quantity that should, under any circumstances, be administered to the human subject. . . . As a near approximation to the truth, an adult person who has taken chloral in sufficient quantity to be influenced by it disposes of it at the rate of about seven grains per hour. In repeated doses the hydrate of chloral might be given at the rate of twelve grains every two hours for twenty-four hours, with less danger than would occur from giving twelve times twelve (144) grains at once; but I do not think that amount ought to be exceeded, except in the extremest circumstances, even in divided doses."

The above is extracted from an exceedingly interesting report on the physiological action of organic chemical compounds, by B. W. Richardson, M.D., F.R.S.[1] From other experiments and observations alluded to in the same paper, Dr. Richardson "has no doubt it will be found, as the chronicle of deaths from chloral hydrate increases, that the mortality of the agent will be greatest when the thermometrical readings are the lowest, and *vice versâ*."

§ 374. *Post-mortem appearances.*—These are stated by Dr. Francis Ogston[2] to be as follows: The bloodvessels of the membranes of the brain are full of blood; the sinuses contain clotted blood; the arachnoid (serous) membrane is œdematous; under this membrane and in the ventricles of the brain clear serum in considerable quantity is found; the brain substance appears somewhat shrunken, but no unusual number of hemorrhagic spots may be noted; the œsophagus is usually slightly reddened and thickened in folds, and its mucous membrane is softened; the mucous membrane about the larynx is œdematous with fine injection of its bloodvessels; the lungs are œdematous and deeply congested; the right side of the heart is

[1] Report of the British Association for the Advancement of Science, for 1871, p. 145.

[2] Edinburgh Medical Journal, Oct. 1878.

engorged and the left comparatively empty. The blood in the heart may be partly clotted and partly fluid; the rest of the blood-making organs, such as the spleen, liver, etc., will be filled with dark blood. In other words, the *post-mortem* appearances are those usually found after death from narcotico-acrid poisons, and when death may be assumed to have been caused by asphyxia.

§ 375. *Chemical examination.*—Chloral.—This compound presents the appearance of a clear and transparent liquid, which gives to paper the appearance of grease spots, which, however, soon disappear on exposure to the air. Its density at 18° C. (64.4° F.) is 1.502. It boils at 94° C. (201° F.) and distils without undergoing alteration. The density of its vapor is about 5.0.

Its odor is penetrating, provoking tears. It has almost no taste. In its anhydrous condition it is very caustic, especially if the skin is exposed to its vapor at a boiling point. It dissolves easily in water in great quantity and without residue. If a few drops of this body be dropped into water, an immediate precipitate is formed at the bottom of the vessel, under the form of an oleaginous liquid, but by a slight elevation of temperature these drops are immediately redissolved. The liquid possesses no acid reaction. If nitrate of silver be added no precipitate of chloride of silver is formed. When even a concentrated solution of chloral in water is boiled with red oxide of mercury no change takes place.

If, instead of gently heating chloral with water, it is put in contact with a few drops of water, it will immediately combine on agitation, attended with the production of heat. A few moments after which a crystalline mass appears. These crystals placed in contact with water are dissolved without residue. This solution contains hydrate of chloral (or chloral-hydrate), which has undergone no alteration.

§ 376. Chloral-hydrate is seen in the form of colorless rhomboidal crystals, or white crystalline plates which are very soluble in water (one and one-half parts), alcohol, ether, chloroform, benzol, and naphtha. It has an aromatic odor and a sharp, disagreeable taste. It melts at about 60° C., boils at 95° C., and at a higher temperature volatilizes without leaving any residue. When the aqueous solution is treated with potassium or sodium hydrate, it becomes turbid and has the odor of chloroform, the chloral hydrate being decomposed into formic acid and chloroform; the formic acid

thus formed combines with the sodium or potassium to form the formiate of the alkali; this is the most important test for chloral, since it serves not only for the qualitative, but also the quantitative analysis; 100 grammes of chloral-hydrate yield 72.2 grammes or 48.1 cubic centimeters of chloroform. In order to estimate the amount quantitatively, the chloral is decomposed by adding an excess of normal standard solution of sodium hydrate; each cubic centimeter of this solution which is neutralized by the formic acid produced, corresponds to 0.1655 gramme of chloral hydrate; it is necessary that the solution should be neutral before adding the sodium hydrate solution. The chloroform resulting from the decomposition of the chloral-hydrate by the alkali can be detected in the manner already described in speaking of chloroform (§ 369).

§ 377. Chloral-hydrate can be *isolated* from *organic mixtures* by following the process recommended by Dragendorff for alkaloids (see § 348), the chloral being readily removed from its aqueous solutions by shaking with naphtha, or preferably with ether; the ether after evaporation leaves the chloral as a residue in crystalline form. This residue can be dissolved in water and tested as above mentioned.

V. *Ether.*

§ 378. Undoubtedly there have been a few cases of death during the administration of ether.[1] Some of these are, as in the case of chloroform, explicable under the supposition of shock from a surgical operation, some from asphyxia due to the exclusion of a proper supply of atmospheric air, and some, and these the largest portion, from disease of the kidneys which usually has not been discovered except at the *post-mortem* examination. Unlike chloroform, ethyl bromide and certain other anæsthetics, etherization carefully performed with due caution as to a proper supply of fresh air for respiration, and when the kidneys are in normal state, so rarely has occasioned death by its direct action, that it is not reasonable to classify ether as a poison; yet its use should always be in the hands of a competent physician.[2]

[1] Cincinnati Lancet and Clinic, New Series, v. 380; Phila. Med. Times, 1880, 545, 547; Lancet, Lond., 1881, ii. 386, 1882, May 27th.

[2] Dr. S. C. Parsons (Surg. N. Y. Dispensary, in the Med. News for Mar. 18, 1882) mentions a case of death following administration of ether, six

VI. *Camphor.*

§ 379. *Symptoms.*—Although camphor cannot be regarded as a very active poison, no well-authenticated case of death resulting directly from its use having, as far as we are aware, been yet reported, it is, nevertheless, capable of producing very dangerous symptoms. These, in the cases which are known, have varied somewhat, but in all there has been more or less evidence of its action upon the brain; vertigo, confusion of intellect, delirium, and somnolence being the most prominent effects. Indeed, the primary action of large doses of camphor is a powerful but not a permanent sedation of the nervous and vascular systems, followed by ataxic phenomena, and remotely by slight and very transient febrile excitement. Dr. Florain has reported the following curious case: A man 56 years of age and of good constitution, took for the relief of priapism, and under a misapprehension of the directions of his physician, an enema containing *ten* drachms of camphor. Immediately afterwards he had sensations of cold alternating with heat in the lower bowels, and these sensations extended along the spine to the neck, and spread over the whole body. He was then seized

fluidounces being used, the operation being for dislocation of the humerus. Death resulted after the patient had recovered from the influence of the ether, *post-mortem* showing lungs congested, with heart, liver, and one kidney healthy, the other being somewhat fatty.

A case of death reported in the Lancet, for May 27, 1882, following the administration of ether after a severe operation of removing a fibroid polypus uteri, death occurring five minutes after operation, and being the result of shock, no indication of asphyxia being found.

A case of death from the inhalation of one ounce of ether given for obstruction of the bowels, is reported by Dr. Robert N. Hartley, in the Lancet for Sept. 4, 1880.

Dr. John B. Roberts, in Phila. Med. Times for June 4, 1881, records a case of death from ether, three and one-half fluidounces being used, death occurring about an hour after the operation, the case being that of a woman, whose heart showed evidence of fatty degeneration. He also reports a case occurring in 1876, in which death occurred subsequent to etherization, two and one-half fluidounces of ether being used, death being the result of asphyxia. In a case of his, in which bromide of ethyl was given for lithotomy, four fluidrachms being given, death resulted from syncope, occurring immediately after the first incision.

On Tuesday, Aug. 16, 1881, a case of death from ether administration occurred at Addenbrooks Hospital, Cambridge, Eng., the patient being a woman fifty years of age, death resulting during administration from asphyxia.

with vertigo, had grotesque hallucinations, an excessive frequency of the pulse, embarrassed respiration, vomiting, and strangury, and was greatly prostrated within two minutes after taking the injection. The delirium increased, the features became pale and decomposed, the eyes fixed, and the pupils dilated. The skin became covered with clammy perspiration, and was ice-cold, the pulse frequent and thready, and the impulse of the heart very feeble. When violently aroused, the patient regained his consciousness for a moment, complained of distressing nausea, extreme chilliness, and great desire to sleep. Vomiting of a yellow watery fluid, smelling of camphor, followed, and was succeeded by great prostration. By the assiduous employment of stimulation, both externally, and internally, as well as purgatives, the patient was rescued from this very precarious situation. He recovered entirely, and the only durable effect of the camphor was seen in the complete anaphrodisia which lasted for several weeks.[1] Two other cases are reported where the camphor was also given in injection (of about a drachm), and which were followed by analogous symptoms. In one of these, the symptoms were very similar to those of an epileptic convulsion.[2] Dr. O. E. Brown, of Kentucky, mentions the case of a young man, who chewed and swallowed about 100 grains of camphor. No symptoms came on for a short time, but he was, perhaps an hour afterwards, suddenly seized with convulsions, and remained unconscious for several hours. He was relieved by bleeding and a warm bath. He gradually recovered his speech, but remained stupid, languid, and wandering all the next day.[3] A few cases are quoted by Drs. Taylor and Christison, in which camphor was taken by the mouth, but they do not differ essentially from the preceding.

§ 380. *Power.*—The smallest dose which appears to have been attended with serious symptoms, is *twenty grains.*[4] In a case related by Wibmer, as much as eight scruples of camphor dissolved in spirit were swallowed by a drunkard. This was followed by vertigo, dimness of sight, delirium, and burning pain in the stomach; there was vomiting, and yet the man recovered. The nature of the poisonous agent cannot fail, in cases where camphor has been taken,

[1] Gaz. des Hôpitaux, No. 41, 1851.
[2] Canstatt's Jahresbericht für 1851, Bd. iv. p. 277.
[3] Bost. Med. and Surg. Journ., xxxvi. p. 868.
[4] *Vide* Taylor on Poisons.

to be discovered, since the odor is so powerful and so well known as to betray itself at once.

§ 381. Dr. Taylor[1] mentions three cases of fatal poisoning from camphor in infants under two years of age; in one of these camphorated oil was given by mistake, in another half a teaspoonful of powdered camphor was followed by fatal results. Dr. Taylor also mentions several cases reported by Dr. G. Johnson, in which very dangerous but not fatal effects followed the use of a strong solution of camphor in alcohol in small doses of twenty drops or more. This solution went under the name of *Rubini's homœopathic camphor*, the strength of which in camphor was not known. It must, however, be admitted that very large doses of camphor may be followed by dangerous but not fatal symptoms.[2] Another case of poisoning by camphorated oil[3] is reported by Dr. Hewetson. Dr. Lamadrid[4] mentions a case in which an attack of acute gastritis followed an excessive dose of camphor.

§ 382. *Chemical examination.*—Camphor is readily detected by its powerful odor, and can be separated from organic mixtures by shaking the acidulated mixture with naphtha, which upon being allowed to evaporate will leave the camphor mixed with more or less fat. It can be separated in purer form by the method recommended by Dragendorff.[5] If much camphor has been taken, however, the odor will be very perceptible in the contents of stomach and intestines.

VII. *Hydrobromate or Monobromide of Camphor.*

§ 383. Brominated camphor, formed by the combination of bromine and camphor, is a violent poison in doses of thirty grains or more.[6] The symptoms are muscular weakness, severe prostration, convulsions, great reduction of the bodily heat (sometimes 30° Fahr.), diminution in the number of respirations and of pulse, sopor, stupor, coma, and death. The symptoms have at first a rapid progress, but, when fatal, death takes place slowly. An insufficient

[1] Op. cit.

[2] *Vide* Trousseau et Pidoux, Traité de Thérapeutique, Paris, 1862, art. Camphre, action toxique, p. 267.

[3] Lancet, London, 1880, 88.

[4] Phila. Med. Times, 1879, 325.

[5] See § 348.

[6] Progrès Med., 1874; London Practitioner, 1874–75; and a monograph by Pathault on Bromure de Camphre, Paris, 1875.

number of cases of fatal results are reported to base any definite conclusions of pathological appearances. The drug has a very sedative influence upon the vaso-motor nervous system, thus interfering with the functions of organic life, especially the respiratory and circulatory systems.

VIII. *Nicotiana Tabacum.* (Tobacco.)

§ 384. *Symptoms.*—The symptoms produced by a poisonous dose of tobacco are nausea, vomiting, a burning heat in the throat and stomach, colic, diarrhœa, frequent urination, extreme giddiness, great anxiety with a disposition to faintness, pallor, coldness of the extremities, and spasmodic trembling; the pulse is small, weak, tremulous, and intermittent; the breathing is labored and stertorous; there is a paralytic relaxation of the voluntary muscles, and clonic spasms of the limbs. The pupils are but slightly affected, and the eyes seem to be sensible to light. This state is succeeded by a general torpor, or utter prostration, which is not coma, but which may terminate in death. Tobacco has produced death by having been criminally mixed with liquor. The dose capable of destroying life, when introduced by the mouth, is not known.

Two cases are related by Dr. Deutsch, in which life was in extreme danger from the swallowing of tobacco. In one, a soldier suffering with a tapeworm, took, by the advice of a friend, some of the extract of tobacco, such as is deposited in smoking-pipes. The quantity swallowed was estimated at an ounce. He was at once seized with the most horrible pains in the stomach, and fell into a state of extreme collapse. The efforts to vomit were ineffectual until an emetic was given to him. After extreme suffering, he slowly recovered. In the other case, a young lady accidentally swallowed the still lighted stump of a cigar which she had been smoking, and suffered greatly from the ordinary symptoms of poisoning by tobacco, together with pain in the stomach, until she was relieved of it by vomiting. A fatal case is reported by Mr. Skae, of a man who swallowed a large mouthful of crude tobacco. In addition to the usual symptoms he had convulsions.[1] A further variation from the ordinary course of symptoms was shown in the case of two females, about eighteen years of age, both of whom

[1] Edinb. Med. Journ., i. 643.

drank an equal quantity of a decoction of tobacco. The one was affected in the usual manner, the other became insensible and was attacked with convulsions; her arteries and veins were distended, the former throbbed forcibly, and the conjunctiva was injected.[1]

Dr. Weaks, of Vermont, mentions the case of a child a few days old, to whom two tablespoonfuls of water impregnated with the smoke of tobacco were given for the purpose of keeping it quiet. It died comatose in eight hours, notwithstanding the most active efforts for its resuscitation.[2]

§ 385. Pereira quotes from Dr. Copland an instance in which half a drachm administered by enema proved fatal, other cases in which one and two drachms had the same effect. In one of these, referred to by Dr. Christison, death occurred in thirty-five minutes. Dr. Tavignot witnessed a fatal result in the case of a robust man fifty-five years of age, who took an enema prepared from fifteen grains of tobacco.[3] Dr. Eberle knew the life of a boy destroyed in less than twenty minutes by a tobacco enema.[4] Several instances also are recorded in which the external application of moistened tobacco leaves produced alarming symptoms or death (vide *supra*, § 6). Two also are said by Gmelin to have resulted from excessive smoking, in one case seventeen, in the other eighteen pipes having been smoked at a sitting.

§ 386. The *post-mortem appearances* are by no means characteristic. In a case minutely described by Dr. Grahl, of Hamburg, the only appearances at all unusual were a diffuse redness of the omentum, and of the outer and inner coats of the intestine, and patches of extravasation in some portion of the mucous membrane, together with an empty condition of the vessels of the abdomen and of the heart.

Where a large quantity of snuff has been taken into the stomach, portions of it may remain entangled in the mucus, and thus be recognized either by its physical characters or by chemical analysis, by the detection of its active poisonous principle called *nicotine*. In Dr. Weaks's case, no odor of tobacco was perceived on opening the body.

[1] Dierbach, Neueste Entdeck., ii. 884.

[2] Boston Med. and Surg. Journ., vol. xlvii. p. 461.

[3] Rev. Méd., Nov. 1840.

[4] Therapeutics, p. 389.

§ 387. *Nicotine.*—This alkaloid has much interest attached to it from its having been the poison used by the Count of Bocarmé in the murder of his brother-in-law, Gustave Fougnies.[1] The nicotine was obtained by Professor Stas from the mouth and stomach of the deceased, and from articles of clothing and furniture. It had been prepared by the hands of the murderer himself, who had devoted several months to the study of the process of eliminating it from tobacco. The *symptoms* produced by it in the human system are not well known. Besides the one already referred to, there is but a single case upon record, and in that, also, the symptoms were not witnessed. From the circumstances it was inferred that the person became suddenly insensible and powerless, and died in from three to five minutes.[2] In experiments upon dogs, Orfila observed that vertigo was first produced, that they then sank down, had tetanic convulsions with opisthotonos, and died in a variable time, according to the strength of the liquid. Anhydrous and pure nicotine, he says, may kill a dog in half a minute, but two minutes are usually required. If somewhat less pure and more diluted, death will follow in about ten minutes, and if still further diluted, the animal may recover. The doses given were from one to twelve drops.

§ 388. *Chemical examination.*—Nicotine is an oily, transparent, colorless liquid, becoming brown and thick upon exposure to the air, and, when pure, exhaling a *slight* smell of tobacco. Its taste is acrid and burning. The vapors that are given off, when it is volatilized at 200°, have so strong an odor of tobacco, and are so irritating, that, according to Orfila, it is difficult to bear them. Nicotine is soluble in alcohol, water, ether, and the oils. Its aqueous solution has a strong alkaline reaction, and behaves in many respects like ammonia water when added to solutions of metallic salts, as, for instance, when added in excess to a solution of sulphate of copper, it produces an azure blue color. It is soluble in any proportion in water, and can be partly precipitated from a concentrated solution by potassium hydrate. It is also quite soluble in dilute acids forming salts with the acids. Nicotine exposed to the air and light soon turns yellow and then brown, a resinous substance being formed. It is readily precipitated from its acid solutions by

[1] *Vide* Appendix for a full report of this case.

[2] Times and Gaz., June, 1858, p. 659; Guy's Hosp. Rep., 3d ser. iv. 345.

phospho-molybdic acid, and from its neutral solutions by Mayer's reagent,[1] terchloride of gold, bichloride of platinum, tannic acid, corrosive sublimate, and solution of iodine in iodide of potassium; if an ethereal solution of nicotine be added to an ethereal solution of iodine, an amorphous brown precipitate is first formed, and in a few hours becomes changed into a crystalline one, the crystals sometimes being one-half of an inch in length (Roussin's test).

§ 389. In all legal cases the physiological test should be performed, since the physiological action of poisonous doses has been so thoroughly studied and is so well known. Frogs are preferably selected for the performance of this test, unless quite large amounts of the alkaloid are at our disposal.[2] From $\frac{1}{200}$ to $\frac{1}{100}$ of a drop is sufficient to show the characteristic action in a frog; in a few minutes it becomes uneasy, and then the limbs assume a peculiar position, the fore legs are drawn back and fastened to the sides, and the hind legs are drawn back against the spine, so that the thigh stands at a right angle from the body and the lower leg is flexed, the feet resting upon the spine; at this time respiration is slower, and muscular twitchings occur lasting for one-half an hour or an hour; after this there is great muscular weakness. With larger doses, such as from one-sixtieth to one-tenth of a drop, clonic spasms are produced immediately, and then the above fixed position of the limbs, slowing and even cessation of respiration, considerable diminution of reflex excitability followed by general muscular paralysis which lasts from twenty to forty hours; the action of the heart is at first slowed, then quickened, and finally slowed again.[3]

§ 390. Nicotine can be *isolated* from *organic mixtures* by the same process recommended in the case of opium (see § 348), naphtha removing it from the alkaline solution.

Although nicotine is volatile, it does not readily escape from the body after death, and it can be detected for some time after death. Melsens,[4] states that he has been able to detect it in the body of an animal seven years after death. In cases of tobacco poisoning, the nicotine is most readily detected in the contents of the stomach

[1] See § 344.

[2] Leonides van Praag, Arch. für path. Anat., viii. page 56, 1855. Rosenthal and von Anrep, Arch. f. Anat. u. Physiol., Physiol. Abth., Suppl., page 167, 1879.

[3] Maschka, Gericht. Medecin, ii. page 462.

[4] Gaz. Hebdom. de Méd., i. 1859.

and intestines, but it can also be detected in the solid viscera, as the liver, heart, lungs, and kidneys. Dragendorff and Taylor have both succeeded in isolating it from the blood.

IX. *Conium Maculatum.* (Common or Spotted Hemlock.)

§ 391. *Its action.*—The poisonous properties of this plant reside chiefly in the leaves, but exist also in other parts. The accounts of its action upon the human system are somewhat contradictory. Some authors attribute to it positive narcotic properties; Orfila quotes the case of a soldier, who having eaten of some broth into which hemlock had been put, went to sleep immediately after his supper. A couple of hours later, he was found still lying on the ground in a profound sleep, insensible. His pulse was extremely slow, the extremities cold, the face swollen and livid, and the respiration labored. He died in three hours. Some cases are related in which delirium and fatal convulsions were said to be due to this poison. On the other hand, these symptoms are not produced by coniine, which is supposed to be the active poisonous principle of the plant, nor do they agree with the observations of other authors, especially of recent date. Yet many of the inconsistencies occurring in medical literature concerning the action of hemlock preparations are explicable on the ground of the uncertainty of the proportion of the active principle contained in the medicinal preparations. Dr. Pliny Earle tried the effect of the extract of conium upon himself. The preparation seems to have been a feeble one, for although the dose was steadily increased until it reached sixty grains three times a day, and seven such doses were taken, the effects were by no means striking. No soporific effect, however, resulted; he experienced merely the sensations of fulness of the head and eyes, a "tendency to vertigo," double vision, and a great feebleness in the limbs.[1]

§ 392. *Symptoms.*—Dr. Hosea Fountain, however, who prepared for himself an extract from the fruit or seed of the plant, and took twelve grains of it, began to experience its effects in half an hour. He had a feeling of lightness in the head, dimness of vision, and *muscæ volitantes* before his eyes; "very soon," he says, "a numb, pricking sensation was felt in the fingers, extending

[1] Am. Journ. Med. Sci., July, 1845.

gradually to the elbows, producing a stiffness of the muscles of the parts, making it difficult to move the forearm and hand. In a few minutes the same sensation was observed in the feet, creeping slowly upward, until it reached the upper part of the thigh. The eyes now began to feel uncomfortable, causing me to brush them frequently, to clear apparent obstructions from the lids. The pulse was soft and feeble, but not more frequent than usual." Having dismounted from his horse, he found so much difficulty in walking that he required assistance, and the lower limbs appeared to be nearly paralyzed. This partial paralysis of the limbs continued throughout the whole day, although the head symptoms disappeared under the influence of tobacco and rest. No *soporific* effect was produced.[1] Ordinarily, the symptoms caused by coniine, the active principle of hemlock, may be thus described: When the dose taken is large enough to produce the peculiar action of this drug, the person experiences physical languor and impairment of muscular movements, disturbed vision and dizziness. If he attempts to walk, his limbs feel heavy and numb, and he may totter or fall from muscular weakness of the legs. Quiet with an uncontrollable desire to keep the eyes closed accompany the other symptoms. The individual may also suffer from a headache in the temples or forehead, and disagreeable sensations of the throat amounting to nausea or vomiting. The muscles of the eye are incapable of accommodating vision of remote or near objects. The pupil may nevertheless be natural in appearance in spite of this disturbance of vision (Husemann), but sooner or later it may become expanded or dilated. The pulse-rate is slow at first, but afterwards becomes rapid. Five fatal cases of poisoning by hemlock are recorded in modern literature.[2]

In an undoubted case of death from eating hemlock, the symptoms were very much like those just described. The man's consciousness and intelligence were not affected, but he lost his sight completely, and was unable to walk. He seemed also to have lost all muscular power in his arms, and the power of deglutition and

[1] Am. Journ. Med. Sci., Jan. 1846.

[2] Husemann, Die Pflanzenstoffe, p. 269; Edinburgh Med. and Surgical Journal, 1845; The Sanitarian, June, 1875; Amer. Journ. of Med. Sciences, Jan. 1846; case of F. W. Walker, reported in detail in the New York Times for April 5th and 6th, 1876, *vide* Appendix.

speech; several efforts were made to vomit, but they were ineffectual. His pulse and breathing were perfectly natural, as well as the heat of skin. Death ensued in three hours after eating the poisonous plants, without convulsions, but apparently from paralysis of the heart and lungs. The lungs are paralyzed long before the heart, so that it is often possible to restore the patient and to save his life by prolonged artificial respiration.

§ 393. The *post-mortem appearances* in this case were not important except the presence of numerous extravasations of dark-red blood beneath the mucous membrane of the stomach. The stomach contained a pultaceous mass formed of a raw, greenish vegetable resembling parsley. Its contents weighed eleven ounces, and had an acid and slightly spirituous odor. The hemlock leaves were identified by their botanical characters, and by the peculiar musty odor of *coniine* which was strongly evolved, on bruising some of the leaves in a mortar with a solution of potassium hydrate.[1]

§ 394. The *hemlock water drop-wort* (Œnanthe Crocata) is a still more energetic poison than the foregoing, but from not being medicinally used does not require notice here. Many accidents have happened from the roots of this plant having been eaten by mistake.

§ 395. *Coniine.*—The active principle of common hemlock is a most virulent poison and a local irritant. A single drop applied to the eye of a rabbit killed it in nine minutes; and, when two grains of the muriate of coniine were injected into the femoral vein of a young dog, it died before there was time to note the interval.[2] It produces paralysis almost instantaneously, but does not appear to interfere at once with the functions of the brain, since, according to Christison, the external senses are little, if at all, impaired, until the breathing is almost arrested; and volition too is retained. The blood undergoes no alteration.

§ 396. The effects of conium have been most thoroughly set forth by Dr. John Harley.[3] The following facts are recited by this experimenter.[4]

"The influence of conium appears to be in proportion, not to the *muscular strength* of the individual, but to his *motor activity.*

[1] Ed. Med. and Surg. Journ., July, 1845. J. H. Bennett.

[2] Christison.

[3] The Old Vegetable Neurotics, the Gulstonian Lectures of 1868. Macmillan & Co., London, 1869.

[4] Quoted from p. 12.

"1. The operation of hemlock in the same individual varies in degree according to his motor activity. A dose of conium, which in the ordinary condition of the patient shall be just sufficient to produce the peculiar effects of the plant in a mild degree, will, during the exhaustion following a profuse seminal discharge for example, operate much more decidedly and intensely. Again, the effect will be found to vary in proportion as the activity of the patient varies. Thus, in those whose bodily vigor declines as the day wears away, a dose which will be followed by no appreciable effect in the early morning will produce decided effects in the evening, and *vice versâ*.

"2. Those leading a sedentary, inactive life are more readily affected by conium than those of active habits. A delicate person of active habits will, therefore, bear a larger dose of hemlock than one possessing abundance of strength with but little energy.

"3. An active, restless child will often take, with scarcely any appreciable effect, a dose sufficient to paralyze an adult of indolent habits; and such as would reduce a powerful muscular man to a tottering condition, and force him to assume a recumbent position, and retain it for a quarter of an hour or more. (See cases.)

"4. The same rule applies to children themselves; to produce a given effect, a dull inactive child requires only one-half the quantity that a lively active one does.

"Upon the cerebrum hemlock is powerless. I have induced its full physiological action again and again hundreds of times, in at least a hundred different individuals of all ages, and have never been able to recognize the least *narcotic*, nor directly *hypnotic*, effects. If sleep followed complete repose of the muscular system as a necessary consequence, then there would be no more powerful or direct hypnotic than hemlock. But other conditions over which this drug has no direct control are required to procure sleep. Under the influence of an effectual dose a child often presents the aspect of sleep. . . . But such an event will rarely happen to an adult. His mind will continue during the whole action of the hemlock as calm and active as was Socrates' when he said to his friend, 'Crito, we owe Æsculapius a cock; pay the debt, and do not forget it.' And he will tell us that he feels a strong desire to keep the eyes closed and remain quiet and undisturbed.

"In poisonous doses the eyes will be completely fixed, and the

pupils dilated; articulation and deglutition impossible; expression and all other power of motion gone, and yet, while there is every appearance of the most profound coma, the perceptive faculties and reasoning powers may be as acute as ever. Such a condition is, I believe, not an uncommon one in other states than that produced by hemlock. . . .

"I have observed that persons who use tobacco freely, usually require a large dose of conium to produce its physiological effects; and the reverse, that those cannot tolerate tobacco who are readily influenced by comparatively small doses of hemlock."

§ 397. *Chemical examination.*—Coniine, like nicotine, is a colorless, volatile fluid, which, upon exposure to air and light, becomes darker and darker in color, and is partly transformed into a resinous substance. It has a very peculiar odor, said by many authors to resemble that of the urine of mice; this odor can be perceived by adding a little potassium hydrate to a few drops of the fluid extract of conium. Coniine is inflammable, and much less soluble in water than nicotine (about 100 parts of water); its aqueous solution has an alkaline reaction, and behaves with the metallic salts in the same manner as the aqueous solution of nicotine; if a saturated solution of coniine in water be warmed, it will become cloudy on account of the greater solubility of the alkaloid in cold than in warm water; it is also soluble in alcohol, ether benzole, chloroform, amyl-alcohol, and naphtha; unlike nicotine and the fixed alkaloids, it will coagulate albumen; it is also soluble in dilute acids, forming salts with them; the hydrochlorate may be obtained in the form of crystals which show a beautiful play of colors when examined by polarized light. The most delicate precipitating reagents are solutions of potassio-bismuth iodide, of phosphomolybdic acid, and the solution of iodine in iodide of potassium, the last reagent precipitating coniine from solutions which contain only one part in eight thousand of water (Dragendorff).

§ 398. The *isolation* of coniine from *organic mixtures* is accomplished in precisely the same manner as that of nicotine, and the alkaloid remaining after the evaporation of the naphtha can be tested by the above tests.

Coniine is eliminated very rapidly by the kidneys and can be detected in the urine during life. Dragendorff has succeeded in detecting it in the blood and in the liver also. It withstands decom-

position of the tissues for a comparatively long period, and has been detected in the body of an animal several months after death (Otto).

The following process has been employed by Dr. Harley[1] for the recovery and detection of very small quantities of coniine when mixed with organic matters. "The substance known or suspected to contain conia is comminuted and exhausted, after a few days' maceration in a percolator, by water acidulated with one-fiftieth of its bulk of sulphuric acid; the filtrate is spread out in a thin layer upon flat dishes, and allowed to evaporate to the consistence of a thin syrup in a warm, dry room, or at the distance of three or four feet before a fire; the residue is mixed with an equal bulk of strong solution of caustic potassa (one part to three of water), transferred to a long tube, and agitated with its bulk of ether several times during twenty-four hours. The ether is then decanted, and the alkaline mixture washed again and again with fresh portions of ether, until the conia is completely removed. Two, or at most three, washings are sufficient for this purpose. On distillation of the ethereal solution, the conia, more or less pure, remains. The impure conia is next shaken with a small quantity of dilute sulphuric acid, which separates the alkaloid from oily or resinous impurities. From this solution of sulphate of conia the base is separated in the usual way, viz., evaporation to a syrupy consistence, mixture with caustic potassa, washing the mixture with ether, evaporation of the ether, and finally distillation of the conia in a current of hydrogen, which may of course be omitted when we only want to determine the presence of conia. If spirit be used in the exhausting process, the ethereal extract will be contaminated by much fatty and resinous matters; hence the advantage of using a watery solution. In searching for conia in organic mixtures the same process may be adopted. The contents of the stomach may be digested for a few days with a sufficiently large quantity of sulphuric or oxalic acid, to prevent decomposition, then strained, evaporated spontaneously, and treated as above. Before we conclude that conia is present, we must isolate an oily matter which possesses, in addition to a conia odor, an intensely sharp biting taste, and which dissolves readily, with loss of odor, in a drop of dilute sulphuric acid. We must pour off this drop into a clean tube, and redevelop from it a strong conia odor by the

[1] Op. cit., p. 80.

addition of a little of a strong solution of caustic potassa. If we depend upon the odor alone we may fall into error."

Dr. Harley[1] relates a case in which it was impossible for him to distinguish in urine from patients who had not taken conium, an ethereal extract treated with potassa from an aqueous solution of conium which he used for comparison. He mentions this fallacy in order to show, "that, in examining the animal fluids or tissues for conia, we must bear in mind that the addition of caustic potash to them will often develop an odor indistinguishable from conia, and that nothing short of the isolation of the principle itself should satisfy us."

X. *Gelsemium Sempervirens.* (Yellow Jasmine.)

§ 399. There are five cases recorded,[2] of fatal poisoning by the drug, and several where serious, though not fatal, symptoms followed various sized doses.[3] Its poisonous principle appears to reside in an alkaloid gelsemin, which, with gelseminic acid, has been chemically isolated by Prof. T. G. Wormley.[4] Five to fifteen drops of the fluid extract of gelsemium will cause muscular weakness and languor in most persons, though the susceptibility varies with the individual. After larger and poisonous doses, half to a fluidrachm of the fluid extract, there succeeds to the above symptom certain nervous phenomena, such as dizziness, disturbed vision, and sometimes frontal headache, and these symptoms may be intensified amounting almost to muscular paralysis and blindness; the gait becomes tottering, the eyelids droop or cannot be opened, the eyeballs squint, and the muscular power of speech is lost; the cutaneous sensibility is diminished, respiratory movements and pulse become slower and weaker, a cold perspiration breaks out, and the bodily temperature is lowered. These symptoms may be observed twenty minutes after the taking of the drug, but in non-fatal cases they usually disappear in a very few hours. Convulsions are usually absent in man, though they have been observed in experiments on the lower animals. In one case reported by Dr. Boutelle[3] toxic symptoms appeared in half an hour, and the patient died in less

[1] Op. cit., p. 18.

[2] Med. Rec., N. Y., 1882, 65; Boston Med. and Surg. Journal, July 3, 1879, Dec. 22, 1881, and Amer. Journ. of Med. Sci., April, 1870.

[3] Bos. Med. and Surg. Journ., Feb. 9, 1871; and idem, April 15, 1869.

[4] Am. Journ. Pharmacy, 1870.

than three hours. In the second, reported by Prof. Wormley, that of a pregnant woman, the symptoms did not appear for two hours, and she died in seven and a half hours. In the former case the blood was found after death to be fluid and dark colored, with no tendency to turn red on exposure to the air nor to coagulate, while in the latter, though the blood in the large veins and heart was dark-colored, a fibrinous clot was found in the heart. Since death was probably caused by narcosis and asphyxia, this discrepancy is not remarkable. No other *post-mortem* lesions of any note, except what might be explained on the above hypothesis, were reported. Prof. Wormley infers from his investigations that the woman in this case could not have been poisoned by a dose larger than one-sixth of a grain.

Dr. Seymour, of Troy, reports two cases of fatal poisoning, in one by a teaspoonful of fluid extract of gelsemium in a single dose, and in the other by twenty-drop doses every four hours, amounting in all to two ounces of the tincture of gelsemium, and a final single dose of half an ounce of the fluid extract of gelsemium. This last case he reports[1] in detail. An empiric prescribed[2] a preparation of gelsemium which fatally poisoned his patient.

§ 400. *Chemical examination.*—The detection of gelsemium in cases of poisoning depends upon the isolation of two of its constituents, viz: gelsemine, an alkaloid, and gelsemic acid, as it was called by Prof. Theo. G. Wormley,[3] who first isolated it. Robbins[4] and Schwartz[5] consider, however, that gelsemic acid is identical with the glucoside, æsculin, while Prof. Wormley, after a careful reinvestigation, disagrees with them,[6] stating that it differs from æsculin in regard to its crystallization, its solubility in water, ether, and hydrochloric acid, and its reactions with corrosive sublimate, sulphate of copper, solution of bromine in hydrobromic acid, and in its physiological action.

§ 401. Gelsemic acid is soluble in 2912 parts of water at the ordinary temperature, in 330 parts of ether, and is rather freely soluble in chloroform; it is also soluble in the alkaline hydrates, the solution having a yellow color by transmitted light, but appear-

[1] Bos. Med. and Surg. Journ., Dec. 22, 1881.
[2] Med. Rec., N. Y., 1882, 65.
[3] Amer. Journ. Pharm., 1870, p. 1.
[4] Ber. d. deut. chem. Gesellsch., 1876, p. 1182.
[5] Inaugural Dissert., Dorpat, 1882.
[6] Am. Journ. Pharm., July, 1882

ing greenish-blue by reflected light; nitric acid dissolves it, giving a yellow colored solution, which changes to a deep red when treated with ammonia water; sulphuric acid dissolves it, forming a yellowish solution, which does not change color on warming to 100° C., and from this solution ammonia water in excess will produce a precipitate of white crystalline needles even with exceedingly small amounts ($\frac{1}{10000}$ grain); corrosive sublimate gives a copious yellow precipitate from which large tufts of needles quickly separate; a solution of bromine in hydrobromic acid gives a green precipitate which quickly changes to a blue and finally to a brown; sulphate of copper gives a dirty brown precipitate which soon changes to a dull red color, and needle-shaped crystals separate; acetate of lead gives a yellow precipitate from which star-shaped groups of crystals, which are insoluble in ammonia water but soluble in acetic acid, separate.[1]

§ 402. *Gelsemine* is a colorless, odorless, non-crystalline solid, fusing below 100° C., and having a bitter taste; it forms salts with the acids, most of which are soluble in water and alcohol; it is soluble in 644 parts of water and readily soluble in ether and chloroform; sulphuric acid dissolves it with a reddish or brownish color which gradually changes to a pink, and if "warmed on a water-bath, it acquires a more or less purple or chocolate color;" potassium bichromate or cerium oxide added to this sulphuric acid solution give a reddish-purple color; nitric acid produces with it a brownish-green color which changes quickly to a deep green.[2]

§ 403. The *isolation* of the above principles from *organic mixtures* can be accomplished in the same manner as mentioned in speaking of opium.[3] The gelsemic acid can be removed from the acid aqueous fluid by shaking it either with ether or chloroform, which solvents upon evaporation will leave it in the form of a crystalline residue. The gelsemine can be separated from the alkaline, but not from the acid aqueous solution by shaking with ether or chloroform, which, after separation from the aqueous fluid and evaporation, will leave it as a residue. These residues can be tested by the above tests.

[1] Wormley, Amer. Journ. Pharm., July, 1882.

[2] Wormley, op. cit.

[3] See § 348.

XI. *Atropa Belladonna.* (Deadly Nightshade.)

§ 404. The root, leaves, and fruit of this plant are all poisonous. The berries are black, and have often been eaten by children in ignorance of their poisonous properties. Dr. Taylor states that they were on one occasion openly sold in the streets of London as an edible fruit. Two persons, who had eaten of them, died; and the man who sold them was tried and convicted of manslaughter. A case graphically described by this author will suffice as an illustration of the symptoms produced. "A boy, aged 14, ate, soon after breakfast, about thirty of the berries, which he had bought in the street. In about three hours it appeared to him as if his face was swollen, his throat became hot and dry, vision impaired—objects appeared double, and they seemed to revolve and run backwards. His hands and face were flushed, and his eyelids tumid; there were occasional flashes of light before his eyes. He tried to eat, but could not swallow on account of the state of his throat. In endeavoring to walk home he stumbled and staggered; and he felt giddy whenever he attempted to raise his head. His parents thought him intoxicated; he was incoherent—frequently counted his money, and did not know the silver from the copper coin. His eyes had a fixed, brilliant, and dazzling gaze; he could neither hear nor speak plainly, and there was great thirst; he caught at imaginary objects in the air, and seemed to have lost all knowledge of distance. His fingers were in constant motion; there was headache, but neither vomiting nor purging. He attempted to get out of bed, with a reeling drunken motion; his speech was thick and indistinct. The pupils were so strongly dilated that there was merely a ring of iris, and the eyes were insensible to light. The eyelids did not close when the hand was passed suddenly before them. He had evidently lost the power of vision, although he stared fixedly at objects as if he saw them. The nerves of common sensation were unaffected. When placed on his legs he could not stand. The pulse was 90, feeble and compressible; his mouth was in constant motion, as if he was eating something. His bladder was full of urine on admission. He continued in this state for two days, being occasionally conscious; when, by a free evacuation of the bowels, some small seeds were passed; these were examined and identified as the seeds of belladonna. The boy gradually recovered, and left the hospital on the

sixth day after his admission."[1] Total blindness is not an unusual effect of this poison. It was observed in a child, seven years of age, found wandering in the streets of London.[2] Sometimes the cerebral symptoms are much more aggravated than in the above case, there being frequently delirium or stupor, which, as well as the other symptoms, is slow in passing off. In a case related in the *Lancet*, a lady was given by mistake a drachm of the extract in soap liniment; she fell into a comatose condition in half an hour; the pupils were widely dilated, the hands and feet cold, and the pulse scarcely perceptible. Her jaws were rigid, and there was no vomiting; the stomach-pump was used, and she recovered in a few days gradually. Another instance in which it caused serious symptoms, from having been put into soup instead of caramel, is mentioned in the *Annales d'Hygiène*. The toxical effects of belladonna are produced when applied locally, or introduced into the rectum. A lady, suffering from hypogastric pain, applied to the abdomen a liniment composed of camphorated oil and extract of belladonna. Forty-eight hours after commencing its use she was seized with delirium. The pupils became dilated, and there were irregular movements, lipothyma, redness of the face, and a fixed stare. The menses flowed abundantly, anticipating their proper epoch by ten or twelve days.[3] A fatal case of the employment of an enema of the decoction of the root is recorded.[4] The extract varies very much in strength, and is sometimes quite inert.[5]

The disturbance of vision described in the above paragraph is a symptom peculiar to a class of poisons and drugs called mydriatics, such as hyoscyamus, datura, and duboisia. This and all other symptoms peculiar to belladonna are attributable to its active principle called atropine, and the intensity of the symptoms caused by the preparations of the plant, as well as the plant itself, will depend upon the proportion of the alkaloid present in them.

§ 405. *Atropine.*—About one-sixth of a grain of atropine is capable of producing unpleasant symptoms. A young man poi-

[1] On Poisons, Am. ed., 1875, p. 725.

[2] Lancet, Dec. 1859, p. 561.

[3] Times and Gaz., August, 1859, p. 173.

[4] Casper's Wochenschrift, Feb. 1845, p. 101.

[5] The susceptibility of persons to this poison varies greatly. A dose which would produce little or no disturbance in one individual may cause unpleasant symptoms in another.

soned himself with two grains of this alkaloid. No trace of the poison could be detected in the stomach or intestines. Dr. Andrew, of the Royal Infirmary, Edinburgh, had a patient who was treated by atropine locally to the eye. She swallowed one morning, by mistake, between five and six drachms of the solution, containing about two-thirds of a grain of atropine. She was immediately sensible of her error, her eyesight failed her as well as her voice, the muscles of her face were convulsively moved, and she complained of a burning heat in her throat and stomach. She was very restless, and unable to stand. Although emetics and purgatives were given to her, the next day she was violently agitated and presented the symptoms of a person with delirium tremens. She recovered, but had double vision, spectral illusions, and various disturbances of the nervous system for a week or two.[1]

§ 406. *Symptoms.*—From the many cases of poisoning by belladonna it may be seen that the constant toxic symptoms of this and of its alkaloid, atropine, are dryness of throat, huskiness of the voice, sometimes even amounting to its complete abolition, called aphonia, increased rapidity of respiratory movements, dilatation or expansion of the pupil of the eye, a peculiar red rash on the skin not unlike that of scarlatina, active delirium with talking, feeble and frequent pulse; the surface of the body, especially of the legs and arms, are cold; sleepiness, stupor, coma, and asphyxia, sometimes accompanied by convulsions, precede paralysis and death. Morphine and atropine, given in fatal doses of each, do not antagonize each other so far as the fatal result is concerned, but a fatal dose of morphine may influence the above described symptoms, in some cases the symptoms of morphinism predominating, while in others atropinism may be most prominent; for instance, the pupil may be contracted by the opium or may be dilated by the atropine, yet it must not be forgotten that just prior to death from morphine alone, the pupil is often dilated and not contracted; so too, the effect of increasing the frequency of breathing from the predominance of atropine poisoning may obscure the diminution of these movements which is peculiar to morphine poisoning. The elimination of atropine is by means of the urine, and a few drops of the latter may be used as a test by introducing

[1] Edinb. Month. Journ., Jan. 1852.

it into the eye of an animal, when the pupil will be dilated, if atropine be present. A very characteristic symptom of poisoning by atropine is that before mentioned, viz., paralysis of accommodation of the eye, by which is meant the inability to so change the focus of the lenses of the eye, that the eyesight can accommodate itself to near or remote objects. This paralysis of accommodation lasts as long as the pupil remains dilated, and may be produced by local application of weak solutions of atropine, and this local paralysis will also occur if general absorption of a sufficiently large dose has taken place.

§ 407. Three or four drops of a solution of atropine, containing two-thirds of a grain to the ounce of water acidulated with acetic acid, were put into the eye of a man with double cataract. In half an hour he had vertigo, and shortly after all the symptoms of poisoning with belladonna, flushed face, dilated pupils, and incessant hallucinations. His bladder became distended, and he was unable to empty it. Violent delirium continued during the night. He recovered in four days, these unpleasant symptoms having gradually disappeared.[1] Dr. Bethune has reported a case, in which a few drops of a solution of two grains of atropine to the drachm was applied three times in one morning to the eye. In the afternoon the patient was attacked with delirium, and had an uncertain gait, sleeplessness, and difficulty of swallowing. On the next day but one he had another attack of delirium, which somewhat resembled delirium tremens, as he saw imaginary persons in the room.[2]

§ 408. The *post-mortem appearances* produced by belladonna and atropine are by no means characteristic. Most generally there is a marked redness of the lungs, meningeal membranes of the brain, and of the retina; there has been observed also in animals, hyperæmia at the base of the brain, in the choroid plexus, and in the lateral ventricles. Congestion of the retina is especially noticed after slow or repeated poisoning. In every case this state of hyperæmia is only the first degree of interstitial hemorrhage which is met with quite frequently. (Tardieu.)

The diffuse redness of the gastric mucous membrane should be ascribed to the brandy which has been used as a vehicle for the

[1] Am. Journ. Med. Sci. (from Gaz. des Hôpitaux), Oct. 1853, p. 540.

[2] Boston Med. and Surg. Journ., April, 1857, p. 201.

poison. (Ibid.) Rosenberger[1] relates a case in which the brain mass, the cerebellum, and the spinal cord were the seat of numerous capillary apoplexies.

§ 409. The dose of belladonna necessarily fatal to human life is not definitely known, and will depend upon the amount of atropine actually present in the preparation under suspicion. According to Winckler,[2] the proportion of alkaloid present in the root of belladonna is about one-half of one per cent., and in the leaves it varies from 0.41 to 0.49 per cent.; according to Flückiger and Hanbury,[3] the dried leaves yield 0.44 to 0.48 per cent. of atropine, and the young roots contain larger proportions of atropine than the old roots, the maximum proportion in the former being 0.60 per cent., and in the latter 0.25 to 0.31 per cent.; the alkaloid resides chiefly in the bark. "Manufacturers of atropine employ exclusively the root."

One grain of atropine internally or subcutaneously administered is a dangerous dose, but has not been followed by fatal results. One decigramme (about one and one-half grains) is the smallest fatal dose recorded, although one-tenth of a grain has given rise to dangerous symptoms, and also from three to five milligrammes (from 0.046 to 0.077 of a grain) when injected under the skin. The physiological effects of atropine last for twenty-four hours, and the larger portion of the alkaloid is not eliminated until this time has elapsed, so that when the drug is given in medicinal doses two or three times a day, the amount of alkaloid in the system at any one time may gradually increase, and toxic effects be gradually produced, thus giving rise to what is called medicinal poisoning.

§ 410. *Chemical examination.*—The evidences of poisoning by belladonna depend upon the detection of portions of the plant, such as the seeds and leaves, when such have been taken, in the vomitus and feces, and upon the isolation and recognition of the active principle, atropine, in the vomitus or urine during life, or in the tissues and blood after death.

Formerly atropa belladonna was supposed to contain only one alkaloid, viz., atropine, but recent investigations of Ladenburg[4] have shown the presence of two alkaloids, which he calls heavy and light

[1] Canstatt's Jahresbericht, 1845, p. 295.

[2] Pharm. Journ., 1872, p, 1029.

[3] Pharmakographia, London, 1874, p. 412.

[4] Berichte der deutsch. chem. Gesellsch., xiii. p. 909.

atropine, the heavy atropine being the ordinary atropine, and the light atropine being identical with hyoscyamine, the principal alkaloid of the hyoscyamus niger, which is isomeric with atropine, and their physiological action is also identical. The datura stramonium, hyoscyamus niger and duboisia myoporoides also contain the same alkaloids and have the same physiological action. At the present time in toxicological analyses the ordinary atropine is the only alkaloid which is sought for in cases of suspected poisoning by these plants.

Atropine as commonly seen is in the form of odorless, white, crystalline masses, solutions of which have an alkaline reaction, a bitter taste, and turn polarized light to the left. It is soluble in 300 parts of cold and in 58 of boiling water, from which solutions animal charcoal removes it; it is very soluble in dilute alcohol and amylalcohol, in 33 parts of chloroform and 36 parts of ether; benzol dissolves a large amount at first, but after the solution has stood for a little while, a portion of the atropine separates in the form of long hair-like crystals, the benzol retaining about two and one-third per cent. in solution. Atropine is readily soluble in dilute acids forming salts with the acid, some of these salts being crystalline; concentrated solutions of these salts are precipitated by the alkalies, the precipitate being soluble in an excess of ammonia; in all analyses for atropine, however, the use of the fixed alkaline hydrates (sodium and potassium) and of lime and baryta water should be avoided, since these reagents decompose atropine, tropine and tropic acid being formed; concentrated acids also decompose it. The principal tests for atropine are the peculiar crystalline precipitates produced in solutions of its salts by a solution of bromine in hydrobromic acid, by picric acid, and by terchloride of gold;[1] one of the most delicate precipitating agents for atropine is Mayer's reagent, which gives a precipitate in solutions containing only one part of atropine in 7000 parts of solution, and this reagent has been recommended by Dragendorff for the quantitative estimation of this alkaloid; bichloride of platinum gives no precipitate with solutions of the salts of atropine as it does with most alkaloids; atropine is also precipitated from its solutions by phosphomolybdic acid, corrosive sublimate, tannic acid, potassium bichromate after a

[1] See Wormley, Microchemistry of Poisons.

while, and solution of iodine in iodide of potassium, if the solutions are not too dilute. The best test is, however, the physiological one, which depends upon the readiness with which a solution of atropine introduced into the eye dilates the pupil; in performing this test comparative experiments should always be made, the animal being placed in the same light and kept there, the width of the pupils measured, and a drop or two of the suspected fluid introduced into one eye; in a very short time, if atropine be present, the pupil of that eye will become dilated, while that of the other eye will remain unchanged. If atropine be introduced into the eye of an animal, the pupil begins to dilate in one or two minutes, if it be injected subcutaneously in from five to ten minutes, and if swallowed in from twenty to thirty minutes.

§ 411. If poisoning has resulted from the ingestion of the berries, as is very often the case in children, the seeds can almost always be detected in the contents of the stomach or intestine; these may be recognized by their kidney shape, the horse-shoe-shaped embryo, which is situated in the centre of the seed, their gray color, their size which is about two millimeters long by one and one-half broad, and their rough, uneven surface.

The coloring matter of the berry is also important, and can usually be seen in the contents of the stomach or vomitus, and its presence is additional confirmatory evidence when the seeds have been detected. A substance having a blue fluorescence is also present in the belladonna, but exists in very large amount in the berry; this is soluble in acid fluids, from which it can be removed by rendering alkaline with ammonia and shaking with amylalcohol, and from the amylalcohol it can be removed by shaking with acidulated water.

§ 412. In order to *isolate* atropine from *organic mixtures* the same process may be used as has been recommended in the case of opium and morphine (see § 348), the atropine being removed from the ammoniacal fluid by shaking it with benzol, care being taken to keep the fluid warm, while extracting with benzol, on account of the liability of the atropine to separate from cold benzol in crystalline form.

Atropine withstands decomposition of the animal tissues for a long time, and has been detected in a mixture of organic substances which had been decomposing for two and a half months (Dragendorff).

XII. *Datura Stramonium.* (Jamestown Weed.)

§ 413. Poisoning by this plant is usually accidental and frequently due to the ingestion of the seeds by children. It is a very common plant in this country, being often cultivated in the garden, and is a household remedy for asthma.

§ 414. *Symptoms.*—These are due to its active principle daturine, which has been proved to be identical with atropine, chemically and physiologically.

All parts of the plant are poisonous, but the seeds and the leaves are most frequently employed. In some countries it has been, and is still, used for the purpose of producing intoxication with unconsciousness, in order to facilitate the perpetration of criminal designs. It has been thus given infused in wine or mixed with food. Poisoning by other species of datura is very common in India. During the year 1848, there were treated for it at the Native Hospital, in Bombay, forty-nine males and two females. The powdered seeds are there employed, concealed in rice or other grain. In many cases three stages of symptoms are observed—delirium, sopor, and coma; in others delirium only is observed. The primary delirium may be vociferous or merely garrulous, the patient usually manifesting excessive timidity. In both this and the soporific stage he is constantly engaged in picking at real or imaginary objects, and sometimes in performing such antics as to render laughter on the part even of friends unavoidable. Dr. J. G. Johnson reports the case of a boy in whom the movements were like those observed in chorea.[1] Several of the movements seem to depend upon perverted vision, by which the power of judging of the distance of objects is destroyed from paralysis of the muscles of accommodation in the eye. Husemann observed a case of poisoning by this plant, in which all black objects appeared to the patient green.[2] In other cases there is complete blindness. The pulse and temperature, although usually natural, undergo in some cases extremes of exaltation and depression. On recovery, the person usually recollects nothing since the meal at which he was poisoned, so rapid are its effects.[3] Dr. Duffin, of London, reported the case of his own child,

[1] Am. Med. Times, i. 22.
[2] Journ. f. Pharm., ii. 191.
[3] Brit. and For. Med.-Chir. Rev., Jan. 1851.

two years old, who died in twenty-four hours after swallowing one hundred seeds without chewing them. She became fretful, and like a person intoxicated; in the course of an hour efforts to vomit ensued, together with flushed face, dilated pupils, incoherent talking, and afterwards wild spectral illusions and furious delirium. In two hours and a half she lost her voice and the power of swallowing, evidently owing to spasms of the throat. Then croupy breathing and complete coma set in, with violent spasmodic agitation of the limbs, occasional tetanic convulsions, warm perspiration, and a scarcely perceptible though not frequent pulse. In other cases the pulse was full and slow, and the general symptoms those of ordinary intoxication, with this remarkable exception of the slowness of the pulse. In a case related by Boerhaave, and in others reported in this country, a scarlet eruption appeared on the face.

Accounts of poisoning, one by the infusion of the leaves, and the other from eating the seeds (which have not a disagreeable taste), may be found,[1] in which great stress is laid upon the eruption, "somewhat like that of *urticaria*," which appears as one of the toxic symptoms, and varies in intensity with the severity of the symptoms. In females it has produced nymphomania. Kurzak observed priapism of an hour's duration in a case of poisoning with stramonium seeds.[2] Dr. Bobierre, professor of chemistry at Nantes, drank by mistake a small quantity of an infusion of the leaves and seeds. In a quarter of an hour he began to feel heavy, and had an uncomfortable feeling of constriction in the neighborhood of the larynx. His pupils were dilated, and the secretion of saliva, perspiration, and urine was entirely suppressed.[3] The external application of the bruised leaves may give rise to the symptoms of poisoning. An overdose of the officinal extract has produced fatal effects.

§ 415. The *post-mortem appearances* after poisoning with stramonium leaves or berries present nothing which can be fairly attributed to the poison. In the cases which have been examined there has been but a very slight deviation from the natural condition, and the appearances in no way differ from those of atropine or belladonna poisoning.

[1] Boston Med. and Surg. Journ., Aug. and Oct. 1872, pp. 81 and 240.

[2] Schroff, Pharmacol., p. 532.

[3] Journ. de Chim. Méd., 1851, p. 539; *vide* also Charleston Med. Journ. and Rev., Nov. 1854. (The urine was probably retained and not suppressed.)

§ 416. *Chemical examination.*—The chemical evidence of stramonium poisoning, as in the case of belladonna poisoning, depends upon the detection of some portion of the plant in the contents of the stomach or intestines, or before death in some cases in the vomitus, urine, and feces. When the seeds have been taken, as is often the case by children, they are readily recognized by their kidney shape, the horse-shoe-shaped embryo, their black or very dark brown color, and their size, which is about four or five millimeters in length.

§ 417. The active principles are the same as in the belladonna. The alkaloid, which was formerly called daturine, is now known to be identical with atropine. The experiments of Ladenburg previously referred to (§ 409), have shown the same two alkaloids in the datura stramonium which exist in the belladonna, viz., the heavy and light atropine, or, in other words, atropine and hyoscyamine. The chemical tests and analysis are precisely the same as for belladonna.

XIII. *Hyoscyamus Niger.* (Henbane.)

§ 418. All parts of this plant are poisonous. The root is long, tapering, whitish, and fleshy, and bears considerable resemblance to parsley and parsnip roots, and has been eaten by mistake for them. Dr. Houlton states that, in a monastery where the roots had been eaten for supper by mistake, the monks who partook of them were seized in the night with the most extraordinary hallucinations, so that the place became like a lunatic asylum. One monk rang the bell for matins at twelve o'clock at night; of those of the fraternity who attended to the summons, some could not read, some read what was not in the book, and some saw the letters running about the page like so many ants.[1] Orfila relates two cases in which paralysis, delirium, and insensibility, together with tetanic symptoms, were caused in two soldiers who ate of the young shoots of this plant.[2] The seeds are still more active. Two young children, having eaten some of them, became actively delirious, and even maniacal, striking and biting all who came in their way. Their faces were red, hot, and swollen, and the pupils dilated. They were gradually restored by the use of emetics, local depletion, and

[1] Lancet, July 6, 1844.

[2] Toxicol. gener., ii. 264.

sinapisms to the extremities.[1] Another similar case is related in the same journal, but, the seeds being unripe, the symptoms were still more alarming.

§ 419. The medicinal preparations usually given are the tincture and extract. Both of these vary greatly in strength. The dose of the former is a fluidrachm, of the latter five grains, on an average. The active principle of the drug is represented by its alkaloid, *hyoscyamine*, the strength of which varies with each lot, because it is not usually prepared in a pure state. The medicinal dose of hyoscyamine varies from one-sixtieth of a grain to one grain, according to the purity. The action of both hyoscyamus and its alkaloid resembles so closely that of belladonna and atropine, that reference is made to these articles (§§ 404–406) ; the only marked difference between these two drugs refers to their action upon the brain ; in this respect, hyoscyamus appears to produce more decided impression than belladonna, and especially produces a quieting effect upon the muscular system ; in fact, it has been used without serious consequences in large doses (one-half grain to one or two grains) in insane asylums in England to control voluntary movements. Its action in these large doses prevents insane patients from getting out of bed or walking. The older preparations of henbane were of such uncertain strength, that the effective dose varied from two hundred to two hundred and fifty grains. The alcoholic extract (U. S. Pharmacopœia, 1880) is made from fresh leaves, and is much more efficient.

§ 420. Some preparations of hyoscyamine in the dose of $\frac{1}{333}$ gr. have reduced the pulse from 79 to 18. In medicinal doses it occasions dryness of the mouth and throat, lowers the pulse, dilates the pupils, and induces sleep.

§ 421. Duboisine, an alkaloid from the duboisia myoporoides, of the order of Solanaceæ, is identical with hyoscyamine. Dr. Norris, of Philadelphia, found that dilatation of the pupil occurred in half the time required after a dose of atropine. $\frac{8}{100}$ of a grain administered by injection under the skin was followed by dryness of the throat, and indistinctness of vision, whereas twice this dose quickened the pulse, flushed the skin and impaired muscular movements, differing in this respect from the action of atropine ; it always differs

[1] Henke's Zeitsch., 1848, 4 H. p. 516.

from this by causing placid indifference and inertness, or even faintness, but no drowsiness. It opposes the sweating effects caused by pilocarpin. In one case mentioned by Dr. Norris, an exceedingly minute portion of duboisine sulphate was applied to the surface of the eye where it melted, and a few moments later the patient became dizzy, her face flushed, and she became wildly delirious, very restless, and picked at surrounding objects. According to Wecker, this alkaloid produces less irritation of the eye's surface than atropine. Another difference is shown by the fact that duboisine acts more promptly in paralyzing the muscles of accommodation in the eye. The medicinal dose of duboisine is stated as one-sixtieth of a grain.[1]

§ 422. Dr. Cabot, of Boston, gave three teaspoonful doses of the tincture of hyoscyamus at intervals of an hour. Ten minutes after the last dose the face began to swell, and become red and polished, the eyes were closed, and the patient was able to speak only with the greatest difficulty on account of the tongue and lips. The red discoloration of the skin extended as far as the navel, and was attended with intolerable itching and burning.[2] Delirium and hallucinations, after fourteen grains in divided doses, were met with by Reinbold, of Hanover.[3]

The only two cases of death alleged to have been caused by hyoscyamus were reported, the one in 1715, by Walther,[4] and the other by Lindern, who is quoted at second hand by Orfila.[5] This result may, therefore, be regarded as extremely rare. A case of poisoning by the fruit of hyoscyamus is recorded in the *British and Foreign Med.-Chir. Review* (1859), with symptoms almost identical with those in the case of Dr. Cabot, related above.

§ 423. *Chemical examination.*—The seeds of the hyoscyamus differ from those of the belladonna and stramonium by being of a grayish-brown color and being smaller (from one to one and one-half millimeters long). The alkaloid hyoscyamine is identical with the light atropine and duboisine, and is found in the belladonna, datura, hyoscyamus, and duboisia.

