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TOXICOLOGY.

MEDICAL JURISPRUDENCE

FORENSIC MEDICINE AND TOXICOLOGY

BY

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TOXICOLOGY.

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INTRODUCTION.

HISTORICAL.

It seems probable that the deleterious qualities of poisonous plants and the effects of the bites of venomous reptiles must have been recognized by prehistoric man at a time long antedating the historical period. The most ancient writings which have come down to us, if they deal with things medical, indicate a knowedge of toxicology in the early civilization of Egypt and India. Duteil[1] has interpreted a passage in an extremely ancient papyrus in the Louvre as follows: "Speak not of the name of J. A. O. under the penalty of the peach," from which it would be difficult to draw any other inference than that the Egyptian priests were acquainted with the preparation of a poisonous substance (hydrocyanic acid) from peach leaves or kernels with which those who betrayed the secrets of the priesthood were destroyed. The papyrus Ebers[2] also makes mention of both mineral and vegetable poisons (lead, antimony, copper, opium, hyoscyamus) without, however, referring to their poisonous qualities.

The translations of the early Sanskrit medical writings contained in the Ayur-Veda (*ca.* B.C. 900?) and the commentaries thereupon (Shastras) of Charaka and Sushruta (*ca.* B.C. 600?) which have been made by Wise,[3] indicate an extensive knowledge of poisons among the Hindus of that early period. The Ayur-Veda contained a division treating of the administration of antidotes for poisons, and of the prevention of the effects of the diseases produced by mineral, vegetable, and animal poisons, and by the bites of venomous serpents, insects, etc. The Shastras of Charaka and Sushruta contain sections devoted to poisons and their antidotes (kalpa, panata) in which the actions

[1] Flandin: "Tr. d. Poisons," Par., 1846, i., 31; Hoefer, "Hist. d. l. Chimie," Par., 1842, i., 226.

[2] "Papyros Ebers," Joachim, Berlin, 1890.

[3] "Commentary on the Hindu System of Medicine," T. A. Wise, Calcutta, 1845.

of vegetable, animal, and mineral poisons are described,[1] including a lucid account of alcoholic intoxication, and the following, which we quote to show that criminal poisoning was then guarded against:

It is necessary for the practitioner to have a knowledge of the symptoms of the different poisons and their antidotes. As the enemies of the Rája, bad women, and ungrateful servants sometimes mix poison with food. On this account the cook should be of good family, virtuous, faithful, and not covetous, nor subject to anger, pride, or laziness. . . . The practitioner should have like qualities, with an intimate knowledge of poisons; and should examine the food to be eaten by a Rája in the cooking room. This should be large, airy, light, and surrounded with faithful servants, and no one should be allowed to enter unless he is first examined. . . . A person who gives poison may be recognized. He does not answer questions, or they are evasive answers; he speaks nonsense, rubs the great toe along the ground, and shivers; his face is discolored; he rubs the roots of the hair with his fingers; and he tries by every means to leave the house. The food which is suspected should be first given to certain animals, and if they die, it is to be avoided."

Apart from references to the venom of serpents,[2] Mosaic history contains but few allusions to poison,[3] but it is not probable that the Hebrews during the Egyptian captivity failed to acquire some knowledge of the action of poisons. The "bitter water"[4] and "water of gall,"[5] used by the Hebrews as an ordeal and for punishment, must have contained some poison, at least in those cases in which it possessed any efficacy.[6]

The Greeks at a very early period were acquainted with the action of poisons and venoms. Homer relates that Ulysses[7] sought to obtain from Ephyra a poison (*φάρμακον ἀνδροφόνον*) with which to anoint his arrows; and, according to Ovid[8] the arrows of Hercules were charged with the venom of the Lernean serpent. It is from the use of poisons in this manner that the word "toxicology," common to most modern languages, is derived.[9] Hecate, the daughter of Perseus, is said by Diodorus Siculus to have been skilled in the preparation of poisons; to

[1] Wise, *l. c.*, pp. 391–412.

[2] Gen. iii. 15; Deut. xxxii. 24, 33. See "Hierobotanicon," O. Celsius, Upsala, 1745, i., 8, 78; ii., 23, 119, 408.

[3] Numbers v. 17–27.

[4] Jeremiah ix. 14.

[5] Cf. also Mark xvi. 18.

[6] "Od.," i., 261.

[7] "Metam.," ix., 158.

[8] *Τόξον*, a bow; *τοξικός*, for the bow; from which Dioscorides (Alexiph., xx.) derived the name *τοξικόν* (*φάρμακον*) to apply to the poison with which the barbarians smeared their arrows.

have invented that called aconite,[1] and to have tested the virtues of her preparations by mixing them with the food of her guests. Having acquired great experience in the art, she poisoned her husband. Her daughters, Circe and Medea, also became proficient in toxicology, and the former profited by her mother's example, and removed her husband by the same means.[2] To protect himself from the poisons and spells of Circe, Ulysses obtained from Hermes an herb with black root and milk-white flower, which acted as a narcotic.[3]

In the historical period there are numerous evidences of an early knowledge of the action of poisons among the Greeks and neighboring nations. It is related by Plutarch[4] that Alexander the Great (B.C. 333) drank the medicine offered by his physician, Philip, and recovered, although he had been warned that Philip had been bribed to poison him. Xenophon (*ca.* B.C. 400) relates that the use of poison was so frequent among the Medes that is was an ancient custom for the cup-bearers to taste of the wine before presenting it to the king, and that among the Persians the children were instructed in the properties of plants that they might know which were deleterious.[5] Of the fifteen orations of Antiphon (born *ca.* B.C. 480) one was in the matter of an accusation of poisoning.[6] Among the Athenians an indictment for poisoning (*φαρμάκων vel φαρμακείας γραφή*) was tried before the Areiopagus, and a malicious intent was a necessary ingredient of the crime, for which the punishment was death. Women appear to have been most addicted to the crime of poisoning in the Grecian period, as they are at the present time.[7] The earliest works extant, treating specially of poisons, are the "Theriaca" and "Alexipharmaca" of Nicander, written between B.C. 185 and B.C. 135,[8] which, although con-

[1] Whether this was the plant now known under this name, or some other (possibly conium) is not known. The Latin writers used the word to refer to poisonous plants in general (Virgil, "Geor.," ii., 152).

[2] Diodorus Siculus, "Hist.," iv., 45.

[3] "Od.," x., 305; v., 236. Theophrastus and Dioscorides consider this *μῶλυ* to have been a species of garlic. Pliny, "Hist. Nat.," xxv., 8.

[4] "Vit. Alexandri," c. 19.

[5] "Cyropædia," i., 3; viii., 8.

[6] *Κατηγορία φαρμακείας κατὰ τῆς μητρυιᾶς.*

[7] See the oration of Antiphon above, and Kennedy, Smith's "Dictionary of Greek and Roman Antiquities," London, 1870, p. 895; Friedreich's "Bl. f. ger. Anthrop.," 1850, i., 65; 1853, i., 40; 1854, ii., 32, 78.

[8] Poems in hexameter verse. The *Θηριακά* of nearly a thousand lines, and the *Ἀλεξιφάρμακα* of more than six hundred lines.

taining much that is fabulous, set forth so wide a knowledge of the effects of many vegetable, animal, and mineral poisons that it is impossible to doubt that the work of Nicander was but the expression in poetical form of information previously obtained by an extensive experience with the agents whose effects he describes, probably by others whose writings, if such existed, have been lost. Indeed, the "History of Plants" of Theophrastus,[1] written about B.C. 300, contains many references to the poisonous as well as the medicinal action of the vegetable poisons, including most of those mentioned by Nicander.

Hemlock was used among the Greeks for the execution of criminals, both at Athens and at Marseilles,[2] then a Greek colony,[3] and was the means of execution of Socrates, B.C. 339.[4] It is also related by Valerius Maximus[5] that the senate of Marseilles kept hemlock at the disposal of those who wished to destroy themselves for reasons which the senate deemed to be adequate. In B.C. 317 Olympias, widow of Philip of Macedon, having captured her rival Eurydice, sent to the latter in her prison a sword, a rope, and a cup of hemlock with orders to choose her mode of death.[6]

"The Materia Medica" of Dioscorides, written in the first century of our era,[7] which, with the commentary of Matthiolus[8] remained the standard work upon the subject down to the sixteenth century, contained a treatise on poisons and their antidotes.[9]

[1] Περὶ φυτῶν ἱστορια. The best editions are those of Bodæus a Stapel, fol., Amstelod., 1644, and Schneider, 5 vols., Lips., 1818–21.

[2] Cicuta quoque venenum est publica Atheniensium pœna invisa, ad multa tamen usus non omittendi. Pliny, "Hist. Nat.," xxv., 95.

Valerius Max., ii., 6.

[4] Diogenes Laërtius, ii. Whether the poison by which Socrates was destroyed was in fact conium maculatum was a question among the earlier toxicologists. Wepfer ("Hist. Cicut. Aquat.," vi. Ed., Basil., 1679, p. 5) gives a full account of the controversy. The evidence appears to be in favor of the affirmative, as is shown by Schultze ("Toxicologia Veterum," Halle, 1788, p. 34, s. 2.

[5] Lib. xxi., cap. 6.

[6] Diodorus, xix., 11; Justin., xiv., 5; Ælian, V. H., xiii., 36.

[7] The first Greek edition, fol., Venet., 1499, ap. Aldum Manutium; the first Latin translation, attributed to Petrus Aponensis, fol., Colle, 1478. Of the numerous later editions probably the best is that of Saracenus, fol., Francof., 1598.

[8] First published in Italian, 1544. Many later editions in Latin, of which the most esteemed is that of Venice, 1565, fol.

[9] Printed as the sixth book, περὶ δηλητηρίων φαρμάκων, in the Aldine, 1499, 1518, Cologne, 1529, and Paris, 1549, editions; and in that of Saracenus as separate treatises, "Alexipharmaca" and "Theriaka."

The search for antidotes was probably coeval with the discovery of the deleterious effects of poisons. The earliest reference to the use of a counter-poison is that to the μῶλυ of Homer, already referred to. Theophrastus and Nicander also direct what remedies shall be used to combat the effects of poisons. It was in the first century B.C., however, that the idea of antagonizing the action of one poison by another, or by habituation, was first developed. According to Galen,[1] Zopyrus, a physician of Alexandria, invented an antidote which he recommended to Mithridates, king of Pontus; and Celsus refers to a similar composition which the same physician prepared for one of the Ptolemies. The extended acquaintance of Mithridates with the action of poisons and of their antidotes is frequently referred to in classical literature, and it is related that when (in B.C. 63) he wished to commit suicide, his constitution had been so long inured to antidotes that poison had no effect upon him and he was compelled to have a mercenary dispatch him with his sword.[2] The Mithridatic antidote is described by Celsus[3] as consisting of thirty-six ingredients. As, according to Pliny,[4] Pompey caused a translation of the work of Mithridates on poisons to be made by Lenæus, it is not improbable that the Mithridatic served as a model for the "Theriac" of Andromachus, and the numerous other theriacs and alexipharmacs, the use of which continued down to the beginning of the eighteenth century.

The earliest reference to criminal poisoning in Roman history was in B.C. 331, when about twenty matrons, including Cornelia and Sergia, were surprised in the act of preparing a poison, which they were compelled by the magistrates to drink, and thus perished. Following this, other matrons to the number of one hundred and seventy were convicted.[5] In B.C. 184 the prætor was directed by the senate to investigate cases of poisoning (de veneficiis quærere) growing out of the baccha-

[1] "De Antid.," ii., 8, vol. xiv., p. 150. Ed. Kühn and Celsus, v. 23, s. 2, p. 94.

[2] Appian, "Mithridat.," 107–111: Dion Cass., xxxvii., 3, 11–13: Plut., "Pompey," 41; Pliny, "Hist. Nat.," xxv., 3; Gellius, "Noct. Att.," xvii., 16. Also Martial:

Profecit poto Mithridates sæpe veneno,
Toxica ne possent sæva nocere sibi.

[3] Celsus, v., 23. Galen, "De Antid.," ii., 9, says forty-four, and Pliny, "Hist. Nat.," xxix., 8, fifty-four.

[4] "Hist. Nat.," xxv., 3.

[5] Livy, viii., 18; Val. Max., ii., 5, s. 3.

nalian orgies, and in B.C. 180 another like investigation was directed during the prevalence of a pestilence. In both a great number of persons were convicted.[1]

The first legislative enactment specifically relating to the crime of poisoning was the Lex Cornelia de Sicariis et Veneficis, passed by the dictator Sulla in B.C. 82, which continued in force, with some modifications, until the fall of the empire. The punishment provided was *deportatio in insulam* and confiscation of property if the prisoner was of high rank, and exposure to wild animals if of low degree. A later provision made the law applicable to druggists (pigmentarii) who carelessly administered poisons, and to women who, even without evil intent, administered poison to produce conception, if the person to whom it was given died. Women so convicted were punished by banishment.[2] In the legislation of Justinian poisoning was regarded as a more heinous crime than murder by violent means.[3]

Although many of the suspected poisonings during the later years of the Republic and the Empire mentioned in Roman literature were undoubtedly false accusations or mere suspicions, the very prevalence of the fear and imputation of poisoning, indicates a widespread popular knowledge of the action of poisons and readiness to use them, which is further proved to have existed at that period by a great number of murders by poison, the historical proof of which cannot be questioned.

Livia, the wife of Augustus, was strongely suspected of having poisoned Marcellus, the son of Octavia, and Caius and Lucius, the children of Julia, and of having even hastened the death of Augustus by administration of poison in A.D. 14, to secure the succession to her son Tiberius,[4] who was believed to have compassed the death of Germanicus by poison in A.D. 19, with the aid of Cn. Piso and his wife Plancina.[5] In this connection Tacitus also states that in A.D. 50 Martina, a notorious poisoner (famosa venefica), and the favorite of Plancina, was sent to Rome for trial, but died suddenly on her arrival at Brundisium.[6] During the reign of Tiberius, Sejanus seduced

[1] Livy, xxxix., 8, 38, 41; xl., 37.

[2] Marcian. Dig. 48, tit. 8, s. 3; Inst. 4, tit. 18, s. 5.

[3] Plus est hominem exstinguere veneno, quam occidere gladio. Dig. xlviii. 8; iv., 3.

[4] Dion Cass., liii., 33.

[5] Tac., "Ann.," ii., 73; iii., 16; Pliny, "Hist. Nat.," xi., 71.

[6] "Ann.," ii., 7; iii., 7.

Livia, the wife of Drusus, the son of the emperor, and with her aid and that of the physician Eudemus, caused the death of Drusus by poison administered by Lygdus in A.D. 23. The facts were only brought to light eight years later upon the information of Apicata, the wife of Sejanus, supported by the confessions, under torture, of Eudemus and Lygdus.[1]

It is stated that Caligula left a large chest filled with several sorts of poisons, which being, at the command of Claudius, thrown into the sea not long after the death of Caligula, the waters were so infected thereby that there died abundance of fish.[2]

Agrippina and her son Nero, aided by Locusta and Xenophon, made elaborate studies in experimental toxicology upon the human subject, and applied the knowledge so gained to the removal of Crispus Passienus, the second husband of Agrippina, the emperor Claudius, Domitia, and Britannicus. Locusta was executed under Galba.[3]

One of the most masterly of the orations of Cicero was in defence of A. Cluentius Habitus[4] (B.C. 66), accused by Oppianicus, son of Statius Albius, of three distinct acts of poisoning, two of which had proved successful, of which accusation he was acquitted. Cluentius himself had previously (B.C. 74) accused Statius Albius of an attempt to poison him, and, it was claimed, had secured his conviction by bribery of the judices. In his defence Cicero also accuses Statius Albius and Sassia, the mother, and one of the accusers of Cluentius, of having poisoned Cluentia, a former wife of Albius, and also makes a countercharge against another accuser of his client, Clodia, the wife of Q. Met. Celer, that she had poisoned her husband, and further, refers to a physician, Clodius, as having been employed by Oppianicus to poison his grandmother, Dinæa.

It is rarely that the nature of the poison used is referred to by Greek or Roman writers. The word "aconitum," as used by Juvenal, Martial, and others, applies to any form of vegetable poison, but in one passage of Pliny it is specially applied

[1] Sueton., "Tib.," 62; Tacit., "Ann.," iv., 3, 8, 11.

[2] Platina, "Lives of the Popes," Ed. Rycaut, p. 2.

[3] Tacitus, "Ann.," xii., 61, 66, 67; xiii., 15, 19; Suetonius, "Nero," 33; Dion Cass., lx., 34; lxiv., 3; Juv., i., 71.

[4] Pro Cluentio. See also Blair, "Lectures on Rhetoric," New York, 1829, pp. 298–311.

to a certain plant.[1] C. Papirius Carbo is said to have committed suicide by cantharides in B.C. 119, and his brother, Cn. Papirius Carbo, destroyed himself in B.C. 119 with a "solution of vitriol" (atramentum sutorium).[2] Pliny states that opium was frequently used as a means of suicide, and cites a specific instance in the case of Post. Lic. Cæcina.[3] The blood of the ox is reputed as poisonous by Nicander and Dioscorides, and Æson was said to have been forced to kill himself by drinking of it.[4] Either the account is fabulous, or, as seems more probable, the poisonous quality of putrid blood was recognized, particularly as Nicander refers to "black" blood. At a much later period (A.D. 364) the death of the Emperor Jovian was probably the subject of the first historical reference to poisoning by carbon monoxid.[5]

That permission to commit suicide by poison was granted during the Empire, as at Athens, is evident by the case of Euphrates, who, having reached an advanced age, asked and obtained of Hadrian the permission to put an end to himself by poison.[6]

Simulation of poisoning was not unkown among the Romans, as Pliny[7] relates that Drusus (B.C. 91) drank goat's blood that the pallor so produced might aid in substantiating his accusation of poisoning against his brother-in-law, Cæpio.

During the interval between the division of the Roman Empire (A.D. 364) and the Renaissance, a period of a thousand years, toxicological science, so far as methods of detection and symptomatology are concerned, made no step in advance, and the armamentarium of the poisoner was only slightly extended by the addition of a few new forms of poisons already known, such as white arsenic, and other chemical compounds discov-

[1] "Hist. Nat.," xxvii., 2. This passage also refers to a method of administration which has been very rarely imitated in modern times: Sed antiquorum curam diligentiamque quis possit satis venenari, cum constet omnium venenorum ocyssimum esse aconitum, et tactis quoque genitalibus fœminini sexus animalium eodem die inferre mortem? Hoc fuit venenum, quo interemtas dormientes a Calpurnio Bestia uxores M. Cæcilius accusator objecit. Hinc illa atrox peroratio ejus in digitum.

[2] Schmitz, in Smith's "Dict.," i., 610, 611.

[3] "Hist. Nat.," xx., 76.

[4] Nicand., "Alexiph.," 312; Diosc., "Alexiph.," cap. xxv.; Diod. Sic., iv., 50. See also Pliny, "Hist. Nat.," xxviii., 41.

[5] Plate in Smith's "Dict.," ii., 615; Hoffmann, "Op. Omn.," i., 229. Gibbon, iii., 232.

[6] Dion Cass., lxix., 8.

[7] "Hist. Nat.," xxviii., 41.

ered by the Greek and Arabian alchemists. The history of this period is lacking in that precision which is essential to distinguish between mere suspicion of poisoning in cases of sudden death of prominent persons and deaths truly attributable to poison. Yet there is no lack of supposed poisonings, although less concealed methods of disposing of obnoxious persons were more freely resorted to than during the comparatively civilized period of the Roman Empire. An indication of the customs of the times is to be found in the methods of termination of the reigns of the eighty-three emperors of the East from Valens (A.D. 364) to Constantine XII. (*obiit* A.D. 1453); of these, thirty-nine died from causes presumably natural, seventeen by assassination, four were killed in battle, two by accident, seven by poison,[1] and fourteen were disposed of by confinement in monasteries.

Constantine the Great is said to have caused the death of his son Crispus in A.D. 326, either "by the hand of the executioner or by the more gentle operation of poison."[2] Nine of the successors of Charlemagne as emperors of the Holy Roman Empire, anterior to the accession of the house of Austria in 1438, died from the effects of poison.[3]

Of the Popes of Rome, up to the close of Platina's history, at the death of Paul II. in 1471, five are said to have died of poison.[4] In the "Acta Sanctorum" of the Bollandists are many

[1] Constantine III., A.D. 641, by his stepmother, Martina (Gibbon, "Rom. Emp.," ed. Smith, London, 1862, vi., 73); Leo IV., A.D. 780 (Robertson, "Hist. Christ. Church," ii., 151); Constantine VII., A.D. 959, by his son, Romanus II., at the instigation of his wife Theophano, who, four years later, A.D. 963, poisoned her husband as she had his father (Smith, "Dict. Biogr.," i., 840; iii., 656, 657; Gibbon, *l.c.*, vi., 103, 104); Zimisces, A.D. 975 (Gibbon, *l.c.*, vi., 107; Smith, "Dict.," i., 469); Romanus III., A.D. 1034, by his wife Zoe (Gibbon, *l.c.*, vi., 109; Smith, "Dict.," iii., 657); Henry A.D. 1216 (Smith, "Dict.," ii., 382).

[2] Gibbon, ii., 352.

[3] Charles the Bald (II.) by a Hebrew physician, Sedecias, in 877; Otho II. at Rome in 983; Otho III. by Stephanie, widow of Crescentius, in 1002 (Robertson, *l. c.*, ii., 435); Conrad III., by Roger of Sicily in 1152; Henry VI. by his wife Constance, in 1197; Frederick II. by an illegitimate son, in 1250; Conrad IV., according to some by orders of the Pope, according to others his physician, John Maurus, of Salernum, administered an enema containing powdered diamond and scammony, in treatment for a fever, whereof he died in 1254; Henry VII. by poison administered in the sacrament by monks at the instigation of Robert, King of Naples in 1313 (Infessura, Diarium, ed. Eccard, "Corp. Hist. Med. Aevi.," ii., 1866. Platina, "Lives of the Popes," ed. Rycaut, p. 304); Gunther, poisoned by a physician of Frankfort in 1349. (See Hoefer, "Biog. gén.," *passim.*)

[4] John XIV., 984 (Robertson, *l.c.*, ii., 419); Clement II., 1046, (Platina, ed. Rycaut, p. 201; Robertson, *l.c.*, ii., 550); Damasus II., 1048 (Robertson, *ibid.*); Victor III.,

references to instances of poisoning which have been collected by Marx.[1]

Rosamond, widow of Albion, king of Pannonia, whose murder by Helmichis she had instigated, sought to poison her confederate, in 575, with poison in wine. He drank half, and, feeling the effects, compelled her to drink the remainder, so that in a few hours both came to their end.[2]

Childebert, King of Austrasia, was poisoned in 596, either by his mother Brunhilde or by Fridegonde, queen of the Franks. The life of the latter "could be summarized in a chronological table of assassinations by steel or poison"; the former was accused of having poisoned her grandson, Thierry, and of having murdered ten kings or sons of kings.[3]

In the first years of the twelfth century Bertrade, wife of Philip I. of France, attempted to poison her stepson, afterward Louis VI., who escaped death by a miracle, and suffered long from the effects of the poison.[4]

Early in the thirteenth century King John of England is said to have caused the death of Maud Fitzwalter in the Tower of London by a poisoned egg.[5]

The Register of the Chatelet at Paris, under date of July 22d, 1390, contains an account of the examination of one Jehan le Porchier, accused of intent to poison the king (Charles VI.).[6]

Charles the Bad (II.) of Navarre is said to have been well versed in alchemy and toxicology. Godefroy of St. Denis and Juvenal Ursinus recite the directions given by Charles to a minstrel, Woudreton, that he shall purchase "sublimed arsenic," which he will find in the shops of the apothecaries, and poison the King of France (Charles VI.), his brother, and two uncles, by mixing it with their food or drink. Woudreton was captured and executed in 1384.[7]

In the writings of physicians during the middle ages are in-

1087 (Platina, 215; Robertson, ii., 668); Benedict XI. (or X.), 1303, (Infessura, *l. c.*, ii., 1865). An attempt was also made to poison Eugene IV. in 1432. (Infessura, *l. c.*, ii., 1876; Platina, *l. c.*, 358.)

[1] "Lehre von den Giften," Gött., 1827, p. 38.

[2] Machiavelli, "Hist. Florence," ed. Bohn, p. 12.

[3] Thierry, "Récits Mérovigniens."

[4] Hoefer, "Biogr. gén."; Prudhomme, "Crimes des Reines de France," Par., 1791, p. 83.

[5] Dixon, "Her Majesty's Tower," London, 1869, i., 46.

[6] Introduction, vol. i., xiii.

[7] Hoefer, "Histoire de la Chimie, ex Mangin, Poisons," p. 36. For other instances of poisoning during this period, see Marx, "Gifte," i., 42, *seq.*

dications in varied forms showing that criminal poisoning was certainly not a lost art. Thus Oribasius includes in the elaborate collection of earlier medical writers, which he prepared at the command of Emperor Julian (A.D. 361), a chapter on poisons, the brevity of which is accounted for by the statement that the dissemination of the knowledge of the action of poisons would be an aid to the commission of crime.[1] The same view was not entertained by Aëtius of Amida, physician to Justinian I., who wrote in the middle of the sixth century, and described the poisons then known, chiefly those already mentioned by Nicander and Dioscorides, and their effects and antidotes.[2] The fifth book of the "de Arte medendi," of Paulus Ægineta (*circa* A.D. 650), treats in the first twenty-six chapters of venoms and in the remaining forty of poisons,[3] and is practically a transcript of Dioscorides.

From the middle of the seventh century to the rise of the school of Salernum in the beginning of the eleventh century, was a period practically without contributions to medical literature by Christian writers, although several Arab physicians of note lived in the tenth and eleventh centuries, whose works contain more or less of toxicological interest, particularly with reference to substances having poisonous qualities, such as corrosive sublimate and white arsenic, which had been discovered by the Arabian and Greek alchemists. The eighth book of the "Almansor"[4] of Rhazes (*ob.* 923 or 932) treats of poisons and antidotes. Corrosive sublimate is described as being highly poisonous, while liquid mercury was found non-toxic in an experiment upon a monkey. In the "Liber Regius of Haly Abbas"[5] (*ob.* 994) directions are given for the treatment of poisoning. Avicenna (980–1037) wrote at length concerning poisons, their action and antidotes.[6] His death was due to a heroic treatment to which he subjected himself, accelerated by a mithridatic containing an excess of opium. The work of Serapion the Younger (*circa* 1070), which is a compilation from previous writers, con-

[1] De venenis neque tutum est indefinite scribere, neque memoriæ literarum commendare omnes curationes neque nocumenta, quæ quisquis ex eis nasci dicit, siquidem qui hæc doceat, causam scelestis præbebit, ut multa mala faciat ("De loc. affect.," iv., 63).

[2] Tetrabiblion IV., serm. i., 10, 45-81.

[3] Ed. Basil., fol., 1532, pp. 304–331.

[4] "Liber medicinalis Almansoris."

[5] Lib. iv., c. 38.

[6] "Kanon," lib. ii., iv.

tains much of toxicological interest. He describes nux vomica, but does not refer to its tetanic action, mentions the depilatory and escharotic power of arsenic trisulfid (Galen), and discusses the bezoar and its virtue as a universal antidote.[1]

Several Arabian and European medical writers during the twelfth, thirteenth, and fourteenth centuries described the actions of poisons and antidotes; among others, Johannes Actuarius, Averrhoes (*ob.* 1206), Maimonides (*ob.* 1208), Nicholaus Praepositus, and Gilbertus Anglus. A few also contributed special treatises upon poisons; Petrus of Abano (1250–1320); Arnaldus of Villanova (*ob.* 1314); Antonius Guainierius (*ob.* 1445), and Santis de Ardoynis (1424–26).

With the Renaissance in Italy poisoning became an art, practised for private vengeance or cupidity, or political murder at the procurement of states as well as individuals. During the fifteenth century no less than fourteen editions of the "De Venenis" of Abano were printed, the rarity of all of which is additional testimony of their popular use.

The despot of that period "protected his bedchamber with a picked guard, and watched his meat and drink lest they should be poisoned," and "no one believed in the natural death of a prince; princes must be poignarded or poisoned" (Symonds). Of the long list of poisonings of prominent persons during the fifteenth and sixteenth centuries, a few may be cited as examples: Polissena, the first wife of Francesco Sforza, and her little girl were poisoned by her aunt; Gian Galeazzo Sforza was poisoned in 1494 by his uncle, Ludovico; Cardinal Ippolito di Medici was poisoned by his brother, Alessandro, in 1535, who also caused the death of the poet Berni by the same means in 1536; Antonio Fiziraga was poisoned at Lodi in 1402; Biordo Michelotti was stabbed between the shoulders with a poisoned dagger by his relative, the abbot of S. Pietro, in 1416; a princess of the house of Este was poisoned by her husband in 1493 to prevent her poisoning him; while Lucchino Visconti was plotting to murder his wife, Isabella Fieschi, she succeeded in poisoning him in 1349; Gian Galeazzo Visconti poisoned his uncle Bernabo in 1385; Pietro Riario, Cardinal of San Sisto, died at Rome in

[1] "De simplicium medicamentorum historia," Venet., 1552, iv., 16; vi., 6; vi., 31. The bezoar (calculi from the intestines of herbivorous animals) continued in vogue as an antidote until the beginning of the eighteenth century. (See Marx, "Gifte," 210–211.

1474, poisoned, it is alleged, by the Venetians; Louisa Strozzi was killed by a corrosive poison in 1534; Ladislas, King of Naples, died in 1414 from the effects of a poison, which a physician of Perugia had placed upon the lips of his daughter, the king's mistress; Leo X. (Medici) died suddenly in 1521, and, according to the testimony of physicians, by poison. The historian Guicciardini, in an essay upon the form of government to which Florence was best suited, speaks of the manner in which tyrants should be dealt with as follows: "The one true remedy would be to destroy and extinguish them (the tyrants) so utterly that not a vestige should remain, and to employ for this purpose the poignard or poison, as may be most convenient; otherwise the least surviving spark is certain to cause trouble and annoyance for the future." [1]

That political murder by poison was considered as quite legitimate in Italy at that time is evidenced by the contents of the secret archives of the Council of Ten at Venice, which have been published by Lamansky: [2]

The second minute in the collection, under date of May 24th, 1419, is an agreement of the Council to a proposition of Michaletus Mudacio to poison Sigismund, King of Hungary, for a specified reward, and that poison shall be furnished him for the purpose. This Mudacio seems to have played a double game with the Council, for after experiments with alleged poisons found to be harmless, and several discussions in the Council concerning him in 1419, 1420, and 1432, the desired end was not attained, and Sigismund died a natural death in 1437.[3] The third entry in the record, September 23d, 1419, relates to an offer by the Archbishop of Trebizonde to procure the death by poison of Marsilius of Carrara, which was accepted and payment of fifty ducats and a horse ordered. In 1450, December 2d, "the Council received a poison in the shape of balls, prepared at their express order, and killing by its odor alone, when thrown into the fire. A distinguished person, discreet and intelligent, subject of another state, having offered his services to poison Count Francesco Sforza,[4] with only the condition that the poison should be furnished him, the Council, in view of the advantages which the State might gain from the death of the Count, decrees

[1] For poisonings in Italy at this period, other than the Borgian, see Symonds, "Renaissance in Italy," i.; Roscoe, "Life of Leo X.;" "Histoire des Papes," Paris, 1842–44; and the historians of the period: Machiavelli, Guicciardini, Sismondi, Varchi, Infessura.

[2] "Secrets d'État de Venise," St. Petersburg, 1884.

[3] See also Lamansky, *l. c.*, p. 159.

[4] Sforza died of dropsy March 8th, 1466.

that the effects of the poison shall be previously tested upon a person condemned to death." The same matter was further discussed by the council during 1451–53, and matters seem to have been pressing in September and November, 1453, when the last references to the project impose the condition that it shall be executed in December. Again, August 24th, 1461, it accepts a proposition of a Manual Sardoun, to poison the Sultan Mahomet II. for a stated compensation.[1] Under date of November 5th, 1477, the Council records its acceptance of the proposition of Bishop Raditch, to cause the poisoning of Sanzach and Ismael, two Turkish generals then besieging Croya. For this service a pecuniary compensation is agreed upon, partly in the form of a pension "until by the intercession of the Council with the Pope, the latter shall have given him (the bishop) a benefice." Again, January 14th, 1478–79, it accepts the proposition of one Lazarus, "the Turk," to poison the wells from which the Pasha and his army take their water; and orders that poison in sufficient quantity be sent him. June 27th, 1492, the Council accepts an offer of one Jacob of Venice, to poison a sea-captain, Barbetta, who had gone over to the Turks. February 13th, 1514–15, orders are sent to the Count of Lessina to poison or otherwise assassinate the Turk Kara-Mustapha. On December 29th, 1525, the Council took measures to avoid awkward complications arising from the misdirection of certain letters relating to poisoning. April 27th, 1527, it deliberates over an offer on the part of Don Babo de Naldo to poison the Duke of Bourbon. October 29th, 1562, the Council orders the rectors of Zara to cause the death of a prisoner, Camillo Pecchiari, by a poison which they sent for the purpose, and which they direct to be given in small and repeated doses, that the death may appear to be due to disease. March 27th, 1563, orders are sent to the Venetian agent in Constantinople, to cause the death of a dragoman, suspected of treachery, by poison. September 20th, 1564, the Council sends letters denying that it caused the poisoning of Giordano-Orsini. May 12th, 1568, the destruction of a wounded prisoner, by the application of poison to his wound by the barber having charge of him, is ordered. February 5th, 1570–71, the Council sent a chest of poison to the proveditor in Dalmatia, with orders to poison the wells and springs from which the enemies of the Republic take their water. At various times during 1574–86 the Council discusses the cases of two Turkish captains, finally causing their death by poison, and sending instructions to its representative at Constantinople to ascribe their deaths to disease. June 3d, 1592, the Council suggests to its envoy at Constantinople, that he poison a dragoman in his employ, whose faithfulness it doubts. During the seventeenth century the secret archives continue to refer frequently to the poisoning of enemies of the Republic. Indeed, as late as December 16th, 1755, the Council finds that the poisons

[1] Mahomet died May 3d, 1481.

in its collection are in great disorder, and orders the Inquisitors to put them in a more safe and serviceable condition.

That the Council at an early date sought to prevent the too vulgar use of poisons is shown by a law passed by it in 1410, in which apothecaries are forbidden under severe penalties to give or sell poisons to any person without an order from the judges.[1]

Three recipes for the preparation of poison are preserved as "secreta secretissima" in the archives under date of 1540–44. One of these shows the poison to have consisted of a mixture of corrosive sublimate, white arsenic, arsenic trisulfid, and arsenic trichlorid, prepared by sublimation.[2]

Of the numerous murders committed by Pope Alexander VI. (1492–1503) and his son, Cæsar Borgia, many were by poison. The unfortunate Zizim, or Djem, son of Mahomet II., who had sought refuge at Rome from his brother Bajazet, was poisoned between Rome and Naples by Alexander in 1495, for a consideration of 300,000 ducats paid by the Sultan. Agnelli, bishop of Cosenza, was poisoned in 1497 by Cæsar, who also caused the poisoning of the Bishop of Cette, in France, 1498, and that of his own near relative, Giovanni Borgia, in 1500. At least five cardinals, Giambattista Orsino; Ferrera, Cardinal of Modena; Michiel, Cardinal St. Angelo; and the Cardinals of Capua and Verona, were removed by poison between 1495 and 1503, to satisfy the rapacity of Alexander. "Having sold the scarlet to the highest bidder, he used to feed his prelate with rich benefices. When he had fattened him sufficiently, he poisoned him, laid hands upon his hoards, and recommenced the game."[3] Alexander himself finally fell a victim to his own poison in 1503, and Cæsar only escaped death at the same time

[1] "Nullo modo vel ingenio possit dare nec vendere talem speciem veneni sive tossici alicui persone de mondo—sine buleta predicta et licentia dominorum justiciariorum," Lamansky, *l. c.*, p. 533.

[2] The materials used in the first sublimation were: "Sulimado, L. 2" (corrosive sublimate); "arsenicho," g. 6; "rexegal, g. 2" (realgar. See Kopp, "Gesch. d. Chem.," iv., 99); "oro pimento, g. 6" (orpiment); "sal armonia, g. 6" (ammonium chlorid. See Kopp, *l. c.*, iii., 237); "sal gema, g. 6" (sodium chlorid); "verde rame, g. 4" (verdigris). Arsenic trichlorid would be produced by the action of the chlorids upon the compounds of arsenic. In the second sublimation "radix napello, g. 4" (aconite), and "aqua de ciclamina, L. 10" (cyclamen enropæum).

[3] Symonds, *l. c.*, 414. As to the Borgian poisonings see also Roscoe, "Life of Leo X.;" Gordon, "Lives of Alexander VI. and his son Cæsar Borgia;" Tomasi, "Vita di Cesare Borgia;" Lamansky, *op. cit.*; Goetz, "Diss. de Mensis Pontificiorum venenatis," Lubeck, 1715.

after a severe illness; for the Pope's butler served the poisoned wine, intended for some wealthy cardinals, to the Pope and his son by mistake.

Catharine di Medicis, wife of Henry II. of France (1533–89) is said to have had in her employ a Milanese named Reni, who served her in the double capacity of perfumer and poisoner.[1]

A story in the memoirs of Henri de Guise, about the middle of the seventeenth century (1648) indicates the popular feeling with regard to poisoning: A soldier being requested to assassinate Annese, the successor of Massaniello at Naples, shrank with horror from the suggestion, but at the same time signified his perfect willingness to poison him.

Nor was poisoning unknown in England during the sixteenth and seventeenth centuries. In 1531 a statute of Henry VIII. (22 Hen. VIII., c. 9) ordered poisoners to be boiled to death. In 1537 particular precautions were taken for the safety of the infant Edward, Prince of Wales: "The food supplied for the child's use was to be largely 'assayed.' His clothes were to be washed by his own servants, and no other hand might touch them. The material was to be submitted to all tests of poison."[2] In 1542 a young woman was boiled alive at Smithfield for having poisoned three families.[3] On September 15th, 1613, Sir Thomas Overbury died in the Tower of London, poisoned by corrosive sublimate, administered in an enema at the instigation of Robert Carr, Earl of Somerset, and his notorious countess, both of whom were tried and convicted of the crime in 1616 and condemned to death, but escaped through the weakness of James I., although their humbler instruments were executed.[4]

In Italy and France during the latter part of the seventeenth and the beginning of the eighteenth century, the use of poison as an agent of secret murder became so common as to warrant the violation of the secret of the confessional and the establishment of special tribunals. During the pontificate of Alexander VII., in 1659, the clergy of Rome informed the supreme pontiff of the great number of poisonings revealed to them in the con-

[1] "Discours merveilleux de la Vie . . . de Catherine de Medicis," 1649 pp. 43, 56, 64, 65, 78, 84, 116, 154. The first edition of this work, incorrectly attributed to H. Estienne, dated 1575, was published in 1574. See also Freer, "Life of Jeanne d'Albert," Lond., n. d., 399, 402–405.

[2] Froude, "His. of Eng.," iii., 246.

[3] Wriotheley's Chronicle, ex Blyth.

[4] Amos, "The Great Oyer of Poisoning," Lond., 1846.

fessions of young widows. An investigation led to the discovery of a secret society, which met at the house of Hieronyma Spara, who, in return for "charity," dispensed an "acquetta" so graduated in potency as to cause the death of those to whom it was administered within the period of time desired. La Spara and thirteen of her companions were hanged, a large number of the culprits were whipped half-naked through the streets of Rome, and some of the highest rank were punished with fines and banishment. The Pope caused the papers to be brought to St. Angelo that the iniquitous secret should not become known. At about the same time, but somewhat later, the still most notorious Toffana carried on a similar traffic at Naples, where she was arrested in 1709, but escaped to a monastery, whence she continued to dispense *Manna di San Nicola di Bari*, until she was again apprehended and executed, after having caused the death of six hundred persons, probably in 1719, she being then about seventy years of age.[1] The most fantastic statements have been made concerning the nature of the poison used by Spara and Toffana. The most trustworthy contemporaneous evidence, however, is that it was an aqueous solution of white arsenic.[2] The statement of the Abbé Gagliani

[1] The Spara is said to have been the pupil of Toffana, as both were Sicilians. If there was any relation between them, however, it was more probably the reverse. La Spara was certainly executed during the pontificate of Alexander VII., probably in 1659. From internal evidence in the letter of Gavelli to Hoffmann (quoted in the next note) Toffana was a prisoner in Naples at least as late as 1718. If she was then seventy years of age she could only have been eleven years old when La Spara was executed. The statement of Keyster, that the Toffana was still alive at Naples in 1730, is, if true, proof positive that Toffana was the successor of La Spara in point of time.

[2] Halle ("Gifthistorie," 1787, p. 80) says that it was prepared by bandits from the froth of the mouths of victims tortured to death; and Blyth ("Poisons," p. 11), after stating that two popes, Pius III. (*ob.* 1503) and Clement XIV. (*ob.* 1775) between whose deaths nearly two hundred and seventy-two years elapsed, were both victims of Toffana, advances the theory of the formation of putrid arsenical derivatives, based upon alleged facts for which we can find no authority. As against these fables we have the contemporaneous statements of Wepfer and Hoffmann: Wepfer ("De Cicuta aquatica," etc., Basil, 1679, p. 295) says that J. A. Bartholin, of Turin, etc., had recently told him that the poison with which certain Roman (female) poisoners had, under the impious cloak of religion, caused much damage and had suffered the merited punishment for their crimes under Alexander VII. was arsenical, and called by them "acquetta." This clearly refers to the poisonings of La Spara. Fr. Hoffmann ("Med. rat.," ii., pr. ii., c. 12, s. xix., ed. Genev., 1753, p. 198), after referring to the poisonings under Alexander VII., reproduces this letter received by him from Garelli, the physician of

quoted and apparently favored by Beckmann,[1] that it was a mixture of opium and cantharides, is not consistent with the further statement of the abbé that it was as limpid as rock water, and without taste; nor with that of Haller,[2] that it was without taste or odor. The absence of taste and odor is also inconsistent with the supposition that it was a putrid product. On the other hand, an aqueous solution of arsenic trioxid is odorless and colorless and practically tasteless.

While information concerning the Spara and Toffana poisonings is only to be found in fragmentary references of an unofficial character, the record of the contemporaneous poisonings in France by the Marchioness of Brinvilliers and her accomplices and imitators is quite as complete as in most cases of the present day. Indeed what would correspond to our "appeal books" in the cases of the Marchioness and one of her accomplices are extant.[3]

In 1665 two Italians, one named Exili, had associated themselves with a German apothecary, named Glazer, in the manufacture and sale of poisons at Paris. A priest, learning of the nefarious traffic through the confessional, informed the government, and the two Italians were lodged in the Bastile, where one died. Glazer does not appear to have been under suspicion at that time. At about the same time Gaudin de Sainte-Croix,

the Emperor: "Your elegant dissertation on the errors concerning poisoning recalls that slow poison with which a famous (female) poisoner now living in the prisons of Naples used fatally in six hundred cases. This is really nothing but crystallized arsenic dissolved by simple decoction in a large quantity of water, to which cymbellaria is added, with what object I know not. This was communicated to me by the Emperor, to whom the minutes of the trial, confirmed by the confession of the poisoner, were transmitted. The solution was known commonly at Naples as Aqua della Toffanina." The dissertation referred to in the above was first published in 1718. Professor Wegler, of Coblenz, in 1814, said that he had in his possession a bottle of the original Aqua Toffana, which was cubical in shape, of blue glass, with the letters S. N. blown in the glass, hermetically sealed, with one end drawn out to a point and the other bent into a hook, and filled with a perfectly clear and transparent liquid. (Jahrb. d. Staatsarznk., 1814, vii., 425.)

[1] "Hist. Inventions," ed. Bohn, 1884, i., 47–63.

[2] Vorlesungen, Bern., 1784, ii., 191.

[3] "Factum pour Dame Marie Magdelaine d'Aubray, Marquise de Brinvilliers, Accusée, contre Dame Marie Therese Mangot, veusve du Sieur d'Aubray, Lieutenant Civil, Accusatrice; et Monsieur le Procureur-Général," Paris, 1676. "Factum du Procez extraordinairement fait à Lachaussée, Valet de Saint Croix, pour raison des empoisonnements des Sieurs d'Aubray," Amsterdam, 1676.

a captain of cavalry, was committed to the Bastile at the instance of the civil lieutenant Dreux d'Aubray, in consequence of an intrigue between the captain and the daughter of d'Aubray, the Marchioness of Brinvilliers. Exili and Sainte-Croix were confined in the same room. At the end of a year Sainte-Croix was liberated, after having learned the secrets of Exili and the address of Glazer, and renewed his connection with the Brinvilliers. Glazer and Sainte-Croix manufactured poisons with which the Marchioness poisoned a maid-servant, her father and two brothers, and attempted to poison her sister with the aid of Sainte-Croix and of his valet, Lachaussée. They also established a commerce in poisons, their most lucrative client being one Reich de Penautier, who was tried for poisoning in 1673.[1] Both Glazer and Sainte-Croix fell victims of their own poisons. The apothecary became ill and died, and Sainte-Croix, too weak to leave his dwelling, set up a furnace there and was found dead beside it.[2] After the death of Saint-Croix the authorities seized the contents of his laboratory, among which were numerous poisons: corrosive sublimate, opium, antimony, lapis infernalis; and papers implicating the Marchioness and Penautier; Lachaussée was seized and executed in 1673, after having confessed to the particulars of the poisoning of the brothers of Brinvilliers. The Marchioness escaped to Belgium, where she was subsequently captured, brought back to Paris, tried and executed, after having confessed her crimes, July 17th, 1676.

The execution of the Brinvilliers did not put a stop to the poisonings, and in 1679 a special commission,[3] the *chambre ardente* or *chambre des poisons*, was appointed to try cases of alleged poisoning, and sat, first at the arsenal, and afterwards at the Bastile, until it was discontinued in 1680. Two years later, 1682, an edict for the punishment of poisoners was registered in the Parliament.[4] The chambre ardente condemned two fortune-tellers, La Voisin and La Vigoureux, who sold poisons, called "succession powders," which, like that of the Brinvilliers, consisted of corrosive sublimate. La Voisin was

[1] "Factum contre Maitre Pierre-Louis Reich de Penautier."

[2] It seems highly probable that their deaths were due to poisoning by hydrogen arsenid.

[3] "Mémoirs sur la Bastille," Paris, 1789, i., 116.

[4] Pitaval, "Causes Célèbres," i., 317.

burnt alive February 22d, 1680. Two priests, named Le Sage and Guibourg, and some forty other persons were tried by the same tribunal on charges of poisoning. Among those arraigned before this tribunal were François Henri de Montmorenci, duke of Luxembourg; the Duchesse de Bouillon, niece of Mazarin; the Comtesse de Soissons, mother of Prince Eugene; the Comtesses de Polignac and du Roure; the Conte de Soisson and the Marquis de Fenquières, whose prosecutions were probably due to the political machinations of Louvois and the Montespan.[1]

La Voisin was and will probably remain the last "poisoner for hire" in a civilized community,[2] as in the present era of phosphorus matches, rat poisons, soothing syrups, and indiscriminate embalming such a traffic would not be lucrative. If the occupation of the mercenary poisoner is no more, it is, however, because the principal, since the beginning of the eighteenth century, has found it more safe and economical to do the sinister work himself—or herself. After a century and a quarter, during which only isolated cases are reported, and during which repeated poisonings by the same criminal were uncommitted or undetected, the theatre of such crimes was transferred to Germany: In 1803 the widow Ursinus, a "Frau Geheimrathin," in Berlin, was brought to trial and condemned to life imprisonment for one of at least four poisonings of which she was guilty.[3] Soon afterward, September 17th, 1811, Anna Margaretha Zwanziger was executed in Bavaria to expiate the death of one of the several victims of her sixty or seventy attempts at poisoning, she having conceded before execution that

[1] Concerning the poisonings of this period, see: Flandin, "Poisons," Paris, 1846, i., 100–120; Pitaval, "Causes Célèbres," La Haye, 1746, i., 267–326; Mémoirs . . . sur la Bastille, London, 1789, i., 113, 115, 116, 125, 127, 136, 140, iii., 306; Anquetil, "Louis XIV.," Paris, 1819, i., 215; Beckmann, *loc. cit.*; Dumas, "Crimes Célèbres" "Lettres de Mad. de Sévigné," ed. 1726; pp. 140, 143, 154, 158–161, 168–170, 196–198; "Letres historiques et galantes par Mad. de C——," Cologne, 1709–11, ii., 101; iv., 376; and the Factums and Memoirs of the trials.

[2] If we except that early manufacturer of soothing syrup, whose case is referred to by Fodéré ("Med. Lég.," iv., 20), who about 1770 "practised baby farming," and who, before execution, confessed that it was her practice to prepare the food of the infants confided to her with a decoction of poppies in place of ordinary water; "which put them to sleep and prevented their crying; soon they did not eat and perished in marasmus." Concerning professional poisoners in India at the present time, see Irving, "Ind. Ann. m. Sc.," Calc., 1862–63, xv., 46, 1864, s. xvii., 1.

[3] Hitzig und Häring, "Neue Pitaval," ii., 161.

"her death would be a benefit to mankind, as it would be impossible for her to abstain from poisoning."[1] On April 20th, 1831, Gesche Margaretha Gottfried was executed at Bremen, having during a criminal career of fifty years killed forty persons by poison.[2] The imitativeness of poisoners, so frequently observed at the present time, was exemplified in the case of Gesche Margaretha Brockmann, who, having read Voget's account of her namesake's career, was prompted to poison her father, brother, sister, and child, for which she was condemned to life imprisonment, that being the maximum penalty in Oldenburg at the time.[3] In the male line the case of Dr. Castaing in France may be cited, he having been executed for one murder by poison, although suspected with good reason of thirteen besides.[4] These were the worthy precursors of Mary Ann Cotton (1873), Maria Katharina Swanenburg (1883), Flannagan and Higgins (1884), Pastré-Beaussier (1886–88), and Sarah Robinson (1889). But the *causes célèbres* of modern times will be sufficiently discussed in the sequel.

Previous to the nineteenth century the methods for the detection of poisoning depended, so far as they were of any value, upon the circumstances attending the administration. Apart from these they were mere superstitions, modified only to a slight extent, even as late as the middle of the eighteenth century, by the more extended knowledge of post-mortem appearances in the cadaver caused by disease which followed the permission to dissect the human subject.

It was believed by the Romans that the bodies of those who were poisoned resisted fire better than those of persons who died from natural causes, and that the heart particularly was incombustible. Thus Pliny[5] tells us that Vitellius, in his oration accusing Piso of poisoning Germanicus, used the argument that the heart of the deceased could not be cremated; and that the defence of Piso was that this was due to heart disease. Suetonius[6] mentions lividity of the body as another sign of poisoning.

Lividity of the surface and incombustibility of the heart

[1] *Op. cit.*, ii., 218–255.

[2] *Op. cit.*, ii., 256–359; Voget, "Lebensgeschichte der Giftmörderin Gesche Margarethe Gottfried," Bremen, 1831.

[3] Vrtjrschr. f. ger. Med., 1855, vii., 300–335.

[4] "Causes Criminelles," Paris, 1828, iv., 1–103.

[5] "Hist. Nat.," xi., 72.

[6] "Caligula," cap. i.

were recognized as evidence of poisoning even to the eighteenth century; and Zacchias, who considers the former a reliable sign, while stating his belief that the latter is fallacious concedes that the opposite opinion is entertained by "that very learned man Fortunius Nattius."[1]

The statements of Zacchias afford a good account of the history of this question up to his time, and its condition at the time, and is a curious combination of ancient superstitions and glimmerings of future developments. After devoting a section[2] to substantiating his opinion that poisons, which are the causes of certain diseases, may be generated in the body as well as introduced from without, he discusses in three sections (Quæst. vi., vii., viii.) the signs of the poisoning and the means of distinction between the effects of those introduced from without and those of such as are produced in the body (diseases). Different poisons cause different signs. It cannot be determined from the signs alone whether a person has died of poison from without or from within.[4] But if we consider a particular case and make use of certain conditions and conjectures, as well as of the signs, we may attain positive indication of administered poison. We may have positive knowledge that a man was not poisoned. The signs of poisoning are divided into antemortem and post-mortem. The former are derived from the condition of the patient at the time, and the circumstances of the attack. Symptoms arising suddenly in a healthy person are indication of poisoning. But various natural foods are poisonous to some persons. All poisons have an offensive and abominable odor and a horrible taste. When poison is taken the gullet is wounded, the mouth at the fauces is constricted, there is itching, burning and inflammation in the mouth, etc., etc. (an account of the symptoms caused by corrosives and powerful irritants). But while the symptoms of poison are usually sudden in their appearance, they may be long delayed. Of the post-mortem signs the most valuable is the lividity of the body,[5] the hair and nails fall off easily or spontaneously, and the latter become black after death.[6] The cadavers of those who die by poison are not eaten by wild

[1] The reference to this "Nattius" is not contained in the edition of Lips., 1630, but is found in those of Frankfurt, 1666 and 1688, Norimb., 1726, and Venice, 1737 and 1751. We have not seen the intermediate editions of Amst., 1651, and Avignon, 1660, 1655, and 1657. We know of no medical writer of this name. Probably the reference is to the work of Fortunatus Fidelio (see vol. i., p. xv.).

[2] "Quæst. Med. Leg.," lib. ii., tit. ii., Q. iv.

[3] See "Causation."

[4] The word "signs" (*signa*) is not used as synonymous with "symptoms" (*symptomata*), but includes these as well as post-mortem appearances.

[5] Maxime in consideratione habet colorem cadaveris quem primum citrinum apparere dicit, aut sublividum; post unius aut vero alterius horæ spatium lividum ac nigrum evadere (Quæst. vii., 30).

[6] We have known an alleged toxicological expert, in the year of our

beasts or birds. The occurrence of froth at the mouth, and the lack of combustibility of the heart are fallacious signs. Among the "less safe" signs are ulcerations (or sphacelation) of the mouth, gullet, stomach, and intestines; discolorations and coldness of the internal organs; and coagulation of the blood in and around the heart.[1]

Poisoning from without may be distinguished from auto-intoxication by the lack of fever and the occurrence of thirst in the former. The existence of contagion points away from poison as the cause of death. Rapid putrefaction indicates death from internal poison, as the bodies of poisoned persons putrefy with difficulty. The occurrence of intermissions indicates disease. Vomiting caused by poison is persistent. The matters vomited are to be tested by administration to hens.

It is not astonishing that in the dawn of modern chemistry Plenck should, in discarding these ancient superstitions, have made the extreme statement that the only certain sign of poisoning is the botanical character of a vegetable poison or the chemical identification of a mineral poison found in the body.[2]

The development of methods of medico-legal recognition of poisoning to the point which it has reached at the present time, and which we believe to be still far short of what it will attain in the future, has followed the advances in those sciences upon which they mainly depend, chemistry and pathology.

Of the two forms of post-mortem investigation of poisoning, toxicological chemistry was the first to acquire a scientific foundation, as chemistry, in its simpler branches, was a well-developed science half a century before the dawn of modern pathology. As mineral chemistry preceded organic, so the detection and identification of mineral poisons became possible and certain at an early date, while chemical proof of the presence of at least some of the vegetable and organic poisons is even at the present time unattainable by the means at our disposal.

In the history of toxicological chemistry six events may be cited as marking important stages in the development of the science: 1st. The first practical application of the previously ob-

Lord 1895, to seriously inform the coroner that he was sure that a death was due to poison because the nails of the cadaver had turned blue.

[1] Itaque signa hæc hactenus in cadaveribus observata et minus tuta sunt, et non habent fortasse aliqua eorum omniumodam veritatem. . . . Tamen in cadaverum dissectione pluries id ipsum apparet, absque eo, quod veneno fuerit extincta (Quæst. vii., 34–37).

[2] "Elementa Med. et Chir. forens.," Viennæ, 1781, p. 36. Unicum *signum certum* dati veneni est *notitia botanica* inventi veneni *vegetabilis*, et *analysis chemica* inventi veneni mineralis.

served properties of hydrogen arsenid to the detection of arsenic by James M. Marsh, in 1836,[1] whose method, in a greatly modified and improved form, still remains the most delicate and reliable test for arsenic. 2d. In 1839 Orfila extracted notable quantities of arsenic from the liver, spleen, kidneys, heart, and muscle of the assassin-suicide Soufflard. This was the first instance of the extraction of *absorbed* arsenic from the human cadaver, previous analyses having been confined to the alimentary canal.[2] 3d. In 1841[3] Fresenius and von Babo devised a scheme for the sytematic search for all mineral poisons, which in a slightly modified form is still the best method, save in exceptional cases where certain poisons may be left out of consideration. 4th. The separation of the vegetable alkaloids from medicinal and poisonous plants, beginning with the investigation of opium by Sertürner in 1805, opened up a new field in toxicology as in materia medica. The earliest case of death from the administration of a vegetable alkaloid was that of C. Auguste Ballet, who was poisoned at St. Claud May 29th, 1823, by his relative and physician Castaing, who was convicted, mainly on moral evidence, and executed. 5th. In connection with the investigation of the cause of death of Gustave Fougnies, poisoned by his brother-in-law, the Count Bocarmé, in 1850, by the forcible administration of nicotin, Stas devised a process for the separation of alkaloidal poisons from the cadaver, which, in the more or less modified form, is that still in use.[4]

[1] "Description of a New Process of Separating Small Quantities of Arsenic from Substances with which it is Mixed" (Edinb. New Philos. J., Oct., 1836, p. 229–236). Scheele had, as early as 1775, obtained a gaseous compound from zinc and arsenic, which he found to be combustible, and to deposit elementary arsenic [arzenikkönig] on the walls of the vessel in which it was burnt ("Phys. u. chem. Werke," Berlin, 1793, ii., 136–137; "Kongl. Svensk. Vet. Ak. Handl.," 1775, xxxvi., 265–294). In 1803 Trommsdorff was at the edge of the discovery and found that arsenical hydrogen was evolved by the action of dilute sulfuric acid on arsenical zinc, and that when the gas was passed through a tube of sufficient length arsenic was deposited on its walls (J. of Nat. Phil., Chem., etc., 2 Ser., i., 1802–3). Serullas in 1821 proposed to utilize the decomposition of the compound of hydrogen and arsenic, obtained by the action of water on an alloy of arsenic, antimony, and potassium, to detect small quantities of arsenic in cases of suspected poisoning (J. d. Phys., etc., 1821, xcv., 154). Marsh was, however, the first to make the reaction practically available, and to suggest a simple form of apparatus, which has been subsequently greatly perfected.

[2] Mém. Ac. roy. Méd., 1840, viii., 376–422.

[3] Ann. d. Ch. u. Ph., 1844, xlix., 308.

[4] Bull. Ac. roy. d. méd. d. Belg., Bruxelles, 1851–52, xi., 202–312.

6th. In 1874 Selmi showed that the substance, supposed upon an insufficient analysis to be morphin, which had been extracted from the cadaver of the widow Sonzogno, of Cremona, after thirteen days' burial, was really not morphin, but a putrid product, a ptomaïn or cadaveric alkaloid.[1]

The history of the development of the pathology of poisoning may in one sense be said to be that of pathology itself, for one of the most important applications of pathology, both gross and microscopic, to toxicology is in the determination of the existence or absence of so-called natural causes of death. So far as positive evidence of poisoning from post-mortem appearances is concerned, the history of toxicological pathology remains to be made. While little or no conclusive evidence of the action of the true poisons can be obtained from the observation of gross appearances, there is good reason to hope that the investigations of microscopic changes caused by the action of toxic agents, which have been but recently begun, may in the near future afford the means of solving some questions which are now unanswerable.

BIBLIOGRAPHICAL.[2]

A. WORKS EXCLUSIVELY TOXICOLOGICAL.

I. Printed Since 1850.

Accettella, N. . Prontuario di tossicologia, 8vo, Torino, 1878.

Agnolesi, P. : Vade mecum di tossicologia, 32mo, Napoli, 1881.

Autenrieth, W. : Kurze Anleitung zur Auffindung der Gifte, etc., 8vo, Freiburg i. B., 1892.

Bandlin, O. : Die Gifte u. ihre Gegengifte, 12mo, 3 v., Basel, 1869–73.

Barillot, E. : Traité de chimie légale, etc., 8vo, Paris, 1894.

Baumert, G. : Lehrbuch d. gerichtlichen Chemie, 8vo, Braunschweig, 1889–93.

Bellini, R. : Della importanza degli studi tossicologici, etc., 8vo, Pisa, 1860.

——— Lezioni sperimentali di tossicologia, etc., 12mo, Firenze, 1865.

——— Manuale di tossicologia, 8vo, Pisa, 1878.

[1] See Ptomaïns, in the division of Special Toxicology.

[2] Theses, dissertations, and articles in medical and other journals are not included. For these, as well as for quite complete bibliographies of individual poisons, the reader is referred to the Index Catalogue of the Library of the Surgeon-General, Washington, 16 vols., 4to, 1880–1895; and the Index Medicus. Partial bibliographies of individual poisons will be found in the division of Special Toxicology.

Benoit, S. : De l'empoisonnement criminel, etc., 8vo, Paris, 1888.
Blyth, A. W. Poisons, etc., 8vo, London, 1884; New York, 1885.
Bocker, F. W. Die Vergiftungen, etc., 8vo, Iserlohn, 1857.
Bonnal. Leçons sur l'empoisonnement, Bordeaux, 1871.
Brouardel, P. and Ogier, J. : Le laboratoire de toxicologie, 8vo, Paris, 1891.
Chandelon, T. : Traite de toxicologie, etc., 8vo, Liége, 1888.
Chapuis, A. : Precis de toxicologie, 16mo, Paris, 1882. Deuxième ed., 16mo, Paris, 1889.
Chevallier, A. : Traité de toxicologie, etc., Paris, 1868.
Chiaja, S. : Enchiridio di tossicologia, etc., Napoli, n. d. Ed. terz., Napoli, 1858 (II.)
Coutance, A. : Empoisoneurs—Empoisonnés, etc., 8vo, Paris, 1888.
Cracau, G. N. B. Gift und Gegengift., 32mo, Leipzig, 1887.
Crosby, T. R. : Chart of Poisons, fol. ; n. p., n. d.
Dragendorff, G. : Die gerichtlich-chemische Ermittelung der Gifte, etc. 8vo, St. Petersburg, 1868. Zweite Auflage, 8vo, St. Petersburg, 1876. Dritte Aufl., 8vo, Göttingen, 1888. Vierte Aufl., 8vo, Göttingen, 1895.
——— *Ibid.* Trad. de l'Allemand par E. Ritter, 8vo, Paris, 1873. Deuzième ed., trad. p. L. Gautier, 1886.
Duflos, A. : Prüfung chemischer Gifte, etc., 8vo, Breslau, 1867.
——— Handbuch der angewandten gerichtlich-chemischen Analyse der chemischen Gifte, 8vo, Breslau, 1873.
Dyrenfurth, M. : Die Gifte und Gegengifte, Berlin, 1879.
Eberle, J. J. : Notes on Toxicology, etc., 12mo, Easingwold, 1876.
Fabbri, A. : Del modo di riconoscere i principali avvelenamenti, 8vo, Firenze, 1851.
Falck, F. A. : Lehrbuch d. praktischen Toxikologie, 8vo, Stuttgart, 1880 (II.).
Froehner, E. : Lehrbuch der Toxikologie für Thierärzte, 8vo, Stuttgart, 1890.
Galtier, C. P. : Traité de toxicologie générale, etc., 8vo, Paris, 1855.
——— Traité de toxicologie médicale, etc., 2 v., 8vo, Paris, 1855 (II.).
Georgantas, A. D. : *Μέθοδος πρὸς ἀνίχνευσιν δηλητηρίων*, fol., Ἐν Ἀθήναις, 1876.
Guérin, G. : Traité pratique . . . de recherches toxicologiques, 8vo, Paris, 1893.
v. Hassett, A. W. M. Handleiding der vergiftleer, etc., 2 v., 8vo, Utrecht, 1850–54.
——— Handbuch der Giftlehre, etc., bearb. v. G. B. Henkel, 2 Th., 8vo, Braunschweig, 1862.
Helwig, A. : Das Mikroscop in der Toxicologie, 8vo, Wiesbaden, 1864; also 8vo, 16 pl., Mainz, 1865.
Hendess, H. : Allgemeine Giftlehre, 8vo, Berlin, 1880.
Hermann, L. : Lehrbuch der experimentellen Toxicologie, 8vo, Berlin, 1874.
Hildebrandt, H. : Kompendium der Toxikologie, 8vo, Freiburg, 1893.
Hoh, T. : Gift und Kontagium, etc., 8vo, Leipzig, 1862.

Horsley, J. : The Toxicologist's Guide, etc., 12mo, London, 1866.
Hugounenq, L. : Traité des poisons, etc., 8vo, Paris, 1890.
Husemann, Th. and A. : Handbuch der Toxicologie, etc., 8vo, Berlin, 1862. Supplementband, 8vo, Berlin, 1867.
Jacob, J.: Poisons et Contrepoisons, Paris, 1877.
Joannes, J. G. : Ἐγχειρίδιον τοξικολογίος, 8vo, Ἀθήναις, 1869.
Klein, J. : Elemente der forensisch-chemischen Analyse, 16mo, Hamburg u. Leipzig, 1890.
Kobert, R. : Kompendium der praktischen Toxikologie, 8vo, Leipzig, 1884. Zweite Aufl., 8vo, Stuttgart, 1887. Dritte Aufl., 8vo, Stuttgart, 1894.
——— Lehrbuch der Intoxicationen, 8vo, Stuttgart, 1893.
Kupferschlaeger, S. : Eléments de chimie toxicologique, etc., 8vo, Liége et Bruxelles, 1879.
Lewin, G. : Toxikologische Tabellen, etc., 8vo, Berlin, 1856.
Lewin, L. : Lehrbuch der Toxicologie, 8vo, Wien, 1885.
Liebermann, L. : Anleitung zur chemischen Untersuchung, etc., 8vo, Stuttgart, 1877. Zweite Aufl., 8vo, Stuttgart, 1883.
Lopez, Arrojo L. : Toxicologia clinica, 2d ed., 8vo, Madrid, 1889.
Lopez, Dueñas S., and Lopez-Giron, J. : Resumen general de venenos, etc., fol., Madrid, 1872.
Luchini, A. : Chimica applicata alla tossicologia, 8vo, Napoli, 1888.
Ludwig, E. : Medicinische Chemie, 8vo, Wien u. Leipzig, 1885. Zweite Aufl., 1895.
——— Chemie und Rechtspflege, 8vo, Wien, 1892.
de Macedo Pinto, J. F. : Toxicologia judicial e legislativa, etc., 8vo, Coimbra, 1860.
Mangin, A. : Les poisons, 8vo, Tours, 1869.
Mata, P. : Compendio de toxicologia, etc., ed. 3, Madrid, 1857. Ed. 5, Madrid, 1875.
Meister, J. C. F. : Leitfaden zu Vorlesungen über Gifte, etc., 8vo, Breslau, 1856.
Mohr, F. : Chemische Toxicologie, etc., 8vo, Braunschweig, 1874.
——— Toxicologie chimique, trad. p. L. Gautier, 8vo, Paris, 1875.
Monferranti, R. : Manuele pratiche dei ricerche tossicologiche, 8vo, Napoli, 1872.
de Montgomery, H. : Essai de toxicologie, etc., 8vo, Paris, 1878.
Naquet, A. : Précis de chimie légale, 16mo, Paris, 1872.
——— Legal Chemistry. Transl. by J. P. Battershall, 16mo, New York, 1884.
Orfila, P. [M. J. B.] : Traité de toxicologie, cinquième ed., 2 vol., 8vo, Paris, 1852 (ii.).
——— Leçons de toxicologie, 8vo, Paris, 1858.
——— Zwölf Vorlesungen über die Lehre von den Vergiftungen, etc., Deutsche v. Händel, 8vo, Weimar, 1858.
——— Lehrbuch der Toxicologie, a. d. franz. m. Zusätz. v. G. Krupp, 2 Th., 8vo, Braunschweig, 1852–53.
Otto, F. J. : Anleitung zur Ausmittelung der Gifte, etc., 8vo, Braun-

schweig, 1856. Zweite Aufl., 8vo, Braunschweig, 1857. Dritte Aufl., 8vo, Braunschweig, 1867. Vierte Aufl., nach dem Tode des Verfassers herg. v. R. Otto, 8vo, Braunschweig, 1870. Fünfte Aufl., 8vo, Braunschweig, 1875. Sechste Aufl., 8vo, Braunschweig, 1884. Zweiter Abdruck, 1892.

——— On the Detection of Poisons. Notes by Prof. W. Elderhorst, 8vo, London, 1857, New York, 1857.

——— Instruction sur la recherche des poisons, trad. d. l. 4ème ed. all. par G. E. Strohl, 8vo, Paris, 1872.

Pascal, N. : Empoisonnements, 8vo, Paris, 1871.

Pelikan, E. : Beiträge zur gerichtlichen Medicin, etc., 8vo, Würzburg, 1858.

Poison (the) Register, etc., 3d ed., 12mo, New York, 1891.

v. Praag, Leonides t'. Opuijrda : Leerboek voor practische Giftleer, 2 Th., 8vo, Utrecht, 1871.

Rabuteau, A. P. A. : Elements de toxicologie, etc., 12mo, Paris, 1873.

Rabuteau and Bourgoin, E. : Elements de toxicologie, etc., 2ème ed., 8vo, Paris, 1887.

Reale, N. : Lezioni di chimica farmaceutica e tossicologica, 8vo, Napoli, 1877.

Reese, J. J. : A Manual of Toxicology, etc., 8vo, Philadelphia, 1874.

Ricci, A. : Prontuario di tossicologia clinica, 8vo, Milano, 1882.

Sainz y Rozas, J. A. : Tratado de toxicologia, etc., 2 ed., 12mo, Zaragoza, 1874.

Sbiriziolo, M. : Trattato . . . di tossicologia, etc., 2 vol., 8vo, Napoli, 1885.

——— Vade mecum degli avvelenamenti, etc., 16mo, Napoli, 1888.

Schmid, W. : Anleitung zur . . . Untersuchungen für Staatschemiker, 8vo, Zurich, 1868.

Schmidt, G. A. : Alfabetisk ordnet Fortegnelse over Gifte og Modgifte, 8vo, Kjobenh., 1883.

Schneider, F. C. : Die gerichtliche Chemie, etc., 8vo, Wien, 1852.

Schneider, K. F. R. : Deutsches Giftbuch, 2d Aufl., 8vo, Wittenberg, 1861.

Sonnenschein, F. L. : Handbuch der gerichtlichen Chemie, 8vo, Berlin, 1869. Zweite Aufl., neu bearb. v. A. Classen, 8vo, Berlin, 1881.

——— Handbook der geregtelijke scheikunde, etc., door. J. F. Snyder, 8vo, Amsterdam, 1872.

Spica, P. : Chimica medico-farmaceutica, etc., 8vo, Feltre, 1892.

Supino, R. : Veleni e contravveleni, 12mo, Milano [1892].

Tanner, T. H. : Memoranda on Poisons, 3d ed., 18mo, London, 1872. 4th Amer. ed., Phila., 1879. 6th Am. ed., rev. by H. Leffmann, Phila., 1888. 7th Am. ed., rev. by J. J. Reese, Phila., 1892.

Tardieu, A. and Roussin, Z. : Étude médico-légale et clinique sur l'empoisonnement, 8vo, Paris, 1867. Deuzième ed., 8vo, Paris, 1875.

——— Die Vergiftungen, etc., Deutsch von F. W. Thiele u. H. Ludwig, 8vo, Erlangen, 1868.

Taylor, A. S. : On Poisons, etc., 2d ed., 12mo, London, 1859. 3d ed.,

12mo, London, 1875. Edited, with notes and additions, by R. E. Griffith, 8vo, Phila., 1848. 3d Amer. ed., 8vo, Phila., 1875.

——— Die Gifte, etc., Uebersetzt v. R. Seydler, 3 Bd., 8vo, Cöln, 1862-63.

Trousseau and Reveil: Compendio di tossicologia, Trad. da P. Pepere, 12mo, Napoli, 1864.

Valenti y Viro, I.: Tratado elemental de toxicologia, etc., 8vo, Barcelona, 1877.

——— Atlas de micro-quimica, etc., 4to, Barcelona, 1878.

Vauthier, J. Z. F.: Les poisons, etc., 8vo, Bruxelles, 1880.

Vitali, D.: Manuale di chimica tossicologica, 8vo, Milano, 1893.

Werber, A.: Lehrbuch der praktischen Toxikologie, 8vo, Erlangen, 1869.

——— Leerbock v. d. pract. vergiftleer. Bew. d. J. L. v. Praag en R. J. Opuijrda, 8vo, Utrecht, 1878.

Winckler, E.: Memoranda der gerichtlich-chemischen Prüfung auf Gifte, 12mo, Weimar, 1852.

Wormley, T. G.: Micro-chemistry of Poisons, etc., 8vo, 13 pl., New York, 1867. 2d ed., 8vo, 17 pl., Phila., 1885.

v. Wyss, H.: Kurzes Lehrbuch der Toxicologie, etc., 8vo, Leipzig u. Wien, 1895.

Yamasaki, Kensu: Mada K. Katai, 12mo, Tokio, 1894.

II. Between 1700 and 1850.

Addison, T. and Morgan, J.: An Essay on the Operation of Poisonous agents, etc., 8vo, London, 1829.

Anglada, J.: Traité de toxicologie, etc., 8vo, Paris, 1835. 12mo, Bruxelles, 1837.

Artus, W.: Anleitung zur Auffindung der Mineralgifte, 8vo, Leipzig, 1843.

Attumonelli, M.: Trattado de' Veleni, etc., 2 v., 4to, Napoli, 1785.

Augé, A.: Table toxicologique, etc., n. p., n. d.

Berlin, N. J.: Nachricht die gewöhnlichen Gifte chemisch zu entdecken, 8vo, Stockholm, 1845.

Bertrand, C. A. H. A.: Manuel médico-légale des poisons, etc., 8vo, Paris, 1817.

Buchner, A.: Toxikologie, etc., 2d Aufl., 8vo, Nürnberg, 1827.

d. Chiaje, S.: Enchiridio di tossicologia, etc., 8vo, Napoli, 1835.

Christison, R.: A Treatise on Poisons, etc., 8vo, Edinburgh, 1829. 2d ed., 8vo, Edinburgh, 1832. 3d ed., 8vo, Edinburgh, 1836. 4th ed., 8vo, Edinburgh, 1845. 1st Am. ed., 8vo, Phila., 1845.

——— Abhandlung über die Gifte, 8vo, Weimar, 1831, 1833.

Cooke, J.: A Treatise on Poisons, etc., 8vo, London, 1770.