[1] The National Dispensatory, Stillé and Maisch, Phila., 1879, p. 517.

[2] Am. Journ. Med. Sci., Oct. 1851.

[3] Casper's Wochenschrift, 1840, No. 8.

[4] Wibmer, Wirkung, etc., iii. 149.

[5] Toxicologie, 5ème éd., ii. 304.

XIV. *Solanum Dulcamara.*

§ 424. The *bittersweet* or *woody nightshade* (S. dulcamara), is said to possess feeble narcotic properties. There is but little testimony to support this view. The green berries may be poisonous, but it is doubtful whether the ripe berries or any other portion of the plant is dangerous to human life. A case is recorded in Casper's *Wochenschrift*, in which a man took, in one forenoon, from three to four quarts of a decoction made from a peck of the stalks, and was attacked with pain in the joints, numbness of the limbs, dryness of the mouth, and palsy of the tongue, the consciousness was unimpaired, the pulse quiet, but small and rather hard, and the skin cool. The symptoms disappeared under the use of stimulants.[1] Orfila relates an instance in which three children were poisoned by the berries of the S. nigrum, the common garden or deadly nightshade. One of them died, and all of them exhibited symptoms analogous to those produced by belladonna. The supposed active principle of these plants is called solania, or solanine, and is found also in the young shoots of the common potato, S. tuberosum, but not in the tuber itself. In some experiments by Dr. Fraas, the effect of this alkaloid upon animals was very variable when administered by the mouth or rectum. Those in which it was injected into the veins we do not consider as conclusive. Two grains of acetate of solanine injected into the rectum of a rabbit produced heaviness, apathy, *dilatation* of the pupils, convulsions, and death in six hours; but twenty grains of pure solanine given to a pig, and five grains to a dog, produced little or no effect.[2] Schroff distinctly states that this alkaloid has no influence upon the size of the pupil.

§ 425. *Chemical examination.*—The active principle of the solanum is the alkaloid solanine, which can be obtained by the evaporation of its solutions partly crystalline and partly amorphous. It is readily precipitated from its solutions by the alkaline hydrates in the form of a gelatinous precipitate. It neutralizes the acids to form salts which are soluble in water but are not very stable.

[1] Lond. Med. Gaz., Sept. 1850, p. 548.

[2] Brit. and For. Med.-Chir. Rev., July, 1854.

Solanine has a bitter taste. Concentrated sulphuric acid gives a light yellowish-red color, which, after a while, changes to a brown. Fröhde's reagent produces with solanine a cherry-red color which changes to a reddish-brown, then to a yellow, and after twenty-four hours to a greenish-yellow with black flocculi. Solanine can be isolated by Dragendorff's method (see under Opium, § 348).

XV. *Physostigma.* (Calabar Bean.)

§ 426. The *Calabar* or *Ordeal Bean* is the seed of the *Physostigma venenosum*, a shrub growing in marshy localities on the western coast of Africa from Old Calabar to Cape Lopez. It is employed by the natives as an ordeal bean in cases of suspected witchcraft. Its poisonous properties were first investigated by Christison in 1855. In 1864 Jobst and Hesse extracted an alkaloidal substance from the cotyledons, and called it physostigmine. Vée and Leven obtained the same substance in a pure state, and gave it the name eserin; Harnack and Witkowski[1] isolated a second alkaloid, calabarin. The greater part of the seed consists of inert substances, with a small quantity of oil. It is odorless, and almost tasteless.

§ 427. *Symptoms.*—Fraser[2] gives as the first symptom violent pain in the epigastrium soon after ingestion of the poison. Then follow eructation, dyspnœa, giddiness, and muscular weakness. In large doses it has been found to act upon the heart, and cause death by syncope. Large doses also cause cramps in the muscles of the chest, and an irregular, slow pulse. In a minimum fatal dose the heart's action is slowed, and, as the circulation continues, the poison acts upon the spinal cord, until its function is destroyed and asphyxia ensues.

The bean seems to have cathartic and emetic properties. Out of forty-five children who were poisoned in Liverpool by eating the beans,[3] thirty-eight suffered with vomiting, and eighteen with diarrhœa. In these cases, vomiting was the first symptom, and was soon followed by muscular weakness, diminished frequency of the pulse, cold extremities, cold sweat, and *facies Hippocratica*. Contraction of the pupil was observed in a few cases. Christison in

[1] Archiv für exp. Path., 1876.
[2] On the Calabar Bean.
[3] See Appendix.

his experiments, took twelve grains of the bean, and experienced violent symptoms, among which were tumultuous and quickened heart's action, and extreme faintness.

§ 428. *Post-mortem appearances.*—There are no characteristic *post-mortem* changes. The blood obtained from the animals poisoned in Fraser's experiments was generally dark in color; but when drawn from the left side of the heart, after a large dose had been taken, it had the scarlet hue of arterial blood. In dogs and rabbits the red blood-corpuscles were changed in form.

§ 429. *Chemical examination.*—Physostigmine is slightly soluble in water, and easily soluble in alcohol, ether, chloroform, benzole, and amylalcohol. The last three solvents extract it from aqueous alkaline solutions. It is easily soluble in dilute acids and alkalies. Its solutions are colorless, but when exposed to the light, and still more quickly if moderately warmed, they become of a beautiful red color, owing to the formation of rubreserine. It is not precipitated from its solutions by platinic chloride or picric acid. There are no characteristic color tests for physostigmine. The best test is the physiological, which consists in introducing a little of the solution into the eye, contraction of the pupil being the result. The same precautions should be taken as in performing the physiological test for atropine, which causes dilatation of the pupil. Calabarine differs from physostigmine in being insoluble in ether.

§ 430. *Isolation from organic mixtures.*—This is very difficult, on account of the ready decomposition of physostigmine in both acid and alkaline solutions by both heat and light, so that these influences must be avoided as much as possible in performing an analysis in cases of suspected physostigmine poisoning. The method of Dragendorff, as described under opium (see § 348) may be employed. Dragendorff has succeeded in detecting it in the contents of the stomach, intestines, saliva, and blood. By long contact with decomposing substances it becomes destroyed. Dragendorff was unable to detect it in blood which had been preserved for three months after death.[1]

[1] Op. cit.

CHAPTER X.

MISCELLANEOUS POISONS.

I. *Nux Vomica.*

§ 431. *Qualities.*—Nux vomica and its chief poisonous alkaloidal principle may be conveniently treated of together. *Nux vomica* is a round flat seed about three-quarters of an inch in diameter, and two lines in thickness. It is covered with fine, silky, gray hairs, and is hard and difficult to pulverize. The powder is of a yellowish-gray color, and has an intensely bitter taste. *Strychnine*, which is the chief active or poisonous principle of nux vomica, is usually seen as a white crystalline powder, inodorous, but excessively bitter.

This bitter taste is so distinct, that it is said that it may be perceived when only one part of strychnine is present in 30,000, or as others assert, in 400,000 parts of water. It is very sparingly soluble in water, but is easily dissolved by ordinary alcohol when boiling. It is deposited, however, upon cooling. Nux vomica also contains the alkaloid *Brucine*.

§ 432. Strychnine is the most violent in its action of all of the convulsive poisons, and, by comparison of its least poisonous doses with those of brucine, is more than thirty-eight times as energetic, and kills an animal in less than one-third of the period required for the latter alkaloid; as compared with the convulsive properties of the alkaloids of opium, it is twenty-four times more active than thebaïne, fifty times more active than laudanine, eighty-five times more than codeïne, and four hundred times more energetic than hydrocotarnine.[1] This less energetic action of brucine is in part explained (Binz) by its less poisonous effect, and in part, because it is more rapidly eliminated by the kidneys. In a medicinal dose of $\frac{1}{64}$ to $\frac{1}{12}$ of a grain strychnine produces in man only the effects of a bitter stomachic tonic, increasing the flow of urine and of saliva; in larger doses its poisonous action will appear, and has the same characteristics in all the vertebrate animals. This becomes an important fact in medico-legal investigations, since the recovery of the alkaloid from the body of a person, whose death has been caused by this poison, may often be in sufficient amount for use in causing the death of a small animal (a frog, for instance), and thus a physiological test may be added to the chemical proof. The phenomena of its toxical action will depend upon the quantity of poison swallowed and the state of fulness or emptiness of the stomach, since the fasting stomach will absorb more rapidly and larger amounts of the poison; occasionally an instance may occur, in which an individual may resist a dose which would be poisonous to the generality of persons. The first evidence of the absorption of a dose dangerous to life is an increased activity of all the senses, such as restlessness of mind and muscles, increasing agitation, sensations of itching, exaggerated sensibility to touch, the eyes showing an intolerance or increased sensibility to light, an increased sensibility to odors and

[1] Falck, Vierteljahr. f. gericht. Med., 1874, p. 195; and Ann. d'Hyg. pub. et de méd. lég., 1876.

taste. Soon after these early signs of nervous exaltation, muscular stiffness accompanied by twitchings will appear, with unsteadiness in walking; the muscles of the jaw are usually the last to be attacked with this stiffness or rigidity, which is often painful, and the temporal muscles, as well as those of the neck, are the seat of little convulsive movements. If the dose be large enough, these symptoms, which usually come on within the first half hour, are followed by more serious as well as severe muscular contractions of the whole body, and the body will be stretched out stiffly with the back arched, and supported sometimes only by the head and heels (opisthotonos), frothy mucus collects about the mouth, the face is pale, articulation interrupted and difficult, the jaws locked with the tongue sometimes between the teeth, *while intellectual activity is perfectly intact;* the limbs become agitated with violent and noisy contractions, which in criminal cases is often compared to the clapping of castanets (Tardieu, case of Pégard). These various phenomena of convulsions, after a longer or shorter continuance, culminate with the characteristic tetanic spasms; the muscles become rigid and violently throw the limbs into forcible contraction, the sole of the foot is turned inwards, and the muscles of the face are distorted in horrible and anxious grimaces. The victim will complain of these painful contractions, and make vain efforts to raise himself, make inarticulate efforts to cry out, and experience a most lively thirst. In men there are painful erections of the penis, and in women corresponding sexual sensations.[1] Respiration is difficult from the muscular rigidity of the thoracic muscles, the pupils are dilated, the neck is swollen, the lips are blue and cyanotic from the tendency to asphyxia, and at the moment of impending death respiration slowly reappears, the lips open, the victim apparently revives, and, if the dose be large enough, the sequence of symptoms will be repeated; but if recovery is to take place, the spasms return with diminished force, and all convulsions will ordinarily cease in a few hours, leaving an intense muscular fatigue, a moral and physical languor, with increased excitability to reflex irritations, and often with a muscular stiffness which may persist for a longer or shorter time. Death takes place from asphyxia caused by the tonic contraction of the thoracic

[1] Trousseau and Pidoux, Traité de Thérapeutique.

muscles, in the most violent paroxysmal convulsions, or from exhaustion during the interval after the fourth or fifth paroxysm. The paroxysms usually last from one to five minutes, and the intervals from five to fifteen minutes; in cases which terminate in recovery, the paroxysms become less and less severe, and the intervals longer and longer, while the reverse is true in those cases which end fatally. Death usually takes place in adult human beings during or after the fourth or fifth paroxysm, but children may die during or after the first, second, or third convulsion. This is an important point in medico-legal cases in distinguishing between the convulsions of strychnine poisoning and uræmic or puerperal convulsions (see § 437). During the intervals, except in rare cases during the fourth, the patient is able to converse intelligently, but a paroxysm may be caused by any unusual jar, such as the shutting of a door, or movement, or even by an attempt to swallow food or drink. The excessive bitter taste, the rapidity of the onset of the symptoms, the rapid progress of the case to a fatal termination or to recovery, the limited number of the convulsions, and the normal condition of the intellect during the interval between the convulsions are the characteristic clinical features of a case of poisoning by nux vomica or strychnine.

§ 433. The rapidity with which the symptoms of poisoning may supervene will depend, in addition to the conditions above related, upon the method by which the poison is introduced. Absorption rapidly follows the introduction by rectal injection, by insufflation into the air passages, by contact with interior surfaces, the serous membranes for instance, and by the subcutaneous cellular tissue; but the most rapid absorption takes place when strychnine is injected into a vein, symptoms appearing in one-third of the time required for its absorption from the stomach. Absorption may also take place from the bladder as rapidly as from the stomach.[1] According to Shuler, the mucous surface of the eye is equally active in power of absorption. One to three grains of strychnine placed upon the inner angle of the eye and left there will rapidly destroy the life of a man. As this poisonous agent is eliminated with the urine, its presence should be detected in that secretion by chemical

[1] Ségalas, Journal de Magendie, 1822.

analysis. According to Moller's experimental researches[1] strychnine may be detected within three minutes in the saliva of an animal, which has received a subcutaneous dose of this poison, while in fifteen minutes all proof of its presence in that secretion disappeared. These experiments were confirmed in certain patients into whose subcutaneous tissue Moller injected about $\frac{1}{10}$ of a grain; but, according to Dragendorff, it is slowly eliminated, in dogs not until several days after its absorption. If this be true with men, it would not be safe to gradually increase or even continue the doses usually considered within the range of medicinal usage, and which the daily experience of medical practitioners shows to be absolutely safe. Leube and Rosenthal oppose these statements of Dragendorff. Recent experiments of Kratter[2] show that after the subcutaneous use of $\frac{1}{10}$ of a grain of strychnine, this alkaloid is found in the urine on an average within an hour, and its elimination ceases within forty-eight hours. Strychnine may be absorbed by the stomach within ten minutes.

§ 434. A case of accidental poisoning with strychnine has been described by Mr. Bennett;[3] the quantity taken was about a grain and a half in solution. The patient when first seen, which was about an hour after the poison had been taken, was in a rigid and trembling state, and the face almost maniacal in its expression. This was soon followed by a violent tetanic convulsion. Between the fits she did not utter any expression of alarm, but would occasionally request a little cold water. The muscles of the jaws remained so rigid between the spasms, that the attempt to introduce the stomach pump was unsuccessful; and although some strong emetics were got down, it repeatedly happened that the attempt of the patient to take liquids was followed by so violent a spasmodic fit as to prevent her swallowing them, and to give that apparent dread of water so well marked in cases of hydrophobia. During the tetanic fits the whole body was stiffened and straightened; the neck violently drawn back, the chest fixed, the eyes protruding from their sockets in a horrible manner, the legs pushed out and widely separated, the muscles of the face convulsed, pulse imperceptible, and

[1] Ugesk. for Läger. R. 3, Bd. xix. p. 161; quoted in Revue des Sciences Médicales, 1876, t. viii. p. 477.

[2] Untersuchungen über die Abscheidung von Strychnine durch den Harn, in Wiener med. Wochensch., 1882, p. 214.

[3] Lancet, 1850, vol. ii. p. 462.

no breathing could be perceived; the face was livid, more particularly the lips, and froth issued from the mouth. The pupil was also dilated during the paroxysm. It was impossible to produce any relaxation of the body during a fit, and, if moved, the whole body maintained its rigid condition. As soon as death had taken place, which was in an hour and a half, the limbs relaxed, the face and lips gradually lost their livid hue, and became, as well as the body, extremely pallid.

In this case alarming symptoms did not arise until about an hour after the poison had been taken, and numerous cases might be referred to in which an interval of an hour or two occurred. The poisonous effects of strychnine are more rapidly developed than those of nux vomica. But usually the former supervene very speedily, being seldom delayed more than fifteen minutes. Death may occur in twenty minutes, as in the case of Dr. Warner, who was supposed to have taken only half a grain,[1] or in half an hour, as in a case reported by Dr. Theinhart, where thirty grains of strychnine were swallowed, and seldom is postponed for more than two hours, if no measures for the removal of the poison have been taken.[2] In a case that occurred near London, a prescription was improperly prepared, so that the young man, for whom it was directed, took a grain and a half of nux vomica, and the same quantity of strychnine. It is stated that "he soon afterwards complained of some extraordinary sensations, and *almost immediately* expired."[3]

It is hardly worth while to enumerate the symptoms of poisoning in the many recorded cases, for details of which the reader is referred to the Appendix, or to any of the references in the foot-note.[4]

[1] Phila. Med. Exam., Oct. 1847, p. 309.

[2] Am. Journ. of Med. Sci., Jan. 1848, p. 303, from Gaz. Médicale.

[3] Am. Journ. of Med. Sci., April, 1854, p. 537, from Pharm. Journ., July, 1852.

[4] Caussé et Bergeron, Contribution à l'étude de l'empoisonnement par strychnine, suivie de l'exposé de l'affaire Toulza, dit Rapala, et de la discussion Medico-légale, dont elle a été l'objet, Ann. d'Hygiène, Paris, 1878, pp. 272 314. Bridges, Poisoning by strychnine, Chicago Med. Journ. and Exam., 1879, p. 48. Rowbotham, Strychnine poisoning, Med. Times and Gazette, London, 1879, i. p. 591. Chaplain, A case of strychnine poisoning, Med. and Surg. Reporter, Philadelphia, 1879, p. 196. Idem, p. 525. Arantham, Case of strychnine poisoning, Southern Clinic, Richmond, 1878, p. 1427. Beates, Poisoning by nux vomica, Medical Gazette, N. Y. 1880, p. 34. Jackson, Fatal case of poisoning by strychnine, Australian Med. Journ., Melbourne, 1880, ii. p. 54. Foucart, Empoisonnement par gouttes

§ 435. *Power.*—The smallest quantity of *nux vomica* which is known to have caused death, is said to be three grains of the alcoholic extract, but it is quite uncertain to how much of the powder this corresponds. Hoffmann (quoted by Christison) states, that two doses of the powder, of fifteen grains each, proved fatal, and other cases are related in which fifty and sixty grains produced death. The smallest quantity of *strychnine* which has proved fatal appears to be one grain.[1] In Mr. Bennett's case, above quoted, about one grain and a half was taken.

Recovery occasionally is observed, even after very large doses. Dr. Thomas Anderson records a case in which seven grains of strychnine were taken without producing fatal consequences.[2] The comparative mildness of the symptoms, and the recovery throw some doubt upon the purity of the strychnine which was used. Dr. Dresbach, of Ohio, attended a man who drank, by mistake, three ounces of a solution of strychnine, containing one grain to the ounce. When seen by Dr. D., about twenty minutes afterwards, he was in the following condition : The whole muscular system rigid, the muscles of the back and legs so rigidly contracted, that it was with extreme difficulty that the man was able to walk, face drawn awry and articulation impeded, a sense of burning about the stomach, tightness about the chest, vertigo, and dimness of vision, lower extremities cold, and perspiration abundant. Chloroform being the only article at hand which seemed likely to be useful, Dr. D. gave the patient at once two drachms, and in less than fifteen minutes the relief, Dr. D. says, was complete.[3] A case, in which a man recovered after swallowing a grain and a half, is reported by Mr. Foster.[4]

amères de Baume (made by digesting St. Ignatius beans in alcohol, about five parts in ten). France Médicale, 1880, p. 498. Richet, D'une mode d'asphyxie dans l'empoisonnement par la strychnine, Acad. des Sciences, Paris, 1880, p. 443 ; Bos. Med. and Surg. Journal, July, 1877, p. 43. Kelly, A case of strychnine poisoning, Nashville Med. and Surg. Journ., 1882, p. 26. Harrison, A case of poisoning, illustrating the antagonism between strychnine and morphine, Lancet, 1882, p. 185. Lefort, De la substitution accidentelle de la strychnine à la santonin dans la pharmacie, Bull. de l'Acad. de Méd., Paris, 1881, p. 460. Delaunay, Influence de la nutrition sur l'empoisonnement par la strychnine. Comptes Rend. de l'Acad. des Sci., Paris, 1881, p. 432.

[1] Med. Times and Gaz., April, 1855.

[2] Am. Journ. of Med. Sci., April, 1848, p. 562.

[3] Am. Journ. of Med. Sci., April, 1850, p. 546, from Western Lancet.

[4] Lancet, 1852, vol. ii. p. 198.

In another case, a girl swallowed two grains of strychnine upon an empty stomach. The poison remained in the stomach fifty minutes before it was removed by an emetic and the stomach-pump. The disturbance was slight, and the girl recovered. Dr. Taylor suggests, that, owing to the symptoms having been very slight in this case, the strychnine was probably not pure.[1] But recently other cases have occurred of recovery from still larger doses, so that it seems more probable in these cases that the absorption is delayed. In one, four grains were taken by mistake. Copious vomiting was produced by emetics in about a quarter of an hour, but the system was violently affected, there being not only excessive tetanic rigidity of the muscles, but frequently recurring convulsions, with other symptoms already detailed. The man recovered entirely in two or three days.[2] In another case, by Mr. Chippendale, a man who had been in the habit of using small doses for an imaginary spermatorrhœa, took four grains of strychnine and four of morphine in an ounce of spirit, with the intention of destroying himself. Tetanic spasms ensued in about half an hour; but in this, as in the preceding cases, the intellect remained unaffected—a fact the more remarkable on account of the large quantity of morphine which had been swallowed. The latter produced none of its peculiar effects, except, perhaps, an itching of the skin, which occurred in the convalescence, and might perhaps be ascribed to it. The man recovered perfectly. The stomach-pump was used one hour after the poison had been taken, and water and animal charcoal were injected into the stomach.[3] A case is reported by Dr. Bly, of a man who, by mistake, took four grains of strychnine at a dose. Tartarized antimony was immediately administered, but ineffectually, and tetanic convulsions and locked jaw succeeded. These symptoms were palliated by chloroform inhalations, so that an additional dose of tartar emetic could be administered. In thirty-five minutes after the poison had been swallowed the emetic operated freely, and the anæsthetic inhalations having been continued, the threatening symptoms ceased in the course of seven or eight hours.[4] In this case it is probable that the

[1] Med. Times and Gaz., Ap. 1854.

[2] Ibid., Ap. 1855. G. Hinnell.

[3] Ibid. See, also, case of compound poisoning mentioned previously.

[4] New York Journ. of Med., Nov. 1859, p. 422.

tartar emetic taken immediately after the poison hindered the absorption of the latter. Mr. Iliff has reported a case in which a female recovered after swallowing two drachms of powered *nux vomica*.[1]

The attention of the reader is again called to the question of compound poisoning, which has previously been discussed, § 34. The peculiar action of strychnine being exerted upon the spinal cord and the voluntary muscles, will of course be antagonized by other energetic drugs which have a more or less paralyzing action upon this nervous centre, such, for instance, as chloroform, chloral-hydrate, morphine, and belladonna; and yet it will be gathered from what has been said in reference to the rapid and energetic action of strychnine, that these antagonists, whose absorption is slower, will not be likely to control the symptoms of its poisonous action, unless previously or simultaneously administered, except in the case of chloroform. Since death is caused by the continued contraction of the thoracic muscles producing asphyxia, provided this latter effect be prevented by an antagonistic drug for a sufficient time after the administration of strychnine, it will be reasonable to assume that fatal strychnine poisoning will be averted. In this connection what has been mentioned in regard to the elimination of the poisonous agent becomes of the highest importance, viz., that the total elimination usually is accomplished within forty-eight hours, and begins within the first hour.

§ 436. *Poisoning by strychnine compared with tetanus.*—It is not always an easy matter for a physician to distinguish between the effects of poisoning by strychnine and a disease called tetanus. A striking symptom in poisoning by strychnine consists in the sudden appearance of the convulsive spasms, and the rapidity with which the attacks and remissions succeed each other, and the short space of time which intervenes between the access of the spasms and a fatal result. We may also mention that the intervals between the spasms are marked by an absolute calm. In tetanus the rigidity of the affected parts is generally permanent, and the access, which is more prolonged than in poisoning, has more the character of paroxysm and of exacerbation rather than succession of attacks. Moreover, the fatal result never happens in tetanus in two or three hours after the access of spasms, as has been noticed in strychnine

[1] Lancet, 1849, Dec. 15.

poisoning. In the former the duration is generally from two to ten days, and in every case exceeds that of strychnine poisoning, if death supervenes.[1] In tetanus, some cause for the disease, such as a wound, can almost always be detected. The facts that in tetanus the symptoms progress slowly, and several hours elapse before a severe convulsion occurs, while in strychnine poisoning usually less than one hour elapses; that in tetanus the jaw is the first to be affected, and the lock-jaw is constant, while in strychnine poisoning, in those cases in which any difference at all can be perceived, the jaw is the last to be affected; that the duration of the symptoms is numbered by days in tetanus, and by minutes or hours in strychnine poisoning; and that the number of convulsions in strychnine poisoning is limited to four or five, will usually enable the physician to distinguish between the two conditions without difficulty.

§ 437. *Poisoning by strychnine compared with puerperal convulsions.*—It rarely happens that a pregnant woman becomes the victim of criminal poisoning by strychnine; when such a case does occur, the question, as to whether the convulsions were due to the pregnant condition or to strychnine poisoning, is sure to arise. The writer is familiar with two cases in which this question arose during the criminal trial, the Hersey case,[2] and the Major case (see Appendix). The following are important points to bear in mind in making a differential diagnosis between the two conditions. (1) When due to the puerperal condition the number of convulsions is usually more than five. (2) In puerperal convulsions, the patient has no recollection of what has occurred, either during the convulsion, or during the intervals, the entire period during which the convulsions took place being a blank, whereas in strychnine poisoning the intellect is intact, except, perhaps, during the last interval and convulsion. (3) Death usually takes place much more quickly after the first convulsion in strychnine poisoning than in puerperal convulsions or eclampsia. (4) When death occurs as a result of puerperal convulsions, it very rarely takes place before the birth or delivery of the child (only a very few cases having been recorded), whereas in strychnine poisoning, this event makes no

[1] *Vide* an excellent account in Compendium de Chirurgie Pratique, A. Bérard et Denonvilliers, Paris, 1841, t. 1er, p. 350.

[2] Case of Geo. C. Hersey, indicted for the murder of Betsy F. Tirrell, Boston, A. Williams & Co., 1862.

difference, but in the two cases with which the writer is familiar, death occurred before the birth of the child. (5) In case of death due to puerperal convulsions no strychnine would be detected by chemical analysis.

§ 438. *Strychnine poisoning compared with epilepsy.*—As a rule, this question would never arise, because the previous medical history of the patient would be known, and because recovery almost always takes place in the case of epileptic patients who are able to be away from home without an attendant, if a convulsion happens to occur at such a time. In case, however, that death does occur during an epileptic paroxysm, the fact that only one convulsion was observed and no lucid intervals, will enable the physician to eliminate strychnine poisoning as the cause of death; if no distinct history of the convulsion can be obtained, the failure to detect strychnine in the body after death will eliminate it.

§ 439. The *post-mortem appearances* are by no means characteristic, though certain negative results can be drawn therefrom, which are not without value when considered in relation to the symptoms observed during life. It may be difficult to prove whether the pathological peculiarities found at the autopsy of persons poisoned by strychnine may not have been due to some other condition. No notice should be taken of those cases in which there is no appreciable trace of pathological alteration of the organs; though this absence of lesion is not without value.[1] The most significant pathological lesions would be found in the nervous centres, but it must also be remembered that there are certain diseases in which identical changes have been observed. (Abercrombie.)

To some extent the degree of rigidity of the body, and the permanence of this condition, are peculiar. Reference is asked to what has been mentioned in a previous chapter on this subject (§ 11). Until the researches of Brown-Séquard and others during the last few years it was supposed that this was a pathognomonic condition, but it seems now to be pretty well established that the condition of cadaveric rigidity depends upon a certain peculiarity of the muscular system, and that, when death has followed the use of convulsive poisons, the early and prolonged condition of the rigor

[1] Tardieu, op. cit., p. 940.

mortis is due to the effects of the convulsions upon the muscular system. Now it is evident, that in any case of death which has been preceded by violent muscular movements, this same early appearance of cadaveric rigidity may occur; whilst, on the contrary, if the convulsions have been prevented by the use of medicaments, or perhaps by artificial respiration,[1] this *post-mortem* peculiarity might be absent.[2] In the case of Cook, poisoned by Palmer, Dr. Harland testified that the body was very stiff, more than dead bodies generally are. The muscles were very highly developed; they were strongly contracted and thrown out. The hands were firmly closed.[3] In another case, reported by Mr. Wilkins, it is stated that seven hours after death the rigidity of the body was so great as to allow it to be lifted by the heels; it is described as being "as stiff as wood."[4] In some cases there have been found signs of inflammation in the intestinal canal, and very generally congestion of the brain and its membranes, and sometimes softening of its substance and of the spinal marrow. The right cavities of the heart are usually contracted, and the blood dark and fluid. In some instances, no doubt, these appearances were due to cadaveric changes, and were not the result of any peculiar influence of the poison. In the Major case (see Appendix) the cadaveric rigidity was very marked two weeks after death, when the body was exhumed by the writer. If death takes place during a convulsion, as is commonly the case, while the action of the respiratory muscles is suspended, there will be found venous congestion in all parts of the body, but this appearance is not, of course, characteristic of strychnine poisoning.

§ 440. *Chemical examination.*—*Strychnine* occurs mostly in the form of prismatic crystals belonging to the rhombic system, when allowed to crystallize slowly, but when it separates quickly from concentrated solutions, the crystals are needle-shaped. It is soluble in about 7000 parts of cold and 2500 parts of boiling water, but is readily soluble in alcohol, amylalcohol, benzol, naphtha, and chloroform; the latter solvent dissolves it the most readily, taking up

[1] *Vide* an article on Hydrocyanic Acid, in London Practitioner for April, 1872.

[2] Croonian Lecture, by Prof. Brown-Séquard, in Proceedings of the Royal Society, May 16, 1861. Also Amer. Journ. of the Med. Sciences, Jan. 1870, pp. 87 *et seq.*

[3] Times and Gazette, May, 1856.

[4] Guy's Hosp. Reports, 3d ser. iii. 484.

nearly twenty per cent. of its weight; it is but slightly soluble in ether; it is readily soluble in dilute acids forming salts which are readily soluble in both water and alcohol. If strychnine is gently heated, it volatilizes unchanged, and can be condensed in the form of a sublimate upon a glass microscope slide or upon the inner surface of a glass tube; this sublimate assumes the characteristic crystalline form if touched with a drop of water. All solutions of strychnine have an exceedingly bitter taste which can be perceived, if a single drop of a solution containing only one part of the alkaloid in 100,000 parts of water be introduced into the mouth, and, according to some authors, it can be perceived when the solution contains only one part in 420,000. It is the most bitter substance known, and it is very difficult to disguise the bitter taste, so that it can only be administered criminally by making the victim believe that he is taking some very bitter medicine; the writer is familiar personally with two instances in which an attempt was made to poison several persons by introducing strychnine into tea, but unsuccessfully, since the introduction of the first teaspoonful into the mouth showed, by the intense bitterness, the presence of some unusual substance, and, if swallowed, the amount taken was only a small medicinal dose. Strychnine is readily precipitated from its solutions in acids by ammonia water, usually in crystalline form; this test (and the same is true of all of the other tests which produce precipitates) is best performed by placing a drop of the solution upon a glass slide, adding a drop of the reagent and stirring the mixture with a glass rod; the crystals formed can then be examined with a microscope. Strychnine treated with a drop of concentrated sulphuric acid forms a perfectly colorless solution which, if stirred with a crystal of potassium bichromate, passes through a beautiful play of colors; first a purple color is formed immediately; this changes quickly to a violet, then to a red, then to a yellow, and finally the mixture becomes colorless, unless too much of the bichromate has been added. This test gives about the same play of colors with curarine, but the red is produced much more quickly and is much more permanent than in the case of strychnine; moreover, in legal cases there is no danger of confounding these two substances, since they are separated from their solutions at different stages of the process for the isolation of organic poisons (see § 348). Strychnine is precipitated by a solution of potassium bichromate in

the form of golden-yellow groups of crystals made up of an aggregation of needle-shaped crystals; this precipitate can be obtained with $\frac{2}{100}$ of a milligramme (less than $\frac{1}{3000}$ of a grain). A solution of iodine in iodide of potassium gives a brown amorphous precipitate, which is soluble in boiling alcohol and separates from it upon cooling in the form of prismatic crystals; this test is delicate for $\frac{12}{10000}$ of a milligramme (less than $\frac{1}{50000}$ of a grain). Picric acid gives with a solution of strychnine a golden-yellow precipitate, which is amorphous at first but gradually becomes crystalline, the crystals having an irregular but peculiar shape; this test is delicate for $\frac{5}{100}$ of a milligramme (less than $\frac{1}{1000}$ of a grain). Ferricyanide of potassium gives with a solution of strychnine an amorphous precipitate, which quickly becomes converted into groups of greenish crystals having a very peculiar form. Corrosive sublimate also produces a precipitate which is amorphous at first, but which soon becomes converted into groups of colorless prismatic crystals. Mayer's reagent gives a precipitate at first amorphous and then becoming crystalline. Terchloride of gold and bichloride of platinum also yield crystalline precipitates with a solution of strychnine. Valuable negative tests are, that neither Fröhde's reagent nor concentrated nitric acid give a colored solution with *pure* strychnine. The physiological test should never be omitted in cases of strychnine poisoning which are investigated legally; an exceedingly small amount of strychnine given hypodermically to a frog will produce the tetanic spasms; care should be taken not to give too large an amount, lest immediate death be produced; $\frac{1}{5000}$ part of a grain will usually suffice to produce the effect after a few minutes in a frog.

§ 441. *Brucine* crystallizes in the form of oblique rhombic prisms which are more soluble in water and alcohol than strychnine; it is insoluble in ether, but soluble in benzol, chloroform, naphtha, amyl-alcohol, and the dilute acids; alkalies precipitate brucine from its solutions in acids usually in the form of an amorphous precipitate which gradually becomes crystalline; it is quite soluble, however, in an excess of ammonia water. Concentrated nitric acid, or concentrated sulphuric acid, which contains a trace of nitric acid, imparts to brucine a beautiful orange-red color which is changed by a solution of stannous chloride to a reddish-violet; this test is delicate for $\frac{1}{100}$ of a milligramme (less than $\frac{1}{6000}$ of a grain). It is pre-

cipitated in an amorphous form, the precipitate gradually becoming crystalline by Mayer's reagent, potassio-cadmic iodide, bichloride of platinum, picric acid, and potassium bichromate. Pure concentrated sulphuric acid and Fröhde's reagent impart to it a pale rose color. It has a very bitter taste, but not as bitter as strychnine, and it is not nearly as active physiologically as strychnine. According to Sonnenschein[1] it can be converted into strychnine by the action of oxidizing agents such as nitric acid, and *vice versa*, strychnine can be converted into brucine by the action of reducing agents such as sulphide of ammonium.

§ 442. The *isolation* of these alkaloids *from organic mixtures* can be accomplished by the same process as recommended in the case of morphine (see § 348), both the strychnine and the brucine being separated from the alkaline solution by shaking with naphtha, benzol, or chloroform. Benzol removes them better than naphtha, and upon evaporation leaves the brucine as an amorphous residue near the edge of the watch-glass, and the strychnine near the centre as a crystalline residue. Chloroform removes them better than either naphtha or benzol, and upon evaporation leaves both in the form of a crystalline residue. These residues can be tested by the above tests, the precipitating reagents being applied to a solution of the residue in water to which a little acetic or sulphuric acid has been added.

§ 443. If both strychnine and brucine are detected, the inference is that the form of poison used was nux vomica or one of the pharmaceutical preparations of nux vomica, while, if either alkaloid alone were present, the source of poisoning must have been that alkaloid or one of its salts. Strychnine withstands the decomposition of animal tissues for a very long period, and has been detected in the body more than a year after death.[2]

The following process for the detection of strychnine in mixed fluids was devised by Prof. Graham and Dr. Hoffmann, for the purpose of testing the presence of this poison in the bitter ales of Burton.[3] It may also be applied to other liquids. Two ounces of ani-

[1] Bericht. d. deutsch. chem. Gesellsch., 1875, No. 4.

[2] See Magoon case. Appendix.

[3] This inquiry was instituted at the invitation of the English brewers of ale who were naturally indignant at the assertion made in a lecture by M. Payen of Paris, that strychnine was there prepared in large quantities, for the purpose, as had been ascertained by the

mal charcoal are to be shaken in about half a gallon of the suspected fluid, and this is to be left at rest for a night, and then filtered through paper. The fluid is thus deprived of its bitterness. The charcoal which contains the strychnine is then to be boiled for half an hour in eight ounces of rectified spirit, and the spirit after being filtered, is concentrated by distillation. The remaining liquor, which is watery, is next decomposed with a few drops of a solution of potash, and agitated with an ounce of sulphuric ether. The ether contains the strychnine in a state of considerable purity, and, on being evaporated, it deposits a white soluble matter, of intense bitterness. If a drop of sulphuric acid be placed upon this residuum, and then a fragment of bichromate of potassium, in the resulting liquid a beautiful violet tint appears at the points of contact, and soon spreads over the whole fluid. This change of color seems to be characteristic of the alkaloid strychnine. The discoverers could detect with it half a grain of strychnine in half a gallon of the pale ale (of Allsop and Son), into which it had been purposely introduced. These gentlemen attest that, after analyzing a large number of samples of pale ale taken indiscriminately from the supplies of various manufacturers, *not one of the varieties of beer*, when tested with the greatest scrupulousness, gave the *slightest evidence of the presence of strychnine.*[1]

II. *Hydrocyanic Acid, or Prussic Acid.*

§ 444. *Qualities.*—The extreme energy of this poison in small doses is well known. The medicinal or dilute acid directed by the United States Pharmacopœia contains two per cent. of anhydrous acid. Very nearly the same proportion exists in the formulas of the British Pharmacopœias. Scheele's acid, for medicinal use, should contain five per cent. of anhydrous hydrocyanic acid, but as sold it is said usually not to exceed the strength of two per cent. The dilute hydrocyanic acid is a transparent, colorless, volatile liquid.

Its *taste* is described by Dr. Christison as acrid and pungent, and

French authorities, of being sent to England, to be there employed in the manufacture of the celebrated bitter beer of that country. *Vide* Adulterations detected in Food and Medicines, A. H. Hassell, London, Longman, Green, Longman, and Roberts, 1861, p. 617.

[1] Med. Times and Gazette, May, 1852.

by others as hot or bitter; but it is probable, as remarked by Dr. Taylor, that the taste may be unperceived, when the dilute acid is taken in a fatal dose, concealed in porter or medicine.

Its *odor* is popularly supposed to resemble that of bitter almonds, but this notion is incorrect. It may have something of this odor, sufficient perhaps to recall it, and this peculiar smell may be recognized by some persons and not by others, but the impression usually made by it is indistinct, with the exception of a peculiar involuntary constriction of the fauces. The character of the odor is an important consideration in cases of supposed poisoning by prussic acid. If distinctly recognized by more than one person about the mouth of the deceased, or upon opening the body, it may afford strong reason for supposing that death was caused by this agent. But, as will be presently seen, this evidence is not obtained in every case.

§ 445. *Symptoms.*—The rapidity with which this poison acts upon the system hardly allows of the observation of successive symptoms. Where insensibility is not immediately produced, it is preceded by faintness, giddiness, loss of muscular power, and sometimes by convulsions. In other cases, the patient being found insensible, it is impossible to know the previous symptoms. When seen at this time, the eyes are fixed and glistening, the pupils dilated and unaffected by light, the limbs flaccid, the skin cold, and covered with a clammy perspiration; there is convulsive respiration at long intervals, between which the patient appears lifeless; the pulse is imperceptible, and involuntary evacuations occasionally take place. The respiration is slow, deep, gasping, and sometimes heaving or sobbing. This description, which is applicable to the greater number of cases, we have borrowed from Dr. Taylor. It should be added, that there is usually rigidity of the jaws, which has sometimes effectually prevented the administration of antidotes.

§ 446. The *period at which death takes place* is usually within an hour, seldom indeed exceeding three-quarters of an hour. A man drank from a phial containing prussic acid, while embracing his wife; he died in fifteen minutes.[1] Seven epileptic patients, who were accidentally poisoned with this acid, died in convulsions within three-quarters of an hour.[2] In most cases, however, death occurs in a few minutes; and, if life be prolonged for a period of an hour

[1] Pharm. Journ., Aug. 1851.

[2] Orfila, vol. ii. 286.

(Lonsdale), recovery usually takes place.[1] The rapidly fatal character of this poison is, indeed, one of its most striking features. From experiments upon animals, it was supposed at one time that prussic acid was, necessarily, almost immediately fatal. Animals poisoned by it die within a few seconds. In man, however, although the symptoms often commence in the act of swallowing, either by direct absorption through the surfaces of the tongue and mouth, or by inhalation and absorption through the lungs, they may also not be perceived for one or two minutes. When absorption occurs by inhalation in an atmosphere containing a small amount of the volatile acid, whether over the fumes from a vessel in the open air, or in a room badly ventilated, the symptoms of intoxication may come on at a later period. The danger of accident will depend upon the amount at any one period present in the system of the individual exposed to its vapor or solution, and if this be sufficient to imperil his life, the symptoms will occur suddenly and with great violence.

§ 447. Upon this fact depends often an important question, bearing upon the voluntary or homicidal nature of the poisoning, since it may become evident from circumstantial evidence, that the deceased has retained consciousness and voluntary power for a certain length of time after swallowing the poison. In Mr. Burnam's case, mentioned further on, insensibility did not occur for two minutes after the poison was swallowed, and that in the largest dose yet recorded. In the case of a girl, aged seventeen, the servant of a chemist, who was seen by the reporter when already insensible, the retention of consciousness for a short period was proved by circumstantial evidence. In turning up the feather bed, after the body had been removed from it, a prussic acid bottle, with the stopper in, was found between it and the mattress, near the centre; it contained about eight drops of the acid. The girl's mistress stated, that about twenty minutes after the girl had left her, she was proceeding up stairs to bed, when, in passing the girl's room door, she heard a moaning noise; she entered the room, and found her lying in bed, with her clothes on, and the bedclothes drawn up to her face, apparently "gasping for breath." She instantly gave the alarm. "The evidence adduced proved, as far

[1] Dr. Fagge reports one fatal case which occurred an hour and a quarter after the poison was swallowed, Guy's Hosp. Rep., 1868, p. 259.

as *could* be proved, that she had swallowed an ounce of the acid, re-corked the phial, thrust it to full arm's length between the feather bed and the mattress, got into bed, and then drew the clothes over her body, and there appeared to have been no convulsions."[1] Dr. Sewell reports, in the same journal, the interesting account of a gentleman who swallowed seven drachms of the medicinal acid, equal to twenty-one grains of Scheele's acid. It was proved that after taking the poison he had walked from the table in the middle of the room to the door, unlocked it, called for assistance, and then, returning to a sofa in the room, stretched himself upon it. Here he was found lying as if in a profound slumber, his legs crossed, his arms by his side, and his eyelids firmly closed. The eyes were more brilliant than during life, and continued so until the next day. His face was livid, and the lips very blue; the muscles were all relaxed.[2] A young man swallowed, in his bedroom, a dose equivalent to 2.54 grains. He then descended thirty steps, and walked about twenty paces before he became powerless. He was endeavoring to open the front door of the house to go out, when he suddenly fell. The only symptoms observed by a person present were, that "he threw his arms about, and made a noise in breathing, fetching it hard; he very soon became still."[3] A case which is characterized by Dr. Taylor as one of the most extraordinary on record in this respect, is that related by Mr. Godfrey: "A gentleman aged forty-four, swallowed, it was supposed, half an ounce of prussic acid (strength not stated), but certainly a quantity sufficient to destroy life. After taking it from the bottle, he walked ten paces to the top of a flight of stairs, descended the stairs, seventeen in number, and went to a druggist's shop at forty paces' distance, where he had previously bought the poison, entered the shop, and said, in his usual tone of voice, 'I want some more of that prussic acid!' He then became insensible, and died in from five to ten minutes after taking the poison." There were no convulsions.

Such cases as these (and more might be quoted) fairly prove the untenable nature of the notion, that any acts indicative of design, committed after the poison has been swallowed, cannot be attributed to the deceased. Many simple acts, like those noted, can be easily

[1] Boston Med. and Surg. Journ., vol. xxxii. p. 528. Leithead.

[2] Ibid., vol. xxxvii. p. 320.

[3] Lowe, Guy's Hospital Rep., 1846, p. 490.

performed in a very short space of time, and scarcely take anything away from the fearfully rapid character of this poison.

Another fallacy, derived from the result of experiments on animals, is the supposition that death from prussic acid is always preceded by a shriek! There is no case in the human subject which attests any such fact; on the contrary, in the vast majority of cases, there are neither general convulsions, as is common in animals, nor any unusual cry; but, on the contrary, death comes on in a placid manner, the patient passing away without a struggle. The convulsions which were observed in the seven epileptic patients already referred to, may, with some probability, be referred to their constitutional predisposition. In a case of suicide by a dose equivalent to eight grains (reported by Dr. J. G. Fleming), the appearance of the body was most strikingly like life, even the natural color had not left the cheek, the features were composed, and the limbs relaxed. There evidently could have been no convulsions.[1]

§ 448. It is obviously unnecessary in a work of this character to enter into a discussion of the many theories advanced by physiological investigators, as to the *modus operandi* of this poison upon animal life. Investigations upon this question, by Preyer,[2] Hoppe-Seyler,[3] Bernard,[4] Meuriot,[5] Wagner,[6] and Lewisson,[7] are quite fully set forth in a recent publication on Materia Medica and Toxicology,[8] and show very conclusively that the action of this poison is direct by its presence in the blood upon the organic life, and suppresses the functions of life either by arrest of the circulation by paralysis of the heart causing syncope, or by interfering with the aeration of the blood in the lungs causing asphyxia, producing a similar and as fatal a result as if the animal life had ceased by suffocation. When the doses taken are small, non-toxic doses, their continuous administration causes a slowing of the heart's action, following almost immediately upon a sudden prolonged arrest of the heart in diastole (or relaxed in opposition to a contracted state of that organ), which may finally result in the complete arrest of

[1] Edinb. Monthly Journ., July, 1846.
[2] Die Blausäure, Bonn, 1870.
[3] Medicinish-chem. Untersuch., Berlin, 1867.
[4] Leçons sur les substances toxiques et medicamenteuses, Paris' 1857.
[5] Archives Générales, 6e s., t. xi.
[6] Lancet, 1877.
[7] Reichert's Archiv., 1870.
[8] H. C. Wood, Phila., 1882, 4th edition.

cardiac movements. In certain experiments of Boehm and Knie,[1] it was observed that if woorara were first given to animals, the injection of prussic acid could be well supported in large doses, provided artificial respiration were maintained. One especial symptom of hydrocyanic poisoning should not be omitted. It is the peculiar forced and prolonged expiratory movement in respiration which is always present.

§ 449. The diseases which are liable to be confounded with poisoning by this acid are sudden apoplexy, cerebral or pulmonary, cardiac syncope, embolism of the lungs, or air embolism in the arteries or veins after child-birth or instrumental abortion. The question of doubt in these cases can always be solved by a careful *post-mortem* examination.

§ 450. Dr. Taylor[2] discusses, as an important medico-legal question, the rapidity with which death may occur after a fatal dose of prussic or hydrocyanic acid. Since its action is so rapid, it becomes important to know for how long a time the individual was conscious and able to move of his own volition. Dr. Lonsdale is the authority for the statement that a drachm of Scheele's acid (equal to about two and a half drachms of the dilute acid of the U. S. Pharmacopœia) would affect the ordinary adult in one minute or less, and three or four times this dose will exert its influence within ten or fifteen seconds. Dr. Taylor cites the importance of this question in the case of Rex *v.* Freeman at the Leicester Spring Assizes, 1829.[3] "The medical question at the trial was: Could this quantity of poison (four and a half drachms of Scheele's solution, equal to about eleven drachms of that of the U. S. Pharmacopœia) have been taken, and the deceased have retained consciousness and volition for a sufficiently long period to have performed these acts herself? (corking the vial and placing the leather and string, which appeared to have gone round the neck of the bottle, in the chamber vessel). Five medical witnesses were examined and the opinions of four of these were strongly against the *possibility* of the acts having been performed by the deceased. This strong medical opinion was set aside by circumstances and the prisoner was acquitted." We can see no reason

[1] Archiv für experiment. Path. und Therap., Bd. ii. p. 137; referred to by Prof. Wood, op. cit.

[2] Treatise on Poisons, Phila., 1873, p. 565.

[3] Medical Gazette, vol. viii. p. 759.

for the assumption that the deceased must have placed the articles spoken of in the vessel after swallowing the poison. Why would it not be more reasonable to assume that this act of volition was performed before swallowing the prussic acid? Two other cases are referred to by Dr. Taylor, in which the individuals, who took the acid for suicide, used similar large doses and set the phials down by their side, drew up the bedclothes smoothly without any signs of disorder about them or the body itself. Dr. Taylor states that "in death from prussic acid the body is usually found lying calm and tranquil without any mark of struggling or convulsions."

The following symptoms of fatal poisoning from a three-drachm dose of prussic acid is reported by Dr. A. B. Kelly.[1] Perfect insensibility, lividity, mucus about the mouth, eyes open and glassy, pupils dilated, respiration hurried and stertorous, no convulsions or vomiting; opthalmoscope, fifteen to twenty minutes after dose was taken, showed that the brilliant red glow of the choroid was entirely absent, a pale violet-gray tint taking its place. The optic disk, of a dull gray-white tone, was ill-defined; the retinal arteries were discerned with great difficulty, as narrow threads; the veins, on the other hand, were easily traced, appearing unevenly and imperfectly charged with blood. The media presented the appearance of baked tapioca; these appearances were observed after artificial respiration, and faradisation of the phrenic nerve were resorted to, and were indicative of death.

§ 451. The *smallest quantity* of anhydrous prussic acid capable of destroying life has so far, from actual observation, proved to be about nine-tenths of a grain. This was the amount which destroyed the life of a woman mentioned by Mr. Hicks.[2] Life was extinct in twenty minutes. This quantity is equal to about fifty drops of a two per cent. solution of the acid. Other cases have been reported in which most alarming symptoms ensued from smaller doses.[3] In any case in which it appears that death has resulted from a small quantity, it is highly important, if we would avoid errors, that the strength of absolute acid should be ascertained by an analysis of the sample remaining.

§ 452. Instances of *recovery from very large doses* have been

[1] Lancet, Dec. 6, 1879, p. 831.

[2] London Med. Gaz., xxxv. 896.

[3] *Vide* Taylor on Poisons.

recorded. Dr. Christison has reported a case in which, with great difficulty, a gentleman was restored who had taken between a grain and a half and two grains of the anhydrous acid;[1] and, still more recently, Mr. W. H. Burnam communicated to the *Lancet* a very interesting history of the recovery of his father from accidentally taking a drachm of Scheele's acid, which was found upon analysis to contain 2.4 grains of anhydrous acid.[2] Mr. Nunnely, also, has reported a case of recovery from one grain and a third of anhydrous acid.[3] One curious fact, in relation to the size of the dose, should not be forgotten, viz., that a comparatively small dose will produce equally fatal results with a large one, it being highly probable, from the cases so far recorded, that all doses over one grain of absolute or anhydrous acid are capable of destroying life with equal certainty and rapidity. The limits of safety, in the use of prussic acid, are easily passed, and the formidable symptoms occasioned by it develop themselves with wonderful rapidity; hence too great caution cannot be observed in its administration with remedial views in medical practice.

M. Regnauld relates the case of a young man who was poisoned by the *vapor* of prussic acid disengaged from a mixture of the ferrocyanide of potassium and sulphuric acid. The symptoms were those of asphyxia, rather than of the nervous prostration which usually accompanies poisoning from the internal use of prussic acid.[4]

§ 453. *Post-mortem appearances.*—It should be admitted, that the peculiarities of these, in prussic acid poisoning, are not well marked, and that the general condition of the body is that usually met with after death from an intense narcotic, or death from suffocation or asphyxia. The face is either livid or pale; the lips and nails blue; and the skin of the neck, back, and shoulders much discolored. The jaws are firmly closed; the muscles of the hands and feet contracted, and cadaveric rigidity comes on sooner, and is more perfect than usual. The eyes have a peculiar brilliant and glistening appearance, the pupils are widely dilated, and foam is sometimes seen about the mouth. Evidence of involuntary evacuation of the bladder and rectum is not unfrequently observed. The veins of the brain are found turgid, and the lungs are congested

[1] Med. Gaz., 1850, 917.
[2] Brit. and For. Med.-Chir. Rev., April, 1854.
[3] Taylor.
[4] Brit. and For. Med.-Chir. Rev., Oct. 1852, p. 561.

with a very dark-colored, but liquid blood. Orfila says that the mucous membrane of the air-passages has generally a dark-red color, which cannot be removed by washing, and the bronchial tubes are filled to their extremities with a bloody froth. More recent authorities would hardly admit this description by Orfila, since experience has shown that this poison usually destroys life by asphyxia or by paralysis of the heart's action (*vide supra*, §448), and the *post-mortem* appearances from hydrocyanic or prussic acid are such as are met with where the death is to be attributed to either of these causes.

The heart is usually found with the right side relaxed and engorged with liquid blood (having ceased its motion during diastole rather than during systole). This is the usual concomitant of death by asphyxia, and presents nothing unusual. The mucous membrane of the stomach is, in perhaps the majority of cases, highly reddened, and this deepening of color may extend for some distance into the intestinal tube. In a case reported by Jochner, and in a few others, a chocolate-colored fluid was found in the stomach. The blood is generally dark and fluid, sometimes of a purplish color. It will readily be seen how insignificant are the pathological alterations found in those who have been killed by prussic acid. There is no one of the appearances here noted which may not be met with in death from many other causes, and especially in sudden death by some mode of asphyxia. The peculiar mottled and ecchymotic appearance of the lung tissue, the engorgement of both ventricles and sometimes of both auricles with dark fluid blood, the congestion of vessels of brain and spinal cord (Tardieu) mentioned by some authorities, are probably due to the asphyxia which is produced in the majority of cases of intoxication by this drug. Certain experiments[1] performed by the writer would seem to prove not only this point, but also that rapid and early *post-mortem* muscular rigidity would follow the majority of deaths which suddenly occur to persons previously in full health and muscular activity. In some of these experiments upon the intoxication of animals by prussic acid, artificial respiration prevented convulsions and certain *post-mortem* appearances usually attributed to this poison.

[1] *Vide* London Practitioner, April, 1872, p. 197. Also a paper by the writer with experiments, Boston Med. and Surg. Journ., Aug. 23, and Sept. 13, 1866.

§ 454. The only circumstance which is at all deserving of attention, and which merits a separate consideration, is the presence or absence of the odor of prussic acid. It may be at once stated that where this odor is unequivocally detected the evidence is satisfactory, since it is of so peculiar a character as not to be readily mistaken for anything else, unless nitrobenzol or oil of bitter almonds be excepted (*vide infra*). Unfortunately, however, it is not always discovered, even in well-attested cases of poisoning by this substance. The odor is sometimes observed about the mouth and nostrils of the deceased, and is not perceived in the stomach. This was the case, in an instance reported by Jochner, of a young man who committed suicide by this poison. On the other hand, the stomach may exhale the odor of prussic acid and none be perceived about the mouth or in the room. This was noted in the case reported by Mr. Hicks, in which, moreover, the examination of the body was not made until *ninety* hours after death. On opening the chest, the odor was more plainly perceived than in any other part of the body, and the fluid contained in the stomach smelt very strongly of prussic acid.[1] In none of the epileptic patients before mentioned was the odor of prussic acid discovered in any part of the body. The inspection was made twenty-four hours after death. Dr. Christison's case of recovery from a large dose may be referred to here, as corroborative of these facts ; the first liquid drawn from the stomach by the tube which he introduced, gave indications of the presence of prussic acid on analysis, but not by any peculiar odor, although there was none other by which it might have been concealed. The stomach of Sarah Hart, poisoned by Tawell, had no odor of prussic acid, yet one grain of anhydrous acid was obtained by distillation from its contents, consisting partly of apple pulp. In the greater number of cases, however, there can be no doubt that it is readily distinguishable, since in some it has been so strong as to seriously affect the bystanders. The circumstances which cause these singular variations have not been thoroughly investigated. It is supposed, very naturally, that the length of time the person has survived after taking the poison, and the interval elapsing between death and the inspection of the body, must, as well as the dose, have an influence upon the preservation of the odor. But it is evident that these con-

[1] Med. Gaz., xxxvi. 460.

jectures are not entirely satisfactory, since not only has the odor been detected after as long an interval as seven days, but, on the other hand, it has not been detected even where the presence of the acid has been demonstrated by chemical analysis. It is probable that in these cases it may have been fixed by a base. It is a matter of some importance to refer to a case mentioned before to show in what way a person with criminal intent can mislead justice by inhalation of some other odor more pungent than that of prussic acid just before death. This, of course, might mislead the senses, but would in no way interfere with the chemical research for the poison.

§ 455. Hydrocyanic acid may be obtained from many vegetables, particularly those belonging to the sub-orders Amygdaleæ and Pomeæ;[1] as from bitter almonds, apple-pips, the kernels of peaches, apricots, cherries, plums, and the flowers of the peach, and cherry-laurel, from the bark of the wild cherry, and the root of the mountain ash. Prussic acid does not exist ready formed in these plants, but is the result of the reaction of water upon amygdalin in the presence of a ferment, emulsin. Hence, if any of the above substances are found in the stomach, the question may arise whether the indications of the presence of prussic acid are due to their decomposition or to the acid swallowed as such. The only manner in which doubt arising from this circumstance can be satisfied, is the obtaining by chemical analysis a larger quantity of the acid from the contents of the stomach, than these substances could yield. It is extremely improbable that death should result from the ingestion of any of these articles, except in such a large quantity as to obviously preclude the idea of prussic acid in substance having been taken.

It has been stated that this acid may be produced spontaneously from unsound cheese; but Dr. Taylor was unable by experiment upon numerous samples of decayed cheese to find any evidence of it. The notion, also, that it may be a spontaneous product of animal decomposition, is timidly advanced by Orfila, but has not yet received the necessary confirmation. It is also said to be produced by the action of nitric acid on alcohol. This fact was clearly ascertained by M. de Claubry, who observed the serious effects of the

[1] Pereira.

vapor upon the health of the workmen engaged in the manufacture of hyponitrous ether.[1]

§ 456. While these objections must be allowed their full force in cases where their applicability can be shown, it by no means follows, where no chemical process further than mere distillation is employed, and where none of the organic matters above mentioned are found in the stomach, that the distinct evidence of the presence of prussic acid, by odor and by the simple chemical reactions to be presently noticed, ought not to be perfectly satisfactory. Moreover, if the mode of death be known, these objections will fall away of themselves. If, however, none of the circumstances preceding death can be ascertained, and neither the odor of prussic acid nor its reactions with the established tests be recognized, it may certainly become a question of serious import, whether the traces of it found afterwards may not be due to some other cause than its ingestion into the stomach. Thus, if the contents of the stomach be subjected to distillation *with an acid*, it may possibly happen that the sulphocyanide of potassium, which sometimes exists in minute traces in the saliva, may be decomposed, and evidence of prussic acid be thus obtained. The evident answer to this objection is the fact, that so many stomachs have been distilled without finding any prussic acid, even when an acid was added previous to the distillation. For the value of these objections, we must refer the reader to the more detailed treatises on Poisons, especially to those of Drs. Christison and Taylor, and to Orfila's *Médecine Légale* and *Toxicologie*, and to what has been already mentioned in a previous chapter.

§ 457. *Bitter almonds.*—A lad of fifteen, the son of a wholesale grocer, got access to a cask of bitter almonds, and consumed a large quantity of them with sugar. After a time, but how long is not known, he felt a pleasing sensation, then became suddenly giddy, fell down, and lost his consciousness and recollection. He was found lying insensible near the cask. Ammonia and carbonate of potassium were successively administered, and the stomach pump employed. By these means he was much relieved. Emetics were then given, and he threw off, in the course of half an hour, as much as *eight ounces*, *Troy*, of bitter almonds.

[1] Ann. d'Hyg., 1839, ii. p. 350.

Another case of poisoning,[1] from eating two handfuls of bitter almonds, is reported, in which the first symptom observed was a convulsion with gasping and labored breathing. The patient recovered after the application of electricity, one pole being applied over the heart, the other over the course of the pneumogastric nerve in the neck. The contents of the stomach were first removed with the aid of a stomach pump. Inhalations of ammonia, beef-tea, and brandy were also administered. The ammonia inhalations and electricity were applied alternately every half hour. As yet, however, no fatal case of poisoning by eating the almond kernels themselves has been recorded.

§ 458. The essential *oil of bitter almonds*, which is the product of the distillation of the crushed almonds with water, contains a large amount of prussic or hydrocyanic acid, which is the product of the chemical action of the ferment emulsin upon the glucoside amygdalin, both of which pre-exist in the almond pulp, but do not yield prussic acid until brought together in contact with water. It has been estimated that 2500 parts of almond pulp contain 100 parts of amygdalin, and yield by distillation 41 parts of essential oil, which contains, when fresh, six parts of anhydrous prussic acid. Hence the essential oil may, when perfectly fresh, contain 12.76 per cent. of anhydrous acid. This, on account of its volatility, escapes very rapidly, if the oil be exposed to the air, and a portion escapes every time the bottle is opened, as is the case with all preparations containing hydrocyanic acid.