Costill, O. H.: A Practical Treatise on Poisons, etc., 12mo, Phila., 1848; Phila., 1864.

Ducatel, J. T.: Manual of Practical Toxicology [condensed from Christison], 8vo, Baltimore, 1833.

Duflos, A.: Pharmakologische Chemie, etc., 8vo, Breslau, 1842.

——— Die chemische Heilmittel und Gifte, etc., Breslau, 1839.

DUVAL, M. : Sur la toxicologie, 4to, Paris, 1806.

DZONDI, K. H. : Ueber Kontagion, Miasmen und Gifte, 8vo, Leipzig, 1822.

FLANDIN, C. : Traité des poisons, etc., 3 vol., 8vo, Paris, 1846–53.

FRANK, J. : Handbuch der Toxicologie, 8vo, Wien, 1800. 2te Aufl., 8vo, Wien, 1803.

——— Manuel de toxicologie, etc., Trad. . . . par. L. H. J. Vrancken, 8vo, Anvers, An xi. [1803].

——— Manuale di tossicologia, etc., Trad. . . . di G. Mathey, 8vo, Milano, 1809.

FRICCII, M. [Frick, M.] : Paradoxa de Venenis, etc., 16mo, Aug. Vindel, 1710.

GIFTBUCH (vollständige), col. pl., 8vo, Sondersheim, 1817.

GMELIN, J. F. : Allgemeine Geschichte der Gifte, Erster Th., 8vo, Leipzig, 1776.

GUÉRIN, DE MAMERS : Nouvelle toxicologie, etc., 8vo, Paris, 1826.

HAGEN : Isagoge ad chemiam forensem, Regiomont, 1789.

HALLE, J. S. : Gifthistorie, etc., 8vo, Berlin, 1787.

HARMAND DE MONTGOMERY, T. : Essai de toxicologie, etc., 8vo, Paris, 1818.

HERGANG, K. G. : Vollständiges Giftbuch, etc., Sondershausen, 1815.

HOULSTON, T. : Bemerkungen über die Gifte, etc., Altenburg, 1786.

HÜNEFELD, F. L. : Lehrbuch der polizeylich-gerichtlichen Chemie, 8vo, Berlin, 1832.

KIRCHER, A. : De venenis liber, etc., 18mo, Grascii, 1739.

KOLBANI, P. : Ueber die herrschenden Gifte, etc., 8vo, Presburg, 1792.

——— Gifthistorie, etc., 8vo, Wien, 1798. Zweite Aufl., 8vo, Wien, 1807.

KÜHN, O. B. : Practische Anweisung zu gerichtlich-chemischen Untersuchungen, 8vo, Leipzig, 1829.

LINDER, J. : De venenis in genere, etc., 12mo, Lugd. Bat., 1708.

LINDES, W. : Praktische Anleitung zu . . . gerichtlich-chemischen . . . Untersuchungen, 8vo, Berlin, 1849.

LINDESTOLPE, J. [LINDER] : Liber de venenis, etc., 8vo, Lipsiæ, 1739.

LOWRIE, W. F. : Toxicologia, etc., 8vo, New York, 1832.

LUTTER, E. : Medicinische und chirurgische Bemerkungen über Gifte, etc., 8vo, Speyer, 1792.

MACLAGAN, D. : Contributions to Toxicology, etc., 8vo, Edinburgh, 1849.

MALLE, P. : Analyse toxique générale, 2 pl., 8vo, Strassbourg, 1839.

DE MAMERS, G. (See Guérin).

Manual of Toxicology, etc., 16mo. London, 1821.

MARC, C. C. H. : Allgemeine Bemerkungen über die Gifte, etc., 8vo, Erlangen, 1795.

MARX, K. F. H. : Die Lehre von den Giften, 2 Th., 8vo, Göttingen, 1827–29.

MEAD, R. : A Mechanical Account of Poisons, 4to, London, 1702. 2d ed., 1708. 3d ed., 1738. 4th ed., 1743. 5th ed., Dublin, 1729. 6th ed., Latin version, J. Nelson, London, 1737. 7th ed., 1750. 8th ed., Leiden, 1737. 9th ed., 1750. Napoli, 1739; 1758. Other editions, Amstel., 1749; Frankof., 1763; Göttingen, Ed. Oedems, 1749.

MÖLLER, H. : Die Lehre von den Giften und den Vergiftungen, 8vo, Quedlingen, 1825.

DE MONTMAHOUX, E. : Manuel médico-légale des poisons, etc., 18mo, Paris, 1824.

MÜLLER, A. G. : Tractatus de venenis, 8vo, Halæ, 1807.

MUTEL, P. : Des poisons, etc., 8vo, Paris, 1830. Also Ital., 8vo, Milano, 1831.

Neues vollständiges Handbuch der Giftkunde, etc., 16mo, Chur u. Leipzig, 1840.

ORFILA, P. [M. J. B.] : Traité des poisons, 2 vol. in 4 pt., 8vo, Paris, 1814–15. Deuxième ed., 2 vol., 8vo, Paris, 1818. Troisième ed., 2 vol., 8vo, Paris, 1827 [1826]. Quatrième ed., 2 vol., 8vo, Paris, 1843.

——— Recherche médico-légale des poisons, 8vo, Paris, 1845.

——— A General System of Toxicology, etc., transl. by J. G. Naucrede, 8vo, Phila., 1817.

——— Toxikologie, etc., bearb. v. J. A. Seemann, u. A. O. S. F. Karls, 2 vol., 8vo, Berlin, Posen u. Bromberg, 1829–31.

——— Tossicologia pratica, etc., comp. da D. L. Michelotti, 12mo, Livorno, 1827.

——— Appendix to the General System of Toxicology, etc., transl. by J. A. Waller, 8vo, London, 1821.

——— Allgemeine Toxicologie, etc., m. Zusätze u. Anm. v. Hermbstädt, 4 Bd., 8vo, Berlin, 1818–19.

——— *Idem.*, Deutsch von O. B. Kühn, 2 Bd., 8vo, Leipzig, 1839.

OTTO, C. : Haandbog i toxikologien, 8vo, Kjobenh., 1838.

PALDANUS, H. L. : Versuch einer Toxicologie, 8vo, Halle, 1803.

PALLAS, E. : Essai sur une nouvelle classification des poisons, etc., 8vo, Paris, 1822.

PLENCK, J. J. : Toxicologia, etc., 8vo, Viennæ, 1785. (A German ed. of the same place and date.) Ed. secunda, 8vo, Viennæ, 1801.

——— Tossicologia, etc., 12mo, Venezia, 1789.

——— Toxicologia, etc., 8vo, Madrid, 1816.

PLOUQUET, F. : Essai sur la nature des poisons, etc., 8vo, Caen, 1809.

PRESTWICH, J. : Dissertation on . . . poisons, etc., 8vo, London, 1775.

REMER, W. H. G. : Lehrbuch der polizeilich-gerichtlichen Chemie, 8vo, Helmstedt, 1803. 3te Aufl., 2 Bd., 8vo, Helmstedt, 1827.

RICORD-MADIANNA, J. B. : Recherches et expériences sur les poisons d'Amérique, etc., 4to, Bordeaux, 1826.

RINALDINI, B. : Trattado di tossicoscopia, etc., 8vo, Pavia, 1831.

ROBINSON : Toxicological illustrations, col. pl., 1835.

ROUPELL, G. L. : Illustrations of the Effects of Poisons, fol., London, 1833.

DE SALLE, E. : Table synoptique des poisons, etc., 2 ed., Paris, 1824.

SCHNEIDER, F. C. : Die gerichtliche Chemie, etc., 8vo, Wien, 1852.

SCHNEIDER, J. : Populäre Toxicologie, etc., 8vo, Frankfurt, 1838.

SCHNEIDER, P. J. : Ueber die Gifte, etc., 12mo, Würzburg, 1815. Zweite Aufl., 8vo, Tübingen, 1821.

SCHULTZE, J. E. F. : Toxicologia veterum, etc., 4to, Halæ, 1788.

DA SILVA PEREIRA E CUNHA, C. A.: Tractado dos venenos, etc., 8vo, Lisboa, 1815.

SOBERNHEIM, J. F. AND SIMON, J. F.: Handbuch der praktischen Toxicologie, 8vo, Berlin, 1838.

STOWE, W.: A Manual of Toxicology, etc., 24mo, London, 1823.

——— A Toxicological Chart, etc., fol., London, 1821. [Ind. cat. mentions 8th, 9th, and 13th eds., London, n. d. There was also a German transl., Weimar, 1821.]

STÜCKE, C.: Toxicologische Tabellen, etc., oblong 4to, Cöln, 1828. 2te Aufl., 1837.

SÜNDELIN, KARL: Handbuch der medizinischen Chemie, etc., 8vo, Berlin, 1823.

TADDEI, G.: Repertorio dei veleni e contravveleni, 2 T., 8vo, Firenze, 1835.

TAYLOR, A. S.: On Poisons, etc., 12mo, London, 1848.

——— *Idem.* Ed. with notes and additions, by R. E. Griffith, 8vo, Phila., 1848.

THOMSON, A. T.: Anleitung zur Erkenntniss und Behandlung der Vergiftungen, etc., bearb. v. A. Remmont, 12mo, Aachen, 1846. [A translation of an appendix to his "Conspectus of the Pharmacopœias."]

——— Versuch über den sichern und wirksamen Gebrauch der Gifte, etc., 8vo, Lipsiæ, 1774.

WIEDEMANN, C. R. W.: Anweisung zur Rettung . . . der Vergifteten, 8vo, Braunschweig, 1797.

ZALACZKOWSKI, T.: Tabella toxicologica, 8vo, Turici, 1836.

III. BEFORE 1700.

ABANO [VEL ABBANO; APONO; APONENSIS] PETRUS DE: Tractatus de Venenis, Mantuæ, fol., 1472; 4to, 1473. Venet., 4to, 1473, 1476, 1485, 1487, 1520, 1537; 8vo, 1550; fol., 1565, 1567, 1595. Paduæ, 4to, 1473. Roma, 4to, 1475, 1484–85, 1490. Mediol., 4to, 1475. Patav., 4to, 1476, 1487. Lipsiæ, 4to, 1489, 1500. Basil., 8vo. 1531. Marpur., 8vo, 1537, 1550, fol., 1565. Rivini, fol., 1548. Argent., 8vo, 1566. Paris, 8vo, 1567. Lyon, 16mo, 1593, 1595. Francof., fol., 1679. La Haye, 8vo, 1742, n. p., n. d. Sæc. xv., ed. ii.

ABRA, H.: De curandis venenis, etc., 12mo, Leovardiæ, 1601; Arnheim, 1603.

ANDROMACHUS: Theriaca, ed. Fr. Tidicæus, 4to, Tiguri, 1607.

ARDOYNIS, SANTES DE: Liber de venenis, etc., fol., Venet., 1492; fol., Basil., 1540, 1562.

ARMA, J. F.: De venenis dialogus, 8vo. Taurini, 1557.

ARNALDUS DA VILLANOVA: De arte cognoscendi venena, 4to, n. p., n. d.; 4to, n. p., n. d.; 4to, Mediol., 1475.

AVERROES: Liber de venenis, fol., n. p., n. d.; 4to, Paris, 1506. [Also in Regimen sanitatis, Leyden, 1517.]

BACCIUS, A.: De venenis et antidotis *προλεγομενα*, etc., 4to, Romæ, 1586.

BULGETUS, A.: De morbis venenatis venenisque tractatus, etc., 4to, Patav., 1657. [In his "De affectionibus cordis," etc.]

CARDANUS, H. : De venenorum differentiis, etc., 4to, Patav., 1563 ; Basil., 1564.

——— De venenis libri tres, fol., Lugd., 1663. [In his "opera."]

CARRERIUS, P. : Quæstio an dentur venena ad tempus ann Conciliatore edita, fol., Patav., 1548 ; Venet., 1548 ; 4to, Venet., 1565.

A CASTRO, R. : De morbis venenatis, etc., Tolosæ, 1636.

CODRONCHIUS, B. : De morbis veneficis ac veneficiis libri quatuor, 16mo, Venet., 1591, 1595 ; Mediol., 1613.

DIOSCORIDES : *περὶ δηλητηρίων φαρμάκων*—De venenis. *περὶ ἰοβόλων*—De venenatis animalibus. [Either separately or as books of his Materia Medica.] Of the great number of editions, the ed. princ. of the Greek text is the extremely rare fol., Venet., 1499, ap. Aldum Manutium, and the most recent and valuable that of Lips., 1829–30, 8vo, ed. Cur. Sprengel. The earliest Latin version is by Petrus de Abano, fol., Colle, 1478. There are also Latin versions by H. Barbarus, fol., n. p., n. d. ; J. Ruellius, fol., Paris, 1516 ; M. Vergilius, fol., Florent., 1518 ; P. A. Matthiolus, fol., Venet., 1554 [with extensive commentaries] ; and J. A. Saracenus, fol. [Francof.-a.-M.], 1598, of all of which there are numerous editions. There are also translations in Italian, Spanish, German, French, and Bohemian. [See Choulant, Handb. d. Bücherkunde f. d. ält. Med., Leipz., 1841, 76–82.]

A FONSECA, R. : De venenis eorumque curatione, 4to, Romæ, 1587.

FORESTI, P. : Liber de venenis et fucis, Lugd. Bat., 1606.

GAEHAUSEN, H. : Decisio trium quæstionum de veneficiis, 4to, Rintel., 1629.

GOCKEL, E. : Libellus de venenis, etc., 8vo, Aug. Vind., 1669.

——— Libellus alter de venenis, etc., 8vo, Aug. Vind., 1669.

GODELMANN, J. G. : Tractatus de Magis, Veneficiis et Lamiis, etc., 4to, Francof., 1591 ; 8vo, Norimb., 1676.

GRATAROLUS, G. : Universale consilium de præservatione a venenis, etc., 8vo, n. p., n. d. [1561].

GREVIN, J. : Deux livres des venins, etc., 2 T. in iv., 4to, Anvers, 1568.

——— De venenis libri duo, etc., 4to, Antw., 1571.

GUAINERIUS, A. : De peste et de venenis, etc., fol., n. p., n. d. [Sæc. XV.]

——— Liber de venenis, 4to, Papiæ, 1518 ; Paris, 1518 ; Lugd., 1525.

LIEBAULT, J. : De præcavendis curandisque venenis, etc., 8vo, Paris, 1577.

LOEBER, V. : Mantissa de venenis, etc., 8vo, Hamburg, 1671.

MATTHIOLUS, P. A. : [See Dioscorides.]

MERCATI, M. : Instruttioni sopra i veleni occultamenti ministrati, 4to, Roma, 1576.

MERCURIALIS, H. : De venenis et morbis venenosis, etc., 8vo, Francof., 1584 ; 4to, Patav., 1588 ; Venet., 1601.

MEYSSONIER, L. : Traité des maladies veneneuses, etc., 7th ed., 4to, Lyon, 1678.

MOIBANUS, J. : Giftjäger, etc., 1567.

NAUDÉ : An magnum homini venenis periculam, 12mo, Romæ, 1632.

NICANDER. Theriaca—Alexipharmaca. The first edition of the Greek text is accompanying that of Dioscorides, in the Aldine fol. of 1499. Other editions of Greek alone are: 4to, Venet., 1523, and 4to, Colon, 1530. Greek and Latin: 8vo, Paris, 1549 [Alexipharmaca alone]; 8vo, Valentiae, 1552 [Theriaca alone], 4to, Paris, 1557; 8vo, Florent., 1764; 8vo, Halae, 1792 [Alexipharmaca]; 8vo, Lips., 1816 [Theriaca]. Latin: 4to, Colon., 1531; 8vo, Francof., 1532. French: Anvers, 4to [with Grevin, *q.v.*]. The best editions are those of J. G. Schneider, Halae, 1792, and Lips., 1816.

[NICANDER]. Scholia vetere in Nicandri Alexipharmaca, etc. Rec. ab. E. Abel., 8vo, Berol., 1892.

PABST [or BABST], M.: Giftjagende Kunst, etc., 4to, Leipz., 1541.

PARAEUS [PARÉ], A.: Des venins et morsure des chiens enragez, etc., 4to, Paris, 1582.

PLINIUS, B.: Carmen de venenis et venenatis, etc., 8vo, Viteb., 1597; Norimb., 1689.

PONA, F.: Trattato de' Veleni e lor cura, 4to, Verona, 1643.

PONS, J.: Avertissement pour la préservation et cure générale contre les poisons, Lyon, 1634.

PONZETTUS, F.: De venenis, etc., fol., Venet., 1492; Romae, 1521, Basil., 1540, 1562.

PRAEVOTIUS, J.: De venenis eorumque Alexipharmacis, etc., 32mo, Mediol., 1646; 24mo, Lugd., 1660; 12mo, Hanov., 1666.

RAMESEY W.: *Οιανασια, και δηλητηρια.* Tractatus de venenis, or a treatise of poysons, etc., 16mo, London, 1661.

——— [RAMSAY, W.]: Life's security, or the names, natures and vertues of all sorts of venoms and venomous things, 8vo, Lond., 1665.

RAMIREZ, C. B. DE S.: De suspicione exhibiti veneni, etc., 4to, Colon., 1671.

RANCHINUS, F.: Traité des venins, etc. [In his Œuvres pharm.], 12mo, Lyon, 1624.

RUDIUS, E.: De morbis occultis et venenatis, fol., Venet., 1610.

SCHARFF, L. B.: *Τοξικολογια*, sive de natura venenorum, etc., 8vo, Jenae, 1678.

——— Antidotus prophylactica, etc., 16mo, Erffurth, 1698.

SCHENCK A GRAFENBURG, J.: De venenis, 8vo, Tübingen, 1597.

STUBING, J.: Liber de venenis, 4to, Vindeb., 1561.

THEATRUM DE VENEFICIIS, etc., fol., Frankf., 1586.

TRILLO, A.: Tratado general de todas las tres especias de venenos, etc., 8vo, Toledo, 1679.

VOETIUS, L.: Tractatus de venenis, 12mo, Venet., 1550.

ZUCCARINUS, M.: Methodus occurrendi venenatis corporibus, 4to, Neap., 1611.

B. MODERN WORKS ON MEDICAL JURISPRUDENCE, ETC., HAVING REFERENCE TO TOXICOLOGY.[1]

Abercrombie, J. : The Student's Guide to Medical Jurisprudence, 12mo, Phila. [London], 1885.

Bartholow, R. : A Practical Treatise on Materia Medica and Therapeutics, 7th ed., New York, 1889.

Beck, T. R. and J. B. : Elements of Medical Jurisprudence, etc., 12th ed., 2 v., 8vo, Phila., 1863. [In this edition the articles on poisons have been revised by B. W. McCready and S. St. John. The first edition (T. R. Beck alone) was in 2 v., 8vo, Albany, 1823.]

Bellini, R., and Filippi A. : Bibliotheca medico-legale, 8vo, Pisa, 1878. [T. iii. is Manuale di Tossicologia.]

Binz, C. : Intoxicationen. [In Gerhardt's Handbuch der Kinderkrankheiten, iii. Hälfte, Tübingen, 1878.]

Briand, J., Chaudé E., and Bouis, J. : Manuel complet de Médecine légale, etc., 10ème ed., 2 v., royal 8vo, Paris, 1880. [The Legal Chemistry by L. Bouis. The 1st ed. (Briand alone) was 8vo, Paris, 1821.]

Buck, A., et al. : A Reference Handbook of the Medical Sciences, etc., 8 v., 4to, New York, 1885, 1889. [Articles on Toxicology by B. V. Abbott, R. H. Chittenden, C. Harrington, W. B. Hills, H. Leffmann, F. Peyre Porcher, R. A. Witthaus, and H. C. Yarrow.]

Casper, J. L., Liman, K. : Handbuch der gerichtlichen Medicin, 8te Aufl., 2 v., 8vo, Berlin, 1889. [The 1st ed. was in 2 v., 8vo, Berlin, 1857–58 ; with an atlas of 9 col. pl. in 4to. There is an English translation, 4 v., 8vo, Lond. (New Sydenham Soc.), 1861–65, from the 3d Germ. ed. (1860) ; and a French translation, 2 v., 8vo, Paris, 1862, also from the 3d Germ. ed.]

Casper, J. L. : Klinische Novellen, etc., 8vo, Berlin, 1863.

Chapman, H. C. : A Manual of Medical Jurisprudence and Toxicology, 8vo, Phila., 1892.

Chevers, N. : A Manual of Medical Jurisprudence for India, etc., 3d ed., 8vo, Calcutta, 1870.

Devergie, A. : Médecine légale, etc., 3ème ed., 3 v., 8vo, Paris, 1852.

Elwell, J. J. : A Medico-Legal Treatise on Malpractice and Medical Evidence, 4th ed., 8vo, New York, 1881.

Eulenberg, H. : Handbuch der Gewerbe-Hygiene, 8vo. Berlin, 1876.

Falck, C. P. : Handbuch der gesammten Arzeneimittellehre, etc., i. Bd. 8vo [all publ.], fol., Marburg, 1850.

Falck, C. P. and F. A. : Beiträge zur Physiologie, . . . und Toxikologie, Stuttgart, 1875.

Filippi, A., Severi, A., and Montalti, A. : Manuale di medicina legale, etc., 2 v., 8vo, Milano, 1889. [The toxicology is contained in vol. i.]

Fodéré, F. E. : Traité de médecine légale, etc., 6 v., 8vo, Paris, 1813.

[1] Including works on toxicological chemistry.

Fresenius, C. R. Anleitung zur qualitativen chemischen Analyse, 16te Aufl., 8vo, Braunschweig, 1894–95.

——— Anleitung zur quantitativen chemischen Analyse, 6te Aufl., 2 v., 8vo, Braunschweig, 1875–87.

Gerhardt, C. [See Binz.]

Gribble, J. D. B., and Heber, P. . Outlines of Medical Jurisprudence for India, 3d ed., 8vo, Madras, 1892.

Guy, W. A., and Ferrier, D. . Principles of Forensic Medicine, 6th ed., 12mo, London, 1888.

Hamilton, A. McL., et al. . A System of Medical Jurisprudence, 2 v., 8vo, New York, 1894. [Articles on poisons by C. E. Pellew, W. S. Haines, and V. C. Vaughan.]

v. Hofmann, E. R. : Lehrbuch der gerichtlichen Medicin, 6te Aufl., 8vo, Wien, 1893.

v. Jaksch, R. [See Nothnagel.]

Kochler, R. : Handbuch der speciellen Therapie, einschliesslich der Behandlung der Vergiftungen, 3te Aufl., 2 v., 8vo, Tübingen, 1869.

Kornfeld, H. : Handbuch der gerichtlichen Medicin in Beziehung zu der Gesetzgebung Deutschlands und des Auslandes, 8vo, Stuttgart, 1884.

Legrand du Saulle, H., Berryer, G., and Pouchet, G. : Traité de médecine légale, de jurisprudence médicale et de toxicologie etc., 2ème ed., 8vo, Paris, 1886. [The toxicology by G. Pouchet.]

Lesser, A. : Atlas der gerichtlichen Medicin, 2 v., fol., Berlin, 1883–91. [Toxicology in vol. i.]

Lewin, L. : Die Nebenwirkung der Arzneimittel, etc., 2te Aufl., 8vo, Berlin, 1893.

Lutaud, A. : Manuel de médecine légale et de jurisprudence médicale, etc., 5ème ed., 12mo, Paris, 1892.

Mann, J. D. : Forensic Medicine and Toxicology, 8vo, London and Phila., 1893.

v. Maschka, J. . Handbuch der gerichtlichen Medicin, 4 v., 8vo, Tübingen, 1881–82. [The toxicology is contained in Bd. ii. and is by B. Schuchardt, M. Seidel, Th. Husemann, and A. Schauenstein.]

——— Sammlung gerichtsärztlicher Gutachten, etc., 4 v., 8vo, Prag u. Leipzig, 1853, 1858, 1867, 1873.

Mende, L. J. K. : Ausführliches Handbuch der gerichtlichen Medicin, etc., 6 v., 8vo, Leipzig, 1819–32.

Nothnagel, H., et al. : Specielle Pathologie und Therapie, 21 v., 8vo, Wien, 1894. [Bd. i., Vergiftungen, von R. v. Jaksch.]

Ogston, F. : Lectures on Medical Jurisprudence, 8vo, London, 1878.

Orfila, P. (M. J. B.) : Traité de médecine légale, 4ème ed., 4 v., 8vo, and Atl., Paris, 1848.

Paris, J. A., and Fonblanque, J. S. M. : Medical Jurisprudence, 3 v., 8vo, London, 1823.

Paulier, A. B., and Hétet, F. : Traité élémentaire de médecine légale, de jurisprudence médicale et de toxicologie, 2 v., 18mo, Paris, 1881.

Penzoldt, F., and Stintzing, R. : Handbuch der speciellen Therapie innerer Krankheiten, 6 v., 8vo, Jena, 1894. ["Die Vergiftungen" are

treated of in Bd. ii. by Binz, Schuchardt, Wollner, Husemann, Moeli, Erlenmeyer, Tuczek, and Liebreich.]

REESE, J. J. : Text Book of Medical Jurisprudence and Toxicology, 2d ed., 8vo, Phila., 1889.

SCHÜRMAYER, J. H. : Lehrbuch der gerichtlichen Medicin, 4te Aufl., 8vo, Erlangen, 1874.

SEYDEL, K. J. : Leitfaden der gerichtlichen Medicin, etc., 8vo, Berlin, 1895.

STILLÉ, A. : Therapeutics and Materia Medica, etc., 4th ed., 2 v., 8vo, Phila, 1874.

TAYLOR, A. S. : A Manual of Medical Jurisprudence, 12th ed., edit. by T. Stevenson, 12mo, London, 1891. 11th Am. ed., edit. by Clark Bell, 8vo, Phila., 1892. [The 1st ed. was 8vo, London, 1836, Elements of Medical Jurisprudence.]

——— The Principles and Practice of Medical Jurisprudence, 3d ed., edit. by T. Stevenson, 2 v., 8vo, London, 1883.

WHARTON, F., AND STILLÉ, M. : Medical Jurisprudence, 4th ed., edit. by R. Amory and E. S. Wood, 3 v., 8vo, Phila., 1884. [Vol. ii. contains the toxicology.]

WIENER, D. : Sammlung gerichtlich-medicinischer Obergutachten, 2te Aufl., 8vo, Berlin, 1893.

WOOD, H. C. : A Treatise on Therapeutics, comprising Materia Medica and Toxicology, etc., 7th ed., 8vo, Phila., 1890.

WOODMAN, W. B., AND TIDY, C. M. : Forensic Medicine and Toxicology, 8vo, Phila. (London), 1877.

v. ZIEMSSEN, H. : Handbuch der speciellen Pathologie und Therapie, 16 v., 8vo, Leipzig, var. dates. [Bd. xv., 2te Aufl., 1880, is devoted to Intoxicationen, articles by R. Boehm, B. Naunyn, and H. v. Boeck. In the English transl., New York, 1881, the supplement contains an article by E. S. Wood.]

GENERAL TOXICOLOGY.

DEFINITION.

No substance is under all circumstances deleterious. The poisonous quality is not one which is essential and necessary, but one which is manifested only under certain conditions. The genus "poison" does not exist as a class apart. Substances which are commonly known to be actively deleterious may, in certain forms and quantities and in certain conditions of the system, act as remedies, and substances which are in daily use as medicines, condiments, or foods may cause poisoning under certain conditions. It has been said that "the idea 'poison' cannot be circumscribed and that consequently its definition is impracticable."[1] But if there be no definition possible for the term "poison" because the same substance may under different conditions be a medicine or a poison, the term "medicine" must be equally incapable of definition. Yet the distinction between the poisonous and the medicinal action is most simple, and is popularly recognized as consisting in the harmful action of the former and the beneficial quality of the latter.

If, therefore, a definition be sought which shall set apart a class of substances called poisons, as a genus is differentiated in a classification of natural history, the search will be in vain. It is with the poisonous quality, not with the thing which exhibits it, that we have to deal.

In defining a poison we have to express the quality of its action and the method by which such action is produced, in so far as it differs from the methods by which similar results may be caused by agents other than poisons.

The effect of the poison is harmful and may be fatal. The damage caused by a poison is due neither to mechanical action, as with a bullet or club; nor to physical action, as with fire or lightning; nor to parasitic growth, as by trichina or tuberculosis; but to an interior action, chemical in its nature, upon some tis-

[1] Lewin: "Lehrbuch der Tox.," 1885, p. 4.

sue or fluid of the body whereby the composition of the tissue or the normal chemical changes constituting life are modified or interfered with. Poisons, like medicines, do not act until after they have entered the circulation.

A poison is a substance which, being in solution in, or acting chemically upon, the blood may cause death or serious bodily harm.[1]

In order that a substance shall be considered a poison within this definition, it is essential that it act after or by reason of its presence in the blood, and therefore chemical agents which may cause death or injury by their destructive action upon the skin or stomach do not come within it. Such substances are "corrosives."

A corrosive is a substance capable of causing death or injury by its chemical action upon a tissue with which it comes in direct contact.

The distinction between poisons and corrosives is in the method of application partly, but mainly in the nature of the subsequent effects. Thus arsenic applied to the skin acts as a true poison, because its local effects are insignificant as compared with the systemic action due to the passage of arsenic so applied into the blood. Sulfuric acid, on the other hand, when applied to the skin may be absorbed to some extent, but the destruction of cutaneous tissue is the principal injury, and resembles that caused by a burn or a scald.

The difference between poisons and corrosives seems, at first sight, hardly to warrant their separation into distinct classes. Indeed, many of the older toxicologists and some of recent date

[1] The following definitions more or less closely resemble that given in the text: "A poison is a substance which, when absorbed into the blood, is capable of seriously affecting health or of destroying life" (Taylor: "Poisons," 3d Am. ed., 1875, p. 18). "Poisons are substances which, introduced by absorption into the organism, cause structural alterations and functional derangements more or less grave, and can even, when their action reaches a high degree of intensity, cause death, or at least put life in danger" (Vulpian, quoted and adopted by Chapius, "Précis de Tox.," 2ème ed., 1888, p. 25). "A substance of definite chemical composition, whether mineral or organic, may be called a poison if it is capable of being taken into any living organism, and causes by its own inherent chemical nature impairment or destruction of function" (Blyth: "Poisons," 1883, p. 23). "Any body which, in consequence of its local chemico-dynamical action, and particularly of its absorption, causes grave or fatal accidents is considered a poison" (Galtier: "Traité du Tox.," 1855, I., i.).

expressly include the corrosives in their definitions of poisons.[1] Nevertheless, the difference is such practically that it must be recognized. A person pours a quantity of concentrated sulfuric acid into the mouth of a child or of a sleeping adult, who swallows a portion, and dies in consequence of the destruction of the stomach caused by the corrosive. This is unquestionably *administration* in the meaning of the statute (see p. 48), and for legal purposes it is not material whether the acid be considered a "poison" or a "noxious thing." Two workmen in a chemical factory quarrel, and one throws the other into a vat of sulfuric acid, in which he is immersed up to the armpits. The victim dies in consequence of the extensive destruction of skin. This is certainly not *administration* of sulfuric acid, be it poison or not, and neither in a popular nor a legal sense would the victim be said to have been poisoned. The nature of the action of sulfuric acid in the two cases is the same, the only difference being in the organ destroyed; but in both cases the application is direct and the action is different from that of the true poisons, which, so long as they remain in contact with the skin or unabsorbed in the stomach or intestines, are inert.

There is another difference between the action of corrosives and that of poisons not included in the definitions. The intensity of the action of the corrosives is directly proportionate to the degree of concentration of the agent. Dilute hydrochloric acid is not only administered medicinally, but is a normal and necessary constituent of the gastric secretion; concentrated hydrochloric acid is a powerful corrosive. With the true poisons the degree of concentration has no influence, unless it be that, within certain limits, a dilute solution may be more rapidly and completely absorbed than one which is concentrated, and may consequently act more rapidly. The intensity of the

[1] "Venenum est quid, quod exigua mole corpori nostro vel ingestum, vel externe applicatum, morbum gravem, aut mortem causat" (Plenck: "Elem. Med. et Chir. forens.," Viennæ, 1781, p. 32). "A poison is any substance or matter (solid, liquid, or gaseous) which, when applied to the body outwardly, or in any way introduced into it, can destroy life by its own inherent qualities, without acting mechanically" (Guy and Ferrier: "Forens. Med.," 6th ed., 1888, p. 319). The following also include "outward application" in their definitions: Van Hasselt: "Handb. d. Giftlehre," 1862, p. 1. Briand, Chaudé et Bouis, 1880, 10ème ed., i., 636. Mann: "Forens. Med. and Tox.," 1893, p. 381. Devergie, "Méd. lég.," 1852, 3ème ed., iii., 8. Mata: "Trat. de Med. y Cir. leg.," 1875, 5ta ed., iv., 145.

poisoning depends upon the amount of the poison which enters the blood.

It is true that certain agents, such as oxalic acid, and even the mineral acids, are both corrosives and poisons, but with these the symptoms of corrosion predominate when the substance is taken in a solid or concentrated form, while these symptoms may be entirely absent and replaced by those due to the poisonous action of the acid or of its soluble salts, when the substance is taken in dilute solution or in combination. Moreover, as most poisons are also medicines under other conditions, there is no reason why the same substance may not be considered as included in the two classes of poisons and corrosives.

But while the expert may answer the question: "What is a poison?" the question: "Is morphin a poison?" cannot be answered by "yes" or "no" without qualification. A quarter of a grain of morphin administered to a normal infant would act as a poison, and might cause death in the absence of treatment. The same quantity of the same drug given to an adult not addicted to its use would produce the indicated therapeutic action, while in an infant or adult habituated by previous medical or toxicophagic use it would be practically inert.

Although all reference to quantity is omitted in the definition, the question of quantity is one which cannot be neglected in determining whether the substance administered in a given case is or is not a "poison" or a "noxious thing." In the popular acceptation no substance is a poison which does not kill in small doses—strychnin and prussic acid are commonly recognized as poisons, while potassium chlorate and common salt are not, simply because small quantities of the former may cause death, while the lethal doses of the latter are very much greater. In many scientific works an attempt is made to include the consideration of quantity in the definition of poisons[1] by the insertion of the words "in small quantity" or an equivalent. But as the quantity varies within wide limits, not only with different poisons, but with the same poison under different conditions, the question of quantity, although of great importance

[1] Hofmann, E. R. v.: "Lehrb. d. ger. Med.," 1891, 5te Aufl., p. 612. Ogston, F.: "Lect. on Med. Jur.," 1878, p. 568. Devergie, A.: "Méd. lég.," 1852, 3ème ed., iii., 8. Mata, P.: "Trat. de Med. y Cir. leg.," 1875, 5ta ed., iv., 145. Schuchardt, B.: in Maschka's "Handb. d. ger. Med.," ii., 7, etc.

in the consideration of each individual case, cannot be regarded in the strict limitations of a definition.

The question whether the substance is capable of producing deleterious effects *in the dose administered* must be considered, whether it be a poison or a "noxious thing." The ruling of Lord Chief-Justice Cockburn in Reg. *v.* Hennah (Cornwall Lent Ass., 1877) criticised by Taylor and by Mann,[1] is undoubtedly sound in so far as he held that "unless the thing was noxious in the quantity administered, it cannot be said that there has been a noxious thing administered." In this case the defendant attempted to administer two or three grains of cantharides, not with intent to murder, or to "injure, aggrieve, or annoy" (unless the object of exciting erotic feelings come within the meaning of one of the latter terms), and hence, it appearing by the testimony (whether truly or not is not here material) that this quantity of cantharides is not sufficient to be "noxious," the Lord Chief-Justice very properly directed an acquittal. In the case of Peo. *v.* Burgess[2] it was also held that in order to sustain conviction for assault in the first degree under sec. 217, 2, of the New York penal code, it must be shown that the life of the person was endangered by administering poison. Casper and Liman, in two cases of administration with malicious intent of phosphorus and of sulfuric acid in extreme dilution, expressed the opinion that these substances in such dilution are not poisons in the medical or scientific sense, whatever they might be held to be from the legal point of view in connection with the intent to cause injury.[3]

It is quite true (as stated by Taylor and by Mann) that the ruling in Reg. *v.* Hennah throws great responsibility upon the medical witness, and further, that it renders a quantitative determination by the chemist highly desirable when it is possible, and in many ways increases the difficulty of proof. Yet the expression of Taylor that "the quantity of a poison wilfully administered ought not to affect the culpability of the person administering it" applies solely to the question of responsibility for intent, which is one entirely foreign to the function of the medical or chemical expert.

[1] Taylor: "Med. Jur.," 11th Am. ed., 1892, p. 74. Mann: "For. Med. and Tox.," 1893, p. 382.

[2] 45 Hun, 157; 5 N. Y. Cr. Rep., 514.

[3] Casper-Liman: "Handb. d. ger. Med.," 8te Aufl., ii., 361-367.

It remains to be considered whether our definition excludes those substances which are not properly poisons. A recent French definition affords an excellent text upon this point. According to it "a poison is any substance which, taken into the interior, or applied to the exterior of the body, is capable of causing death, or at least of destroying or seriously affecting health."[1]

Under this definition not only are corrosives and mechanical irritants, such as pounded glass, poisons, but pathogenic bacteria also. The case of a person who died in consequence of the lodgment of a cork in the air passages, that of a man who swallowed a silver fork, and all cases of injury from "vitriol throwing" or immersion in caustic liquids are cases of poisoning. Indeed, under a strict interpretation of the words "applied to the exterior," a person who is clubbed to death or sandbagged must be considered as poisoned.

If we consider the alimentary canal as the channel of entrance to the economy, we may divide noxious substances into:

(*a*) *Mechanical or Physical Agents—e.g.*, sharp, insoluble bodies such as pounded glass; boiling water, etc.;

(*b*) *Corrosives*, substances which destroy the organs with which they come in contact by virtue of their chemical action thereupon, such as sulfuric acid;

(*c*) *Poisons*, which, being dissolved in the blood, act either upon it or upon deeper-seated organs and tissues;

(*d*) *Bacteria*, which enter the system as individual organisms, whose processes of nutrition produce disease and death in their host. Although the bacteria themselves are not properly poisons, their products, the toxins and toxalbumins are, and are included in our definition.

The definitions of some recent Continental writers appear to us to be needlessly diffuse, and yet in some respects deficient. Thus Kobert:[2]

"Poisons are substances, some inorganic, some organic, either produced in the organism or introduced from without, some unorganized substances pre-existent in nature, which from their chemical nature,

[1] Le poison est toute substance qui, prise à l'intérieur ou appliquée à l'exterieur du corps, est capable de donner la mort, ou tout au moins de détruire ou d'altérer profondément la santé (Lutaud, A.: "Man. d. Méd. lég.," 1892, 5ème ed., p. 454).

[2] "Lehrbuch der Intoxikationen," Stuttgart, 1893, p. 9. This definition is a modification of that of Husemann ("Handb. d. Tox.," Berl., 1862, p. 2) which is in turn derived from that of C. P. von Falck.

under certain circumstances so affect any organ of the living economy, that the health or relative well-being of this economy is thereby temporarily or permanently prejudiced."

The definition of Vitali[1] is still more verbose:

"All substances are poisons which, by their nature or by their form of combination foreign to the human economy, when introduced into it or applied upon it in whatever manner, in certain doses even to the smallest, and without therapeutic indication, are capable of producing death or causing more or less severe disturbances of health, acting on the anatomical elements and on the liquids of the animal economy, and changing the normal transformations and functions of that organism."

In most legislative enactments relating to poisoning, either the necessity of strict definition of the term "poison" is avoided by use of some such general expression as "other noxious things" to include all deleterious agents which may not be considered to be poisons; or the act itself contains a definition, as in the French law, sufficiently broad to cover all possibly harmful substances.

In the Penal Code of the State of New York the definitions of murder and manslaughter[2] consider only the intent, and the circumstances attending the homicide, without reference to the material agent, except in some forms of manslaughter. Thus secs. 191, 194, refer to the use of "any medicine, drug, or substance, or any instrument or other means" for the procurement of abortion, and sec. 200 fixes the liability of a physician who shall, while intoxicated, administer "a poisonous drug or medicine" which causes the death of the person to whom it is given. In cases of homicide, therefore, in this State, the question whether the instrument of death is or is not called a poison is in no way material. It is only in the definitions of the different degrees of assault that the word "poison" is used.

Sec. 217. A person who, with an intent to kill a human being or to commit a felony upon the person or property of the one assaulted, or of another,

1. Assaults another with . . .

2. Administers to, or causes to be administered to or taken by another, poison, or any other destructive or noxious thing so as to endanger the life of such other,

Is guilty of assault in the first degree.

Sec. 218. A person who, under circumstances not amounting to the crime specified in the last section:

1. With intent to injure, unlawfully administers to, or causes to be

[1] "Manuale di Chim. tossicolog.," Milano, 1893, p. 6.

[2] Sects. 183, 184, 188, 189, 193.

administered to, or taken by another, poison, or any other destructive or noxious thing, or any drug or medicine the use of which is dangerous to life or health; or, 2. With intent thereby to enable himself or any other person to commit a crime, administers to, or causes to be administered to, or taken by another, chloroform, ether, laudanum, or any other intoxicating narcotic or anæsthetic agent; or . . . 3, 4, 5—

Is guilty of assault in the second degree.

In other sections of the code, however, the word "poison" is used in such manner that a strict definition of its meaning is necessary:

Sec. 358. A person who willfully mingles poison with any food, drink, or medicine, intended or prepared for the use of human beings, and a person who wilfully poisons any spring, well, or reservoir of water, is punishable . . .

Sec. 402, 403, 404, referring to the sale of "poisons or poisonous substances" by apothecaries or druggists.

The **English law**[1] provides: Sec. 11. Whosoever shall administer, or cause to be administered or taken by any person, any poison or other destructive thing, with intent to commit murder, shall be guilty of felony.

Sec. 23. Whosoever shall unlawfully and maliciously administer to, or cause to be administered to or taken by any other person, any poison or other destructive thing, so as thereby to endanger the life of such person, or so as thereby to inflict upon such person any grievous bodily harm, shall be guilty of felony.

Sec. 24. Whosoever shall unlawfully and maliciously administer to, or cause to be administered to or taken by any other person, any poison or other destructive or noxious thing, with intent to injure, aggrieve, or annoy such person, shall be guilty of a misdemeanor.

The **German law**,[2] after defining and fixing the penalties for the different degrees of murder and manslaughter secs. 211 to 222, without reference to the material agent used, further provides:

Sec. 229. Whosoever shall intentionally administer to another, for the purpose of injuring his health, poison or other substances capable of destroying the health, shall be punished with imprisonment for a term of ten years or less.

If a severe bodily injury has been caused by the administration, the imprisonment shall be for not less than five years, and when death has been caused, it shall be for not less than ten years or for life.

The **Austrian law**[3] (sec. 135) recognizes four varieties of murder (*Mord*): "1. Assassination (*Meuchelmord*), which is accomplished by poison or by other insidious means; 2. Murder for robbery (*Raubmord*) . . . ; 3. Murder by procurement (*bestellte Mord*) . . . 4. Ordinary murder." Administration of poison by force is ordinary (*gemeine*) murder.

[1] 24 and 25 Vict., c. 100.
[2] Strafgesetzb. f. d. Deutsche Reich.
[3] Bürgerliches Gesetzbuch, Strafgesetz über Verbrechen, etc.

The French law[1] defines poisoning in the following comprehensive terms: "Poisoning is every attempt upon the life of a person by the action of substances which can cause death more or less rapidly, in whatever manner these substances may have been used or administered and whatever may have been the consequences."[2]

In the State of New York, and probably in other States of the Union, no restriction or supervision is placed upon the **use of poisons** in trades or manufactures. In Massachusetts, in 1891, a strenuous effort was made to secure the passage of a law regulating the use of arsenic in the manufacture of wall papers,[3] and in this State repeated applications have been made to the Legislature to restrain the promiscuous use of arsenic in embalming liquids. In these cases the opposition of parties whose pecuniary interests were supposed to be affected proved successful. Nor is the **sale of poisons** by persons other than apothecaries and druggists in any way restricted. The only provisions in the Penal Code of the State of New York referring to the sale of poisons are:

Sec. 402. An apothecary or druggist, or a person employed as clerk or salesman by an apothecary or druggist, or otherwise carrying on business as a dealer in drugs or medicines, who sells or gives any poison or poisonous substance without first recording, in a book to be kept for that purpose, the name and residence of the person receiving such poison, together with the kind and quantity of such poison received and the name and residence of some person known to such dealer, as witness to the transaction, except upon the written order or prescription of some practicing physician whose name is attached to the order, is guilty of a misdemeanor.

Sec. 403. [Provides that the poison record book of druggists shall be open to inspection by any person at reasonable hours.]

Sec. 404. [Provides for the labelling of poisons, and further provides that]: a person who . . . sells, gives away, or disposes of, or offers for sale any sulphate or other preparation of opium or morphine, except paregoric and those preparations containing two grains or less of opium or morphine to the ounce, without attaching to the bottle, vial, box, or package containing such sulphate or other preparation of opium or morphine, a scarlet label lettered in white letters, plainly naming the contents thereof, with the name and residence of such person, is guilty of a misdemeanor.

Sec. 405*a*. A person who, except on the written or verbal order of a

[1] Code pénal, art. 310.

[2] See also Briand, Chaudé et Bouis: "Manuel de Méd. lég.," 10ème ed., i., 624–635.

[3] See Arsenical Wall Papers.

physician, refills more than once prescriptions containing opium, morphine, or preparations of either, in which the dose of opium exceeds one-fourth grain, or morphine one-twentieth grain, is guilty of a misdemeanor.

While the druggist may not sell arsenic, or phosphorus, or strychnin without the formalities prescribed, "Rough on rats," which contains ninety-six per cent. of white arsenic, and numerous other rat and vermin poisons containing large proportions of the poisons named are sold without question. And, while the apothecary may not refill more than once a prescription in which the dose exceeds one-twentieth grain of morphin, he may sell "soothing syrups," which contain from half a grain to a grain to the ounce *ad libitum*.

In France, Germany, and other European countries the sale or use of poisonous substances by manufacturers and others as well as by druggists is restricted and under government supervision.[1]

Although the wording of statutes treating of poisoning avoids the necessity of a strict definition in criminal cases, except in the instances above referred to, questions arise in civil actions in which the lack of such definition has proved an embarrassment. Thus in the case of U. S. Mutual Accident Assoc. *v.* Nora Newman,[2] payment of an accident insurance policy containing a provision in which the company excepted from among the risks against which it insured "voluntary or involuntary taking of poison, or contact with any poisonous substance" was contested. The deceased, James E. Newman, had died from inhalation of illuminating gas, which the company claimed was a "poisonous substance." The case was decided adversely to the company, largely upon the evidence of a professor of chemistry in a Virginian university, who defined a poison as "a substance which, when taken into the body, is capable of destroying some part or parts of the body, so as to leave them permanently incapable of performing their functions"[3]—a definition under which the case of the man who was killed at the Eddystone lighthouse by melted lead dropping

[1] See Legrand du Saule, Berryer et Pouchet: "Méd. lég. et Tox.," 1357–1361. Dubrac: "Jurisprudence méd. et pharm.," 2ème ed., 1893, 412–460, 704. Kornfeld: "Ger. Med.," 1884, 162 *ff.*; Pharm. J. and Tr., 1868–69, n. s., x., 121–128. E. S. Wood: Fifth Ann. Rep. State Bd. of Health, Lun. and Char., Mass., 1884, 256–264.

[2] Insurance Law Jour., xvii., 97. See also Paul *v.* Traveller's Ins. Co., N. Y. Rep., 112, 472.

[3] Ins. Law Jour., *l. c.*, p. 104.

into his open mouth, and in whose stomach a lump of lead weighing nearly eight ounces was found,[1] was one of poisoning; but under which, if the person recover, hydrocyanic acid is not a poison. In consequence of this decision, which we believe to have been based upon an improper definition of the word "poison," the companies have subsequently added the words "voluntary or involuntary inhalation of any gas, vapor, or anæsthetic" in their policies.

CAUSATION OF POISONING.

Poisonings may be classified either with reference to the origin of the toxic agents producing them, or according to the motive or lack of motive attending their introduction into the system.

With reference to their origin poisons may primarily be divided into two classes: Those which originate within the human body; and those which are introduced from without.

I. Endogenous Poisonings.—These are produced either by retention in the system of a poisonous substance, normally produced therein and constantly excreted therefrom in a state of health; or to the formation within the body of a pathogenic poison.

Poisoning by retention is caused by interference with any of the excretory functions. If the skin be varnished, death results from arrest of the excretory function of the perspiration. When the action of the kidneys is seriously interfered with by organic disease, or by mechanical or non-mechanical suppression, the phemomena of uræmic poisoning are soon manifested, and death is only delayed by a vicarious action of the skin or intestines for a limited period. The well-known effects of asphyxia, whether mechanical or due to the inhalation of an inert though irrespirable gas, are due in part to deprivation of oxygen, but also to accumulation of carbon dioxid in the blood. Although in obstruction of the bowels the most violent symptoms are due to local inflammatory action, the reabsorption of substances such as skatol, indol, and sulfur compounds, usually discharged with the alvine dejections, is the cause, to some extent, of the systemic disturbances.

[1] Spry: Phil. Trans., Lond., 1756, xlix., pt. 2, 477.

Auto-intoxications are also the result, not of interference with elimination, but of the production in the system either of some normal excretory product in excessive amount, such as oxalic acid, lactic acid, etc., or of substances which, probably not produced in a state of health, are endowed with more actively toxic properties, such as acetone, oxybutyric acid, etc. It is probable that many, if not all, non-communicable diseases are truly auto-intoxications.

Communicated poisonings are caused by the introduction into the system of an organism which in its development generates poisonous substances. Several pathogenic bacteria have already been shown to produce definite alkaloidal or albuminoid poisons (tetanin, typhotoxin, etc.) to whose action the phenomena of the corresponding diseases are due.

The study of endogenous poisoning is within the domain of pathology. Its present forensic interest is relative only, and is limited to the recognition of resemblances between the methods of action and chemical characters of members of the two classes of poisons. In this respect the subject is one of vital importance, particularly with regard to alkaloidal poisonings.

It is within the limits of possibility that a person might, with murderous intent, administer to another a poison such as tetanin, or a culture of the bacillus producing it. Such materials are, however, within the reach of very few persons; and there are no means by which they or their effects could be distinguished from natural results, were they criminally administered.

II. Exogenous Poisonings are caused by the introduction into the system, through one of the channels of absorption to be mentioned hereafter, of a substance possessing the qualities of a poison, whether it be organic or inorganic, natural or synthetic, animal, vegetable, or mineral in its origin.

It is with this class of poisonings that forensic toxicology has directly to deal.

With reference to the circumstances attending their origin, poisonings may be classified for convenience of comparison into:

I. Homicidal.

II. Suicidal.

III. Accidental.

Among the **homicidal** cases are included not only such as have been pronounced to be such by the finding of a trial jury, but all which have been the subject of criminal proceedings, if intent to cause death has been alleged, whatever may have been the result of the trial.

Accidental poisonings are all such as occur without intent to cause death, and among them are consequently included so-called accidents, due to criminal negligence or ignorance, which, strictly speaking, are homicidal, and have in many instances been the subject of trial for manslaughter. Yet the origin of such a case more closely resembles that of a poisoning in which the degree of negligence has been less, than one in which those has been deliberate intent to kill.

Accidental poisonings may be subdivided into:

(*a*) INDUSTRIAL POISONINGS, which are the natural consequence of contact with poisonous substances used in manufacturing processes, such as poisoning by lead among the hands in white-lead factories, by phosphorus in match factories, by mercury among mirror silverers and thermometer makers, etc. Such cases come more within the domain of public hygiene than in that of forensic toxicology, yet they may be of legal interest when the manufacturer is guilty of culpable negligence in exposing his workmen to unnecessary and avoidable danger, or in cases of alleged homicide in which the defence seeks to account for the presence of the poison in the cadaver by attributing to it this origin; or as affording an opportunity for the study of chronic poisoning, which, in homicidal cases, may be produced by the repeated administration of very small doses.

(*b*) POISONINGS FROM ENVIRONMENT arise from contact with poisonous substances or inhalation of poisoned air, not as a consequence of employment in a particular trade, but from the use of materials containing toxic agents. Thus arsenical poisoning, usually non-fatal and of the chronic type, may be caused by wearing clothing colored with arsenical dyes, or by inhabiting rooms the walls of which are covered with arsenical paint or paper; or lead poisoning by inhabiting apartments freshly painted with white lead, etc.

(*c*) FOOD POISONINGS are the result of eating articles of food which have been rendered poisonous either by the unintentional or fraudulent admixture of mineral poisons, or by the genera-

tion in them of putrid poisons. Such cases are of frequent occurrence, and in them usually a number of persons are attacked at the same time, sometimes an entire family and sometimes hundreds of persons in a community. As instances of this class of poisoning the Bradford lozenge cases, in which two hundred persons were poisoned, and the Hyères poisonings, in which over four hundred persons suffered; in both instances, in consequence of arsenic being used in mistake for plaster of Paris, which was intended to be used as an adulterant; also the constantly recurring poisonings by lead and copper, fraudulently or negligently mixed with food, and the numerous cases of cheese, sausage, ice-cream, fish, and meat poisonings.

(*d*) MEDICINAL POISONINGS are unfortunately of frequent occurence and are not seldom the subjects of suits for malpractice or of prosecutions for manslaughter. For their occurrence either the physician, the pharmacist, or the empiric is responsible. The first in many instances is excusable, as when a patient dies from the effects of chloroform administered as an anæsthetic with every possible precaution, or when a person manifests an unusual susceptibility to the action of a drug, administered in doses usually perfectly safe. But cases of severe poisoning and even of death from the effects of powerful drugs administered by physicians in heroic doses, either through ignorance, foolhardiness, or mistake, are by no means rare. For poisonings caused by errors in writing prescriptions, the pharmacist is as much to blame as the physician. For the pharmacist's error of mistaking one bottle for another in compounding a prescription there is no more excuse than for him who pleads that he did not know the gun was loaded. For the recurrence of poisonings caused by the administration or application of powerful remedies by incompetent and ignorant persons, and for the wholesale destruction of infant life by opiates in proprietary nostrums, a community which permits the one to practise or the other to be sold has itself to blame.

(*e*) OTHER CRIMINAL POISONINGS WITHOUT INTENT TO KILL arise from the administration or taking of drugs which have or are presumed to have abortifacient properties to or by a pregnant woman; or for the purpose of facilitating the commission of another crime, as by the administration of chloroform to permit the accomplishment of robbery or rape; or to prevent pilfer-

ing, as by mixing tartar emetic with whiskey; or from mere malice, as by purposely discharging chlorin gas into an inhabited apartment.

The wilful poisoning of domestic animals, a misdemeanor under secs. 660, 661 of the New York Penal Code, may be classified here.

STATISTICS OF POISONING.

Statistics of cases of poisoning can at best be but fragmentary and incomplete, as in all countries the great majority of non-fatal accidental and medicinal poisonings escape record entirely, while in such records of vital statistics as are officially kept, particularly in the United States, either the nature of the poison is not determined with sufficient accuracy, or all cases are classed under the general head of poisoning. Confessedly such records take notice only of fatal cases. The legal reports refer only to the very small fraction of cases in which conviction of criminal poisoning is followed by appeal.

	1880	1881	1882	1883	1884	1885	1886	1887	1888	1889	1880–89
Poisonous foods	16	12	15	15	27	11	26	32	25	21	200
Bromin and compounds	4	2	2	2	3	2	1	3	1	4	24
Potassium iodid	4	1	2	1	3	4	7	4	2	3	31
Cantharidin	4	4	3	..	2	1	1	2	1	1	19
Rhus toxicodendron	..	2	2	4	..	2	4	5	1	3	23
Iodoform	2	2	20	11	5	4	11	9	2	10	76
Phenol	25	28	17	24	20	13	16	15	11	10	179
Lead	21	23	26	32	27	36	20	31	27	30	273
Mercury	6	3	9	6	38	28	14	17	21	23	165
Arsenic	32	19	16	17	25	11	12	18	15	26	191
Tobacco	1	1	..	4	9	5	2	4	5	3	34
Santonin	2	..	3	..	3	..	1	2	2	3	16
Soda and salicylic acid	1	..	2	3	5	2	4	2	2	2	23
Quinin, etc	3	2	..	1	8	4	..	2	8	6	34
Caffein	..	..	..	2	1	2	3	..	1	4	13
Camphor	1	2	1	3	1	4	1	3	1	1	18
Cannabis indica	1	..	..	3	7	4	3	4	.	..	22
Hyoscin, etc	1	..	2	2	5	1	1	1	1	6	20
Stramonium	3	5	4	6	..	4	3	1	4	1	31
Aconitin	9	2	12	7	4	5	3	5	5	3	55
Atropin	8	23	12	12	16	17	14	11	10	8	131
Strychnin	17	15	9	13	19	9	6	7	7	14	116
Chloral hydrate	9	2	5	7	8	2	1	5	2	1	42
Alcohol	10	3	4	17	3	9	9	16	18	26	115
Cocain	..	..	..	..	5	11	24	23	28	23	114
Opium	12	22	21	17	14	17	13	9	11	12	148
Morphin	11	11	21	27	27	18	17	25	10	17	184

The medical, pharmaceutical, and chemical journals contain reference only to such cases as present points of novelty or of exceptional scientific interest, and consequently indicate a preponderance of certain forms of poisoning over others which is misleading. Kobert[1] gives the preceding table showing the number of cases referred to in the medical journals during the years 1880–89.

In this table the large number of food poisonings indicates the interest in that subject now taken by the medical profession, and the two hundred and seventy-three cases of lead poisoning are probably, with very few exceptions, cases of chronic poisoning having hygienic interest, but of little forensic importance.

That medical literature affords no adequate information upon this subject is evidenced by the results of an attempt which we have made to determine the number and character of accusations of murder, manslaughter, and assault by poisoning in the State of New York during the fifteen years 1879–93. Accounts in the public press, verified and amplified by inquiry of the district attorneys of the several counties, and by examination of the county records, show that sixty-four such accusations were made during that period in twenty-four counties (pop. 4,026,278); of which twenty-one were by arsenic, eleven by Paris green, eight by opiates, two each by corrosive sublimate, strychnin, and potassium cyanid, and one each by cupric sulfate, carbolic acid, chlorid of lime, oxalic acid, and laudanum. In thirteen the nature of the poison was insufficiently determined. Of these sixty-four cases there are but three to which we have found any reference in medical literature.

The statistics of fatal, accidental, and suicidal cases are somewhat more satisfactory, although it is probable that a large proportion of so-called "accidental" poisonings are classified among the deaths from natural causes, and the nature of the poison in suicidal cases is frequently determined by the "guess" of the coroner's physician. Such statistics are of value, however, to indicate the influence of locality upon the kind of poison most frequently causing death and most frequently used by suicides. The following table, compiled from the returns of the coroners of the county of New York, may be divided into three periods: 1841–43, 1866–80, 1889–92.

[1] "Intoxikationen," p. 32.

	1841, 42, 43		1866		1867		1868		1869		1870		1871		1872		1873		1874		1875		1876		1877		1878		1879		1880		1881		1882		1883		1884		1885		1886		Total 1867-1886		Total 1887-1892	
	A	S	A	S	A	S	A	S	A	S	A	S	A	S	A	S	A	S	A	S	A	S	A	S	A	S	A	S	A	S	A	S	A	S	A	S	A	S	A	S	A	S	A	S	A	S
Corrosive	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	2	..	..	..	1	..	2	..	..	..	1	..	..	1	..	..	..	1	..	..	..	..	..	..	..	..	..	2	4	..	1
Acid	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	1	..	..
Sulfuric acid	1	1	..	..	..	..	..	..	..	..	3	1	1	..	..	..	..	1	..	2	..	..	..	1	..	..	..	2	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	16	..	..
Hydrochloric acid	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	1	1	..	..	..	..	..	..	2	..	..	2
Ammonia	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	1	..	..	..	..	..	..	1	..	1	1
Potash	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	1	..	..	..	..	..	..	..	1	..	1	..	..	1	..	..	..	..	..	1	..	..	..	..	4	1	1	
Oxalic acid	..	..	..	..	1	..	..	..	1	..	..	1	..	1	..	..	..	1	1	3	..	..	..	1	..	1	..	2	1	2	..	..	..	1	1	..	..	2	..	..	..	3	11	1	3
Irritant	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	1	..	..	..	3	..	1	..	..	..	..	1	1	..	..	..	..	..	1	4	2	2
Arsenic	5	8	..	..	4	..	2	..	1	..	..	..	..	1	1	..	2	..	3	..	3	..	1	..	4	..	2	1	2	..	3	..	11	..	15	8	15	8	5	..	..	3	31	2	21	
Paris green	..	..	..	..	5	..	8	..	13	..	1	10	..	12	..	21	..	22	1	21	..	25	1	22	1	15	1	18	..	15	..	17	..	21	..	18	1	29	3	2	..	39	4	42	
Rat poison	..	..	..	..	1	..	..	..	..	..	..	..	..	..	1	..	1	..	..	2	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	2	4	..	
Phosphorus	1	..	..	..	..	..	1	..	..	..	..	..	..	1	..	..	..	..	2	1	..	3	..	1	..	1	..	1	..	..	..	..	3	..	6	1	..	..	..	..	..	..	5	1	4	
Corrosive sublimate	2	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	1	..	..	1	..	..	..	..	1	..	..	..	1	1	..	..	..	..	1	3	1	1	
Lead	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	3	..	4	3	..	6	..	3	..	..	..	..	17	1	19	
Copper	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	2	2	..	
Morphin	2	1	..	..	2	..	1	..	2	2	2	..	3	3	3	2	..	3	3	1	6	2	1	1	2	1	1	3	4	5	1	..	..	..	1	4	5	6	3	..	21	32	10	8
Opium	3	5	..	..	..	..	2	..	1	2	1	..	2	..	7	11	..	3	8	2	6	..	5	1	7	..	5	..	1	7	4	17	7	..	7	7	3	7	3	..	11	33	31	22
Laudanum	13	26	..	..	12	..	6	..	5	..	6	..	8	1	7	11	6	1	11	..	5	1	4	4	5	3	5	1	2	3	2	..	..	..	1	..	..	1	..	..	14	77	1	1
Opiates	..	..	..	..	6	..	10	..	8	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	24			
Narcotic	..	..	..	..	..	..	..	..	..	..	..	..	1	..	5	..	..	..	..	1	..	..	..	..	..	2	1	1	..	..	..	..	..	..	1	..	..	1	..	..	..	7	4	1	1
Paregoric	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	1			
Poisons not opiates	..	..	..	..	11	..	10	..	10	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	31			
Tr aconite	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	1	..	..	2	..	..	..	..	..	..	..	..	..	..	..	..	2	..	1	..	..	..	3	1	..	3
Strychnin	1	..	..	..	1	..	..	..	..	..	3	1	1	..	..	..	..	1	..	2	..	..	..	1	..	..	..	2	..	..	1	..	1	..	..	..	2	..	..	..	..	1	12	2	1
Tobacco, nicotin	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	1	3	..
Belladonna	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	1	..	..	3	..	..	..	..	..	..	1	..	..	..	..	..	..	1	3	..	1
Colchicum	1	..	..	..	..	..	..	..	..	..	..	..	1	..	1	..	2	..	1	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..			
Kerosene	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	1	..	1	..	2	..	..	..	..	..	..	..	..	..	..	..	..	..	..			
Chloroform	..	..	..	..	..	..	..	..	1	1	..	1	..	1	..	1	2	2	1	..	1	1	..	1	..	2	2	3	1	1	1	1	1	..	2	1	3	7	..	..	15	9	9	6
Alcohol, etc	2	..	..	..	..	..	..	..	..	..	1	..	1	..	..	..	..	..	..	..	..	..	..	..	1	..	1	..	2	..	2	..	..	..	1	..	..	..	..	..	..	..	1	..	1	
Ether	..	..	..	..	..	..	..	..	..	..	..	..	..	1	1	..	..	..	..	..	..	1	..	1	..	..	..	..	..	..	..	..	1	..	..	2	1	1	1	..	..	3	1	1	2
Chloral	..	..	..	..	..	..	..	..	..	..	..	1	..	1	..	1	..	..	2	..	..	2	1	1	1	1	..	2	..	2	1	..	..	..	1	..	..	..	..	..	..	10	6	..	1
Prussic acid	..	1	..	..	..	..	1	..	..	..	3	..	1	..	1	..	1	..	2	..	4	1	4	1	1	..	2	..	..	2	..	1	3	..	3	..	..	..	..	..	..	21	..	6	
Potassium cyanid	..	..	..	..	2	..	..	..	3	..	..	..	..	..	..	..	1	1	3	..	2	..	1	..	2	..	5	..	2	..	..	..	4	..	2	..	3	..	3	..	1	22	..	12	
Illuminating gas	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	1	..	1	..	..	..	..	..	..	..	1	1	1	2	33	11	30	4	25	19	53	11	13	3	157	15	
Charcoal gas	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	2	1		
Carbon dioxid	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	1	..	1	
Turpentine	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	2			
Carbolic acid	..	..	..	..	..	..	..	..	..	..	1	..	1	..	1	1	..	1	1	..	1	1	..	1	1	1	..	..	..	1	2	..	..	12	..	5	10	9	13	6	7	6	23	32
Creosote	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	2			
Not specified	..	..	7	18	..	4	..	5	..	4	..	..	..	2	..	..	..	1	..	1	1	..	..	..	..	..	..	2	..	..	..	41	..	..	..	..	..	2	2	11	24	43	2		

One poisoning by each of the following: Tartar emetic (A. 1841–43); tr. gelsemium (1867, S.); chamomile, lime water (1871, A.); Dover's powder (1872, A.); yellow wash (1872, S.); soothing syrup, sulfur dioxid (1873, A.); fusel oil (1874, A.); oil of tansy (1874, S.); cholera mixture, magnetic zinc, hyoscyamus (1875, A.); oil of bitter almonds (1876, S.); castor-oil bean, "Laville" (1877, A.); McMunn's elixir (1878, S.); nitrous fumes, rhus (1879, A.); stramonium, wormwood, hydrogen, poisoned wound (1880, A.); nitric acid (1880, S.); pyrogallic acid (1891, A.); sewer gas (1892, A.); pennyroyal (1892, S.). In 1842 four were accidentally poisoned by tr. sanguinaria, and in 1877 four by nitrate of mercury. Among the poisonings by arsenic in 1890-92 are included 20 by "rough on rats." The only references made to homicidal poisoning are one by laudanum in 1841–43, and one by morphin, in 1879. The data for 1841–43 are from Beck, "Infant Therapeutics," pp. 136-143; the remainder from the reports of the New York City Board of Health.

The most noticeable features of these statistics are: 1st. The large number of poisonings by illuminating gas in the last period. The large predominance of accidental over suicidal deaths by this agent is due to the fact that only those cases in which the suicidal intent was obvious are classified as suicides. At the present time illuminating gas probably causes more deaths in this city than any other poison. 2d. The large number of poisonings by "Paris green." During the period 1866–80 morphin and the preparations of opium caused 251 deaths and Paris green 238; while in 1889–92 the opiates were fatal in 75 cases and Paris green in 86.[1] The predominance of Paris-green poisoning is peculiar to the United States, where it is traceable to the very general use of that dangerous agent for the destruction of vermin. In England, poisonings by the opiates, by the cyanic poisons, and by the mineral acids outnumber those by arsenic. In France at the present time phosphorus is the agent most frequently employed in criminal poisoning,[2] and the number of illicit administrations of the salts of copper and of cantharides is much greater than in other countries, while crimes in which the cyanic poisons and the opiates are used are of very infrequent occurrence. In Germany and Austria, on the other hand, poisonings by the cyanics are only exceeded in number by those by illuminating gas and the mineral acids. In the latter country the mineral alkalies are more frequently the cause of death than elsewhere. In Finland carbon monoxid, strychnin, and the mineral acids are the poisons most frequently used. In India poisonings by arsenic still maintain the predominance which they formerly had in Europe, but are very nearly equalled, and in some districts surpassed, by the use of the opiates. In India also poisonings by mercurials, aconite, and dhatura are more often met with than in Europe or America. The table on page 60, compiled from various sources, indicates the relative number of poisonings by the commoner poisons in various countries:

The *motive* for homicide by poison is only exceptionally revenge or pecuniary advantage. Murder by poison is most frequently by or for a woman.[3] In Prussia in the years 1863–77,

[1] See Arsenical Greens.

[2] Tardieu: "Empois.," 1875, p. 167.

[3] In this connection see Friedreich's Bl. f. ger. Med., 1850, I., i., 65; 1853, III., i., 40; 1854, IV., i., 78; II. 32.

	I.	II.	III.	IV.	V.	VI.	VII.	VIII.	IX.	X.	XI.	XII.	XIII	XIV.	XV.	XVI	XVII.	XVIII	XIX	XX
	New York City, 1841-43.	New York City, 1865-80	New York City, 1889-92.	Massachusetts, 1878-1886.	England, 1835-35.	England, 1840.	England and Wales, 1863-67.	England, Suicides, 1871-80.	England and Wales, 1881.	England, 1848-72.	France Criminal, 1825-189	France, Criminal, 1851-62.	Prussia, Suicides, 1869.	Vienna, Suicides, 1871-75.	Berlin, 1876-78.	Finland, 1870-86.	Finland, 1879-93.	Madras, 1885-89	Bombay, 1885-89	Bengal, 1885-89
Carbon monoxid...	..	18	203	13	2	..	....	....	..	..	....	.		.	155	.	27	....	..	2
Mineral acids.....	2	16	3	1	34	..	71	170	20	..	86	47	18	17	93	2	18	.		1
Arsenic	13	37	57	35	185	32	83	78	10	..	823	287	3	2	12	7	9	179	211	175
Paris green.....	..	238	86	26	..	..	....	....	..	..	1	..	1							
Phosphorus.....	1	11	5	4	..	..	15	35	15	2	336	267	11	13	40	5	11			
Mercury	3	5	2	5	15	..	58	51	6	2	11	8	..	..	..	2	6	35	17	6
Copper	..	2	..	..	..	..	3	8	..	..	267	159	..	..	2	.	..	....	.	2
Morphin.....	3	53	18	)	(2	..	32	....	..	2	....	..	5	8	9	1	11	....	..	29
Laudanum	39	91	2	60	133	..	....	....	..	7	....	3								
Opium, etc.....	8	107	53	)	(63	75	600	424	146	11	21	6	..	..	3	1	5	25	81	266
Strychnin	1	13	3	6	2	..	41	183	21	4	23	7	..	2	4	4	21	....	..	7
Aconite.....	..	4	3	3	2	..	6	10	4	1	....	..	..	..	..	..	1	18	1	6
Dhatura	..	..	..	..	..	..	....	....	..	..	5	..	..	..	1	..	..	14	22	2
Cantharides	..	..	.	1	1	..	2	1	1	..	59	30								
Oxalic acid.....	..	16	4	3	19	..	66	159	16	2	....	..	..	..	19					
Chloroform.....	..	24	15	5	..	..	....	) 25	(2	..	....	..	1	..	6					
Chloral..... ...	..	16	1	9	..	..	....	) 0	(20	..	....	..	..	..	7	..	1	....	..	1
Cyanic poisons	1	47	18	7	31	..	182	357	46	2	8	4	6	40	40	1	8	....	..	4
Carbolic acid.....	..	14	55	7	..	..	5	188	50	3	....	..	..	..	2		7	....	..	1
Total.....	83	872	614	214	543	349	1,620	1,000	569	48	2,123	792	103	120	432	30	136	631	857	1,141

I., Beck, *l. c.* II., III., Repts. N. Y. City Board of Health. IV., Tr. Mass. Med.-Leg. Soc. (no report for 1885). V., Lond. Med. Gaz., 1839, xxv., 294. VI., 6th Ann. Rept. Reg. Gen. Gr. Brit. (1844). VII., *Ibid.*, 1869. VIII., Blyth, "Poisons," p. 31; cases in 1,000; (1) and chlorodyne. IX., Ph. J. and Tr., 1883 (3), xiii., 1,084. X., *Ibid.*, 1872 (3), ii., 910; cases of death or injury due to error or negligence. XI., Lacassagne, Arch. d. l'Anthrop. crim., Par., 1886, i., 260-264. XII., Tardieu, "Empoisonnements," 2ème ed., 164-165. XIII., Falck, "Toxikologie," p. 18. XIV., Hofmann, "Ger. Med.," 5te Aufl., 662; also 18 by alkalies. XV., Lesser, "Atl. d. ger. Med." p. 1. XVI., Ann. d'Hyg., 1873, 2 s., xli., 240. XVII., Fagerlund, Vierteljsch. f. ger. Med., 1894, 3 f., viii., Supphft. 48-93. XVIII., XX., Gribbel and Hehir, "Med. Jur. for India," 400-401.

67.7 per cent of those accused of poisoning were women.[1] In France during the years 1825–80, 70 per cent. of the criminal poisonings were by women; 43 per cent. were caused by domestic dissensions, 24 per cent. were by mothers upon young children, 10 per cent. due to adultery, 9 per cent. for vengeance, 9 per cent. due to cupidity, and 5 per cent. to unrequited love. Only 30 per cent. of the poisonings occurred in cities.

In the United States the apparent rarity of crimes by poison in large cities is a subject for serious reflection. In the city of New York during the past fifty years there have been but seven trials for murder by poison, four of which resulted in the conviction of murder in the first degree,[2] one in the second degree,[3] and two in acquittal.[4] During the same period there was one conviction for manslaughter[5] and two for assault in the first degree,[6] and two cases of murder and suicide.[7] In the three cases of murder by poison which (after an interval of twenty years, during which no similar cause was tried) have been the subject of trial during the past four years, the body of the deceased was buried upon the certificate of a physician in two instances, and after an entirely insufficient investigation by the coroner in the third; and it was only after periods of 43, 53, and 98 days that, at the instance or suggestion of unofficial persons, the bodies were exhumed and submitted to proper examination. There is little room to doubt that the more frequent occurrence of trials for poisoning in rural communities is due rather to the greater detective efficacy of public rumor among these, than to exceptional rarity of crimes of this nature in large cities of such heterogeneous population as New York.

The inadequacy of our present police and legal machinery to the prevention and detection of secret poisoning is further shown by the occasional detection of a murder by poison constituting the last of a series of similar crimes, escape from the consequences of the earlier of which has finally led to carelessness on the part of the perpetrator and to the awaking of suspicion by the sheer number of rapidly succeeding deaths, all leading to

[1] Falck: "Lehrb. d. Toxik.," p. 18.

[2] Williams, 1854; Stephens, 1859; Harris, 1892; Buchanan, 1893.

[3] Meyer, 1894.

[4] Heggi, 1872–73, Lebkuchner, 1888.

[5] Nichols, 1893.

[6] Volkmer (Joseph and Mary), 1879; Burgess, 1886.