It is, therefore, a dangerous substance from the very fact of the uncertainty of the amount of the contained poison. In certain patent medicines made from wild cherry the hydrocyanic acid present may reach a dangerous amount. Heat used in cooking may volatilize a considerable portion of the prussic acid in the bitter almond oil, and hence secure a certain immunity from its baneful influences, but when used without heat, caution should be exercised. It should be remarked here, however, that the peculiar flavor and taste do not depend upon the amount of prussic acid present, since a distillation of the oil in the presence of potassic hydrate will remove the prussic acid, and without dispelling

[1] Med. Times and Gazette, London, July 9, 1881.

the almond flavor. Even with this precaution the essential oil, like all other essential oils, will cause an intoxication like that of alcohol. There has been at times some doubt expressed as to the fact of this purification being sufficient. Much of the difference of opinion which has reigned as to this question arises from the circumstance, that by merely agitating the oil with a large excess of lime or caustic potash, and distilling it, the prussic acid is not sufficiently separated. A salt of iron should be employed, which fixes it more effectually. Dr. Maclagan made experiments upon dogs with the oil thus rectified, and found that when no trace of prussic acid could be detected by the iron test, it was not poisonous. The following are the most prominent of his conclusions: 1. That the poisonous action of the unrectified oil is essentially due to the hydrocyanic acid which it contains. 2. That the oil really free from prussic acid, in doses of a few drops, does not act as a poison on animals generally. 3. That although the rectified oil, in doses of a drachm and upwards, does prove fatal to rabbits, yet in dogs (whose organization renders them much better subjects for testing the probable effects of the substance on man), doses of the oil even as large as three drachms, entirely or nearly free from prussic acid, produce no other effect than a little vomiting, and do not cause death, or even dangerous symptoms. The same results have in the main been obtained by other chemists, particularly Wöhler and Frerichs.

Why the deadly ingredient should be allowed to remain in it, if it can be so readily removed, it is not easy to understand. The placing of restrictions upon the sale of this and other articles of a pernicious character, for the purpose of flavoring or ornamenting articles of food, appears to us to be of still more urgent importance than any restraint upon the sale of arsenic and similar poisons, which cannot be employed except for destructive purposes. In the latter case means are readily found to obtain the required poison; while in the former, experience has shown that equal brilliancy of color and delicacy of flavor can be obtained from harmless substances, as from the deadly poisons in universal and daily use in this country and England. Prussic acid is too potent a poison to be distributed to cooks and confectioners, disguised with the pleasant odor of bitter almonds; the most ordinary prudence and humanity would seem to demand that it should not be used thus freely and incautiously.

§ 459. The following are a few of the instances in which the oil of *bitter* almonds has produced fatal results :—

A child, eight and a half years old, took a teaspoonful of *ratafia*, containing seven drops of the oil of bitter almonds. She became immediately insensible, but had no spasms ; the limbs were relaxed, the jaw, however, firmly closed ; the eyelids closed, but the eyes brilliant and glassy, although without expression. Cold affusion, emetics, and stimulants restored her, and in twenty minutes her consciousness returned.[1] The general symptoms of poisoning with the oil of bitter almonds resemble very closely those by pure prussic acid, the principal difference being that in the former they are perhaps less instantaneous or immediate in their accession, and that the duration of life is somewhat longer. Nevertheless, cases of very rapid death from this oil are recorded.

Dr. Taylor mentions the particulars of a case referred to him, in which it was probable that the whole duration of the case did not exceed *seven* minutes, and the man was not seized by the peculiar symptoms of poisoning until *five* minutes after he had taken the dose. During this time he was conscious and self-possessed, and replied rationally to questions put to him.

In a case related by Mertzdorff, in which two drachms of the ethereal oil of bitter almonds were swallowed, death occurred in half an hour. Another one, related by Dr. Taylor, is remarkable not only for its termination in a similar short space of time, but from the fact of the smallness of the dose, which was only *seventeen drops*. A druggist swallowed half an ounce of "almond flavor," equivalent, it is said, to thirty drops of the oil. He fell insensible in less than half a minute. This case presents, moreover, this peculiarity, that there was a temporary remission of the symptoms. He was sensible for a few minutes, and spoke on the nature of his attack, but gradually again relapsed into a delirious and apparently very happy state. His eyes were extremely brilliant, but the pulse was quick and intermittent, and the whole body cold. He gradually recovered from the effects of the poison.[2] This case is a very extraordinary one ; the peculiar effects cannot well be attributed to the smallness of the dose, since, as we have already seen, little

[1] Lancet, June 8, 1844.

[2] Lancet, Sept. 1839, p. 930. Mr. Chavasse.

more than half the quantity has proved fatal, and in this case it was strong enough to produce almost immediate insensibility. Mr. Iliff has reported a case in which death must have been very rapid. It is that of a young woman who poisoned herself in the Zoological Gardens, London. A small phial containing a drop or two of the oil of almonds was found in the pocket of her dress with the cork pushed in.[1] In addition to the similarity in symptoms, the *post-mortem* appearances from this substance resemble those of prussic acid as well as that of other narcotics. The same placid and natural expression of countenance, and the same purplish color and fluid condition of the blood, are here found. But the odor is uniformly present, generally more or less about the mouth, but, in all the cases yet reported, very characteristic and penetrating into the stomach and cavities of the body generally. It is very persistent, and may be discovered several days after death.

§ 460. The oil of bitter almonds is about four times as strong as the medicinal hydrocyanic acid of the United States Pharmacopœia. It is of a yellow color, has a bitter, acrid, burning taste, and is slightly soluble in water. The almond flavors and essences so much used in cooking are solutions of the oil in spirit, and may prove highly dangerous in the hands of ignorant people. Enough prussic acid is contained in less than an ounce of most of these flavors, unless purified as above mentioned, or exposed to the air for some time, to produce fatal effects, and it is evident that smaller quantities might have the same result in the case of children.

§ 461. There is a case related by De Keyser, which appears to show that the application of this oil to the skin may be dangerous. A lady used about half an ounce of it by mistake for an oil intended to make her hair grow. That the vapors of the oil did not occasion the symptoms is possible, because the waiting maid, who assisted her, was unaffected. She was seized with coldness extending from the head and spine to every part of the body, followed by ringing in the ears and unsteadiness, deafness, swimming of objects before the eyes, and impaired power of moving the limbs. She fell insensible, and in a state of collapse resembling syncope. She gradually regained her consciousness, but the sense of coldness con-

[1] Lancet, April, 1850.

tinued for several hours.[1] It is not improbable that she may have inhaled the vapor.

§ 462. *Apricot kernels.*—These also may yield hydrocyanic acid. At Arles, a child ate two or three apricots; but, not content with this, also took the kernels inclosed in the fruit. Very soon after he was seized with convulsions, and died in spite of every attention.[2]

§ 463. *Peach kernels.*—Dr. Keating, of Philadelphia, has reported a very interesting case, in which he succeeded, by affusion of cold water, in restoring a child three years of age, who had eaten a quantity of peach kernels. The child was seized suddenly, and when seen was found insensible, with slow, deep, sobbing respiration, no convulsion of the limbs, but slight twitching of the mouth, icy-cold extremities, finger-nails livid, hands slightly clenched, eyes prominent, and pupils dilated. A strong odor of prussic acid was perceived about the mouth. An emetic brought up a quantity of peach kernels, emitting the characteristic odor. It should be here observed that in these cases the hydrocyanic acid would be reasonably slow in being disengaged from the kernels in the stomach, and thus postpone the severe symptoms which arise from the ingestion of the acid itself. Another case is reported, in which the kernels of the cherry proved fatal to a child of five years, after forty hours' illness.

§ 464. *Cherry-laurel water.*—The following case of poisoning by this liquid is remarkable for the slowness with which the symptoms supervened, and the unusually long duration of life. A hypochondriac of advanced age drank one morning an ounce and a half of cherry-laurel water. The symptoms of poisoning did not come on for *three* hours. Then the hands and feet became paralyzed, and the head fell forward upon the chest. Involuntary discharges from the rectum and bladder took place. The extremities, though cold and immovable, were not insensible. The pulse was small, the voice hoarse but distinct, and the intellect perfect. The patient observed with pleasure the incessant progress of the weakness; he died in the evening without pain or convulsions. On section, the blood was found gluey, and of a peculiar dark color; but no odor of

[1] Journ. f. Phamakodyn., 1857, p. 588.

[2] Quoted in the Am. Journ. Med. Sci., Jan. 1853.

bitter almonds was detected. The celebrated trial of Capt. Donellan, in 1781,[1] on a charge of poisoning Sir Theodosius Boughton with this liquid, is no doubt familiar to the reader. It is not definitely known how much hydrocyanic acid is contained in cherry-laurel water, since it varies with each preparation. It is known, however, that in summer, the distillate from cherry-laurel leaves contains almost twice the amount of acid as that distilled in winter, the amount gradually increasing from winter to summer. The leaves do not evolve the hydrocyanic acid, unless broken or bruised. Cherry-laurel water also becomes weaker by being kept. The medicinal dose is from forty minims to a fluidrachm.

§ 465. *Cyanide of potassium.*—This substance, as also probably all the salts of hydrocyanic acid, is equally destructive and rapid in its effects with the free hydrocyanic acid. The symptoms are exactly similar to those produced by the latter poison, as are also the *post-mortem* appearances. Dr. Finnell, however, reports having met with intense redness of the gastric mucous membrane in three cases of fatal poisoning by this salt.[2] But Dr. Schauenstein found a dark-red color of the membrane with bloody points in only two out of five cases,[3] and attributes this peculiarity to the caustic operation of the alkaline solution in a concentrated state, and when the stomach contains but little food. The odor of prussic acid is less striking, and less frequently perceived in poisoning with this salt. In a case of sudden death from it reported by Casper, there was no unusual odor, although the nature of the poison was detected by chemical analysis.[4] The reaction of the contents of the stomach is always alkaline, and, according to Schauenstein, prussic acid can always be detected in them by the addition of formic acid. The reason of this is that the cyanide of potassium is a very unstable salt, so that it is easily decomposed by weak acids ; even the carbonic acid in the air is sufficiently strong, hence the odor of hydrocyanic acid evolved from a bottle containing cyanide of potassium. This explains also why, in some cases, we may have the effect of a caustic, since a large portion of the cyanide may have been converted into carbonate of potassium. The quantity capable of proving fatal may be stated at from two and a half to five

[1] Taylor, op. cit., p. 593.
[2] Am. Med. Times, i. 33.
[3] Prager Vierteljahr., lxv. anal. 14.
[4] Vierteljahrschrift, July, 1854.

grains, since the former quantity is equal to one grain of anhydrous prussic acid. The fatal dose must necessarily vary with the strength of the preparation, and this is very different for different specimens. The strongest is made by saturating a solution of potassa with prussic acid. A man, aged thirty, died in a quarter of an hour after taking fifteen grains, prescribed for him by his medical attendant by mistake for the ferrocyanide.[1] Dr. Perry related the following case to the Boston Society for Medical Improvement: A nurse administered this poison by mistake to a child, who had a slight cough, instead of a cough mixture, which stood near the bottle of solution of the cyanide. The immediate effects of the dose were vomiting and convulsions; then insensibility, locked jaw, coldness of extremities, which were pendulous and without muscular power; diminished frequency of respiration (twelve to sixteen per minute), the pulse small but distinct, sixty per minute; the circulation languid, pupils dilated, sphincters paralyzed; the teeth closed so firmly and continuously that only once or twice could anything be poured into the mouth. Dr. P. saw the child in fifteen minutes after the accident, and found it in a warm bath and insensible. It was treated by stimulants and the inhalations of ammonia. Death was sudden, and no *post-mortem* examination was made. The child lived *one hour and a half.*

A case is reported by Dr. C. E. Ware, of a woman who died in less than an hour from taking seven grains of this salt in a teaspoonful of liquid. Death occurred by gradual syncope.[2] In Vienna, Dr. Schauenstein met with five cases of fatal poisoning by this substance, in the course of eighteen months. In all of them the death seems to have been sudden. In one case, in a young girl, strong tetanic spasms came on directly after the poison had been taken.[3] The same symptom, with severe abdominal pains, occurred in a case reported by Prof. Wagner, of Leipzig.[4] The root and the juice of *Cassava* (Jatropha Manihot) produce symptoms identical with those of prussic acid, but, in general, they are less intense. De Keyser relates that three children who had eaten

[1] Henke's Zeitsch., Bd. 45, H. 1, p. 6.

[2] Boston Med. and Surg. Journ., Dec. 1856, p. 387.

[3] Br. and For. Med.-Chir. Rev., Oct. 1859, p. 530.

[4] Archiv f. phys. Heil., 1859, p. 417.

of the former, and an adult negro who had drunk about six ounces of the latter, recovered.[1]

§ 466. *Chemical examination.*—The best tests for hydrocyanic acid are equally adapted to its detection in its simple state and mixed with organic liquids. In the latter case, if it cannot be detected in the vapor by the tests, the liquid must be filtered and distilled as mentioned below. The three principal tests for hydrocyanic acid are called the iron, silver, and sulphur tests, and are performed as follows:—

§ 467. *The iron test.*—This test is founded upon the formation of Prussian blue, and the following is the method recommended by Tardieu.[2] Make a mixture of solutions of a protosalt of iron and of a sesquisalt (the sulphate or chloride). Add a few drops of the mixture to the suspected liquid and agitate; no precipitate should appear. Then add to the liquid thus treated a quantity of a solution of potassa or caustic soda sufficient to turn litmus paper clearly blue (this shows when the solution has become alkaline); an immediate black or greenish precipitate appears, which, if no prussic acid exists in the suspected liquid, is composed exclusively of a mixture of protoxide and sesquioxide of iron; whilst, if there should be contained in the liquid any prussic acid, there would appear, mixed with these two bodies, a small proportion of Prussian blue. If the first precipitate (black or green) only occurs, the addition of a slight excess of hydrochloric acid will instantly dissolve the two oxides, and will make a very clear solution. If, on the contrary, the liquid contain prussic acid, the addition of hydrochloric acid will have the effect of bringing out more beautifully the intense color of Prussian blue. In this last case, however, it must be observed that the yellow color of the liquid, due to the presence of a dissolved sesquisalt of iron, may be so intense as to momentarily mask the color of Prussian blue, and to give to it the appearance of a greenish precipitate (a mixture of the two colors yellow and blue). This is an important cause of error, especially when a minute quantity of the prussic acid is present, but the Prussian blue can always be plainly seen after the mixture has been standing for a sufficient length of time for it to completely

[1] Journ. f. Pharmakodyn., 1857, p. 586.

[2] Op. cit., p. 1198.

settle, when it will form a distinct blue coating upon the bottom of the vessel. It can then be collected upon a white filter paper, washed, dried, and preserved as one of the *corpora delicti*. Carson observes that this test will detect hydrocyanic acid when it is mixed with common salt, or other chlorides which interfere with the reaction of nitrate of silver. It is, on the whole, a delicate test when properly employed; but a frequent cause of failure in its application is the addition of too much potassa, or of the iron salt.

§ 468. The *vapor* of prussic acid may also be detected by this test, by means of the following expedient: Put a drop of the solution of potash in a small white saucer, and invert it over another containing a portion of the suspected liquid. After two or three minutes, or more, if the acid be much diluted, remove the upper saucer and drop on the potash a drop of the solution of the iron mixture; add one or two drops of dilute muriatic acid, to dissolve the surplus oxide of iron, and if hydrocyanic acid is present, a trace of Prussian blue will appear. This test may be conjoined with the silver test, both in its application to the liquid and to the vapor, for if the cyanide of silver formed in that test be decomposed by muriatic acid, prussic acid being liberated will, of course, give the reactions just described.

§ 469. *The silver test.*—The nitrate of silver causes, in a liquid containing prussic acid, a clotted white precipitate of the cyanide of silver, which is known by its solubility in boiling nitric acid. This property distinguishes cyanide of silver from the chloride, the latter being insoluble in boiling nitric acid. It is insoluble in cold nitric acid, and also in water; though it is quite soluble in ammonia. If a sufficient quantity of nitrate of silver be added, all the prussic acid will be thrown down as cyanide of silver (thus removing the odor of prussic acid). If the cyanide of silver is heated in a small straight tube closed at one end, a very strong and penetrating odor is evolved by the decomposition of the salt; the gas evolved burns, when lighted in contact with the air, with a beautiful reddish-purple color, having a green tinge on the outside of the flame. This latter portion of the test can only be performed when a large amount of hydrocyanic acid is present, and consequently quite a large amount of cyanide of silver is at our disposal.

The *vapor* of prussic acid may be detected also by the silver test. A watch-glass may be moistened with nitrate of silver, and

inverted over a vessel containing this acid; very soon an opaque white film of the cyanide of silver forms upon the moistened spot. Dr. Taylor states that one drop of the officinal acid (containing less than $\frac{1}{50}$th of a grain of the anhydrous acid) produces speedily a visible effect.

M. O. Henry has also suggested the following method of determining whether the precipitate contains the cyanide of silver: from one-third to one-half of a grain of the precipitate should be boiled for five or six minutes in a small tube with half its weight of chloride of sodium or potassium, and six or seven fluidrachms of distilled water. If the precipitate contains a cyanide, an insoluble chloride of silver and a soluble cyanide of sodium or potassium will be formed. The latter being filtered, treated with a small quantity of freshly prepared green hydrated oxide of iron, is heated again and filtered. It then contains a ferrocyanide of potassium or sodium, and, if treated by a salt of the sesquioxide of iron, gives a Prussian blue, and, if by the sulphate of copper, a chestnut-colored precipitate.

§ 470. *The sulphur, or Liebig's test.*—This test was first proposed by Liebig, and is the most delicate one yet discovered, as it will not only indicate the presence of prussic acid when no *odor* can be perceived, but when the other tests have failed to detect it. Dr. Taylor says that he detected clearly as small a quantity as the 0.3930th of a grain, and that in an experiment in which ten drops of a liquid, containing only one 473d part of a grain of anhydrous prussic acid, produced the characteristic reaction with hydrosulphuret of ammonia in five minutes.

§ 471. Dr. Taylor[1] says: "The iron and silver tests may be applied first, and these should be followed by the sulphur test, as the latter always contaminates the liquid to be tested. . . . Although we at present know of no vapor but that of prussic acid, which will thus affect the sulphur-test, it appears to me that we should not be justified in relying upon infinitesimal results, which admit of no kind of corroboration. The question is here much the same as in reference to the detection, by the process of Marsh, of minute traces of what is alleged to be arsenic, when the quantity is too small to be separated by Reinsch's process. The silver test

[1] On Poisons, p. 674.

cannot be relied upon for detecting small quantities of prussic acid in organic liquids or solids, unless the film of cyanide of silver is converted into sulphocyanate of ammonia. When, however, we have produced the colored results by the iron and sulphur tests, there can, it appears to me, be no reasonable doubt of the presence of the poison. With either result, separately, as applied to the vapor, there may be room for objecting to the conclusion that prussic acid has been certainly detected."

§ 472. The manner of applying the sulphur test is as follows: "If a small quantity of hydrosulphuret of ammonia (containing a little excess of sulphur) be added to a few drops of the solution of prussic acid, and the mixture be gently warmed, it becomes colorless, and, on evaporation, leaves sulphocyanate of ammonia, the sulphocyanic acid being indicated by the intense blood-red color produced on adding to the residue a solution of a persalt of iron; this color immediately disappears on adding one or two drops of a solution of corrosive sublimate. This test is very delicate, and it therefore requires some care in its application; thus, if the boiling and evaporation be not carried far enough, the persalt of iron will be precipitated black by the undecomposed hydrosulphuret of ammonia; and if the heat be carried too far, the sulphocyanate of ammonia may itself undergo decomposition and be lost."[1] "The great utility of the sulphur test, however, is in its application to the detection of the minutest portion of prussic acid when in the state of *vapor*. In this respect it surpasses any process yet discovered. In order to apply it we place the diluted prussic acid in a watch-glass, and invert over it another watch-glass, holding in its centre one drop of the hydrosulphuret of ammonia. No change apparently takes place in the hydrosulphuret; but if the watch-glass be removed after the lapse of from half a minute to ten minutes, according to the quantity and strength of prussic acid present, sulphocyanate of ammonia will be obtained on gently heating the drop of hydrosulphuret, and evaporating it to dryness. With an acid of from three to five per cent. the action is completed in ten seconds. The addition of one drop of persulphate of iron to the dried residue brings out the blood-red color instantly, which is intense in propor-

[1] The terms in the text, "hydrosulphuret of ammonia," and "sulphocyanate of ammonia," should more properly be sulphide of ammonium, and sulphocyanide of ammonium.

tion to the quantity of sulphocyanate present. When the prussic acid is excessively diluted, the warmth of the hand may serve to expedite the evolution of the vapor."[1] The tests for the *vapor* are equally applicable to organic mixtures and to the detection of the poison in the blood, secretions, or soft tissues.

§ 473. Buchner[2] gives an account, in connection with the murder of the Countess Chorinsky, of the detection of prussic acid in the murdered woman. No odor of prussic acid could be detected in the blood, which remained in a fluid state for five days, and remained free from putrefaction for a considerable time. Some of the blood mixed with water was distilled; the first portions of the distillate evolved the vapor of prussic acid, easily recognized by its odor, and presented the usual reactions of prussic acid. Buchner considered the sulphur test of Liebig the most delicate. Prussic acid was detected even after fifteen days.

M. Henry and H. Hubert have also proposed the following very satisfactory test: the cyanide of silver, having been prepared according to the method referred to above, and thoroughly dried, is introduced into a tube from five to seven inches long, closed at one end, and containing at its closed extremity a little iodine, about half the weight of the cyanide. On heating this end of the tube very gently, beautiful snow-white crystals of iodide of cyanogen are deposited upon the cool portions of the tube.[3]

§ 474. The spectroscope has been suggested as a means for determining the peculiar changes of the blood after prussic acid intoxication. It is too early yet to give any directions for examination by this instrument, nor have any results yet been attained to warrant its use as a reliable agent in the investigations of poisons in legal medicine. For an interesting memoir on this subject, reference is given to a paper by Victor Fumouze, "Les Spectres d'Absorption du Sang;"[4] also Stokes[5] gives an analysis of a paper by Preyer[6] on the absorption-spectra of blood and prussic acid, etc. Also in "Traité d'Histologie," etc., Frey, Spillman, et Ranvier, an appendix by the latter upon "Des Substances Colorées du Sang et de leur Analyse Spectrale."[7]

[1] Taylor on Poisons, p. 548.
[2] Revue des Cours Scientifiques.
[3] Bull. de l'Acad. de Méd., xxii. 350.
[4] Paris, G. Baillière, 1871.
[5] Glasgow Med. Journ., 1868, p. 70.
[6] Die Blausaüre, Bonn, 1868.
[7] Paris, F. Savy, 1871.

§ 475. It is important to remark here that it is in the tissues especially that prussic acid may be found, though no odor of the agent can be detected in the blood, and that when no suspicion of the poisonous agent is held by the chemical expert, he should, first of all, examine for this evanescent or volatile poison. Taylor[1] mentions the case of a dog's stomach which had been exposed to the air for twenty-four hours after death by prussic acid intoxication, and then thoroughly washed under a current of water, yielding the cyanide of silver in the course of chemical analysis.

§ 476. *Detection after death.*—Notwithstanding the readiness with which prussic acid undergoes decomposition, it has been detected in the body after death. Rieckher detected it in the contents of the stomach by the sulphur test, twenty-four hours after death.[2]

Mr. West was able to detect it on distillation by the odor, and the silver and iron tests, *twenty-three* days after death, although no pains had been taken to insure its preservation.[3] In the following case it will be seen that it was detected after about the same period of time.

A young man of Tours having purposely poisoned himself with medicinal hydrocyanic acid of the twelfth degree, of which he appeared to have swallowed about twenty-five grammes, M. Brame was called upon, after the lapse of *three weeks*, for the purpose of trying whether it was possible to detect hydrocyanic acid in the body. He was able to detect and estimate a considerable quantity of this poison which had remained in the stomach. By the addition of neutral and pure nitrate of silver, there was formed an abundance of a flocculent and yellowish precipitate, which, well washed and dried under the air-pump, and then heated for a few seconds on a sand-bath, assumed a gray color. This precipitate was soluble in ammonia and cyanide of potassium. Treated while hot with potassium hydrate, cyanide of potassium was formed, with which it was easy to obtain hydrocyanic acid and Prussian blue. Suspended in water, and subjected to the action of a current of hydrosulphuric acid, it gave rise to a clear and limpid solution of

[1] Medical Jurisprudence, London, 1865, p. 302.

[2] Canstatt's Jahresbericht für 1852, Bd. vii. p. 49.

[3] Prov. Med. Journ., July 23, 1845.

sulphocyanic acid when the sulphuret formed had been separated by filtration. By means of hydrochloric acid, hydrocyanic acid could be obtained from it of a very powerful odor, and the vapor of which formed a white precipitate in a solution of nitrate of silver; the precipitate was soluble in ammonia. The first precipitate, heated in a lamp, in a narrow tube closed at one end, gave hydrocyanic acid and a few drops of water, etc. This same precipitate, gently heated with caustic potassa, gave rise to no disengagement of ammonia.

In this case the hydrocyanic acid had remained in the stomach three weeks after burial. It did not appear to have entered into any chemical combination. There was a very considerable quantity of it, for, says M. Brame, "I was able to collect about 0.60 of cyanide of silver, or nearly 0.120 of hydrocyanic acid."[1] In the "Eaglesham" poisoning case it was detected in the stomach of the body which had been buried fourteen days;[2] and in a recent German case three weeks after death.[3]

§ 477. When no odor of prussic acid can be perceived emanating from the mouth, or after opening the body, from the contents of the stomach or intestines or from the serous cavities of the body, the vapor tests may show the presence of the acid. But even when the vapor tests do not reveal its presence, we must not infer positively that no acid is present, but must resort to the distillation process. This process consists in placing a portion of the suspected material, mixed with a sufficient amount of water if necessary to render it fluid, in a retort which is connected with a condenser, the tube of which ends in a flask or receiver. If the fluid is not acid, a sufficient amount of tartaric acid should be added to give it a decided acid reaction. The retort is then heated, care being taken not to allow the temperature to rise over 105° C. or 110° C. As soon as two or three cubic centimeters of the fluid have collected in the receiver, the latter should be removed and another one put in its place. In this first distillate, if prussic acid were present in the suspected substance, the odor can usually be perceived, even when it could not be detected in the original substance, and in some cases

[1] The Chemist, Feb. 1855, from Comptes Rendus, No. 20, Nov. 13, 1854, by M. Brame.

[2] Edinb. Med. Journ., iv. 163.

[3] Brit. and For. Med.-Chir. Rev., April, 1860, p. 531.

where the vapor tests could not be obtained; with this distillate the above tests should be employed both by the vapor and the liquid method. The distillation should be continued, until the distillate gives no further turbidity with a solution of nitrate of silver, and during the process the receivers should be changed as soon as a few cubic centimeters have been collected.

III. *Nitro-benzol.*

§ 478. Nitro-benzol, or the so-called "oil of mirbane," also contains a flavor and taste similar to that of bitter almonds or prussic acid. Small doses of this substance are poisonous when swallowed or inhaled. It was first mentioned by Casper in 1856 as a poison.[1] The vapor is more poisonous by inhalation than is the liquid when swallowed.[2] Poisoning by this substance differs from that of prussic acid, in that the symptoms come on more slowly and are not so pronounced. They come on with a general feeling of lassitude without convulsions usually. "The eyes are bright and glassy, the features pale and ghastly, the lips and nails purple, the skin clammy, and the pulse feeble."[3] After several hours, the patient becomes unconscious with stiffened muscles, followed by general convulsions. A fatal case[4] is reported by Mr. Nicholson. Dr. Taylor[5] reports a case of fatal poisoning which was the subject for an inquest in the Isle of Man, and another which was brought into the London Hospital. There were no immediate symptoms in the second, but the boy, aged 17, said that he felt drunk, and became stupid and comatose, dying without the occurrence of vomiting or convulsions twelve hours after he had swallowed the poison. Dr. Letheby reported another case where death was caused by inhaling the vapor of nitro-benzol, which was spilt over the wearing apparel of the victim. The symptoms were similar to those cases in which the poison was swallowed.

§ 479. The delay in the appearance of the symptoms is not always of long duration. It depends upon the condition of the nitro-benzol; if the poison is taken in pure form, it is not soluble in water, and therefore does not mix with the fluids of the stomach, so that it is

[1] Vierteljahrschrift, Bd. xvi. p. 1.
[2] Taylor, op. cit.
[3] Ibid.
[4] Lancet, 1862, i. p. 135.
[5] Op. cit.

absorbed very slowly, and the symptoms may be delayed for many hours after taking the poison, but, on the contrary, if the poison be taken in spirit in which it is readily soluble, the symptoms may come on almost immediately after swallowing it. The blue livid appearance of the skin and mucous membranes may appear sometime before the patient feels very sick. Vomiting very frequently occurs, the vomitus having the peculiar odor of nitro-benzol which resembles that of the bitter almond. The pupils are dilated and do not contract when exposed to a strong light. There is often pain in the stomach and abdomen, and incontinence of the urine and feces. In many cases, however, the blue color of the skin and the deep sleep are the only symptoms. Death may occur in four or five hours, but usually does not in fatal cases until after the lapse of from eight to twenty-four hours.

§ 480. The *post-mortem appearances* consist chiefly in the very powerful and very persistent odor of nitro-benzol about the body, the dark-brown or even black color of the blood, which also coagulates with difficulty. This is an important point in distinguishing nitro-benzol from prussic acid poisoning; in the latter the blood is usually cherry-red in color. The blood also, when examined with the spectroscope, shows the absorption band of hæmatin which is not present in normal blood.

§ 481. *Chemical examination.*—The principal tests for nitro-benzol are: (1) its powerful odor resembling that of bitter almonds; (2) its insolubility in water, so that it floats upon water in the form of oily drops; it can easily be removed from such a mixture by shaking with naphtha which readily dissolves it, and, upon allowing the naphtha to evaporate in a watch-glass at the ordinary temperature, the nitro-benzol will be left as a residue; (3) it can be converted into aniline by being dissolved in alcohol, and this solution treated with a little metallic zinc and hydrochloric acid; the nascent hydrogen thus evolved converts the nitro-benzol into aniline, which can be separated by shaking the mixture with ether, removing the ether and allowing it to evaporate, when the aniline will be left as a residue in the form of an oily fluid which will react to the ordinary tests for aniline.

§ 482. In legal cases the chemical examination enables us to readily distinguish between prussic acid and nitro-benzol. In the first place, in cases of poisoning by the latter substance, the odor

is perceptible for a much longer time than in prussic acid poisoning, and in the second place with this powerful odor we are unable to obtain the vapor tests for prussic acid, which would not be the case in prussic acid poisoning.

IV. *Aniline and Aniline Salts. Poisonous Action of Substances belonging to the Benzol Group.*

§ 483. From Dr. Starkow's investigations[1] it appears that the nitro-substitution products of these substances, if soluble, are more poisonous than the substances themselves, and produce a special action on the blood. The blood of an animal poisoned with bi-nitro-benzol, if subjected to the examination by the spectroscope, shows an absorption-band between the red and the orange in the spectrum, and corresponds to the Frauenhofer line *C*, in addition to the two normal oxyhæmoglobin bands; reducing agents, such as ammonic sulphide, change the position of this band a little to the right, and ammonic hydrate causes it to entirely disappear. The blood of an animal, poisoned with (mono-) nitro-benzol, or nitro-naphthaline, shows the same band, but if these substances are mixed with blood outside of the body, the band does not immediately appear as in the case of bi-nitro-benzol, but only after the lapse of several hours. These mono-nitro-substitution products are less active poisons than the bi-nitro-substitution products, but are much more active than the simple benzol, aniline, etc., or the chlorine substitution products, as chloro-benzol, none of which give rise to the above described absorption band, although chloro-benzol, when mixed with blood, causes a disintegration of the blood-globule, and the speedy appearance of hæmoglobin crystals.

§ 484. Aniline acts on the blood like ammonia and phosphuretted hydrogen, and decomposes the hæmoglobin, hæmatin being produced, whose peculiar absorption bands can be seen with the spectroscope either in the blood of animals poisoned by it, or in blood to which it has been added outside of the body. The chemical action of nitro-glycerine upon the blood-pigment is analogous to that of nitro-benzol, and the toxic action of both is nearly similar, three grains being sufficient to kill a dog of moderate size in two or

[1] Virchow's Archiv, iii. No. 4; and Journ. de Pharm. et de Chimie, April, 1873; abstract by the writer in Boston Med. and Surg. Journ., July 10, 1873.

three days' time. Nitric and sulphuric acids act on the blood in a manner somewhat similar to that of the nitro-compounds, the blood-pigment being decomposed and the hæmatin spectrum produced, an effect not caused by hydrochloric or phosphoric acids.

Thus the action of many of the organic nitro-compounds is evidently due to the radical nitryl which they contain, as nitro-glycerine, and the poisonous action of nitro benzol cannot be due entirely to the formation of aniline in the system, as has been stated by some writers.

V. *Nitro-Glycerine* (Glonoin), *Nitrite of Amyl, of Ethyl, and of Potassium.*

§ 485. Nitro-glycerine is a substitution compound formed by the action of nitric acid on glycerine. According to Dr. Taylor,[1] this heavy liquid has been used in Sweden in mining under the name of "blasting oil," and in some instances by mistake has been taken by the miners with serious consequences. In one case a miner swallowed "two mouthfuls," which was followed by a painful feeling in his throat, after which he drank a quantity of milk; when seen by a physician in an hour and a quarter after swallowing the liquid, he was suffering with faintness, difficult breathing, and oppression at the chest. In five hours vomiting and purging set in, and shortly before death the lips became livid, and the man lay quietly as if asleep, breathing feebly and occasionally with a deep sigh. The *post-mortem* examination revealed great congestion of the brain, as well as of the lower lobes of the lungs. The lining membrane in the air passages had a reddish-brown tint. The greater curvature of the stomach presented a similar appearance with some ecchymosis.[2] A case attended by serious symptoms caused by homœopathic pellets of Glonoin (supposed to be the first trituration) was observed by the writer. As nearly as could be ascertained about thirty to fifty of these pellets were swallowed by a boy of three years of age, who was found lying on the floor conscious and calm. He was seen by the writer within twenty minutes after the time he was supposed to have swallowed the pellets; he was lying upon a sofa with pallid but peaceful countenance, feeble,

[1] Treatise on Poisons, Phila., p. 642.

[2] Dr. Taylor has taken the above account from Husemann's Jahresbericht, 1872, p. 533.

rapid pulse, and slow movements in breathing which were not full, but shallow. He could not cause any contraction of the muscles below the waist, and could not stand. He complained of a slight headache, but there was no other apparent symptom. No remedy was given, but in six or seven hours he was up on his legs, and had no further serious trouble.

§ 486. Nitro-glycerine (known under the popular name of Glonoin) is a nitro-substitution product, acting somewhat like nitrite of amyl, potassium, or ethyl; these nitrites probably cause serious symptoms from the presence of nitrous acid in their feeble combinations.[1] Taylor's statement that it is insoluble in alcohol is probably a mistake, since its alcoholic solution (one per cent.) is kept by many apothecaries in England and in this country, though the substance itself is not mentioned in the Pharmacopœia. The medicinal dose of this solution is stated as half a drop to two or three drops; a single drop has caused unconsciousness, abolition of pulse and insensibility, followed by rapid recovery.[2] The effect of inhalation of the volatile nitrites is intense suffusion of face, fulness of head and unconsciousness; at first, the pulse is very rapid and tumultuous, though feeble, but the blood pressure is weak, and the bodily temperature remarkably lowered. These symptoms increase for a few moments after the inhalation has ceased. Though the effects of nitrite of amyl and other volatile nitrites are very rapid and apparently severe, instances of death following their use are rare, and there is no recorded case of their criminal use. Death from this class of drugs would be caused by asphyxia, and the *post-mortem* appearances in the blood, as shown by the spectroscope, are due to this cause, the chocolate-colored blood from poisoned animals showing in the spectrum the absorption bands of methæmoglobin.[3]

§ 487. *Chemical examination.*—Nitro-glycerine, or more properly speaking, trinitro-glycerine, can be easily detected by its explosive

[1] Richardson, in British and Foreign Medico-Chirurg. Review, July, 1867.

[2] Brit. Med. Journ., 1880, i. 406.

[3] Hoppe-Seyler, Zeitsch. für Physiol. Chem., Bd. ii. ū. iii., quoted by Dr. Wood in his Mat. Med. and Toxicology, Phila., fourth ed., p. 359.

For detailed and elaborate experimental studies with amyl nitrite, reference is made to the following works: T. Lander Brunton, Journ. of Phys. and Anat., vol. v.; H. C. Wood, American Journ. of Med. Sci., July, 1871; Amez Droz, Archives de Phys. normale et pathologique, Sept. 1873; Pick, Central, f. d. med. Wissen., 1873.

properties when heated or struck; for the performance of this test a capillary tube containing a very small fraction of a drop of the suspected substance may be introduced into the flame, when it will either explode or flash brilliantly. By boiling with potassium hydrate, nitrate of potassium is produced, and can be obtained in crystalline form. By long contact with hydriodic acid or sulphuretted hydrogen water it is decomposed, and glycerine is formed. Nitro-glycerine also responds to some of the tests for nitric acid, especially those with sulphate of iron and brucine in the presence of sulphuric acid (see § 52). Nitro-glycerine is insoluble in water and dilute alcohol, but is soluble in concentrated alcohol (at least 95 per cent.), ether, methylalcohol, amylalcohol, and chloroform.

§ 488. Nitro-glycerine can be *isolated* from *organic mixtures* by treating the mixture with absolute alcohol until the whole has an alcoholic strength of at least 95 per cent. after rendering feebly acid with sulphuric acid, allow this to digest for twenty-four hours, filter and evaporate until about one-sixth of the original volume remains; this residue should be shaken with ether which will remove the nitro-glycerine from the fluid residue, and upon being allowed to evaporate spontaneously will leave the nitro-glycerine in the form of an oily fluid in the watch glass; this residue can be tested as above mentioned.

VI. *Aconite.* (Monkshood; Wolfsbane.)

§ 489. *Symptoms.*—The leaves and root of *Aconitum napellus* contain one of the most extraordinary and fatal poisons known. The former have proved fatal when eaten by mistake for salad; and the latter, from its resemblance to horseradish, has given rise to many unfortunate accidents. The root is tapering, about the thickness of the finger at its upper part; its color externally is brown, internally it is white and fleshy. Its taste is bitter, but after a few minutes a marked numbness and tingling are perceived on the lips, tongue, and fauces. The leaves, when chewed, have the same taste, and produce the same feeling of numbness.[1] Dr. Isaacs has reported the case of an apothecary's clerk poisoned by the inhalation of the dust of aconite-root, which he was pulveriz-

[1] Pereira. For several cases, *vide* Headland, Lancet, March, 1856, p. 341.

ing. The effects of the drug were first manifested by numbness and a prickly sensation of the tongue and difficulty of swallowing, with dryness and a sense of constriction in the fauces. About an hour afterwards there was some difficulty of respiration, with diminution of the force and frequency of the pulse, greatly dilated pupils, loss of voice, and prostration of strength. Very slight convulsions occurred, at repeated intervals, for about five hours, when the patient was supposed to be dying. The countenance was hippocratic, and the pupils very greatly dilated, the pulse 36 and feeble, and the breathing correspondingly slow. There was also, from the first, great oppression in the cardiac region. Two weeks afterwards the aphonia still continued.[1] The pharmaceutical preparations most in use, and which, therefore, are most apt to be either accidentally or intentionally employed, are the ordinary and the saturated tinctures, the alcoholic and fluid extracts, and the abstract of aconite. There being several formulæ for these preparations, they are variable in strength, owing to the variable quantity of aconitine contained in the prescribed dose. Thus, two persons, who took twenty-five minims of the tincture, died;[2] while another, who swallowed an ounce and a half of the tincture, prepared according to the French Code, survived. The active principle, called *aconitine*, which is very variable in its poisonous strength, prepared by Mr. Morson, of London, is so powerful that, according to Pereira, one-fiftieth of a grain has endangered life; but, on the other hand, a case is reported by Dr. Golding Bird, in which, although two grains and a half of this alkaloid were taken, the patient recovered, after having very dangerous symptoms. Pereira states, also, that there is a spurious aconitine, which is inert or nearly so, sold in the shops, since he took one grain of it without perceiving the least effect upon the tongue or otherwise.[3] The effect of a slight increase in the medicinal dose is well seen in the following case, communicated to Pereira by Dr. Redfern. The patient, who was suffering with acute articular rheumatism, took

[1] New York Journal of Medicine, Sept. 1859, p. 191.

[2] The tincture made from the root is about five times stronger than that from the fresh leaves.

[3] The preparations sold under the name of this alkaloid are altogether unreliable; a number of experiments were conducted by the editor with a sample which appeared to be not much stronger than the powdered root itself.

five drops of the tincture three times a day for two days, without marked relief. On the third day the dose was increased to six drops, at the same intervals. Two doses of this amount were taken; and an hour after the second he was found in a state of extreme restlessness, and complaining of great pain in various parts of his body. To use his own expression, he felt as though his skin were too tight for his body. He described his sensations as intolerable. At this time there was much frothing at the mouth with violent retching at intervals. The surface of the body was cold, and bathed in profuse perspiration, which ran down his face in streams. The pulse, though at first 150 in the minute, fell to between 50 and 60 in a few minutes, and was so small and com pressible as scarcely to be felt at the wrist. He recovered under the use of brandy and water and external warmth. The following case of poisoning by aconite and chloroform simultaneously swallowed presents so striking a description of the poisonous effects of aconite that it is reproduced in detail from the report of Dr. John E. Blake:[1] "A young lady . . . . took by mistake something more than one drachm of a mixture containing equal parts of the tincture of aconite root and chloroform." (The chloroform would probably have no controlling effect upon the deadly action of the aconite.) " . . . . The burning taste of the chloroform caused the error to be at once perceived. The patient took a potion of mustard and water, and immediately walked to my house, accompanied by a young lady friend, ... about one-eighth of a mile. I allow for walking this distance not more than five minutes, and believe that not more than fifteen to twenty elapsed from the time the dose was taken before I saw her. She had the presence of mind to bring the bottle with her, by which I was at once informed of the exact nature of the poison, presuming from the word liniment" (on the label) "that the aconite was of the *root*, and was probably in large amount. This presence of mind on the part of the patient, and the perfect calmness which never deserted her, and which was also exhibited in a striking manner by her friend, helped me much in this sudden emergency, and contributed to save her life."

"There were absolutely no symptoms at this time. I had in

[1] New York Medical Journal, April, 1875, p. 371.

my pocket between fifteen and twenty grains of sulphate of zinc, which I gave her. . . . . I went down stairs for a stomach-pump. Returning to the chamber, and finding that no vomiting had taken place, I introduced the tube of the stomach-pump at once, and pumped in something over a pint of warm water. On drawing this water back, it returned so impregnated with the poison that the strong odor of chloroform was very perceptible. The aconite gave the water a dark tinge. I thus passed rapidly through the stomach nearly two quarts of water, and am convinced that none of the poison remained. Enough, however, had been absorbed to do deadly work, and numbness of the tongue and cheeks began to be perceived, before the operation of evacuating the stomach was finished, rapidly extending itself over the shoulders and back and down the arms. . . . . Stimulants were administered, which Miss C. was able to swallow. . . . . I had scarcely left the bedside . . . . when the patient, articulating with great difficulty, said: 'All is dark now, Doctor, I cannot see at all;' and at once became unconscious. Not more than two minutes before this she had been able, with slight assistance, to totter, with faltering and uncertain step, from the sofa to the bed. . . . . Dr. Thomas arrived . . . . an hour after the poisonous dose was taken; . . . . the patient's situation . . . . was, briefly, as follows: her breathing had become imperceptible, she was growing cold, and she was *pulseless*. *No pulse whatever could be detected, even in the axilla, and she remained without any trace of pulse for a period of over three hours.* This is a point about which there can be no mistake, one to which our attention was *carefully* and *constantly* directed." By artificial respiration with oxygen gas, by electrical stimulation, and by keeping the head low, the patient gradually recovered from this death-like syncope, the pulse at the wrist showing an almost imperceptible thrill more than three hours from the time the dose was taken. This reaction was succeeded by a comatose condition, with an over-excitability of the skin, a feeble current of the electric battery caused her to cry out. Five hours after the commencement of the poisonous symptoms, some urine was obtained and examined; this resulted in finding it loaded with albumen and fragments of renal casts. The electrical stimulation was continued at times during the succeeding eleven hours, "since, if discontinued for more than ten minutes, the pulse

would flag at once, and coma come on." Some urine, collected again seventeen hours after the beginning of the poisoning, showed copious amounts of albumen and casts, but during the next few days these gradually disappeared. "For several days she could recall nothing whatever of what had passed, and could not imagine how she came to be where and as she was. Slowly, however, all has come back, and she remembers pretty well all that occurred up to the time of her being insensible." She fully recovered. Other cases of poisoning are referred to in the foot-note.[1]

§ 490. The first and most usual *symptoms* are a burning and numbness of the lips, mouth, throat, and stomach, followed by tingling in various parts of the body, loss of sensation, vertigo and dimness of vision, tremors, great prostration, sense of fulness in the throat, speechlessness, hurried respiration, and death in a state of collapse. Vomiting and purging are also usual symptoms, but are not observed in every case. General convulsions are unusual, as we find that, in fifty-three cases collected by Dr. Tucker, of New York,[2] they are mentioned as having occurred only in seven. In four out of twelve more recent cases there were convulsions. The mind remains perfectly clear, there being in general neither stupor nor delirium. The latter symptom was seen in three cases of the number collected by Dr. Tucker. When applied to the eye, the preparations of aconite are said to have the effect of contracting the pupil. In seventeen out of twenty cases, however, in which the poison was swallowed, it is stated by Dr. Tucker that the opposite effect was observed. Sometimes the sight is temporarily impaired. The symptoms of poisoning by aconite usually arise within a few minutes after it has been taken ; and when death takes place, it is, in the majority of cases, within three hours. The quantity of aconite, or any of its preparations, capable of pro-

[1] Stewart, Case of poisoning with aconite and fusel-oil, Edinburgh Med. Journ., 1879, 904; O'Brien, Case of poisoning by aconite, Med. Rec., N. Y., 1879, 128; Carleton, Case of aconite poisoning, Bos. Med. and Surg. Journ., 1879, 544; Elliot, Case of aconite poisoning, Lancet, London, 1878, ii. 917; Pike, idem, 1880, i. 12; Hardman, idem, 1882, i. 37; Samson, A case of poisoning by aconite, Chicago Med. Rev., 1882, 91; Van Peski, Empoisonement par le nitrate d'aconitine, Gaz. Hebd. de Sci. Med. Montpellier, 1880; Unique case of poisoning by aconite, Brit. Med. Journ., 1881, 64.

[2] For these valuable tables, *vide* New York Journal of Medicine for March, 1854.

ducing death, is, for the reasons before given, unknown. The case related by Dr. Easton,[1] in which twenty-five minims of the tincture were taken, shows probably the smallest dose which has proved fatal. Another case, in which twenty-five drops proved fatal, occurred in January, 1853. A gentleman, feeling himself unwell, stepped into a drug store, and was given by a medical student, a friend of his, who was attending, this amount of the tincture, under the supposition it was the proper dose. He expired about four hours after taking it, with the symptoms of poisoning by aconite. Dr. Male, of Birmingham, it is stated, died from the effects of not more than *eighty* drops, taken in ten doses, during a period of four days, the largest quantity taken at once being *ten* drops. An excise officer in England died in consequence of tasting Fleming's tincture of aconite. He said he thought he had swallowed about a teaspoonful. He did not complain at the time, but in the course of a few hours was cold and pulseless. The remedies applied were ineffectual, and he soon expired.[2]

§ 491. *Post-mortem appearances.*—The few cases in which *post-mortem* examinations have been made, have revealed nothing peculiar, the most constant appearance being congestion of the vessels of the head and of the lungs. It is, of course, evident that no conclusion can be drawn from such imperfect data as these.[3] Of the fifty-three cases collected by Dr. Tucker, in which aconite in

[1] Glasgow Med. Journal for July, 1853.

[2] Am. Journ. Med. Sci., April, 1852, p. 553.

[3] In the trial of John Hendrickson, Jr., for the murder of his wife by poisoning, at Bethlehem, Albany County, New York, Dr. Swinburne, who made the post-mortem examination, inferred from the emptiness of the stomach and small intestine; the corrugation of their mucous coat, and the presence of a reddish, viscid mucus in the stomach, that *vomiting* had taken place, and that this vomiting was produced by *aconite!* Dr. Salisbury, who had charge of the chemical analysis of the organs of the deceased, testified that he tested a *small portion of the stomach and a small portion of the duodenum* for prussic acid, most of the mineral poisons, then for morphine, strychnine, "stramonine," and other poisons, none of which he discovered. He then inferred the presence of *aconitine*, from the fact that, after digesting a small portion of the stomach and duodenum in alcohol, evaporating, filtering, and purifying, finally with *animal* charcoal, and then testing the filtered solution by boiling in sulphuric acid, it was "*turned to a deep port-wine red color.*" We beg leave to refer the reader for well-merited strictures upon the medical and chemical evidence given upon this trial, to the candid and able review of it by Prof. Charles A. Lee, in the American Journal of the Medical Sciences for Oct. 1844.

various forms and in all variety of poisonous doses was taken, twenty-seven recovered, and twenty-six died. In all those who recovered, emetics and external and internal stimulants were employed.

From a number of experiments performed upon animals, it would seem to us as if many of the pathological effects, especially convulsions, noticed or observed by other writers might be explained by the asphyxia caused by this agent, and especially on account of the persistent pulsation of the heart for twenty minutes after the respiratory efforts had been suspended. It was also observed that the vomiting occurred most generally when the vital powers were much reduced; and that after death, very slight, if any, signs of irritation of the internal coat of the stomach were apparent.

§ 492. *Chemical examination.*—The detection of the poison in cases of aconite poisoning depends upon the discovery of portions of the plant, if such were taken, by the microscope, upon the isolation of the alkaloid aconitine, upon getting the peculiar physiological effect, the numbness and tingling sensation upon the lips, tongue, or skin, when a portion of the extract is applied to these surfaces, and, when a sufficiently large amount of poison is present, upon getting the complete physiological effect by administering a portion of the isolated alkaloid to an animal. The exact chemical nature of the alkaloid aconitine is still somewhat in dispute, and there are several different preparations of it in the market, these differing in their activity and in their chemical properties. The alkaloid, which is prepared according to the directions given in the German Pharmacopœia, and which is called the "German aconitine," has the following properties: It is but slightly soluble in water, requiring about fifty parts of boiling water for its solution; it is quite soluble in alcohol, ether, and chloroform, but insoluble in naphtha; it is also soluble in benzol and amylalcohol; it has an alkaline reaction, and is soluble in dilute acids, from which solutions, when not too dilute, it can be partially precipitated by ammonia water, potassium hydrate, and potassium carbonate. When aconitine is gently heated in a watch-glass with one or two cubic centimeters of phosphoric acid solution, the solution assumes a reddish color, and, if very carefully heated to a higher temperature, a violet. Concentrated sulphuric acid dissolves aconitine, forming immediately a yellow-colored solution, which, if a considerable amount of the alkaloid is present, changes soon to a brown and gradually passes

through a reddish-brown to a violet, then to a colorless solution in twenty-four hours; this test is delicate for $\frac{7}{10}$ths of a milligramme (about $\frac{1}{100}$th of a grain). Phosphomolybdic acid precipitates aconitine from very dilute solutions in the form of a gray precipitate which becomes bluish after a while. Tincture of iodine, terchloride of gold, tannic acid, and Mayer's reagent precipitate it from moderately dilute solutions, and corrosive sublimate, potassium bichromate, picric acid, and sulphocyanide of potassium from concentrated solutions.[1] The principal reliance must, however, be placed upon the physiological test, $\frac{1}{100}$th of a grain sufficing to produce the peculiar sensation when applied to the skin, and $\frac{1}{1000}$th of a grain when applied to the tongue. Dr. Headland has proposed the application of the physiological test to the detection of this poison. If $\frac{1}{20}$th of a grain be obtained it will be enough; $\frac{1}{300}$th of a grain will poison a mouse with characteristic symptoms; $\frac{1}{100}$th, a small bird; $\frac{1}{1000}$th of a grain causes tingling and numbness on top of the tongue; $\frac{1}{100}$th, dissolved in spirit and rubbed into the skin, causes loss of feeling lasting for some time.[2]

§ 493. The *isolation* of aconitine from *organic mixtures* is best accomplished by Dragendorff's method.[3] If we are tolerably certain from the symptoms or history of the case, that aconite or aconitine was the cause of the poisoning, the process can be shortened by only shaking the acid solution with naphtha to remove certain impurities, then after removing the naphtha, rendering alkaline and shaking quickly with benzol, which can be decanted, after it has separated from the water, into several watch-glasses and allowed to evaporate; the residue in one watch-glass should be used for the sulphuric acid test, that in another should be dissolved in a drop or two of very dilute sulphuric acid (1 : 50) and the aconitine precipitated by phosphomolybdic acid, that in a third dissolved in the same way and treated with a drop of platinic chloride, which will not precipitate aconitine but will a large number of other alkaloids, and that in a fourth should be dissolved in dilute acetic acid for the performance of the physiological test.

§ 494. Aconitine resists decomposition of the animal tissues for quite a long period, and has been detected by Dragendorff in the

[1] Dragendorff, op. cit., p. 202.

[2] Lancet, March, 1856, p. 343.

[3] See § 348, p. 356.

blood one month and twenty-three days after death, and in the stomach two months and nine days after death. It can be isolated from all of the tissues and fluids, and has been detected not only in the contents of the stomach and intestines, but in the urine, feces, liver, blood, and kidneys. The remarkable symptoms occasioned by the plant, and the discovery of a portion of that which has been taken, will generally be sufficient to explain the cause of death. The only case in which it was criminally administered is that which is related by Dr. Geoghegan, where the deceased had eaten for his dinner some greens dressed by the prisoner. The latter was convicted upon the medical and general evidence, no trace of the poison having been discovered in the body. He confessed before his execution that the powdered root of aconite had been mixed with pepper, and sprinkled over the greens.[1] A recent case of poisoning by the alkaloid aconitine has occurred in England, and may be found in detail in the Appendix.

VII. *Lobelia Inflata.* (Indian Tobacco.)

§ 495. This plant, which in the hands of empirics has been productive of so much mischief, is very similar in its effects to ordinary tobacco. It is a powerful nauseating emetic. It causes severe and speedy vomiting in most cases, attended with continued and distressing nausea, sometimes purging, copious sweating, and great general relaxation, extreme prostration, anxiety, contracted pupils, insensibility, and occasionally death preceded by convulsions. As an emetic, the dose is from ten to twenty grains, as an expectorant, from one to five grains. The poisonous principle, called lobeline, a viscous, transparent oil with a strong alkaline reaction, is a volatile alkaloid, and has been obtained in a pure state by M. Bastick.[2] The trial of Samuel Thomson for the murder of Lovette by this drug is given in considerable detail in the chapter on "Malpractice."[3] Other cases of death from its administration have occurred, both in this country and in England, where the peculiar practice of Thompson has been extensively introduced by a person named Coffin, and is there called "Coffinism." Dr. Letheby, in his testimony upon

[1] Dublin Journal, July, 1841.

[2] Gaz. des Hôp., July, 1851.

[3] § 1083 (old edition, third). In this case the symptoms of poisoning by lobelia are well described.

an inquest held upon a man who died from the effects of a lobelia emetic given him by a greengrocer, stated that within three or four years there had been, in England, *thirteen* cases of poisoning with it.[1] When any of these cases were brought to trial, however, the culprits usually manage to escape. But in 1856 one was convicted and sentenced to three months' imprisonment.[2] Dr. Beck says "that thousands of individuals in the United States have been murdered by the combined use of capsicum and lobelia, administered by the Thomsonian quacks." "The founder of what has been called '*Coffinism*,'[3] an individual who styles himself, 'A. S. Coffin, M.D., Professor of Medical Botany,' declares in his 'Botanical Guide to Health, and the Natural Pathology of Disease,' 17th ed., 1850, that lobelia 'is not a poison,' 'that it never operates upon those who are in perfect health;' and he says that the powdered leaves or pods may be given in doses of a teaspoonful every half hour, in a cup of vervain tea or pennyroyal, and repeated until it operates as an emetic; and he adds, 'Never mind Hooper, but give enough'!!"[4]

§ 496. *Symptoms.*—When lobelia is given in large doses (more than a fluidrachm of the wine or fluid extract), symptoms of headache, nausea, and vomiting, distress at the pit of the stomach, and a difficulty of breathing, dependent upon the constraint of the respiratory muscular movements, will supervene. These are soon followed by difficulty in swallowing and speaking, the voice sounding hoarse and feeble; the pulse becomes feeble and irregular; muscular weakness, heavy sleep interrupted by hallucinations occur, with peculiar prickly sensations in the skin and pain in the urethra accompanied by diuresis. The action of lobelia is cumulative, or in other words, the depressing poisonous effects are apt to follow the continued use of medicinal doses. One large dose provokes vomiting and an increased flow of urine with diarrhœa, which need not necessarily prove fatal.

§ 497. Lobeline (the active principle of lobelia) has been obtained by Mr. Procter by the process described in the United States Dis-

[1] Med. Times and Gaz., 1853, ii. 568.

[2] Taylor on Poisons, 3d Am. ed., p. 736.

[3] *Vide* Pharm. Journ., Sept. 1, 1849, and Feb. 1, 1851.

[4] Pereira. For cases, *vide* Med. Gaz., Aug. 1849 and 1850; Lancet, 1849, June, 1850, and Pharm. Journal, Aug. 1851; also Med. Gaz., 1851, p. 384. See also a case of poisoning in Appendix.

pensatory (Wood & Bache). This alkaloid is a yellowish liquid, lighter than water, of somewhat aromatic odor, and very acrid durable taste. It is soluble in water, but much more copiously in alcohol and ether; and the latter readily removes it from its aqueous solution. It has an alkaline reaction, is soluble and forms crystallizable salts with sulphuric, nitric, and hydrochloric acid, and a very soluble but uncrystallizable salt with acetic acid. It forms an insoluble compound with tannic acid, which instantly precipitates it from its solutions. By a boiling heat it is entirely decomposed, losing all its acrimony; but when combined with acids, it may be boiled in water without undergoing a change. By experiments upon animals it seems to give the narcotic without the emetic action of the plant lobelia.[1] Its action resembles that of the alkaloid of tobacco, called nicotine. Prof. Procter introduced a grain dissolved in water into the stomach of a cat; the animal immediately seemed weak and feeble, and remained without muscular movement for an hour with dilated pupils. It did not vomit nor have diarrhœa, and did not recover for fifteen hours.

§ 498. *Chemical examination.*—In addition to the properties mentioned in the preceding section, lobeline is readily soluble in naphtha; if the naphtha solution be poured into a watch-glass, which has been previously moistened with hydrochloric acid, and allowed to evaporate, the residue which remains will not be crystalline, but will be a yellowish amorphous mass; this property distinguishes it from coniine; if this residue be immediately dissolved in a little acidulated water, it will be precipitated by Mayer's reagent and by phosphomolybdic acid. Its solutions in water are precipitated by a solution of bichloride of platinum, terchloride of gold, corrosive sublimate which gives a precipitate soluble in an excess of the reagent, solution of iodine in iodide of potassium, potassium bichromate, and picric acid. The chloride of lobeline treated immediately with Fröhdes reagent produces after about two minutes a deep violet color, which gradually increases for about two hours and remains unchanged for about twelve hours, but later changes to a brown and

[1] Am. Journ. of Pharmacy, ix. p. 105, and xiii. p. 1.

Lobelia has been used in certain quack medicines as an anti-tobacco agent, to prevent by its substitution the desire for chewing. In this way some persons have suffered from the narcotic effects of this drug.

yellow ; if the chloride residue has been exposed to the air for some time, it does not give this reaction.

§ 499. Lobeline can be *isolated* from *organic mixtures* in the same way as nicotine and coniine.

Volatile or Essential Oils.

§ 500. These are more or less narcotico-irritant poisons, in addition to the specific action belonging to any individual one belonging to this class.

VIII. *Cedar Oil.*

§ 501. The oil of the common juniper (Juniperus Virginiana) has an action upon the system similar to that of savin, except that it appears to have a more decided narcotic influence. Dr. Wait reports four cases of poisoning by this oil, two of which proved fatal. The quantity taken in each case was from half an ounce to an ounce, and in three of them it was swallowed with the view of bringing on abortion. The patients were seized with convulsions, and vomited a fluid having a strong odor of the oil. After the convulsions had subsided, they fell into a comatose condition. The *post-mortem* appearances in the two fatal cases were not very striking. There were several small red patches upon the lining membrane of the stomach, and the duodenum showed marked signs of inflammation. The uterus in each case was in a healthy, gravid state. The odor of the oil was distinctly perceived on opening the stomach.[1]

IX. *Savin.*

§ 502. The leaves of this plant have, in the fresh state, a strong, peculiar, and heavy odor, especially when rubbed, and a nauseous, resinous, and bitter taste. The dried tops are of a yellowish-green color, and are less odorous than the fresh ones. The *oil of savin* is a limpid, almost colorless liquid, having the unpleasant odor of the plant, and a bitter, acrid taste. The medicinal dose is from two to six drops. But the use of the oil or of the dried leaves of this plant in medicine, is exceedingly restricted. From the frequency, however, with which it is resorted to for the purpose of procuring abortion it is necessary to notice its effects. The oil of savin and

[1] Boston Med. and Surg. Journ., 1849.

the fresh leaves, when applied to the skin, exercise a powerful rubefacient and even vesicant action. Swallowed in large doses, it occasions vomiting, purging, and other symptoms of gastro-intestinal inflammation. Administered internally in small quantity in the form of the infusion of the leaves or of the essential oil, the tongue perceives a peppery or bitter taste, which is followed by a persistent sensation of freshness on the lips and slight twinges in the stomach.[1] The irritating or pungent juice seems to reside in a little gland situated on the back of the leaves in a shallow groove. When administered in large doses, a fluidrachm or more, signs of irritant poisoning are manifested, such as heat at the stomach, epigastric and abdominal pains, vomiting of greenish matters, abundant and often bloody stools, flow of saliva, and in fact the well-known signs of gastro-intestinal irritation. The nervous phenomena are those of a general stimulation followed by prostration and collapse. The circulation is at first excited and then becomes feeble, accompanied by a sensation of cold with a rapid and small thready pulse. The respiration, at first hurried, becomes slow and stertorous, and insensibility then succeeds, sometimes preceded by convulsions. In the first period of excitement or stimulation the flow of urine is increased and the strong odor of sabine is quite distinct in it; oftentimes the urine contains blood, and hemorrhage may occur from nose or lungs; but the congestion is principally noted in the genito-urinary apparatus, and hemorrhage may take place from the uterus, and, in the pregnant female, the fœtus may be expelled, though this does not always happen. It seems pretty evident that abortion does not occur except after dangerous and fatal doses, as suggested by Tardieu and Hamelin; yet the researches of Goltz, Schlœsinger, Hoffmann, and Van Basch ascribe to sabine the property of a direct stimulation upon the uterine organs through the spinal nerve centre. Whatever may be the explanation of its action, there is no doubt of its causing a decided local congestion of the pelvic viscera and the genito-urinary organs, from which there is a relaxed condition of the involuntary muscular fibres, and differing in this latter respect from rue, which causes strong contraction of the muscles. Pereira says, that, according to his observation, it is the most certain and powerful emmenagogue of the whole materia medica.