[7] Deffarge, 1879; Fritz, 1885.

the pecuniary advantage of the same survivor. The defendant Meyers was under indictment for a similar offence in Ohio, where he would have been tried for murder had he escaped conviction in New York, and if the theory of the State be correct, the crime for which he was convicted was but one of many schemes for the defrauding of life-insurance companies, some of which at least were attended with murder. In November, 1888, Sarah Jane Whiteling was convicted of murder in the first degree for having killed one of her children by poison, having previously destroyed another child and her husband by similar means in order to collect a paltry life insurance upon each. The three deaths occurred on March 20th, April 24th, and May 26th of the same year. In 1888 Sarah J. Robinson was convicted of murder in the first degree after two trials, for one of a series of probably eleven poisonings by arsenic at Somerville, a suburb of Boston. The victims were relatives; the motive the collection of life-insurance money. Instances of repeated poisoning are, however, by no means peculiar to this country. In Germany the cases of Anna Margaretha Zwanziger (3 murders and several attempts), the widow Ursinus (4 murders), Gesche Margaretha Gottfried (15 murders), and Gesche Margaretha Brockman (3 murders), are historical. In Holland the poisonings attributed to Maria Catherine Swanenburg extended between 1877 and 1883. Besides the 4 persons for whose murder she was convicted in 1885, she had destroyed 19 others, while 36 persons were made ill by poison administered by her, of whom 5 twice, 2 three times, 1 four times, 1 five times, and 1 six times; and had attempted to kill 14 others, 1 twice and 1 three times. In Belgium in 1894 the wife of a prominent government official was charged with successive poisoning of a sister, an uncle, and a brother. In France, Pel (1884) was suspected of having destroyed five persons by poison in a period of twelve years, and Pastré-Beaussier (1886–88) was accused of having caused the death of three persons and the serious poisoning of twelve others. In England, two women (Reg. *v.* Flannagan and Higgins, Liverpool Winter Assizes, 1884) were convicted of murder, having caused the death of four persons by arsenic extracted from fly-paper. Taylor[1] refers to the case of Mary Ann Cotton (Durham Lent Assizes, 1873), convicted of the murder

[1] "Poisons," 3d Am. ed., 180.

of a stepson, who had previously at various times destroyed her mother, fifteen children, three of her husbands, and a lodger by poison. In all of the cases cited the object was the same, the obtaining of insurances upon the lives of the victims. The case of the notorious "Dr." Cream, or Neill, executed for the murder of Matilda Clover in England in 1892, shows that the wholesale poisoner may extend the field of his operations over two continents.

ABSORPTION OF POISONS.

That poisons administered by the mouth, or by inunction, pass into the blood and tissues was known early in the present century. Voigtel[1] in 1804 quoted several instances in which mercury was obtained by distillation from the bones of those who had taken corrosive sublimate for some time. Buchner[2] relates that at a still earlier period Pickel had found mercury in the brain of a similar patient, and that Eckel had obtained mercury from the blood, urine, and saliva of a syphilitic patient under treatment by inunction, and in the perspiration of another treated by internal administration. Orfila was the first to demonstrate the presence of arsenic in the blood, organs and urine after its administration by the stomach, first in a series of experiments upon animals in 1839,[3] and later in the same year in the human subject in the case of Soufflard.[4] Since that time analyses have shown the presence of a great number of poisons in the blood, tissues, and urine either in their own form or in their products of decomposition, after their administration by the stomach, by inhalation, and otherwise, and if any still remain whose absorption has not been thus proved, it is either because the necessary investigations have not been undertaken, or because of the inadequacy of our methods of research. In some instances in which the usual chemical reactions have failed to show the presence of a given poison in the blood its existence there has been otherwise demonstrated. Thus while liquid reactions may fail to show the presence of lithium and thallium in the blood and tissues after the administration of small doses,

[1] "Handb. d. path. Anat.," 1804–5, i., 153–258; ii., 10, 110.

[2] "Toxikologie," 2te Aufl., 1827, 544, 538.

[3] Mém. Ac. roy. de méd., 1840, viii., 376.

[4] "Toxicologie," 5ème ed., i., 403, and Bull. Ac. de méd., 1838–39, iii., 664.

their presence there has been distinctly shown by spectroscopic examination of the ash. In the case of alkaloidal poisons reactions approaching in delicacy the spectroscopic tests are wanting, and for many the known reactions require very appreciable quantities of the substance. If the presence of snake poison in the general circulation has not been demonstrated by chemical means, its presence there may be inferred from the fact that when injected in the distal end of a limb whose communication with the general circulation is temporarily cut off by a ligature its action is slight and local only, but on removal of the ligature its rapidly fatal action is exerted. The "incarcerated" hypodermic use of cocain to produce local anæsthesia in a portion of a limb separated from the general circulation by a bandage is now generally practised.

At the present time the view of Bernard, that poisons only act after absorption into the blood[1] is so universally accepted that we have considered it as the distinguishing element in the method of action of the true poisons, in contradistinction to the local chemical action of the corrosives, and to the physical action of mechanical and thermal irritants. (See Definition, p. 43.)

When a poison is directly introduced into the circulation, as by hypodermic or intravenous injection, or application of its solution to a wounded surface, it is clear that absorption is dispensed with, and that consequently the action of poisons or medicines so administered is more rapid than when they are taken by the mouth.[2] Poisons so introduced begin to act immediately, as the time required for their transmission to the organ, tissue, or morphological element upon which they exert their action is practically insignificant; and with rapidly acting poisons a fatal termination may follow in a very short time. Thus ani-

[1] Pour qu'un poison agisse, il faut qu'il soit arrivé dans le système artériel, car ce n'est que lorsqu'il sera parvenu dans le réseau capillaire, au moyen des artères, que ses effets se manifesteront. Qu'on place, en effet, sur le cerveau l'un de ces poisons dont l'action sur le système nerveux est si puissant et cause si promptement la mort, de la strychnine ou de l'acide cyanhydrique, par exemple; aucune action immediate ne se produira, et à la longue seulement un effet local pourrait se montrer dans certains cas. Il faut, ainsi que nous venons de le dire, pour que les effets toxiques de ces substances se manifestent, que l'absorption les ait amenées dans le courant artériel, qui les conduit aux capillaires ("Substances toxiques et médicamenteuses," 1857, p. 47).

[2] Arsenic is apparently an exception to this rule (see Arsenic: Absorption).

mals have been killed in half a minute by injection of cobra poison (Fayrer) or of hydrocyanic acid (Preyer).

Almost all poisons also act more energetically when thus introduced, by reason, partly, of the immediate entrance of the entire dose, which, being gradually absorbed from the alimentary canal, may be partly removed by elimination before absorption is complete, and partly because by hypodermic injection the entire quantity administered certainly enters the circulation, while a part of that given by the mouth frequently escapes absorption entirely.

Substances, however, which are themselves non-poisonous, but which are acted upon by the gastric secretion, with formation of a poisonous derivative, are inert when introduced directly into the circulation, but actively poisonous when taken into the stomach. Thus the neutral crotonglycerid, which exists in the fresh seeds of *croton tiglium*, is inert when injected hypodermically, but is decomposed when taken into the stomach with liberation of crotonic acid, which acts energetically. Potassium myronate, a constituent of mustard, and amygdalin, a glucosid from the bitter almond, may be given to herbivorous animals under the skin without producing any ill effects, but, if given by the mouth, they are decomposed in the intestine by the action of bacteria therein contained, the former with the liberation of oil of mustard, the latter with the formation of hydrocyanic acid, whose actions are then manifested.[1]

On the other hand, there are some poisons which, although extremely active when introduced hypodermically, are either inert or much less violent in their action when taken by the mouth. Such are the venoms of serpents, curare, ergotic acid, which are either excreted more rapidly than they are absorbed, or are converted into less harmful derivatives in the alimentary canal or liver.

The **rapidity** with which absorption takes place depends upon: (1) The diffusibility of the substance; (2) its condition, whether in solution or solid, and, in the latter event, whether readily or difficultly soluble; (3) other conditions existing which favor or impede solution and diffusion; (4) the absorbent surface.

The influence of variations in diffusibility upon the rapidity

[1] Kobert: "Intoxikationen," 1893, 22, 30, 359.

of absorption is, in the case of solid poisons, dependent in large measure upon variations in conditions influencing solution, as in their case solution is preliminary and prerequisite to diffusion or absorption. The rapidity of absorption by the pulmonary mucous membrane is due in part at least to the rapid diffusibility of gases and vapors. Vapor of chloroform when inhaled, even if largely diluted with air, is rapidly absorbed, and has been known to produce unconsciousness and muscular resolution in animals in twenty-five seconds, while in the human subject, administered for the purpose of causing anæsthesia, it frequently accomplishes that object in less than half a minute, and in some cases has destroyed life in less than a minute.[1] But, when taken into the stomach, liquid chloroform, by reason of its sparing solubility and its slight diffusibility under such conditions, causes insensibility only after the lapse of half an hour or more. Thus Smith[2] relates the case of a woman who swallowed about an ounce of chloroform, after which she walked about the streets for an hour, when she was found leaning against a building, and walked to the police station, where she became unconscious. She had walked between one and two miles before becoming unconscious.

In general terms it may be stated that anything which favors solution of a poison accelerates its absorption, while any condition which impedes its solution retards absorption. Insoluble substances are non-poisonous. Even if they be substances which in a soluble form are actively poisonous, when in an insoluble modification or combination they are inert. Thus, while the yellow variety of phosphorus is one of the most virulent of poisons, the red allotropic modification, which is the same substance chemically, and differs only in its physical properties, is practically harmless, and that principally because it is not dissolved by the oils and fats which are solvents of the yellow modification. Indeed, the object of administering chemical antidotes in cases of poisoning is usually to bring about the formation of an insoluble, and therefore non-poisonous, combination. But, while the toxic activity of a given substance is proportionate to the quantity which can be brought into solution, it by no means follows that readily soluble substances are more

[1] Lyman: "Anæsthesia," New York, 1881, p. 195.

[2] Phila. Med. News, 1891, lix., 688.

poisonous than related substances of more difficult solubility. Indeed, the reverse is known to be the case in many instances, as in the series of alcohols of which ordinary alcohol is a member the higher forms are less soluble and more actively poisonous than the lower.[1] (See Absorption by the Stomach, below.)

Absorption by the Lungs.—The absorption of gases and vapors which are soluble in or act chemically upon the blood takes place with great rapidity. Indeed, volatile liquid poisons are sometimes more rapidly fatal when their vapor is inhaled than when the liquid is injected directly into a blood-vessel. Thus Preyer,[2] experimenting with hydrocyanic acid, found that a rabbit died in twenty-six seconds after inhaling the vapor of the concentrated acid for three seconds, while in another rabbit into whose jugular vein 1 c.c. of a sixty-per-cent. acid was injected, although the interference with respiration was immediate, the spasms only appeared in twenty-nine seconds, and death followed in sixty-one seconds after the injection. In guinea-pigs death followed inspiration of air charged with vapor of hydrocyanic acid in five, six, and sixteen seconds. It is clear that here absorption was practically instantaneous.

The action of poisonous gases when inhaled in tolerably concentrated form, besides being favored by rapid absorption, is also aided by interference with the principal channel of their excretion. With gases which may be inhaled undiluted with air, elimination by this channel is entirely arrested, and the effects are very rapidly produced. Thus muscular resolution has been produced by nitrous oxid in twenty-five seconds. In the case of indifferent gases the interference with elimination of carbon dioxid is the only cause operative, and several minutes elapse before the accumulation of that gas in the blood causes asphyxia.

Absorption by the Mouth.—Absorption by the buccal mucous membrane is usually of slight practical importance, as the poison is rarely retained in that cavity, or given in a form which permits of any extensive absorption during its brief sojourn there.

Poisons of great activity, however, enter the circulation

[1] In this connection see also Richet, C. rend. Soc. biol., Paris, 1893, 9 s., v., 775.

[2] "Die Blausäure," ii., 1870, 60, 62, 71.

from the mouth or lips or the nasal mucous membrane with such rapidity that their contact with those surfaces may cause death before an act of swallowing is attempted, or even possible. Thus Preyer[1] found that rabbits fell unconscious in twenty-three seconds after the application of hydrocyanic acid to the tongue, and in twelve seconds after its application to the nostrils. Nicotin also, when applied to the mouth in doses of two to four drops, may cause poisoning either immediately or within half a minute.[2] In cases of tobacco poisoning from excessive smoking, in which the smoke is not inhaled, the absorption is by the buccal mucous membrane except in so far as it may occur by swallowed saliva. Arsenic is also absorbed from the mouth under exceptional circumstances, as in those instances in which arsenical symptoms were traced to smoking tobacco or cigars impregnated with arsenic.[3]

Absorption by the Stomach.—It is usually from the stomach in cases of homicide that the poison first enters the circulation, the absorption being afterward supplemented by the more perfect action of the intestines. When the stomach is empty its absorbent power is more active than when it contains food, and particularly so in the morning before breakfast. As water and alcohol are absorbed to a great extent by the stomach, substances dissolved in those liquids, particularly if the solution be dilute, are rapidly absorbed. It has been shown by numerous experiments, and observations that some poisons, as hydrocyanic acid or strychnin in solution, or alcohol may, under favorable conditions, be absorbed from the stomach in ten seconds or less after their first introduction. It is probable also that all liquid poisons and crystalline substances in solution are absorbed from the stomach with equal rapidity, and that when a considerable interval of time elapses between the taking of the liquid into the stomach and the manifestation of symptoms, as is the case usually with arsenic, the delay is not due to slowness of absorption but to the time required for the tissue changes which are the proximate causes of the symptoms which they produce.

Substances, also, which are insoluble in water or inert are

[1] *Loc. cit.*, p. 69.

[2] Husemann u. Hilger: "Pflanzenst.," 2te Aufl., ii., 1,173.

[3] Schlegel: J. d. pract. Hlk., 1827, lxiv., 13. Friedreich: Bl. f. ger. Anth., 1858, ix., 3 Hft., 40. The latter a case of alleged homicide.

sometimes converted into soluble and poisonous derivatives by the action of the gastric secretion, or of constituents of the food. Thus, while calomel is insoluble in water, it is dissolved to an appreciable extent in the gastric juice by the combined action of the peptic ferment and acid, and to a still greater extent if the food contains a large quantity of salt. On the contrary, certain constituents of the food may by their chemical or physical action delay the process of gastric absorption, as salt interferes with the absorption of silver nitrate by its conversion into the insoluble chlorid, and oils delay the solution of arsenic. It must also be noted that many poisons provoke vomiting, and that a large proportion of the dose swallowed may be thus expelled before sufficient time for its solution and absorption has elapsed.

Absorption by the Intestine.—As the small intestine is the essential organ of absorption of the products of digestion, so is it also a most active absorbent for poisons and medicinal agents which, having reached it, are more rapidly absorbed than from the stomach. Poisons, such as phosphorus, which are soluble in oils but not in watery liquids, escape absorption in the stomach entirely, and only enter the circulation indirectly from the intestine, by the channel of the thoracic duct.

Absorption from the **rectum** is with most poisons in solution quite as rapid as from the stomach and apparently more rapid with some. Thus Galtier [1] cites the experiments of Roselli and Strombio, who found that strychnin introduced into the rectum in alcoholic solution caused tetanic spasms in dogs more rapidly than the same quantity given in the same form by the stomach. Rectal medication by means of suppositories is now frequently practised, as it was by enemata formerly. Morphin and belladonna are more rapid in their action when given by the rectum than when swallowed, and the numerous accidents that have followed the use of infusions of tobacco as enemata attest the facility of the absorption of nicotin by the rectum. Instances of homicidal administration of poisons by the rectum are of infrequent occurrence. (See Sulfuric Acid, Arsenic, Mercury.) Suicides have in a few instances selected this method of administration.[2]

Poisons absorbed from the stomach and intestines are car-

[1] "Toxicol. gén.," 1855, p. 8.

[2] Hofmann: "Lehrb. d. ger. Med.," 5te Aufl., 615.

ried by the gastric and mesenteric veins to the portal vein, by which they enter the liver, where they are to some extent retained[1] or whence they pass by the hepatic veins into the general circulation.

The duration of absorption from the alimentary canal will depend largely upon the form in which the poison is taken and upon the extent to which that not immediately absorbed is either rendered insoluble by the action of antidotes or removed by purgation, vomiting, or the stomach pump. When opium, which causes neither vomiting nor purging, is taken in the solid form in large amount, and no successful effort to remove it from the stomach is made, its gradual solution and absorption must continue until death puts an end to the process. In the case of a woman[2] who died sixty hours after taking an unknown quantity of white arsenic, that poison was found in substance in the stomach. It goes without saying that, if life continue, the same conditions which diminish the rapidity of gastro-intestinal absorption increase its duration.

Absorption from the Skin.—The skin with its cuticle or epidermis intact is practically impervious to watery liquids which do not act upon it chemically. There are, however, some poisons which act directly upon it, producing dermatitis, which in a mild form may be limited to a simple redness or erythema, attended with more or less itching and going on to the formation of vesicles, pustules, or bullæ, and, in extreme cases, even gangrene. Among these are arsenic and the juices of certain plants. The epidermis is, on the other hand, permeable to oils and fats, and mercury and some other medicines, incorporated with fats in the shape of ointments, are readily absorbed when rubbed upon the cuticle. The menstruum best adapted to this purpose is lanolin (a fat, in which glycerol is replaced by cholesterol), obtained from wool. Finely divided powders, when rubbed upon the cuticle, are also absorbed, probably by being dissolved in the oily sebaceous secretion. Metals, also, whether in the shape of solids or vapors, are absorbed through the cuticle, as is evidenced by the occurrence of lead poisoning among plumbers and others who handle that metal, and by the physiological action of mercury produced by exposure of the skin to the vapor of that metal.

[1] See p. 82.

[2] Paterson: Edinb. Med. Journ., 1857, iii., 394.

The abraded skin, exposing the very vascular subcutaneous cellular tissue, becomes a very active absorbent surface, and solutions applied to it are to all intents and purposes under the same conditions as when hypodermically injected.

Absorption from Genito-Urinary Organs.—Several cases are reported of women who have been poisoned by arsenic introduced into the vagina with murderous intent.[1] The occurrence of mercurial poisoning in women, due to the use of corrosive sublimate solutions for vaginal and uterine irrigations, has been the subject of several treatises.

The male urethra and mucous membrane of the glans and prepuce are actively absorbent surfaces. So far as we are aware no modern instance is recorded of criminal introduction of poison by these channels, although Zacchias relates[2] that Ladislas, King of Naples, was killed by poison communicated during coitus.

The mucous membrane of the urinary bladder absorbs only slowly and imperfectly. Stillé[3] states that atropin may be absorbed from the bladder even after it has been eliminated from the system by the kidneys. On the other hand, Falck[4] found that 0.03 gm. (gr. $\frac{1}{2}$) of strychnin nitrate in solution introduced into the bladder of a dog did not produce the slightest symptoms of poisoning, while very much less quantities (from 0.75 to 3.9 mgm. per kilo) caused death by hypodermic injection, by the rectum, and the empty stomach.

The experiments of Cazeneuve and Lépine[5] show that strychnin is not absorbed from the uninjured bladder. They injected 0.04 gm. (gr. $\frac{6}{10}$) into the bladders of dogs by means of a Dieulafoy syringe; the neck of the organ having been previously ligated. No symptoms whatever were manifested for sixteen to twenty hours, after which they developed rapidly, and terminated suddenly in death. As at the autopsy they always found the bladder inflamed in the neighborhood of the ligature they properly attributed the later effects to absorption from this inflamed surface.

Absorption through other Channels.—Substances in solution injected into parenchymatous organs pass into the cir-

[1] See Arsenic.

[2] "Quæst. med. leg.," Venet., 1737, i., 170.

[3] Wharton and Stillé: "Med. Jur.," 4th ed., ii., 11.

[4] "Toxikologie," 1880, p. 8.

[5] Ct. rend. Ac. Sc., Paris, 1881, xciii., 445.

culation as rapidly as when injected into the subcutaneous tissue. The serous membranes are actively absorbent. Cases of medicinal poisoning have resulted from the introduction of atropin solution into the auditory canal, and mineral acids have been poured into the ear with murderous intent. The corneal and palpebral conjunctiva is an absorbent surface, as is proved by absorption of atropin applied to it, even in persons in whom there is closure of the lachrymal duct; but absorption takes place more slowly than from mucous surfaces.

That the fœtus may be killed by poison taken by the mother is proved by instances in which arsenic, phosphorus, lead, mercury, and copper have been detected in the tissues of the fœtus, either after the death of the mother or after dead birth.

DISTRIBUTION OF POISONS.

The distribution of poisons in the system, *i.e.*, the relative proportion existing in different organs and tissues of the body at various stages of absorption and elimination, has as yet been studied with regard only to a few mineral poisons, such as arsenic, antimony, copper, and lead. Concerning the first two the number of observations is still small, and with regard to the last two the investigations have been for the most part in cases of chronic poisoning. Investigations hitherto made show that the distribution is different with different poisons and very probably not the same in acute as in chronic poisoning, or in poisoning by repeated doses. (See Arsenic: Distribution.)

METHODS OF ACTION OF CORROSIVES AND POISONS.

Two varieties of action are commonly recognized: a local action, that produced at the point of application, whether external or internal; and a remote action, manifested in some organ, tissue, or fluid to which the deleterious agent is not directly applied. The local action of the corrosives is intense, and their remote action insignificant. With true poisons the reverse is the case. (See Definition, p. 45.)

The **local action** is most intense with the mineral acids and alkalies. These substances disorganize those tissues with which they come in contact by chemical decompositions which

they bring about in dead and living tissues alike. The injuries caused by them during life closely resemble those caused by heat; to all intents and purposes they are burns (see Vol. I., p. 641). Some substances act both as corrosives and as true poisons, the local or remote action predominating according to the degree of concentration. This is true of oxalic acid, mercuric chlorid, and phenol, substances which have a great tendency to enter into chemical reaction with the nitrogenized constituents of the body, thereby causing destruction of the cellular elements of tissues with which they come into contact, either by direct application or by the channel of the circulation. The local action of such agents is manifested in erythema, eruptions, swelling, with or without œdema, extravasation of blood, and even formation of pus, and by sensations of pain, prickling, itching, burning, or of cold, when they are applied to the cutaneous surface; and in the symptoms of gastro-enteritis, more or less intense, when they are taken by the mouth.

In the case of arsenic, and possibly also of other poisons, the effects at the point of application, although in one sense local, are *direct* only to a limited extent, and depend rather upon absorption of the poison and its return by the circulation to the part affected. Gastro-enteric inflammation is caused by arsenic administered by other channels than the mouth, and consequently without direct application to the gastro-enteric mucous membrane.

The **remote action** of poisons, manifested after their entrance into the circulation, is local in the sense that it is exerted upon organs or tissue elements with which the substance is thus brought in contact. The view formerly held by some that poisonous influences in the case of rapidly acting poisons, such as hydrocyanic acid and nicotin, must, because of their rapidity of action, be transmitted by the nerves is no longer entertained. Even the most rapidly fatal poisons do not cause death with such rapidity that their transmission by the blood to the part affected is impossible.

Concerning the method of action of poisons upon cells and tissue elements or their constituents but little is known. A few poisons are, however, known to cause changes in the composition, chemical changes, in certain constituents of the tissues and fluids which render them unfit to perform their normal func-

tion. Thus carbon monoxid combines with the red coloring matter of blood corpuscles to form a compound more stable than that which the same pigment produces with oxygen, and thus extinguishes life by interference with the transfer of oxygen from the lungs to the tissues. It has also been suggested that the poisonous action of arsenic is due, in part at least, to interference with normal chemical processes brought about by modifications of oxidation.[1]

Rabuteau[2] has sought to show that in the case of the mineral poisons a relation exists between their atomic weights and their toxic activity, the latter being the greater as the former is more elevated; or that they are the more active the lower their specific heat.

Recent investigations of the actions of those organic poisons whose chemical constitution is known indicate that their toxic activity depends upon their molecular structure, and in the case of certain series of related or homologous compounds is increased or diminished by introduction of certain groups or atoms, or in the case of isomeric bodies by variations in their positions. Thus Gibbs and Reichert[3] have shown that the lethal doses of propyl, butyl, heptyl and octyl alcohols are progressively diminishing quantities. Bauman and Kast,[4] investigating the relative activity of the sulfones, found that those containing the radical methyl (CH_3) only are therapeutically inactive, while the ethyl (C_2H_5) derivatives have hypnotic actions increasing in activity with the number of ethyl groups contained in their molecules. The influence of orientation has been studied by Brieger and by Gibbs and Hare,[5] who found that the paratoludins are much more actively poisonous than the ortho compounds. But that, on the other hand, of the three isomeric diphenols, catechol (ortho) is the most actively poisonous, and resorcinol (meta) the least so. The lethal doses of the three are given as catechol 5, quinol (para) 10, and resorcinol 100.[6]

[1] Binz and Schultz: Arch. f. exp. Path. u. Pharm., 1879–82, xi., 200; xiii., 256; xiv., 345; xv., 322.

[2] "Toxicologie," 2ème ed., 1887, p. 11. See also Richet: C. rend. Ac. Sc., Paris, Oct. 24th, 1881, and Blake: *ibid.*, April 23d, 1888.

[3] Amer. Chem. Journ., 1894, xvi., 443–449.

[4] Zeitschr. f. physiol. Chem., 1889, xiv., 52–74.

[5] Arch. f. An. u. Phys., Phys. Abth., 1889, Supplb., 271–291; 1890, iii-iv., 344–359.

[6] For further information upon this subject see O. Loew: "Ein natürliches System der Giftwirkungen," München, 1893.

Circumstances and Conditions Modifying the Action of Corrosives and Poisons.

External Conditions.—Usually conditions independent of the toxic agent and of the subject have little influence upon the action of a poison except in so far as unfavorable circumstances, such as extremes of temperature, may diminish the resisting power of the individual, although it has been observed that when bromids are given in large doses bromism is more frequent in the colder than in the warmer half of the year.[1]

The physiological action, however, of gaseous substances is markedly influenced by the *pressure*. Thus oxygen, which, under the ordinary conditions of dilution and pressure in which it exists in the atmosphere, is a prime necessary of life, becomes an active poison under increased pressure, causing death in tetanic spasms resembling those caused by strychnin.

Conditions of the Poison—Quantity.—Even with the most active poisons there is a limit of quantity below which the substance is entirely inert. When the quantity is increased beyond this a point is reached where the effects produced are such as may be desirable under certain conditions and not deleterious. The quantity then represents a *medicinal* or *therapeutic dose*, and the effects are designated as *physiological* or *medicinal*. A further increase in quantity causes effects which, either exceed those desirable therapeutically in degree or in their nature are menacing to life or health; when the quantity taken is a *toxic dose*. And finally, by further increase a quantity is reached which, in the absence of antidotal interference, would extinguish life—a *fatal* or *lethal dose*.

The question: What is the lethal dose? of this or that poison is one almost invariably asked in homicidal cases, and one difficult of a definite answer. The question usually refers to the amount swallowed. This, even in non-fatal cases, may be greatly in excess of quantities which have in other instances been known to cause death under conditions otherwise closely similar. The actual dose of a true poison is the quantity which is absorbed, not that which has been temporarily placed in that

[1] Féré: C. rend. Soc. biol., Paris, 1893, 9 s., v., 277. See also Kossa: Arch. f. exp. Path. u. Ph., 1895, xxxvi., 120.

intermediate receptacle, the stomach, and subsequently more or less completely expelled by vomiting or otherwise. The minimum quantity of any poison capable of causing the death of a healthy adult is not definitely known, whether reference be had to the amount introduced into the stomach, or to the quantity which has passed into the circulation, or whether the poison referred to be one which, like arsenic, is almost invariably introduced by absorption, or one which, like morphin, is frequently administered hypodermically.[1] Yet we cannot doubt that the quantity of any poison, at one time operative, and capable of causing death under given conditions, has a definite minimum limit. This is, however, at present an unknown quantity. The sum of our knowledge upon this point is that a certain small quantity of the poison has been known to cause death under circumstances more or less favorable to its action, and that in other instances persons have been known to recover after having taken much larger quantities.[2]

Physical Conditions.—With the *corrosives* the most important physical condition in this connection is the *degree of concentration*, to which the gravity of the injury is directly proportionate. With the true *posions* the *degree of solubility* exerts the greatest influence upon the rapidity and intensity of action. The dictum *corpora non agunt nisi soluta* is pre-eminently true of poisons. Both poisons and corrosives act more energetically when they are *warm* than when cold.

Age of Poison.—The length of time which has elapsed since the preparation or solution of the poison may in certain cases have permitted it to become almost or entirely inert by decomposition. Thus solution of hydrocyanic acid rapidly deteriorates by exposure to light and air, and the dilute acid of the Pharmacopœia is rarely met with in pharmacies of the two-per-cent. strength called for; solutions of morphin form an excellent culture medium for certain moulds, which convert the alkaloid into the less active oxydimorphin; and solutions of aconitin lose their activity rapidly.

[1] See Arsenic and Morphin, Lethal Dose.

[2] We deem it necessary in this connection to caution the legal reader against acceptance of reports of cases of death from small doses of poisons recorded in medical literature. In the great majority of "small-dose cases" either the quantity actually taken is open to question or other and more probably operative causes of death existed in the subject.

Conditions of the Subject—Species and Race.—There is probably no poison which acts with equal intensity upon all forms of life. The germicidal activity of mercuric chlorid is at least five hundred times as great as that of arsenic trioxid, while the two poisons are fatal to man in about equal doses. Of the albuminoid substances having poisonous qualities, some (the toxalbumins) are produced by bacteria, and are poisonous to animals; while others (the alexins or antitoxins) are produced physiologically or pathologically in animals, and are poisonous to bacteria. Ascarides are not poisoned by strychnin, which destroys their host. Quinin is more poisonous than strychnin to infusoria and to diatoms, while the reverse is the case with vertebrates. Neither alkaloid has any considerable influence upon the bacteria of putrefaction. A rabbit will bear more atropin or morphin than a man whose weight is fifty times as great. Goats are not affected by quantities of nicotin or of lead sufficient to destroy human life. Amygdalin is poisonous to rabbits but not to dogs. Frogs are very susceptible to the action of the digitalis glucosids, which have very little effect upon toads. Instances such as these, which might be multiplied, indicate that great caution is necessary in drawing deductions applicable to the human subject from experiments upon animals. While the result of such experiments are frequently of great value in establishing the identity of certain poisons (see Physiological Tests), and in studying the method of action of poisons and medicines under conditions not easily realized otherwise, they should only be considered as indicating the action upon the human subject when supported by corroborative evidence obtained from observations made upon man either in suicidal or accidental poisonings or by experimentation within the limits of propriety and safety. In some cases of homicidal poisoning evidence also has been furnished by the poisoning of domestic animals that have eaten the remains of the food in which the poison was administered or the vomit of the victim, and by examination of their bodies.

Differences in the actions of certain poisons have also been observed in different races of men. Thus opium, which causes a sleepy, dreamy condition in Caucasians and Chinese, is said to affect Malays and Japanese with murderous mania. It is also

said that narcotics in general are more active in the inhabitants of warm climates than upon northern races.[1]

Whether these differences are due to varying peculiarities in organization of the individuals of different races, or to other causes referred to in this section, is not determined.

Age.—The doses of medicinal substances properly administered to children are not only absolutely smaller than those given to adults, but are also less relatively to the weights of the two, the susceptibility of the child being greater than that of the adult. The narcotics particularly are much more active upon young than upon adult subjects. In old age the resistant power of the individual diminishes, and changes, particularly in the walls of the arteries, of a character favoring a fatal action, are more frequently present. Old persons have in several instances been killed by doses of drastics and of digitalis which would have caused only the usual therapeutic actions in persons of middle age.

Some exceptions to the rule that young individuals are more susceptible to the action of poisons than those that are full grown have been observed. Thus children take calomel without salivation longer than do adults;[2] and new-born puppies withstand larger doses of strychnin than full-grown dogs.[3]

Condition of Health.—It may be said, in general, that persons in health are better capable of withstanding the action of poisons than those whose physical condition is impaired by excesses or by disease. Some poisons also, by similarity of their effects with those caused by certain diseases, may bring about the death of a person so affected when taken in small doses. Thus ordinary medicinal doses of opiates may cause a fatal result in a person already on the verge of apoplexy. Tartar emetic or chloroform may suddenly extinguish the life of a person having organic disease of the heart. Persons in a starving condition have been fatally poisoned by quantities of poisonous berries and of putrid food, which would at most have provoked vomiting in a person in a well-nourished state.

In such cases a question may well arise as to whether the disease or the poison was the cause of death. When there is no question of the administration of the poison, and death has

[1] Husemann: "Tox.," 29.
[2] *Ibid., op. cit.*, 31.
[3] Bert and Demant, *ex* Loew: "Syst. d. Giftwirk.," 88.

followed after the manifestation of the symptoms usually caused by it and within the usual time in which that poison causes death, the disease is to be looked upon as a condition, and the death should be attributed to the poison. But it is possible that a diseased person may take a poison, manifest the symptoms attributable to the combined or associated actions of the poison and disease, and yet die of the disease. Thus, through the negligence of a physician, a child of about four months of age was given four drops of deodorized tincture of opium at 9 A.M. Between noon on that day and the next morning the child manifested marked symptoms of opium poisoning, for which it was treated by the usual methods by another physician. During the two following days the pupils regained their normal size, the child cried frequently, had a temperature ranging from 103° to 105.5° and died eighty-eight hours after administration of the opiate, with symptoms of compression of the brain. The autopsy and the clinical history showed that, while the child had been dangerously poisoned by opium, it had recovered from the effects of the narcotic and had died of acute hydrocephalus.

The theory of death from poisoning by improper medication has also been advanced in homicide cases of death alleged to be caused by wounds. Thus, in the case of Stokes, it was claimed by the defence that the death of the deceased was not due to the pistol-shot wound which he received, but to the action of morphin administered by the surgeons who attended him.[1]

On the other hand, patients suffering from certain diseases manifest, apart from habituation, a tolerance for particular medicines in doses which would be poisonous to a healthy person. Thus large doses of opiates are frequently given to persons suffering from dysentery, tetanus, cholera, or alcoholic mania without the production of poisonous effects; syphilitics bear doses of mercury or of iodin which would cause symptoms of poisoning in healthy individuals; and strychnin has been administered to a hemiplegic to the daily amount of three grains without injurious consequence. But this immunity has a limit, and when the quantity of the poison is further increased toxic effects are produced.

Mental influences may also cause an explosion of symptoms

[1] Peugnet: Papers Med.-Leg. Soc., New York, 1882, 2d ser., 294–333.

of poisoning in a person saturated with a drug taken medicinally or habitually. This is frequently observed in attacks of delirium tremens, and Féré [1] has reported a case of acute bromic poisoning in an individual who had been under the drug for three years, which was clearly due to moral shock.

Continued Action.—The corrosives frequently cause death by mechanical stricture or closure of the openings of the stomach and by destruction of the gastric follicles, weeks or months after the primary action has ceased, by the intensity of the inflammatory processes set up. Some poisons, also, as arsenic, may establish a gastro-enteritis of sufficient intensity to destroy life after the last traces of the poison have been eliminated from the system.

Cumulative Action.—The action of a poison or medicine may be cumulative either in the sense that repeated small doses produce effects different from those which would result from the administration of a single dose, even much greater in amount; or in the sense that, during the administration of small doses, the effects of a single large dose may be produced. In the first sense all medicines and poisons are certainly "cumulative," and so-called "chronic" poisonings are the results of such repeated small doses. The occurrence of poisonings cumulative in the other sense is most exceptional. It is stated that the glucosids of digitalis "have the remarkable property that during the continued administration of small doses the effects of a single large dose appear." [2] The same is said to be the case with colchicin.[3]

Idiosyncrasy-Intolerance.—By *idiosyncrasy* in the medical sense, or *idiocrasy*, is understood a peculiar susceptibility, causing peculiarities of effect from the ingestion of certain substances, and from external influences. Idiosyncrasies of odor, taste, smell, and sound are not of interest here.

Many instances are recorded of persons upon whom ordinary articles of diet produce most unusual effects. Strawberries have been known to cause febrile symptoms and convulsions. A case is reported by Fergus [4] of a person who on three different occasions experienced severe symptoms on merely tasting a strawberry. Once, in less than three minutes, failure of the heart's

[1] C. rend. Soc. Biol., Par., 1893, 9 s., v., 277.

[2] Husemann u. Hilger: "Pflanzenstoffe," p. 1,240.

[3] Lewin: "Toxikol.," 7.

[4] Lancet, 1869, ii., 563.

action set in and prolonged fainting ensued. Lobsters and oysters have been frequently known to cause urticaria, and we have met with a lady in whom the most violent attacks of vomiting and sickness always followed the eating of a cooked oyster, while the raw bivalve could be eaten in moderate quantity with impunity. Honey has been known to cause severe poisoning in several cases. In these, however, the toxic effects are traceable to the fact of the bees frequenting poisonous plants, as in the case of the honey of Trebizonde, collected from a species of rhododendron, the *Azalea pontica*.[1] Cocoa and beans have also been known to cause poisoning in some persons.[2]

Medicinal and toxic agents also act more energetically than usual upon certain persons, and in some cause symptoms varying from those usually produced. The wide variations in the symptomatology of arsenical poisoning may be cited in support of the latter statement. Instances of intolerance of certain drugs are frequently met with: rhubarb, manna, and even mineral waters have been known to produce violent vomiting and purging in small doses; the balsams and turpentines occasionally produce urticaria; belladonna, even in very small doses, has caused a scarlatinal rash and toxic symptoms, and even the application of the ordinary belladonna plaster has produced dilatation of the pupil and marked dryness of the throat. Idiosyncrasies with regard to the action of mercurials, quinin, and Epsom salt have also been observed.[3] It is particularly with the modern synthetic remedies, chloral, chloroform, sulfonal, etc., that marked variations in the action upon different individuals, and even upon the same individual at different times, have been observed. It is probable, however, that in many of these cases the amount of the dose actually taken, or the purity of the product, is questionable, or some other cause of death was also present.[4] The following statement of Leydel[5] concerning the

[1] Pavy: "Food and Dietetics," Phila., 1874, p. 321. See also Bidie: Madras Q. J. M. Sc., 1861, iii., 399. Coleman: New Jersey M. Reptr., Burlington, 1852, vi., 46.

[2] Kobert: "Intoxikationen," 23.

[3] See MacDonnell: "Wood's Reference Handb. of the Med. Sci.," iv., 2. Ascherson: "Wchnschr. f. d. ges. Heilk.," Berlin, 1837, iii., 817. Betancourt: Phil. Med. Times, 1881–82, xii., 72. Ford: Mich. Med. News, Detroit, 1878, i., 98. François: Gaz. méd. d. Strassb., 1863, xxiii., 77. Smith, R.: Edinb. Med. Journ., 1876–77, xxii., 601–603.

[4] In this connection see Börnträger: Viertljschr. f. ger. Med., n. F., 1889, lii., 306–323; 1890, liii., 19–66.

[5] Leitfaden d. ger. Med., Berlin, 1895, 107.

toxic action of non-synthetic poisons is in the main correct: "That in adults a peculiar idosyncrasy toward particular poisonous substances exists is not as yet proven. Reference is had as a rule to the different methods of application, which in the case described causes a more rapid and more powerful action, than in others in which equally large doses are in question."

Habit-Tolerance.—A much more important condition is the tolerance of certain poisons which is gradually established by their habitual use. The increasing quantity of morphin or of the opiates which is taken by those addicted to the opium habit is well known, and quantities are finally taken by them which would suffice to destroy several non-habituated persons, and would have proved fatal to them if taken at the outset. Habitual use of alcohol, hashish, cocain, nicotin, arsenic, chloral, etc., also establishes a tolerance of those poisons. Not only that, but the poison becomes a necessity, the deprivation of which causes serious effects, in some instances, as with arsenic, resembling those produced in normal individuals by the ingestion of a poisonous dose of the same poison. It is probable that a similar habituation may be produced with many poisons and medicines other than those customarily used by toxicophagists. Thus Kobert[1] refers to a feast in China at which all the Europeans were attacked with purging, while all the Chinese remained unaffected, in consequence of the food having been broiled with castor oil according to the Chinese custom.

The immunity from the action of poisons established by habit is, however, never complete, and when the dose taken surpasses a certain limit the symptoms of poisoning are manifested. Numerous deaths of morphin, cocain, chloroform, and arsenic habitués caused by overdoses have been recorded.

Action of the Liver upon Poisons.—By an elaborate series of experiments and observations Roger[2] has demonstrated that the liver acts as a protector of the organism against poisoning; that most of the substances carried to it by the portal vein are arrested, some are retained, others eliminated, and others profoundly modified. Many mineral substances, such as iron, copper, lead, arsenic, etc., accumulate in its parenchyma, and alkaloids, nicotin, quinin, morphin, cocain, atropin, hy-

[1] "Intoxikationen," p. 23.

[2] "Action du foie sur les poisons," Paris, 1887.

oscyamin, strychnin and veratrin, and curare and ammonia are arrested in their absorption by the liver. Injections of these poisons into the portal vein act with only half or a third the intensity of similar quantities injected into the jugular. The peptones, putrid poisons, and the toxic products of intestinal fermentations are also arrested by the liver. The normal portal blood of the dog has double the toxic power of the blood of the hepatic veins of the same animal when injected into the peripheral circulation of rabbits. The protecting influence of the liver is directly proportionate to the amount of glycogen present. This accumulation, or deposition of poisons in the liver, which renders that organ the best situation in the cadaver for the detection of absorbed poison, can only be explained on the supposition of the conversion of the dissolved poison into a less soluble combination, which is deposited. That such compounds are formed in the liver and elsewhere has been demonstrated. The metallic poisons combine with albuminoids to form sparingly soluble *albuminates*, and de l'Arbre[1] has shown that many alkaloids form compounds with the biliary acids, which are for the most part difficultly soluble. Roger[2] considers it probable that the alkaloids may enter into combination in the liver with glycogen to form sparingly soluble conjugate compounds similar to the glucosins obtained by Tanret[3] by the action of glucose and ammonia upon each other under pressure. It has been demonstrated that the quantity of glycogen in the liver is rapidly diminished, frequently to the point of entire disappearance, by the action of many poisons, such as arsenic, antimony, phosphorus, morphin, strychnin, and chloroform;[4] and that with several poisons, such as chloroform, carbon monoxid, etc., the disappearance of glycogen is accompanied by glycosuria. Külz[5] has found that in rabbits strychnin causes the disappearance not only of the liver glycogen, but also the much more resisting muscle glycogen, which he attributes to the muscular activity due to the tetanizing action of the poison. The in-

[1] "Verbind. einzelner Alkaloide mit Gallensäuren," Diss., Dorpat., 1871.

[2] *Loc. cit.*

[3] Journ. d. Ph. et de Chim., 1885, 5 s., xii., 105.

[4] Saikowsky: Archiv f. path. Anat., etc., Berlin, 1865, xxxiv., 79. Rosenbaum: Arch. f. exp. Path. u. Ph., 1882, xv., 450. Demant: Ztsch. f. physiol. Ch., 1886, x., 441. Kimpel: Sitzber. d. phys.-med. Gesell. z. Würzb., 1893, 135.

[5] Marb. Festschr., 50jähr. Jubil. C. Ludwig, p. 119.

solubility of any of the compounds whose formation in the liver is presumed to occur is not absolute, and consequently the action of the liver is not to prevent but to mitigate the action of the poison, to extend the time during which the poisonous agent passes into the general circulation. Moreover, the alkaloidal salts of the biliary acids, although difficultly soluble in water, are readily soluble in the presence of an excess of the sodium salts of the same acids which exist in the bile; and it is well known that not only alkaloidal but other poisons enter into the bile and are thus discharged into the intestine to be either excreted with the faeces or reabsorbed gradually and during several hours later.

Antidotal Actions by the Animal Economy Itself.—Many poisons, after introduction into the body, are to a greater or less extent converted by chemical reactions into compounds whose toxic powers are less marked. These changes are brought about by several different methods. In some cases it is a mere *neutralization*, as when absorbed acids are converted into their alkaline salts. By *oxidation* phosphorus is to some extent converted into the phosphates, which are normal constituents of the body, and the highly poisonous sulfids are oxidized to the harmless sulfates. By *reduction* potassium chlorate is converted into the much less poisonous chlorid; and chloral [trichloraldehyde, ($COH.CCl_3$)] is first reduced to trichloralcohol ($CH_2OH.CCl_3$), which is subsequently further modified as described below, by *synthesis* or formation of *conjugate derivatives*. Thus the poisonous phenols combine with the sulfates to produce non-poisonous ether-sulfuric acids which appear in the urine; the camphors, naphthols and chloral, are eliminated in the form of conjugate derivatives of glycuronic acid ($C_6H_{10}O_7$), itself a derivative of albuminoid substances; and benzoic and salicylic acids and their congeners form non-poisonous glycocol conjugates. By *decomposition* certain poisonous glucosids are split up into non-poisonous substances.

The seat of these transformations in the body has been determined only imperfectly. Some, such as the conversion of benzoic into hippuric acid, take place in the kidneys. The liver is probably an active agent in this respect, and is known to be the seat of the formation of the glycuronic conjugates. Other changes, particularly decompositions such as that of the glucosids, occur in the alimentary canal.

It is clear that with the organic poisons the occurrence of such transformations in the system is of great forensic interest. The organic poison found by the chemist in the cadaver in the form in which it was administered is only the residue which has escaped modification, and it is quite conceivable that the tranformation may have been complete, so that only the products of the chemism of the system upon the substance introduced remain. As the exact nature of these products derived from many organic poisons is as yet unknown, and the possibility of their formation from other substances than the poisons from which they are known to be derived is in most instances still unexcluded, the difficulties in the way of proving the presence of absorbed organic poisons are great, except with a few which resist modification, or whose changes in the body have been more carefully studied.

Compound Poisoning.—When two or more poisons are taken together, or in rapid succession, the action of each may be modified by that of the other or others; but in what manner and to what extent cannot be predicted, save in some exceptional instances. The variations in the combined effects of strychnin and morphin are well illustrated in the following cases: A man, twenty-nine years of age, took 0.2 gm. (gr. iij.) of strychnin, 3.9 gm. (℥ i.) of opium, and an unknown quantity of quinin. Twelve hours after he complained only of "feeling queer." Later he manifested symptoms of strychnin poisoning of a mild type; after which he became profoundly narcotized and died forty hours after taking the poison.[1] Marvin[2] has reported a case in which death was caused by 0.65 gm. (gr. x.) each of morphin and strychnin, and in which the symptoms of morphin poisoning could hardly be recognized. In Beatty's case[3] a woman of twenty-two years took 0.1 gm. of strychnin (a packet of Battle's vermin killer), and immediately afterward 7.4 c.c. (℥ ij.) of laudanum, and then 2 gm. (℥ ss.) of red precipitate. In three hours she was found suffering from narcotic poisoning. She recovered under treatment, and at no time manifested any symptom of strychnin poisoning. Almost the same proportion of strychnin and laudanum were taken, with very different results, in a case reported by Harrison:[4] A man of

[1] Chicago Med. Journ., 1860, xviii., 635.
[2] Med. Herald, Louisville, 1879, i., 4.
[3] Lancet, 1871, ii., 907.
[4] *Ibid.*, 1882, i., 780.

fifty-four years, who had been drinking, took a package of Battle's vermin killer (0.1 gm. strychnin) and 5.6 c.c. (3 iss.) of laudanum. In an hour he had a spasm, and afterward had violent tetanic convulsions, from which he recovered under treatment, without having manifested any symptom of narcotic poisoning. A case intermediate in character between these has been reported by King,[1] of a man of seventy years who took 0.26 gm. (gr. iv.) of morphin and "some" strychnin. He suffered violent tetanic spasms and during the intervals had all the symptoms of opium poisoning. After a most violent convulsion he went to sleep and slept nine hours, and subsequently recovered.

A similar variation, although not so marked, has been observed in three cases of poisoning by compound liniment of belladonna and aconite, in which, clearly, the relative proportion of the two poisons was the same, although the absolute quantity varied with the dose.[2] In Simpson's case the pupils are said to have been contracted, although both belladonna and aconite cause dilatation.

When one poison is given before another the effects of that last administered may be modified. Thus Preyer[3] has shown that animals under the influence of atropin are not killed by doses of hydrocyanic acid sufficient to cause the death of individuals not so prepared, and Stillé[4] states that double the quantity of hydrocyanic acid necessary to cause death, injected hypodermically into a dog under the influence of ether, failed to produce any evidence of poisoning for three-quarters of an hour during which ether was being inhaled, but caused the usual symptoms of poisoning and death on cessation of the etherization. Or if atropin be administered first and morphin afterward, the quantities may be so adjusted that the influence of the morphin upon the pupil will be masked and the clinical history of the case will record a combination or succession of effects of the two alkaloids.

If the substances administered differ materially in the rapidity of their action the victim may either succumb to the more

[1] Louisville Med. News, 1880, x., 160.

[2] Richardson: Brit. Med. Journ., 1885, i., 327. Lipscomb: *Ibid.*, 1888, i., 694. Simpson: *Ibid.*, 1882, i., 774.

[3] "Die Blausäure," i., 73.

[4] Wharton and Stillé: "Med. Jur.," 4th ed., ii., 62.

rapidly acting poison or corrosive, without having manifested the symptoms of that which behaves more slowly, or may recover from the effects first caused and subsequently die from the action of the slower poison. Thus Ludwig [1] cites the case of a man who died in five hours from the effects of caustic potash which he had taken along with Paris green and phosphorus. And Maclaren [2] reports the case of a man of twenty-two years, who took a combination of strychnin and phosphorus, and after having recovered completely from a violent attack of strychnin poisoning, died in five days with all of the symptoms caused by phosphorus. When complex mixtures containing several poisonous ingredients are taken, life may be prolonged for a longer period than is usual in fatal poisonings by the most active ingredient, and death may result after an illness the clinical history of which is rather a combination or succession of the effects of the different constituents of the mixture than that of any one. A case illustrating this, in which a woman of sixty-eight years died in four days from the effects of a dose of chlorodyne, has been reported by Leighton.[3] Much will, however, depend upon the magnitude of the dose of a given mixture. Thus while in most cases of chlorodyne poisoning (see Opium) the clinical history is either of the complex type just referred to, or that of profound narcotism, in Browne's case [4] of a man who committed suicide by swallowing three bottles of chlorodyne, the patient was found in a dying condition and expired soon after with manifestations of a distinctly cyanic character.

ELIMINATION OF POISONS.

Poisons existing in the blood of living beings are separated therefrom and discharged from the body, in a more or less modified form, by all of the excretory organs, and by other channels not physiologically excretory. The mere expulsion of the contents of the stomach by vomiting is not, strictly speaking, elimination, but operates rather to prevent absorption.

As a rule, elimination takes place principally by the urine, except with gaseous poisons, which are eliminated mainly by

[1] Med. Jahrb., 1880 (reprint).
[2] Glasgow Med. Journ., 1881, xv., 320.
[3] New Engl. Med. Mthly., 1882–83, ii., 270.
[4] Austral. Med. Journ., 1879, n. s., i., 587.

the lungs. The excretory function of the liver also plays an important part in the elimination of many poisons which pass into the bile, and are so either separated in the alvine dejections or reabsorbed in a modified form to be subsequently eliminated by the kidneys. Elimination also takes place by the perspiration, the serum of vesications and of serous effusions, the saliva, the pancreatic, gastric and intestinal secretions, and the milk. The excretory action of the stomach is notable with some poisons, such as arsenic and iodin. The latter, when administered by other channels, is eliminated principally by the gastric follicles and vomiting.

The duration of elimination and the relation between the rapidity of absorption and that of elimination are questions of great forensic and clinical interest. The beginning of elimination is probably coincident with or very shortly follows the beginning of absorption. Potassium iodid may be detected in the urine ten minutes after its administration; and potassium ferrocyanid has been found in the same excretion two minutes after having been swallowed. The rapidity of elimination and its duration vary with different poisons, and also with the same poison according to the form and manner in which it is taken and variations in the species and physiological conditions of the subject. As a rule, metallic poisons are eliminated more slowly than alkaloidal or synthetic poisons. With some poisons, such as the copper salts, absorption is rapid and elimination quite slow. With others the rapidity of elimination as compared with that of absorption from the intestinal canal is so great that the substance, although actively poisonous when injected into the circulation, is inert when given by the stomach. Thus curare is non-poisonous when swallowed by mammals if the function of the kidneys is unimpaired, but produces the same symptoms of poisoning as it does when injected into the blood if administered to an animal in which the renal arteries have been previously tied. The functional activity of the kidneys consequently is an important factor in influencing the effects produced by a given dose of poison, and persons whose kidneys are diseased may be expected to suffer more serious effects than those whose kidneys are normal from the same dose of a given poison, for the same reason that serious disease of the kidney causes auto-intoxication, *i.e.*, deficient elimination of the poison.

The rapidity of renal elimination is diminished and its duration is increased with the age of the individual. Brouardel[1] administered a gram of salicylic acid during a day to each of three healthy persons, aged respectively twenty, forty-five, and seventy years. The acid was detectable in the urine of the first in one hour after breakfast, in that of the second in the evening, and in that of the third only on the third day. The elimination was completed in the first the day after the administration, in the second in four days, and in the third in nine days.

TREATMENT OF POISONING.[2]

Removal of Cause.

The first indication in a case of true poisoning is the removal from the system of any poison which may yet remain unabsorbed, or its conversion into some insoluble or inert derivative or compound. The former end is attained by the administration of emetics or purgatives, or by physical means; the latter by the action of the so-called chemical antidotes.

Removal of the Poison from Wounds.—As absorption from wounds is very rapid, attempts at the removal of poisons so introduced must be made soon after the introduction to be of any avail. The means adapted to this end are squeezing or sucking out the poison, or washing it out, preferably with dilute hydrogen peroxid if the presence of putrid substances or bacteria be suspected; the application of tincture of iodin to "post-mortem" or "dissection" wounds; and the application of the actual cautery to the poisoned wounds inflicted by venomous animals. If the wound be in an extremity the intensity of the poisoning may be diminished if a ligature be placed to compress the veins above the injury immediately after its infliction, and venesection practised below.

Removal of Poison from the Stomach.—This is accomplished by the action of emetics or by mechanical means, which should always be promptly resorted to in cases of poisoning,[3] except it be known that the poison is already com-

[1] Ann. d'hyg., 1894, 3 s., xxxii., 483.

[2] For treatment of corrosion by acids and alkalies see pp. 345, 469.

[3] It is to be as strictly avoided when a corrosive has been swallowed.

pletely removed from the stomach, in which case the administration of emetics is not only not beneficial but harmful; or when it is known that the poison has been administered by some channel other than the stomach.

The most available *emetic* is apomorphium chlorid, which acts within three or four minutes, may be always within the immediate reach of the physician, and may be administered in spite of the resistance of the most obstinate suicide. The physician's pocket case should always contain compressed tablets of 0.007 gm. (gr. $\frac{1}{10}$) of the officinal salt, one or two of which may be dissolved in about thirty drops of water and injected hypodermically within a very few minutes. Immediately after the injection the usual mechanical methods of provoking emesis should be adopted if possible, such as tickling the fauces with a feather, bending the body forward and manipulating the epigastrium from the sides toward the median line, and the administration of lukewarm water into which some butter or lard has been melted. If an emetic is to be given by the mouth, zinc sulfate should be used in doses of 1.5 to 2 gm. (gr. xx.–xxx.), or, in cases of phosphorus poisoning, cupric sulfate in doses of 0.2 to 1. gm. (gr. iij.–xv.). Tartar emetic should never be used.

A more prompt and radical method of removing poison from the stomach is by the use of the *stomach pump* or, preferably, the *siphon*. The latter consists of a rubber tube of about 8 mm. internal diameter and 2 m. in length, one end of which is closed and rounded, and has near it a single lateral opening. The rounded end, well oiled, is introduced into the pharynx and pushed gently down the œsophagus into the stomach, with the aid, if possible, of an effort of swallowing on the part of the patient. A funnel is now placed in the free end of the tube and through it from half a litre to a litre of lukewarm water, or, preferably in poisoning by the alkaloids, infusion of tea or coffee, is poured into the stomach, the tube being compressed to closure near the funnel before the latter has been completely emptied. The funnel is now removed, the free end of the tube depressed while still closed, and the pressure released, when the contents of the stomach are siphoned into a vessel placed for that purpose. More than a litre of liquid should not be used at one time, and the operation may be repeated two or three times with fresh liquid. The pipe should be introduced in adults to

a length of 45 to 50 cm. from the teeth. Occasionally the opening of the siphon in the stomach becomes plugged by solid particles of food or of the poison too large to enter it. In that event, the tube should be withdrawn after a few gentle movements in and out for a short distance and of rotation, by which vomiting is usually mechanically induced. A siphon may also be extemporized from an ordinary œsophageal sound having a rubber tube fitted to the free end. The sound should, however, have but a single lateral opening, and being of a harder material than the soft-rubber siphon is not so desirable. The introduction of the pipe is sometimes interfered with by spasms of the œsophagus; when this occurs the end of the pipe may be smeared with an ointment containing cocain, as suggested by Lafosse.[1]

Removal of Poison from the Intestine.—If the poison has already passed into the intestine the administration of a purgative may be desirable, particularly if the substance taken be known to be one which is slowly absorbed from the intestine. The purgative used should be one which has no solvent action upon the poison, and if possible one which tends to render it less soluble. Thus castor oil should not be given in phosphorus poisoning, as the oil dissolves that element, and sodium or magnesium sulfate is to be selected in lead poisoning, with a view to the formation of the insoluble lead sulfate. A recto-colonic tube may be used with advantage in some instances, and the rectum and colon washed out.

Antidotes to Prevent Absorption.—These may act either physically or chemically. The most generally useful of those acting physically is animal charcoal in moderately fine powder, which has the property of absorbing and retaining alkaloidal and also, to a less degree, mineral poisons. To be of service it must be freshly burnt or must have been kept in well-closed vessels after having been burnt. Vegetable charcoal possesses the same property, but in a much less degree. It may be added to the liquid used in washing out the stomach. Either form of charcoal is itself non-poisonous and is not contra-indicated in any form of poisoning.

Albumen, in the form of white of egg, is readily obtainable and is never harmful. Its action is partly mechanical in im-

[1] Bull. gén. de thérap., etc., Paris, 1885, cix., 128.

prisoning the particles of a still undissolved poison and preventing its adherence to the walls of the stomach, and partly chemical in forming sparingly soluble compounds with many of the mineral poisons and with some of those of vegetable origin. If the albumen be not spontaneously vomited after a few moments it should be followed by an emetic, particularly in poisoning by corrosive sublimate, to prevent its resolution by the gastric secretion.

Milk is not so advantageous as albumen, as it is distinctly contra-indicated in poisoning by phosphorus or arsenic, by reason of the solvent action of its fat upon the former, and of its alkali upon the latter. Moreover, the albuminoids which it contains do not combine with poisons so readily as do those of white of egg.

Other demulcents, such as mucilage, gelatin, tragacanth, etc., act only mechanically. The chemical antidotes are administered with the view of forming sparingly soluble or insoluble compounds, such as tannin produces with the alkaloids, glucosids, and antimony and zinc compounds; ferric hydrate with arsenic; slaked lime with oxalic acid; common salt with silver nitrate, etc.; or of converting a harmful substance into one which, although soluble, is comparatively harmless, as magnesia usta forms magnesium sulfate with sulfuric acid, and vinegar converts caustic potash into potassium acetate.

Symptomatic Treatment.

To combat the effects of absorbed poison the efforts of the physician should be directed to the counteraction by physical or antidotal means of individual effects which are menacing to life, and thus bridging over a critical period until the natural processes of the economy have either eliminated the poison or rendered it harmless.

The objects to be attained are most frequently to prevent cessation of respiration or of the heart's action, or to re-establish one of those functions after a short interruption. The **physical means** adapted to these ends are:

Artificial Respiration.—This is one of the most valuable means of preventing death by all poisons which cause death by affecting the respiration. It should be resorted to whenever

there is threatened arrest of respiration, and should not be abandoned as useless so long as the action of the heart is perceptible.

Before proceeding with artificial respiration the freedom of the upper air passages from obstruction must be assured. Mucus and fragments of vomited matter are removed from the pharynx, if present, by the finger covered with a handkerchief. In syncope and narcosis the tongue falls back, and pressing upon the epiglottis closes the entrance to the larynx. To free this opening the operator, facing the patient, places his two thumbs on the two sides of the upper jaw below the zygoma, and with the fingers behind the angles of the lower jaw draws it and the attached soft parts as well forward as possible.[1] If the laryngeal opening is closed by œdema which cannot be overcome by the finger, intubation of the larynx may be practised or tracheotomy performed. The latter operation is also sometimes called for when the jaws are fixed by trismus. The blowing of expired air into a tracheal tube is not desirable, owing to its large percentage of carbon dioxid. Air from a bag with properly constructed valves may, however, be used, provided the apartment be warm and the air be filtered through a cotton plug.

If an assistant be available, the best method of artificial respiration in cases of poisoning is that of Sylvester, combined with Heiberg's manipulation. The patient is placed upon his back; one operator, standing or kneeling astride the hips, leans forward and, without pressure upon the body, holds the tongue forward in the manner above described, while the second, kneeling above the patient's head, seizes the arms near the elbows and draws them above the head, holds them there for two or three seconds, and then, bending the arms down, presses them against the sides of the chest to expel the air for the same length of time. The movements are repeated about fifteen times a minute. The operators should alternate with each other and will be materially aided by a third or fourth, particularly in opium poisoning, in which artificial respiration may have to be continued for a number of hours. A single operator should select Marshall Hall's method while waiting for further assistance. He kneels beside the patient and with the right forefinger maintains the tongue depressed and drawn as far forward

[1] Heiberg: Berl. kl. Wochenschr., 1874.

as possible, while with the left hand he rolls the body upon its face and presses upon the abdomen to expel the air, then rolls it upon the back to permit entrance of air by the return of the elastic walls of the chest to their normal position.[1]

The **faradic current** is a powerful aid to or substitute for artificial respiration. The cathode of a faradic battery of moderate power is fitted with a double (bifurcated) electrode, the two parts of which are applied over the phrenic nerves on each side of the root of the neck above the clavicles and behind the sterno-cleido-mastoid muscle, which is pressed somewhat forward; the flat anode is applied to the epigastrium. The current is closed for about two seconds at a time, at intervals also of two seconds. While the current is closed the head, shoulders, and arms are fixed by an assistant, and pressure is exerted upon the abdomen in the intervals. From time to time a short pause is made in the faradization to observe whether and to what extent natural respiration is established.

When failure of respiration is threatened from paralysis of the respiratory centres due to insufficient blood supply, as evidenced by coma accompanied by a slow, feeble pulse, the action of gravity may be utilized to favor the blood supply to the brain by inclining the patient with the head downward. This is the opposite to the "ambulatory treatment" adopted in the threatened coma from cerebral congestion produced by morphin poisoning.

The **warmth** of the body must be maintained in poisonings by agents, such as chloral and other hypnotics and narcotics, which cause diminution of the body temperature as evidenced by the indications of the thermometer and the coldness of the nose and extremities. The room in which such patients are treated should be warm—not less than 70° F. Artificial heat should not be applied to the patient too suddenly. The temperature should be gradually raised by simply placing the patient in bed with sufficient covering, and the application of hot-water bottles or bags to or near the soles of the feet, or by friction with warm towels. If a warm bath be used it should not be resorted to until after the body temperature has approached the normal. All liquids introduced into the body should be previously warmed to about 100° F.

[1] For other methods of artificial respiration see Satterthwaite, "Wood's Ref. Handb. Med. Sc.," i., 377–380.

The use of the **cold douche** or **ice bags** applied to the head in cases of narcotic poisoning attended with cerebral congestion, as in opium poisoning, may diminish the sopor and tendency to coma, but, if incautiously applied, may operate detrimentally in further diminishing the already low body temperature. If they be used the body must be kept warm, either as indicated above or by immersion in a bath of about 100° F.

Stimulants other than warmth and electricity are frequently indicated—either mild stimulants such as tea and coffee, or more powerful agents such as brandy, ether, acetic ether, or camphor hypodermically. Inhalation of ammonia serves as an active respiratory stimulant in syncope, but it must be remembered that death has been caused by the use of this agent in too concentrated a form for this purpose.

Transfusion of Blood is rarely possible, of questionable utility, and attended with danger. **Bleeding**, followed by **infusion** of a saline solution such as Little's[1] in double the quantity of the amount of blood removed, has, however, been found in experiments upon animals to be of benefit in poisoning by hydrocyanic acid, carbon monoxid, and nitrobenzol, by removing a large proportion of the absorbed poison.

Physiological Antidotes—Antagonism of Poisons.—The idea that for each poison there is a counter-poison is as old as the Ayur Vedas, which antedate B.C. 600, and is not yet eradicated from the popular mind. Yet there are no two poisons whose actions are in all respects opposite or antagonistic. The nearest approach is with muscarin and atropin, but even with them the antagonism is not complete, although the administration of atropin rapidly counteracts the menacing symptoms caused by muscarin. Physiological antidotes are of value only when they tend to counteract an effect of the poison which is dangerous to life, as when chloral or chloroform is given in strychnin poisoning to prevent or mitigate the tetanic spasms. On the other hand, there are many cases reported in medical literature as cases of morphin poisoning which are in truth poisonings by atropin and morphin, the former alkaloid having been administered as a physiological antidote.

[1] Sod. chlor., ʒ i. ; potass. chlor., gr. vi. ; sod. phosph., gr. iij. ; sod. carb., gr. xx. ; alcohol, ʒ ij. ; aq. dest., ℥ xx.

In poisonings or corrosions attended with great pain it is not only the part of humanity, but also serves to maintain the resisting force of the system, to allay the pain by the hypodermic administration of morphin unless its use is directly contra-indicated.

PROGNOSIS OF POISONING.

The action of a poison or corrosive may result in death, in complete recovery, or in partial recovery.

Death from the primary action of poisons and corrosives may be due to *exhaustion* caused by persistent vomiting, violent convulsions, or severe pain; to *cerebral paralysis* affecting the respiratory centres; to *cardiac paralysis;* to *œdema* of the lungs or of the glottis; to *internal asphyxia* by modification of the hæmoglobin of the blood; to *diminished body temperature;* to *hemorrhage* caused by perforation of blood-vessels by corrosion; or to more remote effects, such as suppression of urine, etc.

Complete recovery results in most cases of non-fatal poisoning in which the toxic agent is eliminated, and in which the pathological changes produced are either insignificant in kind, or of such nature that progressive repair follows the removal of the cause.

Partial recovery is practically recovery from the primary effects of the deleterious substance, followed by prolonged illness or death from its secondary effects. Thus death from *starvation* frequently occurs months after the primary effects of the mineral acids and alkalies have disappeared; *atrophy* and *degeneration* of various organs follow as secondary effects of several poisons, of the gastric follicles by arsenic, of the liver and kidneys by phosphorus, of the extensor muscles by lead, etc.; *necrotic changes* are produced in certain organs, as in the maxillary bones by phosphorus, in the teeth by mercury, and in the extremities by ergot: *cataract* follows ergot poisoning, *amaurosis* is caused by quinin; and many poisons cause as secondary effects great sensitiveness to external influences of certain organs, which persists for months or years.

OF THE EVIDENCE IN CASES OF MURDER BY POISON.[1]

The proof in a trial for murder by poison is divisible into three parts:

1. The deceased died from the effects of a certain poison.

2. Said poison was administered to the deceased by the defendant.

3. Said administration was with a deliberate and premeditated design on the part of the defendant to cause the death of the deceased.

The evidence upon which a crime of this character is established is of necessity circumstantial. It is within the limits of possibility that a person might, unobserved by the murderer, be an eye-witness to the administration, knowing the nature of the substance administered. But in such an event, unless the witness, while knowing the nature of the substance administered, was ignorant of its capacity to cause death, or was under physical or moral restraint, he would be an accessory to the crime; or its consummation, except the poison were one of those acting with extreme rapidity, would be seriously jeopardized by interference with the administration on the part of the witness or with the effects of the poison on the part of medical assistance summoned by him. And even in a case so improbable the question as to whether the substance administered was actually the cause of death would still remain to be solved by expert evidence. But the intentional killing by poison is a crime secret in its essence, and while in many cases there have been witnesses to the administration of the substance containing the poison, and in many others an innocent party or the victim himself has been the agent of administration, the incorporation of the poison with the vehicle has been an act previously performed in secret by the real criminal—an act which can be proved only by circumstantial evidence.

With the second and third parts of the above division the toxicological expert has to do only to the extent of furnishing evidence with regard to individual facts, which, along with

[1] For an excellent article on the legal aspect of this subject see Wigmore: "Med. Leg. Journ.," New York, 1888, vi., 392–413.

those developed by the moral evidence, are to be weighed by the jury in determining these points.

Questions for the toxicologist bearing upon administration or intent arise in every trial for murder by poison, and always have an important, sometimes a crucial, influence in determining guilt or innocence. In some instances the distinguishing facts are easily observable and become evident in the course of the analysis, as when Paris green in substance, or arsenic and copper in the proportion to constitute that pigment, is found in the cadaver. Under these circumstances proof of the possession of Paris green by the deceased and of white arsenic by the accused would have very different significance from that of evidence tracing Paris green into the hands of the defendant. At other times the solution of such collateral questions may involve investigations independent of the main analysis and call for all the resources of the expert. In a case, for instance, in which it was claimed by the defendant that he had administered five grains of quinin and a sixth of a grain of morphin to the deceased, the absence of quinin became quite as important an element as the presence of morphin, and its importance depended upon the relative delicacy of the tests for the two alkaloids and the influences of changes produced in them in the body during life and after death.

It is chiefly in the endeavor to obtain evidence upon intricate and novel questions that the expert stands in danger of travelling too far into the domain of theory, into which he is usually led as far as he can be made to go by the counsel who employs him. The introduction of scientific evidence of too finely spun a character usually has the opposite effect to that intended; the expert assumes a position in his direct evidence which he is unable to maintain in its integrity in the cross, and, by basing his inferences upon data the correctness or sufficiency of which is open to question, invites the submission of opinions at variance with his own by the opposing party, with the ultimate result of causing the jury to disregard entirely a mass of conflicting evidence upon points which at best they can understand but imperfectly.

The question whether the deceased came to his death from the action of a certain poison depends for its solution entirely upon expert evidence, to obtain which in the most complete

and satisfactory form possible requires the united services of the clinician, the pathologist, and the chemist. The evidence of the cause of death is obtained from:

1. The symptoms manifested during life.

2. The gross and microscopic appearances observed in the cadaver.

3. The existence of the poison in the body, the vomited matters, the excreta, or the remains of articles of food, medicine, etc.

EVIDENCE OF POISONING FROM THE LIVING BODY.

From the History of the Attack.

In the great majority of criminal and suicidal poisonings the dose administered or taken is large, and consequently the **attack is sudden**, whether the victim be in a condition of health or of illness, and with most poisons progresses rapidly. But in several forms of disease and of internal injuries which prove rapidly fatal the attack is equally sudden,[1] as in cerebral hemorrhage, organic disease of the heart, the rupture of internal organs, in certain forms of Bright's disease, etc., most of which, however, are attended with well-marked pathological changes, easily recognizable at the autopsy. On the other hand, if the poison be administered in small and repeated doses, the onset is more gradual, and the clinical history more closely resembles that of some natural disease. Thus in the Havre poisonings by arsenic[2] the attacks were so insidious that the investigation as to their cause originated in a suit brought by the tenant against his landlord, which led to an examination of the house in which the poisonings occurred, and the lease of which was the subject of litigation, as to its alleged unsanitary condition.

The **relation of time** between the taking of a meal, or of medicine or drink, and the manifestation of symptoms is always one of great forensic importance, and frequently aids greatly either to indicate the guilty party or to prove the inno-

[1] Schuchardt, in Maschka's "Handb. d. ger. Med.," ii., 34–42, reports several cases of sudden death from "natural" causes in which there was suspicion of poisoning.

[2] Affaire Pastré-Beaussier, Brouardel and Pouchet: Ann. d'hyg., 1889, 3 s., xxii., 137, 356, 460.

cence of one wrongly suspected. Poisons usually (although by no means always) act within an hour after their administration; and for every poison the period of time which elapses between the administration and the appearance of its effects varies, according to circumstances, within comparatively narrow limits. If, for instance, a person be attacked with symptoms of arsenical poisoning about half an hour after having eaten a meal, the probability that the poison was contained in some article of food or drink then taken is worthy of serious consideration; and if on analysis one of these is found to contain arsenic, we have then the demonstrated combination of an effect and a cause capable of producing such effect.

But if, on the other hand, a person be accused of administering poison to another in a certain article of food, drink, or medicine which is found to contain the poison, the accusation is to be looked upon with grave suspicion if a day or more have elapsed between the time when the administration was alleged to have occurred and the manifestation of its effects.

The nature of the symptoms is to be considered in this connection, whether they are those of a poison whose action is always rapid, or those of one whose effects may be delayed for several hours.

In poisonings by repeated doses, also, the connection of opportunity of administration and the occurrence of symptoms is of importance. Thus in the Havre poisonings mentioned above the symptoms abated in some of the victims when they went out of the reach of the accused, and again made their appearance after their return; and in a case reported by Kornfeld,[1] in which a woman was convicted of the murder of her husband by arsenic, the deceased, who worked away from home, suffered an attack only when he visited his home, or his wife visited him.

In multiple poisonings, where poison is mixed with an article of food of which several persons partake, all usually suffer from its effects in varying degrees of intensity according to the amount eaten by each, and his or her condition of age, etc. If several persons eating of a particular dish or drinking of a beverage manifest the same symptoms of sudden illness, there is great probability that the cause of the disorder is a poison either mixed with or generated in the food or drink taken.

[1] Friedreich's Bl. f. ger. Med., 1885, xxxvi., 149-160.

But, while it seldom occurs that a number of persons are simultaneously attacked with disease suddenly, such instances have been known, and Taylor[1] has recorded several cases.

It is not to be inferred, on the other hand, that because symptoms of poisoning are observed only in some of those partaking of a particular article of food or drink and not in others who have taken as much or more, the symptoms are due to disease and not to poison. The poison may be one for which those who escape from the effects have established a tolerance by habit;[2] or the toxic agent may have been incorporated as a solid in such manner as to accumulate in certain portions of the dish; or the administration may have been intentionally limited to one or more of the persons apparently partaking equally of the same food.[3]

From the Symptoms.