[1] Hamelin, Dict. Encycl. des Sciences Médicales, Paris, 1878.

He quotes, from a German author, the case of a woman who swallowed an infusion of savin to occasion abortion. Violent and incessant vomiting was induced, which was followed in a few days by excruciating pain in the abdomen, abortion, dreadful hemorrhage from the uterus, and death.

Two other fatal cases are given by Dr. Christison, in one of which abortion was produced. In two others, related by Dr. Taylor, the women being respectively in the seventh and eight month of pregnancy, violent and fatal gastro-intestinal inflammation was induced, and abortion followed. In one of these cases, furnished by Mr. Letheby, the symptoms resembled those of narcotic poisoning; the woman was found lying on her back, perfectly insensible, and breathing stertorously. Although the power of producing abortion cannot be denied to this drug, it is the result of general observation that this effect ensues only when it is taken in such doses as to endanger life by the violent inflammation set up in the stomach and intestines, and that it may even then destroy the life of a pregnant female without bringing on the premature expulsion of the child. In a case reported by Dr. Hinds, a woman five or six months advanced in pregnancy brought on premature labor by repeated doses of oil of savin. Before and after this event she suffered greatly from purging, vomiting, and intense pain, which terminated in a dangerous attack of peritonitis. She was actively treated, and recovered.[1]

Mitscherlich has made some experiments on animals[2] which show that the oil of savin is a powerful poison. It is absorbed into the system, as he determined from its peculiar odor, which he detected in the exhalations from the lungs, the cavities of the body, in the blood, and in the urine. The bloodvessels of the intestinal canal, after death from this drug, were injected, and the epithelial coat was in places thrown off; but, according to Mitscherlich, the signs of inflammation in the alimentary canal were not so great as to induce the belief that death from poisoning by the oil of savin was induced by its local action; he inclined to attribute the fatal results to the absorption of the poison itself. He noticed, also, a congestion of the kidneys and that muscular irritability (contractility) continued for a long time after death.

[1] Times and Gazette, Nov. 1857, p. 524.

[2] Mitscherlich, ii. p. 659.

There is a case related by Kopp,[1] of an infant who died from the effects of breathing an atmosphere charged with the vapor of oil of savin, which had been left all night in a bottle uncorked near the cradle.

§ 503. *Post-mortem appearances.*—After death there are found, in general, undoubted evidences of inflammation of the stomach and intestines. In one of Dr. Christison's cases, the inside of the stomach was red with patches of florid extravasation, and there was extensive peritoneal inflammation, with fibrinous effusion. The contents of the stomach had a green color. In the case communicated by Dr. Salisbury to Dr. Beck (*vide* "Abortion"), where the examination was made from twelve to fourteen hours after death, the stomach was found softened and *perforated*, its contents emptied into the cavity of the abdomen, and there were signs of extensive peritonitis. The perforation was about the size of a fifty-cent. piece, and was situated in the region of the greater curvature, near the cardiac orifice. For several inches around the perforation, the stomach was very much corroded, thinned, and softened, so that it was easily torn. The œsophagus and upper part of the small intestines are described as inflamed. Evidence of the presence of savin in the intestinal canal was obtained, and a phial was discovered in the room, still containing half a drachm of the oil of savin and tincture of lavender. In a case occurring to Mr. Lord, of Hampstead, the œsophagus presented a dark, aborescent injection, with slight patches of ecchymosis, and in the stomach there was a large patch of redness about three inches in length; the vessels of the mucous membrane were considerably injected, forming infiltrated patches, especially about the lesser curvature, but there was no ulceration or erosion. Here, also, a large quantity of a greenish fluid was found, of the appearance and consistency of green pea soup, which was found on examination under the microscope to be due to the presence of finely triturated savin powder. The intestines, also, were highly inflamed, the duodenum being of the color of cinnabar, and there was also some peritonitis.

§ 504. *Detection.*—According to Pereira, powdered savin may, on account of its green color, be mistaken for bile, but, when mixed

[1] Richter, Aüsfürlich, Arzneim, Supp. Band., p. 408.

with distilled water, it entirely subsides, and provided no bile be intermixed, the supernatant liquor will be devoid of a green color.

If savin have been given in the form of infusion or decoction, it may be impossible to detect it, but, when the oil has been administered, it may be separated by distillation. Furthermore, it has been already stated, savin in powder may be recognized by means of the microscope, the circular pores being visible, and the acuminated shape of the leaves. The odor, also, may aid in its recognition.

X. *Taxus Baccata.* (Yew.)

§ 505. The leaves and berries of the common yew have been known for ages as poisons. Although Orfila gave them to animals in many cases without effect, numerous cases of accidental poisoning by them are known. It is usually classed among the acrid narcotics, although, in most cases of poisoning by it, which have been reported, acridity has appeared to be the least essential of its properties. In the case of a lunatic who died fourteen hours after chewing yew leaves, the symptoms were giddiness, sudden prostration of strength, vomiting, coldness of the surface, spasms, and irregular action of the heart.[1] Similar effects were seen in a child who died four hours after eating the berries.[2] Brandis says, a young woman took, as an abortive, the leaves of the yew, and fell into the sleep of death without convulsions. Indeed, one might have supposed her to be really sleeping, for her cheeks preserved the hue of life, and a quiet smile played over her face.[3] In Henke's Journal,[4] an interesting history is given of the poisoning of eleven persons by a decoction of yew leaves. They had partaken of it as a prophylactic against hydrophobia, some of their dogs having been bitten by one supposed to be rabid. In half an hour all of them were seized with giddiness, confusion of sight, pain in the head, nausea and vomiting, and then fell asleep. Two of them, however, died within about an hour, without either pain or convulsions, but with a smile upon their countenance. The rest recovered without further symptoms. The *post-mortem appearances*, in these and the preceding cases, threw no

[1] Dr. Mullan, Dub. Hosp. Gaz., 1845.
[2] Hurt, Lancet, Dec. 10, 1836.
[3] Blumenbach's Med. Bibliothek, Bd. iii. p. 684.
[4] Erg. Heft. 43, p. 127.

light upon the manner in which the poison affected the system, except from the negative evidence of the absence of any well-marked signs of inflammation. In Mr. Hurt's case, however, it is stated that, besides patches of redness upon the mucous membrane of the stomach, it was also much softened.

XI. *Oil of Tansy.* (Tanacetum.)

§ 506. The few cases that have been reported of poisoning by the oil of tansy indicate that its appropriate position is among the narcotico-irritant poisons. It has been often taken for the purpose of inducing abortion, but does not seem to possess this property, which is popularly attributed to it. A fatal case of poisoning with half an ounce of this oil is recorded in the *Am. Journ. Med. Sci.* for May, 1835. Frequent and violent clonic spasms were experienced, with much disturbance of respiration. No signs of inflammation in the stomach and bowels were found upon section. Death occurred in two hours after taking the poison.

§ 507. The *symptoms* of poisoning by the oil of tansy are a sense of heat and discomfort in the stomach and bowels, attended with giddiness, muscular weakness and prostration, disturbances of the circulation and respiration similar to those observed after the other previously described essential oils, death taking place after coma from asphyxia, though sometimes preceded by convulsions.

§ 508. The fatal *dose* is not definitely agreed upon, but serious symptoms may follow a dose of fifteen minims to a teaspoonful.

There have been quite a number of deaths caused by this oil taken inadvertently, or for the purpose of promoting the menstrual discharge. A number of cases are referred to in the foot-note.[1]

A young lady took a teaspoonful of the *oil* by mistake for the *essence* of tansy, for the purpose of promoting the catamenial discharge. She complained of dizziness, and became insensible in ten minutes, was seized with convulsions, her respiration was laborious and her pulse irregular. She died in one hour and a quarter after taking the oil. Another young lady of the family took of the

[1] Smith, Detroit Lancet, 1881–2, 147; Wetzell, Amer. Med. Bi-Weekly, Louisville, 1879, 58; Stephens, Nashville Med. and Surg. Journ., 1879, 320; Jewett, Boston Med. and Surg. Journ., 1880, 237; Stuart, Peoria Medical Monthly, 1881–2, 294; Gallaher, Pittsburg Med. Journ., 1880–1, 22; Tidd, Detroit Lancet, 1881–2, 100.

medicine at the same time, but vomited very soon, and suffered no inconvenience.[1] In a case which came under the notice of Dr. Dalton, of Lowell, recovery took place in consequence of spontaneous vomiting having occurred. Nevertheless the girl remained insensible and convulsed for some time after it. The most interesting and detailed case is that related by Dr. Dalton, Jr., of Boston. The quantity taken was a little less than an ounce and a half, and death took place in three hours and a half. The girl, when first seen, had fallen out of bed, in convulsions, and was entirely unconscious. The cheeks were highly flushed, the eyes open and brilliant; the pupils widely dilated and insensible; the skin was warm; pulse full, rapid, and strong; respiration hurried and stertorous, and obstructed by an abundance of frothy mucus, which filled the air passages and was blown from between the lips in respiration; the breath had a strong odor of tansy; convulsions occurred every five or ten minutes, in which the respiration was suspended, the arms raised and rigidly extended, and the fingers contracted. In the intervals between the convulsions there was no return of consciousness, and the jaws remained clinched so that it was impossible to administer any medicine by the mouth.

§ 509. The *autopsy* was made ten hours after death. The countenance was natural, the cadaveric rigidity was very strong, and there was only a slight discoloration of the dependent parts. The brain was not congested in any part, nor was there any effusion. Neither was there any appearance of congestion in the lungs. The interior of the heart exhaled a distinct odor of tansy, as did also the cut surface of the pectoral muscles. There was a strong odor of tansy in the peritoneal cavity. "The stomach contained about twelve ounces of a semi-fluid, yellowish-gray substance, consisting of partially digested food—potato, cranberries, onions, etc.—mixed with an abundance of small, yellowish-brown, glistening oil-globules, exhaling a powerful odor of tansy; mucous membrane generally pale, not vascular in any part, but throughout nearly the whole of the great pouch brownish and much thinned and softened, so that for a considerable space it was nearly or quite destroyed. There was an old, whitish, slightly puckered cicatrix of the mucous membrane on the posterior wall of the stomach, near the smaller curva-

[1] Am. Journ. Med. Sci., July, 1852, p. 279.

ture, but no other morbid appearance."[1] A four months' fœtus was found in the womb, not in the least disturbed. A two-ounce phial, containing still five drachms of the oil of tansy, was found in the pocket of the girl's dress; and a mug from which it had apparently been drunk, mixed with water, was also found, smelling very strongly of the medicine.

Half an ounce of oil of tansy, taken by a pregnant female to procure abortion, produced a partial loss of consciousness, and convulsions. It failed, however, of its purpose.[2] No other means have as yet been proposed, or are perhaps necessary, for the detection of this oil in cases of poisoning by it, than its powerful and peculiar aromatic smell.

XII. *Cocculus Indicus.*

§ 510. *Symptoms.*—This is the fruit of *Anamirta Cocculus.* The kernel, which is the only poisonous portion of the berry, has no smell, but an intensely bitter taste. It contains an active principle, called *picrotoxin*, which is an exceedingly active poison. It appears, from the experiments of Glover, Routh, and Falck, that the prominent symptoms produced by it in animals are salivation and tetanic convulsions, which usually terminate fatally, although the dose required to kill is much greater than that of other poisonous alkaloids, as much as forty grains of it being required to kill a dog. Cocculus indicus is chiefly used for the purpose of taking fish and of sophisticating malt liquor. It is also used for the destruction of lice. Several fatal accidents have occurred in this country from it. Dr. Thompson reports one case from its *external* application. A child, aged six years, whose head, after the hair had been cut close, was washed with an alcoholic tincture of cocculus indicus, was seized, in less than half an hour after its application, with tetanic convulsions. The pupils during the spasm were exceedingly contracted, and in the interval between the attacks were dilated to the fullest extent. By touching the eyelids, the spasm could be produced at pleasure. The case was treated with energy, but the child died in a few hours. On *post-mortem* exami-

[1] These appearances would hardly be called morbid, but probably were due to a *post-mortem* digestion of the stomach by its own secretion.—(Ed. third ed.)

[2] Bost. Med. and Surg. Journ., Dec. 1857, p. 383.

nation, no changes of any note were observed. A younger sister of the deceased, who had also been submitted to the same cleansing process, was likewise attacked in a similar manner. Under the use of counter-irritation by mustard, and injections of the tincture of assafœtida, she recovered, the convulsions gradually subsiding about three hours after the attack commenced. The next morning a scarlatinous eruption, which gradually faded during the day, appeared upon the body and arms.[1]

§ 511. The following account of several cases of poisoning by the internal use of this substance has been kindly communicated by Dr. Fish, formerly assistant resident physician at the Philadelphia Hospital, Blockley. A strong decoction of this berry (two ounces to a pint of water) is used in that institution for the destruction of vermin upon the paupers. The vessel containing it was unfortunately placed near some tonic infusions in use by several patients. Through the ignorance of the nurse, a wineglassful of this decoction was given to each of three persons, and two tablespoonfuls to three others, by mistake for their usual medicine. Two of those who took the largest quantity were seized with convulsions about twenty minutes after they had taken the poison, and died in about half an hour. This happened in the evening, and their muscles were still contracted the next morning. Both of these men were much reduced by intemperance and disease. The remaining four, who were seized within a few moments of each other, and within half an hour after they had taken the poison, presented the following symptoms: faintness, mental confusion, giddiness, dimness of vision, nausea, excessive thirst, severe pain in the abdomen, and in one case insensibility. The pulse was much weakened, and the respiration was slow and labored. Emetics were given to them, and, after the operation, mucilaginous drinks and stimulants. They all recovered, but suffered greatly from headache during the rest of the day.

Another case is mentioned in Traill's Outlines; and one is referred to by Taylor, in which the *post-mortem* examination distinctly revealed traces of gastro-enteritis, due to the irritant action of the poison. In this case, however, the patient lived until the nineteenth day.

[1] Philad. Med. Examiner, April, 1852, reported by William B. Thompson, Senior House Surgeon, Emigrants' Hospital, Ward's Island.

§ 512. The following case presents a curious question in the administration of poisons. The prisoner was indicted for administering poison, and it was proved that two *cocculus indicus* berries had been given to a child nine weeks old. The child, after having swallowed them, threw up one by vomiting, and the other passed through her body in the course of nature, and was found next day in her clothes.

Two medical witnesses, called on the part of the prosecution, proved that the *cocculus indicus* berry is classed with the narcotic poisons; that the poison consists in the presence of an alkaloid, which is extracted from the kernel; that all the noxious properties are in the kernel; that it has a very hard exterior or pod, to break which much force is required.

One of the witnesses added that the berry, if the pod is broken, is calculated to produce death in an adult human subject, though he did not know how many berries would be required for the purpose; that he thought the poison contained in the kernel of two berries, if the pods were burst, and if retained on the stomach, might produce death in a child nine weeks old, but that the berry could not be digested by the child, and that it would pass through its body, without the pod being burst, and so would be innocuous (as had, in fact, happened in the present case).

The counsel for the prisoner objected that the berries were not poisonous within the meaning of the statute, for that, though the kernel of the berries contained poison, yet the pod rendered the poison innocuous. The judge (Vaughan Williams) overruled the objection, and left the whole case to the jury. Verdict, guilty.

Judgment of death was recorded, but execution was stayed in order to submit the point raised by the prisoner's counsel to the consideration of the judges. The discussion before them in the Exchequer Chamber is given in detail by the reporter, and is quite interesting.

The counsel for the prisoner observed that the indictment was founded on the statute 1 Vict. c. 85, sect. 2, which makes it a capital felony to administer to, or cause to be taken by, any person, "any poison or other destructive thing," with intent to commit murder. The real question is, whether the berries in the state in which they were administered were "poison." The prisoner

thought he was giving a destructive thing, but did not do so. It was inquired of the counsel by the judges what he would say if arsenic was given in a globule of glass? Again, if arsenic was put in a paper envelope, and that wrapped in oiled paper and administered? He contended that in "such states it could not be a destructive thing." But it was replied, if a person gives poison in too small a dose, you would say that it was not within the statute, as it could not be destructive. If you are right in so saying, persons might give doses of arsenic and speculate on the size of the dose. Finally, Chief Justice Wild remarked, "The question here is whether the prisoner administered poison with intent to murder. The kernel of the berry was a poison, but he administered it in a condition in which it was not capable of doing injury. Is that administering poison? If a person administers poison with intent to murder, but accompanies it with something which prevents it acting, we all think it is the offence provided for by this enactment, and that the conviction must be affirmed." Justice Alderson said, "This is very different from the case of a person administering an innocent thing and thinking it poison; there he does not administer poison at all; here he does." The other judges concurred in affirming the conviction.[1]

§ 513. Orfila concludes, from experiments undertaken by him,[2] that, 1st, powdered cocculus poisons dogs; 2d, it acts upon the nervous system very much like camphor, but especially attacks the brain; 3d, *picrotoxin* is the active principle of the berry.

Bonnefin[3] concludes that the convulsive action of the poison was caused indirectly by stimulation of the cerebro-spinal axis; and these effects seem analogous to the action of veratrum viride.

"The experiments of Zschudi present the following comparative results of administration of strychnine and picrotoxin, both substances being given in two-grain doses:—

[1] Am. Journ. Med. Sci., April, 1851, from Regina *v.* Clanderoy, Carrington and Kirwan's Nisi Prius Reports, vol. ii. p. 709.

[2] Toxicologie, t. ii. p. 648.

[3] Thèse pour le Doctorat en médecine, 1851.

Strychnine.	*Picrotoxin.*
Causes tonic spasms.	Tonic and clonic spasms alternately.
Kills in three minutes.	Kills more slowly, *i. e.*, in an hour and a half.
Does not act upon the brain.	Acts in some degree as a narcotic upon the brain.
Never causes vomiting.	Excites frequent vomiting.
Does not act upon the secretions of saliva or bile.	Increases both in a very remarkable degree."[1]

§ 514. *Post-mortem appearances.*—These are not sufficiently striking to be of any very great assistance in determining poisoning by this drug; though in a few cases there have been observed signs of congestion of the brain and nervous centres.

§ 515. Dr. Taylor states that "porter, ale, and beer owe their intoxicating properties in some instances to a decoction of extract of the berries of cocculus indicus."[2] For a very interesting view of this matter, reference is made to Hassall's book on "adulterations detected in food and medicine."[3] It appears that cocculus indicus is often put into malt liquors to give a false strength to them. In his testimony before the Parliamentary committee he stated that he had ground many cwts. of cocculus indicus "to go into the poor man's drink."

In England cocculus indicus can be purchased at the druggist's under the name of "*multum.*" Mr. Simonds, in his testimony before the same committee, stated, "In the suburbs of London I may mention that it is a common practice with the publicans to adulterate beer on Saturday nights."

Child,[4] in a work which passed through eleven editions, gives, in a receipt for porter, "a quarter of an ounce of cocculus indicus."

Morris,[5] in a receipt for making twenty-five quarters of malt, gives six pounds of cocculus indicus.

Not only is beer itself adulterated, but frequently the very materials from which it is made are adulterated, as, for instance, the hops and malt.

§ 516. *Chemical examination.*—The detection of poisoning by cocculus indicus depends upon the isolation and recognition of its

[1] Stillé, Mat. Med., and Therapeutics, from Canstatt's Jahresbericht, 1848, p. 137.

[2] Med. Jurisprudence, p. 324.

[3] Op. cit.

[4] On brewing porter.

[5] Brewing malt liquors.

active principle picrotoxin, which is not an alkaloid, but a non-nitrogenous neutral substance, which crystallizes in the form of small quadrilateral prisms or in tufts of fine needles which have a silky lustre; these crystals are odorless, have an exceedingly bitter taste and a neutral reaction. It is soluble in 150 parts of cold and 25 of boiling water, very soluble in water containing a little acid, and soluble in alcohol, ether, amylalcohol and chloroform; ammonia water also dissolves it. Concentrated sulphuric acid dissolves it with a saffron-yellow color, which is changed by a trace of chromate of potassium to a violet. It is not precipitated from its solutions by salts of barium, iron, copper, lead, silver, gold, or platinum. A very small amount is sufficient to kill a fish; according to Blasner, one centigramme (about $\frac{1}{6}$ of a grain) will kill a fish of 200 or 300 grammes (about one-half a pound).

§ 517. In order to *isolate* picrotoxin from *organic mixtures* the ordinary method for alkaloids may be used as recommended under opium (see § 348), the picrotoxin being removed from the acid solution by chloroform. Amylalcohol and ether will also remove picrotoxin from acid solutions.

§ 518. The best method for the separation and detection of picrotoxin in beer or ale is that recommended by W. Schmidt, slightly modified by Dragendorff.[1] This method depends upon the principle that the picrotoxin is not precipitated by basic acetate of lead, and also that it can be removed from its solution in acidulated water by shaking with amylalcohol or ether. The beer or ale is first evaporated to the consistence of a syrup, then a sufficient amount of warm water added to render it fluid, and mixed with animal charcoal (five or six grammes for a bottle of beer). After several hours it is filtered, the charcoal washed, and the filtrate treated with basic acetate of lead in excess, filtered, the excess of lead in the filtrate separated by passing a current of sulphuretted hydrogen gas through it; the sulphide of lead thus formed will carry down a number of impurities; filter from the sulphide of lead and shake the filtrate, which should not be more than one-third of the volume of the original fluid, after allowing the sulphuretted hydrogen to escape, with five or ten per cent. of amylalcohol; after this has separated, it should be decanted and the shaking repeated with

[1] Pharm. Zeitsch. f. Russl., i. pp. 304 and 414.

a new portion of amylalcohol; the mixed amylalcohol fluids should be evaporated by gentle heat and the residue extracted with dilute alcohol (50 per cent.), filtered, and the filtrate evaporated; this residue is dissolved in boiling water to which a few drops of sulphuric acid have been added, the solution decolorized with animal charcoal, filtered and the cooled filtrate shaken with ether, which will remove the picrotoxin and leave it, after evaporation, as a residue. The picrotoxin can be purified by crystallizing it alternately from its solutions in water and alcohol. The sulphide of lead and the animal charcoal left upon the filter paper in the above process may retain a little of the picrotoxin and should be washed, dried, and extracted with ether. By this method Schmidt has obtained on one occasion four decigrammes (more than six grains) of picrotoxin from a bottle of beer.

XIII. *Digitalis Purpurea.* (Foxglove.)

§ 519. The leaves of this plant are the part usually employed, although the seeds contain also a large proportion of its active principle, which is called *digitaline*. The symptoms produced by digitalis in a poisonous dose have some peculiarities by which they may be distinguished from those caused by other poisons enumerated in this class. It is characterized by its remarkable property of causing slowness of the pulse. This symptom, which is quite familiar to physicians, was experimentally produced in nineteen patients in Andral's clinic, the object being to test the efficacy of the pills of digitaline recommended by Homolle and Quevenne. In all of them the pulse was reduced gradually about twenty-five beats, after the use of the pills for a period of time, which varied with each one.[1] The effects of this poison are cumulative; it may remain some time without any obvious effect upon the system, and then display its properties suddenly in a violent manner, and also after being discontinued its action upon the system does not immediately cease.

As mentioned in the opening chapter (§ 3), digitalis is sometimes tolerated with impunity in certain conditions, especially that of alcoholic intoxication. Instances where large doses of tincture of digitalis were unattended with serious results were reported by the medical witnesses at a coroner's inquest at Ramsgate, England.

[1] Union Méd., 1852, Nos. 52 and 53. [2] Med. Times and Gazette, Aug. 23, 1873, p. 206.

§ 520. The following is an example of poisoning with the tincture of digitalis, where a teaspoonful was taken in a glass of water. The symptoms did not manifest themselves until five hours after the dose had been taken; then they commenced with a feeling of nausea, which increased until violent and frequent vomiting took place. There were great præcordial distress, intense frontal headache, dimness of vision with dilated pupil, ringing in the ears, cramps in the muscles, very powerful but at the same time irregular and intermittent pulsations of the heart with diminished frequency (44 in the minute), the pulse strong and tense, the respiration sighing, the thirst uncontrollable, retention of urine, sleeplessness, and great debility. The next day, in addition to these symptoms, there was violent delirium, and from that time they continued very gradually to decrease for ten or eleven days. The pulse was very long in regaining its frequency.[1] A case is recorded by M. Caussé of a young woman who was pregnant, and who secretly took a large quantity of the expressed juice of digitalis, either to diminish the œdema of her limbs, or to produce abortion. The latter effect was produced and death followed in twelve days, probably as much from the want of medical care as from either the abortion or poison.[2] The tincture is often quite inert, or very feeble in medicinal properties; most of the fatal cases of poisoning by digitalis which have occurred have been either from the leaves in substance or infusion, or from digitaline. A decoction of the leaves, prescribed by a quack in London, to the amount of six ounces, proved fatal in twenty-two hours. Dr. Leroux relates a case of poisoning by digitaline which nearly proved fatal.[3] The dose taken amounted to 0.03 gramme, which is equal to about half a grain. In another instance, in which 40 granules, equal to about two-thirds of a grain, were supposed to have been taken, the patient recovered under the use of emetics and stimulants.[4] The symptoms, however, were not as urgent as in the preceding case. In still another case which recovered, the dose of digitaline taken was also 40 granules, but the symptoms were somewhat different from those enumerated above. Within an hour the person, who was a female, states that she was seized with dizziness, debility,

[1] Union Médicale, No. 112. 1851.
[2] Bull. de Thérap., lvi. 101.
[3] Union Médicale, No. 99. 1852.
[4] Dr. Ghereau, Union Méd., Jan. 10, 1854. Quoted in Ed. Monthly Journ. for August, 1854.

vomiting, and cold sweats, and was unable to feel her pulse beat, or to pass her urine ; she lay incapable of moving or speaking, her eyes felt strained, and actually projected considerably ; she had hallucinations, also continual nausea, extreme tenderness of the epigastrium, and great thirst ; and the pulse fell to 46 in the minute.[1]

§ 521. *Digitaline*[2] occurs in short and delicate needle-shaped crystals, and possesses an intense and persistent bitter taste. It is but slightly soluble in water, soluble in twelve parts of cold and six of boiling alcohol of 90°, less soluble in absolute alcohol, and nearly insoluble in ether. It is soluble to almost any extent in chloroform, which is its proper solvent, and which serves for a reagent in testing its purity. It is rapidly soluble in a solution of chloral hydrate, the solution becoming greenish-blue in color. The concentrated mineral acids dissolve it, hydrochloric producing an emerald-green color, sulphuric acid a green, which, if subjected to the action of bromine fumes, changes to a dark red, nitric acid a yellow, and *aqua regia* a yellow which changes to an obscure green ; a mixture of equal parts of nitric and sulphuric acids produces a rose color which becomes changed to a deep violet. When heated on platinum, it melts, swells up, becomes brown, and disappears without leaving any traces. About one-ninth of a grain was administered to an adult without any toxic effect being produced. The method of obtaining digitaline from the powdered leaves of the plant is also described in the same journal.

Nativelle received the grand prize of Orfila for his researches above detailed, but his results have been opposed by other authorities, such as Gubler,[3] Roucher,[4] and Schmiedeberg,[5] who assert that digitaline is a complex substance, and that it does not represent the active principle of digitalis. There is no good evidence of any fatal dose of Nativelle's digitaline. Flückiger and Hanbury[6] state, however, that one sixty-fourth of a grain taken by an adult person once or twice a day may produce alarming symptoms.

§ 522. Poisoning by digitalis is marked by signs of irritation of

[1] Annuaire de Thérap., 1858, p. 102.

[2] Journal de Chimie Médicale, May, 1873. Article by Nativelle ; an abstract of his paper is presented in the Boston Med. and Surg. Journ., July 10, 1873, p. 35, by the writer.

[3] Bull. d'l'Academie Roy. de Méd., vol. xxxvii. p. 404.

[4] Gaz. Méd., 1874.

[5] Archiv für experiment. Path. und Pharm., Bd. iii. p. 19.

[6] Op. cit., p. 424.

the stomach, such as severe vomiting, with sensations of heat in the head, disturbance of sight, and sometimes vertigo. If the patient be quiet and lying prone, the pulse will be full and strong, but after muscular exertion it becomes rapid and weak. Another sign of its irritant action may be abundant salivation, and on continuation of these symptoms of irritant action on the stomach, signs of muscular weakness and syncope occur with a feeble pulse at the wrist, and a tumultuous beating of the heart. The eyes protrude and are bloodshot, and the pupils are dilated. According to Tardieu,[1] the color of a fire appears blue to the person poisoned by digitalis; after the vomiting has ceased, the pressure over the pit of the stomach causes pain. The tongue is coated with a white envelope and is red at the tip, at other times it is dry and retracted. Breathing is sighing, deep and irregular. Sometimes there is diarrhœa of the same character as the vomitus, such as glairy liquids of a greenish color. Vomiting may continue as often as fifty times in the first few hours. Hiccough and involuntary evacuations accompanied by spasmodic movements precede the fatal result, which may not occur until the eighth or tenth day, though most usually it occurs within the first two or three days. Tardieu cites one case where the victim lived twelve days. Barth[2] relates one case of an invalid who drank about one and three-quarters pints of the tincture of digitalis, and expired in three-quarters of an hour exhausted with repeated vomiting. Twenty grains of the extract of digitalis has caused death in nine days.[3] The above symptoms refer only to acute poisoning.

§ 523. *Chronic* or *slow* poisoning follows the continued administration of small medicinal doses, in which case, though the medicine has been tolerated for several days or even weeks, violent symptoms of poisoning suddenly appear with syncope, or acute frontal headache with obscuration of vision. In one case Tardieu cites that a paralysis of one-half the body followed this syncope, which latter was repeated, accompanied by vomiting and diarrhœa, convulsions, delirium, marked slowing and feebleness of the pulse, and death. In these cases of slow or subacute poisoning death may be rapid or may not occur for several days.

[1] Op. cit., p. 753.

[2] Bull, de la Société Anatomique, p. 685. 1849.

[3] Tardieu, La Clinique, Paris, 1867,

Tardieu states that in the thesis of Ducroix,[1] it is cited that two-thirds (nineteen) of twenty-four very severe cases of poisoning by this drug recovered, so that it should be observed that death does not always follow the very severe symptoms above related.

§ 524. The *post-mortem appearances* do not bear testimony to the signs of irritation which are so severe in the history of the symptoms. It is not uncommon to find no other than perfectly healthy organs, though sometimes thickening of the walls of the stomach and a few congested spots of violet-colored redness may be observed; sometimes also there may be an increase of the serous fluid in the pericardium, and a state of congestion and serous infiltration between the meningeal surfaces. The pathological effects are, therefore, not very marked, but in one case where these have been reported, there were merely an injection of the "external membranes of the brain, and some redness of the mucous membrane of the stomach." It need hardly be said that this evidence amounts to nothing. In M. Caussé's case, the mucous membrane of the stomach near the lesser curvature of the pyloric orifice was covered with purplish patches. This was the only lesion referable to the action of the poison. Two cats, accidentally shut up in a room where this plant had been spread out to dry, were found the next morning dead. They had eaten of the leaves. Their bodies were very much relaxed, and, it is stated, putrefied very soon.

§ 525. *Chemical examination.*—In cases of digitalis poisoning careful search should always be made in the vomitus and feces, or in the contents of the stomach and intestines, for portions of the plant; the vomitus and feces will usually have a bright green color due to the coloring matter of the plant; this green color can be distinguished from that caused by copper compounds by its not being changed to an azure-blue color by ammonia-water, and from bile by its not passing through the changes of color characteristic of bile pigment, when treated with concentrated nitric acid. The bright green color is imparted to the contents of the stomach or vomitus by all of the preparations of digitalis, but portions of the plant can only be detected when the powdered leaves, or the infusion or decoction of the leaves or some other portion of the plant

[1] De l'émpoisonnement par la digitale, Paris, 1865.

[2] Archiv für Pharmacie, Oct. 1858, p. 16.

have been taken. At the present time, the leaves are the only portions of the plant which are used medicinally. The upper surface of the adult leaf is green, but pale and more or less silvery in the young leaf, and contains very short, transparent hairs, which have a very brilliant and crystalline appearance; the under surface of the leaf is pale and very pubescent, containing a much larger number of the brilliant hairs than the upper surface; the leaves also have another kind of vegetable hair, which is jointed, not crystalline in appearance, and usually made up of two or three segments; both of these varieties of hair may be easily detected by microscopic examination in the powdered leaf, or in the contents of the stomach, vomitus, etc., when the powder, decoction, or infusion have been taken, except when the two last preparations have been very carefully strained, which is not usually the case.

§ 526. Digitaline, as usually found in the market, is a mixture of several substances called by Schmiedeberg and Dragendorff, digataline, digitaleine, digitonine, and digitoxine; in chemico-legal investigations the first two substances are sought for, and may be recognized by the following properties and tests. *Digitaline* may be obtained in crystalline form as mentioned above, but is usually seen in the form of spherical masses; it is soluble with difficulty in water even at a boiling temperature; it is readily soluble in alcohol and in a mixture of alcohol and chloroform; it is slightly soluble in absolute ether, pure chloroform and benzol; benzol will remove it from acid solutions; concentrated hydrochloric acid colors it yellowish-green; concentrated sulphuric acid a greenish-brown, which changes to a beautiful violet-red color if exposed to the action of bromine vapor, and if water is then added to an emerald-green. *Digitaleine* is soluble in all proportions in water, and is readily soluble in alcohol and ether; it is insoluble in benzol, but slightly soluble in chloroform which removes it from acid solutions; concentrated hydrochloric acid gives it a light yellow color; concentrated sulphuric acid colors it reddish, which changes to a violet when treated with bromine vapor, and then to a green if diluted with water. The most important test, however, for both digitaline and digitaleine is the physiological test, which is best performed with frogs, since the heart can be exposed, and the action of these principles upon the heart be seen; very small amounts suffice to diminish

the number of pulsations and finally to cause cessation of the heart's action, this cessation taking place during the systole.

§ 527. Both digitaline and digitaleine can be isolated from organic mixtures by the process recommended for the opium alkaloids (see § 348), the digitaline being separated by benzol from the acid fluid, and the digitaleine by chloroform, and are left as a residue after the evaporation of these fluids.

XIV. *Veratrum Album and Veratrum Viride.*

§ 528. Both these drugs, known as white and green hellebore, are exceedingly active poisons, and belong, from their peculiar characteristics, to the class of poisons called by M. Tardieu hyposthenic ant. M. Oulmont,[1] in his observations on man and animals, found that, although the general sedative action of veratrum *viride* was the same as that of veratrum *album*, it differs essentially from the latter in the following points:—

It never has so intense an action on the digestive canal, nor produces inflammation of its mucous membrane, and the vomiting is not so severe or lasting.

The poisonous dose of green hellebore is much larger than that of the white hellebore, and the action of the former is not so quickly manifested.

§ 529. *White hellebore.*—The active principle of this drug is veratrine, and the symptoms of poisoning most commonly noted are, a sense of burning heat in the stomach with a feeling of constriction and heat in the mouth and throat, great anxiety, nausea, violent purging, vomiting, tenesmus, intestinal pain, great prostration, diminution and feebleness of the pulse and respiratory movements, muscular tremors, dilatation of pupils, depression of temperature, coldness of the extremities, convulsions, and insensibility.

Some instances are recorded in which purging was absent, and again, others, in which there was no vomiting. As hellebore is frequently used in the so-called worm powders, not a few cases of poisoning may occur from an overdose ignorantly administered or taken. A number of fatal cases of poisoning with this agent have been reported.[2]

[1] Bull. Gén. de Thérapeutique, etc., t. lxxiv. p. 145, May 30, 1869.

[2] Beiträge zur Gerichtl. Arzn., iv. 47; Taylor on Poisons, 3d Am. ed.,

§ 530. *Green hellebore.*—The symptoms of poisoning caused by this drug are not unlike those produced by white hellebore. The distinctions noticed by Oulmont have already been related. A few fatal cases of poisoning by this drug, called also *Indian poke*, have been reported.[1] M. Oulmont mentions instances of poisoning by this agent where a persistent attack of hiccough was induced.[2] An active principle called veratroidine has been isolated from this drug. This alkaloid is soluble in ether, but a resinous principle called viridia, which is insoluble in ether, has also some of the peculiar properties of veratrum viride. The name veratroidine was given because this alkaloid resembles that from the white hellebore known as veratrine. It has also been asserted that viridia was not unlike the resin of white hellebore, called by Simon, *gervina*.[3]

The most striking difference between these two *hellebores* consists, from experiments by Oulmont on animals, in the great irritation of the alimentary canal by veratrum album, which also causes not only vomiting and purging, but a positive inflammation which may even produce death; the veratrum viride, on the contrary, may cause vomiting, diarrhœa, and even fatal prostration, without showing *post-mortem* any signs of inflammation in the alimentary canal.

The strength of the tincture of both these poisons is variable, as well as that of the different alkaloids and resinoids; hence, it is not easy to state a maximum therapeutic or a minimum toxical dose.

§ 531. *Chemical examination.*—The detection of poisoning by white or green hellebore depends upon the detection of some portion of the plant, which has characteristic properties, by the microscopic examination of the contents of the stomach or intestines, or upon the isolation and recognition of the active principle veratrine.

Veratrine as usually seen is probably a mixture of several alkaloids, but it may be obtained pure in crystalline form. It is white, crystallizes with difficulty from alcohol in the form of colorless prisms, which melt at about 115° C. and at a higher temperature

p. 497; Gaz. Hebd., vol. viii. No. 31; British Pharmaceutical Journal and Trans., Feb. 1868.

[1] Am. Journ. of Med. Sciences, July, 1865; Am. Journ. of Pharmacy, Sep. 1865.

[2] Bull. Gén. de Thér., lxxv. p. 526.

[3] *Vide* an article by Dr. E. Peugnet in the Medical Record, N. Y., May 1, 1872; also articles by Wm. Bullock, Am. Journ. of Pharm., Sept. 1865, March, 1866, and in Am. Journ. of Med. Sciences, Jan, 1870, by H. C. Wood, M.D.

volatilize. It is readily soluble in alcohol (about three parts) and chloroform (about two parts); it is also soluble in benzol, amyl-alcohol and ether (about 10 parts), but soluble with difficulty in naphtha, and water; it is readily dissolved by dilute acids, salts of veratrine being formed which are soluble in water, and from such solutions the veratrine is easily precipitated by alkalies and alkaline carbonates in the amorphous form at first, the precipitate gradually becoming crystalline. The slightest amount of veratrine brought in contact with the nasal mucous membranes creates violent sneezing. Concentrated sulphuric acid dissolves veratrine, forming a solution which is yellow at first, but changes gradually to an orange, then to a blood-red, and after about one-half an hour to a beautiful carmine-red color which is quite permanent; this change is hastened by heating or by exposure to the action of bromine vapor. If veratrine is first mixed with a little sugar and then treated with concentrated sulphuric acid, the mixture is at first yellow but quickly changes to a grass-green color, then to a beautiful blue and finally to a dirty violet color; this change begins at the outside of the mixture and progresses inwards, and can be hastened by the addition of a little water (Weppen's test). "Concentrated hydrochloric acid dissolves the pure alkaloid without change of color; but, if the solution be heated to the boiling temperature, it quickly acquires a red color, which ultimately becomes very intense and resembles that of a solution of permanganate of potash. Under this reaction, if only a drop of the acid be employed, almost the least visible quantity of the alkaloid will manifest itself."[1] (Trapp's reaction.)

§ 532. The *physiological test* for veratrine is an exceedingly important one in legal cases. The experiments of Weigelin[2] have shown that veratrine dissolved in very dilute acetic acid acts very energetically upon frogs in a dose of four-tenths of a milligramme; violent retching is produced very quickly, and the action of the heart becomes slower, the pulsations being diminished from 60 per minute to 32 after the lapse of ten minutes, and to 8 after the lapse of ninety minutes, when the heart's action becomes very irregular and sometimes ceases entirely. Tetanic convulsions are caused by larger doses, such as two milligrammes, which dose produced in

[1] Wormley, op. cit.

[2] Dragendorff, op. cit., p. 210.

medium-sized frogs immediate retching, in fifteen minutes tetanic spasms, and death in one hour.

§ 533. Veratrine can be *isolated* from *organic mixtures* by the process of Dragendorff, recommended for the opium alkaloids (see § 348). It is removed from the alkaline solution by shaking with naphtha or benzol, preferably the latter solvent; the residue left after the evaporation of the benzol can be used for performing the above tests.

§ 534. M. Prévost[1] contrasts the effects of this alkaloid upon the muscular system of frogs, in a way which may serve to test the presence of this poison as distinct from that of strychnine.[2]

The differential effects are as follows:—

In Strychnine Poisoning.	*In Veratrine Poisoning.*
1. Convulsions occur at the commencement.	1. Spasmodic contractions occur at the commencement.
2. The initial convulsion is succeeded by a series of convulsions.	2. The initial convulsion is prolonged, and usually terminates in a series of faint fibrillary twitches.
3. Extremely slight peripheral excitation causes a convulsion.	3. It is difficult to originate spasmodic contractions by excitation of the periphery.
4. The most feeble peripheral excitation always originates general convulsions.	4. Excitation often causes contractions that are altogether limited to the excited portions; occasionally, however, the contractions are general.
5. Convulsions disappear on destruction of the spinal cord.	5. Spasmodic contractions may be caused even after destruction of the spinal cord by irritation of either the nerves or the muscles.
6. Convulsions cease in limbs separated from the trunk, and consequently from the spinal cord. In parts so separated, excitation of nerve-trunks or of muscles causes merely normal contractions.	6. In limbs separated from the trunk, and consequently from the spinal cord, spasmodic contractions may be caused by irritation of the nerve-trunks or of the muscles.
7. Convulsions occur in limbs separated from the circulation by ligature of the bloodvessels, provided the nerve-trunks are intact.	7. No spasmodic contractions occur in limbs separated from the circulation by ligature of the bloodvessels.

[1] Récherche Expérimentale relative à l'Action de la Veratrine.

[2] It may be well to state here that many of the alkaloids can be recognized from their effects upon the smaller animals.

XV. *Quinine.*

§ 535. The occasional occurrence of alarming and even fatal effects from the use of sulphate of quinine renders it necessary that we should briefly notice them. When given in larger doses than usual, continued for too great a length of time, or in persons peculiarly susceptible to its influence, it produces considerable cerebral disturbance, and may occasion severe headache, vertigo, deafness, diminution or loss of sight and of speech, delirium, coma, and great prostration.[1] Dr. M'Lean relates four instances in which complete blindness was produced, from which, however, the patients partially recovered in the course of a year. In one of these cases, three drachms were given in the course of 36 hours; in another, an ounce; in the others the quantity was somewhat less, being in one three drachms and a half in three days, and in the other the quantity is merely said to have been "large."[2] In the same journal will be found the paper of Dr. Baldwin detailing his experiments upon animals, and giving the particulars of a case which came under his notice, where he considered that death resulted from the use of quinine, although not administered in large doses. Death was preceded by extreme restlessness, dilatation of the pupils, blindness, and convulsions. The disease was intermittent fever. Without presuming to deny the accuracy of Dr. B.'s opinion of the cause of death in this case, we may state that we have seen precisely the same alarming cerebral symptoms in a child seven years of age, with intermittent fever, which could not have been caused by quinine, as none had been taken in any form. A man with acute rheumatism, under the care of M. Récamier at the Hôtel Dieu, after taking about 100 grains of the sulphate of quinine in hourly doses of from four to five grains, was suddenly attacked with delirium and died in a few hours. A similar case occurred under M. Husson's care, but the patient recovered. The whole quantity given was 93 grains, the symptoms coming on after the last dose.[3] Four cases are collected by M. Mélier in which it is said that this drug was fatal; and Guersant has given an account of a physician who died poisoned by sulphate of quinine, after taking nearly five

[1] Wood and Bache.

[2] Am. Journ. Med. Sci., April, 1847, 515.

[3] Gaz. des Hôp., Dec. 1842.

ounces of it in the course of eight or nine days. In this, as in other similar cases, sight and hearing were lost, the limbs were cold, the breathing slow and labored, and the pulse feeble, irregular, and infrequent. Death took place by coma preceded by delirium.

536. *Post-mortem appearances.*—In experiments on animals poisoned by large doses of quinine, the only peculiar *post-mortem* appearances were engorgement of the venous sinuses within the cranium.

XVI. *Daphne mezereum.*

§ 537. The berries of this plant, which resemble those of the red currant, are actively poisonous. In two cases reported by Dr. Schwebes, the symptoms were nausea and vomiting, followed by complete narcotism; there were convulsive movements of the eyes and upper extremities occurring at short intervals, the pupils were contracted and scarcely sensible to the stimulus of light. The children were restored by cold affusion to the head and other appropriate treatment. Dr. Christison mentions the case of a child, aged eight years, which proved fatal, and three others which recovered. The symptoms were similar to those described.

CHAPTER XI.

POISONOUS GASES.

I. *Carbonic Acid Gas.*

§ 538. *Effects.*—This gas when pure is irrespirable; the irritation produced by it upon the glottis being so great that it closes, and respiration becomes impossible. When, however, it is mixed with the air, it may be respired, and then produces symptoms somewhat similar to those of asphyxia, but which, nevertheless, are due to its specific narcotic action upon the system through the mucous membrane of the lungs. Under the present head we include cases of poisoning by charcoal fumes, which, however, contain, besides carbonic acid, carbonic oxide and traces of carburetted hydrogen. The first symptoms produced by this gas are heaviness of the head, a sensation of weight or pressure upon the temples, ringing in the ears, and a disposition to sleep. Then nausea and sometimes vomiting follow; the respiration becomes slower, difficult, and sometimes stertorous; the pulsations of the heart, which are at first tumultuous, then become irregular, and finally slower; the muscles are paralyzed, and the individual falls into a comatose condition, which may last several hours before life is extinct. Occasionally, secondary phenomena, such as nasal or pulmonary hemorrhage and pneumonia, are observed. Still more serious consequences may result, as in the case of a man whose attempt to destroy himself with charcoal fumes was interrupted after he became insensible. An inflammation of the sciatic nerve occurred, followed by general paralysis, delirium, and death.[1] The general appearance of the body varies in different

[1] Arch. Gén., 5ème sér., ix. 476.

cases, according to the rapidity of death and the length of time elapsing before it is seen. Sometimes the face is red and swollen, the eyes bright and glistening, the limbs flexible, and there are red spots in various parts of the body; in others, on the contrary, there may be remarkable pallor, and a tetanic stiffness of all the muscles.

§ 539. The body has sometimes an appearance of complete repose in natural sleep; but sometimes, also, the features are swollen, discolored, and distorted. The internal appearances are a vivid red, or sometimes a violet color of the blood, or, again, this fluid may be black and thick; the soft solids are everywhere of a brighter color than natural; the lungs are voluminous, and of a brownish-black color on their exterior, and red internally; the body retains its heat and flexibility for a considerable time, and putrefaction occurs more slowly than after other modes of death.[1] The presence of carbonic acid in the air of a room where persons have died or been more or less affected by it, may be detected by the white precipitate formed by it with lime-water or a solution of subacetate of lead. The *proportion* in which it exists may be detected, as recommended by Dr. Taylor, by introducing into a measured quantity, in a graduated tube over mercury, a strong solution of caustic potash. The degree of absorption will indicate the proportion of carbonic acid present.[2]

§ 540. Death from the inhalation of carbonic acid and coal gas is almost always, where it is not suicidal, produced accidentally. There can be no doubt, however, but that a person may be thus destroyed by criminal design when asleep, this gas being of so insidious and oppressive a nature, that the individual may pass readily, without waking, from natural sleep into a state of fatal coma. Attendant circumstances may awaken a suspicion of wilful poisoning, but there is evidently nothing in the medical aspect of the case by which death can be attributed to the action of another rather than to that of the individual himself. The study of the effects of carbonic acid and coal gas upon the system, under the various circumstances where it is inhaled, is important only as enabling us to refute unjust suspicions of other violent causes of death, and especially of poisoning by other agents. Such suspicions are

[1] Briand, Méd. Lég., p. 414.

[2] Med. Journ., p. 529.

very apt to be entertained. Dr. Christison relates a case, in which a man and woman who had survived the effects of the gas generated from a pan of burning coals in their apartment, while at the same time four other persons in the room perished, were imprisoned on suspicion of having conspired to murder their companions. Similar cases have frequently been the subject of examination before the coroner's inquest.

§ 541. Carbonic acid is disengaged not only during the combustion of fuel, but may be present in deleterious quantity in the atmosphere from other sources. Thus it, combined with other impurities from exhalations, has been the cause of death by the non-renewal of the air where a large number of persons are confined in a close apartment, and are obliged to respire the same air repeatedly; it is disengaged in breweries during the process of fermentation, and in green-houses, from the plants, during the night; persons have been frequently destroyed by it who, for the sake of warmth, have laid themselves down near the vents of lime kilns; and it is well known that it accumulates in the shafts of coal mines, and has there been the cause of death to large numbers of persons. The only one of of these cases that can well become the subject of medico-legal inquiry is that in which death results from the gases evolved by the combustion of fuel. We use the word *gases* since, as already remarked, it is by no means certain that the fatal effects are always due to the disengagement of carbonic acid gas. Carbonic oxide, which is also evolved, is still more rapidly poisonous than carbonic acid gas.[1]

§ 542. Investigations[2] (1872) have conclusively shown that death from the inhalation of coal gases is due to carbonic oxide, which latter forms, with a certain portion of the blood, a combined salt, that will prevent the blood from absorbing oxygen; whereas the inhalation of pure carbonic acid gas causes death by preventing the

[1] M. Chevalier, in the October number of the Ann. d'Hyg. for 1854, has related a case of poisoning by the vapors of carbon. He shows that three or four per cent. of carbonic oxide will suffice to destroy a strong dog, that would not have been killed by less than thirty or forty per cent. of carbonic acid in the air. Warm-blooded animals may be destroyed by one per cent. of carbonic oxide.

[2] Cl. Bernard in Liquides de l'organisme, Paris, 1859; Physiologie generale, Paris, 1872, p. 50; Physiologie Opératoire, Paris, 1879, p. 444; Anæsthésie et Asphyxie, Paris, 1875, p. 407.

elimination of the same gas, and also by depriving the system of oxygen, thus causing death by asphyxia.

§ 543. M. Demarquay[1] states from his own observation and experiments, if carbonic acid is injected into the veins, taking care not to inject with such force as, by entering the cavities of the heart, to cause death by mechanical distension of that organ, it is absorbed in large quantity and quickly eliminated. Introduced into the system by the lungs, carbonic acid produces none of the toxical properties so commonly attributed to it. In fact the mammalia can breathe a long time, without any serious inconvenience, atmospheric air or oxygen one-fourth or one-fifth of whose volume consists of carbonic acid; in man a slight disturbance may be caused after the lapse of a time varying with the susceptibility of individuals, but lasting long enough to cause the development of certain therapeutical effects which have indicated the use of the agent. Finally, the lesions after death caused by the inhalation of this gas, whether in man or animals, do not in the least resemble those caused by the toxical agent, carbonic oxide, with which it has often been confounded.

Most of the accidents caused by coal gas, confined air, vapors from vats, laying aside all account of carbonic acid, should be in a large measure attributed either to carbonic oxide, sulphuretted hydrogen, alcoholic fumes, or even to other gases, as yet little known, which have their origin from these sources.

§ 544. Carbonic acid is simply irrespirable. It is, however, unlike nitrogen or hydrogen in this respect, and is more poisonous than these gases because it prevents the inspiration of oxygen and the necessary respiratory functions. Respiration essentially consists in the interchange of gases between the blood and air, and, from the fact that this interchange cannot take place, more in consequence of an absence of the necessary amount of oxygen in an air saturated with carbonic acid than from the poisonous character of this gas, according to the physical law of gases, it may be seen that the inspiration of pure carbonic acid is a material obstacle to the respiratory process, and consequently produces asphyxia. On the other hand, hydrogen and nitrogen cannot supply the deficiency of oxygen and cause the normal changes in the blood necessary to

[1] Essai de Pneumatologie Médicale; Recherches, etc., sur les Gas, Paris, 1866, p. 458.

support life, but this is not the result from the same physical law of diffusion of gases, because being of a different nature from the eliminated gases; yet this interchange can go on only for a limited period, and thus disturbances may be caused that are inconsistent with life.

On the continent of Europe, and especially in Paris, self-destruction by the vapors of charcoal is one of the most common forms of suicide. In England and the United States this agent is seldom resorted to, while accidental death from the gases escaping from burning coal or the smothered combustion of wood is very frequent.

§ 545. *Qualities.*—Carbonic acid gas when not heated, is heavier than common air, and will therefore be found in greatest quantity near the floor after combustion has ceased; but during combustion, or while the air is still warm, it will be equally diffused through the apartment. Dr. Taylor found, by experiment, that, in burning a quantity of charcoal actively in an open brazier raised above the floor, in a large apartment, the proportion of carbonic acid was nearly equal in air taken a foot above and a foot below the level of the source of combustion, there being no current to affect the results. The inferences which he draws from this and from other considerations are—1st. That in a small and close apartment individuals are equally liable to be suffocated at all levels, from the very equal and rapid diffusion of carbonic acid gas during combustion. 2d. That in a large apartment, unless the gas be very rapidly diffused by a current of air, the air around the source of combustion may become impregnated with a poisonous proportion, while that at a distance might still be capable of supporting life, because carbonic acid requires time for its perfect and equable diffusion in a very large space.[1]

§ 546. The following case may serve to show the circuitous route by which carbonic acid may find its way into bedrooms:[2] A man and his wife were found dead in their bed-room; the first in an easy bent position on his right side, on the floor; the latter in a similar position, and her countenance wore a mild and placid expression. No marks of violence were found upon the bodies, and

[1] Med. Jur., p. 535.

[2] An account of two cases of poisoning with carbonic acid, in remarkable circumstances, communicated by Jos. Law, Esq., Surgeon, etc., Ed. Month. Journ., March, 1853.

with the exception of slight suggillations on the man's back, the skin was perfectly natural in color and appearance. A *post-mortem* examination and a chemical analysis were made without any indication of poisoning being detected. There was a singular and intolerable smell in the house, strongest in the chamber. It was found, upon further inquiry and examination, that a straw mattress had been burnt in the cesspool of an adjoining yard a few days previously, the embers of which were still in a state of ignition, and, when stirred, gave off dense volumes of smoke and a disgusting smell resembling that in the house. The walls of this cesspool and of the foundation of the house were of loose stones, and under the influence of a strong west wind the products of combustion had found their way through the foundation into the boarded walls of the house, and thence into the chamber. No sulphuretted hydrogen could be detected in the gas which still escaped into the room, but sufficient carbonic acid to fatally contaminate its atmosphere. Briand enumerates several instances in which carbonic acid, coming from fires lit in an apartment other than that occupied by the deceased, has nevertheless penetrated into it and been the cause of fatal accidents.[1] In one of these cases a man and his wife were found dead in bed, suffocated by gas produced by the charred woodwork in the neighborhood of a fire in a room at the opposite end of a long corridor on the same floor. The gas had worked its way under the floor until it found a vent in a crack of the flooring in their apartment. In other instances the gas was driven through stove-pipes, and from one chimney-flue to another on different floors.

§ 547. *Illuminating gas.*—The ordinary illuminating gas, which consists chiefly of light carburetted hydrogen, contains also vapors of volatile, liquid carburets of hydrogen, carbonic oxide, and other elements. Light carburetted hydrogen is in itself hardly poisonous, but the composite gas, which is now everywhere so freely used for burning, has frequently caused fatal accidents. Still, the atmosphere may be very offensively loaded with it, and yet be breathed for a short time with impunity. It does not appear to act merely as an asphyxiating agent, but rather like a narcotic.

[1] Méd. Lég., p. 418.

The first symptoms are nausea, headache, noises in the ears, and great prostration. All of these become aggravated; the breathing then becomes oppressed, the limbs are paralyzed, and death is preceded by coma and convulsions.

§ 548. The *post-mortem appearances* are, generally, intense injection of the vessels and redness of the serous membranes of cerebrum and spine, redness of the bronchial mucous membrane and of the lungs, and a dark color of the blood. In the fatal cases which occurred at Strasburg, and which are reported by M. Tourdes, the bronchial tubes were filled also with a white, thick, and viscid froth, streaked with blood.[1]

The following case is reported by Gartner, of Stuttgart.[2] The gas affected a lady, her servant maid, and also an English pointer dog. The lady was first seized; her illness began with an affection of the head, sickness, vomiting, and purging of thin rice-water-like stools, in which whitish flakes were observed. After twelve hours she recovered, but felt very drowsy. On the fifth day she experienced pain in the back part of the head, lassitude, vertigo, tinnitus aurium, and loss of appetite, accompanied by a loaded tongue, a small pulse of 90, and cessation of the menses. Blood of rather a dirty dark-red color, presenting no buffy coat, was abstracted from a vein. Next day the patient was worse; she was quite insensible, and lay with closed eyelids; the eyes were turned up, the pupils were much contracted, and unaffected by the light; the face was not swollen; there was trismus; the arms were flexed at the elbow-joints; the respiratory movements were very feeble; the pulse was hardly perceptible; the skin was warm, but insensible to the touch. She was, however, restored by venesection and other remedies.

In the servant girl similar symptoms occurred, but not with much severity, which may be attributed to the fact that the atmosphere of her chamber had not been so strongly impregnated with the gas. She had severe cramps of the extremities, great jactitation of the hands, flexion of the arms at the elbows, great restlessness, and inclination to yawn. Her blood presented no buffy coat. Latterly

[1] Ann. d'Hyg., t. iii. p. 457. *Vide*, also, Devergie, Méd. Lég., t. iii. pp. 72 and 75.

[2] Ed. Month. Journ., Oct. 1854.

she had a non-febrile bloody diarrhœa. She recovered in fourteen days from the date of her seizure.

The dog was found insensible and quite stiff, as if dead, but it soon recovered.

A man employed to clean a covered passage for water, into which a gas-pipe had leaked several days before, was sickened by the smell of the air, and fell with his head under the water. In about five minutes he was with difficulty dragged out, by means of a noose round his neck. He was pale and breathed feebly, but gradually his warmth and pulse became natural; the breathing, however, was oppressed and guttural, and the muscles of the trunk and limbs spasmodically contracted. Death took place in about seven hours. The body, which was examined thirty-five hours afterwards, was rigid, and everywhere seemed congested with blood. It exhaled a strong aliaceous odor, and the stomach and intestines were distended with a gas which took fire and burned when flame was applied to it.[1]

§ 549. The danger arising from the inhalation of illuminating gas varies with the kind of gas and with the thoroughness of its purification; the principal danger arises from the carbonic oxide which the gas contains; ordinary illuminating gas made from bituminous coal and well purified consists chiefly of hydrogen (40–50 per cent.) marsh gas (35–40 per cent.), carbonic oxide ($4\frac{1}{2}$–$7\frac{1}{2}$ per cent.), olefiant gas and other hydrocarbons (4–8 per cent.), and usually very small amounts of carbonic acid gas and air. The so-called water gas made by passing steam over anthracite coal heated to a white heat yields a gas which burns with a colorless flame and contains 40 or 50 per cent. of carbonic oxide; this is mixed with a highly luminous gas made from naphtha, petroleum, or cannel coal to give it illuminating properties, but even then it contains a very large percentage " of carbonic oxide, which is one of the most active of poisons, producing when inhaled speedy death, and according to Leblanc, ' one vol. of it diffused through 100 vols. of air totally unfits it to sustain life; and it appears that the lamentable accidents which too frequently occur from burning charcoal or coke in braziers and chafing-dishes in close rooms, result from the poisonous effects of the small quantity of carbonic oxide which is produced and escapes combustion, since the amount of carbonic acid thus diffused through

[1] Annuaire de Thérap., 1857, p. 288.

the air is not sufficient in many cases to account for the fatal result.'[1] When it was proposed to supply the *Invalides* in Paris with water gas, a commission was appointed, consisting of Messrs. Dumas, Chevreul, and Regnault, eminent chemists, to investigate it. They found that it contained from thirty to forty per cent. of carbonic oxide, and reported 'that it would be dangerous to the occupants of the institution to introduce, even by way of experiment, gas obtained from the decomposition of water by the *Kirkham* process.'[2] This gas was the odorless carbonic oxide and hydrogen mixture. H. Letheby says,[3] 'Seligue, in 1840, obtained permission to use the gas in the towns of Dijon, Strasburg, Antwerp, and two of the faubourgs of Paris, and Lyons. At Strasburg an accident occurred which put a stop to its use. The gas escaped from the pipes into a baker's shop, and was fatal to several persons; and not long after an aeronaut, named Delcourt, incautiously used the gas for inflating his balloon. He became insensible in the car, and those who approached to render him assistance fainted and fell likewise. The use of the gas has therefore been interdicted on the Continent.'"[4]

§ 550. *Chemical examination.*—The detection of this form of poisoning after death depends chiefly upon the recognition of the carbonic oxide in the blood by the spectroscope, since the coloring matter of the blood unites with it to form a compound which gives characteristic absorption bands when examined by the spectroscope. In cases of poisoning by carbonic oxide or any gaseous mixture which contains it, the blood has the bright-red, almost rose-color, which remains several days;[5] this color is due to the union of the blood pigment, hæmoglobin, with the carbonic oxide, which has replaced the oxygen normally combined with the hæmoglobin, thus depriving blood pigment of one of its physiological functions, viz., that of carrying oxygen through the body. If blood which contains this compound of hæmoglobin and carbonic oxide be, after suitable dilution with water, examined with the spectroscope two absorption bands similar to those of the natural blood pigment will

[1] Bloxam's Chemistry, p. 118.

[2] London Journal of Gas Lighting, June 10, 1856.

[3] London Journal of Gas Lighting, May 20, 1862.

[4] Extract from an article by the writer published in vol. iii. Public Health Papers of the American Public Health Association.

[5] Huenefeld, Blutproben vor Gericht und das Kohlenoxydblut, Leipzig, 1875.

be seen, but differing from them by the fact that, when treated with sulphide of ammonium, they do not disappear or rather coalesce; moreover, this compound is a very stable one, and is not destroyed by drying, so that it can be detected, according to Eulenberg and Jaederholm, in blood after it has been dried for several weeks by moistening with water and examining with the spectroscope.

II. *Sulphuretted Hydrogen Gas.*

§ 551. This is the principal deleterious gas, which is evolved from privy wells, and from foul drains and sewers. Its familiar and extremely offensive odor affords such unmistakable evidence of its presence, that unless a person is obliged to inhale it, or is exposed to it in a concentrated form, accidents will rarely occur from it. The consideration of its effects, and the means of obviating them, is evidently more the subject of medical police or hygiene, than of legal medicine. A few observations may not, however, be misplaced. When not existing in a very large proportion in the atmosphere, it may be breathed for a certain time with comparative impunity, giving rise merely to lassitude, loss of appetite, and sometimes a typhoid febrile condition. Again, when inhaled in a greater quantity, the symptoms are acute and oppressive pain in the head and pit of the stomach ; and for this reason, this gas has received from the French the name of "*plomb des fosses.*" If after experiencing these sensations the individual does not immediately withdraw from his position, he loses his consciousness, and falls, completely deprived of sensibility and the power of motion ; a reddish froth runs from the mouth, the body is cold, and the face livid ; the eyes are dull, and the pupils dilated and immovable ; the pulse very irregular, and almost imperceptible ; convulsions ensue, and the person dies comatose.

In a case reported by Dr. Radcliff, of Baltimore, a man who descended into a privy-sink nearly eighty feet deep, already almost emptied by machinery, was overcome by the gases and fell to the bottom, where he remained for two hours before he could be extricated. Meanwhile cold water was repeatedly thrown upon him. Fifteen minutes afterwards he was much asphyxiated and depressed, with hurried and difficult respiration, but he recovered rapidly.[1]

[1] Am. Journ. Med. Sci., Oct. 1858, p. 377.

§ 552. The *post-mortem appearances* usually described are the following: A proneness to rapid putrefaction; an offensive odor from all parts of the body; the blood dark and liquid; the right side of the heart congested, and the muscles of the body of a dark color, and insusceptible to the stimulus of galvanism.

This is not the only noxious gas evolved from privies and drains, but it is that which is the most destructive to life. There are also ammoniacal emanations, which are extremely irritating to the respiratory mucous membrane; and nitrogen gas, which sometimes accumulates in enormous quantity, but which, although irrespirable, is perhaps not in itself noxious.[1]

III. *Nitrous Oxide Gas.*

§ 553. This gas, more properly named protoxide of nitrogen, has been used very extensively by dentists and somewhat by surgeons for minor operations as an anæsthetic. It deserves but little mention here. Its action is due most probably to the property which it possesses of supplying the place of a portion of the oxygen in the blood with an innocuous respirable gas incapable of supporting life, but not directly interfering with the elimination of carbonic acid gas. If pure, and properly administered, this gas can do no harm unless the inhalation be prolonged beyond the period that respiratory movements are manifested; even then the removal of the inhaler may be immediately followed by inspiration of air, which soon takes its proper place in the lungs and blood, producing the normal changes necessary to the support of life. In this respect it resembles ether, which may also cause asphyxia, if administered by an inexperienced person. If in making the gas too great heat be used, deutoxide of nitrogen or poisonous nitrous fumes may be formed in the retort holding the nitrate of ammonium (the substance most commonly used in the manufacture of the gas). Yet this is quite soluble in water, through which the gas is generally, and should always be, conducted before passing into the gasometer. The present plan of condensing nitrous oxide and confining the condensed gas in an iron chamber avoids the risk which might occur from the decomposition or fouling of the water when kept in gasometers.

[1] *Vide* Brand, Méd. Lég.

There has been as yet no reliable record of death immediately caused by the use of this anæsthetic gas.[1]

[1] For further particulars with regard to this gas, reference may be made to Dental Cosmos, Jan. 1869; Med. Gaz., N. Y. (extract from the Loudon Lancet), Dec. 11, 1869; Gazette Hebdomadaire, Paris, Dec. 10, 1869; Dict. Enc. des Sciences Médicales, t. vii. pp. 690, 691; Boston Med. and Surg. Journal, N. S., vol. v. p. 91; Edinburgh Med. Journ., Jan. 1871; N. Y. Med. Journ., Aug. 1870; Am. Journ. of the Med. Sci., July, 1870, p. 61.

CHAPTER XII.

APPENDIX.

CASES OF POISONING ILLUSTRATING IMPORTANT MEDICO-LEGAL QUESTIONS.

[COMPILED BY CHARLES HARRINGTON, M.D.]

Case I.—Poisoning by Nitrous Fumes.

(Annales d'Hygiène Publique, 44, p. 345.)

§ 554. A workman, named Clementz, employed in a morocco factory, was found one day in one of the rooms lying flat on his back, hardly breathing. On removal to his house he regained consciousness, but in a few hours was dead. His fellow workmen had no doubt but that the real cause of his death was the inhalation of a red vapor, which came from a certain operation which he had carried on. His widow spoke of bringing an action for damages against his employer, who declared that it was his belief that death had not been caused by the fumes, but that the man had been sick already some time; that he drank, etc. etc. It was, therefore, deemed indispensable in the interest of the truth that an examination and chemical analysis should be immediately made. The viscera were well preserved, and emitted no putrid odor. The endocardium was notably inflamed. The liver presented neither lesion nor change in volume. The stomach and small intestines contained a few bits of macaroni and some white muscular tissue resembling veal. The whole intestinal canal was intact. The bladder was empty. Microscopic examination of the organs revealed no changes except in the lungs. The latter were almost entirely disorganized and swam in black blood which was partially coagulated. The tissue was so soft that in some places it was detached like jelly. Some portions of the left lung near the heart had, however, retained the normal structure. At these places the lung was merely congested, and when washed in a stream of lukewarm water it showed a yellow color. Not only did all the pulmonary tissue and the bloody fluid in which

it was bathed react strongly acid, but a marked nitrous odor was plainly perceptible on incision with the scalpel or with sharp glass. A piece of paper, wet with a solution of iodide of potassium and starch, held in the vessel containing the lungs was in a few seconds turned blue. The lung tissues were finely divided, and, together with the blood which had come from them, were submitted to chemical examination, which proved the presence of nitric acid. Among the articles used by the deceased in his work were nitric, hydrochloric, and sulphuric acids.

Case II.—Poisoning by Oxalic Acid.

(Boston Medical and Surgical Journal, Oct. 14, 1875.)

§ 555. S. S. A., an American, fifty-three years of age, and by occupation a carpenter, left home on a Wednesday morning to collect money that was due him. Nothing peculiar was noticed in his manner before leaving home. He returned about noon, and went directly to his barn (five minutes' walk from the house), took his horse from the sleigh, and put the blanket on without removing the harness. He also put the sleigh in the barn. He subsequently told his family that he lay on the hay about two hours, as he did not feel well. At two o'clock P. M. he walked to his house; his gait was slow and unsteady. When he entered the house he complained of intense pain in his stomach. His face and hands were livid and "cold as a stone." He could hardly speak, saying but a word or two at a time. He asked if there was any chalk or magnesia in the house. He took all that was at hand of those remedies. He also took a quantity of "pepper tea" and "ginger tea." He was drawn up on a lounge before a very hot fire, and external warmth was applied. He would not allow any one to mention that he was sick, but persisted in saying that he should soon be better. His condition remained about the same Wednesday night and Thursday. Whatever he drank, and he drank a good quantity of water, was soon vomited. Thursday noon, with assistance, he got up from the lounge, got into a chair, and was taken to his bedroom. At two o'clock in the afternoon he was seen by Dr. Marion by request of the wife, who said she was afraid he had pneumonia, thus showing that she suspected nothing. Mr. A. was lying on his back, partly dressed. His face and hands were livid and very cold.

There seemed to be no capillary circulation, indeed, there was no pulsation in the radial artery, and it was extremely feeble in the brachial. The patient's mind seemed clear, his pupils were normal. The tongue, which he protruded slowly when asked to, was slightly coated. His speech was labored, but a word or two being articulated at a time. Dyspnœa was marked; tracheal râles were very loud, so loud that a satisfactory examination of the chest was impossible. Even the heart sounds were inaudible. The patient had urinated and defecated.

Suspecting something wrong, Dr. Marion asked him if he knew any reason why he should be taken so very sick. "Yes," he said, "I have taken about two tablespoonfuls of oxalic acid, thinking it was whiskey." Drs. Marion and Hosmer in consultation concluded that antidotes would be of little avail, since it was now more than twenty-four hours since the poison was taken, and the patient looked as if he must soon die. Carbonate of ammonia was prescribed to be administered by the stomach, freely if it was retained; also brandy and nutritive enemata.

His condition remained the same until Friday morning, when he became delirious. In his delirium he got out of bed alone. He died at two P. M. on Friday.

Autopsy, twenty-six hours after death.—Rigor mortis complete. Lividity not as marked as during life, except on the dependent portions of the body. About ten ounces of bloody serum were in the right pleural cavity; the lungs were œdematous, otherwise healthy. The heart was flabby and larger than normal; the walls of the left ventricle were very much thickened. The left cavities of the heart were empty; the right, together with the venæ cavæ and pulmonary vessels, was filled with dark, soft coagula. The spleen was rather large, dark, and very soft. The kidneys were of about the normal size, and in the gross appearance nothing abnormal appeared, except that they were rather friable. The stomach contained about three ounces of straw-colored fluid. The stomach was of medium size, fleshy to the feel, if not a little stiffened, and had a somewhat dryish look upon the external surface, like a specimen which, having been in spirit, had been removed and left for some time exposed to the air. This same surface had a very marked appearance of fine lines running parallel lengthwise, finely set, and giving it a somewhat wrinkled look; and it had to a considerable extent a chalky-

white look, like a specimen that had been in a solution of corrosive sublimate. All of the veins about both curvatures were crowded with coagulated blood, and were very firm to the feel. The mucous membrane was quite rugous, and of a color throughout that strikingly resembled dark putty; there was nowhere any redness, abrasion, lymph, or any other indication of inflammation. In consistence it was firm, as were all of the other tissues.

Case III.—Poisoning by Oxalic Acid.

(The Lancet, September 28, 1867, p. 394.)

§ 556. Sarah S., aged thirty-four, married, and previously in good health, took voluntarily, on May 22, half an ounce of oxalic acid. Directly afterwards she felt a burning pain in the throat and epigastrium, and was giddy; in about fifteen minutes she was very sick; an hour afterwards she was purged violently, and passed blood in the stools; she also vomited blood. On admission to the King's College Hospital she had severe pain in the stomach, retching, great thirst, cold extremities, sunken eyes, and pallid countenance. Chalk and water, and afterwards half a drachm of carbonate of magnesium, were given her, with the effect of lessening the pain; and vomiting was kept up by a mustard emetic and warm water. At first her pulse could not be felt, but the circulation was soon restored, hot bottles being placed at her feet, and stimulants given internally. Vomiting continued through the night, and she passed much blood and mucus by the bowels. Milk was given her, but rejected. Her pulse was 104. Next morning she had great pain over the abdomen, and was very sick; her voice was very harsh; pulse 72. Enemata of milk and brandy were given.

The record for May 25 was that for the past two days she had had less pain and sickness; her mind had always been clear; slept better; purged a little.

On the 27th there was no purging, no blood in stools, tongue clean; slight headache, less pain, no sickness. On the 29th she was much worse; had severe pain across the abdomen, and had been purged a great deal. Constant sickness came on, and she died in a few hours, apparently from exhaustion. Her diet consisted of iced milk and beef-tea, with a little brandy occasionally.

Autopsy, twenty-three hours after death.—Body fairly nourished;

no lividity; slight rigidity of the extremities; tongue and mouth normal; epiglottis red and eroded on the under surface. Most of the mucous membrane of the œsophagus was stripped off, especially at the lower part. The stomach was congested and distended. At the lower part of the great curvature was a slough, irregular in shape, about the size of a crown-piece. The wall was thicker than usual here, and the peritoneal coat was opaque. The slough had not begun to separate. All through the intestines the walls were congested and thicker than usual. In the lower part of the ileum, for about three feet, and leaving off suddenly about six inches from the cæcum, were numerous sloughs of an ochre-yellow color, and involving the whole of the bowel, and not Peyer's patches or solitary glands only. The serous covering was congested. No ulceration in the large intestine, but the solitary glands were enlarged. There was no peritonitis. The trachea and bronchi were normal; lungs rather congested at their bases. The heart and liver were healthy. The cortical portion of each kidney was rather swollen and œdematous, and in some of the tubes there was an increased quantity of cells. The mesenteric glands were normal.

It is worth while noting that in this case the autopsy showed the tongue and mouth of the deceased unaltered in appearance, whilst a great slough was found at the lower part of the greater curvature of the stomach. It is rare for fatal cases of poisoning by oxalic acid to be unattended by a whitened and softened state of the mouth, and it is also rare, perhaps, for the acid to produce such strong corrosive action upon the cardiac and intestinal mucous membranes as was noticeable in this case.

Case IV.—Poisoning by Carbolic Acid.

(The Lancet, June 21, 1873.)

§ 557. A girl, aged seven, was admitted into the General Hospital, Birmingham, having ten minutes previously swallowed, by mistake, about half an ounce of carbolic acid diluted with twice that amount of glycerine and water. On admission the child was quite insensible; pulse hardly perceptible; pupils contracted; conjunctivæ insensible to touch; temperature below normal; respiration catching, and about sixteen to the minute. The stomach was washed out by

the stomach-pump. Some gruel and oil were given, and an enema of brandy administered, and the heat of the body maintained by appropriate means. The child died comatose one hour and fifteen minutes after admission.

Autopsy, twenty-four hours after death.—Rigor mortis was persistent. After ligaturing the venæ cavæ close to the heart, the right side was found to contain three ounces of blood, which was of dark color and quite fluid. This was the case also in the large vessels. The left ventricle had its walls firmly contracted, and contained about two drachms of blood. No abnormality was noticed in the lungs or any abdominal viscus, except the alimentary canal. The abdominal cavity smelt strongly of carbolic acid. The bladder contained about half an ounce of urine, which smelt strongly carbolic and responded to the tests for that acid. It contained no albumen. The tongue, mouth, pharynx, larynx, œsophagus, stomach, and the first two inches of the duodenum appeared quite unaffected by the drug. The small intestine for three feet was much changed. It was covered with a white eschar, the valvulæ conniventes having suffered most. The mischief seemed confined to the mucous membrane. The brain contained much venous blood, as did also the sinuses. The blood was quite fluid. There was no effusion. The ventricles were quite dry. There was no cerebro-spinal fluid whatever. The whole brain had a strong odor of carbolic acid.

Case V.—Poisoning by Carbolic Acid.

(The Lancet, March 1, 1873.)

§ 558. On February 13 a woman, aged forty-four, who was suffering at the St. George's Hospital with bronchitis and emphysema, received by mistake a dose of strong carbolic acid instead of senna. She was found by the house physician, who was immediately called, sitting up in bed, laboring under great dyspnœa, and apparently suffering acute pain. She was unable to speak, but kept her hands firmly pressed over the region of the sternum, as if to indicate that there was the seat of pain. She was very restless, and groaned continuously. The breath smelt strongly of carbolic acid, the interior of the mouth and lips was charred white, and there was a brown eschar on the chin. The pulse was quick (140 in the minute) and feeble. An emetic of sulphate of zinc mixed with

olive oil was immediately administered; she swallowed it with great difficulty. The endeavor to get her to drink warm water mixed with oil was without success, as she had lost the power of deglutition. No vomiting ensued, and she soon sank into a state of stupor, the breathing became slow and stertorous, and the pulse excessively feeble. The stomach-pump was introduced and warm water injected. She gradually sank, and died comatose fifty minutes after taking the poison. The amount of poison taken was not known at the time, but it was afterwards ascertained to have been nearly a fluidounce of the impure commercial carbolic acid.

The *post-mortem* examination was made twenty-eight hours after death. There was a brown stain on the chin extending to the angle of the mouth. Old pleural adhesions existed on both sides. The left lung was greatly congested, the right emphysematous; the bronchial mucous membrane of both was injected, and the tubes full of frothy mucus. The left ventricle of the heart was strongly contracted, the right partly so; the organ was normal. Larynx and trachea natural. The mucous membrane of the mouth, œsophagus, and stomach was converted into a soft white material, giving the organ very much the appearance of being covered with a thin layer of white lead. This easily peeled off, exposing a bright-red surface beneath. These appearances ceased at the pylorus. The stomach was strongly corrugated, and contained about two ounces of brown fluid smelling powerfully of carbolic acid. There were a few congested patches in the duodenum. The ventricles of the brain contained about an ounce of clear fluid; the organ was otherwise natural. All the other viscera were natural. The blood was uniformly fluid, and on exposure became of a bright-red color. No smell of carbolic acid could be detected in any of the viscera with the exception of the stomach.

Case VI.—Poisoning by Chlorate of Potassium.

(Annales d'Hygiène Publique, Jan. 1880, p. 543.)

§ 559. Monsieur X., aged 67, in good health, took on April 6, on account of lumbar pains, at 8.30 A. M., a part of a package of salt which had been delivered to him as a purgative by the servant and gardener of a health officer of a neighboring commune in the latter's absence. Mme. X., put some of the salt in a glass; the

amount used was conjectured to be about 35 grams. The glass was then twice filled with water somewhat hot, and the man drank the contents. Half an hour afterwards he had a copious dejection and vomited; towards eleven o'clock he vomited about a cupful of yellowish-green liquid. At half-past-eleven he had another dejection, and a little later vomited copiously; the vomitus was green. He had also a whitish dejection, and voided an unknown amount of urine. Soon afterwards, feeling weak, he went to bed, and complained of pains in the abdomen. At half-past-two, his wife, who had been out, became alarmed at the condition in which she found him. The lips were œdematous and blue. At 3.40, a health officer, who was called in, found him already dying; cyanotic hue, rigidity of the limbs, bronchial râle, and cold sweat. Death occurred at four o'clock.

The *autopsy* was performed eighteen hours later. Cadaveric rigidity was present. The back and loins were slate-colored; there was a small ecchymosis on the left ear. The digestive tract, free throughout its length, showed signs of inflammation of the peritoneum; it was tied just below the cardia and again at the lower end of the rectum, removed, and preserved in an hermetically sealed jar. The liver, spleen, and kidneys were engorged with blood of a deep green color. The lungs were normal; heart flabby, and filled with blackish-brown blood partly coagulated. Examination of the stomach showed a hemorrhagic patch near the pylorus, honeycombed, brown, and about as large as a five-franc piece. Some urine was in the bladder and it was preserved, as were also a reddish serous fluid from the peritoneal cavity, and the blood from the heart. These three fluids, together with those of the digestive canal, and the balance of the salt above mentioned, were submitted to chemical analysis. The salt was found to be chlorate of potassium, and it was detected in the urine but not in the other fluids.

Case VII.—Poisoning by Alum.

(Annales d'Hygiène Publique, 39, p. 192.)

§ 560. May 15, 1872, V. M., 57 years of age, who had suffered for several years from gastric troubles, wishing to purge himself, as was his habit, with Epsom salts, sent his son to an apothecary to procure the same. Towards midnight he took about 30 grams

dissolved in a glass of water. The symptoms which ensued were as follows: Feeling of constriction and burning in the mouth, throat, and stomach; nausea interrupted by but one fit of bloody vomiting; no movements of bowels; extreme discomfort, then unbearable anguish. Pulse small and frequent; respiration quick; repeated syncope. The mind and senses were unaffected.

Attributing all these symptoms to an unusual action of Epsom salts, the patient did not send for a physician until morning. The latter came at seven o'clock, and soon discovered the case to be one of poisoning, but having no knowledge of what the patient had taken, could not determine the nature of the poison. He ordered a half bottle "Limonade de Rogé," a strong infusion of coffee, enemata of castor oil, and poultices to the abdomen. The patient was already in a most desperate condition; pulse intermittent and wiry; skin cold; deglutition almost impossible. Death occurred eight hours after ingesting the poison.

The *autopsy* was made on the 17th. Decomposition slight; rigor mortis absent. Bluish discoloration of cutaneous veins of the thighs. The scalp, skull, and sinuses were anæmic; meningeal veins contained a little blood. Brain substance normal. Mucous membrane of the mouth, pharynx, and œsophagus covered with a grayish-yellow coating, and easily detached. Tongue and tonsils swollen. An effusion of coagulated blood was observed between the trachea and œsophagus and in front of the thyroid cartilage. Peritoneum inflamed throughout; the cavity contained considerable brownish serous fluid. Omentum injected; its veins and those of the mesentery full of black blood, and hard. External surface of stomach grayish and strongly injected; veins filled with black blood, which was hard and friable, as though due to the action of acids or astringents. Mucous membrane gray and shrivelled; at the pyloric end disorganized and inflamed. The contents of the stomach consisted of a brownish fluid which attacked the polished surface of the scalpel; near the pylorus was an abundance of gray powder. The organ was somewhat contracted. Duodenum thickened, contracted, and of a gray color; in its upper part was more of the gray powder. Several bright-red exudations were observed under the peritoneal covering of the small intestine; notable injection of both the large and small intestines. In the first half of the latter were two oval gangrenous spots measuring ten and fifteen centimeters in the long

diameter. No well-marked alteration in the large intestine; the descending colon contained some semi-solid fecal matter of a greenish-brown color. Liver small, grayish, and hard; its peritoneal coat covered with a fibro-albuminous exudation of a yellowish-gray color. Liver substance anæmic. Biliary ducts contained calculi as large as a pepper-corn. Gall-bladder dilated, hypertrophied, and gray; it contained some pale-yellow bile, in which swam about 130 calculi as large as pepper-corns. Spleen small and contracted. Kidneys much injected. Bladder empty. Lungs normal; pleural cavities contained considerable bloody serum. Heart enlarged; walls thin; cavities contained some soft clots like currant jelly.

Chemical examination of the stomach and intestine proved the poisonous substance to be potash alum, which had probably been calcined.

This case is the first ever reported of fatal poisoning by alum.

Case VIII.—Poisoning of a Family by Carbonate of Barium.

(Vierteljahrsschrift für gericht. Med. und öffentl. Sanitätswesen, Bd. xxviii. p. 248.)

§ 561. On March 29, 1874, the family B., of Hamburg, celebrated the sixty-eighth birthday of the father. At five o'clock in the afternoon chocolate and cake were passed around. A piece of the cake was given to a canary and another to a dog. Five minutes afterwards the canary died without having shown any symptoms of disease. The dog was seized with vomiting and diarrhœa, and on the next day seemed very sleepy. Within from about a quarter to a half an hour after eating the cake the family were seized with similar symptoms. Herr B., aged 68, Frau B., aged 58, Frl. B., aged 19, and Frl. R., a friend, aged 17. The first to be seized were the young ladies, who turned pale, and complained of a peculiar sensation of stretching of the skin. In a short time this was followed by violent vomiting and diarrhœa. Shortly afterwards Frau B. was seized with the same symptoms; she described the sensation in the skin of the face to be as though she had washed in hot water. Herr B. was taken with general discomfort and rumbling in the abdomen. Meanwhile, they were visited by two persons, who suspected as a cause of the symptoms the cake which already was half eaten. In consequence of their suspicions they

declined to eat any, and remained well. Following their advice the three ladies, who had already taken "Hoffmann's Drops" without effect, drank large quantities of milk; but Herr B. firmly refused to take any. After drinking the milk increased vomiting occurred, which, however, soon diminished. In the evening the ladies were better, though on the next day they felt weary and devoid of strength, and complained especially of weakness in the legs. Herr B., on the contrary, suffered during the whole evening, and not before ten o'clock could he be persuaded by his wife to drink milk; violent vomiting followed. In the course of the night diarrhœa set in; toward four o'clock in the morning, on going to the water-closet, he fell to the floor, and required the assistance of his wife and daughter to get back to bed on account of loss of power in his legs. Toward morning the vomiting ceased, but a constantly increasing paralysis from below upwards was developed. At ten o'clock he was only able to nod his head. At two in the afternoon he was found by his physician with a congested face, bolstered up in a half-sitting posture in bed; voluntary motion of the arms and legs was quite impossible, and articulation was difficult. Sensation in the paralyzed parts was unimpaired, and reflex action was very manifest on tickling the feet. The sphincters were unaffected; the breathing was somewhat increased in frequency, and there were tracheal râles; the sensorium was affected. Pulse and temperature were normal; there were no pains even in the region of the stomach. In the evening, at nine o'clock, twenty-eight hours after eating the cake, he died. He had eaten no more of the cake than any of the others who were seized.

The *autopsy* was deferred until sixty hours after death, when decomposition had made such progress that the results were quite negative. The only noticeable features were the prominent dark color of the gray substance of the brain, and the apparently complete absence of injury to the mucous membrane of the throat, œsophagus, stomach, and intestine.

In order to obtain more knowledge concerning the nature of the poison, the experiment of feeding birds with the cake was tried. A few minutes after eating they were seized with unsteadiness in the legs, and were soon unable to stand without falling. Flying then became impossible, and in a few minutes more the birds were lying dead. From several experiments it was noticed that the paralysis

did not appear simultaneously on both sides; the birds always fell on the same side. A dog was given 20 grm. of the cake, and in 2½ hours was seized with vomiting and diarrhœa, but recovery followed. The same symptoms occurred in birds and a dog which were fed on the flour from which the cake was made.

The analysis of the flour showed the presence of 10 per cent. of carbonate of barium, and of 0.3 per cent. of sulphate of barium; the cake yielded 2.74 per cent. of the carbonate and of 0.43 per cent. of the sulphate of barium. The contents of the stomach of the deceased, amounting to but 8 grm., yielded perceptible traces of the barium. At about the same time (April 6 and 7) it became known that several persons in two separate parties had become ill after eating cake. Both cakes came from the same confectioner, who obtained his flour at the same shop as the family B. Consequently the two specimens of cake were analyzed for barium, although two chemists had already pronounced them free from injurious substances. Carbonate of barium was found in both in very considerable amount. The flour from which all the cakes were made came from a shop where drugs, paints, and chemicals were sold. A wilful and malicious adulteration seemed improbable, while a careless admixture was more to be conjectured. It is probable that it had been the intent to adulterate the flour with chalk, and that the barium had been accidentally substituted.

Case IX.—Poisoning by Phosphorus.

(Annales d'Hygiène Publique, 1880, p. 256.)

§ 562. On April 27, 1880, D., a reporter, 47 years of age, entered the Pitié in the service of Dr. Gallard. On the 26th in the evening, suffering from some mental trouble, he had determined to poison himself. He bought a thousand matches and soaked the ends in a half liter of red wine, which he had previously warmed. He then removed the wood of the matches, sweetened the infusion, and drank the wine and the dregs. The wine had a well-marked alliaceous taste. It is impossible to state whether all the phosphorus of the matches was dissolved (or detached), since, after withdrawal, they were immediately burned.

In order to determine the amount of phosphorus contained in 1000 matches, a lot of similar matches were submitted to a chemist

for analysis; the result showed that a thousand matches contained 0.58 gram of phosphorus. On soaking the matches in wine and analyzing the liquid and the dregs, the amount of phosphorus in solution and in the sediment was found to be 0.523 gram for a thousand matches.

As soon as he had taken the poison, the man began to carefully note his symptoms. He found that his pulse was quickened and counted 104 beats; he noticed also that his temperature was rising, and soon felt a burning sensation, not very intense, however, in the pit of the stomach. His breath had an alliaceous odor, but there being a light in the room, he noticed no phosphorescence. Being seized with regret at what he had done, he acknowledged everything to his young son, who hastened to call in a physician. The latter prescribed an emetic, milk, and spirits of turpentine to be taken by the spoonful every hour. Vomiting occurred ten hours after taking the poison; until that time there had been no vomiting or nausea, but afterwards he vomited nearly all he had taken. He had two copious dejections of an alliaceous odor after taking the emetic. Several times he had painless contractions in the right arm, causing flexion of the hand and forearm. All the turpentine taken was almost immediately vomited.

On arrival at the hospital the patient was somewhat calm; temperature 38° C (100.4° F.); pulse 108. The epigastric pain was diminished, and the vomiting less frequent; but everything taken was soon ejected. On the way to the hospital he had had contractions of the right arm. Respiration was quiet and regular; there was nothing abnormal discovered on auscultation of the lungs and heart. The breath was strongly alliaceous. Iced milk, and twenty capsules of turpentine were prescribed.

The patient passed a quiet night; had a number of cramps in the right arm; vomiting was renewed, but the epigastric pain was much diminished. There was nothing remarkable about the stools except that they had a strong alliaceous odor.

Analysis of the vomited matters showed them to contain a certain amount of phosphorus, shown by the blackening of nitrate of silver paper by the vapors, although no sign of phosphorescence was given by the Mitscherlich apparatus, which fact may, however, be explained as due to the presence of turpentine. The analysis revealed small quantities of phosphorus. The matters vomited on the

28th were greenish in color and contained bile; there was no odor of phosphorus, but a strong one of turpentine. Analysis showed a minute amount of phosphorus.

In the evening of the 28th, the breath was less alliaceous; the epigastric pains, the cramps, and the vomiting were all sensibly diminished. The urine, which was rather dark, contained no albumen. Ice and turpentine continued; julep with extract of thebaia, 0.05, given. On the 29th, the patient had passed a quiet night; more nausea; he took, and retained, some broth, though he had but little appetite. The tongue was covered with a yellowish granular coating; the epigastrium was still sensitive to pressure. The skin was somewhat jaundiced, and, more particularly so, the conjunctivæ. The liver was of normal size. There was still a little generally increased excitement. The urine was of a light mahogany color, and contained albumen and biliverdine. More contractions. Temperature at night, 39.6° C. (103.3° F.). On the increase of temperature the patient felt better; he was able to eat and retain his food and iced drinks. The jaundice increased; no vomiting; no diarrhœa; no abdominal pains. The administration of turpentine was continued. On the 30th the jaundice was increasing, and the urine was more albuminous. The treatment was continued.

May 1st. Constipation; urine very scanty, turbid, and contained much albumen and biliverdine. The breath had a peculiar well pronounced odor, resembling that of mice. Turpentine continued—twenty capsules of 0.40 grm. Insomnia and headache. May 2, increased jaundice; more appetite; increasing weakness; dry tongue; obstinate constipation. Thirty grams of castor oil were administered; turpentine continued, but thebaia omitted. The weakness and the jaundice increased. The hepatic dulness was increased four centimeters. There were vague pains in the right hypochondrium. The urine was very scanty and thick, and there was great difficulty in voiding it. Considerable prostration; insomnia; intellect clear; pulse 120; heart's pulsations violent. Mouth dry; hemorrhagic crusts on the tongue; petechiæ on the chest. Severe pains in the right hypochondrium; jaundice increased. Respiration quickened; nothing found on auscultation. In the evening the pulse was 124. A very abundant hemorrhage occurred from the intestines. Prostration was rapidly increasing. There was very great dyspnœa. Very copious hemorrhages oc-

curred from the mouth and anus. Death took place at nine o'clock the next morning.

The *autopsy* was performed forty-eight hours after death. There was a general yellowish discoloration of the body, with greenish spots about the umbilicus and on both sides. There was no rigidity of the upper limbs, nor of the head; rigidity was present in the lower limbs. *Lungs.* Large and emphysematous. The internal surface of the bronchi was red. *Heart.* There was about a tablespoonful of yellowish fluid in the pericardium; no adhesions. Heart substance somewhat yellow, but no fatty degeneration. *Liver.* General yellow discoloration; in places more marked. Surface somewhat wrinkled, and marbled with green. The cut surface resembled that of nutmeg liver. The *kidneys* were somewhat congested, especially in the medullary portions. *Digestive tract.* Little redness on the epiglottis and on the surface of the larynx; some rugosity in the lower portion of the œsophagus; the longitudinal folds were more marked than normally. The œsophageal mucous membrane appeared a little congested. On opening the stomach a blackish fluid, which was bloody and of a putrid odor, was found; the fluid contained no particles of phosphorus. The gastric mucous membrane was vascular and somewhat thickened; on the greater curvature were some blackish spots beneath the mucous membrane, but there was no tissue alteration. The *intestines* contained a blackish liquid similar to that found in the stomach. *Brain.* Nothing noteworthy discovered in the brain and meninges. Ecchymoses were observed on the pleura, on the lungs (two very large ones), in the stomach, under the pericardium, on the mesentery, and intestines. The ecchymoses varied in size from small points up to a five-franc piece; on the intestines they produced in places a marbled appearance. Analysis of 100 grams of the fluids from the stomach and intestines yielded 0.00058 gram of phosphorus. Microscopic examination of the liver showed extreme fatty degeneration with interstitial hepatitis. Examination of the kidneys showed fatty degeneration, which was not so advanced as in the liver, in that the cells retained their form; the glomeruli were very little altered. Some of the tubules were filled with blood from slight hemorrhages.

Case X.—A Case of Phosphorus Poisoning.

(Vierteljahrsschrift für gericht. Med. und öffentl. Sanitätswesen. Bd. xxv. p. 25.)

§ 563. Frau H., a strong robust woman of 56 years, had, with the exception of a light attack of rheumatism some years before, and occasional headaches, always enjoyed perfect health up to November, 1861. In the beginning of that month she complained of pain in the back and of nausea, and, without feeling particularly sick, went to bed. On Monday, November 4, her husband cupped her; she was much relieved, and was able to be up and about. After a purgative she felt on Wednesday perfectly well, though she had some headache. In the evening she ate some soup prepared by her daughter-in-law; she remarked nothing concerning the taste. The daughter-in-law pressed her to take more, and poured out from another vessel a cupful, which she refused. She then ate some fish, which her husband had brought from a restaurant, and expressed herself pleased with the same. The husband ate what was left. The vessel, from which the woman had eaten the soup, was not then in the room, but he found the cup of wine soup and drank it after his wife had remarked that the soup had not agreed with her and had brought on pain in the stomach. This was said before she ate the fish. On the following day a physician was called in; she repeated the same to him, and added that she was still very sick; she complained of pain in the back, cardialgia, thirst, and nausea; the face was pale; skin cold; pulse small. She complained of pain from the back to the pit of the stomach, and that she had vomited during nearly the whole night. The physician was unable to talk much with her on account of her extreme restlessness. At four in the afternoon she was ice cold and very anxious and restless. She felt "as though her heart were being torn from her body." At five she was beyond hope; she was cold and in great anguish. Afterwards she became quieter and in a half an hour died. After death her skin was observed to be quite yellow in color. Although there were suspicions of poisoning entertained by a few, they did not reach the ears of the authorities, and the woman was buried. The suspicion, however, grew and spread, and the authorities caused an exhumation and examination to be made. The autopsy was performed on Dec. 19th, forty-two days after death. Decomposition had advanced to a stage naturally to be expected. The examination

of the head showed nothing remarkable. The larynx and trachea were not abnormal. In both pleural sacs were about twelve ounces of a bloody fluid, which was reserved for chemical analysis. The heart was pale, flabby, and completely empty. In the pericardium was about a spoonful of bloody fluid. The great vessels were empty; the lungs empty and not abnormal. The œsophagus showed nothing abnormal, but it was kept for chemical examination. The stomach contained a moderate amount of a brownish, slimy paste, in which numerous whitish earthy particles were observed. The gastric mucous membrane showed no change in color or character where these particles lay, nor were there anywhere any ulcerations or exfoliation of the epithelium. The color of the stomach at the fundus was much reddened, apparently from hypostatic congestion. The duodenum and upper part of the jejunum contained particles similar to those in the stomach; the rest of the intestinal canal showed nothing abnormal. The stomach and intestinal canal and their contents were reserved for chemical analysis, as was also the liver, which was as yet very little decomposed. The spleen, pancreas, omentum, mesentery, kidneys, and uterus showed nothing remarkable. The bladder was empty, and therefore no urine was obtainable for chemical examination. The chemical examination was completed and reported on Jan. 2, 1862. The stomach and contents gave off an odor of volatile fat acids, but nothing which suggested poisoning by phosphorus, prussic acid, alcohol, chloroform, or volatile alkaloids. The contents showed more the character of products of decomposition of the stomach than of food; no starch granules were to be found on microscopical examination. On chemical examination of the stomach and contents, no metallic poison, no sulphuric acid, prussic acid, morphine, narcotine, brucine, veratrine, strychnine, meconic acid, oxalic acid, chloroform, or alcohol were detected. The same was true of the intestine. The white particles spoken of above were examined microscopically and found to differ from one another; some appeared to be vegetable in character. On chemical examination of the duodenum, during the process of digesting in very dilute acid, a luminous point was observed on stirring the mass; this came to the surface and burned like phosphorus. This phenomenon did not occur again. A portion of the substance heated in a flask gave off vapor which turned a piece of paper moistened with nitrate of silver deep brown, and made a film having a metallic lustre and

resembling that produced by the action of phosphorus vapor on nitrate of silver. The substance was then further examined for phosphorus, which was discovered. On examination of the lower portion of the small intestine and the large intestine, the phenomenon above described occurred again. Free phosphorus was also detected here by chemical means. No free phosphorus was detected in the liver. Examination of the stomach for phosphorus yielded but a trace. The estimated amount of phosphorus in the intestines amounted to 0.0940 grm. (1½ grains); the exact amount was probably more. The amount which was administered was probably considerable; the frequent vomiting had probably removed more or less. The discovery of the poison after such a long interval is very remarkable.

It was shown by the legal inquiry that the poison had been administered by the daughter-in-law in the soup, and that she had disguised the taste by adding a large amount of sugar.

Case XI.—Poisoning by Phosphorus Paste.

(Medical Times and Gazette, March 25, 1882.)

§ 564. Isabella S., aged 18, by occupation a laundress, was taken to the out patient department of the Royal Free Hospital on January 12th by her sister, who stated that several days previously her sister (either accidentally or, as there was some reason to suspect, intentionally) had taken phosphorus paste in some hot broth, and had since become very ill. On entrance the patient was seen to be a fairly nourished, well-developed girl, looking sallow and as if in pain. She stated that on January 8th she partook of some broth, in which was placed some phosphorus paste spread on bread. After taking the broth she became very thirsty, and experienced a burning sensation in the stomach and chest, and a peculiar taste in the mouth. The food was taken at about 8.30 P. M. At midnight she felt sick, but did not vomit until 4 P. M. on the 9th. The vomitus was dark green. She partook of milk, tea, and beef-tea before admission to the hospital. On the morning of the 9th she complained of headache, giddiness, burning in the fauces and mouth, and at the pit of the stomach; and during the afternoon was sick. The vomitus on being thrown away at night exhibited phosphorescence, and her sister remarked on the "fire-

works." A rash came out on the face, especially at the angles of the mouth, appearing as red spots with dark centres. On the 12th it appeared as small erythematous puncta at the roots of the hair, on the forehead, and at the angles of the mouth. The symptoms were unaltered, and on the 12th she sought admission to the hospital. She was then collapsed and jaundiced; pulse 80, feeble; temperature 98.2° F. She complained of severe pains in the chest and epigastrium, with general tenderness of the abdomen. Extreme thirst, anorexia, free perspiration, conjunctivæ yellow, tongue furred and of a strawberry appearance. Heart and lungs normal on auscultation. Liver dulness was increased; area of dulness painful on pressure. Slight cough; throat congested; mucous membrane reddened and thickened.

Treatment.—Mucilaginous drinks and magnesia; fomentations to abdomen.

Diet.—Beef-tea, eggs, and brandy.

On January 13th she had diarrhœa; stools green with some dark streaks; condition unaltered. Pulse slow, feeble, 80; temperature 97° F.; no retention of urine; trace of albumen in the urine. In the evening the pulse was 140, feeble; temperature 99.4° F. Pain increased and more general. Vomitus consisted of matter resembling coffee-grounds, and bile mixed with mucus. Slight delirium. At about 4 P. M. on the 14th sudden collapse occurred, and she speedily sank after vomiting some blood.

The *autopsy* was performed thirty-six hours after death. Body of a pale young female. Skin of face and body of a dusky color; posterior part of trunk, legs, and arms covered with confluent purpuric patches. Minute hemorrhages in the mesocolon and in the peritoneum covering the posterior part of the abdomen; a few also in the mesentery and walls of the small intestine, all of small size. Peritoneal cavity contained about half a pint of ale-colored fluid. But a very small portion of the left lobe of the liver about the size of a half crown was visible below the ribs. Liver fairly firm, and of a reddish color speckled with yellow; on section it looked remarkably fatty. Under the microscope the cells were seen to be completely fatty and broken down. Spleen, some punctiform hemorrhages were visible. Lungs, tissues of mediastinum, especially around the roots of the lungs, were filled with small, black hemorrhages. Subpleural hemorrhages abundant also at bases and back

of lungs. Heart, hemorrhages were observed around the bases of the great vessels. Ventricles fairly contracted and firm; tissue pale and opaque. Stomach contained about three pints of "coffee-grounds" fluid; few small hemorrhages. No source of hemorrhage detectable. Kidneys, when examined microscopically, in salt solution, almost completely disintegrated; hardly a perfect cell present; fluid filled with free nuclei and abundant fat drops. The brain was normal.

CASE XII.—A CASE OF POISONING BY ARSENICAL FLY-PAPER.

(Vierteljahrsschrift für gericht. Medicin und öffentl. Sanitätswesen, Bd. xxii. p. 243).

§ 565. The victim P. A. had been in perfect health up to his wedding-day in June, 1871, and up to July 8th of the same year. A slight fever, which he had had at Whitsuntide, lasted but a few days, and bore no resemblance to the case about to be described. On July 8th at four o'clock in the afternoon he went home and drank some coffee which had been prepared by his wife. He noticed no peculiar smell or taste. In about a half hour he complained of burning and pain in the stomach, vomited violently and copiously, and was obliged to drink constantly. This state, with the exception of the vomiting, continued on the 9th, 10th, and 11th, yet on the 10th and 11th he was able to work in the field, though he still complained of great weakness. On the 12th he drank more coffee, which was given him by his wife, and immediately vomited again; he complained anew of increasing pains, and vomited a bile-colored vomitus in copious amount. He was unable to go to work in the field, but toward evening took care of his cow. On the 13th and 14th he was unable to work, complained of weakness, great burning in the stomach, distressing thirst, and vomiting. On the 15th he went to a physician; his tongue was quite clean, there was no tenderness in the abdomen except in the region of the stomach, and there was no diarrhœa. He went home to bed, took the prescribed medicine and immediately vomited. He suffered constantly from distressing thirst, burning, and vomiting. These conditions lasted during the two following days. On the 18th the physician prescribed medicine to control the vomiting. His wife administered the medicine, which he immediately vomited. On the 19th he was somewhat better; in the evening his wife gave him more coffee,

which after a time he vomited. The vomiting continued and did not cease until death. He died in the night and was buried on the 22d. On account of suspicions of poisoning he was exhumed on August 12th (23 days after death) and an autopsy was held.

The *autopsy* showed, among other conditions, the following, which are of interest:—The odor of decomposition was very strong, the odor from the abdominal cavity was, however, but moderately foul. The abdominal wall presented a leathery resistance to the knife. The stomach was empty; the mucous membrane of the same was covered with a dark greenish-gray mucus. The mucous membrane was nowhere decomposed or ulcerated, somewhat raised with blisters (gas), and in the lower curvature was very strongly colored grayish black. On cutting into it it could be readily peeled off. The mucous membrane of the upper portion of the small intestine was covered with the above described mucus, and was also partially pink colored; it was free from ulcers and foreign accumulations. The large intestine was empty down to the lowest portion, which contained a small amount of grayish mucus. The mucous membrane of the large intestine was slate-gray, in spots reddish. The consistence of the liver was diminished, the color dirty brown-red, in spots gray-yellow. The spleen was broken down and dirty green; the kidneys were softened and dirty red-brown. The bladder was empty; the mucous membrane of the same was pink. The heart and great vessels were empty. The other *post-mortem* appearances were not remarkable.

The organs of the abdomen were submitted to analysis, and they, as well as the contents of the intestine, were found to contain arsenic. The intestinal contents amounted to 208 grms., and contained 4.16 grms. of arsenious acid. The presence of this poison, added to the fact that diseases having similar symptoms could be excluded by the results of the autopsy, leaves the consideration of anything but poisoning useless. A brief *résumé* shows that the attacks of vomiting came on always immediately after drinking coffee, etc., given to the man by his wife. It is interesting also to note that the stomach contained no foreign matters (arsenical preparations), and at the same time that the arsenite of sodium, which is a component of fly-paper which the accused had purchased, is easily soluble especially in hot water (in this case coffee), and imparts no characteristic taste or smell to the same.

The finding of the court in this case was that death was occasioned by poisoning by arsenic administered repeatedly in coffee by the wife.

Case XIII.—Poisoning by Arseniuretted Hydrogen from the Separation of Silver from Lead Ore.

(Vierteljahrsschrift für gerichtliche Medicin und öffentliches Sanitätswesen, Bd. xviii. p. 269.)

§ 566. Dr. Trost, of Aachen, communicates a case of poisoning by arseniuretted hydrogen, by which nine persons were affected, of whom three died. At the lead works at Stolberg near Aachen, it was the custom to extract silver from lead ore by means of an old, expensive, and long process. Director Herbst, in Kall, formulated a new process for the extraction, which was as follows:—

To the molten silver-containing ore is added 1½ per cent. zinc; the mass is then heated to a higher temperature, since the melting point of zinc is higher than that of lead. The silver will by this means form an alloy with the zinc, and the alloy will appear on the surface on cooling. The alloy is then skimmed off and treated with hydrochloric acid, first cold, and then by moderate heat. The chlorine unites with the silver to form insoluble chloride of silver and with the zinc to form soluble chloride of zinc, while the hydrogen is set free.

This process was carried out in September, 1869, at the lead-works at Stolberg. On September 1st, at 8 A. M., several hundred weight of the alloy were treated in an iron kettle with several hundred weight of hydrochloric acid. The place in which the operation was carried on was a shed open on one side; in the roof were holes for ventilation. The mass in the kettle was stirred with an iron bar, and then heated. Although the formation of arseniuretted hydrogen had been considered (since zinc and lead almost always contain arsenic), yet it had not been thought that the danger was so great as it afterwards proved. It may here be stated that by the legal examination of the materials used they were shown to contain large amounts of arsenic; the hydrochloric acid in particular contained 0.027 per centum. The operation was therefore performed in nothing less than a Marsh apparatus on a large scale, the gas evolved being diffused in the room. On the first day of the operation towards noon, a number of workmen, who had stood

near the kettle, were seized with symptoms of poisoning; the symptoms were, however, not much noticed. In the evening several others were seized, and on September 2d (the second day of the experiment) the operation had to be discontinued on account of the sickness of the workmen. The symptoms were the same in all the cases, and the treatment which was carried out by several physicians was without special influence on their course.

Of the six cases of recovery one lasted but a day, one a little over two months, and four still longer. It will be enough to describe the course of those cases which ended in death.

1. Leonard M., 37 years old, worked during both days, adding the hydrochloric acid and stirring the mass. Toward evening of the second day he was seized with a feeling of weight in the limbs, headache, abdominal pains, nausea, and vomiting. In the night the feeling of weakness increased, the vomiting was more frequent; he had several liquid stools, bloody urine, delirium, and stupor. On the morning of September 3d he was found unconscious, with swollen face, the skin and the conjunctivæ colored yellow. The patient could be roused from the stupor for but a few seconds, and that with the greatest difficulty. The extremities were cold; the face covered with cold sweat; deglutition impossible; pulse small, 150 to 160. During the day he passed some bloody urine. Death occurred towards nine in the evening.

2. Th. S., 40 years old, oversaw the operation from its beginning to its discontinuance. In the evening of September 2d he was seized with the same symptoms as Leonard M. On the morning of September 3d his condition was worse, and the abdominal pains more violent. In addition, he had pain in the region of the kidneys, greater sense of weight in the limbs, dry and coated tongue, great thirst, but slight excretion of urine, and that bloody. The face was swollen; the skin of the entire body and the conjunctivæ colored brownish-yellow; the head was hot. In the course of the day he had several brownish-black stools. As he passed no urine, the catheter was applied, but nothing but coagulated blood smelling of urine was obtained. The morning pulse was 110; the evening 130. On September 4th pulse 130; body temperature 38½° Centigrade; stools and urine as on the preceding day. September 5th and 6th, increased weakness; beginning delirium; restlessness; swollen abdomen; pulse 130 to 140; stools and urine as before. September

7th, sensorium dulled; stupor; face sunken, tongue, and gums black; deglutition very difficult; pulse 160. Death occurred between three and four o'clock on the morning of September 8th.

3. Michael R., 36 years old, had worked at the kettle and was seized with the symptoms on the first day. The symptoms were like those of the two cases above. Death took place at 9 o'clock on the evening of Sept. 3d.

Autopsies were performed in all of the above cases and with the following results:—

1. Leonard M. *Autopsy thirty-six hours after death.*—Among the external appearances the following were worthy of note: skin dirty yellow; about 30 grm. of a blackish fluid smelling like garlic ran from the nose and mouth on turning the corpse; the whites of the eyes were colored yellow, and the pupils were widely dilated. The mucous membrane of the mouth and lips was colored dirty yellow.

The internal appearances of especial interest were as follows: The dura mater not injected; the pia mater much so. A slight layer of bloody serous fluid on the surface of the convolutions. The mucous membrane of the trachea, larynx, tongue, and mouth was colored greenish-yellow; that of the œsophagus dirty-yellow. The liver was moderately large, and of a greenish-yellow color; the tissue of the same hard and not rich in blood. Both kidneys were firm and congested; both colored dark-red. Bladder empty. The larger abdominal bloodvessels filled with dirty colored dark blood. The tissues of the body in general were colored dirty yellow.

For chemical analysis were reserved the fluid from the nose and mouth, pieces of the lungs, heart, larynx and trachea, liver, spleen, both kidneys and the stomach.

2. Theodor S. *Autopsy twenty-eight hours after death.*—External appearances of especial interest were as follows: Skin, especially that of the face, was of a yellowish color; whites of the eyes dirty yellow; pupils half dilated. Lips and gums dirty blue. Abdomen somewhat swollen. Internal appearances: Pia mater injected; thin layer of serous fluid on the brain's surface. Auricles of the heart contained about an ounce of dark coagulated blood; the left ventricle was empty; the right contained a teaspoonful of coagulated blood. The pleural cavities contained about four ounces of dirty bloody fluid. The mucous membrane of the larynx and trachea was colored dirty yellowish-green, as well as that of the mouth

and fauces. The mucous membrane of the œsophagus was dirty yellow. The stomach contained a spoonful of yellowish mucus. The liver was normal in size, firm, yellowish-brown in color, and not congested. The right kidney much congested, dark-red in color, normal in size; the left increased by a half in size, much congested, and of a brownish-black color. The bladder contained about a spoonful of bloody urine. There were reserved for chemical analysis the fluid from the pleural cavities, the heart, portions of the lungs, larynx with trachea, portions of the liver and spleen, both kidneys, and the stomach.

3. Michael R. *Autopsy sixty hours after death.*—Among the external appearances were the following: The skin was bluish-green in color, and in places raised in blisters; whites of the eyes dirty yellow. A dirty dark-red fluid ran from the nose and mouth, on turning the corpse. The thorax, abdomen, and scrotum were distended. The internal appearances of interest were as follows: Dura mater not injected; pia mater much so; the surface of the brain covered with a thin layer of bloody serous fluid. The cerebrum on removal broke down to a dirty yellow pap. In the pleural cavities was but a little dirty, dark bloody fluid. The lungs were bluish-green in color. Heart empty. The great vessels contained a little dirty, dark-red blood. The mucous membrane of the larynx and trachea, mouth and nose was of a bluish-black color; that of the œsophagus was dirty yellow. The liver was enlarged, not very firm, contained not much blood, and was of a slate-blue color. The coats of the stomach dirty yellow, the stomach contained some fluid. The mucous membrane of the posterior wall of the same was, for an area of two square inches, colored black-gray, and was easily detached. The spleen was enlarged and congested; the tissue soft and blackish-red in color. The kidneys normal in size, much congested, and of a dark-red color. Bladder empty. The large abdominal bloodvessels contained some dark, dirty blood. The tissues of the body generally were of a dirty yellow color.

There were reserved for chemical analysis the fluid from the nose and mouth, portions of the lungs, the heart, larynx, and a portion of the trachea, part of the liver with the gall-bladder, the spleen, kidneys, and stomach.

The portions reserved in each case were analyzed, and arsenic was discovered in all, especially in the viscera.

From the above cases it will be seen that the symptoms of poisoning from respiration of the gas and from internal administration of the arsenic are very similar. And the *post-mortem* appearances of the two are also very similar, though in the former the local appearances are more prominent in the air-passages, and in the latter in the stomach and intestines. In these cases the poison was introduced into the blood by the mouth, etc., and lungs. The gas in the body was resolved into arsenic and hydrogen; the arsenic was carried to the various parts of the body by the blood, upon which it, meanwhile, had its effect, and thus indirectly on the organs. The finding of the court in each of the above cases was that deceased came to his death by poisoning brought about by respiring arseniuretted hydrogen.

Case XIV.—Poisoning by Arsenic in Coffee—Narcotic Form.

(Maschka's Handbuch der gerichtl. Med., vol. ii. p. 260.)

§ 567. Four persons soon after drinking coffee in the afternoon of February 26th, were seized with abdominal pains and frequent vomiting. Frau N. was as though unconscious, and could not move. Minna Z., a young girl six years of age, lay in a condition of supposed sleep; three hours after drinking the coffee she awoke, complained of pain in the abdomen, requested drink, took some sweetened water, and again fell asleep. On proceeding later to give her some medicine prescribed by the physician, she was found to be dead. Frau N. and her servant girl were for four days very ill; they vomited frequently. Herr N. was sick several days, but not confined to his bed. The autopsy of the child occurred on the day after its death. A foamy and somewhat bloody fluid ran from the nose and mouth; nothing abnormal about the mucous membrane of the mouth. The cavity of the abdomen contained about 60 grm. of yellowish clear fluid. The stomach gave off no unnatural odor; it was dilated with gas. The contents of the stomach and duodenum consisted of a grayish-yellow, faintly acid fluid containing a small amount of food and mucus. The mucous membrane was pale, the glands decidedly swollen; otherwise nothing remarkable. The organs of the abdomen were generally anæmic, but not abnormal. The lower part of the omentum was considerably injected, as was also the peritoneal coat of the small intestines. The small intestines

contained a grayish-yellow slimy fluid. The mucous membrane was pale, the follicles swollen. The large intestine contained a yellow thin material like thin pap. The large bloodvessels of the abdomen contained a moderate amount of dark fluid blood. There were no remarkable appearances about the organs of the chest. The pia mater was richly injected, œdematous, and easily detached. The sinuses contained fluid blood. There was nothing abnormal about the brain.

The chemical examination proved the presence of arsenic in the coffee, and in the organs of the young girl. It was found in the stomach and contents, intestines and contents, brain, lungs, heart, spleen, liver, and kidneys. There was no odor of decomposition from the stomach and intestines on the ninth day.

CASE XV.—POISONING BY ARSENIATE OF COPPER. (Vert de Mittis.)

(Annales d'Hygiène Publique, Jan. 1880, p. 23.)

§ 568. Leontine Puthomme, aged 17, took on December 7, 1878, at 5 P. M., fifty grams of *vert de mittis* in water. Vomiting occurred in a half an hour and was assisted by the administration of an emetic. On the following day she was admitted to the Lariboisière Hospital. Vomiting was incessant; there was coldness of the extremities, prostration, and a small weak pulse. Epigastric pain very pronounced. Dejections diarrhœic, and yellow. She complained of dimness of vision; there was no characteristic eruption and no paralysis. She died on the evening of the twelfth. Prostration steadily increased from the first day. There was no delirium until several hours before death. Vomiting and diarrhœa had ceased since the previous evening.

The *autopsy* was performed on December 14th. The integrity of the mucous membrane of the digestive tract was quite unimpaired. There was a slight congestion of the pharynx without any ulceration. The mucous membrane of the stomach was pale, and of perfectly normal appearance; the same is true of the mucous membrane of the intestines. The liver was yellow, and fatty degenerated. There were no well-marked lesions in the other organs. Chemical analysis of the organs, vomited matters, and urine, gave the following results, which are expressed in milligrams:—

	Arsenic.	Copper.
1. Brain	2.4	1.81
2. Stomach and Pancreas—		
in 42 grams	0.4	
in 47 grams		0.20
3. Liver	13.3	9.02
4. Heart	trace	0.51
5. Lungs—		
in 100 grams	0.70	0.47
6. Intestines—		
In 100 grams	0.50	0.30
7. Kidneys	1.50	0.76
8. Hair, 9 grams	0.10	0.00
9. Mammæ—		
in 60 grams	0.20	
in 64 grams		0.30
10. Muscles—		
in 40 grams	0.10	
in 55 grams		0.12
11. Urine, 69 c.c.	0.17	trace
12. Vomitus, 286 grams	0.28	0.25
13. Vomitus, 0.977 grams	1.41	0.79
14. Vomitus, 16.54 grams	2.494 grams	0.843
15. Vomitus, 2.409 grams	0.526 grams	0.316

Analysis of vert de mittis shows it to contain 22.64 per cent. of arsenic, and 14.93 per cent. of copper; a portion of the arsenic is present as arseniate of sodium; 100 grams of the vert de mittis contains 4.82 grams of arsenic in this form. The girl ingested 50 grams of the substance which, by the above analysis, corresponds to 11.32 grams of arsenic, and 7.46 of copper.

Case XVI.—Poisoning by Arsenic.

(From the short-hand reports of the trial.)

§ 569. Miss H., a woman of about 38 years, strong and well, took from her brother on May 1, 1879, at 10 o'clock P. M., a cachet of what purported to be soda, which he said was to settle her stomach, although there was nothing the matter with that organ. She soon went to bed, and at about midnight awoke in great distress. She complained of a "stopping of all her functions" and of a creeping sensation. At her request she was rubbed. The brother then brought her some "Jamaica ginger," which she drank. She then slept until between 5 and 6 A. M.,

when she awoke and complained of weakness and exhaustion. The brother then said he would give her some mustard, mixed something and gave it to her. Soon afterwards she called her aunt, complained of nausea, and asked for some mustard. This was given her, and she vomited. The brother, who was meanwhile away from the house, returned at noon and carried her some tea. He then went to a physician, told him something of the case, and was ordered to give magnesia once in two hours. This he gave at three o'clock in milk, and at five in water. When preparing this he was each time alone in the pantry with the door shut. She grew worse and vomited. At seven o'clock she received a third dose, and the vomiting continued. The brother then started leisurely for a physician. When seen by the latter she was lying on her right side and back with the legs slightly flexed; she was quiet, pale, and somewhat bluish under the eyes. She had nausea, but no pain; the body was cool; pulse weak at right wrist, almost wanting at left. Hot bricks and mustard paste were ordered. Jamaica ginger and whiskey were given; also subnitrate of bismuth for the nausea. The vomitus was about a pint of thick, slimy, greenish fluid. At midnight she was in no pain, and fully intelligent. Respiration was difficult and micturition impossible; she had several loose discharges. She was given a teaspoonful of whiskey, which she swallowed; she could take no more. Soon afterwards she was dead, just before death she was unconscious and nearly pulseless; pupils dilated. The autopsy was performed by the medical examiner on the following day. There was no cadaveric smell. There was a general blueness of the skin, especially of the ears. There was some fluid on the brain surface. The lungs contained air and much dark bluish or black fluid blood. Heart normal. The stomach looked dark blue before removal; it was red or bluish-red at the upper part. The vessels of the stomach and intestines were unusually full. The mucous membrane of the stomach was reddened throughout, and marked in places by spots of a brighter red. The intestine was reddened for two or three feet. There were some particles of white powder on the gastric mucous membrane and some gritty particles on the intestinal. The lower portion of the intestinal was normal. The liver was normal in size, congested, and fatty degenerated. The kidneys were congested; one kidney had a congested patch under the capsule. Spleen nor-

mal. There was dark fluid blood everywhere. The heart was full, but contained no clots. The contents of the stomach were about eight ounces of a dark brown fluid.

The *post-mortem* appearances were, therefore, not inconsistent with the theory of arsenic poisoning. The absence of pain during the sickness and the creeping sensation in the skin were thought to have been the result of administration of morphine. It was known that the accused had arsenic and other chemicals in his possession, and that on the day previous to the sickness he had purchased eight grains of morphine, of which but six remained. The organs of the deceased were submitted to Drs. E. S. Wood and H. B. Hills of the Harvard Medical School for chemical analysis. Arsenic was found in the following amounts:—

In the stomach	5.78 grains.
In the intestine	3.86 grains.
In the liver	2.77 grains.
In the brain	a trace.
Total about	12½ grains.

The prisoner's counsel made a very able plea, and in the absence of conclusive proof, the accused was acquitted.

Case XVII.—Poisoning by Arsenic with Fatty Degeneration of the Liver, Kidneys, and Gastric Glands.