Can a case of poisoning be diagnosed from the symptoms alone? The medical witness should not answer this question without qualification. If among the symptoms we include the detection of the poison in the excreta, as the existence of sugar in the urine is included among the symptoms of diabetes, then the question is answerable in the affirmative in whatever sense the word "diagnosis" may be used. But if the poison be one which is not detectable in the excreta with certainty, the question must be differently answered according to the value given to the word "diagnosis." In attending a case of poisoning or of disease the physician, partly from positive indications exhibited by the patient, and partly by exclusion, reaches a "diagnosis" upon the accuracy of which sufficient reliance may be placed to warrant the adoption of the treatment appropriate to the form of poisoning or the disease indicated. Such a "working diagnosis," made by an experienced clinician, is correct in the great majority of cases, but the symptomatology of poisoning by the commoner poisons so closely resembles that of some disease produced by so-called

[1] "Poisons," 3d Am. ed., 87–91.

[2] See p. 82.

[3] Instances of the cunning with which one victim may be singled out from among many are to be found in Taylor, "Poisons," 3d Am. ed., p. 88. Maschka, Friedreich's Bl. f. ger. Med., 1855, vi., Heft 4, 66–69. Wormley, "Micro-Chem. Pois.," 2d ed., 40.

natural causes that a mistaken diagnosis is a possibility, even with the most careful and expert physician. While the symptoms, excluding chemical examination of the excreta, do not afford positive proof of poisoning, they frequently indicate the strong probability of its existence, and the certainty that the patient is suffering either from the effects of the particular poison or poisons to which they point or from one of very few diseases presenting a closely similar clinical picture.

Although the clinical history is therefore not to be relied upon alone to prove with absolute certainty that a poisoning has occurred, it is of the first importance, in conjunction with the results of the autopsy and of the chemical examination, to establish that fact to a demonstration, and consequently, whenever it is practicable, the manifestations during the life of the patient should be observed with the utmost minuteness and accuracy. In a case of morphin poisoning it may be claimed that death was due to cerebral apoplexy, or to uræmic coma consequent upon Bright's disease. If the patient has during life manifested the symptoms which may be due to one of the three causes, and if, after death, the anatomical lesions caused by apoplexy and Bright's disease are found to be absent, and those due to morphin poisoning (and to other causes also) are found to be present, and if morphin be found on analysis of the cadaver or of the vomited matters, the agency of morphin as the cause of death cannot be questioned as between the three causes mentioned. Further, symptoms afford one of the surest means of answering the question, "Was the poison introduced during life?" There is no analytical method by which undertaker's arsenic can now be differentiated from that taken during life, but it is clear that a history of the symptoms of arsenical poisoning during life is strong evidence of ante-mortem administration.

The symptoms of poisoning present no general characters which permit of a distinction between poisonings and disease—indeed many diseases are endogenous poisonings. The question presented to the clinician in the treatment of poisoning and in the medico-legal proof of its occurrence is one of differential diagnosis, which, of course, varies with the poisons producing different effects. The points of distinction to be observed in a case of strychnin poisoning are not the same as those which

customarily present themselves when arsenic has been taken. General considerations upon this important branch of the proof of poisoning are idle. The effects of each poison must be considered in comparison with the symptoms of those diseases whose symptomatology is similar to that of the form of poisoning in question, and therefore we defer consideration of this branch of the subject to the division on special toxicology.[1]

From the Detection of the Poison in Excreta, etc.

The most direct proof that poison has actually been administered or taken during life is its detection in the matters vomited by the victim, or in the urine or fæces passed during his lifetime.[2] In non-fatal cases these, along with suspected articles of food or medicines, are the only materials for chemical analysis.[3]

The **vomited matters** frequently have easily determinable qualities which are of great diagnostic value—as, for instance, their luminous appearance in phosphorus poisoning, their alkaline reaction when the alkalies or potassium cyanid have been taken, their intense acidity in corrosion by the mineral acids, their characteristic odor in poisoning by phenol, prussic acid, or chloroform, etc. If the poison be heavy and have been taken in the solid form and is not readily soluble, it may be found deposited in the bottom of the vessel containing the vomit or washings of the stomach, as has been frequently observed in arsenical poisoning. As a rule, the quantity of poison expelled will diminish with each succeeding vomit, yet Ryan[4] has reported an instance in which the stomach pump was used after three acts of vomiting, and in which the quantity of arsenic in the material removed by the pump was greater than that in the matters last vomited.

It is quite possible that the poison may be completely removed by vomiting and elimination and yet death result from its action. In that event, clearly, the poison may be detectable in the urine and vomit, but not in the cadaver. Such a case is

[1] See also Tardieu, "Empoisonnements," 2ème ed., 25–55. Taylor, "Poisons," 3d Am. ed., 94–117. Kobert, "Intoxikationen," 37–44.

[2] It goes without saying that there must be no question as to the identity of the matters examined, nor any possibility that the poison may have been subsequently added.

[3] For a few poisons the blood may be examined with the micro-spectroscope.

[4] Lancet, 1851, i., 410.

reported by Taylor[1] in which a man died seventeen hours after taking about one ounce of arsenic.

It is, unfortunately, exceptional in homicidal cases that the vomit is preserved for analysis. This defect in the medical conduct of the case may sometimes be supplied by an examination of the vessel into which the vomit was received, or of the floor, ground or fabrics, upon which it may have been spilled. The surface of a board floor may be removed by planing, and portions of the carpet or other textile fabrics may be cut out. Samples should be taken for analysis not only from the places where the vomit was deposited, but also from other portions for comparison.

The detection of the poison in the **urine** or **fæces** passed during the patient's lifetime removes all doubt concerning the cause of the symptoms observed, provided the analysis is properly made and the purity of the chemicals used is placed beyond a doubt. The negative result of such an examination does not by any means prove that the symptoms observed are not due to poison. Even arsenic, whose detection by an experienced analyst is certain, has been found to be absent from the urine in cases of undoubted poisoning by the white oxid, either because the elimination has been completed, or because of an apparent intermission in the elimination; and instances of failure to detect morphin in the urine of patients who had unquestionably taken large doses, and even when its presence in the vomited matters was demonstrated, are numerous.[2]

The examination of **vessels** and of articles of **food** and of **medicine** frequently affords evidence of the manner of the administration, and may serve to connect a particular person with the crime. It must be remembered, however, that the detection of poison in an article of food alleged to have been taken is not of itself evidence that the poison has been actually taken. Several cases have occurred in which the symptoms of poisoning have been feigned and poisoned food has been submitted for examination to substantiate a false accusation.

The number and variety of articles which, in a case of alleged homicide, may be submitted for analysis for the purpose of tracing administration probably reached the maximum in the

[1] Guy's Hosp. Rep., 1837, ii., 77.

[2] See Elimination, p. 87, and also Elimination under Arsenic, Morphin, Strychnin, etc.

Maybrick case, in which one hundred and seventy-four articles, other than portions of the body and excreta, were delivered to the analyst.

DUTIES OF THE PHYSICIAN IN A CASE OF SUSPECTED CRIMINAL POISONING.

The nature of the physician's relations with his patients and the course of his daily practice are such that the idea of poisoning does not suggest itself unless forced upon his recognition by the circumstances of the case, and even then is reluctantly entertained. That he should be frequently deceived in attributing the effects of poison to other causes is natural, and, be it said, it is preferable that he should be sometimes misled by the craft of the poisoner than that he should go into every household in the double capacity of physician and detective. Yet, as it is the physician's function to ascertain not only the present condition of his patient but also the cause of his ailment, he should, when called to attend a case of poisoning, recognize or at least suspect its true character. If the case be not plainly one of accident or suicide, upon him may then devolve a responsibility more serious than that attending the most desperate case of disease or of injury—a responsibility not of his own seeking and entirely beyond the ordinary functions of his profession. He may be suddenly placed between an intending murderer and his victim, with the possible intent on the part of the former to render him an unconscious accessory to his crime; and should the deed be consummated, upon him the fulfilment or the frustration of the ends of justice will largely depend. That a responsibility so onerous, with the subsequent annoyance and interference with regular practice, should be shirked and evaded by many is to be expected, and we believe that many a poisoner has escaped punishment through a disinclination on the part of the attending physician to assume the responsibilities of the position into which he has been thrust by his attendance upon the victim, and to incur the risks and discomforts which an accusation would entail. Two cases in illustration: A physician of ability greater than the average and of unquestionable integrity, being called in attendance upon a woman, was so thoroughly convinced that she was being poisoned with arsenic by her hus-

band that he accused him of the attempt and forbade him visiting the house in which his wife lay ill until she should be either recovered or dead. Yet upon her death he signed a certificate attributing the death to "malaria and gastritis," and permitted the only undertaker in the village to fill the cadaver with arsenic before tardily communicating his suspicions to the prosecuting officer. Had he had a proper sense of his responsibility, either a murderer would not have escaped the penalty of his crime through the impossibility of a distinction between undertaker's and ante-mortem arsenic, or an innocent man would not have been subjected to a year's imprisonment and the chances of two trials for his life. In another case a physician who had attended the husband and one child during their last illness, refused to attend the second child of a woman who subsequently confessed to having poisoned all three and was convicted of the murder of the last; his refusal being clearly based upon a suspicion of foul play in the two previous deaths, which he had certified as being due to "inflammation of the bowels" and "congestion of the bowels." Yet not a word did he breathe of his suspicions, except to the woman herself, until after her confession.[1]

The duties of the practitioner in this connection we apprehend to be:

1st. To rescue the patient from the effects of the poison or corrosive already taken.

To accomplish this a "working diagnosis" of poisoning by x must be arrived at. To reach this the symptoms will have been studied to the exclusion of those diseases having similar symptomatology, and the history of the inception and cause of the disorder previous to the physician's visit will have been in-

[1] It is but fair to the medical profession to state that an equal disinclination to assume the responsibilities of a prosecution in cases in which a *prima-facie* case seems to be clearly established is sometimes exhibited by those upon whom this duty devolves under the law. Upon one occasion the author, while in attendance upon a grand-jury hearing in one of the northern counties of this State, had the novel experience of being waited upon at the hotel table by the woman in the case, who was out on $250 bail. Her husband had died after a short illness, marked by the symptoms of arsenical poisoning. Arsenic was present in the unembalmed body, and the immoral circumstances of the case indicated the existence of a strong motive to the commission of the crime by the wife. Nevertheless, neither district attorney nor grand jury found sufficient cause for proceeding further, and the woman emigrated to a Western State with her paramour.

quired into. The attendant and possibly the patient will have been questioned, and from the nature and manner of the answers received the physician may frequently obtain indications which will govern his future course. Should there be the slightest doubt in the mind of the medical attendant that the case is not one of accidental poisoning, he should take for examination the urine and vomit and any remains of medicine or food taken by the patient. Portions of these he should examine, with all proper precautions, by the simpler tests for the poison which he believes to have been taken—but by no means should he use all of the samples taken for this examination, the purpose of which is solely the verification of his diagnosis for the government of his treatment. The chemical knowledge and manipulative skill in analytical methods of practitioners of medicine are rarely if ever adequate to the test of cross examination upon a criminal trial.

2d. To prevent any further administration. From the information obtained in fixing his diagnosis and from his observation of the surrounding circumstances the physician may in most cases determine what means to adopt to accomplish this end. He may, further, with proper discretion, ascertain whether any person visiting or in attendance upon the patient has any reason for enmity; if the attack was traceable to the taking of a particular meal or dose of medicine, by whom was it prepared or administered, and in whose care or keeping was it. He will have to decide from the degree of his conviction that a certain person is the author of the poisoning, from the condition of the patient, and from the nature of the surroundings whether and to what extent he should communicate his suspicions to the patient or to any member of the household. Under no circumstances, however, should he give expression to any suspicion which he may have, nor encourage the expression of such suspicions by others, as a matter of gossip or public rumor. There is always the possibility of his being mistaken, and in that event the injury which may be done to an innocent person is irreparable, as such rumors travel rapidly, and any subsequent retraction will probably not reach all who have heard the suspicion originally expressed. In some cases it may be proper for the medical attendant to insist upon the absence from the sick-room or from the house of a certain person to whom circumstances

point with sufficient directness as the author of the poisoning. In other cases, particularly those in which he finds no one in the household to whom he deems it wise to communicate his suspicions, the physician may demand either that the patient be removed to a hospital, or that a trained nurse of his own selection shall be called in.

3d. In case of death, to avoid interfering with a proper investigation by giving a certificate of death or permitting the undertaker to embalm the body.

As no dead body may be buried without a certificate setting forth the cause of death from the attending physician, or a permit from the coroner or medical examiner, the physician who still entertains the belief that the deceased may have died from the effects of poison and signs a death certificate attributing the death to "gastritis" or "apoplexy" or some other disease becomes morally if not legally an accessory after the fact. According to the degree of his doubt he has one of two courses to pursue—either to insist upon a post-mortem by which he may determine whether any sufficient natural cause of death exists, or to turn the case over to the proper authorities for investigation. Should he adopt the former course, which has the advantage that if the death was really due to disease the expression of an unfounded suspicion of poisoning has been avoided, the physician must consider his qualifications for personally making such an examination. As a rule the practitioner is not fitted to properly conduct a medico-legal autopsy, and this may become one, and it will be much preferable for the proper conduct of the inquiry, if one becomes necessary, and for the future comfort of the physician himself, that he should call in the assistance of one skilled and experienced in such work. If the services of the coroner's physician or medical examiner can be obtained unofficially or semi-officially so much the better, as, not only should he be expert in such matters, but if the case be one of poisoning he may then and there take charge of it officially.

As legislatures in this country have been very respectful of the liberties of the "funeral directors," they are entirely unrestricted, and unless expressly forbidden so to do by some one in authority, it is their custom to inject into every cadaver a more or less extensive collection of poisons as soon as possible after death, sometimes within two hours. Poison so introduced can-

not at present be distinguished from that existing in the body before death, and consequently it is the plain duty of the physician, when he believes that death has been due to poison, to expressly state to the relatives and to any undertaker that he may have reason to believe has or will have charge of the funeral that the body should not be embalmed, and if necessary to request of the coroner that he forbid any treatment of the body before the autopsy.

While in attendance upon a case of poisoning the physician should bear in mind that every detail of symptoms, treatment, etc., may become of importance upon a subsequent trial, and that he may be called upon months or even years afterward to recite his observations. He should therefore make full and accurate notes of such cases, from which he may subsequently refresh his memory.

EVIDENCE OF POISONING FROM THE DEAD BODY.

The examination of the dead body of a person supposed to have been poisoned is divided into three parts: 1st. **The autopsy proper,** at which the appearance of the body and its surroundings, and the existence or non-existence of any departure from the natural conformation of tissues or organs detectable by the unaided eye are observed. 2d. **The pathological examination,** by which the presence or absence of minute changes in the tissues, caused by disease or by the action of poisons, is determined by the use of methods involving the use of the microscope. 3d. **The analysis,** whose object it is to separate and demonstrate the presence of poison in the cadaver, if such exist.

Evidence from Position and Surroundings.

Before proceeding to the autopsy proper the coroner or medical examiner should, particularly when the person has been found dead, make note of the position of the body and its surroundings. Observations then made may frequently be of great service in differentiating between poisoning and some other cause of death, or in determining whether the case is one of suicide, accident, or murder.

Note should be made of the following points: 1st. The date

and place of the examination. 2d. The names of the persons present. 3d. The position of the body. 4th. Whether clothed or not, and the condition of the dress. 5th. Had the deceased vomited, and if so the relation of stains of vomit to the cadaver. 6th. Stains of vomit upon the clothing or other articles, with the marks placed by the examiner upon such articles for identification, and the name of the person into whose custody they are given. 7th. Similarly as to stains of urine and of fæces if present. 8th. Description of any cup, bottle, or other vessel, paper or weapon, found near the body, and its position with regard to the body; with notes for identification as in 7th. 9th. Had stains of vomit, etc., been made while the body was in the position in which it was found, or had the body been subsequently moved, and any appearance which would indicate whether such movement occurred before or after death? 10th. The temperature of the room. 11th. In cases of exhumation also note the position of the grave, the nature of the soil, the condition of the casket, and transcribe any inscription upon the coffin plate.

If the autopsy be made upon an exhumed body the possibility of fortuitous impregnation with poison should be borne in mind. The condition of preservation should be accurately noted, as well as any evidence of the cadaver being or having been moistened by ground water. Samples of the earth from above, below, and each side of the coffin should be taken, and particularly samples of any colored article, such as artificial flowers, and of any portion of the grave clothes or casket trimmings which is colored.

THE AUTOPSY.

Direct and positive evidence of death from poisoning is not to be expected from post-mortem appearances alone. Although intense inflammation, softening, and corrosion of the œsophagus and stomach, accompanied by a marked alkalinity of the contents of the latter, may indicate that corrosion by a mineral alkali has occurred, there is no true poison whose action produces characteristic lesions, and many may cause death without leaving any evidence of their effects in structural change of organs or tissues.

Nevertheless, very important evidence in two directions is obtainable by a post-mortem examination: either as corroborative of other proof of poisoning directly, or bearing upon the possibility of death having been due to other causes.

Certain appearances or conditions observable at the autopsy may by their existence serve to confirm the theory of poisoning by a certain agent, or by their absence or the presence of their opposites may even exclude the possibility of a specific poison from consideration as a possible cause of death. Thus a congested condition of the cerebral vessels is present after death caused by the opiates, as well as after death by suffocation from any mechanical cause; a dark color of the blood is inconsistent with the theory of death from carbon monoxid or illuminating gas; and an acid reaction of the contents of the stomach, with absence of the odor of peach blossoms, is antagonistic to the supposition that death was caused by potassium cyanid.

The second purpose served by an autopsy and a pathological examination is probably the more important in cases of supposed poisoning. It is the proof of the existence or non-existence of natural causes of death whose symptomatology resembles the effects of a given poison more or less closely, but which are attended with changes in the appearance of organs or in the structure of tissues which are observable with certainty in a properly conducted autopsy, aided by a micro-pathological examination. Thus, in a case of poisoning by morphin, the pathologist may exclude the possibility that death may have been due to cerebral hemorrhage or to disease of the kidneys. The presence of lesions in such cases materially weakens the proof of poisoning, although it must not be forgotten that kidney lesions, for instance, are quite common, and that a person suffering from kidney disease, recognized or unrecognized during life, may be poisoned by morphin. Indeed poisoners have been known to take advantage of an epidemic of Asiatic cholera to remove their victims by the action of arsenic.

Autopsies—by Whom and How Conducted.[1]

The practitioner of medicine, not specially engaged in pathological investigations, should avoid assuming the responsibili-

[1] See also vol. i., pp. 351–382, 300–303.

ties of a medico-legal autopsy. Indeed, even coroners' physicians are more frequently found ignorant than informed of the details which should be regarded in an autopsy after death supposed to be due to poison. Whenever it is possible the post-mortem examination should be conducted by a skilled and experienced pathologist, and the chemist who is to make the analysis should be present.

The autopsy should be made as soon as possible after death, the earlier the better for the success of both pathological and chemical examinations. If made after putrefactive changes have modified the structure of the tissues, microscopic alterations indicative of certain diseased conditions can no longer be observed, and in a still more advanced condition of decay but few observations of modifications in gross appearances can be made, and the autopsy becomes merely the means of securing material which the chemist may examine for mineral poisons. The pathologist should, however, not discourage the making of an autopsy even in a case of supposed death from vegetable or volatile poison merely because a month or two have elapsed since the burial. In the Harris case the body was exhumed fifty-three days after death, and in the Buchanan case forty-three days after, and both bodies were found in a condition of preservation sufficient not only to permit of accurate observation of gross appearances, but also to a certain extent of the minute structure of the organs and tissues.

The autopsy, consisting of the **external** and **internal examinations**, should be made with due regard to directions given in the chapter on medico-legal autopsies;[1] and the notes of the observations made should be written at the time by an assistant at the dictation of the examiner, should be minute and accurate in all respects, and should be verified by the examiner immediately after the completion of the autopsy.

Under no circumstances should any person upon whom suspicion of criminality in the case under examination attaches be permitted to be present at the autopsy,[2] and should any professional man be present in the interests of an accused or sus-

[1] See vol. i., pp. 351–382.

[2] At the autopsy upon the body of Cook, Palmer, who was subsequently executed for his murder, was present and made two attempts to interfere with the proper removal of the stomach for analysis. "Times Report of Trial," p. 21.

pected person, his function should be strictly limited to that of a spectator.

External Examination.

The exterior of the cadaver should first be carefully inspected and note taken of the following points: 1. The sex and apparent age. 2. The identity. If identified, by whom; if unknown, any marks, scars, etc., useful for identification should be noted.[1] 3. The exact time after death when the autopsy is made if known; if not, the apparent time. 4. The degree of rigidity. 5. The condition of preservation. 6. Whether the hair is loosened or colored at the roots. 7. The condition of dilatation of the pupils (in early autopsies). 8. The general color of the surface. 9. Any discolorations of the surface, and their exact location and appearance. 10. Any wounds, particularly incisions or punctures, made for embalming, over the carotid, brachial, femoral, or tibial arteries or in the abdominal walls, or evidences of hypodermic injections. 11. Any particles of foreign matter found between the teeth or in the mouth should be preserved. 12. If an autopsy has been previously made, note the fact, and the condition in which the organs are found.

Internal Examination.

In this place it is only necessary to allude to the precautions which should be taken in the removal of parts for analysis. These should consist of: 1. The stomach, ligated at both extremities and unopened. 2. The intestine, from the duodenum to the lower rectum, also ligated and unopened. 3. The entire liver. 4. Both kidneys (preserved in separate jars). 5. The entire brain. 6. A large piece of muscular tissue from the thigh. 7. Any urine which remains in the bladder, which may be collected before opening the abdomen by passing a catheter into the bladder and receiving its contents, expelled by pressure upon the abdomen, into a phial. 8. A portion of the blood from the heart and great vessels. Before placing the liver, kidneys, and brain in their respective jars as described below they are to be examined as to their gross appearance, and the

[1] See "Identity," vol. i., p. 416 *seq.*

small portions required for pathological examination removed and preserved in the strongest obtainable alcohol, apart from the remainder of the organs destined for chemical analysis. If any peculiar odor be observed on opening the body it is advisable to add to the above: 9. The lungs.

Each of these parts should be placed by itself in a new glass jar, which has been well rinsed out with water, and which is to be closed with a glass cover[1] or stopper, or with a cork, tied over with moistened bladder or parchment paper. A cord or tape is then to be tied about the jar and cover and so fastened by sealing-wax bearing a private imprint, that it is imposible to gain access to the contents without breaking the seals or cutting the cords or tapes. No preservative should be added, unless the parts must be kept for several days before transmission to their destination. Under these undesirable conditions alcohol only may be used, and if used, a sample of at least a liter should be separately submitted for analysis.

In removing and transmitting the parts destined for analysis the examiner should exercise the minutest care to prevent the possibility of error, contamination, or loss. Each organ as removed, and after examination as to its gross appearance and removal of such fragments as may be required for microscopic examination, should be transferred to its jar and immediately sealed by an assistant, under the observation of the examiner, and with his private seal. In making the sections of organs after their detachment they should never be placed upon a table, nor upon any surface of whose cleanliness the examiner is not personally assured, but should be supported either by the hand of the examiner or upon a new and clean dish or plate. Nor should any part intended for analysis be permitted to pass, even for a moment, into the hands of any person other than the examiner or the chemist. After having been properly sealed the jars containing the viscera should remain in the custody of the examiner until they are transmitted by him to the chemist, and, immediately before their transmission, the integrity of the seals should be verified. If no preservative have been used, and if more than forty-eight hours have elapsed since the autopsy, when the jars containing the viscera are sent to a distance it is advisable to release the pressure upon the covers sufficiently to

[1] The so-called "anatomical jars" are the best for the purpose.

allow any accumulated gas to escape, as sometimes the pressure so produced has been sufficient to cause the fracture of the jars when they are subjected to agitation in transit.

The stomach and intestines should not be opened until they reach the chemist's laboratory, and when they are opened he should proceed with the analysis for volatile poisons as expeditiously as possible. It is highly desirable that the pathologist and chemist should both be present when the stomach and intestines are opened, and that the former should then note the appearance of the mucous membrane and take sections of the tissue, samples of the contents and scrapings from the surface for microscopic examination. If such joint work be not possible, the chemist should carefully note the condition of the gastric and intestinal walls, their degree of preservation, whether thinned or thickened, or softened, the color and appearance of the gastric and intestinal mucous membranes in detail, locating any peculiarity in color or texture; and should send to the pathologist sections cut through all the coats of the stomach and intestines, taken particularly at any point where ulceration, thickening, or other unusual appearances are observed.

In exceptional instances the body has been more or less completely eviscerated and the internal parts removed. Thus in the Affaire Gloeckler, in 1846,[1] the internal organs were removed from the cadaver and thrown into a privy, whose contents were subsequently spread upon a field, where the heart, liver, and one kidney were found. In the Shann case, in New Jersey in 1893, all the internal organs of the trunk except the bladder and one kidney were removed.[2] In such instances any

[1] Devergie, "Méd. lég.," 3ème ed., iii., 563.

[2] We are indebted to Dr. J. Stockton Hough, of Trenton, N. J., for an opportunity to consult the stenographer's minutes in this extraordinary case. Mrs. Mattie C. Shann, a widow, was tried for the murder of her son, John F. Shann, at the Mercer County (N. J.) Oyer and Terminer, August, 1893, and acquitted, the motive alleged by the State having been to obtain the payment of insurances upon the life of the deceased, which had been written some six months before his death. The deceased, about twenty years of age, and residing with his mother at Princeton, was taken sick about February 22d, 1893, took to his bed March 6th, and died early on the morning of April 18th, having been attended during his illness by the family physician, Dr. Bergen. The physician diagnosed the case as one of gastritis, which he assigned as the cause of death in his certificate. The symptoms were those of gastritis, plus a very bad sore throat and an ulcer in the in-

remaining contents of the thorax, abdomen, or pelvis which may be found should be carefully preserved for analysis, as well as the brain and large portions of muscular tissue. Diligent search should be made for the missing organs, and the appearance of what remains of the cadaver should be minutely noted, as to how the evisceration was performed, whether by a skilled hand or otherwise, and whether the body had been embalmed previous to evisceration.

Post-Mortem Appearances of Toxicological Significance.

In the Blood.—Many poisons act chemically in or upon the blood, and produce changes in its appearance observable at

side of the mouth, which almost perforated the cheek before death. In the prescriptions written for the patient during his illness calomel was the only mercurial given, of which the total quantity used in the Shann family during this period was $2\frac{6}{10}$ grains. A druggist, during John's illness, refilled a prescription for antiseptic tablets containing corrosive sublimate three or four times for the defendant or her seventeen-year-old daughter, Mabel. There was evidence that John had taken other medicine than that ordered by Dr. Bergen, the nature of which did not appear, and, it was claimed by the defence, had contracted syphilis during the preceding Christmas holidays. The body was laid on a cooling board, in a room in which the defendant and her daughter slept, by an undertaker, a cousin of the deceased, the following morning. Cloths dipped in "Utopia" embalming liquid were laid upon the stomach, hands, and face and about a t aspoonful was injected into the sore upon the face, but the body was not embalmed, the defendant desiring that it should not be done. The body was last seen in an unmutilated state by the undertaker at about 10 A.M. on the 19th, and at about the same time by the local agent of the life insurance company. From that time until about 3 A.M. on the 20th, when the body had been mutilated, it was not seen, so far as the evidence shows, except by the defendant and her two daughters. One of these, Mamie Kelly, alleges that she saw the body intact at two, five, and nine o'clock on the 19th, at five o'clock in the presence of another person who, however, flatly contradicts her in his evidence. At about 5 P.M. on the 18th an agent of the insurance company asked to see the body and was refused permission by the defendant; and at about eleven o'clock on the evening of April 19th the defendant was informed that the insurance company's physician would make an autopsy on the following morning. At three o'clock on the morning of the same night the defendant awakened one of her male lodgers, to whom she related the particulars given below, and who at her request looked at the body and found blood stains on the sheet upon which it lay, and on the canopy which covered it. At about seven to eight o'clock the same morning (April 20th) Dr. Bergen examined the body sufficiently to see that the abdomen had been cut open and its contents removed. The body was transferred to Trenton by the county physician that evening and an examination was made the following day, April 21st, when it was found that all the contents of the abdominal and thoracic cavities had been removed, with the exception of one

the autopsy. The blood is found to be FROTHY from the presence of bubbles of gas, if the autopsy be made soon after death from hydrogen peroxid, which may cause death by the mechanical action of the gaseous oxygen which it liberates on being decomposed in the blood. A similar appearance has been observed in animals poisoned by diazobenzene salts, which generate free nitrogen when decomposed in the blood, and also in some cases of death from chloroform and ether in the human subject.

The COLOR of the blood is dark after death from the action of poisons which interfere with the proper oxygenation of its coloring matter, as morphin, strychnin, or carbon dioxid, or which act chemically upon it, with formation of methæmo-

kidney, the bladder, which had been slit, about three inches of the rectum, and parts of the œsophagus and trachea. Some of the brain, the kidney, and some bloody matter from the abdomen were put into one jar together, and about two and a half inches of the rectum in another. These were analyzed by Professor Wormley, who found about $\frac{1}{4}$ grain of mercury in the contents of the first jar and about $\frac{1}{50}$ grain in the piece of rectum. On May 13th the body was exhumed and the remainder of the brain, the bladder, the fractions of œsophagus and of trachea, the larynx, both tonsils, both parotid glands, an ulcer in the cheek, the calves of both legs, and a part of the muscles of the right thigh were removed, placed in six jars and submitted to Professor Cornwell, who found them to contain mercury. No trace was found of the viscera removed from the body. The officers of the insurance company denied any knowledge of their removal. The defendant related that during the night of the evisceration "three persons dressed in storm coats, slouch hats, and whose general appearance (but not with much particularity) she described, came to her front door, rang the bell; she arose, went down, opened the door, admitted these three men. That they insisted upon seeing the body of her son—she refused; they continued to insist, and in answer to some question, she told them where the body lay. Two of them went upstairs and entered the room where the body lay. They came down after some length of time, she being meanwhile detained by a third person at the foot of the stairs by violence, who threw her twice against the wall when she attempted to go upstairs; that then they departed and left. Whether they had anything with them or not she was unable to say. During this time the lamp was on the table in the hall, which she had brought down, burning. She says that her condition of mind under the circumstances was such that she thought these persons came from the insurance company to make some examination or otherwise, and that she was so affected by it that she was unable to make any outcry, or do anything so as to arouse any of the boarders in the house, or her daughter, or Aunt Eliza who slept in a room about eight feet from where she stood, detained by one of these three men" [charge of Judge Abbott]. There was some collateral evidence in corroboration of minor points of this testimony, but no direct evidence in support of it by any of the six boarders in the house at the time; and the daughter, Mabel, slept, without awakening, within a few feet of the cadaver during the entire period.

globin, or hæmatin, or of sulfhæmoglobin, as potassium chlorate, and hydrogen sulfid. It is bright red after poisoning by carbon monoxid. (See Physical Examination, p. 124).

Unusual COAGULATION of the blood is observed after death from castor oil beans, jequirity, or the mineral alkalies; while the blood is fluid when death has been caused by strychnin, morphin, chloroform, etc.

Certain poisons cause a more or less complete destruction and solution of the red blood corpuscles, sometimes so extensive that the blood in thin layers appears perfectly transparent and uniform in consistency, while normal blood obstructs the passage of light, even when in very thin strata. This solution of corpuscles is observed after poisoning by hydrogen arsenid and by certain fungi.

In the Brain and Its Coverings.—Congestion of the cerebral and meningeal blood-vessels and effusions of serum into the ventricles and beneath the arachnoid are usually found after death caused by acute poisoning by alcohol, morphin, cocain, nicotin, coniin, strychnin, etc.

In the Thoracic Organs.—Effusions into the pericardium are found after death from many poisons. An unusually contracted condition of the left heart is sometimes found after death from digitalin and veratrin; and endocardial ecchymoses are frequently observed after death from arsenic. The heart muscle is the seat of fatty degeneration in death from arsenic, antimony, and phosphorus.

The lungs are congested and sometimes œdematous in poisoning by morphin, nicotin, pilocarpin, and certain fungi.

In the Abdominal Viscera.—It is here chiefly that post-mortem appearances of toxicological interest are to be found. On opening the abdomen, the characteristic odor of a given poison may sometimes be observed; the garlic odor of phosphorus, the peach-blossom odor of hydrocyanic acid, the turpentine-like odor of savin, the characteristic odors of phenol, camphor, alcohol, etc. The odor is also to be noted on opening the thoracic and cranial cavities.

The PERITONEAL SURFACE of the abdominal organs, particularly the stomach and adjacent parts, is found reddened, inflamed, ecchymotic, and the blood-vessels injected after death from the action of powerful irritants and corrosives. The

peritoneal cavity may contain serous, purulent, or bloody exudations, or even the contents of the stomach, if the walls of that organ have been powerfully attacked or perforated by a corrosive.

The appearance of the external surface of the STOMACH and its degree of distention are to be noted, the organ being held by its two extremities before a good light. If its size departs from the normal it is to be measured and its dimensions noted. The contents are then removed by holding the stomach by its two extremities over a clean, weighed beaker, and making an incision with a scissors at the most dependent part. The beaker and contents are weighed and the weight of the latter determined by subtraction of the weight of the beaker. A mark is placed on the outside of the beaker at the level of the contents that their volume may be subsequently determined. The color, consistency, and odor of the contents are noted and portions of the solid part removed for microscopic examination (see below). The reaction is determined. An alkaline reaction may be due to caustic alkalies, alkaline earths, or potassium cyanid, or to the administration of alkaline antidotes. An intensely acid reaction may be due to acids or to acid salts. The contents are observed in the dark; they are luminous in the presence of phosphorus provided alcohol or oil of turpentine be not present.

To examine the mucous surface of the stomach it is divided from end to end along the greater curvature and spread out, with the mucous surface upward, upon a new and clean plate or dish. The characters of adherent particles are first noted carefully after examination with the unaided eye, a hand magnifier of about two diameters and the microscope. In this manner valuable information is sometimes rapidly obtained. Arsenic trioxid, if taken in substance, is frequently found in the shape of octahedral crystals, accompanied or not by white amorphous particles, and sometimes tinged yellow by formation of the trisulfid during putrefaction. Crystals of white arsenic accompanied by black particles of carbon indicate that the poison was taken as "rough on rats" or some similar combination of arsenic and soot, in substance. Green, metallic particles may be found adherent to the walls of the stomach in poisoning by Paris green, and green scales when death has been caused by cantharides. Small fragments of wood with sulfur attached

have been found when match heads have been taken, and fragments of various seeds and other vegetable tissues after nux vomica, stramonium, savin, aconite, tobacco, and other poisonous vegetable drugs have been taken in substance. Sometimes also particles of a peculiar food substance, or of one peculiarly modified, as by the presence of mould, may serve to identify the vehicle in which the poison was administered.

After any particles adherent to the walls of the stomach have been examined and collected, the mucous surface is to be washed with a small quantity of distilled water (which should be preserved for examination along with the contents). The appearance of the mucous membrane is to be carefully noted, and any departures from the normal located. Excoriations, ulcerations, erosions, and even perforations are found after death from the immediate effects of corrosives, or marked thickening, ulceration, absence of epithelium, and contraction of one or both orifices after death from their secondary effects. Irritant poisons, whether mineral, animal, or vegetable: arsenic, antimony, potassium cyanid, croton oil, savin, many ethereal oils, cantharides, etc., produce more or less intense inflammation, marked by redness, sometimes diffused over the entire mucous surface and darker in the depressions between the rugæ, sometimes of a dotted or petechial character; and by more or less injection of the blood-vessels.

With corrosives and some poisons similar changes are observed in the œsophagus, pharynx, and mouth. With volatile corrosives and poisons, such as hydrochloric acid, ammonia, chlorin, etc., the larynx and trachea are the seat of intense inflammation; and may be eroded if a liquid corrosive has been aspirated.

The INTESTINES are to be dissected from the mesentery, divided at the ileo-cæcal valve, and the small and large intestines separately examined in the same manner as the stomach, the contents being removed either by "stripping" each portion between the index and middle finger from above downwards, or preferably, by the handle of the scalpel after the gut has been opened by the enterotome. Irritant poisons produce more or less extensive intestinal inflammation, particularly in the upper part of the small intestine and in the large intestine. Or the upper part of the small intestine may be peculiarly colored:

green by Paris green or the salts of copper; brown by iodin, potassium dichromate, or phosphorus; yellow by picric acid, lead chromate, or nitric acid; black by silver.

The LIVER, HEART, and KIDNEYS are the seat of fatty degeneration in poisoning by phosphorus, arsenic, antimony, iodin, and certain fungi.

The KIDNEYS are the seat of active hyperæmia in poisoning by cantharides, turpentine, iodin, etc. They crackle on section and contain crystalline deposits of calcium oxalate after death from oxalic acid or the oxalates; and they contain reddish-brown casts, visible to the unaided eye, after death from mineral acids or alkalies, potassium chlorate, etc.

In poisoning by the compound ammonias (hydroxylamin, metatoluidin, etc.), the kidneys and SPLEEN are pigmented, and pigmentary, muriform masses are found in the latter organ.[1]

EVIDENCE FROM THE PRESENCE OF POISON IN THE CADAVER.

If post-mortem introduction of poison be excluded, the most satisfactory evidence of poisoning is from the demonstration of the presence of the toxic agent in the cadaver.

This may be attained by one of three methods or a combination of them: The *physical* examination, by which the poison is identified, when present in substance, by its appearance, crystalline form, color, etc., or by spectroscopic or other physical examination; the *chemical* analysis, by which the dissolved or absorbed poison is separated in a form which permits of its identification by its physical characters and chemical reactions; and the *physiological* examination, by which the nature of the poison is determined by observation of its effects upon animals, by "life tests." These three methods are used jointly, and together constitute a **toxicological analysis.**

CONDITIONS OF A TOXICOLOGICAL ANALYSIS.