(Transactions of the Massachusetts Medico-Legal Society, vol. i. No. 1, p. 43.)

§ 570. Harry C., aged four years, previously in good health, was taken on the morning of Nov. 19, 1877, with vomiting and gastric distress. As the physician who was called failed to recognize the true nature of the attack, and no appropriate remedies were administered, the case may be said to have progressed without treatment. According to the statements of the child's attendants the symptoms above mentioned continued with increasing severity until the morning of the 21st, about forty-four hours after the onset of the attack, when death occurred, preceded by stupor and convulsions. The vomited matters, which were dark-colored towards the last, were thrown away. Suspicions having been excited as to the cause of death, the medical examiner, Dr. Pinkham, was summoned to investigate the matter and an autopsy was made thirty-four hours after death, Dr. Lovejoy assisting.

The body was found to be well nourished. It presented the usual appearances resulting from hypostatic congestion. There was no rigor mortis. A careful examination was made of all parts accessible without section, and nothing abnormal discovered except slight parching of the lips, and some excoriation on the inside of the right cheek.

On section of the head the dura mater was found firmly adherent to the calvaria, as is usually the case in young children. There was a moderate amount of serum in the lateral ventricles, but perhaps no more than normal. The brain and its membranes were in appearance healthy. On laying open the abdominal cavity, attention was immediately arrested by the large size and bright yellow color of the liver, the condition of which was obviously one of extreme fatty degeneration. On raising its lower margin the gall-bladder was seen to be distended with bile, some of which had transuded, staining the tissues beneath. The spleen was of normal size and color. The kidneys were of somewhat lighter color than usual, this appearance being especially observed in the cortical portion after section ; but some of the pyramids of Malpighi were very dark. The stomach was removed entire, ligatures being placed above and below, and it, together with the liver, kidneys, and spleen, was sent to Dr. W. B. Hills, of the Harvard Medical School, for a chemical and microscopical examination.

The intestines were carefully examined, and it was found that the mucous membrane of the duodenum was intensely red and swollen, and covered with a tenacious coating of mucus. These conditions obtained also in the jejunum and ileum, decreasing from above downwards. The mucous membrane of the colon was somewhat reddened. The rectum was partially distended with solid feces, its mucous membrane being normal in appearance, or nearly so. The bladder was empty and contracted. The heart and large blood-vessels appeared healthy. The right auricle was distended with black, somewhat firmly clotted blood; the left auricle was empty. The right ventricle contained a small amount of blood, partially clotted. The left ventricle contained a small clot, a portion of it decolorized. The valves were normal. The larynx, trachea, bronchi, lungs, and pleuræ were healthy in appearance. The spinal cord was carefully examined, and no indications of disease or injury were discovered in either it or its membranes.

Dr. Hills returned the following report, which is presented in a somewhat condensed form: The stomach contained two and one-half ounces of a dark-brown, viscid liquid, which on standing deposited a reddish-brown sediment containing many dark-red bits of substance of varying size, which, on microscopic examination, proved to be little clots of blood. The color of the liquid was found to be due to admixed blood and bile. In addition to blood corpuscles the microscope showed epithelial cells from the glands of the stomach, a large amount of granular matter, and a considerable number of crystals of arsenious acid. In the course of four or five hours' searching perhaps three hundred were discovered.

The mucous membrane of the stomach was observed to be somewhat swollen and covered with a closely-adhering coat of mucus. It was for the most part pale in color, but reddened in patches particularly over the greater curvature, the redness assuming principally a clotted form. There were also a few small dark patches near the pylorus due to blood effused beneath the mucous membrane. Under the microscope the liver appeared to be in a state of extreme fatty degeneration, a fact obvious, indeed, to the unaided eye. There was quite extensive fatty degeneration of the kidneys, and also of the epithelium lining the peptic glands. One-fifth of the contents of the stomach and one-fifth each of the liver, kidneys, and spleen were tested together for the mineral poisons by appropriate methods. The result of this analysis was the discovery of arsenic. A portion of the liver (one-fifth) tested separately for arsenic failed to show its presence. The contents of the stomach, tested as in the first instance, revealed unmistakably the presence of arsenic. No separate examination was made of the spleen or kidneys. It was not thought advisable to make a quantitative analysis without a special order from the medical examiner or court. It was estimated, however, that there might be one-half grain of white arsenic in the contents of the stomach.

Case XVIII.—Poisoning by Arsenic.

§ 571. We are indebted to Prof. George F. Barker, Professor of Physiological Chemistry and Toxicology at Yale College, for the following testimony, which he gave in the case of Horatio N. Sherman, poisoned with arsenic by his wife. As the case has

never yet appeared in any work on toxicology, as Mrs. Sherman was convicted mainly by the evidence of the chemical and medical experts, and as we have a copy of the chemical evidence corrected by the professor himself, we make no apology for introducing verbatim in this work the evidence of the chemical expert as given in court.

After certifying to the identity of the stomach which had been committed to his hands by Dr. Pinney, Prof. Barker describes the chemical process by which he assured himself of the presence of arsenic in the viscera from Mr. Sherman's body.

The stomach was ligatured at both orifices; these ligatures I removed. I then opened the stomach by making an incision along the lesser curvature; I emptied the contents into a clean dish and proceeded to wash the inner surface of the stomach with distilled water, for the purpose of ascertaining its condition; I found it to be considerably congested throughout, there being patches showing distinct inflammation, about the cardiac orifice, and at the pylorus. The inner surface was nowhere softened or ulcerated. The contents, which were poured out, consisted of three or four ounces of a dark, offensive, slimy mucus, containing apparently bile. The second package in the box, on taking off the paper, was found to be a roll of new cloth, containing a portion of a human liver. This presented no unusual appearance, being perfectly healthy, with the exception of a small cyst on one side; after making notes of these results, I carried the materials from my laboratory to that of the medical college, and placed the liver in a new, clean glass jar, under double lock and key. I should here mention that in this package was a fragment of a large intestine, the mucous surface of which appeared slightly inflamed. The next day two-thirds of the stomach, and the contents of the stomach, were taken by me from the jar for the purpose of examination. At the conclusion of the examination of these organs a portion of the liver was likewise examined. The result of the examination was the finding of arsenic in both these organs. On the third of June I reported to the grand juror of Derby the presence of arsenic. Subsequently I examined the remaining third of the stomach and a weighed portion of the liver (six and two-third ounces) for the purpose of obtaining the quantity of arsenic present in that organ. The method of analysis followed in all these cases was as follows:—

The portion of the organ to be examined was finely divided, placed in twice its bulk of distilled water, strongly acidulated with pure hydrochloric acid, a few grammes of potassium chlorate were added from time to time, and the whole gently heated. In the course of eight or ten hours the mass was dissolved and a clear yellow liquid was obtained. This was filtered, heated till the odor of chlorine disappeared, and then a washed stream of hydrogen sulphide gas was passed through it for from twelve to sixteen hours. The liquid was again filtered, and the precipitate thus obtained collected upon a filter, washed till free from chlorine, dried on the water-bath, oxidized with pure nitric acid, and then heated with pure sulphuric acid, the heating being continued till the mass was completely charred. Water was then added and a little pure hydrochloric acid, and the whole was gently heated and filtered and the residue washed. Through the filtrate and washings hydrogen sulphide gas was again passed for twelve hours. The precipitate thus produced was filtered off and washed, and upon it was poured upon the filter some pure ammonium hydrate and sulphydrate, previously heated to boiling. The filtrate was received in a porcelain capsule, evaporated to dryness with a gentle heat, and the residue oxidized by repeated evaporations with pure nitric acid; pure sodium carbonate and nitrate were then added, the whole dried carefully and heated to complete fusion. After cooling, the fused mass was dissolved in cold water, the solution filtered, pure sulphuric acid was added, and it was evaporated till the fumes of the acid appeared. To it was then added a saturated solution of sulphurous acid, and it was again evaporated until fumes appeared. It was then diluted with water, and through it was passed washed hydrogen sulphide gas; a heavy yellow precipitate of arsenous sulphide was thus obtained. This was filtered off, washed, and dissolved in pure ammonium hydrate poured on the filter. The filtrate was received in a previously weighed porcelain capsule and evaporated to dryness on the water bath; the capsule was then reweighed, and the increase in weight showed the amount of arsenous sulphide which had been added; treated in this way, the six and two-third ounces of liver examined in the second case yielded six hundred and two thousandths of a grain of sulphide of arsenic, which quantity I now produce. (The professor exhibited a small vial containing the article.) This quantity corresponds, by calculation, to four hundred and eighty-five

thousandths of a grain of white arsenic, or nearly half a grain. Calling the weight of the liver four pounds, which appears to me to be a fair average—the quantity obtained in two separate and distinct examinations of the same liver being the same relatively to the size taken in the two cases—my opinion is, that this entire liver contained nearly five grains of white arsenic.

Prof. Barker resumed: The sulphide of arsenic produced in the bottle was from a weighed portion of the liver; another portion of sulphide of arsenic was obtained from an unweighed portion; this was oxidized with pure nitric acid by repeated evaporations; the excess of acid was then removed by evaporation with distilled water; the residue was dissolved in distilled water; a small portion was removed and tested with ammonio-silver nitrate; on the addition of this reagent, a brick-red precipitate of silver arsenate was produced. This was inclosed in a small tube, and is contained in the tube herewith produced. [A framed card was here shown, with this tube among others placed on it, with its contents, and marked "silver arsenate."] The rest of the solution was evaporated with a saturated solution of pure sulphurous acid in water, until all odor disappeared; two portions of the liquid thus obtained were removed; one of them, on being tested with ammonio-nitrate of silver, gave a canary-yellow precipitate of silver arsenite, which rapidly became dark olive-brown on exposure to light; this precipitate is here produced, marked "silver arsenite;" the second small portion taken was tested with ammonio-cupric sulphate; a light-green precipitate of copper arsenite, or Scheele's green, was thus produced; this is here marked "copper arsenite." The remaining portion of the liquid was divided into two equal parts. One of these (A) was evaporated nearly to dryness, mixed with sodium carbonate in excess, and thoroughly dried. The dried mass was then intermixed with a perfectly dried mixture, composed of one part of pure potassium cyanide and three parts of sodium carbonate. Several portions of this mixture were then heated in hard glass tubes, having a bulb blown on one end. Each of these gave a black, lustrous, mirror-like deposit of metallic arsenic. One of these tubes is here produced, marked "metallic arsenic by the reduction test." A second similar tube was prepared, sealed, and the portion containing the arsenic heated; the deposit was readily volatile and condensed in the cooler portions of the tube in a brilliant crystalline

sublimate, consisting of distinct octahedral crystals of arsenous oxide or white arsenic. This tube is here produced. [A magnifying glass was produced, and the crystals distinguished by the witness.] Other portions of the dry mass were then tested by heating them in a stream of carbonic gas. (Fresenius and Babo's test.) Each of the portions thus heated gave a bright mirror of metallic arsenic, similar to that obtained in the reduction test. One of these tubes is here produced. No other substance but arsenic gives this test. A second tube was heated, the deposit volatilized, and a ring of crystals of white arsenic obtained as before. The tube containing these crystals, with some of the metal not oxidized, is here produced. The other half (B) of the liquid was divided into two portions; one of these was tested by the method of Reinsch; *i. e.*, it was acidulated with hydrochloric acid, and in it strips of copper were boiled. These strips became covered with a steel-gray arsenical coating, which finally became black. One of these strips is here produced, marked "metallic arsenic on copper—Reinsch's test." Another copper strip was heated in a small tube, and afforded a sublimate of white crystals of arsenous oxide, distinctly octahedral by the microscope. These crystals I here produce, marked "arsenous oxide by Reinsch's test."

The second half of this portion was tested by the method of Marsh; it was added to a flask from which pure hydrogen was being evolved by the action of pure sulphuric acid upon pure zinc; the gas as evolved was freed from acid vapors and from moisture by passing it over solid potassium hydrate and calcium chloride; it was then passed through a long tube of hard glass drawn out at intervals. Before adding the solution the purity of the materials had been tested by heating this tube to redness, and allowing the gas to pass for an hour; no deposit was obtained in the tube. After adding a portion of liquid to be tested, a hair-brown deposit of metallic arsenic appeared in the narrow part of the tube just beyond the heated portion, deepening in color until it finally became black; two of these constricted portions of the tube containing the black deposit of metallic arsenic are here produced, marked "metallic arsenic, in tube, by Marsh's test." At the same time the flame of the burning hydrogen at the end of the tube became bluish-white, and deposited upon a piece of cold porcelain held in it brown

mirror-like spots of metallic arsenic; two of these pieces of porcelain containing the spots are here produced. The other deposits in the constricted portions of the tube were then tested; the deposit was found to be readily volatile and oxidizable, giving a ring of crystals of white arsenic; this tube is here produced, marked "arsenous oxide, Marsh's test." Other portions were submitted to the action of hydrogen sulphide gas passed through the tube. On gently heating it, the black deposit of metallic arsenic changed to the bright yellow of arsenous sulphide, or yellow arsenic. Two specimens of the deposits thus obtained are exhibited. The tubes contain a portion of the black metal, and beyond this a portion of the yellow produced from it. The same tests in the same order were applied to the portion of arsenous sulphide obtained from the contents of the stomach, and a series of products obtained in the same way are here exhibited at the top of the card, marked as before, in the same order on the card. During several of these tests, whenever the metallic arsenic vapor escaped into the air, the peculiar odor of garlic was observed. Besides arsenic, I obtained, both from the contents of the stomach and from the liver, sulphide of bismuth; that sulphide was converted into subnitrate, and a portion of it from each organ is here exhibited; the tubes are marked "bismuth subnitrate." All these tests throughout were made in duplicate. In order to ascertain whether the materials employed in this investigation were pure, I took, before the second examination of the portions of the body mentioned, a fragment of beef's liver, equal in size to the fragments of human liver previously taken, and examined it in the same way, using the same materials and the same apparatus, except where it was replaced by new articles. No trace of arsenic was obtained in this examination. My opinion, therefore, is, that the material and vessels employed were pure and clean. My opinion, also, is, that the yellow precipitate obtained from the weighed portion of the same human liver is also sulphide of arsenic.

To Col. Wooster.—Did not attempt to weigh the arsenic in the stomach as I did in the liver: arsenic, in chemical language, refers to a metal which is black and lustrous, and may be combined with oxygen or sulphur. When combined with oxygen, the form known as oxide is produced; it bears the same relation to arsenic that iron rust does to iron. [Here followed an explanation of the method

in which metallic arsenic is obtained, for the enlightenment of the jury.] It is a question whether metallic arsenic is poisonous. It is a general principle that no metal in itself can be poisonous. The sulphide of arsenic and white arsenic are both poisonous, though the white arsenic is more so than the yellow.

Q. Have you any means of telling what quantity of arsenic there was approximately in the stomach you examined?

A. The tissue of the stomach contained no arsenic; the contents of the stomach contained an amount which, though I did not weigh it, I estimated—[Objected to this as a mere guess. Objection overruled, the court holding that the professor ought to know.] It is fair to assume that comparison by bulk is correct. Judging from the bulk obtained from the stomach, the quantity present must have been about one-tenth of a grain.

Q. How long after arsenic is taken does it ordinarily find its way to the liver?

A. White arsenic taken into the stomach is removed therefrom by absorption, and as it has not been detected in either the lymph or the chyle, this absorption must take place by the blood. It has been detected in the urine within one hour after it has been taken. The liver acquires its maximum quantity, in the opinion of several authorities, in from fifteen to eighteen hours after administration.

Q. Does it pass off or remain in the liver?

A. It is eliminated from the liver, and may entirely disappear in from eight to fifteen days after being taken; depending upon the quantity and other circumstances.

Q. What is the maximum quantity the liver of an ordinary adult will contain?

A. I think that the maximum quantity the human liver may contain is the quantity I have found in the present case.

Q. From the presence of this quantity of arsenic, have you an opinion as to how long it must have taken before death?

A. At least fifteen hours; how much longer I cannot say.

Absorption and elimination of arsenic go on together; if there was vomiting and purging, a portion of the arsenic taken Monday night would have been removed before death on Friday. A considerable portion may have been eliminated, during the five days supposed, by the kidneys. Moreover, the arsenic is distributed through the entire body, and the analysis of a single organ or two

does not discover the arsenic elsewhere contained. For both these reasons, the amount obtained cannot have been the whole amount taken, and must be less.

Q. Is arsenic a cumulative poison?

A. The question of cumulation depends upon the definition of the word cumulative. If by cumulative is meant the accumulation of a substance in the body when it is taken more rapidly than it can be eliminated, then arsenic or apple-pie may be regarded as cumulative. If by cumulative be meant a substance which enters into combination directly with certain tissues, so as to form a compound less readily removed than the original substance taken, or not removed at all, then arsenic in my opinion is not a cumulative poison. I have heard the testimony of Dr. Beardsley in relation to the sickness of this man; the quantity of arsenic found in his stomach may have been, in my opinion, either the last traces of a very large quantity taken on Monday night, or the residue of a smaller quantity taken subsequently; which I cannot tell, but the latter appears to me the more probable. The first effect of arsenic when administered is local. It is specified as an irritant, but not a corrosive poison—that is, it inflames the parts it comes in contact with, without destroying, softening, or perforating them; the local effects thus first produced cause local symptoms; these appear generally in an hour after the poison has been swallowed. They are burning pain in the stomach which, as the poison passes down, extends along the intestinal tract. It increases in severity, is accompanied by great thirst, dryness and constriction of the throat, vomiting, and purging. The matters vomited are dark, bilious, and offensive. By this time more or less of the poison has become absorbed; it enters the blood and produces a second class of symptoms called remote, the action being apparently mainly upon the blood corpuscles; these symptoms are characterized by great prostration of strength, anxiety and depression of mind, a peculiar lividity of the face, a blue line under the eyes, the intellect being as yet unaffected, and the mind clear; death may ensue at this stage of the action, due to the prostration; usually taking place under these circumstances, in from one to eight days; if life be continued, the effect of the arsenic is apparent upon the nervous system and brain; stupor, passing into profound coma, may develop itself in case the action is primarily upon the brain; delirium and convulsions, passing into tonic spasms, in case the

spinal cord is also involved, may precede death; all of these symptoms may not be present in any one individual; the patient may recover from all these symptoms, which may be called primary, and may die from the secondary effects of the poison years afterwards; white arsenic, when taken into the stomach, is dissolved both in the stomach and intestines by the gastric and intestinal juices; spread out upon the posterior surface of these intestines, is a set of blood-vessels which compose what is known as the portal system; the arsenic thus dissolved passes into these bloodvessels through their walls; these vessels unite, thus forming the large trunk known as the portal vein, which empties itself directly into the liver, so that this organ is not only more fully supplied with blood than any other, but it is supplied with blood charged with arsenic from the intestines; hence the liver is more likely to contain this poison than any other organ. It passes from the liver into the general circulation, and thus produces its remote effects. Most probably it does its fatal work on the blood itself, disintegrating the blood-corpuscles, thus rendering the blood unfit to perform its functions. From two and a half to three grains of arsenic would kill an adult; less quantities than that have been fatal; and more has been taken without producing death. Arsenic would not probably be found if death ensued from the secondary effects. Persons have died from the primary effects of arsenic in eight days, and no trace of the poison has been found in the body on analysis. Constriction of the throat, when arsenic is taken, occurs from one-half hour to an hour ordinarily.

Q. Assuming that the liver you examined was Horatio N. Sherman's, of what do you think he died?

[Objected to, and admitted as the witness's opinion, based on his own analysis.]

A. My opinion is, that, considering the symptoms as described and the presence of arsenic as stated, Horatio N. Sherman died from the effects of a poisonous dose of arsenic.

I found in a locked drawer, upon my return from New York in July, a package, which I now produce; a white paper, tied with a red string, with the word "bismuth" written on it, which I found to contain sub-nitrate of bismuth upon opening it. Forty grains of it were examined for arsenic, with a negative result. The bismuth was pure.

The white crystals of white arsenic not appearing distinctly in

the tubes on the card, I have placed beneath them black paper to make them distinctly visible. I did not mention the effect of the poison on the action of the heart and lungs; the pulse during great prostration is generally small and rapid; sometimes, when the prostration is great, the pulse is less rapid than usual; the respiration is difficult, requiring almost a voluntary effort to breathe; it is sometimes quick and sometimes slow; the action of the poison on the lower intestines is to produce tenesmus, *i. e.*, straining without result; inflammatory action produces a dry and hot skin at the first stages; after collapse the skin is cold and clammy, and sometimes moist; the inflammation causes dryness and a folding up of the mucous membrane; there is also more or less faintness; sometimes entire syncope; purging may be wanting; convulsions are exceptional; the same is true of delirium.

Cross-examined by Mr. Watrous: I commenced the analysis on the 6th of November, 1871, and obtained the sulphide on the 23d of the same month; took the 6⅔ ounces from a portion of the liver left at my office; first received it on May 13th; from that time until November 6th it was in my laboratory hidden behind some other bottles, in a sealed glass jar, in a cupboard which was locked; the laboratory was locked; only myself and the janitor had keys; was not in the laboratory daily; have no recollection of any one being present while I was there; opened the bottle only once from May 13th to the 30th of June; it was not labelled; no one was present when I opened it; opened it in the daytime; gave the janitor instructions in regard to it; did not point it out to him; I pointed to the door, and said that that was not to be opened by him or any one else by his knowledge; told him I had an important investigation in progress, and it mustn't be interfered with; didn't tell him what I was looking for; was looking for poison—not for arsenic. I was told that arsenic was suspected; was not requested to examine it in order to see if arsenic was there; looked for all metallic poisons; could have discovered antimony as readily as I did arsenic; was not told there was a suspicion of anything but arsenic; did not start in my investigation with that in view. Never made any experiment with the view of finding arsenic.

[The witness here appealed to the court to make an explanation, in reply to questions asked him; and the court held that the

professor had a right to make any explanation to make his answers clear.]

Q. If you did not find arsenic either designedly or by accident, how did you find it?

A. I was looking for metallic poisons, and found arsenic. [The witness was going on to make an explanation, when Mr. Watrous objected, but finally withdrew his objection.]

The explanation: The examination is for metallic poisons, and not for one or two or three, but all. And if one is found, it is found without especially looking for it. If arsenic had been there alone, some other process than the one I followed might have been better. I looked at the jar occasionally to see that it had not been disturbed; don't know how many kinds of chemicals I mixed with the human liver during my analysis; think it was not fifty, nor twenty-five, nor fifteen; possibly ten; cannot tell exactly, without counting.

[Mr. Watrous asked witness to count them up.]

Witness: If I have counted correctly, I used ten chemicals in the analysis; no means of getting at the bulk of the chemicals used. The following is a list of chemicals I used in the analysis: Water, hydrochloric acid, potassium chlorate, hydrogen sulphide, nitric acid, sulphuric acid, ammonia, ammonium sulphydrate, sodium nitrate, sodium carbonate, and sulphurous acid. Tested the sulphuric acid and found it pure; sulphuric acid does sometimes contain arsenic; this is not true of sulphurous acid. I used half a fluidounce, about four hundred grains of sulphuric acid during the tests; know what I obtained was sulphide of arsenic.

It is not settled among chemists whether arsenic is a metal; it is not absolutely known that it is not itself a compound; the elements of it, if it is a compound, are of course not known; know that the process through which I put the liver would not produce arsenic; I have gone through the same process many times, and never generated arsenic; I know as well as I know anything, that arsenic was not generated in this case; I believe it as much as I believe I exist; I cannot prove that I exist; it is not claimed that chemists can manufacture diamonds or things that cannot be told from diamonds; have heard of the Wharton trial; have read the testimony of chemists claiming to be such, whose testimony differed as to the existence of antimony; the main difference was whether it was enough to cause death, not about the existence of it; arsenic

in bismuth is very uncommon now ; it is found sometimes with bismuth ; if bismuth in which arsenic was put had been placed in the stomach, I should have found them separately ; bismuth is not a poison ; it would not kill a man if taken into his stomach ; subnitrate of bismuth won't kill.

Case XIX.—Poisoning by Corrosive Sublimate.

(Annales d'Hygiène Publique, 1880, p. 387.)

§ 572. A young man, twenty years of age, having fulfilled his duty to his country on the day for drawing lots for conscription, went to a house of prostitution to indulge for the first time in sexual intercourse. A few minutes afterwards, his friends in joke persuaded him that he had contracted a "venereal disease which would go through the blood." Being overcome with syphilophobia, acting under the influence of profound ignorance, and wishing to check the disease at its beginning, he believed in the certainty and rapidity of a radical cure by swallowing at a draught a bottle of medicine which had been prescribed for one of his friends. The bottle contained either some "Van Swieten's Mixture" or an amount of solution of mercuric chloride requisite for a liter of that preparation. The amount swallowed was about 80 grams. The probabilities are that the young man swallowed about 0.800 gram of corrosive sublimate. Soon after the ingestion he was taken with severe pains, soon followed by salivation and vomiting, three phenomena, which clearly denote that the solution was concentrated and acted first as a caustic. Very soon afterwards could be observed eschars inside the lips, cheeks, on the soft palate, and throat. The odor of the saliva was very strong. In the pathogeny of the salivation, after 36 or 48 hours, two factors could be distinguished : the local condition from cauterization of the mouth, and the general condition of mercurial intoxication. The general condition was alarming ; the patient was pale and exhausted, deeply depressed by pain, insomnia, want of nourishment, and terror. On the following days there was stomatitis from the cauterization and excessive absorption of mercury. He refused everything but ice, which was the only thing which eased him. He was very agitated, and groaned continually. There was no fever, even on the seventh or eighth day, showing that the depressing influence of the mercury was the dominant factor. He

continued to be salivated and to vomit; the expectorations were tinged with blood, and contained shreds of mucous membrane altered and blackish. The vomiting commenced a few minutes after ingesting the poison, the diarrhœic stools, which contained bile and blood, appeared on the following days. All the symptoms persisted. There was nothing, however, during the first few days to indicate that the patient was in any immediate danger. The temperature was 37.2° C. (99° F.). It was on the sixth day that the patient began to fail; there were no particular appearances, but the temperature fell to 36.6° C. (97.8° F.) with no symptoms of perforation, or peritonitis, or of internal hemorrhage. The principal cause of the fall in temperature was the depressing influence of mercurial intoxication. The patient succumbed on the ninth day; all the symptoms had gradually become intensified. There was never any delirium; sensation and motility were unimpaired. There was no anuria, but there had been albuminuria since the fifth day. There was general lowering of temperature reaching 35.2° C. (95.3° F.) at the time of death. Such were the symptoms and course of this case, which was characterized at the autopsy by the remarkable state of fluidity of the blood, the absence of lesion in the structure of the liver, the quick congestion and granular fatty degeneration of the kidneys, whose tubules were filled with fat, and the corrosion, the destruction in places, and strong injection of the mucous membranes of the œsophagus, stomach, and intestines. The injury to the mucous membranes was in accord with the local irritation due to the caustic nature of the poison.

Death occurred without a struggle in the midst of a general collapse and great enfeeblement of the circulation; it occurred with an abruptness which suggested internal hemorrhage.

Case XX.—Poisoning by Acetate of Lead.

(Maschka's Handbuch der gerichtl. Med., vol. ii. p. 281.)

§ 573. In 1868, a man sixty-nine years of age, had married a woman of twenty-five, and on March 24th of the same year he transferred his property to her. A few days afterwards he began to feel ill, and on March 31st, by the advice of his wife, a surgeon was called in. The latter noted yellowness of the conjunctivæ, loss of appetite, eructations, mucous râles, weakness, and vertigo;

dejections normal; thirst not increased; pulse 80–90; tongue coated. On April 2d, he was somewhat better; the vertigo and weakness were diminished. In the evening there occurred vomiting, rapid dissolution, small pulse, cold extremities, gurgling respiration, and death. There were no pains, cramps, or retraction of the abdomen.

Twenty days after burial the body was exhumed on account of suspicions of poisoning by acetate of lead, of which a considerable amount was in the house. The body was undergoing dry decomposition; skin easily detached; abdomen green. Suggillations on the back, breast, arms, and thighs. Eyes sunken. Mucous membrane of the mouth pale. Brain pulpy and soft; in the lateral ventricles a small amount of fluid. Membranes opaque; bloodvessels and sinuses empty. Lungs engorged. Pericardium contained some bloody serum. Heart normal. Mucous membrane of the œsophagus discolored and covered with mucus. Stomach entire; mucous membrane somewhat loose, but otherwise normal; no trace of inflammation, ecchymosis, or erosion. The stomach contained a greenish fluid with some food. The intestinal canal was dirty brownish-red in color; mucous membrane normal; intestinal contents were greenish-red fluid. Liver pale, firm, somewhat smaller than normal; gall-bladder contained a little bile. Spleen friable and of normal size. Kidneys normal. Bladder empty.

The stomach and contents, and the intestine and contents were examined chemically. The former amounted to 567.3 grams; the latter 11.68. Reckoning the whole amount of poison by that found in an aliquot part of each, it was found to be 0.870 grm. metallic lead, corresponding to 1.60 of the acetate.

Case XXI.—Fatal Poisoning by Chromate of Lead.

(Vierteljahrsschrift für gericht. Med. und öffentl. Sanitätwesen, Bd. xxi. p. 60.)

§ 574. On March 14, 187–, between nine and eleven o'clock in the forenoon two young boys, aged respectively one and three-quarters and three and a half years, ate a number of small yellow objects, which had been used for decorating a cake formed like a bee-hive, and which represented the bodies of bees. In the afternoon between two and three o'clock both were seized with violent vomiting and great prostration. The vomitus was at first yellow in color; it was unfortunately thrown away. The vomiting continued until eleven

o'clock, but during the last three hours was not so frequent. At six in the evening, when the physician arrived, the two were in bed and presented a very sick appearance. Their faces were much reddened; they complained of much thirst, and were very restless. There was no diarrhœa and no pain. It was not unreasonable to ascribe these symptoms to poisoning, for the children had had nothing but bread and milk for breakfast, and meat-soup, beef, and potatoes for dinner, of which the other members of the family had eaten and had remained well. The poison was attributed to the above mentioned "bees," of which they had been given seven, of which number but one remained. They admitted that they had eaten the other six.

Analysis of the "bees" conducted by an apothecary showed them to be composed of gum tragacanth and chromate of lead.

The treatment consisted at first of the administration of calcined magnesia in mixture. On the following day (March 15th), the faces of both were red and hot; they were listless, and on being questioned showed signs of discomfort, but made no complaint of local pain; the sensorium was apparently affected. Carl, the younger, had some diarrhœa, and towards noon convulsions, during which the face was livid. The convulsions were more frequent towards evening. Ice was applied to the region of the stomach and given in small amounts internally.

On the 16th, at nine o'clock in the morning, the younger child died. The elder had constant redness and heat in the face, was listless and almost stupefied. The skin of the breast and abdomen was markedly erythematous. The evening temperature in the axilla was 39.5° C. Ice and bicarbonate of sodium solution were given internally; ice poultice applied to the gastric region.

On the 17th, the pulse was irregular and intermittent; there was Cheyne-Stokes respiration. Evening temperature 39.6° C. Treatment; ice as before, and subcutaneous injection of quinine. The latter was given because, in spite of great thirst, deglutition was extremely difficult.

On the 18th, the erythema was still present; bad smell from the mouth; stupor; difficult deglutition. Evening temperature 39.2° C. Treatment as on preceding day.

On the 19th, collapse; very foul odor from the mouth; deglutition almost impossible; sensorium much affected. Death occurred at eleven o'clock in the forenoon, five days after ingesting the poison.

Autopsies were performed in both cases. Among the *post-mortem* appearances observed on examination of the body of the younger child thirty-nine hours after death, the following may be noticed.

The bloodvessels on the surface of the brain were strongly injected; the longitudinal sinus empty; bloodvessels of the base of the skull overfilled. On section of the brain tissue single bloody points were seen.

The lungs were congested; on the surface were some small points of emphysema. The heart contained much blood, fluid and coagulated. The intestinal canal was pale, and only occasionally marked by single injected bloodvessels. The liver was bright colored with pale and irregular spots exteriorly and interiorly; on microscopic examination a marked fat formation was seen, especially at the above-mentioned spots (fatty degeneration). The stomach contained a small amount of yellowish-green fluid. Its inner surface showed velvet-like opaque swelling of the mucous membrane, especially marked on the cardiac half. The mucous membrane was marked throughout with red points, which in some places were grouped; in the vicinity of the cardiac orifice it was pale yellow, and the color could not be wiped off. The duodenal mucous membrane was pale and in folds; occasional bloody points were seen. The spleen was large and of normal color. The kidneys were normal. The mucous membrane of the bladder was pale; the bladder was empty.

The *autopsy* of the second child was performed 27 hours after death. The skin of the face was yellowish in color. The bloodvessels of the surface of the brain were very full; the longitudinal sinus contained but little blood; the bloodvessels of the base of the skull were full. The brain substance contained much blood. The dura mater was very adherent.

The lungs were pale red; bloodvessels of the heart very full. The œsophagus was much injected and brownish-red; its mucous membrane was disintegrated and purulent throughout, the destruction being most marked at the upper end. The mucous membrane of the larynx and upper portion of the trachea was purulent and partially disintegrated; the tissues beneath the mucous membrane were deeply reddened. In the right tonsil, which contained much blood, was a pus cavity. The walls of the stomach were somewhat thickened; the mucous membrane at the greater curvature was in overlying folds and colored dirty pale red; at the pyloric end the

color was brownish-red with darker spots. The mucous membrane was loosened and easily rubbed off. On careful examination numerous small blood-points could be seen under the mucous membrane; these were grouped at various points, and caused the redness. The mucous membrane was already disintegrated in several places.

The mucous membrane of the duodenum was ulcerated, loose, and easily rubbed off. At the upper part it was strongly injected, and at one place there was a perforation as large as a lentil; at several points the wall was almost perforated.

The liver was fatty degenerated. The spleen was eleven centimeters long; the exterior and the cut surface were light brown; the consistence was soft. Both kidneys were very rich in blood and on section showed small pus drops, which apparently came from the calices. The bladder was very full; its mucous membrane strongly injected.

The chemical analysis of the organs gave in both cases perfectly negative results. Analysis of the bees showed them to consist of 0.270 grm. of gum tragacanth and 0.0042 grm. of chromate of lead. As has been said before, the number of bees ingested was probably six, and allowing to each an equal share of the same, the amount of the poison ingested by each was 0.0126 grm.

Case XXII.—Fatal Poisoning by Inhalation of Dust from Yarn colored by Chromate of Lead.

(Vierteljahrsschrift für gerichtl. Med. und öffentl. Sanitätswesen, Bd. xvii. p. 29.)

§ 575. A few days before Jan. 1, 1876, a weaver, B. in E., bought a lot of yarn colored by chromate of lead for manufacturing blankets. Some of the yarn was wound on bobbins before New Year, and on the 2d day of January, B. and his journeyman G. began to work with the same at two looms, while Frau B. and the bobbin-girl L. continued the winding. They worked from between seven and eight o'clock in the morning until after ten at night. During the work the yarn gave off so much dust that, according to B.'s declaration, their faces and hair were quite yellow, and they had a bitter taste in the mouth. Their expectorations were colored yellow. Frau B. called the taste "sweetly bitter." The journeyman became sick about eight days after beginning the work. He was seized with

headache, ringing in the ears, pains in the chest and stomach, loss of appetite, inclination to vomit, and constipation. He was ill six weeks when he was obliged to give up work and consult a physician. Meanwhile Frau B. was seized with pains in the chest and loss of appetite, but these symptoms disappeared after continued drinking of warm milk.

In the third week after beginning work, B. was taken with pains in the spine, nausea, proneness to vomit, weakness and loss of sleep. Eight or ten days afterwards he was obliged to cease work and call in a physician on account of severe cramps in the abdomen and constipation. The bobbin-girl also was sick with diarrhœa, loss of appetite, and pains in the chest.

The symptoms of B. and of his journeyman were the same; yellow coated tongue, yellow sputa, complete loss of appetite, nausea, occasional vomiting, pain in the abdomen, especially in the umbilical region, general weakness, and obstinate constipation. The feces were colored yellow. The physician gave as his opinion that these symptoms were caused by inhalation of the dust, and were characteristic of chronic lead poisoning. A weaver in another town, who had used the same kind of yarn at the same time, suffered from similar symptoms and became unfit for work. The above cases improved gradually and the patients recovered; but B.'s nine weeks' old boy was taken sick at the same time, and died in consequence of inhaling and swallowing the dust. This child was at the beginning of the work about fourteen days old. On noticing the dust given off from the yarn the parents sought to protect the child from the same by covering its face with a white woollen cloth, which, however, did not in any way hinder respiration. This cloth and the child's bed were said to have always looked yellowish. The basket in which the child lay stood by day between the two looms, which were about six yards apart. At night the child was taken up stairs into into its mother's bed. The child been from birth active and healthy, and, in spite of the fact that it could not be suckled, had thriven well on suitable food, so that there was no ground for ascribing the *post-mortem* appearances of the stomach to ill-nourishment, etc. About seven weeks from the commencement of the spinning, the child suddenly became very sick. It had at first pallor of the face, and in a few days elevated temperature over the whole body. It had several yellow diarrhœic discharges daily with restlessness and

frequent screaming, during which it dug its hands under the pillows. With these screaming fits appeared reddening of the breast and abdomen. At first it would drink, but refused food; later on it drank with some effort, and on the day of its death swallowed with difficulty. The lips were dry, respiration quickened, and death came slowly. Neither domestic remedies nor medicine were given the child, partially on account of the popular belief among the laity that not much should be done for small children, but more particularly because the parents had considered the child to be perfectly protected by the cloth from any injurious effects of the dust. Death occurred on Feb. 24, after from six to eight days of illness. It is remarkable that the child during six weeks' exposure to the dust remained well, and only six or eight days before death was taken suddenly ill, and grew worse just when no work was going on. The cloth was kept over the child up to the time of death, and it is probable that the cloth became gradually impregnated with dust, which on sifting through was inhaled by the child until death. A proof of this is that on chemical examination after the autopsy the dust was found in the hair and in the jacket of the child, and, moreover, 0.036 grm. of chromate of lead was found in the respiratory tract and œsophagus. The rubber nipple which the child had used was found free from chromate of lead, most probably because the dust which had adhered to it was sucked off and swallowed. The yarn on analysis yielded 11.83 per cent. of chromate of lead.

Among the *post-mortem* appearances the following may be noticed: The hands were clenched convulsively. The omentum was finely injected; between the fundus of the stomach and the spleen was about a half a coffeespoonful of chyme; in the fundus a hole about the size of a large pea. The stomach contained chyme similar to that mentioned above. The stomach had a jelly-like character, especially marked about the perforation. The mucous membrane of the right half was much corrugated (rugæ) and had a pale red color; the left half was everywhere softened and smooth, and its color was in some places bluish and in others pale coppery. The bloodvessels of the upper curvature were somewhat filled with blood; those of the greater curvature more so. The duodenum contained a light yellow, thin mucus, the jejunum a pale yellow and watery mucus, and the ileum a light yellow, thick mucus. The jejunum was pale in color; the ileum marked

by very fine vessels; the mesentery of the former was pale, that of the latter injected, and its glands were much developed and hard. The intestinal mucous membrane showed neither marked reddening nor ulceration. The outer surface of the large intestine was injected; the inner was pale. It contained at the end of the rectum a little yellowish mucus. The liver was normal; the gall-bladder contained a teaspoonful of orange-colored bile. Both kidneys, especially the right, contained much blood. The lungs were healthy, and contained much black, fluid blood. The right auricle of the heart, its left half, and the pulmonary arteries, contained black, fluid blood. The brain could not be examined on account of advanced decomposition. The blood in the same was not increased in amount, and was black and fluid.

The chemical analysis of the organs and fluids was negative in its results, excepting in the air passages and œsophagus, where, as has been already mentioned, 0.036 grm. of chromate of lead were found.

The death of the child was caused by exhaustion following perforation and softening of the stomach brought on in consequence of swallowing and inhaling chromate of lead.

Case XXIII.—Poisoning by Sulphate of Copper—Hæmoglobinuria—Death—Autopsy.

(New York Medical Record, May 27, 1882.)

§ 576. A well-nourished woman, aged forty-six, took about one ounce of sulphate of copper dissolved in tea, at 9 P. M., December 13th, with suicidal intent. As soon as she had swallowed it, she felt a burning sensation in her stomach and all over her body, and becoming alarmed she told a neighbor, who summoned a policeman. He took her at once to a station-house. On the way she vomited three or four times, and before reaching it began to feel very weak and to suffer from severe pain and cramps in her stomach. At the station-house she was given stimulants, and, on the arrival of the ambulance-surgeon, large draughts of warm water and flour. This produced free emesis. At 11 P. M., two hours after taking the poison, she was taken to Bellevue Hospital.

On admission she was very weak and unable to stand or to walk. Her surface was cool and dry; pulse, full, strong, 112 per minute;

respiration natural, 24 per minute. She complained of faintness, and of cramps in her stomach and legs. She was put to bed at once, and the tube of the stomach-pump having been introduced the stomach was washed out with warm water. It was noticed that the material first obtained from the stomach was grayish-green in color. This was afterward found to contain copper. After a few minutes yellow ferrocyanide of potassium was obtained, and twenty grains added to one pint of warm water. This was thrown into the stomach, and on being drawn out, the water was seen to have assumed a brown color, indicating that the chemical change to ferrocyanide of copper had taken place. This injection was therefore continued, the amount of the potash salt being decreased as the brown color became less marked. When the water returned perfectly clear and colorless, the process was stopped. During this time (about forty minutes) the patient had complained constantly of pain in her stomach and of cramps in her legs; and had had several fluid evacuations. Before the tube was withdrawn three ounces of a mixture of castor-oil and olive-oil were thrown into the stomach. A portion of this was rejected. Patient was then given half an ounce of whiskey hypodermically, as the pulse had become more weak, small, and rapid—128 per minute. She was ordered twenty grains bismuth in milk, four ounces every two hours.

December 14th.—Patient vomited several times during the night, and had three loose brown movements not containing blood. She complained much of abdominal pain and of cramps in her legs. This morning she has less pain, but is very tender over the epigastrium. The cramps occur at longer intervals. She complains of frontal headache and feels very weak. There is no febrile movement, and her pulse is regular, strong, 100 per minute; respiration normal; skin cool and dry. Her mental condition is good; no affection of the senses; the pupils are normal and react to light. Ordered poultices to abdomen; a mixture of bismuth in mucilage; and small amounts of milk frequently repeated. During the day the patient was quite comfortable, did not vomit, had less pain, but had several diarrhœal movements attended with tenesmus. Her urine was diminished in quantity, but, as it was passed with the movements, could not be measured. Its color was normal.

December 15th.—Patient had a quiet night and slept. This morning she had some pain and tenderness in the epigastrium, but

has not suffered from cramps since yesterday afternoon. She has not vomited and to-day takes her milk readily. She had during the night, and continues to have to-day, occasional painful fluid movements, small in amount, brown in color, containing gray masses of feces, but no blood. Her headache continues. At noon to-day she complained of pain in the hypogastrium, and began to pass small amounts of urine frequently. Micturition was attended with burning pains in the urethra. The urine was found to be very dark, reddish-black in color, almost like ink. It is turbid, does not transmit light, has a specific gravity 1.014, is acid, and contains a large amount of albumen. On boiling, the coagulum was lighter than ordinary albumen, and floated to the top of the specimen. The substratum of urine was changed to a lighter color. On the addition of acetic acid, the albumen was partly dissolved, and the substratum of liquid regained its original color. Heller's test also demonstrated the presence of albumen. Microscopic examination of the thick sediment, which collected on standing, showed the presence of a large amount of fine granular matter stained brown. This was in irregular masses, and also in the shape of casts. There were a few epithelial casts, and some epithelial cells from the kidney. Careful examination failed to detect the presence of any blood-corpuscles. A portion of the granular matter was dried upon an object glass, common salt was rubbed into it, a hair laid across, a cover-glass applied, and a drop of glacial acetic acid allowed to enter beneath the cover. The slide was then warmed and subjected to examination under the microscope with power of 600 diameters. Crystals of hæmatin were to be seen, though not in great numbers. A specimen was then examined with the spectroscope, when the two absorption bands between D and E, characteristic of oxyhæmoglobine, were distinctly brought out. These tests were sufficient to establish the fact that the patient had developed hæmoglobinuria. Microscopic examination of the blood showed a slight relative increase of white corpuscles. The red corpuscles were not decolorized, and no microcytes were visible. The same treatment was continued with the addition of small doses of opium to allay the abdominal pain and control the action of the bowels.

December 16th.—This morning the patient is found to be moderately jaundiced. She passed a quiet night; had no movements, and did not vomit. She made about forty-five ounces of urine

during the past twenty-four hours; appearance and contents the same as yesterday. To-day the abdominal tenderness continues, but is less marked over the hepatic region than elsewhere. There is no fever, but the pulse is more rapid, 120 per minute, and is now quite small. Respiration is also increased to 30 per minute. Her general condition is better than yesterday, on account of the cessation of all gastric symptoms and of the diarrhœa. She takes her milk with relish. She is perfectly conscious and rational, but is more quiet, and seems inclined to sleep. The only complaint is that of painful micturition. When questioned she says that she has slight headache. Opium stopped.

December 17th.—Patient had a restless night and refused nourishment. This morning she seems to be weaker, and is rather somnolent. She complains of frontal headache, and of pain in the lumbar regions, but says that the abdominal pain has ceased. The abdomen is relaxed, not tympanitic, but pressure develops tenderness all over it. She has not vomited, and has had no movement during the past twenty-four hours. She retains her milk, but has no desire for food, her tongue being still thickly coated and gray. Her throat is not sore. Eyesight and hearing are perfect, and sensation good. Her pulse is feeble, small and rapid, 120 per minute, respiration 30, and for the first time since admission there is some fever, temperature being 100½°. There is a slight increase in the jaundice, the color of the skin being a peculiar grayish yellow. She passed fifty ounces of urine during the past twenty-four hours. It presents the same chemical characteristics, and is almost like black ink in color. Her motions indicate weakness, but there is no paralysis.

Toward evening it was evident that the patient was failing rapidly. Her mental condition was stupid, so that she could not be induced to make various motions that were desired to test coördination. There was evidently general paresis, as she could no longer turn herself in bed or hold a cup. Sensation was not impaired. At four P. M., temperature 101¼°; pulse very small but regular, 128; respiration, 34. Stimulation was given by the rectum, but was rejected, and the discharge was followed by a movement containing semi-solid black feces. She is now unable to swallow. At 9 P. M. the patient had sunk into a semi-comatose condition, and, when aroused, seemed unable to make any voluntary motions.

Sensation was decidedly impaired, or else she was too stupid to notice irritation. Pupils were contracted and no longer reacted to light. Nutritive enemata were not retained, and the urine was passed unconsciously. Temperature 98¾°, pulse 140, respiration 40. From this time she lay in a state of coma, and at two A. M., December 18th, died.

Autopsy twelve hours after death.—Body.—Rigor mortis extreme. Whole surface uniformly jaundiced. No ecchymoses. Brain.—The membranes were tinted a grayish-yellow color. A considerable amount of clear serum was present beneath the pia mater. No meningitis. No marked congestion. Surface of the brain was a pale gray color, evidently tinted like the membranes. Brain was wet, but there was no excess of fluid in the ventricles. Substance normal. Cord not examined. Thorax.—On opening the body it was noticed that the muscles, connective tissue, fat, and all the organs presented a grayish-yellow appearance. All the blood in the body was firmly clotted in the veins. The clots were of a light brownish-red color, resembling chocolate, and very consistent, so that they retained their form on being taken out of the veins. There was no fluid blood in the body.

Pericardium.—No adhesions, no excess of fluid. Heart normal size. Both ventricles were filled with dark clots. Heart-muscle flabby, and on section fatty. A mottling was noticed beneath the endocardium, most marked on the papillary muscles. Microscopic examination showed well-marked fatty degeneration of the muscular tissue. Aorta atheromatous. Pleuræ.—Old adhesions on both sides. No fluid in the cavities. Lungs.—Slight yellowish-brown coloration of the surface of the lungs. No subserous ecchymoses. Both lungs extremely œdematous. The fluid squeezed from them was yellow in color, substance of lungs normal. Abdomen contained no fluid. Spleen normal in size and consistence. Stomach contained a large quantity of semi-solid material, greenish-white in color, apparently half-digested milk. This was tested and contained no copper. There was but slight evidence of gastritis, consisting only in mild congestion. No ecchymoses and no ulceration.

Œsophagus.—Pseudo-membranous patches of gray color, and ulcerations near the stomach. Intestines.—Duodenum empty. It was deeply stained with bile. The ductus communis choledochus was pervious. Jejunum in its upper three-quarters was free from

inflammation. It contained green semi-solid masses. Ileum contained thick hard masses, green in color. These masses did not give any reaction on the addition of aqua ammonia. For sixteen inches along the lower part of the ileum there was extensive ulceration, with the formation of sloughs and loss of substance on the mucous coat. The ulceration was unevenly distributed through the ileum, and not confined to the region of Peyer's patches. It extended upward into the jejunum, but was less marked than below. The large intestine contained large masses of pasty dark-green feces, which distended it greatly. When these were washed out, numerous losses of substance were seen on the surface of the mucous membrane. There were no distinct ulcers.

Liver.—Uniform brownish-yellow color, soft in consistence, normal size, somewhat fatty. Microscopic examination showed extensive fatty degeneration of the hepatic cells. The fat existed in large globules and not in small molecules. Gall-bladder was moderately distended with very thick black bile; this bile was found to contain copper. The bladder contained a small gall-stone: gall-duct and bile-ducts not obstructed. Pancreas normal. Kidneys slightly enlarged; capsule adherent; surface mottled with very dark brownish-red and gray colors, giving a marbled appearance; substance soft. Cut surface nearly uniform dark brownish-red, and little distinction to be noticed between the cortex and medulla. Cortex was swollen, and striated appearance not present. The mucous membrane of the pelvis was gray in color, was coated with a thin layer of mucus and studded with minute ulcers, round in shape. Microscopic examination showed the tubules of the kidneys to be filled with granular matter stained red, and similar to that found in the urine. The cells lining the tubules were swollen; the Malpighian tufts were compressed and a free space existed between the tuft and its capsule, such as is said to appear when a large amount of albumen has been excreted. The capsule of the tuft was moderately thickened; the capillary walls were thickened; no granular matter in the vessels. Ureters normal. Bladder contained some dark-red urine. The mucous membrane was uniformly stained a pink color; no cystitis.

Uterus.—At each cornu, small intramural fibromata. The Fallopian tube on the right side was distended with dark-red fluid; its

fimbriated extremity obliterated; on the left side there was a small cyst in the broad ligament.

The medico-legal importance of the case warrants its publication. The number of cases of sulphate of copper poisoning reported is small, and in but a few is there a record of an autopsy. In no reported case is there any mention of the occurrence of hæmoglobinuria.

Case XXIV.—Poisoning by Sulphate of Copper.

(Maschka's Handbuch der gerichtl. Med., vol. ii. p. 291.)

§ 577. Franz H., sixteen years of age, took on October 11, 1870, an unknown amount of powdered sulphate of copper mixed to a paste with water. A half an hour afterwards he was carried to the hospital with severe vomiting and great weakness, thirst, contraction in the throat, choking, and pain in the epigastrium. The latter was sensitive on pressure. The face was pale, the lips blue at the angles of the mouth, the tongue coated and bluish. The temperature of the skin was diminished, the extremities cold, nails cyanotic, pulse small and increased in frequency. The vomitus was blue, and contained particles of the poison. He had several loose, greenish-yellow stools containing no blood. Urine scanty; blood and albumen absent. During the night he was very restless; on the next day complained of very severe headache, pain in the epigastrium, and burning in the mouth and œsophagus. There was no more vomiting. Urine was scanty, and contained albumen, blood, and bile pigment. The patient went home, but was again brought to the hospital on October 15th. He had marked icterus, pallor of the mucous membranes, diminished temperature, frequent pulse, great weakness, cardiac oppression, restlessness; the stools were pappy, brownish-red, and streaked with blood; tenesmus, very bloody urine. The region of the stomach and liver sensitive; liver enlarged; abdomen retracted. The jaundice decreased somewhat up to the 18th. There was then apathy, cold sweat, collapse, and death.

The *autopsy* was performed two days after. The skin was pale with a tinge of yellow. Rigor mortis in the lower extremities. There was not much blood in the sinuses, or in the brain and its membranes. The veins of the neck contained but a few drops of fluid blood. The mucous membrane of the trachea and of the œso-

phagus was pale. The left lung was pale; œdematous in the upper lobe; the lower lobe was swollen, hard, and friable, and contained a quantity of foamy fluid. The right lung contained the same kind of fluid. The heart and great vessels contained almost no blood. The heart substance was pale, soft, and easily torn. Stomach and intestines distended; the serous coat of the same slate-gray in color. The spleen was normal. The liver was of normal size; its substance yellowish-brown in color, soft, easily torn, fatty, and moderately full of blood. The gall-bladder contained a few drops of dark tenacious bile. Kidneys swollen, the cortex yellowish, the pyramids pale brown. The bladder contained some urine; mucous membrane normal. The coats of the stomach were firm, the coronary vessels only moderately injected. The stomach contained a kilogram of brown, feebly acid liquid. The mucous membrane was swollen, thickened, and covered with tenacious mucus; along the greater curvature it was greenish in color. In the fundus was an eschar as large as a cent piece, where the mucous membrane was dirty brown, villous, and easily detached. On detaching it, the muscular coat was exposed. Otherwise there was nothing abnormal about the mucous membrane. The mucous membrane of the small intestine was pale and not abnormal. That of the large was swollen and pale gray. The large intestine contained some thin slimy feces.

Case XXV.—Case of Suicide by Antimony.

(Boston Medical and Surgical Journal, December 18, 1856.)

§ 578. Miss —, 21 years of age, of a very nervous temperament, was a believer in spiritualism, and a trance speaker. While in one of these trances, some time since, she predicted that she should die during the month of October, and was, in that month, during several days, very ill. She, however, recovered, but again predicted her death at a certain time. Late in the specified day, November 25th, she said, while in a trance, "soon all will be over," and then drew upon paper a "casket," which her believing friends supposed was intended to represent her coffin. At six o'clock in the evening, while at the tea-table, she complained of pain in the lower part of the left side, and soon retired to her room, where she was immediately attacked with vomiting, followed after several hours by purg-

ing, both of which continued, accompanied by burning in the mouth, dryness of the throat, and great thirst. At one o'clock that night she died, apparently of exhaustion, without convulsions or any cerebral symptoms. The autopsy was made thirty-nine hours after death by Dr. Calvin Ellis with the assistance of Dr. Z. B. Adams. There was no decomposition of the body, which was quite rigid. Considerable bluish discoloration about the back of the neck and the hands. The brain was not examined. The other organs were normal, with the exception of the lower part of the small intestine, where the solitary follicles and Peyer's patches were considerably elevated, and of a pale yellowish color. The stomach contained twenty-six ounces of a gruel-like, acid fluid, the reddish color of which was possibly owing to the admixture of blood during its removal. This fluid was examined by Dr. J. Bacon, who reported that "the stomach contained a considerable quantity of antimony. The chemical reactions indicated that tartar emetic was the preparation of antimony taken. No other poison was detected."

The account of the symptoms, being obtained from the friends of the deceased, is necessarily defective; still it agrees very well with that given of similar cases by good authorities. None of the marked appearances of inflammation described by various authors were noticed. The development of the glands of the small intestine was similar to that seen in various affections attended by active catharsis. Dr. Cabot, at the meeting of the Society for Medical Observation, before which the case was read, spoke of having seen similar appearances, in Paris, in children, who had died while under the influence of antimony.

With regard to the spiritual condition of the patient, when she predicted her own death, and at the time of taking the poison, comment is unnecessary.

Case XXVI.—Poisoning by Tartar Emetic.

(The Lancet, Jan. 21, 1854.)

§ 579. E. S. S., aged sixteen years, complained of feeling bilious, and was advised by a nurse to take a dose of tartar emetic; one pennyworth was therefore procured at a chemist's near her residence on Sunday, Nov. 21st, and two-thirds taken the same evening at six. Within a quarter of an hour after, vomiting came on, very

sharp; and a little while after, smart purging. These symptoms continued for about three hours. The girl complained of pain down the œsophagus, and described it as "burning her." She then fell asleep. The matter vomited was described as being very dark. On the Monday morning she had some tea, and did not appear to be so ill as to attract attention. Whilst her mother and father were out, on the same afternoon (about four), a neighbor was called in, as she said "she felt as if she was dying." Medical aid was summoned, brandy and water ordered, beef-tea clysters exhibited, and everything tried to rally her. Her pulse was thin and cord-like. She kept continually throwing her head back, and screaming. Skin warm and moist; pupils dilated; knees drawn up. She lingered till Tuesday morning, when death closed the scene. During the six or eight hours previous to her death she was quite delirious.

Post-mortem examination thirty-six hours after death.—The features were placid; the throat appeared swollen; there was also green discoloration in each iliac region, but predominant in the right. The lungs were slightly congested; the heart healthy and contained about six drachms of fluid blood; the left ventricle was so contracted as almost to close its cavity; liver pale; spleen healthy; gall-bladder half full of very thick green bile; kidneys healthy but congested; the stomach was removed for further examination; the duodenum, jejunum, and ileum were smeared with a thick grumous fluid and quantities of mucus; there were no traces of inflammation; the bladder was half full of urine; the hymen was quite perfect; uterus healthy, but right ovary contained four cysts, the largest able to contain a large horse-bean, the smallest a pea. They were filled with a glairy, straw-colored fluid; the ovary was twice its natural size, and had several coagula of blood in its substance; besides being generally congested, the Fallopian tubes and morsus diaboli were also much congested. The stomach contained about sixteen ounces of thick grumous fluid; there was a large patch of greenish discoloration on the posterior part of the great curve of the stomach, near the œsophageal opening, penetrating to the peritoneal covering, at which part the structure was softened, and blood effused under the mucous coat, as likewise in some eight or ten places near the great curve, but very slight traces of the poison were obtained by the usual and appropriate tests.

At the inquest, the nurse said she told the parents of the girl to

get a dose of tartar emetic, but she obtained a pennyworth, and the quantity served was stated to have been ninety grains. The residue of the powder when weighed was twenty-five grains, so that sixty, or at least forty, were swallowed. The smallest dose on record producing death is fifteen grains (1854). Most of the poison had passed off in vomiting and by stool. Verdict, "accidental death," with caution to the nurse and parents, etc.

Case XXVII.—Poisoning by Sulphate of Zinc.

(Gazette Medica di Lombardia, 1848; and Brit. and For. Med.-Chir. Review, April, 1849.)

§ 580. A strong woman, aged 25, took by mistake for Epsom salts, a solution of an ounce and a half of sulphate of zinc. She instantly vomited, and then became affected with almost incessant retching and purging for half an hour, which continued afterwards, at short intervals, for three hours, and then gradually diminished. The pulse was frequent and small, and extreme prostration existed, accompanied with distressing restlessness and anxiety; the temperature of the skin was diminished; great pain in the abdomen, limbs, etc., existed, as well as a sense of burning in throat and stomach. She died thirteen and a half hours after taking the poison, retaining her intellectual faculties to the last. On examination, forty hours after death, the following were the chief appearances observed: great lividity of the skin, congestion of the brain and its membranes, congestion of the lungs, flaccidity of the heart, the inner surface of the stomach covered with a yellowish pultaceous matter, on the removal of which an uniform yellow ochrous color was observed, except towards the great curvature, where it became reddish; a gelatiniform softening (*ramollissment*) of the mucous membrane prevailed, exposing in some parts the submucous cellular tissue. The small intestines were somewhat injected, and contained yellowish matters.

Case XXVIII.—Poisoning by Chloride of Zinc.

(The Lancet, September 3, 1864, p. 267.)

§ 581. T. B., aged sixty-three, was admitted into the Royal Albert Hospital, Davenport, on the evening of the 18th of May,

1864. After a quarrel in a public-house, she swallowed an ounce and a half (as nearly as could be ascertained) of Burnett's solution of the chloride of zinc, which she had purchased for the purpose of poisoning the bugs. Almost instantly she experienced great pain in the stomach, and vomited freely. Shortly afterwards she was much purged. Medical aid was procured, and mustard, white of egg, and mucilage administered. An hour and a half after she had taken the poison she was brought to the hospital. On admission she was much collapsed, with cold extremities and clammy sweats; vesication of the lips and tongue; pulse very small and quick. She complained of burning pain in the œsophagus and stomach, of giddiness, and of loss of sight. Her voice was gone, and she could only express herself by whispers. She vomited repeatedly, and was violently purged. The matter vomited after admission consisted of the remedies administered with mucus, but no blood. The motions were thin, and of a dark brown color. Had been an habitual spirit-drinker. Brandy was administered both by mouth and by injection; dilute hydrocyanic acid, bismuth, and mucilage were given to allay the frequent vomiting; while opium and brandy were given as an enema. During the night she had occasional slight fits, losing consciousness, and having twitchings of the facial muscles. She never rallied from the collapse, the vomiting and purging continuing up to the time of her death, fourteen hours after admission.

Autopsy, forty-eight hours after death.—Body in good condition, presenting a very natural appearance. Slight cadaveric lividity of the shoulders and back. The mucous membrane of the lips and tongue was abraded. The mucous membrane of the œsophagus was entirely destroyed and removed, except in some parts of the lower third of its extent, where it was softened and hung in shreds. The mucous membrane of the epiglottis was abraded, with swelling and congestion of the fauces and larynx. The peritoneum was injected; no lymph or serum effused. The stomach was of a slate color externally; veins much enlarged; coats thickened, and of leathery consistence. Internally, the mucous membrane was of an ash color, corrugated, and destroyed entirely. There was no appearance of ulceration or perforation. The duodenum and intestines were congested. Heart normal; left side gorged with blood. Lungs congested.

It may be remarked that notwithstanding the great heat of the

weather at the time, the body presented the appearance of a quite recently dead person; no decomposition had commenced. The intestines were examined and detached, but no disagreeable odor was apparent, owing evidently to the antiseptic powers of the poison taken.

Case XXIX.—Cases of Poisoning by Hellebore.

(Annales d'Hygiène Publique, tome xlviii. p. 465.)

§ 582. I. Morgagni reports a case of death eight hours after ingestion of two grams of black hellebore. The deceased had had severe pains and vomiting. On autopsy the digestive canal was found inflamed, the large intestine more than the small; the latter showed alternate constriction and relaxation. There was no gangrene. Forty-two hours after death the limbs were flexible.

II. A servant near Saint-Brieuc who had been somewhat ill for two or three months went to consult a quack who gave him root of Solomon's seal, leaves of ground ivy, and root of black hellebore. He boiled these in cider and reduced the decoction to a half-liter; he then drank a glassful. His master out of curiosity drank the same amount. Forty-five minutes later symptoms of poisoning manifested themselves in an alarming manner. The servant, believing the pains to be due to the beneficial action of the compound, drank a second glass. The symptoms grew worse; vomiting, delirium, most violent contortions accompanied by extreme coldness, which nothing could overcome, then death. It is to be noted that the violence of the symptoms followed a regular course which coincided perfectly with the amounts of the compound taken by the two individuals; the master, who drank but one glass, died in two and a half hours, while the servant, who took two, died forty-five minutes sooner. The *post-mortem* examination was made 16 hours after death. The same changes were present in both cases, but more markedly in the cadaver of the servant. The lungs were engorged; mucous membrane of the stomach considerably inflamed, blackish-brown, and almost gangrenous. It is somewhat remarkable that the œsophagus and intestines showed no change.

III. On November 5, 1850, Dr. Mavel was called in great haste to a family where six persons had been taken suddenly ill a half hour after dinner. The food which they had eaten did not vary from that of the previous day. On his arrival he found the father

and mother abed and two children of five or six years, a son-in-law, and a tailor, who had worked at the house since the day before, all vomiting and suffering from colicky pains. The mother had eaten nothing but soup, and was at the same time the most dangerously sick. The others had eaten pork, potatoes, and cheese. The mother, a woman of seventy years, had ordinarily enjoyed good health. When seen by the physician her face was bluish and anxious, tongue cold, general coldness of the skin, no pulse, eyes dim, absence of sight, vomiting of greenish matter, colicky pains. If she had been the only one sick, Dr. Mavel would have called it a case of cholera, but the fact that six persons were affected after the same repast convinced him that he had to deal with poisoning. He administered an emetic to favor the vomiting, had the patient wrapped naked in hot blankets, which were renewed every ten minutes, and had the thighs rubbed until sinapisms could be procured. This treatment was carried out, and during the following days her condition was better. When she had regained her faculties she informed Dr. Mavel that she was the sole cause of the poisoning. Her son-in-law had been troubled with urticaria which he thought was itch, and he had been advised to rub himself with a decoction of white hellebore. He obtained two of the roots which she boiled in a saucepan, which was afterwards put away without having been cleaned. It was in the same vessel that the soup had been made.

Dr. Mavel made no mention of the other patients, and it is presumable that recovery occurred with all.

IV. In 1860 a woman of thirty-five years living in the hamlet of Boiserie, poisoned her elder brother by repeated small doses of an extract of white hellebore, extended over a period of two months. Encouraged by the success of her first crime, she resolved to take the life of a younger brother. This she accomplished in fifteen days. At the same time she was experimenting on her mother, a woman of seventy years. Suspicion began to fall on her, and she allowed her victim to recover. She then set fire to the house, intending that her mother should perish in the flames. Chemical analysis of the organs of the two brothers left no doubt as to the cause of death; a considerable amount of the active principles of white hellebore being found. The woman was condemned to hard labor for life.

V. A chemist took four grams of the tincture of green hellebore,

an amount equivalent to sixty centigrams of the powder. The symptoms observed by the physician who was called, were vomiting, cold and clammy skin bathed in sweat. He administered an ounce and a half of brandy which eased the vomiting; a large sinapism was applied to the epigastrium and a bottle of hot water to the feet. Warmth returned gradually to the surface, the patient slept about a quarter of an hour, and woke up in a very satisfactory condition. This was the first authentic case of poisoning by green hellebore.

Case XXX.—Poisoning by Mushrooms.

(Tardieu, l'Empoisonnement, p. 978.)

§ 583. Mme. R., aged forty, and her daughter aged twenty, living in the little village of Saintry near Corbeil, gathered a lot of mushrooms of which they made a dinner. A few hours later the daughter complained of vertigo, and felt as though she had taken opium. She drank some coffee, and was calm and comfortable until three o'clock in the morning, when she awoke with colic and vomiting; she was given more coffee. At eight o'clock A. M. the physician found her in the bath assisted by her mother, who was just beginning to feel the same symptoms. There were no traces of mushrooms in the stools. Wine of antimony was given. The vomiting continued, but the alvine discharges were less frequent, tongue neither dry nor cold; no great thirst; abdomen not distended or tender; temperature of body and extremities normal; physiognomy very little altered; pulse normal.

The mother had micturated frequently, but the daughter had voided no urine since eating the mushrooms; there was a suspension of its excretion.

The two women were cheerful and bright, and talked gayly about their sea bath, their plans, and so forth, but their conversation was frequently interrupted by vomiting. Their condition did not appear at all grave. At six o'clock P. M. thirst very great, and only momentarily relieved by abundant cold drinks; vomiting less frequent but more fatiguing, and followed by prostration and faintness. Extremities cold, sensation blunted; numbness and soreness in legs and loins; sight uncertain; lips and tongue cold; indifference of the two women toward each other. At eleven o'clock thirty

leeches applied to anus; iced drinks and sweet almond oil given. No amelioration of their condition, but they were apparently calmer, although frequently interrupted by groans and vomiting; intense thirst.

About thirty-six hours after eating the mushrooms, vomiting was less frequent with the mother, but she did not seem relieved, and asked for an emetic. The daughter was calm, but still continued the vomiting. During the day they became worse, and the indifference toward each other became more complete. Intense thirst; weaker eyesight; incoherence of ideas; facies hippocratica. With the daughter the eyes were turgid, sight dim, pulse slow but regular. She died murmuring names of those dear to her. At night the mother's eyes were sunken; lips and tongue cold and purple; complexion like that observed in cholera; pulse hardly perceptible; heart beat very feeble but regular. The symptoms persisted until six o'clock the next morning, when death ensued. The remedies given in these cases were all rejected by the stomach. Dry friction and friction with camphor oil failed to bring about any reaction. "Antiémetique de Rivière" with ten drops of laudanum seemed to do more harm than good; ice internally and emollient fomentations were soothing.

A servant girl who ate a few morsels of rare mushrooms vomited eight or ten times sixteen hours afterwards, but was not much indisposed. Another who had tasted some cooked ones was not ill until forty-eight hours afterwards, but caused a great deal of anxiety.

Case XXXI.—Suicidal Poisoning by Cantharides.

(Tardieu, sur l'Empoisonnement, p. 1223.)

§ 584. A man aged forty, somewhat insane for two years, and suffering with general paralysis and trembling swallowed at about 7.30 A.M. fifteen grams of dough containing eight grams of powdered cantharides. Fifteen minutes later ipecac and lukewarm water were administered; repeated copious vomiting followed. The cantharides in the vomitus, which was muco serous in character, was in such large amounts that it was supposed that none could have remained in the stomach. Towards ten o'clock small blisters formed on the lips; the mucous membrane of the mouth was in-

tensely red. After vomiting the body temperature was less. Towards five in the afternoon slight fever set in; temperature and pulse increased. This lasted about twelve hours, when it began to diminish steadily. Ten hours after the ingestion of the poison there was considerable meteorism. The penis, and especially the glans, was livid, but without erection; the urine was slightly bloody. After the disappearance of the febrile symptoms the limbs became somewhat rigid and the intellect more benumbed than before. Meanwhile, stupor and dyspnœa appeared; the patient continued in this state until 8.30 A.M. when he died (25 hours after taking the poison). He had as remedies only mucilaginous and albuminous drinks.

Autopsy.—Skull thin; bloody serum between dura mater and arachnoid. Vessels (particularly the venous) of the meninges filled with blood. Arachnoid and pia mater inflamed and thickened throughout. Considerable serum in the convolutions and lateral ventricles. White and gray substance softened. Heart and lungs normal; pulmonary artery near its origin red internally. The stomach contained part of the administered drinks. Mucous membrane of the stomach dotted throughout with red; here and there ecchymoses mostly near the pylorus and cardiac orifice; at those places a few particles of cantharides. The small intestine distended by gas; nothing abnormal; no fluid contents. Large intestine normal; liver enlarged; left kidney somewhat contracted, red and congested; right kidney and ureter normal; left ureter reddish internally; bladder very much thickened, compact, and resisting; mucous membrane injected and red.

Case XXXII.—Accidental Poisoning by Tincture of Cantharides.

(Tardieu, l'Empoisonnement, page. 1224.)

§ 585. October 13, 1851, a soldier, desirous of entertaining six friends who had done him a service, offered them a mixture of honey, alcohol, and water. The ingredients had been taken from the military infirmary. Three hours previously they had eaten a very hearty meal, with which they drank about ten liters of wine. The mixture was divided equally and each drank about 200 grams in two bumpers within ten minutes, when they separated for roll-

call and went to their barracks. After a short time (from forty-five minutes to two hours in the several cases) the seven soldiers experienced a sensation of weight and burning pain in the epigastric and umbilical regions, soon followed by violent colicky pains, nausea, and copious vomiting. At the same time each felt a severe burning pain in the penis, especially at the extremity, accompanied by frequent desire to urinate. All showed signs of excitement, got out of bed, and ran about the room tormented by profuse vomiting and dejections. Frightened by these symptoms, and fearing that something was wrong, the soldier who had appropriated the things from the infirmary sent for the veterinary surgeon whose property they had been. The surgeon soon discovered that the supposed alcohol was a tincture of cantharides, which had been prepared about 15 or 20 days, and that the quantity stolen and consumed was about 600 grams.

Poisoning being evident, he administered to each a strong emetic and sent them to the hospital, where they arrived at 2 A. M. Dr. Tassart noted the following symptoms; face pale and with an expression of anxiety and terror; heat and constriction in the throat; great pain in the epigastric and umbilical regions increased by pressure; frequent vomiting; intense thirst; pains in the hypogastric and lumbar regions; inclination to urinate every two or three minutes with emission of a few drops of bloody urine accompanied by terrible pain, compared by the patients to that which would be produced on introduction of a red-hot iron into the urethra. This pain had its maximum of intensity at the membranous portion and at the meatus. One of the patients experienced for about five minutes a painful erection. Pulse quick, small, and weak. Skin moist and slightly cold. No headache, delirium, or convulsions. They were given flaxseed-tea and a camphor preparation; also hip-baths. At seven in the morning the same symptoms persisted with the exception that the alvine dejections had entirely ceased three hours previously. The dejections were easier, without pain or tenesmus, and free from blood. Tongue red at the edge and yellowish in the centre. Marked injection uniformly diffused at the pillars and pharynx. All the patients complained of a painful sense of constriction in the throat reaching the upper part of the œsophagus, and of a sensation like the strong pressure of a coin against the posterior wall of the pharynx. Thirst still very intense;

two of the patients had difficulty in retaining drink. Ischuria as before. Urine bloody and passed drop by drop; very albuminous. The patients, some lying on the side with flexed legs, others with the vessel with body bent forward, made desperate attempts to urinate. In all a complete absence of erection; slight headache; pulse somewhat more rapid than normal, small and weak. Liniments of camphor and opium were applied to the epigastrium and perineum; enemata containing camphor and opium; baths.

In the evening there was notable amelioration of the symptoms. Vomiting less frequent with three of the men, and entirely stopped with the others. Constriction of the throat and colicky pains less pronounced. Desire to urinate was less frequent and less urgent, but still accompanied by pain. Urine a little less bloody but still very albuminous. No dejections; no erections. On the 15th, condition still better; patients had some sleep; headache disappeared. Abdomen but slightly sensitive on pressure; no vomiting. Desire to urinate felt but once an hour. Urine less red; some whitish film; emission less painful; no erections or dejections. Pulse normal. Dejections had been checked three hours after entrance into hospital and had not returned. Enemata diminished. On the 16th there was but a slight constriction of the throat; hypogastrium no longer sensitive to pressure. Urine normal in color; slight burning sensation on passage; still somewhat albuminous. Appetite regained; thirst disappeared. Broth, soothing drinks, and baths were given.

On the 17th all symptoms had disappeared; all the organs performed their functions. On the 23d the patients were discharged perfectly cured.

Case XXXIII.—Poisoning by Laudanum.

(From the records of Medical Examiner Dr. F. A. Harris, Suffolk County, Massachusetts.)

§ 586. On May 14, 1881, A. G. C. entered a hotel in Boston, at 4.30 P. M. and engaged a room under the name of Smith. He went out at eight o'clock and returned at nine. During the night, heavy breathing and groaning were heard in his room, which was found to be locked. The same sounds were heard in the morning, and the room was entered without difficulty, as the door was found

to have been unlocked. The man was discovered in a dying condition, and medical assistance was promptly summoned, but death ensued before the arrival of the physician. A bottle containing laudanum was found in his pocket, and entries made in his diary showed him to have been subject to melancholia.

The *autopsy* was made twenty-four hours after death, and the fol lowing were some of the appearances found. Rigor mortis marked Lividity of dependent portions. A light froth issued from the nostrils. Punctiform ecchymoses were seen over both shoulders. The lungs were engorged, and frothy serum flowed from the cut surfaces; they were uniformly dense. The heart, especially the right half, contained a considerable quantity of fluid and clotted blood. The spleen and kidneys were congested. The liver, stomach, intestines, and bladder were normal. The stomach contained a quantity of yellowish fluid resembling pea-soup. *The brain presented no unusual appearance.* The liver, kidneys, brain, stomach, and contents were submitted to Prof. Edward S. Wood of the Harvard Medical School, for chemical analysis; meconic acid and morphine showing the presence of the laudanum were detected in the contents of the stomach.

Case XXXIV.—Poisoning by Morphine. Detection of the Poison in the Body.

(Maschka's Handbuch der gerichtl. Med., vol. ii. p. 441.)

§ 587. A seven months' old boy, who had had convulsions several times, was ordered, on the occasion of a fresh attack, to have given him a half a grain of calomel. The powder was given, convulsions set in, and the child died in a state of stupor two days afterward without having vomited. The physician found the powder to be bitter, and suspected morphine poisoning. An autopsy was performed. The tongue was covered with mucus; not swollen. Pupils not dilated. Stomach contained a brownish flocculent mucus; walls easily torn; mucous membrane pale green, epithelium easily stripped off. Duodenal mucous membrane somewhat reddened. Small intestine pale, distended; contained a thin, pappy, light-yellow substance; mucous membrane nowhere reddened, glands healthy. Large intestine pale; contained pappy feces colored by bile. Omentum and mesentery normal. Liver pale, anæmic, yellowish-gray.

Kidneys pale and healthy. Spleen normal. Bladder empty. The vena cava contained considerable dark fluid blood. Larynx and trachea contained some whitish foam; mucous membrane pale. Lungs œdematous; bronchi contained white foam. Œsophagus empty and pale.

Chemical examination showed traces of morphine in the stomach and its contents, and in the intestine. There was none of the poison in the liver, spleen, etc.

One of the powders contained 0.477 grain of morphine.

Case XXXV.—Poisoning by Chloral Hydrate.

(The Lancet, Feb. 18, 1871, page 226.)

§ 588. J. M., female, aged forty-six, married, in comfortable circumstances, and whose constitution presented unmistakable evidence that she was approaching the critical period of life, had for the past seven years been occasionally addicted to excessive indulgence in stimulating liquors. In 1863 she received several mental shocks, and about the same time she also severely taxed her nervous system by too assiduously nursing a sick mother. It was subsequent to this that she first indulged to any extent in the above-named vice. She was first seen by Dr. Hugh Norris in November, 1869, on the occasion of her suffering a severe attack of hysteria, complicated with spinal irritation; he did not learn till some months afterwards that she was the victim of dipsomania. When opposed in her desire for stimulants she became extremely violent, and, being a powerful woman, no inmate of the house escaped her assaults, or came off scathless in her struggles to gratify her desires. No treatment, moral or medical, appeared of any avail. The various diffusible stimulants and antispasmodics were in vain tried for her hysterical symptoms, and no sedative produced effect but the chloral hydrate. Of this a nightly dose soothed her more or less, and for months previous to her decease it was necessary to administer this drug almost every evening in order to procure her any sleep whatever. Just how much chloral she took it is impossible to state, but between October 13, 1870, and the day of her death, January 12, 1871, she had forty-five single chloral draughts *from Dr. Norris*, none, however, exceeding forty grains, and some only from twenty to thirty grains. He attempted several

times to substitute other sedatives, as bromide of potassium or henbane, but without success. Nothing calmed her restlessness or soothed her neuralgia but chloral or stimulants; the one or the other she was resolved to have. Dr. Norris preferred the former, as a very small indulgence in fermented liquors was sufficient to "set her going;" and, when she once commenced, her house was like Pandemonium. The chloral did not appear in any way to injure her health, and it certainly added to her comfort; and by its administration he hoped to control her passion for stimulating drinks. After the 16th of December, he had altogether ceased the supply of chloral draughts until the 3d of January, when he was hurriedly called to see her late at night, as she had taken a large quantity of a concentrated preparation of sarsaparilla, and was supposed to be dying. She was found in a fit of hysteria, and it was learned that she had swallowed (probably on the chance of its containing some stimulant) fully ten ounces of Townsend's extract of sarsaparilla. At her own request she took a draught containing thirty-six grains of chloral, which she had obtained from a neighboring chemist. She received from the physician a dose of this drug each night but one that she continued to live, usually one-half drachm a day, except on the 8th she had twenty grains in the morning and forty grains at night; on the 9th, ten grains in the morning, and thirty grains at night; on the 10th, ten grains in the day and forty grains at night; and on the 11th, forty grains at night. On the 12th, Dr. Norris was astonished to learn that she had died almost suddenly at noon. On making minute inquiry as to the circumstances attending her death, he was appalled to learn that she had been taking from the druggist above mentioned an extra draught (containing at first thirty grains, and latterly thirty-six grains of chloral) each night for a month past, *in addition to those which he had given her;* also that during the night of the 10th she had taken no less than *three* draughts, each containing thirty-six grains, *in addition* to his draught of forty grains, and ten grains in the morning; and that during the night of the 11th, she had taken two such draughts, in *addition* to his of forty grains. He had seen her after the 3d of January almost daily; and though she suffered much from sickness (a prominent symptom for some years past) he found her pretty nearly in her usual condition, save that on the 9th she complained much of neuralgia in her hands and wrists; on that day she had suffered a

considerable loss of blood from the menstrual discharge. He had called on the 11th, the evening preceding her decease, but did not see her, as she was dressing to go out to tea with a friend; he was told, however, that she had been better the whole day.

The following is an account of the occurrences of the previous two days, as collected from her husband's statement at the inquest. On the 9th she neither ate nor drank, and, as she complained of pains in her stomach, she took a draught (containing ten grains of chloral). On the 10th she was very much excited, and tore one of the blankets in pieces by kicking. She declined to take any medicine during that day. On going to bed she said, "I *must* have something to put me to sleep." She had one of the forty-grain draughts. About midnight she awoke and insisted on having another draught. On being refused she became very excited, and lay on the ground writhing. During the night, however, by dint of the most earnest entreaties, she managed to obtain three other draughts, each containing thirty-six grains of chloral. After the second of these she became very sick and obtained the third draught on the pretext that, as she had thrown up the second, it could not have benefited her. She had on a late occasion, when taking two a night, exclaimed, "Oh, if you only knew what good they do me, you would give me bottlesful of them." On this night she slept calmly for an hour or two after each draught, but at daylight she was observed to talk in her sleep, a symptom never before noticed. This soon passed off, and she slept quietly. When she awoke she said she was better, and she *appeared better than she had been for a long time;* she had beef-tea and toast for breakfast, and ate more than she had done for several days. Her husband was absent on business nearly all day on the 11th. On his return he found that she had gone to have tea with a friend, having previously drunk half a glass of beer. Between 10 and 11 P. M. she had one of the forty grain draughts; and between 1 A. M. and 2 A. M. a second containing thirty-six grains. About 3 A. M. she complained much of feeling cold, but went to sleep, and awoke about 5 A. M., when she talked cheerfully. After a time she exclaimed, "I feel that pain at my stomach again." She appeared in great agony, but got up, went down stairs, took some pepper and hot water, which gave immediate relief. She returned to bed, got warm, and fell asleep. About daylight she awoke, and asked for another draught, which,

after some opposition, was given her. She soon went to sleep, but awoke between 10 A. M. and 11 A. M., went down stairs without dressing, and took her breakfast, consisting of a cup of tea and two slices of bread and butter. She then returned to bed and gave her husband instructions to execute some commission for her, as he was going into the town. On his return, after about an hour's absence, he found her sick upstairs, and begging for brandy to stop her vomiting. As aforetime stimulants had failed to relieve sickness, he refused. He left her (in bed) for about five minutes, and on his return she was lying dead on the floor, with a vessel by her side in which she had evidently been vomiting.

In the absence of Dr. Norris, his assistant, Mr. Edwards, saw her immediately, and found her warm but quite dead, the pupils being dilated. Four hours afterwards when seen by Dr. Norris, the body was still warm, as were the sides of the face and neck, notwithstanding the coldness of the weather. All the muscles were quite flaccid, and the joints perfectly limp. The pupils were fully dilated. At the *autopsy*, made a hundred hours after death, there was not the slightest odor of decomposition, except in the air displaced from the lungs on moving the body. The following appearances were observed: There were scarcely any puncta sanguinea in the white portion of the brain, which was very fresh and firm, and little if any fluid in the ventricles. The liver was much enlarged, slightly congested, and somewhat leathery. The kidneys were large, but not apparently diseased. The heart tissue was somewhat pallid; the ventricles were empty; the auricles partially distended by dark, semi-coagulated blood. The stomach, which was not opened, contained two or three ounces of fluid matter. The body was well nourished. All the remaining organs appeared quite healthy, but very firm, and not decomposed in the slightest degree. There was no perceptible odor of chloroform. A hundred and thirty hours after death the stomach and contents, together with portions of the lung, liver, heart, kidney, and spleen, were submitted to Mr. Stoddart, of Bristol, for analysis. The several portions were preserved in an extraordinary way. After more than a week had elapsed since death, there was not the slightest sign of decomposition, nor any unpleasant odor. By chemical analysis the chloroform resulting from the decomposition of the chloral hydrate was detected in the liver and contents of the stomach. No

blood was sent for analysis. In the other organs no chloroform was detected. The contents of the stomach had no perceptible odor of chloroform until after the addition of an alkali.

Case XXXVI.—Poisoning by Nicotine.

(Procès du Compte et de la Comptesse de Bocarmé. Paris, 1851.)

§ 589. (On account of the great interest which this trial excited, we have subjoined the following succinct history of the case, as presented by the Attorney-General of the Court of Appeals of Brussels.)

Act of Accusation.

The Attorney-General of the Court of Appeals of Brussels represents that the court, by a decree of the 16th April, 1851, transmitted to the Court of Assizes of the province of Hainaut the names, first of Alfred Julien-Gabriel-Gérard-Hyppolite Visart, Count of Bocarmé, aged thirty-two years, landholder, born at the Camp of Weltevreden in Java, etc.; second, of Lydia Victoire-Joseph-Fougnies, aged thirty-two years, wife of Count Bocarmé, born at Péruwelz, and both living at Bury, accused of the crimes enumerated in the articles 301, 302, 59 and 60 of the penal code.

In consequence, the Attorney-General has drawn up the present act of accusation, in which the following facts and details are set forth:—

The Count Hyppolite Visart de Bocarmé, belonging by birth to one of the first families of Hainaut, married, in 1843, at Péruwelz, the daughter of an ex-grocer who had two children, and whose son, having lost his right leg by amputation, had not a very strong constitution. The accused, therefore, even before the contract of marriage, foresaw that the end of Gustave Fougnies, his brother-in-law, was more or less near; and after having secured to himself the property of his wife by will, he did not hesitate to consult Dr. Semet regarding the chances of life or death which Gustavus might have.

But Gustavus also began to think of marriage. He had already entertained the idea in 1846, and he was on the point of carrying it into execution, in the month of November last, when he died suddenly at the mansion of Bitremont, where the prisoners resided, and in the very apartment where he had been dining with them. They

communicated the intelligence the next day to Madame Dudzeele and her daughter, to whom Gustavus was about to be married; and the Countess herself charged a servant to "go and tell the two hussies that her brother had died of apoplexy." But the state of the body indicated a very different kind of death, since the autopsy disclosed upon the anterior part of the nose a deep contusion, upon the left cheek a number of scratches, which appeared to have been made by the finger nails; over the left maxillary region there was a corrosion involving the cuticle, and which seemed to have been caused by some caustic fluid; in fine, upon the tongue, in the mouth, throat, and stomach, there were numerous traces of the passage of a similar substance.

The physicians (experts) concluded from these observations that a corrosive liquid had been poured during life into the mouth of Gustavus Fougnies, and had produced a cauterization of the whole of that cavity and part of the pharynx; that a portion of this liquid, either spilt or rejected, had burned the left side of his neck; and that the marks of violence on the face proved that efforts had been made to force down the liquid, and to stifle the cries of the victim.

Moreover, the Count presented upon the second phalanx of the middle finger of the left hand two wounds, which involved the skin, and which were evidently the result of a bite, for the marks of two teeth were visible in the lower wound, which was deeper than the other.

At the time the investigation took place, on the 22d November, at the chateau of Bitremont, there was also apparent upon his fingers and under his nails a red discoloration, which was only too evidently connected with the scratches of which the face of Fougnies offered numerous traces. All this required an explanation, which was far from being satisfactory; and chemical analysis speedily demonstrated that Gustavus Fougnies had been poisoned by nicotine, a narcotic alkali, extracted from tobacco, and which is one of the most deadly poisons. The prosecution was prepared to show that the accused had for ten months previously made this poison a particular study; that he had, some days before the death of Gustavus, procured by his labors two small phials of it, which, since that event, have not been found. Moreover, the Countess herself formally accused her husband of having poisoned her brother; and although the Count himself now acknowledges that he extracted the nicotine which destroyed Gustavus, without, however, explaining by whose means it

had been administered, we think it may be useful to present a summary of the facts which instigated, preceded, accompanied, and followed the crime on the 20th of November.

In marrying Lydia Fougnies, whose patrimony he had over-estimated, Count Bocarmé was far from gaining an opulent position, since he only received from his father-in-law a yearly allowance of 2000 francs, and he brought on his own side 2400.

Such feeble resources did not well accord with so grand a domestic establishment, with numerous servants, and especially with the irregularities of the accused, who in a short time had a second household in the environs of Brussels. He therefore found himself obliged to resort to daily loans from his notary, to whom he owes nearly 43,000 francs; and although M. Fougnies, the father, died in 1845, leaving his daughter a revenue of 5000 francs well secured, this increase of fortune was far from assuring the future of the accused, since their expenses were every day increasing, and they had even drawn since 1846, without repayment, to the amount of 95,000 francs.

All this did not prevent them from owing dribbling debts to the amount of 7000 francs, some of which dated back to the same epoch, and in which we see domestics or mere journeymen figure for sums of thirty, twelve, ten, and three francs. In fine, they had so completely lost their credit, that the Count was reduced to pledge for 400 francs, at a pawnbroker's in Brussels, ornaments belonging to the Countess, and which are still there. The ruin of the accused was thus imminent, unless the death of Gustavus, on which they had so long counted, should occur, to re-establish their dilapidated fortune.

But Gustavus did not die; he had even formed new projects of marriage, which seriously vexed the accused, and which they sought to break, by means of the notary, Cherquefosse. The Countess herself had written to her brother two letters, which were found after his death, and which contained some slanders against Miss Dudzeele, which she had used in an anonymous letter of the month of August. These attempts, however, had resulted in nothing, and there only remained to the Count the last resort, and the most efficient means for attaining his end.

After having cultivated poisonous plants in 1849, he presented himself, in the month of February, 1850, under the assumed name

of Bérant, before Löppens, Professor of Chemistry at the Industrial School of Ghent, and begged to be informed of the proper apparatus for extracting the essential oils of plants, remarking that he had seen the American savages poison their arrows with the juice of certain plants, and that he wished to make some experiments for the benefit of his parents, who lived in the United States. He consulted Löppens particularly with regard to the mode of distilling the essential oil of tobacco, that is to say, *nicotine;* and he ordered from the brazier, Vandenberghe, according to the instructions of the professor of chemistry, an apparatus of brass, which he wished to be ready by the 11th of March.

On his return to Ghent, in the month of May, the accused showed Löppens the first sample of nicotine, which had not proved efficient. He then recommenced the operation under his supervision, and after having labored two days in his laboratory, he succeeded in obtaining two drops of pure nicotine.

He returned again, after some time, with another sample, which had not succeeded any better than the first. Löppens then gave him new instructions; and the accused announced to him at last, on his third visit, in the beginning of October, that he had obtained the most deadly effects on animals.

Nothing now remained but to procure the necessary substances and instruments to operate on a larger scale, and to follow the procedure of Schlœsing, which Löppens had pointed out as the best, and which Pelouze and Frémy describe in their course of General Chemistry.

But these purchases made new journeys to Brussels necessary, which the accused visited on the 16th and 28th of October, and after laboring without interruption two days and two nights, he at length succeeded, on the 10th of November, in obtaining two phials of nicotine, which he was to employ on the 20th, and which could not be found after the death of Gustavus. With regard to the chemical instruments which had served for its preparation, the Count had taken care that they should immediately disappear. The servants of the establishment could give no information with regard to them, and it was not till six weeks after that they were discovered in a secret place, where the Count had mysteriously concealed them.

This precaution, all will agree, does not well accord with

scientific labors, or with researches made for the benefit of another continent.

There is, moreover, the false name of *Bérant*, which the Count always assumed in his interviews with Löppens and Vandenberghe, although he did not conceal his true name at the pawnbroker's shop in Brussels. We may then safely conclude that he had already, in the month of February, meditated the crime which he committed in the month of November, and of which his own mother would seem to have had a presentiment, since she said one day to her daughter-in-law, that Hyppolite was capable of anything, that he might do some mischief by his chemistry, and that she expected nothing else but to see him some day brought before the Court of Assizes. The diligence with which he labored night and day, moreover, clearly indicated the object he had in view, especially at the period when the idea of marriage had taken possession of Gustavus, and the Countess herself had avowed the object, since she said in so many words, at one of her examinations, "My husband speculated on the death of Gustavus; it was his fortune that he coveted—it was that which made him decide upon his death; he had lived too long, in his estimation. During the first days of November, I knew that the poison was prepared for Gustavus; I knew, moreover, that the poison was nicotine. My husband himself told me this in the rear wash-house, the day I saw the large matrass in the vessel of oil, and where he told me he made cologne water. I used many entreaties to know what he was really making, and he at last admitted that it was nicotine. Some days after, he told me, that the first time an opportunity presented, he would not miss Gustavus; and on the 20th of November, on learning that he was coming to Bitremont, he declared to me," added the Countess, "that he would do the business for him that day."

Gustavus, in fact, arrived at ten o'clock; it only required a single word to save him, and yet the Countess passed the whole day with him without informing him of the dangers which impended. She even gave orders which would insure the execution of the crime, by removing those whose habitual presence would have prevented it. Thus, she made the oldest of the children, and his governess, dine in the room of the latter, instead of admitting them to her own table where they dined every day, and she had caused supper to be prepared for the two smaller children in the apartment

of the nurses, instead of in the kitchen as was their custom. It is true that one can hear from the kitchen what passes in the dining-room. She also sent her coachman, Vandenberghe, to Grandmetz, with a letter to the ladies of Dudzeele, although he had, by the arrival of Gustavus, an additional horse to take care of, and although the letter had no other object than to inquire of the ladies what price they would ask for their agricultural implements. There was no urgency in the message, but the distance to travel over required the absence of the coachman for four or five hours; and when afterwards the Countess ordered her chambermaid, Emérance Bricourt, to serve at table instead of Vandenberghe—she was careful to order her to withdraw after the second service. Emérance did not again appear in the dining-room until the time when she supposed they would need a light, and the accused to whom she came to offer it, answered both at the same time, "*No, no, not yet.*"

On withdrawing, Emérance was going to the kitchen, where the coachman was dining, who had returned from his trip to Grandmetz. The Countess followed her and saw her go up to the nursery, where she found the two nurses, Justine Thibaut and Virginia Chevalier. She had also ordered Vandenberghe to accompany, as far as the road to Leuze, a distance of about one kilometre (nearly equal to three-quarters of an English mile) the cook, Louisa Maes, who was returning home. Vandenberghe had set out on the road with Louisa, but he was not long in perceiving that it would be too late for the girl to travel alone, and, as she had no money to pay for lodging on the way, he had returned with her to the house, and informed his master and mistress, who were still in the dining-room with Fougnies. Gustavus had already manifested an inclination to leave. The Count had even ordered Francis Deblicquy, the gardener, to get the carriage ready, but the stable was locked, and Vandenberghe had the key. He had scarcely returned to the house, when the Count went to the kitchen to give the same orders which he had given to Deblicquy. The coachman then took the lantern and went to the stable, and the Count returned to the dining-room.

Justine Thibaut was coming down stairs at this moment to get some supper for the children, although the Countess had ordered them away from the kitchen on this occasion, as already stated.

Arriving upon the last steps of the stairs, she heard a fall in the dining-room, and the voice of Gustavus, who cried for help, exclaiming "*Oh, oh, forgive me, Hyppolyte!*"

She then ran to the kitchen, crossing the office which separated it from the vestibule and dining-room, when she saw the Countess go out of the dining-room and enter the office, closing the doors of the two apartments, so as to prevent the cries of Gustavus from reaching the kitchen. The girl, being still more frightened at this sight, hastened to reach the court by a circuitous way; she then passed opposite the windows of the dining-room, from whence issued stifled cries, and went up to the children's apartment by the old back stairway. Emérance, whom she found there, then went down to offer her services; but she heard no more noise, and the Countess made her go up again on seeing her at the bottom of the stairs.

The marks of violence observed upon the body exclude the idea of accident or of suicide. They prove, on the contrary, a violent struggle; and, when we reflect, that, to make the victim swallow the poison, it was necessary at the same time to open his mouth and restrain the movements of his head to the right and left, which he would otherwise make, it is nearly impossible to admit that the crime was the act of one person only.

How, indeed, can we conceive that the Count Bocarmé, whose left hand, imprinted with a double bite, was held in the mouth of Gustavus, and whose right hand was fully employed in steadying the head and arms, could of himself, and without foreign aid, pour into his mouth a phial of nicotine?

Another person was, therefore, necessarily a participator in the act, and there were only the Count and the Countess in the dining-room at the moment when Justine heard the fall and the cries of Gustavus. The accused wrote as follows, on the 12th of last March, to a correspondent in Paris: "My wife has requested you to engage M. Berryer: do not do it; and, if the engagement is made, suspend it until a new order is received from me, but let her continue in the belief that he is engaged. On this recommendation both her life and mine depend. Only imagine that this wretched woman, after having poisoned her brother, can find no better defence now, when we are both in prison for the deed, than to charge the whole upon me, and to accuse me of the most atrocious crimes. Do not answer this note, which I secretly slip in the accompanying letter. Re-

member, that all the letters we receive are opened. If Berryer shall have engaged to come, explain to him what I have stated to you in this note; show him that the hostile attitude assumed towards me by my wife, is only the result of a moral constraint, occasioned by the position in which she finds herself placed, and that his aim should be to defend us both equally against the accusation, and not take up for my wife the hostile position she has assumed in regard to me; this would give great plausibility to the charge, and lead us inevitably to the scaffold."

This note, which the accused had fraudulently slipped into a letter, intended to be shown, was not for the Judge of Instruction. It expressed then the secret thoughts of Count Bocarmé better than they were ever explained in his interrogations; and those thoughts entirely agree with the nature of the crime of which he is accused; it also well agrees with the disclosure the prisoner had made to the keeper of the prison, since he told him, on returning from his first examination, that it was the Countess who had turned the poison into the mouth of Gustavus; that she had made two different attempts in doing it, and had even spilt it on the clothes of her brother.

This explains why she went a few minutes afterwards to wash her hands with soap in the kitchen; why she immediately placed the clothes of Gustavus, and those of her husband, in a wash-tub full of water; why she caused them to be wrung and washed in lye at midnight, in her presence, by the cook, L. Maes. This also will explain why she caused the crutches of her brother to be washed with hot water; why she even caused them to be burned, saying that she could not bear the sight of anything that had belonged to him; why she had burned his waistcoat and cravat, at the very moment the officers of justice arrived at Bitremont. This will also serve to explain why she caused the floor of the dining-room to be washed the same night, and in her presence; why, the next day, she herself poured oil upon the spots, that they might not be recognized; and why she said, with evident satisfaction, to Emérance, at the time they were making the autopsy, that everything went on well, and that they had discovered nothing, and would bury Gustavus on the morrow.

These facts are too numerous and too direct for any one to doubt of her being an accomplice, especially when placed in connection with

the extraordinary declarations of her husband, with the special character of the crime, and with the measures the Countess had taken to insure its execution. This complicity dated as far back even as the time when she had written, and signed with the false name of *Bérant*, all the letters addressed to Löppens, and the brazier, Vandenberghe; and she had even counterfeited his handwriting in several of these letters.

The Countess alleges, it is true, that if she passed the night in effacing the traces of the crime, it was only to save her husband and the father of her children. But it is very difficult to admit the excuse in regard to so odious a crime, and one, too, committed against her own brother.

Especially it is difficult to admit it in connection with the almost daily acts of violence which the Countess had to complain of, and to which her husband added the grossest immorality, since we have seen that he obliged her to receive the fruit of his adultery at the château of Bitremont.

She also maintains, that if she concurred in preparing for, or aiding the poisoning, she had only done it under the threats of her husband, and under the influence of moral constraint. But then, why did she not at least apprise her brother, when a single word might have saved him? Why did she profane his dead body, by ordering the coachman, Vanderberghe, to deluge it with vinegar? Why apply an opprobrious epithet to the ladies Dudzeele, when she directed a servant to inform them of the death of Gustavus? All this denotes too clearly a common purpose to attain the same object, which might profit both the accused, and which even the uncle of the Countess openly proclaimed in his deposition, explaining the reasons why he had not been present at the house the next day, in compliance with the invitation he had received. "I was," he said, "too indignant against them on account of their infamous conduct, and this indignation has its foundation in my deep conviction that they murdered Gustavus."

In conclusion, Alfred Gabriel Gérard Hippolyte Visart, Count Bocarmé, and Lydia Victoire Joseph Fougnies, wife of Bocarmé, are charged with having wittingly made an attempt upon the life of Gustavus Fougnies, their brother and brother-in-law, at Bury, on the 20th of November, 1850, by means of substances which would cause death more or less promptly, or at least with having been

accomplices in this act, whether they gave instructions to commit it, or procured the substance, or did any other act to carry it into execution, knowing the object intended; whether they knew of, or aided, or assisted the author or authors in, those acts which prepared for or facilitated the deed, or those which consummated it.

Regarding which, the Court of Assizes of Hainaut will decide.

For the Procureur-Général.

May 3, 1851. (Signed) E. D. CORBISIER, Substitute.

The discussions respecting this case occupied the court during twenty-one sittings (from May 27 to June 15). M. Bocarmé was found guilty by the jury, and condemned to death; and Madame Bocarmé was acquitted.

CASE XXXVII.—POISONING BY NICOTINE.

(Taylor on Poisons, 1875, p. 809.)

§ 590. In June, 1858, a London gentleman swallowed a quantity of nicotine from a bottle, and almost immediately afterwards was seen in the act of falling to the floor. He was carried to an adjoining room, but, before this could be reached, he was dead. The symptoms noticed were that deceased stared widely; there were no convulsions, and he died quietly, heaving a deep sigh in expiring. The symptoms, therefore, resembled those of prussic acid. The quantity taken could not be determined. The deceased appears to have been rendered immediately insensible, and to have died in from three to five minutes after taking the poison. The *appearances* observed were a general relaxation of the muscles, prominent and staring eyes, bloated features, great fulness with lividity about the neck. There was no odor resembling nicotine or tobacco perceptible about the body. When examined between two and three days after death, putrefaction had occurred, especially in the course of the veins. The swelling of the neck was found to arise from an effusion of dark liquid blood. The scalp, as well as the membranes of the brain, was filled with dark-colored blood. The lungs were engorged and of a dark purple color. The cavities of the heart were empty, with the exception of the left auricle, which contained two drachms of dark-colored blood. The stomach contained a chocolate-colored fluid (reserved for analysis); the mucous membrane was of a dark crimson-red color as a result of the most intense congestion. There

was no odor except that of putrefaction. The liver was also congested and of a purple-black color. The blood throughout the body was black and liquid; but in some parts it had the consistency of treacle. Nicotine was found in small quantity in the contents of the stomach, also in the liver and lungs; but as the organs had been placed in contact with the stomach, it could not be inferred that the poison had been absorbed and deposited in them.

Case XXXVIII.—Poisoning by Conium.

(New York Times, April 5, 6, and 13, 1875.)

§ 591. Mr. F. W. Walker, 65 years old, having suffered for two years from a nervous affection of the facial muscle, which cauterization, strychnine, and other remedies failed to alleviate, was advised by his physicians to take certain doses of the fluid extract of conium. He was instructed to take fifty minims every half hour until he felt the effects, and then to discontinue the drug. He purchased an ounce vial of the medicine, and, on his return home, took a dose, and calling his wife to his side, dictated to her a description of his symptoms under the operation of the drug. This dictation of symptoms was his invariable habit when taking medicines prescribed by physicians, or taken under his own course of treatment, he having been at times his own physician. The paper which his wife wrote at his dictation tells its own story and reads as follows: "At 4.10 P. M. took fifty minims Squibb's fluid extract of conium. At 4.40 P. M. effect very decided in dizziness, relaxation of muscles and limbs. Fifty minims more then taken. Difficulty of walking immediately, and want of power to control movements. Forced to lie down, but no mitigation of spasms. Limbs and legs weak. Unable to hold head. Speech thickening some. Pain and heaviness in top and back part of head. Pulse fifty-six. At 5.15 took fifty drops. Some nausea. Some tremor at the base of clavicle and in muscles across the chest just above the sternum. No diminution of spasms about the eyes nor of photophobia. At 5.25 drowsiness, inclined to sleep. At 5.40 P. M. eyes difficult to open, speech difficult, fulness in throat, prostration nearly complete. Diplopia vastly increased. At 6.10 P. M. nausea, twitchings on right side, trouble to articulate, eyes closed, fulness almost to suffocation in throat. Pulse about sixty.

In past six—." At this point he stopped dictating. He was then seized with nausea but was unable to vomit; he then wanted electricity applied, but while trying to apply it himself, he fell back dead.

Autopsy was performed about sixty hours after death. In the brain was considerable venous congestion. Falx cerebri slightly thickened and opaque on its anterior portion. Arachnoid and pia mater very much thickened and vascular, but tearing readily from the brain substance. Cruræ cerebri slightly soft. Right crus presented a discolored appearance, and, owing to its softness, was torn on the removal of the brain. Right vertebral artery much larger than normal, and had undergone calcareous and atheromatous degeneration. Left vertebral smaller than normal, and had also undergone degeneration. Basilar artery larger than normal, and with calcareous and atheromatous degeneration. Post cerebral and middle cerebral had both undergone calcareous and atheromatous degeneration. A small foreign growth found on pia mater, just in the fork at the commencement of the cruræ cerebri. Puncta vasculosa not more than normal. Choroid plexus very vascular and thickened; otherwise brain appeared healthy. Weight of brain 57 ounces. Chest: lungs very much congested, perfectly full of blood. Lung tissue very soft and in places easily torn. Heart weighed nineteen ounces; valves perfect; walls healthy. Left ventricle perhaps a trifle thicker than normal in proportion to the whole. Coronary arteries quite free. Aorta: but one small point of atheromatous degeneration visible about half an inch from its origin, and about as large as a pin's head. No fluid in the pericardium. Abdominal viscera found normal excepting an undue amount of fat.

No chemical analysis was made.

Case XXXIX.—Poisoning by Gelsemium.

(American Journal of Pharmacy, January, 1870, p. 14.)

§ 592. On the 30th of January, 1869, three teaspoonfuls of fluid extract of gelsemium were administered to a young healthy married woman several weeks advanced in pregnancy, who at the time complained of no serious illness. In two hours after taking the dose, the patient complained of pain in the stomach, nausea, and dimness of vision. These symptoms were soon succeeded by great restless-

ness, ineffectual efforts to vomit, and free perspiration over the body. At the expiration of about five hours the pulse was found feeble, irregular, and sometimes intermittent; there was great prostration, with irregular breathing and slow respiration. The skin was dry; extremities cold; the pupils expanded and insensible to light; the eyes fixed; inability to raise the eyelids. The vital powers rapidly gave way, and, without convulsions, death occurred in about seven hours and a half after the poison had been taken.

Post-mortem appearances.—Eight days after death the body presented the following appearances, as described by Dr. J. H. Stephenson, who made the autopsy: Countenance natural as in sleep. No emaciation, and body in a perfect state of preservation. Cadaveric rigidity very slight. The back of the neck and between the shoulders, extending the full length of the spine, as also the depending parts of the thighs and arms to the elbows, presented a congested appearance. The membranes and substances of the brain and medulla oblongata were normal. The adipose tissue remarkably thick, and highly tinged throughout with bilious matter. Lungs slightly collapsed, natural in appearance, and superficial veins congested. Heart normal in size, superficial veins injected, and the cavities greatly distended with dark grumous blood, inside of which was found a well-defined membrane identical in appearance with that found in diphtheria and pseudo-membranous croup. The abdomen presented no tympanitic distension. Stomach was slightly distended with gas, and contained a small quantity of ingesta. Peritoneum and intestines in a healthy condition. Liver and investing membrane normal; left kidney congested. The uterus was slightly enlarged and contained a fœtus of about five weeks' development.

A small quantity of the contents of the stomach, which escaped from the organ at the time of the dissection, was collected separately in a small bottle, the stomach with the balance of its contents was placed in a larger bottle. These bottles, with their contents, were carefully sealed and remained undisturbed until the 17th of May. At this time the contents of the bottle containing the stomach were found to have undergone considerable decomposition. A little pure alcohol was added to the decomposing mass, and it was then allowed to remain until the 13th of June, when the chemical examination of the contents of both bottles was commenced. On analysis of their

contents by Dr. T. G. Wormley, both gelsemic acid and the base, gelsemine, were found.

It will be observed that in this case, only three teaspoonfuls of the fluid extract were taken. Presuming it to have had about the same strength as a preparation examined by Dr. Wormley, the quantity of the alkaloid contained in this amount could not have much exceeded the sixth part of a grain. This would seem to indicate the alkaloid to be one of the most potent poisons at present known. On comparing the intensities of the reactions of the several reagents applied to the base recovered from the stomach with those obtained by the same reagents from solutions of the alkaloid of known strength, it was inferred that the quantity of the base recovered in this case did not much, if any, exceed the fiftieth part of a grain.

The fact that the stomach with its contents had undergone considerable decomposition, and also that the chemical examination was not made until some months after death, would seem to indicate that the poison is not readily destroyed by decomposition, and that it may be recovered after comparatively long periods, even when taken only in small quantity.

Case XL.—Poisoning by Belladonna.

(The Lancet, July 16, 1870, page 83.)

§ 593. Ann H., aged 66, swallowed about a teaspoonful of belladonna liniment on March 19, at 11 A. M. At 11.30 a friend found her wandering in her mind, and, having ascertained what had happened, administered an emetic, but without inducing vomiting; subsequently two other emetics were given, but all failed to have any effect. At 2 P. M. she was taken to the Royal Infirmary, Bristol; she was then delirious, talking fast, and throwing her arms about in an excited but feeble way; her pupils were widely dilated and insensible to light; her pulse was 126. The stomach pump was used, but very little fluid was brought up. At 2.45 she was very restless, tossing about in the bed, picking at the bed-clothes, and throwing about her arms in a meaningless way, but partially conscious of what was said to her. She wished to sit up in bed. She could swallow fluids with difficulty, and drank some strong coffee. Pulse 116; respiration natural, rather slow. She was

ordered a draught containing twenty minims of tincture of opium, and a hypodermic injection of acetate of morphine (one-third of a grain) was given soon afterwards. A "calabarized disk" was placed in one eye (the left); in about twenty minutes the left pupil had contracted to a pin's point, the right pupil remaining widely dilated. At 3.30 she was less active in her movements, and seemed more drowsy. The morphine injection (one-fourth of a grain) was repeated. At 4.30 the right pupil was still widely dilated, the left remaining contracted. Her pulse was 100, of good strength; breathing slow. She was more comatose, and breathed stertorously if undisturbed for a few seconds. Another subcutaneous injection of acetate of morphine (one-third of a grain) was given. At 6.15 she was deeply comatose, and apparently had ceased to breathe; no respiration was visible for a full minute, but her pulse was moderately full at 100; her hands were blue, and feet tending to get cold. Artificial respiration by Silvester's method was resorted to, when she began to breathe again regularly but very slowly, about four respirations occurring in a minute. Enemata containing ammonia, brandy, and coffee were given, and galvanism to the respiratory muscles was resorted to. At 8.30 she remained deeply comatose; no reflex movements could be obtained; respirations were five or six per minute; pulse 100; cervical veins were much distended; lips were blue, and the heart's impulse diffused and weak. Brandy and coffee enemata were given every hour, and galvanism was used at intervals. At 1.30 A. M. on the 20th, respirations were six or seven per minute; pulse 100, but variable, at one minute good, at another very weak and small. Venous distension in the neck had subsided. The right pupil remained dilated widely and insensible to light, and the left contracted till the time of her death, which took place at 3 o'clock, sixteen hours from the time of her having taken the poison. At the *post-mortem* examination, thirty-four hours after death, the lungs were found full of blood; the right side of the heart contained but little black blood, the left side being very firmly contracted. The brain was slightly congested. The stomach and other organs presented nothing abnormal. At the time of the autopsy both pupils were equally dilated.

The patient took, in divided doses, eleven-twelfths of a grain of acetate of morphine subcutaneously, and forty minims of tincture

of opium by the mouth. Not the slightest effect was noticeable in the right pupil, *i. e.*, the one which was not acted on by the Calabar bean. Excepting that this pupil was dilated, the patient's condition exactly resembled that of opium poisoning after the delirium had subsided into coma. No evident result was traceable to the morphine administered, except that the coma deepened, and the breathing got slower; but these symptoms doubtless were due to the belladonna, uninfluenced by the medicinal doses of opium and morphine.

Case XLI.—Poisoning by Atropine.

(British Medical Journal, Oct. 5, 1878, p. 516.)

§ 594. Charles S., aged 45, a gardener, swallowed a teaspoonful of liquor atropiæ sulphatis on August 6, 1875, and was taken to the Fulhane Hospital. He said that he was attending an ophthalmic hospital for an affection of the eye, and received some drops as well as a larger bottle of medicine for internal use. He was in the habit of pouring out his daily allowance of the latter into an ounce and a half phial, and carrying it in the same pocket with the atropine. About 1 P. M. he left work, and when he took his medicine, by mistake drank from the wrong bottle. He felt nothing particular, but told the clerk, who brought him home, what he had done. He was then quite sensible; there were no symptoms or signs whatever except excessive nervousness. The left pupil was dilated from a previous application of the atropine. Mr. Greenway gave him an emetic of ipecacuanha wine, but this not acting quickly enough, it was followed by twenty grains of sulphate of zinc in mustard and water, and he was told to walk about. At 1.39 P. M. his pulse was 140, small and regular; temperature 98°. He staggered as he walked, and complained of giddiness. The pupils were not affected. No vomiting supervening, his stomach was washed out with the stomach-pump. He lost power over his legs, and became quite helpless and unconscious; his pulse became excessively weak and irregular; he was given a drachm of tincture of digitalis in brandy. At 2.15 he began to talk quickly, and looked flushed. At 2.45 the pulse was 148, full; the skin was dry. The face was very much flushed. He had very violent delirium, requiring three men to hold him. The pupils had gradually enlarged till they were

nearly fully dilated. The tongue was dry and brown; he could swallow after some compulsion. Vomiting continued at intervals, and black coffee was given him now and then. His feet became cold; mustard was applied to them. At 3.15 P. M. the pulse was 156, smaller; temperature 99°. He was quieter and had a tendency to sleep. The pupils were the same and insensible to light. He did not retract his legs when they were pricked or pinched. When spoken to he did not seem to hear, being evidently quite unconscious. At 4 P. M. the pulse was 144. He seemed generally the same, except that when his name was shouted in his ear, he opened his eyes and turned his head in the direction of the sound. For the first time the respiration seemed labored and quick, accompanied by flapping of the cheeks when he was permitted to sleep. He now moved both arms and legs at times; twitching of some of the muscles was noticed. He had a mustard poultice over his cardiac region, and small quantities were administered. An enema of castor oil was given. At 7 P. M. the pulse was 140; respiration 40 per minute. The tongue was as before, the face less flushed, and the skin still quite dry. He was decidedly more conscious, as, when told to put out his tongue, he did so, and said "yes" when called. He caught at imaginary objects. On account of his precarious state, Dr. Milner Fothergill was requested to see him. He advised that the treatment be continued.

Dr. Fothergill spoke to him, and he answered fairly intelligibly. At 9.15 P. M. the pulse was 140, small and regular; temperature 102°; respiration 50 per minute, laboring. On cursory examination of the chest, a few moist *râles* were heard about the bases. He recognized some friends who came to see him. The skin and tongue were as before. As soon as he fell asleep he commenced to talk unintelligibly. The pupils were as before. Neither bowels nor bladder had acted. At 10.35 P. M. the pulse was 140, full and regular; temperature 102.4°; respiration 60. The bowels had acted slightly. He was inclined to become violently delirious as Dr. Fothergill had foretold. His general condition was unchanged. He had been having eggs, coffee, milk, etc., and a little brandy at intervals. At 11.45 the catheter was passed, but the bladder was empty, and there was no dulness over the pubes. He had considerable tympanites, and tenderness over the abdomen.

August 7, 1.30 P. M. The pulse was 132, full and regular; tem-

perature 103°; respiration 56. He passed a fair amount of urine in the sheets. His skin, pupils, and tongue were as before reported. He complained of great thirst, to allay which ice was given. At 2.45 the pulse was 130, full and regular; temperature 102.6°; respiration 48. He was now nearly conscious and intelligible in his answers, but directly he fell asleep he commenced to talk and mutter. At 4.30 the pulse was 92, small, and slightly irregular. He now remembered taking the poison, and knew where he was. He looked very pale. At 6.30 the pulse was 115; temperature 100.4°; respiration 30. The bowels had acted very freely three times. Ammonia and bark were ordered. At 11.45 the pulse was 140, full, and regular; temperature 102.2°; respiration 36. He complained of soreness at the pit of the stomach. The bowels acted twice again. He had perspired for the first time. The tongue and mouth were less dry. On applying the light of a candle to the pupils, the right contracted slightly; the left did not. He said he had had very pleasant dreams. At 9 P. M. the pulse was 144, full; temperature 102.4°; respiration 52. The skin was now moist. He passed some urine of an amber color; specific gravity 1015; no albumen. At 10.40 P. M. the pulse was 156, small; temperature 100.6°; respiration 52. The pupils were still much dilated. He complained of no pain.

August 8, 10 A. M. He passed a fair night, sleeping at intervals. He vomited once; the bowels acted four times. He took his food well. He complained of some pain over the right side of his chest, and had commenced to cough and expectorate. He was very thirsty. The tongue was as before. The left pupil still dilated; the right was much smaller, and contracted on looking at a bright light. On examining the chest in front, both lungs were resonant and otherwise normal. Behind, at the base of the left lung, a limited area of crepitation was found but no dulness. Over the right base was small crepitation, extending upwards to the spine of the scapula, and very slight dulness. The sputum was rust-colored and scanty. He was ordered a mustard-poultice to the right base, and three grains of carbonate of ammonium in decoction of senega every three hours; also two drachms of brandy every hour, with milk, beef-tea, etc.

August 9, 10 A. M. Pulse 160, regular; temperature 100.2°; respiration 60. He slept fairly well during the night, but in the

morning complained again of pain in the right side of the chest. On examination of the chest dulness as far as the spine of the right scapula was found, and tubular breathing and bronchophony, but no crepitation. Over the left lung posteriorly there was rough breathing, but otherwise the respiration-sounds were normal. At 11 P. M. a sudden change for the worse set in, and the symptoms increased until death, which occurred at 2.40 A. M.

The *autopsy* was performed sixty-five hours after death. The body was well nourished. The abdomen was swollen, tympanitic, and had a green color. The right side of the chest was duller than the left. There was very little hypostatic congestion. The pupils were slightly dilated. There was no rigor mortis. The pericardium contained no fluid. The heart was enlarged and very soft; the right half was much distended with dark coagulated blood; the coagula extended into the venæ cavæ. The left side of the heart was empty, and the muscular walls were soft. The mitral valve admitted three fingers. The pulmonary valves were competent and healthy. The aorta was slightly incompetent and atheromatous.

The middle lobe of the right lung was healthy and crepitant throughout; the lower lobe was hepatized and non-crepitant. There was slight exudation of blood-stained material on section. There was no pleuritic effusion or adhesion at the base. The liver was natural in size, extremely soft, somewhat pale in color, and fatty on section. The spleen was about five inches long, very soft, and friable. The kidneys were also very soft, breaking down on removal. The cortex was congested, and the capsules very adherent. The bladder contained a small amount of urine. The œsophagus had a patch of congestion at the upper extremity, and at the cardiac end some congestion extending into the cardiac end of the stomach, which was also congested as far as its middle. The duodenum was congested at various points. At the lower end of the ileum there was a patch of redness of the size of the palm of the hand. No breach of surface existed. The pia mater was excessively congested. The brain on section appeared to have more bloody points than usual; the lateral ventricles contained some bloody serum. The substance of the brain was very soft.

Case XLII.—Poisoning by Calabar Beans (Physostigmine).

(The Lancet, August 20 and 27, 1864.)

§ 595. In the early part of August, 1864, nearly sixty children were poisoned in Liverpool by eating Calabar beans. A carter had thrown the rubbish, which he had been clearing from the hold of a vessel, upon some waste ground, close to a densely populated low neighborhood. The rubbish contained a great quantity of the beans, which were soon discovered by the poor half-starved children of the vicinity, and greedily eaten, though not very palatable, the taste being bitter and rough, and not unlike that of a horse-chestnut. They are enveloped in so hard a rind, that it was only by fixing them on a stone and breaking them with another that the contents could be got at. It was difficult to ascertain the exact number of beans eaten by each child. Two, three, and four seemed to have been the most frequent numbers; the smallest amount eaten was half a bean; one child was said to have eaten twelve, and yet recovered. Most likely the sickness, which in her case set in at once, saved her. Nearly all of the children were taken to the Southern Hospital, the names of forty-six being on the books. From ten to fifteen were taken to the Southern Dispensary. Their ages varied from two to ten years, the majority being under seven. The symptoms manifested themselves, as nearly as could be ascertained, in from half an hour to an hour and a half after ingesting the poison.

The state of the little sufferers on their arrival at the hospital, as described by Dr. Cameron, who was there at the time, and Dr. Wolliston and Mr. Evans, the house surgeons, was as follows: They were very pale and cold, staggered when they attempted to walk, quite prostrated with extremely feeble pulse and cold clammy skin, presenting all the appearances of the nervous system having received some great shock, amounting to complete collapse in the worst cases. Vomiting commenced early in most of them; purging occurred only in a few. In about two-thirds of them the pupils were contracted, and there was double vision. A few complained of pain in the stomach and bowels, but not at first. The treatment consisted in emetics of sulphate of zinc and mustard, the use of the stomach-pump, and afterwards, where the prostration continued, the administration of brandy. The children suffered greatly from thirst, and so drank readily the large quantities of

warm water offered to them. In the worst cases there was great drowsiness, whilst others complained of giddiness. Only one case proved fatal, that of a little boy of six years; he had eaten six of the beans, and lived only ten minutes after his admission to the hospital, and about two hours from the time of taking the nuts. He presented during that time all the symptoms above detailed in an aggravated form, except the sickness, which even the emetics and stomach pump failed to produce. He died by syncope.

At the *post-mortem* examination on the following day all the viscera were found to be healthy. The stomach and duodenum were filled with a pulpy substance, evidently the beans in a partially digested state; a few red spots dotted the mucous membrane of the stomach here and there, not more than might be caused by the mustard or sulphate of zinc. There was no reddening of the coats of the intestines; there had been purging which had removed all the feculent contents, leaving only a whitish semifluid emulsion of the beans. The heart contained blood and clot in each of the four cavities, indicating death by paralysis of the muscles of the heart. The bladder was perfectly empty and contracted. The brain and spinal marrow, and the other organs of the body were examined and found healthy. With the exception of the one case, all the children recovered sufficiently in a few hours to be able to be removed to their own homes.

The stomach and a portion of the intestines with their contents were removed by order of the coroner for analytical examination by Dr. Edwards. He reported that, "although in this instance circumstances favored the detection of the poison in the intestines after death, yet, in the case of a minimum fatal dose, or a prolonged purging before death, nothing could be found in the body to account for death, or to identify the poison."

A woman, aged thirty-eight, mother of one of the children made ill, went home, and deliberately ate half a bean "to see what it was like." During the evening she experienced some griping pain in the bowels, but passed a good night, and no alarming symptoms occurred till noon of the following day, when she felt trembling and faintness, and was admitted into the hospital. Her symptoms were staggering, double vision, sickness, feeble pulse, and pale countenance. She was so low that she was put to bed, and remained

under stimulant treatment for twenty-four hours before she was discharged.

There was nothing in the appearances at the *post-mortem* examination of the young boy, except the peculiar contents of the intestines, which could have guided a medical practitioner in his opinion as to the cause of death; and had the patient survived a little longer, and the residue of the poison been removed by purging, there would have been really nothing to have distinguished between death by this poison and death by cholera.

Case XLIII.—Poisoning by Strychnine.

(From reports of the trial of David L. Magoon.)

§ 596. Mrs. D. L. Magoon, of Raymond, N. H., a woman of advanced years, died suddenly in the afternoon of August 31st, 1874. Her husband, David L. Magoon, on that day took from his pocket a bottle of "essence of peppermint," poured some out into a tumbler, added hot water, and carried it to her in her bedroom where she lay sick. On account of its extreme bitterness she was unable to drink the whole, and he threw the remainder away. In less than an hour she was dead. She had frequently complained of cold and shivering, and at these times everything tasted bitter.

On her death nothing peculiar was noticed about the position of the body. The face wore a peaceful expression; the hands were clenched unusually tight; the body was so stiff that the clothes had to be cut off.

A little less than a year later (August 12th, 1875), the body was exhumed on account of suspicions of poisoning due to remarks let drop by the husband, and to the finding of a strychnine bottle in his pillow case; an examination was, therefore, made. The organs were found in a good state of preservation; the stomach was remarkably well preserved. The latter organ contained a half ounce of fluid; it was with its contents submitted to Dr. Hayes, of Boston, and Dr. E. S. Wood, of the Harvard Medical School, for chemical analysis. On September 3, 1875, the body was again exhumed, and a third of the liver and a fourth of the intestines removed and submitted for analysis. The stomach was found to contain 0.15 grain of strychnine; the portion of liver contained 0.23 grain; the poison was detected in the intestine but it was not estimated.

The prisoner confessed his guilt after a disagreement of the jury, and died, probably by suicide, before the time appointed for a second trial.

Case XLIV.—Poisoning by Strychnine.

(From the report of the trial of Edwin W. Major.)

§ 597. Ida Major, of Wilton Centre, N. H., a young woman eighteen years of age, the mother of four children, and about to bear a fifth, died on December 20th, 1874, under very suspicious circumstances. On that day between four and five o'clock she had eaten supper, after which she went to bed; her husband, Edwin W. Major, gave her some "medicine," which, he explained, would taste very bitter. Shortly before seven o'clock her husband called in two of the neighbors, who found her complaining of violent headache, blurring of the eyes, and dry mouth; she had had convulsions at 5.30, and shortly after their arrival she was again seized with the spasms. During the spasms she frothed at the mouth and uttered shrieks, but was perfectly conscious. The convulsions continued until 7.20 P. M. when she died. She spoke fifteen minutes before death. On laying out the body the feet were found to be so contracted that the stockings were with difficulty removed; the arms could not be straightened and the chemise had to be torn off.

The *autopsy* was performed two days afterwards. The face was lifelike and florid; body very stiff; feet arched and turned outwards. Flexor muscles of arms contracted like those of lower limbs, but not to such a marked degree. Brain normal; the veins were congested. Lungs, heart, kidneys, bladder, pancreas, gall-bladder, and intestines were normal. Pregnancy was far advanced; labor had apparently commenced. The external surface of the stomach was normal in appearance, except that the bloodvessels, especially the veins, were engorged. The mucous membrane was covered with mucus; the vessels, especially along the lesser curvature, were filled with blood. There was no loss of substance of the mucous membrane. The congestion was most marked upon the lesser curvature near the cardiac end and at the pylorus; the blood was normal in color, being red in the arteries and dark in the veins. The contents of the stomach amounted to four ounces, and contained potato starch, oil globules, vegetable fibre, amorphous matter, and

sarcinæ. The organ and its contents were sent to Dr. E. S. Wood, of the Harvard Medical School, for chemical analysis. Twenty days later the body was exhumed and the liver removed for analysis. The lower extremities were still as stiff as on the day of the autopsy; the arms were somewhat relaxed. The upper part of the spinal cord was exposed; it was normal, but the bloodvessels were engorged.

The chemical analysis detected 1.72 grains of strychnine in the contents of the stomach, 0.51 in the stomach, and 0.23 in the liver.

The guilt of the accused was proved at the trial, and he was subsequently executed.

CASE XLV:—POISONING BY CYANIDE OF POTASSIUM, AND DETECTION OF THE SAME IN THE BODY EIGHT DAYS AFTER DEATH.

(Vierteljahrsschrift für ger. Medicin und öffentl. Sanitätswesen, Bd. xxix. p. 49.)

§ 598. On April 19th, in the evening, von J. entertained a stranger at his inn; the guest not making any appearance on the following day, and the door of his room being locked, the police were summoned. They found the lodger lying dead in bed, and the physician who was called could not state whether he had been poisoned or had died from apoplexy. In front of the bed was found a small bottle apparently containing oil, which was preserved by the police.

The *autopsy*, which was performed nearly five days after death (April 24), gave the following results: The corpse, which was that of a man of about thirty years of age, strong, and well nourished, showed no other trace of decomposition than a slight green discoloration of the abdomen. The color of the anterior surface of the body was somewhat yellowish, while that of the posterior surface was light red with bluish red marbling. *Post-mortem* rigidity was not much marked in the limbs. The face was somewhat livid, and the eyelids closed; pupils dilated. The mouth was half open, and its mucous membrane was of a pale red color. The nails of the strongly contracted fingers were colored dark blue. On account of suspicions of poisoning the cavity of the abdomen was the first to be opened, whereupon a distinct and unmistakable odor of prussic acid was perceived. There was no abnormal fluid in the peritoneum.

The omentum was dry and traversed by distended venous bloodvessels. The stomach was much distended by gas, and its anterior

surface and that of the intestinal coils, which were pale reddish and pale brownish in color, were traversed by a network of numerous fine, highly injected bloodvessels. On the posterior upper surface of the stomach near the pylorus was a very even dark-colored expansion about as broad as the hand. The contents of the stomach consisted of about 100 grm. of a watery, turbid, bloody fluid. The mucous membrane was regularly brownish-red in color and very brawny, but could not be stripped off. It was swollen by imbibition of watery blood, and particularly so at the above-mentioned expansion where the color was deeper. Erosions were nowhere present; the bloodvessels were moderately filled. The stomach and its contents gave off a strong odor of prussic acid together with a decomposition smell. The small intestines, which exteriorly and internally resembled in color, etc., the stomach, contained a small amount of a red slimy fluid. The vessels of the mesentery and the inferior vena cava were overfilled with dark, almost black, blood resembling cherry-juice. The spleen was outwardly deep steel-blue; the cut surface dark red. It contained an enormous amount of blood, while its tissues were nearly normal in character. From the cut surfaces of the bluish-red colored kidneys and brownish-red liver a thin, almost black, blood flowed. The cut surfaces of the liver and spleen gave off a plainly perceptible odor of prussic acid.

The pericardium was exteriorly traversed by a network of fine over-filled bloodvessels, while the inner surface showed no remarkable injection. The pericardium contained about 20 grms. of a yellow serous fluid. The auricles and ventricles, the coronary vessels, the pulmonary arteries, the venous trunks of the thorax, and the veins of the neck were full of dark, fluid blood. The tissue of the heart was normal. After removal of the heart about 300 grms. of blood collected in the thoracic cavity. The larynx contained some red, foamy fluid, and its mucous membrane and that of the trachea were of a brownish color and somewhat injected. The much-expanded lungs were dark steel-blue in color; the lower and posterior portions were almost black. They felt elastic, and on pressure yielded a sound of crepitation. On the surface were found single bright colored blood extravasations, circular and elongated, varying in size from a pea to a lentil. The lung-tissue throughout contained air, and from the cut surfaces flowed dark blood and bloody foam on gentle pressure; a considerable amount flowed with-

out pressure. The bronchi contained considerable red, foamy liquid; their mucous membranes were moderately injected. The pleural sacs contained no fluid.

The dura mater was bluish, moderately moist, and somewhat glossy; many of the ramifications of the bloodvessels on the outer and inner surfaces were quite full. The superior longitudinal sinus contained about 5 grms. of somewhat lighter blood. The pia mater was normal excepting a strong injection of the ramifying bloodvessels. The cerebrum was of normal consistence; on its pale-red cut surfaces were seen innumerable bloody points and streaks. The brown-red colored venous plexuses of the empty ventricles were filled with blood. The cerebellum and other parts of the brain were also rich in blood; the bloodvessels at the base of the skull contained much fluid, almost black, blood. Directly after opening the skull the odor of prussic acid was unmistakable, and much more so on examination of the brain.

The bottle, which was mentioned near the beginning of this case, contained a few drops of watery, whitish, cloudy liquid, which smelt of ammonia and prussic acid. The presence of ammonia was shown by the white fumes produced on bringing a glass rod dipped in hydrochloric acid to the mouth of the vial; that of prussic acid was shown by the iron test. The further analysis proved the liquid to be a very concentrated solution of cyanide of potassium.

It was decided that the deceased had come to his death by probable poisoning by cyanide of potassium. In order to make perfectly sure it was determined to submit the organs to chemical analysis, and on account of the ease with which the poison decomposes the analysis could not long be delayed. On the 27th of April, at least eight and perhaps nine days after death, the analysis was begun. The stomach and intestines showed by the color and smell only moderate decomposition, but the odor of prussic acid was no longer perceptible. The distillate from the contents of the stomach and portions of the stomach itself gave the characteristic reactions of prussic acid. The analysis of the liver, spleen, kidneys, and blood yielded the same reactions. How much of the poison the deceased had taken could not be determined, and the amount found in the body was not quantitated. The microscopical and spectroscopical examination of the blood gave no important results.

The comparatively slight degree of decomposition five days after death, the marked *rigor mortis*, which by a number of authors is stated to be characteristic, the dark color and fluidity of the blood, with the universal congestion of all the organs, especially the heart and lungs, the injection of the mucous membranes of the larynx, trachea, and bronchi, and the foamy contents of the same, are peculiarly interesting, in that without the appearances of the stomach and the chemical results, the supposition of death from suffocation might have been entertained. It must, however, be further remarked that the dark-blue coloration of the lungs, spleen, and kidneys, and also fine network of injection of the pericardium have, as far as is known, not been met with in cases of suffocation. The most interesting point in the case is that the poison could be so easily detected at so long a period after death.

Case XLVI.—Prussic Acid Poisoning from Ferrocyanide of Potassium and Aqua Regia.

(Vierteljahrsschrift für gerichtl. Med. und öffentl. Sanitätswesen, Bd. xxvi. p. 57.)

§ 599. On Sunday April 9, 1876, Udo B. of U., an unmarried merchant, was found dead in his bed at five o'clock in the afternoon. On the table was a small vial containing a yellowish liquid and labelled hydrochloric acid. The deceased was clothed in his shirt, drawers, and socks, and was covered with the blanket. Since he was often heard to express his intention to kill himself, suicide was thought probable. A medico-legal examination was ordered, and on April 11th, forty hours after death, it was made.

On the bosom of the shirt and on the collar were numerous wine-colored and dark violet spots. *Rigor mortis* was present, particularly in the extremities and abdominal muscles. The facial expression was calm, the face pale. The skin at the right nostril was brownish-red, rough, and leathery. Beneath the lower lip to the right was a brownish-red spot 1½ ctm. long, where the skin was dry and leathery. The lips were brownish-red and dry. The inner surface of the lower lip was colored deep red on its right half. The epidermis at these places came away in shreds on being handled. The abdomen in the gastric region was retracted; lower down it was distended.

The organs of the abdomen were normal as regards position. The stomach was flaccid and dirty gray in color; on attempting to remove the organ a great hole was torn near the greater curvature; it contained about 60 grms. of dirty brown liquid. The inner surface of the stomach was colored dirty blackish-gray; towards the fundus uniformly red. The finer and coarser bloodvessels were very full; the blood in the veins was coagulated and of dry and smeary quality and black color. The finer veins appeared dotted on account of the breaking up of the coagula. The mucous membrane was soft and easily torn. The exterior of the duodenum was for a distance of 12 ctms. from its commencement uniformly red in color. The duodenal mucous membrane was softened; it was reddened in blotches; its bloodvessels were full. In the posterior wall of the duodenum near its commencement there was a place about a square centimeter in area and colored dark-red. In the centre of this spot there was a perforation as large as a pea.

The liver was enlarged, flabby, and pale red; its cut surface was uniformly reddish-yellow; it contained much blood. The kidneys were of normal size and flabby; the capsules somewhat opaque and easily detached; the surface after removal of the capsule was smooth. On section the cortex and medullary substance were found to be dark-red and filled with dark blood. The lungs contained much blood. The ventricles contained fluid blood of a dark color; the left auricle a clot dark in color, dry, friable, and circular. The posterior wall of the left auricle was of a slate color, dry, and hard.

The tongue was pale; on the dorsum dirty brownish-red, dry, and rough. The palate, pharynx, and uvula were white; the mucous membrane of the œsophagus dirty gray in color, soft, easily torn, and strongly injected; the blood in the injected vessels black, dry, and friable. The mucous membrane of the larynx and trachea much reddened; that of the arytenoid cartilages, and vocal cords easily detached.

The diploë contained much blood, the longitudinal sinus likewise, the blood dark and fluid. The arteries of the brain were empty.

The brown leathery spots on the nose and upper lip, the corrosion in the cavity of the mouth, the throat, larynx, œsophagus, and stomach, the softening of the stomach, the coagulation in the bloodvessels of the œsophagus and stomach, the formation of a peculiar dry clot in the left auricle, and the character of the wall of the

auricle next to the œsophagus are unmistakable evidences that a mineral acid had been taken.

It transpired that the deceased had vainly endeavored to procure some cyanide of potassium, that he had read works on chemistry, and that he had inquired about the technical use of ferrocyanide of potassium. He had been seen at his window an hour before the discovery of the corpse, which lay covered in a quiet position. These facts taken together made it strongly probable that death was not caused by mineral acid alone, but by a quickly acting poison.

The chemical examination of the liquid in the bottle showed it to consist of equal parts of hydrochloric and nitric acids. The amount contained in the bottle was 63 grm., and the capacity of the bottle was 100 grm. The contents of the stomach on standing deposited a brownish-red heavy sediment. On testing the fluid for hydrochloric acid with nitrate of silver a heavy cheesy precipitate was obtained; on performing the test for nitric acid with sulphuric acid and ferrous sulphate, instead of a brown color, a blue precipitate was obtained which was soluble in hydrate of potassium. A few drops of the fluid were then dried and ignited; the residue was alkaline and partially soluble; the soluble portion contained potassium and sodium, the insoluble was oxide of iron. Some of the fluid diluted with alcohol was distilled and the distillate received in a weak solution of nitrate of silver; the resulting precipitate contained cyanide of silver. When almost completely distilled, the smell of prussic acid was perceived. The residue in the retort contained cyanide of iron. The production of Prussian blue on addition of iron salts to the fluid from the stomach, the potassium and iron in the residue after ignition, the forming of hydrocyanic acid on distillation and of cyanide of iron in the residue showed that either decomposition products of ferrocyanide of potassium were present, or the salt itself, or both. In order to determine this point the fluid was tested for free prussic acid. For this purpose a part of the fluid was mixed with bicarbonate of potassium and distilled in an atmosphere of carbon dioxide. Free prussic acid was discovered in the distillate, and the ferrocyanide of potassium in the residue.

The presence of the salt and its decomposition products, and of the acids shows that the salt and acids had both been employed. Contact of these had formed prussic acid. The deceased had un-

doubtedly taken the salt in solution; the swallowing of the acids was associated with development of the gas and coughing, whence the eschars on the face and the spots on the shirt and collar. Death must have resulted quickly.

Case XLVII.—Poisoning by Oil of Bitter Almonds (Prussic Acid).

(Maschka's Handbuch der gerichtl. Med., vol. ii. p. 325.)

§ 600. A healthy strong man of 24 years, drank from a bottle containing one and a half pounds of oil of bitter almonds, and died in about five minutes. The *autopsy* was performed in twenty-four hours. Cadaverous smell was hardly perceptible. Rigidity was marked in the extremities. The pupils were moderately dilated. On both breasts and upper arm, in the left groin, and over the whole posterior surface were diffuse, bluish-red blotches (suggillations). The eyelids and lips were closed; the jaws were firmly shut. The sinuses and vessels of the dura mater were filled with black fluid blood. The arachnoid was somewhat opaque. Vessels of the pia mater strongly injected. The cerebrum was rich in blood; the ventricles contained about 15 grms. of serum. The plexus was not immoderately full. The cerebellum and medulla oblongata contained considerable blood. The whole brain was hard. The lungs were elastic and normal; they contained considerable dark cherry-red, fluid, foamy blood. The heart was normal; the right ventricle contained about 15 grms. of dark fluid blood; the left was somewhat enlarged and nearly empty. The great vessels contained but a small amount of blood. There were no serous exudations in the pericardium or pleural cavities. The stomach and intestines were exteriorly bluish-red; otherwise normal. The kidneys were normal and much engorged. The liver and spleen were moderately rich in blood. The large bloodvessels of the abdomen moderately filled. There was no exudation into the peritoneal cavity. From all the large cavities of the body, and from the ventricles of the brain, there arose such an intolerable sweetish smell that several persons present at the examination vomited, and all present suffered from headache and weakness, which persisted on the following day.

The chemical examination was performed thirty-six hours afterwards, and 0.480 grm. of anhydrous prussic acid obtained.

Case XLVIII.—Poisoning by Nitrobenzol. Death in Nine and a half Hours.

(Maschka's Handbuch der gerichtl. Med., vol. ii. p. 335.)

§ 601. Three young workmen prepared a bottle of "Schnaps," which was one part alcohol, two parts water, and (according to their story) but twenty drops of nitrobenzol. The latter was added to impart a taste of almonds to the mixture. M. G., 19 years old, drank about a third of the whole amount between seven and eight o'clock in the morning. Each of the others took four or five swallows, and three work-girls took each a taste. Of these persons but one suffered violent abdominal pain; the others remained perfectly well. Shortly after 8 o'clock a blueness was noticed in M. G.'s face; at 9.30 he vomited, and had pain in the abdomen. At ten he lost consciousness, fell down, and had twitching of the arms and hands. At 11.30 he regained consciousness, took some milk and an emetic, vomited repeatedly, and was carried to the hospital. The skin was cold; pulse 100, weak, and irregular; respiration slow and superficial; complete loss of consciousness; trismus, stiffness of the neck, fibrillary twitchings in the masseters, spasmodic flexion of the arms. The skin was grayish-blue (too gray for ordinary cyanosis), pupils dilated and almost incapable of reacting, lids closed. The eyes rolled very slowly from out inwards. A strong smell resembling that of bitter almonds came from the mouth. Involuntary dejections. The stomach was washed out, by means of the stomach-pump, with about five quarts of water. The water on its return was milky, and without a distinct smell. He improved somewhat after being bled at 2 P.M. (60.0 grms.); answered now and then, and could swallow. The blood which flowed from the vein was dark and thin. Under the microscope the blood-corpuscles were rather brighter. After 3 P.M. somnolence; respiration irregular. Death occurred at 5.30 P.M.

The *autopsy* was performed forty hours after death. Rigor mortis marked; numerous red blotches (suggillations) over the whole body. The dependent parts bluish-red; face dirty grayish-yellow. The vessels of the skull were very full; the cortical substance of the brain was comparatively anæmic, the medullary substance hyperæmic; the pons and medulla were pale. The blood in the jugular veins was fluid and brownish-red. Marked catarrh of

the whole throat; the mucous membrane was brownish-red; there was a bluish-red injection of the mucous membrane of the larynx and trachea. Mucous membrane of the epiglottis much swollen. The lungs were full of brownish and black blood. Pericardium empty; right auricle much dilated and contained soft, dark, brownish-red coagula; left ventricle contained but little blood; heart substance dry, hard, and somewhat streaked with yellow. Liver was markedly reddened and contained much dark fluid blood. On incision a distinct bitter almond smell was perceptible. The spleen was firm and congested. The kidneys showed marked passive hyperæmia; considerable cloudy urine exuded from the pyramids on pressure. The stomach was moderately distended; its mucous membrane was covered with tenacious grayish-yellow mucus, which had the same smell as the liver. The mucous membrane was very thick; some imbibition of blood but no hemorrhages. The pancreas was much congested. The serous coat of the intestine had imbibed some blood; the mucous membrane was diffusely reddened and swollen; the single follicles swollen. The bladder was full of clear urine which had no particular odor.

Case XLIX.—Poisoning by Aconitine.

(The Lancet, March 18, 1882, p. 455.)

§ 602. On Wednesday, March 18th, 1882, at the Central Criminal Court, before Mr. Justice Hawkins, George Henry Lamson, aged twenty-nine, surgeon, was indicted for the wilful murder, on December 3d, 1881, of his brother-in-law, Percy Malcolm John, aged nineteen. Percy Malcolm John, who was a cripple with curvature of the spine and paraplegia, had property to the extent of £3000, half of which at his death would revert to the prisoner's wife. John had, for three years prior to his death, been at Blenheim House School, at Wimbledon, kept by Mr. W. H. Bedbrook. On December 3d, 1881, John was, with the exception of his paralysis, in good general health, and on that day had taken his meals, breakfast, dinner, and tea, in company with Mr. Bedbrook among others. On Saturday, December 3d, Lamson called on John at Blenheim House at 6.55 P. M., and their interview took place in the dining-room of Blenheim House in the presence of Mr. Bedbrook. Mr. Bedbrook offered Lamson some wine, which he accepted, and

Lamson then asked for some sugar, as the wine (sherry) was rather strong, and he said "sugar would destroy the alcoholic effects." A basin of white sugar was brought, and Lamson put some of it into his sherry. Lamson then produced a Dundee cake and some sweets, of which all three partook. At 7.15 P.M. Lamson produced a box of gelatine capsules from his pocket, and said "Oh, by the way, Mr. Bedbrook, when I was in America I thought of you and your boys. I thought what excellent things these capsules would be for your boys to take nauseous medicines in." Lamson then gave a capsule to Mr. Bedbrook, and filling another with sugar handed it to John and said, "Here, Percy, you are a swell pill-taker; take this, and show Mr. Bedbrook how easily it may be swallowed." John swallowed the capsule; the prisoner soon said, "I must be going," and at 7.21 P.M. left the house. A little after 8 P.M. John complained of heartburn, and soon after said, "I feel as I felt after my brother-in-law had given me a quinine powder at Shanklin." He was carried up to his bed-room, and about 9 P.M. was found in great pain and vomiting. He complained that "his throat appeared to be closing, and the skin of his face felt drawn up." At 11.30 P.M. he died. John was treated by linseed poultice to the abdomen, white of egg beaten up with water, and two hypodermic injections of morphine of one-sixth of a grain and a quarter of a grain respectively.

The *post-mortem* examination of John's body was made on December 6, 1881, by Dr. Little and Mr. Berry of Wimbledon, and Mr. Bond of the Westminster Hospital. The spinal disease was found to be old and inactive. There were some old adhesions of the lung. The lips and tongue were pale. The cerebral meninges, liver, kidneys, spleen, and the mucous membrane of the stomach were much congested. The mouth and lips were pale. On the under surface of the large end of the stomach were six or eight yellowish-gray patches, a little raised, about the size of a small bean, and towards the smaller end were two or three similar ones. The heart was almost empty, but healthy. The lungs were congested, the posterior parts very much so.

The analysis of the viscera, vomit, and the articles of which John might have partaken, were conducted by Dr. Stevenson of Guy's Hospital, in conjunction with Dr. Duforé of the Westminster Hos-

pital, and the results obtained by the one were verified by the other. The results were briefly as follows :—

1. Portions of the liver, spleen, and kidneys treated by Stas's process gave evidence of slight traces of morphine, and the alkaloidal extract when placed upon the tongue produced the numb tingling sensation which is characteristic of aconite, and which, for the sake of brevity, we will call aconitism.

2. The contents of the stomach similarly treated produced aconitism.

3. The stomach itself similarly treated showed the presence of an alkaloid, but the extract failed to produce aconitism.

4. The urine gave evidence of morphine and aconitine, and the extract obtained from an ounce of the urine killed a mouse in thirty minutes, when injected under its skin ; the symptoms being exactly similar to those produced by injecting a minute quantity of a solution of Morson's aconitine.

5. A mixture of the extract from 1, 2, and 3, when injected under the skin of a mouse, killed it with similar symptoms in twenty-two minutes.

6. The vomit was found to contain muscle, starch, onion, vegetable pulp (probably apple), raisins, candied peel, and pine-apple essence. It contained neither morphine nor quinine, but very marked aconitism, which lasted for over six hours, was produced by a minute quantity of the extract, and when injected under the skin of a mouse, it produced powerful symptoms in two and a half minutes, and killed it in a quarter of an hour.

Dr. Stevenson considered that the vomit contained as much as a quarter of a grain of aconitine.

In the beginning of 1881, Mr. Bedbrook received a letter from Lamson, who was then in America, and a box containing a dozen pills. The letter requested Bedbrook to give the pills to John, as Lamson had heard of cases in America similar to that of John being benefited by the pills in question. The deceased had taken one of the pills, and the next morning complained of being very unwell, and said he should take no more of the pills. On August 28th Lamson bought three grains of sulphate of atropine and one grain of aconitine of Mr. Albert Smith, a druggist of Ventnor. On August 29th, Percy John, who was staying in the house of Mrs.

Jolliffe at Ventnor, was taken ill with diarrhœa and prostration, and a feeling "as if he were paralyzed all over."

Lamson was living with his father in Ventnor between August 6th and October 23d, 1881, and was in the habit of going to the house where Percy John was living, and had actually called upon him on the 29th. On October 13th he bought twelve quinine powders (containing a grain and a half each) of Mr. Littlefield, a druggist of Ventnor. John at the time of his death was taking quinine powders, which had been supplied to him by Lamson. On November 11th, Lamson had bought of Bell & Co. half an ounce of a mixed solution of morphine and atropine, containing forty grains of morphine and one grain of atropine, and on the 16th of November he bought the same amount of a similar solution. On November 16th he also asked for five grains of digitaline, which were not given to him, because the sample in stock was not thought to be good, and on the 20th of November he asked for one grain of aconitine, with which the assistant in the shop refused to serve him. On November 24th he purchased two grains of aconitine of Messrs. Allen and Hanbury.

Among the effects of the deceased were found twenty quinine powders numbered from 1 to 20. Numbers 1, 2, 3, 4, 5, and 6 were in larger papers than the rest, and the powders were nearly uniform in weight, containing about a grain and a half of quinine each. Nos. 7 to 20, inclusive, were wrapped in smaller papers, and varied in weight from six-tenths of a grain to one and eight-tenths of a grain. Nos. 16, 17, and 19 differed in appearance from the rest, having an admixture of a pale, fawn-colored substance. They all contained aconitine. No. 16 contained 0.83 grain of aconitine and 0.96 grain of quinine. One-fiftieth of a grain of the aconitine contained in this powder killed a mouse in six minutes and a half. One of the pills which had been sent by Lamson from America was found to contain nearly half a grain of aconitine. Some of it injected into the back of a mouse killed the animal in less than five minutes, and the aconitism produced by a small quantity on the tongues and throats of the experimenters lasted for over seven hours.

Witnesses were called to prove that Lamson would benefit pecuniarily by the death of deceased, and that at the time of John's death he was a bankrupt without a penny in the world. No wit-

nesses were called for the defence. The counsel for the defence was unable to bring forward any solid arguments in refutation of the evidence and the opinions of the experts, and the jury, after deliberating thirty-five minutes, returned a verdict of guilty. Sentence of death was passed in the usual form.

Case L.—Poisoning by Lobelia.

(New York Journal of Medicine, Nov. 1844.)

§ 603. We have selected the following case of lobelia poisoning on account of its brevity. "The defendant, Riley Drake, was charged with having produced the death of Miss Lucina Frost, by 'the grossly ignorant, careless, and unskilful administration of lobelia to her.' Dr. A. H. Brownson, sworn, says: 'He was called to visit Miss Lucina Frost on the 11th of September, 1843. Found her laboring under febrile excitement. Considered her complaint a case of bilious remittent fever. Continued to attend her as a physician until Thursday, the 28th of September, when she was convalescent, and had been for several days. Patient had some appetite. Witness also testifies that lobelia is a violent emetic, which, if taken in large doses and not discharged from the stomach, will act as a fatal poison. Thinks an emetic, of any kind, would have been very improper for the deceased when he last saw her.' Dr. Brownson's testimony was corroborated in every point by that of Dr. P. R. Brooks, who was called to see the patient two or three times in consultation with Dr. Brownson. Nancy Sutcliffe, sworn, says: 'She has known the deceased about eight years. Was there about a week before her death, and up to the time of her death. Saw Riley Drake there on Sabbath (September 24th), when the patient asked him if he thought she was getting better, and he said not. He (Drake) had something to say to her every time he was there. On Thursday, she (deceased) said to him that Brownson told her that her fever had turned, and that she was better. Drake said that Brownson was mistaken, that her fever had not turned, and that she would never get well under Brownson's treatment. Deceased asked Drake if he could help her, and he said he could. Her father would not give his consent to have Drake. Friday night, September 27th, Drake came in, and she (deceased) told him if he thought he could help her, she wished he would. He

gave her some medicine, to prepare her stomach for an emetic. The next morning he came in and gave her an emetic. He gave her small seeds steeped in water. Witness saw the seeds. She vomited twice. After vomiting, appeared to feel better. Probably a teaspoonful of seeds was given. Nothing else was given. Drake did not come again till afternoon. Patient appeared better till noon, when she became distressed for breath. Seemed filled up on her stomach, and continued so until Drake came in the afternoon. He ordered her ginger-tea, which was made, and *three teacups full* given. He then steeped *lobelia* in a teacup, and gave her that, *seed and all.* Teacup was about half full. She drank about half of it; and then wanted some physic. He gave her some. She then said she thought she ought to take more. She then took the *rest* of the *lobelia.* He then gave her some red stuff, and then some *nerve-root tea.* She died in half an hour after the lobelia was first administered.' The evidence was confirmed in every essential particular by that of Olive Fairchild and Charles Gearnsey, with the additional fact that the deceased was severely *convulsed* after taking the emetic on Saturday, just previous to her death. Dr. Stephen D. Hand, sworn: 'Witness is a practising physician, residing in Binghamton, Broome County, New York; says he was called to examine the body of Miss Lucina Frost, on Sunday, September 31st, 1843. Was informed that she had died on the Saturday previous, September 30th. External appearances of the body natural. Examined the stomach and other internal organs. Found a *tablespoonful* of *lobelia seeds* in the *stomach.* Mucous membrane of stomach softened and much inflamed. Intestines also considerably inflamed. The heart and other organs healthy. Witness had no doubt but the lobelia contained in the stomach killed the patient. Thinks there was enough there to destroy the life of any person, unless thrown off. All parts of the lobelia plant contain the same properties. Thinks, from the description given of the patient by Drs. Brownson and Brooks, that an *emetic* of *any kind* would have been very *improper* under the circumstances.' The testimony of Dr. Hand was corroborated by that of Drs. West, Brooks, and Cook, who were also present at the *post-mortem* examination. Drs. Thomas Jackson and N. S. Davis, both residents of Binghamton, were also sworn in regard to the properties of lobelia, which they

stated 'to be an active narcotico-acrid poison, when taken in large doses.'

"This closed the evidence on the part of the prosecution, when the defendant called Charles Gearnsey, Haman Gearnsey, Samuel Martin, Harry Martin, Alvah Parsons, Nathaniel Broughton, Charles Elliot, Sherlock Black, Rhodia Gearnsey, George Doolittle, Uriah Doolittle, James Russel; all of whom testified that they were personally acquainted with Riley Drake, and considered him a skilful physician of the *Thomsonian* school. The defendant then called the following *Thomsonian* and *Botanic* doctors to prove the qualities of the lobelia. Folkert Van Vleck, sworn: 'Lives in Hamilton, Madison Co. Is a physician of the Thomsonian order. Twelve years' practice. Have had as many patients as I could attend to. Have used lobelia in almost every case of inflammation and fever, and usually with good success. In case of remittent fever, should use lobelia as an emetic, and afterwards in broken doses. My patients have always recovered. Lobelia produces an emetic effect on the healthy stomach. It will not produce inflammation under any circumstances. Did not hear the testimony of Dr. Brownson. Heard Dr. Hand's. Thinks that lobelia would not produce the effect described by Hand.' Cross-examined: 'Says he lost only one *fever patient*. Has lost some with consumption. Gives lobelia in consumption. Has been present at a *post-mortem* examination. Thinks that a single dose of lobelia could not be taken so as to produce death; it might be by repeating the dose. Cannot tell whether tobacco is a *poison*, or not. Thinks cicuta would produce nausea. Has never studied surgery, anatomy, etc., and does not deem it necessary.' William Rose, sworn: 'Is a botanic doctor. Has used lobelia for thirty years in all cases where emetics were needed. In 1825, had one hundred cases of scarlet fever, in which he used lobelia, and did not lose a patient. Has no knowledge of its possessing poisonous qualities. Understands by *narcotic* poison, that which stops the blood. Don't know how opium produces death. Thinks arsenic would produce death quicker than cicuta.' Jabez Jeffers, also a botanic physician, gave similar testimony to that of Dr. Rose. Thomas W. Griffin, sworn, says: 'He is a Thomsonian physician; has practised eighteen years as such. Says he uses three articles, viz: lobelia, cayenne pepper, and barbary bark, in all cases, and in all *stages* of the disease, and under all *circum-*

stances, and *always* with *good* effect. Thinks that lobelia is not a poison.' The testimony being closed, the case was ably argued by Lieut. Gov. Dickinson, for the defendant, and by the Hon. Joshua A. Spencer, in behalf of the people, when it was submitted to the jury by Judge Morrell. The jury, after an absence of a few hours, returned with a verdict of guilty. Judgment was, however, suspended until the next term of court."

Case LI.—Poisoning by Lobelia—Perforation of the Stomach. Death.

(British Medical Journal, 1882, ii. p. 23.)

§ 604. A fatal case of poisoning by lobelia inflata occurred recently at Carlisle. From the evidence, it appears that the deceased, John Richardson, a joiner, had been for some months suffering from heart disease, and had frequently complained of a severe burning pain in the stomach. He was a man of intemperate habits, and an enormous eater. On the day of his decease, he was not so well as he had been, and said he had taken an emetic which had not acted. He was asked of what kind, when he replied "one of Dr. Coffin's prescription, and there is lobelia in it." There was a book on the table, and pointing to it, he said he had been recommended to try that system when in Lancashire. The *post-mortem* examination was made by Dr. Hair and Dr. Carlyle twelve hours after death. The pupils were slightly dilated. The lower jaw was firmly fixed. The abdomen was greatly distended. A quantity of fluid, probably about two pints, having a milky appearance, was found in the peritoneal cavity. The intestines were in places much congested. The stomach was next examined, and an aperture about the size of a goose-quill was found in the lesser curvature; and it was probably through this aperture that the fluid found in the peritoneal cavity had escaped. In the stomach itself there was about half a pint of yeasty looking fluid, in which, on careful examination, lobelia seeds and pieces of cayenne could be detected. The lungs were healthy, but the heart was fatty. The head was not examined, as sufficient evidence to account for death was afforded by the perforation of the stomach. The jury returned a verdict that the deceased died of perforation of the stomach, induced by the action of an emetic containing lobelia which he had injudiciously taken.

Death from lobelia inflata administered by Coffinites are of frequent occurrence. Their dictum is that "Heat is life, and the want of heat disease and death." In accordance with their principles, their drugs are lobelia and cayenne. It is asserted by them that lobelia cannot kill; but it has been shown over and over again that, when not rejected, it acts as a powerful toxic agent, and kills with the greatest certainty.

Case LII.— Poisoning by Savin.

(The Lancet, 1845, i. p. 677.)

§ 605. A young woman, M. A. N., aged 21, had enjoyed remarkably good health up to twelve hours before her death. On the night of Tuesday, April 22d, she supped with her lover, by whom she was far advanced in the family way. She was then cheerful and hearty, but about three o'clock in the morning she awoke her mother, with whom she slept, and complained of a violent pain in her stomach. She was very sick, and her mother, believing that it arose from a too hearty meal, got up and gave her some brandy and water. The sickness, however, was not diminished, and she subsequently became insensible. About eight o'clock her mother sent for the parish surgeon, whose assistant came to see her; he prescribed some ordinary medicines, which, from her insensibility, they were unable to administer. Fearing that there might be some danger, the mother sent for another surgeon, Mr. Newth, who got to her about eleven o'clock. She was lying on her back, perfectly insensible, breathing laboriously, and with stertor; she was foaming at the mouth, her countenance was turgid; the eyes shut, with the pupils much contracted; her limbs were also convulsed, and the mother told him that she thought she had been suddenly taken in labor. He therefore made an examination *per vaginam*, but found that the *os uteri* was only slightly dilated; the membranes, however, were ruptured, and the uterus was still acting vigorously, each contraction being accompanied by a convulsive tremor. Believing it to be a case of puerperal convulsions, he bled her, and applied cold to the head, but this did not in any degree restore her. As the *os uteri* became more fully dilated, he found that the head of the child presented, and that it was gradually descending. About three o'clock, while the labor was thus progressing, she

uttered a low moan, and in a few minutes ceased to breathe. Mr. Newth immediately sent for the forceps, and delivered her of a male child, which was dead; as far as he could judge from its appearance and the mother's report, deceased was somewhere between the term of the seventh and eighth month of pregnancy.

Twenty-four hours after death, Mr. Newth made an examination of her body. It bore no marks of violence, was well formed, and rather robust. On opening the head, he found that the vessels upon the surface of the brain were much gorged with black fluid blood, and, in proceeding with sections of the cerebral substance, discovered that the gray matter was infiltrated in various parts by the same dark-looking blood; this he especially noticed in the anterior part of the corpus striatum. The choroid plexus was also gorged; the ventricles were empty. The lungs were a little congested on the dependent parts, but otherwise healthy. He did not examine the heart, nor did he pay attention to any of the abdominal viscera besides the stomach. This, on being opened, appeared rather paler than usual, excepting one or two spots, which were red, as if blood had been effused into the mucous tissue. It contained about four ounces of fluid, which he secured, and took to H. Letheby, Esq., M. B., for examination. This fluid had a dark brownish-green color, smelt of digesting matter, and exhibited an acid reaction. Oil of savin was obtained from the fluid by distillation. A little of the sediment of the original bottle was afterwards examined under the microscope and compared with the powder of the dried plant; their structure appeared to be perfectly identical. The rest of the fluid was filtered, and the residue digested with ether, by which means there was obtained a green solution, containing resin and the green coloring matter chlorophyll.

Case LIII.—A Case of Digitalis Poisoning.

(Vierteljahrsschrift für gerichtl. Medicin und öffentl. Sanitätswesen. Bd. xxiv. p. 278.)

§ 606. Two young German conscripts, who had on examination been declared healthy and fit for military duty, went to a "liberator" to get the means of avoiding the same. He explained to them that healthy recruits, who could not be exempt, but who wanted to be, must be sick; and gave them a medicine which would bring on a

diseased condition—an abdominal affection—in consequence of which exemption would be gained without any permanent injury to health. Each obtained a box containing one hundred pills, with directions to take four pills twice daily. A short time before entrance to the army, perhaps eight or ten days, they began to make use of the pills. They entered the army on December 12th, 1874, and three days afterwards one of the two, *G. von K.*, announced himself as sick, and was treated a few days in barracks, then on the 18th admitted to the hospital. After three weeks' treatment he very suddenly and unexpectedly died. Shortly before his death the officer, under whose command the two recruits belonged, reported that a rumor had spread that the two were employing some means to appear sick in order to avoid duty. In consequence of this rumor, and on account of the inexplicable death, a legal examination of the body was made. The autopsy revealed no sufficient cause of death, and the suspicion of poisoning was strengthened. The finding of a box containing thirteen pills in the stocking of the dead recruit strengthened the suspicion, and the other recruit C. K. was arrested, since he was suspected of using the same means to attain the same end as G. von K. had in view. The recruit C. K., directly after entrance into the army, was declared by the staff-surgeon to be temporarily unfit for duty on account of deficient circumference of the chest, and this report had been confirmed by the commission. In addition to the above defect the sick appearance of the soldier was particularly noticeable. The recruit himself had no certain knowledge as to the real reason of his exemption, and had continued the use of the pills; yet on account of the appearance of serious symptoms he had for a day discontinued their use. On receipt of the news that his friend was dead, he threw away his pills out of fright. This he admitted at the examination, when, after a firm denial, he was pressed to an open confession. He admitted that he had used the pills, and described the effects to be exactly like the symptoms of the deceased.

The *autopsy* was performed twenty-two hours after death, and was negative in its results; the organs and tissues were in a state of health. There were no remarkable external appearances. The body was well nourished; the panniculus adiposus was normal; the muscles were pale. The blood was thinly fluid, nowhere coagulated, and dark cherry-red in color. The organs of the

chest were perfectly normal; the lungs and heart floated when placed together in water. The lungs were nowhere adherent, and contained air throughout; the lower and posterior portions contained more blood than the upper—hypostatic congestion. In the lower portion of the trachea was some foamy liquid. The heart was well covered with fat; the heart substance and valves were nowhere abnormal. The right half of the heart was filled with dark fluid blood; the left was empty. The abdominal organs showed nothing remarkable; the omentum was normal. The contents of the stomach consisted of a greenish-yellow turbid fluid with single white flakes, and amounted to about 200 ccm. The mucous membrane of the stomach showed circumscribed areas of injection; in these areas the bloodvessels were much developed and enlarged; here and there were small ecchymoses. The contents of the duodenum consisted of a thick, almost mushy, mass, which was much mixed with mucus; the color was cloudy yellow. The greenish color of the stomach contents was here less observable. The mucous membrane of the duodenum, and of the rest of the small intestines, showed the same hyperæmic areas as the stomach. The contents beyond the duodenum were of a more solid consistence, and the lower portion of the small intestine and the whole large intestine contained feces of a yellow color and good consistence. In the small intestine several dead ascarides were found. Nothing remarkable was observed in the liver; yet the color of the contents of the gall-bladder was dirty brownish. The spleen was perfectly normal in every way. Nothing abnormal could be discovered in the kidneys, ureters, or bladder. The latter was moderately filled with clear urine. The dura mater was normal; the pia mater at the sagittal suture showed opacities and patches. The brain was somewhat anæmic, and the sinuses contained but little blood. For chemical analysis were reserved the stomach and contents, a portion of the œsophagus, a piece of the liver, a portion of the duodenum, contents of duodenum, and blood from the heart; also the pills found in the stocking. The stomach, part of the œsophagus, and portions of the liver and duodenum were analyzed together, and yielded a residue of digitalis. The contents of the stomach gave the reactions of digitalis, and several very small green particles found in the stomach were reserved for the microscope. Digitalis was also present in the contents of the duodenum. Analysis of the blood gave negative

results. Chemical and microscopical analysis of the pills showed them to consist of digitalis powder; the particles from the stomach were the same. The physiological test was also carried out, and with success.

The symptoms, which the deceased had suffered, are described in the report of the examination. He complained chiefly of loss of appetite, nausea, constipation, headache, and dizziness. On examination, the ill appearance and a strong smell from the mouth were very noticeable; the tongue was heavily coated; pressure over the stomach occasioned so much complaint of pain that there arose a suspicion of exaggeration. There was no fever. The pulse was slowed to 56 per minute. There were no other symptoms. In spite of appropriate diet and medicine his condition did not improve. On December 21st the pulse was still slower, 52 per minute. On December 26th he vomited, and on emptying the vessel greenish slimy masses had been noticed; he was, therefore, given a vessel in which to preserve the vomit, in case of its happening again, for examination. This vessel was not used, and no vomiting had been noticed. His sick condition remained the same till the end of the month, and he lost more or less strength. He complained of darkness of vision, noises in the ears, and great weakness. Examination of the pupils showed that they reacted well and were of equal size. On January 9th he complained of swelling in the neck and difficult deglutition. Examination showed no reason for the swelling, and the physician wished to make a further examination of the abdomen with the patient in an upright position, but the patient could not stand on account of faintness, which disappeared after a few minutes of rest. In the course of this day and the preceding he complained of hiccough. Early in the afternoon he wanted to go to stool; he had hardly arisen from his bed when he sank down, and in a few minutes was dead.

Case LIV.—Poisoning by Digitaline.

(Tardieu, l'Empoisonnement, 1875, p. 809. Annales d'Hygiène Publique, 1864, vol. ii. p. 105.)

§ 607. On November 17, 1863, Madame de Pauw died under circumstances which excited suspicions of poisoning. These suspicions were directed against Dr. Couty de la Pommerais, who was

pecuniarily interested in her death. The body was exhumed and examined thirteen days after death. It was in a remarkably good state of preservation; it was well nourished and plump, and showed no evidence of disease. There were no marks of violence. The skull was intact; brain normal. Nothing remarkable about the mouth and throat. Lungs perfectly healthy; no hyperæmia, no inflammatory changes, no tubercles. Heart normal, and contained considerable half-coagulated blood. No fluid in the peritoneal cavity. Liver, spleen, and kidneys normal. The stomach and intestine showed in places several suffusions of blood and hyperæmic points; there was nothing pointing towards acute or chronic inflammation. The sexual organs showed no evidence of disease or violence. In the uterus was a fœtus of seven or eight weeks.

The conclusions drawn from the autopsy were that there were no pathological changes to account for death from natural causes, and that the absence of any characteristic changes and the appearances in the digestive tract furnished reasonable grounds for suspecting death from poison, which could be proved only by chemical analysis. The stomach, intestine, lungs, heart, liver, spleen, and kidneys were reserved for analysis. The floor of the sleeping-room of the deceased was also examined. Various substances found in the possession of the accused, and his books and papers were investigated.

Among 900 chemical and pharmaceutical preparations (mostly homœopathic remedies) was a large quantity of deadly poisons out of all proportion to the ordinary needs of a physician and more especially of a homœopath. There were four vials of white arsenic, three of corrosive sublimate, besides sulphate of copper, black hellebore, to the amount of 125 grams, powdered nux vomica 250 grams, powdered stramonium 250 grams, powdered aconite 350 grams, powdered belladonna 250 grams, powdered digitalis 125 grams, prussic acid, muriate of morphine, strychnine, digitaline, etc. etc. It was shown that the accused had on October 4, 1861, purchased 50 centigrams of digitaline, on June 11, 1863, 1 gram, June 19th, 2 grams; total 3.50 grams, of which but 0.150 (or less than one-twentieth of the whole amount) was found. So large an amount of this poison to be used in that space of time is out of all proportion to the ordinary needs of not only a physician, but even of a very busy apothecary, since the poison is of such a dangerous character that it can be given only in very small doses

(one milligram or less); 3.50 grams are, therefore, at least 3500 doses.

Witnesses testified that the deceased had gone about her duties and had been in good health up to the evening before her death. The first symptoms appeared in the night, and were repeated violent vomiting and rapidly increasing weakness. The physician found her pale, very agitated, bathed in cold perspiration; she complained of intolerable headache; the pulse was irregular, intermittent, and hardly perceptible; the heart's action was tumultuous, irregular, and ceased from time to time.

On undertaking the chemical analysis, it was noticed that the stomach and intestine were not at all decomposed and gave off no odor; the other organs were undergoing decomposition. The latter were analyzed for mineral poisons, but with a negative result. The stomach and intestine were examined for organic poison. Various extracts of these organs were made and employed for physiological tests.

Scrapings from the floor where the deceased had vomited were analyzed; no mineral poisons were detected, and the alcoholic extract was further employed with the others above mentioned.

The first experiment was performed with the last-mentioned extract. A strong medium-sized dog was taken; incisions were made in the integument of the thigh and 5 grams of the extract were introduced into the connective tissue; the wounds were then closed. Before the operation the heart-beats numbered 110 per minute. The dog remained apparently well during the first 45 minutes. He then lay down and began to lick the wounds. At the end of two hours and twenty-five minutes he vomited three times, the vomitus being mucus and a little bile. He then lay down again very weak and anxious. His heart's pulsations were 94 per minute, irregular and intermittent, now ceasing, then tumultuous. Respiration quickened and somewhat intermittent. One hour later repeated vomiting; pulse 74. Seven hours from the beginning of the experiment the dog was quite exhausted; on being urged to move he would retch and vomit; pulse 68, and still more irregular and intermittent. Twelve hours later he was quite cold, but in full possession of consciousness; pulse 40, weak and very irregular. Respiration superficial, frequent, intermittent. Three hours later the dog died almost without any agony and without any coma.

Death occurred in 22 hours from the beginning of the experiment. On *autopsy* the organs were found to be normal; black, thick, partially coagulated blood was contained in the auricles and ventricles. The auricles were dilated, the ventricles contracted. Apex and vicinity bright red. Two grams of the extract mixed with water were introduced by means of a funnel into the stomach of a rabbit. The pulsations of the heart were slowed, irregular, and intermittent. Respiration embarrassed, and shortly before death intermittent. In two hours and five minutes the pulse was 41. Death occurred in two hours and forty-five minutes. On *autopsy* the organs, excepting the heart, were found normal. The ventricles were contracted, auricles dilated; apex bright red. The heart wall showed several abnormal small raised red spots.

Five grams of the mixed extracts from the stomach and intestine were introduced into the thigh of a strong medium-sized dog. The pulse was 102 before the operation. In two and a half hours the animal was depressed and anxious, and lay down. Respiration was stertorous and intermittent; pulse 84, irregularly intermittent. Vomited twice. In six hours, pulse 55; respiration superficial and labored. He was restless and uttered faint cries; fully conscious. Twelve hours later, pulse 70, dog much better. In twenty-four hours from the beginning of the experiment the pulse was 90, and still somewhat intermittent. Perfect recovery followed.

Experiments made with an alcoholic extract from scrapings from the floor, where there was no vomitus, were without result. The extracts from the floor differed chemically from each other. That containing some of the vomitus was brown in color, had a rancid oily odor, and a bitter taste. Tannic acid threw down a precipitate; sulphuric acid produced a purple-red color; hydrochloric a green. The other was not bitter, and did not react with acids, as did the first. Attempts to separate the active principle of the poison from the extracts were fruitless.

The preceding experiments showed that the poison was one having a special action on the heart. They were repeated with frogs. The hearts of three frogs were exposed in order to enable observation of the action. No poison was given to the first frog; the second received six drops of a solution of digitaline (0.010 gm. digitaline in 5.00 gm. water) under the skin; the third was given 0.050 gm. of the poisonous extract from the floor. The following

table shows the comparative rapidity and rhythm of the heart's contractions:—

		Frog No. I.	No. II.	No III.
After	6 minutes.	42	20	26
	10 "	40	16 irregular.	24 irregular.
	20 "	40	15	20
	28 "	38	0	12 very irregular.
	31 "	36	0	0

The hearts of the two poisoned frogs showed contracted ventricles and dilated auricles. The heart muscles showed nó microscopic changes. This experiment was repeated several times, and the slowing of the heart and the irregularity of the rhythm were constant phenomena. Notwithstanding the amplitude of the slowed heart beats, the blood was toward the end not completely ejected. These symptoms, and the condition of the heart after death are in full agreement with the characteristic phenomena of digitalis poisoning.

The results of these experiments, the lack of abnormal *post-mortem* appearances, the previous good health of the woman, the finding of forged letters proving the accused to have anticipated the death of the deceased, the possession and use of such a quantity of the deadly poison by the accused, and the fact that he was pecuniarily interested in the woman's death, made a very strong case against him. He was declared guilty, and subsequently executed.

Case LV.—A peculiar Case of Poisoning by Carbon Monoxide.

(Vierteljahrsschrift für gericht. Medicin u. öffentl. Sanitätswesen, Bd. xxix. p. 272.)

§ 608. On September 27, 187– towards 5.30 o'clock in the afternoon two workmen, C. H. and W., were engaged in a paper factory filling a large iron kettle with rags. The kettle was of spherical form and 2.40 meters in diameter; it was in a large airy room with a number of others of the same sort. The kettles rotate on an axis, and at the upper part of each is an opening just large enough for a man to go through into the interior. The kettles are filled by means of a hopper-arrangement coming through a trap door in the floor above, which is about 0.75 meter from the uppermost surface of the kettles. The partially cleansed and finely cut

rags are fed loosely from the hopper into the kettle, after which a man goes through the opening and treads them down more firmly. The amount necessary to fill a kettle varies according to the character of the rags, from 1000 to 1500 kgr. Slaked lime in the form of milk of lime is then introduced through a tube; the amount used varies according to the rags, and requires from 100 to 200 kgr. of calcined lime for its preparation. The opening is then closed and the kettle rotated by machinery; steam is introduced through a pipe in the axis and the resulting lye runs off through an escape-pipe. The plugging of these pipes by rags is prevented by sieves. The operation requires for completion from four to twelve hours.

When the workmen had filled the kettle with rags C. H. went inside in order to tread them down; meanwhile W. left the factory to do an errand. On returning after a short interval he missed his comrade; receiving no reply on calling out, and finding no reason for his absence from the room, he climbed into the kettle. There he noticed that the rags were more firmly packed, filling only one half of the kettle; lying on the rags was the apparently lifeless H. After vainly trying to rouse him he climbed out without having felt in the least unwell, brought a lamp, and called another workman, J. H., to his aid. W. again climbed in while J. H. held the lamp at the opening in order to light up the interior. It was then about six o'clock. When W. had convinced himself that H. was dead, he went to give information at the office. Meanwhile several workmen came into the room, and the workman J. H., a very strong man, climbed into the kettle and lifted the corpse, so that the others standing above could draw it out. After about half an hour, during which efforts to revive the deceased were fruitless, W. again climbed into the kettle to continue the packing. To his horror he found a second corpse, that of J. H. who had lifted out the first, and who in the general excitement had not been missed. The death of C. H. had not been ascribed to the development of gas but to apoplexy, and that J. H. might require assistance in getting out of the kettle had not been considered. W. while trying to revive J. H. became unwell, and was barely able to get out of the kettle. The symptoms were those of suffocation, and disappeared only after long exposure to fresh air.

This led to the conclusion that poisonous gas must have been developed in the kettle and caused the two deaths. In order to

remove the second corpse with more ease and less danger, the kettle was turned on its axis till the opening came nearer the floor. The overseer then introduced a burning lamp, and the upper portion of his body into the cavity to discover the where abouts of the corpse; on finding it he withdrew and instructed the workmen as to its position. A workman now introduced his arm into the opening up to the shoulder for the purpose of reaching and drawing forth the body. A second man stood next to him with one hand inside to seize the body as soon as it should be brought within reach; a third stood by ready to assist. The overseer stood with a lamp about three yards from the hole, when suddenly a violent explosion occurred, more or less severely injuring the three workmen, and especially the first mentioned. The contents of the kettle were ignited, but soon quenched by means of a powerful stream of water, then removed from the vessel. Examination of the wet and partially charred rags revealed the presence of a large amount of fragments of lime. This gave an important hint towards explaining the cause of death, which, on the supposition that C. H. had performed the operation in the usual manner, was very mysterious. The theory of the overseer was that C. H., in order to avoid slaking lime and thus save an hour, had thrown in the lime before filling with rags, intending to let the steam slake it. The workman W. had noticed nothing of the sort, yet it had been possible for H. to throw in lime while W. brought rags. The director admitted it to be very possible that a certain amount of water had been left in the kettle from the last cleaning.

The theory of the development of carbonic oxide and hydride from dry distillation of the rags was strengthened at the autopsy by the very noticeable characteristic light-red blotches (suggillations) on the body.

The *autopsy* was performed 62 hours after death. The same bright red color was observed in the interior of the body, especially marked in the blood and muscles. This red color was not so marked in the body of J. H. Decomposition was comparatively far advanced in both bodies; *rigor mortis* was present in J. H. only in the lower extremities. In both cases vomitus was found in the respiratory tract; in the body of C. H. but a small number of doughy clumps were found in the foamy mucus which filled the trachea, but larger masses were present in the mouth, nose, and

œsophagus. In the lungs of J. H., as far as the medium-sized bronchi, fine food particles and foam were discovered; the trachea contained more of the same, and immediately above the bifurcation was completely plugged by a strongly wedged mass of food. The lungs of both were, as is usual in suffocation, œdematous, and on the surface emphysematous. No ecchymoses were found on the lungs, heart, or pericardium. The bloodvessels of the skull and brain of C. H. were very full; those of J. H. not in so marked a degree. The spectroscopic examination of the blood was unfortunately not carried out.

The investigation of this case included consideration of the nature of the poisonous gas. It was already evident that when inhaled, it was either incapable of supporting life, or capable of exercising an active injurious influence or both, and that it was an explosive. As to its development: admitting the presence of a certain amount of water in the kettle, which, if not left from the previous cleaning, may have been present as dampness in the rags (hygroscopic, etc.), it is evident that, when brought into contact with the unslaked lime, hydrate of calcium will be formed with the development of a great amount of heat. The rags in contact with the lime would thus be subjected to a sort of dry distillation, or, owing to a lack of oxygen, to an incomplete combustion. Under such circumstances gaseous compounds would be developed, of which carbon monoxide and marsh gas are the most important. Both of these, and especially the former, are specifically lighter than air, with which they easily mix; both are colorless and odorless; both may be inhaled without any irritating effect on the mucous membrane. The marsh gas of itself produces no injurious effects when inhaled, though it is incapable of supporting life. Mixed in certain proportion with air, and ignited, it explodes violently; the explosion does not occur when the proportion of air is less than six times, or more than fourteen times, that of the gas. The carbon monoxide, on the contrary, is not only incapable of supporting life, but acts as a poison on respiration. Mixed with air in a proportion of five per cent., it soon brings on unconsciousness and death. Neither in the pure state nor when mixed with air is it explosive; it burns in the air with a blue flame to carbon dioxide. The explosion which occurred in this case is easily explained, and the reason of its non-occurrence on the first exposure of the lamp to the atmosphere of the kettle is evidently

that the proportion of marsh gas in the kettle was too small or too large for an explosion to occur. The *post-mortem* appearances, especially those of C. H., were characteristic of poisoning by carbon monoxide. The bright-red color and the fluidity of the blood in both cases, and the bright-red color of the suggillations on the body of C. H., the bright-red muscles of both in spite of advanced decomposition, have been mentioned. The character of the blood is especially characteristic of poisoning by the carbonic oxide. The body of J. H. was not so marked as that of C. H., nor was the blood so light in color; decomposition was farther advanced. The relatively few characteristic appearances of carbonic oxide poisoning in the case of J. H. and the fact of the trachea having been firmly plugged by vomited food, had an important bearing on the verdict of the examiners.

The finding was in substance as follows:—

1. The death of C. H. was caused by inhalation of carbonic oxide gas developed in the rags by the heat produced by the change of quicklime to milk of lime.

2. The death of J. H. was due directly to inhalation of vomited food and indirectly to poisoning by carbonic oxide gas.

3. The explosion in the factory was due to the ignition of a mixture of marsh gas (light carburetted hydrogen) and air.

The points of peculiar interest in the above case are first the short duration, and second, the manner in which the gas was developed.

Case LVI.—Poisoning by Illuminating Gas.

47 (Vierteljahrsschrift für gerichtl. Med. und öffentl. Sanitätswesen, Bd. xxv. p. 276.)

§ 609. On March 20th, 1875, it was reported that a number of persons dwelling in a house in Bernburg had become very sick during the night, and that some had already died. The physician who was called, noticed on entering a smell of illuminating gas, which was strongest in the rooms inhabited by the family H. He was met in the sitting-room by H., who was staggering and half insensible. In the sleeping room adjoining lay H.'s wife in bed, gasping and unconscious; in another bed lay his father dead, cold, and already stiff. The children, aged respectively one and ten years,

had suffered from convulsions, unconsciousness, and vomiting, and had already been carried to another apartment. The affected persons were immediately removed from the house, and later to the hospital, where the usual methods of resuscitation were resorted to H. and his children recovered gradually; his wife died in two hours despite the most energetic stimulant treatment.

Investigation revealed the following facts: The house was devoid of gas supply. On the day before the unfortunate occurrence the tenants had noticed an odor of gas in several rooms on the ground-floor, and several had complained of headache, stupor, nausea, and vomiting. The windows were opened, and the smell and symptoms disappeared. No further attention was given to the trouble, and the odor was ascribed to rock oil. At four in the morning H. awoke, noticed again the penetrating smell, and vomited violently. He staggered to the outer door, where he was so confused and insensible that he was unable to answer the questions put by several soldiers passing by. In a short time he returned to his room, fell to the floor, and lay there several hours. On return of consciousness he entered the bedroom, where he found his father dead and his wife dying. He called the landlord, who opened the windows, carried the children to another apartment, and called in help.

The symptoms of those affected were nausea, stupor, difficulty of speech for days, impairment of memory, weakness, and depression, the usual symptoms of poisoning by illuminating gas. The death of H., senior, was apparently quiet; Frau H. remained for some time in deep coma with stertorous respiration.

All doubts as to the cause of death were soon settled, for on making an excavation in the street, a break was found in the gas-main, from which point the gas had penetrated through the soil and into the house.

The *post-mortem* examination was made forty-eight hours after death. Numerous pink spots (suggillations) were present, not only on the dependent parts but also on the dorsum of the feet and abdomen. Rigor mortis was marked. Decomposition almost absent. The muscles were of a beautiful bright-red color. Blood for examination was easily obtained from the arteries of the old man by means of an ordinary syringe. It was light red and fluid. The arteries of the woman contained stringy coagula; the

veins contained dark half-coagulated blood, which was with some difficulty drawn into the syringe. The spectroscopic examination of the blood of the man gave the characteristic absorption bands, which were not affected by addition of sulphide of ammonium, and thus characteristic of blood containing carbon monoxide.

The examination of the blood of the woman did not give good results, which was due probably to the fact that she had lived two hours in an atmosphere free from the poison.

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