The expert selected to perform the analysis must, of necessity, be a chemist of experience.[2] He should also be a graduate

[1] Pilliet: Compt. rend. soc. biol., Paris, 1894, 10 S., i., 331.

[2] As recently as 1861 (Phar. Jour. and Trans., 1860-61, n. s., ii.,

in medicine and familiar with the methods of physiological experimentation. Indeed, the peculiar experience and training requisite to enable one properly to conduct an intricate toxicological analysis are such that only most exceptionally can persons truly competent be found, except among those who in the larger institutions of medical and pharmaceutical instruction have devoted themselves specially to this subject.

In some countries, as in France, the analysis is entrusted to two experts, who conduct the operations jointly. In a circular of February 6th, 1867, the French Minister of Justice deprecates the multiplication of experts in the following language: "Under the pretext of reaching greater certitude and of carrying to the mind more entire conviction, magistrates show themselves much too ready to call in at once and without distinction two and even three experts. This is a regrettable tendency against which we must contend. In fact, the expert investigation does not bind either the magistrates or the defence, and the number (of experts) which always delays the solution of questions involved is no guaranty of the value of the work.

"It seems evident to me, in fact, that expert investigations draw their force much less from the number of specialists consulted than from the well-known merit, knowledge, and integrity of the expert. It frequently happens that a report offers all the more security when it is signed by a single expert, because the responsibility falls upon him alone, because he has verified everything himself, and because no opinion is expressed as a sort of compromise.

"It is therefore, I repeat, by knowing how to choose the expert to whom a mission is confided, and not in multiplying their number, that one assures all the importance which it should have, to this aid to the information which is necessary in many cases. . . .

"It is hardly necessary that I should add that all the rules which I have laid down apply to chemical analyses."[1]

In these views we heartily concur, as we have found that

614) two medical men in England analyzed (?) portions of the body of a child and swore positively to the presence of arsenic before the coroner. The jury demanded a further examination by a chemist, which was made and demonstrated the absence of arsenic.

[2] Dubrac: "Traité de jurispr. méd.," etc., Paris, 1893, pp. 193, 194.

the collaboration of two experts greatly increases the time necessarily consumed in the analysis and in the exchange of views concerning the propriety of the steps to be taken, without in any way rendering the result more reliable than if reached alone by the more experienced analyst.

Still more superfluous, and even subversive of accuracy of the results of so delicate an investigation, is the presence during the analysis of the purely medical examiner which was required in Russia.[1]

No person whose ability or integrity is open to the doubt implied by the requirement of testimony at the mouth of two witnesses, or whose perception of color is deficient, should ever be entrusted with a toxicological analysis, either alone or in conjunction with another.

To insure the physical impossibility of admixture of foreign substances by mistake or intentional interference, the analysis should be conducted in a room in which no other work is done at the same time, in which there are no poisonous substances other than those possibly present in the articles under examination and certain chemicals necessarily used, and which no person other than the analyst is permitted to enter. The windows should be sealed, and also the door during the absence of the analyst, and the integrity of the seals verified each time they are broken.

The vessels used in each analysis should be new. Each should bear a distinctive number or letter, and the notes of the analysis should refer to these in such manner that at no time can there be any doubt of what may be the contents of each vessel.

No solvent or chemical is to be considered as pure unless proved to be so by the proper tests, or by purification by the analyst himself.

When it is possible it is highly desirable that samples of the poison actually separated from the articles examined shall be preserved in as many of its distinctive forms as may be.[2] This is always possible if the poison separated by analysis is mineral in nature, but with the volatile and vegetable poisons, when present in small quantity, it is frequently impossible or un-

[1] Dragendorff: "Ermittelung v. Giften," 1te Aufl., 1868, p. 2.

[2] See Forensic Questions, 4.

desirable. Thus when morphin has been separated in very minute quantity it is preferable to use all of the material remaining after the application of the chemical tests to portions, in the application of a life test, rather than to reserve a part, as notable quantities are required to produce the physiological action, and as evidence of this character is more satisfactory to the minds of a lay jury than that based upon delicate chemical reactions.

Finally, we would caution the analyst against undue haste. No step in the analysis should be taken except after mature reflection and appreciation of its possible influence upon the operations to follow. Nor should the analyst permit himself to be led into the expression of an opinion as to what poison may be present before he is perfectly certain that it is present. All "tails," such as filtrates, washings, remainders after extraction, etc., should be preserved until the end of the analysis.

PHYSICAL EXAMINATION.

It sometimes happens, when a poison has been taken in the solid form, or is a substance having a characteristic odor, that portions may remain in the stomach sufficient to permit of identification or to afford indications of its probable character, from its crystalline form, botanic character, color, odor, etc. Such a *preliminary physical examination* of the contents of the stomach and of fragments adherent to its walls (see p. 119), or between the teeth, and of articles of food, medicine, etc., supposed to have been the vehicle of administration should never be neglected.

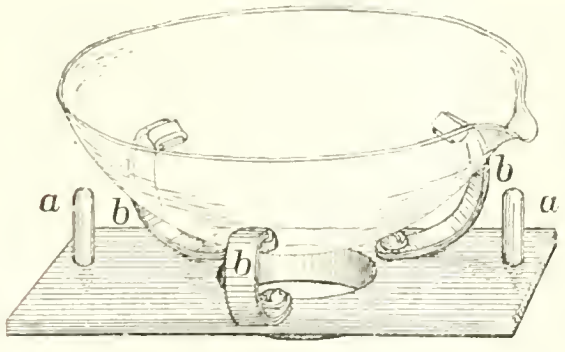

FIG. 1.—Microscope Super-stage for Examining Crystals, etc.

Very frequently the nature of a substance is indicated by its *crystalline form*, either in its own form or in precipitates obtained by the action of reagents. The crystals are almost always small and require the use of a microscope for the determination of their forms. In some cases

also the polarizing microscope is of service. We have found a super-stage of the form shown in Fig. 1, very convenient for microscopic examination of objects in dishes, tubes, etc. For tubes the body of the microscope is tilted back and the tube placed against the pins *aa*. Dishes and other round-bottomed vessels are supported by the three projecting pieces *b*, whose points are covered with small sections of rubber tubing to prevent slipping.[1]

A spectroscopic examination of blood or urine may in some cases promptly furnish valuable evidence. Thus after death from illuminating gas or carbon monoxid the blood presents the characteristic spectrum of carbon monoxid hæmoglobin,[2] and in poisoning by sulfonal the urine shows that of hæmatoporphyrin.[3]

The action of certain substances upon polarized light may also be utilized. Thus copaiva, salicylic acid, salol, arbutin, antifebrin, and chloral render the urine lævogyrous. Certain crystals are also brilliantly colored when examined with polarized light.

CHEMICAL EXAMINATION.

The purposes of the chemical analysis are: 1. The separation of the poison from the constituents of the viscera, vomit, etc., in a condition of purity sufficient to permit of the proper application of the chemical or physiological tests suitable for its identification; and 2. The recognition of the identity of the poison by its physical, chemical, and physiological characters. It is with the methods of separation only that we have to deal in this place. The individual reactions of the various poisons will be considered in the division on Special Toxicology.

The methods adapted to the extraction of poisons from organic mixtures vary with the nature of the poison. For analytical purposes, poisons and corrosives may be classified into:

I. Mineral Acids and Alkalies.—These cause extensive destruction of tissue, recognizable at the autopsy. With

[1] See also "Micro-sublimation," p. 152, and Wormley: "Micro-chemistry of Poisons," 2d ed., Phila., 1885. Erhard: "Die giftige Alkaloide u. d. Ausmitt. auf mikroskopischem Wege," Passau, 1866. Valenti y Vivo: "Atlas de micro-quimica," etc., Barcelona, 1878. Sedgwick: St. Andrew's M. Grad. Assoc. Trans., 1867, i., 200–207.

[2] See vol. ii., p. 31.

[3] See vol. ii., p. 33.

the possible exception of nitric acid, they are either in their own form or in other forms of combination normal constituents of the body, and hence require to be quantitatively determined in cases of supposed death or injury from their action in which analytical investigation is desirable.

II. Volatile and Gaseous Poisons are such as are separable from acid or alkaline mixtures by mere distillation.

III. Organic Poisons, including all non-volatile substances which are decomposed by powerful reagents, and which must consequently be separated by the action of solvents or precipitants not capable of modifying their composition, except in so far as they may be converted into combinations from which they may be released without decomposition.

IV. Mineral Poisons.—Substances of such stability that they remain in a more or less modified form of combination after the action of reagents capable of destroying or removing the organic material with which they are mixed.

While conditions may exceptionally exist in which it is permissible to limit the analysis to the search for a particular poison or group of poisons, in the great majority of cases the work should be so conducted that the search for a particular poison shall not render the detection of another impossible. It is desirable also that a portion of materials submitted shall be set aside, if the quantity be sufficient, in case of accident. For these reasons the objects examined should be divided, except under the conditions mentioned below. The division, however, should be so made as to permit of calculations of quantity in an entire organ from the amount found to be present in a portion thereof. The material, if solid, should be finely divided by a hashing machine, mill, or mortar, thoroughly mixed, weighed, and the portions removed for each analysis also weighed. If liquid they should be both weighed and measured, and the weight and volume of portions taken after thorough stirring also weighed and measured. In some cases the entire organ or substance must be submitted to analysis, as when a liver is to be examined for organic poison, or when the stomach contents or other material are very small in quantity. Under these circumstances the chances of detecting the poison are so remote that they should not be diminished

by operating upon any less material than the entire quantity available.

If the amount of material available be large, it may be divided into three portions, one of which is set aside with the addition of sufficient purified alcohol[1] to prevent putrefaction; the second is examined, first for volatile poisons by distillation, and the residue for mineral poisons, and the third directly for organic poisons.

In most analyses, however, it will be found necessary to use the entire quantity, of some portion submitted at least, for the detection of both organic and mineral poisons, with or without the previous removal of a fraction for the search for volatile poisons. When this is to be done the analysis for organic poisons must necessarily precede that for mineral poisons. All "tails," *i.e.*, filters, washings, the final aqueous liquid after the extraction of vegetable poisons by immiscible solvents, and the residues remaining after the search for vegetable poisons are to be united with the residue of distillation left after the search for volatile poisons, and the materials so reunited examined for mineral poisons.

No reagent is added in the search for volatile or organic poisons which interferes with the subsequent detection of mineral poisons, and in proceeding as above directed there are but two possible causes of partial loss of material. During the heating necessary to distillation certain organic poisons may suffer partial or complete decomposition, hence only a portion of the material should be used for this purpose. There is necessarily a slight loss of material in collecting the various "tails," distributed in a number of vessels, and consequently an error in the quantitative determination of mineral poisons. But as this error is not great, and is in favor of the defence, it may be disregarded.

Examination for Volatile Poisons.

The poisons included in this analytical group are: Phosphorus, the cyanic poisons, chloroform, chloral, alcohols, ethers, hydrocarbons, phenol, nitrobenzene, carbon disulfid, camphor and essential oils, iodin, bromin, and hydrochloric acid.

[1] See p. 133, note 1.

The substances to be examined, finely hashed if solid, and made up to a liquid measure of about 300 c.c. are acidulated with tartaric acid if not already acid, and placed in the distilling flask *a* of the apparatus shown in Fig. 2. The stopcock *b* of the carbon dioxid generator *c* is at first closed. The dis-

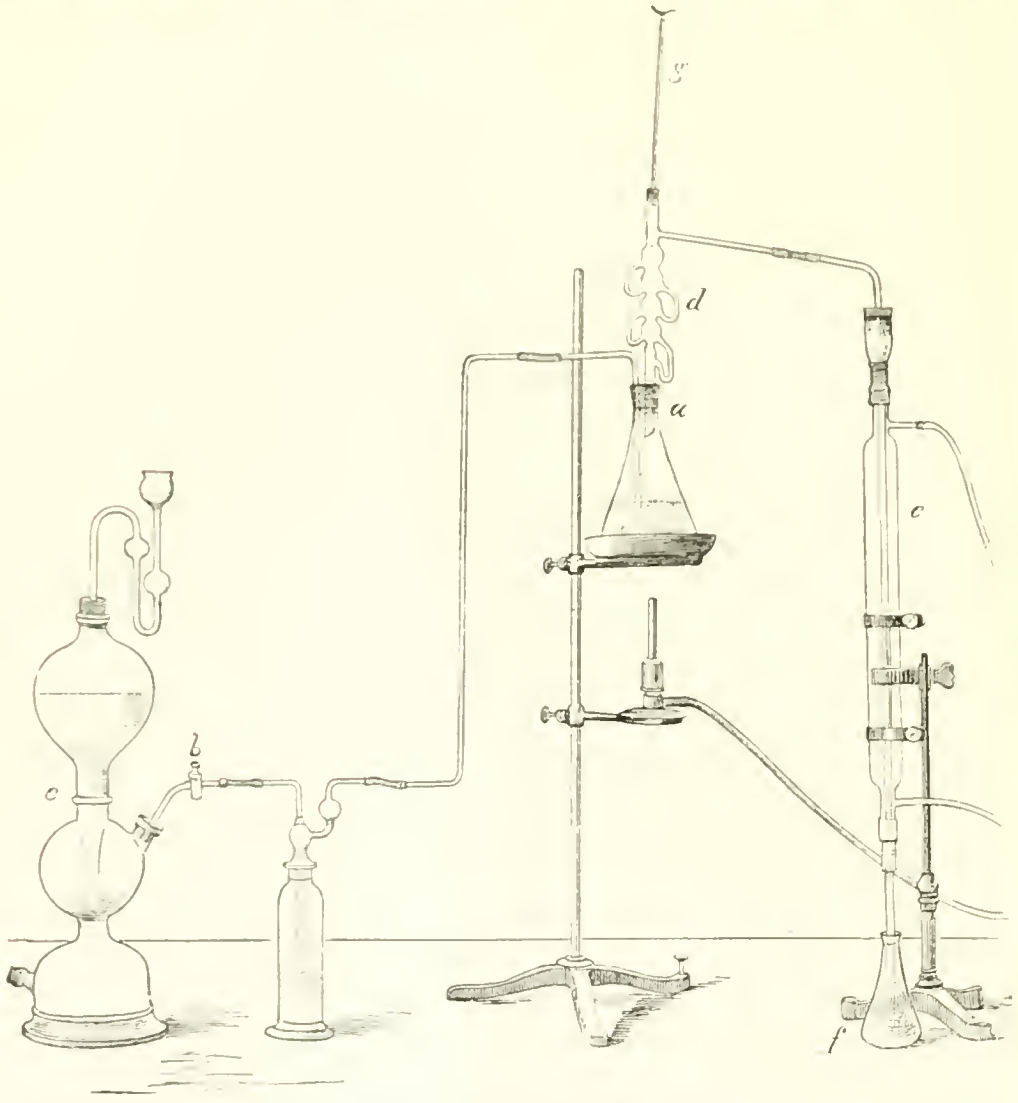

FIG. 2.—Distilling Apparatus for Volatile Poisons.

tillation is conducted in a dark room and with screens so placed, if necessary, that after the burner is lit the fractioning tube *d* and the upper part of the condenser *e* are in total obscurity. The heating, which must be moderate and gradually increased at first to avoid bumping and frothing,[1] is continued until 250

[1] Bumping may be prevented by adding a few short pieces of capillary glass tube to the *cool* contents of the flask; and frothing, in some measure, by the addition of a fragment of paraffin.

c.c. of distillate have been collected in separate portions of 50 c.c. each in five numbered flasks placed at *f*.

During the distillation the fractioning tube *d* and the condenser *e* as well as the connecting tube are to be carefully examined for the phosphorescence produced by phosphorus (see Phosphorus); the temperature indicated by the thermometer *g* is to be noted when the distillate begins to collect and at each change of the receiving flasks, and the several distillates are to be examined by the tests for the volatile poisons to be subsequently described. The reaction of the distillate is to be noted from time to time. If any odor of turpentine be observable either in the original material or in the distillates, or if any pronounced odor capable of masking that of turpentine be present, the distillation must be further conducted for the detection of phosphorus in the manner directed in the special consideration of that poison. Otherwise the apparatus is allowed to cool, 100 c.c. of water holding enough magnesia to render the contents of the flask alkaline are added and two further distillates of 50 c.c. each are collected.

In the distillate from the acid liquid alcohols, ethers, chloroform, hydrocarbons, camphors, essential oils, phenols, nitrobenzene, hydrocyanic acid, iodin, bromin, hydrochloric and acetic acids are to be searched for; and in the distillate from the alkaline liquid, chloral, ammonia, anilin, and volatile bases, including nicotin and coniin.

Examination for Alkaloids, Glucosids, etc.

The processes at present in use for the separation of alkaloidal poisons from complex organic mixtures are either processes by precipitation, or by solution in solvents immiscible with water. The former, while valuable for pharmaceutical purposes and for investigation, are not adapted to the needs of forensic toxicology.

The various methods of extraction by immiscible solvents are variations of, and improvements upon, the method first used by Stas in the Bocarmé case,[1] which was founded upon the following facts:

[1] Poisoning by nicotin, at Bury in Belgium, in 1851.

1st. The tartrates (and most other salts) of the alkaloids are readily soluble in water and in alcohol.

2d. The alkaloids are separated from aqueous solutions of their salts by ammonia and other powerful bases.

3d. The alkaloids thus liberated pass, more or less completely and readily, into ether or other liquids immiscible with water, when these are shaken with aqueous liquids containing the free alkaloids.

Method of Stas.[1]—The material is treated with twice its weight of strong alcohol and 0.5 to 2 gm. of tartaric acid or oxalic acid, preferably the former, and warmed to 60°–65° (140°–149° F.), cooled, filtered, and the filtrate evaporated, preferably in a vacuum over sulfuric acid, or in a current of air, at a temperature not exceeding 35° (95° F.). The remaining liquid is filtered, and filtrate and washings are evaporated in vacuo. The residue is taken up with strong alcohol, and the alcoholic solution again evaporated spontaneously or in vacuo. The residue is dissolved in the smallest possible quantity of water, gradually alkalized with monosodic or monopotassic carbonate, and agitated with four or five times its volume of ether, which is, after separation, decanted and allowed to evaporate at the ordinary temperature.

The modifications suggested by Julius Otto, and constituting the **Stas-Otto method**, properly so called, consisted in agitating the final aqueous liquid while still acid with ether for the purpose of removing fats and other substances, other than alkaloids, soluble in ether, and in adding ammonium chlorid to the alkaline aqueous liquid after extraction by ether, for the purpose of causing morphin (which when crystalline is practically insoluble in ether and soluble in aqueous liquids alkalized with soda) to crystallize.[2]

Prollius[3] adopted the method of Stas, for the extraction more particularly of strychnin, with the modification that chloroform was used in place of ether.

Method of Uslar and Erdmann.[4]—The substances under examination are digested for one to two hours at 60°–80° (140°–

[1] Bull. Acad. roy. d. méd. d. Belg., Brux., 1851–52, xi., 202–312 (p. 304).

[2] Ann. d. Chem., 1856, c., 44, and "Ausmitt. d. Gifte," 3te Aufl., 1867, pp. 37, 42.

[3] Arch. d. Pharm., 1857, lxxxix., 168.

[4] Ann. d. Chem., 1861, cxx., 121. Ztsch. f. an. Chem., 1862, i., 267.

176° F.) with water faintly acidulated with hydrochloric acid, and the solution filtered, treated with a slight excess of ammonium hydroxid, and evaporated to dryness. The residue is extracted three or four times with hot amyl alcohol (boiling point 132° C.); the filtered amylic solution is shaken with hot water, acidulated with hydrochloric acid, and the aqueous solution, after separation, is treated with a slight excess of ammonium hydroxid and again agitated with hot amyl alcohol; the amylic alcohol layer is then separated and evaporated.

In the later **Stas-Otto** process,[1] the use of amyl alcohol for the extraction of morphin (of which it is the best solvent yet known) is adopted as preferable to the crystallization method above described.

This method has more recently been modified by **Marmé**,[2] particularly for the extraction of morphin, by agitating the aqueous liquid, while still acid, with hot amyl alcohol, then rendering it alkaline with ammonium hydroxid and again agitating with the same solvent. The purpose of this modification is to remove coloring matters and other impurities, and thus purify the acid solution before extraction of the morphin from the alkaline solution (see below).

The processes of **Huseman**[3] and of **Thomas**[4] differ from the preceding in that they use acidulated water for the first extraction in place of alcohol, the former acidulating with sulfuric or hydrochloric acid, the latter with acetic acid. In both methods the filtered aqueous extract is directly alkalized with potash or soda, and extracted by agitation with chloroform. In both also the alkaline, aqueous liquid is subsequently examined for morphin.

Method of Dragendorff.—This process has been gradually extended by its author from the simple form in which it was first suggested for the separation of strychnin and brucin, to its present elaborate form to permit of the separation of a great number of alkaloids and glucosids.[5]

The process is conducted as follows: The materials are ex-

[1] 3d and later editions.

[2] Pharm. Ztg., xxviii, 334. Ztschr. f. an. Chem., 1883, xxii., 634.

[3] "Handb. d. Tox.," Berlin, 1862, p. 202.

[4] Chem. News, 1862, 341, 352.

[5] Arch. Sudebn. Med., 1865, i. Ztsch. f. an. Chem., 1866, v., 474. Pharm. Ztschr. f. Russl., 1866, v., 85; 1867, vi., 663. "Ermittelung v. Giften," 4te Aufl., 1895, pp. 149–153. See also Nowak: "Sitzber. math.-natur. Cl. d. k. Ak. d. Wissensch.," Wien, 1866, lxvi., 143.

tracted at 50° (122° F.) with water acidulated with sulfuric acid (not more than 5 c.c. of diluted acid 1:5); the extract is filtered and concentrated to a thin syrup on the water-bath. The residue is gradually mixed with three to four volumes of strong alcohol, and filtered cold after standing twenty-four hours. The alcohol is distilled off. The aqueous liquid remaining, diluted to 50 c.c. and again filtered if necessary, is then, while still acid, successively agitated with (1) petroleum ether, (2) benzene (C_6H_6), (3) chloroform, (4) petroleum ether, each solvent being separated and evaporated apart, furnishing the "acid residues" I., II., III., IV. The aqueous liquid is then rendered alkaline with ammonia, and successively agitated with (5) petroleum ether, (6) benzene, (7) chloroform, (8) amyl alcohol, which are in like manner separated and evaporated to leave the "alkaline residues" V., VI., VII., VIII. Finally the aqueous liquid is evaporated with powdered glass, and extracted with chloroform, which on evaporation leaves the final residue IX. These various residues are then to be examined by the suitable reagents for those alkaloids or glucosids which are extracted by each solvent.

We have found that for chemico-legal purposes a process embodying certain portions of almost all of the above is more advantageous than any one of them—a process which might be described as the Stas-Erdmann-Uslar-Otto-Dragendorff-Marmé method. The reasons for the adoption of the following combination are (1st) Alcohol is a better menstruum for the first extraction than water, because the alcoholic solution may be evaporated at a low temperature, not exceeding 35° (95° F.), within a reasonable time and without the possibility of loss of material by mould or putrefaction which attends similar treatment of an aqueous extract, while the concentration of a watery solution necessitates a higher temperature, which may cause decomposition of even so stable an alkaloid as morphin. (2d) Benzene is a better solvent of strychnin than ether, and almost as good a solvent of atropin. (3d) Amylic alcohol is at present the only available solvent for morphin. It is open to the objection that it also dissolves many other substances, including the cadaveric alkaloids, with equal facility, but until some other solvent not open to these objections shall have been found we are compelled to its use. The objection is to some extent met by ex-

traction of the acid liquid with amylic alcohol before proceeding to the removal of morphin from the alkaline liquid (Marmé). This treatment of the acid aqueous solution with amylic alcohol is postponed until immediately before the extraction of morphin and after the extraction of other alkaloids, in order to avoid the possibility of loss of these alkaloids by their passage from the acid liquid into amylic alcohol (see note, p. 136).

The modification which we have found most serviceable in cases in which all *delectable*, non-volatile organic substances are to be sought for is the following: The substances, finely hashed if solid, are extracted with alcohol[1] acidulated with tartaric acid,[2] at about 35° (95° F.),[3] and washed with alcohol until the washings are no longer acid. The alcoholic filtrate and washings are evaporated spontaneously, either in a current of warm air or in vacuo, to the consistence of a syrup.[4] From three to four volumes of strong alcohol are then gradually stirred in[5] and the solution is strongly cooled and filtered, after having stood twenty-four hours. The alcoholic filtrate is again evapo-

[1] The alcohol used must be purified by dissolving in it about 2 gm. to the litre of tartaric acid, distilling after twenty-four hours, repeating the same operation a second time, and again distilling after twenty-four hours' contact with potassium carbonate. In the last distillation the vapors are passed through a slightly ascending tube about 60 cm. long filled with fresh, granular animal-charcoal.

[2] If the material be strongly acid it is preferable to extract it first with alcohol alone until disappearance of the acid reaction and afterward with alcohol and tartaric acid, and then to carry the alcohol and alcohol and acid extracts through the subsequent stages of the process separately. An aqueous solution of the acid (1 : 10) must form no precipitate with $CaSO_4$, $BaCl_2$, or $(NH_4)_2C_2O_4+NH_4HO$, and must remain unaltered by H_2S even after addition of NH_4HO.

[3] This temperature should not be surpassed at any stage of the process, except as subsequently noted in the evaporation of amylic alcohol.

[4] If the materials under examination contain a large amount of fat they will, according to Focke (Arch. d. Pharm., ccxxii., 307; Ztsch. f. an. Ch., 1884, xxiii., 604) retain a considerable proportion of any strychnin, and a less proportion of morphin, should these be present. To avoid this he suggests that at this point the residue of evaporation of the alcohol be mixed with about ten volumes of water and treated with baryta water in excess. After standing several hours dilute sulfuric acid (1 : 5) is added in slight excess, the liquid is stirred and filtered after an hour, barium chlorid in slight excess is added to the filtrate, which is again filtered, evaporated to a small volume, the residue extracted with absolute alcohol, the extract filtered after having stood for some time and again evaporated.

[5] If the alcohol be gradually added during stirring the precipitate formed is granular. If it be gummy, from too rapid addition of alcohol, it must be subsequently dissolved in a very small quantity of water and again precipitated with alcohol.

rated as before, and the residue dissolved in as small a quantity of water as possible.[1] The acid aqueous liquid is transferred to a separator (Fig. 3), an equal volume of petroleum ether[2] is added, and the two liquids are shaken together repeatedly, at short intervals, for an hour. The two layers are then allowed to separate.[3] The aqueous layer is drawn off through the stopcock below, and the petroleum ether decanted from above into a glass capsule (Fig. 1) in which it is allowed to evaporate spontaneously; while the aqueous liquid, returned to the separator, is similarly treated with a second portion of petroleum ether. Usually the first petroleum ether leaves little or no residue on evaporation. If this be the case the treatment with benzene may be proceeded with after decantation of the second petroleum ether. If a perceptible residue be left by the first petroleum ether, the extraction with that solvent is repeated until nothing more is extracted. In the residue of evaporation of the petroleum ether (I.) the following will be found if present: *Piperin*,[4] *phenol*, *picric acid*, *salicylic acid*, *benzoic acid*, camphors, essential oils, capsicin, cardol, lobeliin.

The acid aqueous liquid is then similarly agitated with benzene,[5] and the benzene extracts are similarly separated and evap-

[1] Croton oil and nitroglycerin are to be sought for in the portion here insoluble in water.

[2] The true petroleum ether, or rhigolene, is neither necessary nor desirable. A product of sufficient purity, volatility, and solvent power may be obtained from commercial gasolene by strongly agitating with dilute sulfuric acid, then with water, drying by contact with calcium chlorid, adding a little lard, and distilling slowly on the water-bath. Those portions are collected which pass below 70° (140° F.).

[3] It sometimes happens with petroleum ether, frequently with benzene, that the two liquids form an emulsion, from which they separate spontaneously with extreme slowness. When this occurs the separator should be allowed to stand over-night. The aqueous layer is then drawn off as far as possible from below, and the separator given a slight shake, and again allowed to stand. A portion of the solvent will separate and may be poured off. The emulsion is then transferred to a dry filter in a funnel over a second separator, a drop of alcohol is added and the emulsion stirred in the filter with a glass rod. The liquids will either pass through the filter and form separate layers in the separator below, or the emulsion will contract, and the solvent separate in a layer in the filter, which may be removed by a pipette. Patience is necessary. If the extract by any of the solvents be cloudy it must be filtered through a small filter moistened with the same liquid.

[4] The substances whose names are given in *italics* pass only in traces into this solvent and are more perfectly extracted by one of those used subsequently.

[5] The commercial "crystallizable benzol, free from thiophene" is sometimes sufficiently pure. It should give no brownish tint with concentrated sulfuric acid, nor any blue color when agitated with the same acid and isatin. To purify benzene it should be agitated with

orated. The benzene residue from acid solution (II.) may contain: Caffein, cantharidin, anemonin, *santonin*, caryophyllin, cubebin, phenol, quinol, catechol, resorcinol, salicylic acid, picric acid, piperin, *colchicin*, colchicein, digitalin, gratiolin, cascarillin, elaterin, *populin*, *colocynthin*, *geissospermin*, chrysamic acid, absynthin, oils (anemone, ranunculus), *quassin*, *menyanthin*, *ericolin*, *daphnin*, *cnicin*, *sabadillin*, *meconin*, *antipyrin*.

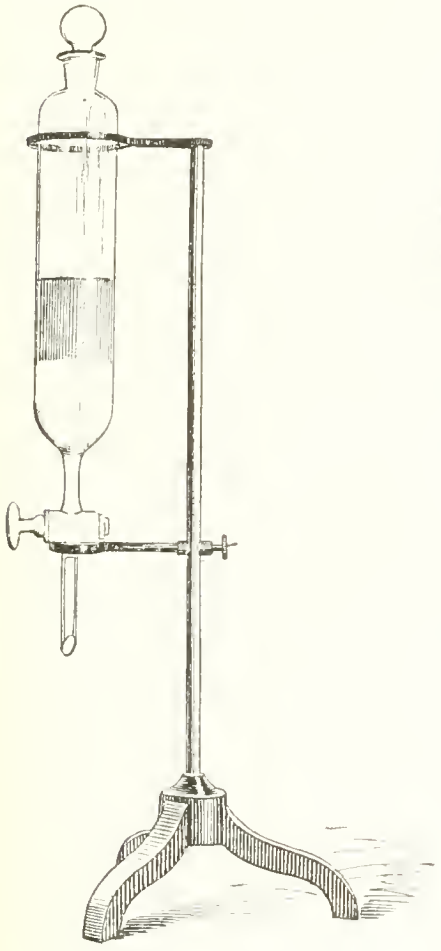

Fig. 3.—Separator.

The acid aqueous liquid is next agitated with chloroform,[1] and the chloroform layers drawn off from below and evaporated spontaneously. The residue left by the chloroform from acid solution (III.) may contain: Cinchonin, theobromin, papaverin, narcein, hydrastin, chelidonin, solanidin, æsculin, gelsemic

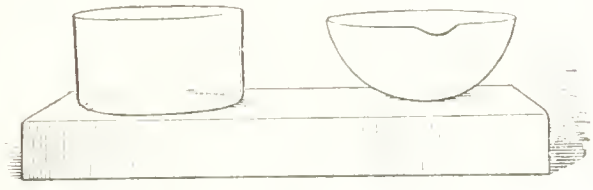

Fig. 4.—Glass Evaporating Dishes.[2]

acid, picrotoxin, helleborein, populin, quassin, menyanthin, ericolin, daphnin, cnicin, benzoic acid, digitalein, digitalin, *convallamarin*, saponin, senegin, lycaconitin, myoctonin, aspidospermin, pereirin, *brucin*, sanguinarin, quebrachin, geissospermin, syringin, *colchicin*, *colocynthin*, *meconin*, anthemin,

about one-tenth its volume of concentrated sulfuric acid until a sample no longer gives the indophenone reaction with sulfuric acid and isatin. The benzene is afterwards agitated with water, dried by contact with calcium chlorid and distilled slowly, that portion being collected which passes at 80°–82° (176°–180° F.).

[1] It should be purified by agitation with dilute caustic potash solution, separation, and distillation shortly before use.

[2] The round form is more convenient for the microscopic examination of the residue. The vertical-walled form is preferable with ether solutions, to avoid "crawling" of the liquid.

santonin, *ergotinin*, gentiopicrin, veratrin, jervin, *antipyrin*, acetanilid.

The acid aqueous liquid is then agitated once with ethylic ether,[1] and the treatment repeated if the first ether layer leaves a residue on evaporation. The residue of evaporation of the ether (IV.) may contain: oxalic acid, meconic acid, ergotinin, arbutin, gallic acid, pyrogallol, β-naphthol, chrysophanic acid, naphthalin.

The aqueous liquid is next rendered alkaline by ammonium hydroxid in slight excess, and agitated cold with petroleum ether as before. The residue of evaporation of the ether (V.) may contain: *strychnin*, *quinin*, *delphinin*, *sabadillin*, *brucin*, *aspidospermin*, *pereirin*, *veratrin*, *emetin*, *gelsemin*, *quebrachin*, *oxyacanthin*, cocain, coniin, methylconiin, piperidin, amins (methyl-, ethyl-, propyl,- and amyl-), capsicin, sarracenin, nicotin, lobelin, spartein, quinolin, anilin, toluidin, picolin, urechitoxin, kairin, *thallin*.

The alkaline aqueous liquid is then agitated in a similar manner with benzene. The residue of the evaporation of benzene from the alkaline liquid (VI.) may contain:[2] atropin, hyoscyamin, strychnin, ethyl- and methyl-strychnin, quinin, *cinchonin*, quinidin, cocain, narcotin, codein, thebain, sabadillin, taxin, brucin, physostigmin, pilocarpin, veratrin, sabatrin, delphinoidin, *lycaconitin*, myoctonin, aconitin, *antipyrin*, thallin.

The alkaline aqueous liquid is then agitated with chloroform, in the residue of whose evaporation (VII.) there may be: cinchonin, cinchonidin, papaverin, narcein, laudanin, berberin, lycaconitin, *muscarin*, *morphin*, pelletierin, antipyrin.

The aqueous liquid is then rendered acid by the addition of dilute sulfuric acid[3] and agitated with cold amylic alcohol, which

[1] The ether used should be purified by agitation with water and redistillation, first from quicklime, then from tartaric acid, and finally from metallic sodium.

[2] This residue frequently contains a crystalline substance which is not an alkaloid, but whose nature we have not been able to determine from lack of material. The crystals are brilliant, frequently present beautiful pink, green, blue, and violet iridescence, and often do not appear until after the residue has stood for twenty-four or forty-eight hours. They are sometimes plates, more usually flattened prisms with ends truncated obliquely at angles of about 86° and 94°. This material is not soluble in dilute hydrochloric acid and consequently does not remain in the residue purified as directed below (see p. 138).

[3] Sulfuric acid, which should not be added in great excess, is preferred to hydrochloric, acetic, and tartaric acids at this point to avoid so far as possible any loss of morphin by the

is drawn off after separation and immediately evaporated over the water-bath. If no considerable residue remain the next step is proceeded with. If a residue remain the agitation with amyl alcohol[1] is to be repeated until an alcoholic layer leaves little or no residue. The purpose of this treatment is principally the removal of substances other than morphin soluble in amylic alcohol. The amylic alcohol layers on evaporation (on the water-bath at first, subsequently spontaneously) leave a residue (VIII.) which may contain: *sabadillin*, meconin, meconic acid, *oxalic acid*, *morphin*, colchicin, colocynthin, and convallamarin. They will also leave considerable quantities of arsenic if the body has been embalmed.

The acid aqueous liquid being returned to the separator, an equal volume of hot amyl alcohol is added and then ammonium hydroxid to alkaline reaction, and the liquids are strongly shaken together. The residue of evaporation of the amyl alcohol (IX.) may contain: morphin, solanin, salicin, *convallamarin*, *saponin*, *senegin*, *narcein*.

sparing solubility of its salts in amylic alcohol, that of the sulfate being less than that of the others. We have found that 25 c.c. of amylic alcohol, agitated under similar conditions with acid solutions, dissolved 0.0143 gm. of the acetate, 0.0098 of the chlorid, 0.0059 of the tartrate, and 0.0045 of the sulfate. With *hot* amyl alcohol Wormley (Univ. Med. Mag., May, 1890, Reprint, p. 8) obtained somewhat different results: acetate 0.005, sulfate 0.0028, chlorid 0.0025.

[1] Objection has been made to the use of amyl alcohol on the grounds that it is disagreeable to the operator, that it itself frequently contains poisonous bases and furfurol, and that it is a good solvent of the ptomains, whose exclusion is, of course, highly desirable. The first objection concerns no one but the chemist, the second is removable by proper purification of the solvent. The third is more serious, but until some solvent of morphin not open to this objection is found, we are compelled to the use of amyl alcohol, and to reliance upon subsequent purification of the extract.

The impurities liable to be present in amylic alcohol (iso-amylic) are basic substances (pyridin and related bodies) and furfurol. The former may be removed by distilling the alcohol two or three times from tartaric acid. The removal of the latter, which interferes with the crystallization but not with the reactions of morphin, is more difficult. We have been unable to obtain satisfactory results with the very tedious method suggested by Udransky (Zeitsch. f. phys. Ch., 1889, xiii., 248–263). An alcohol free from furfurol may, however, be obtained from Gustave Claudon, 18 Ave. Victoria, Paris, at the moderate price of six francs the kilo. Much more costly products from German manufacturers contain furfurol in relatively large amount. The alcohol should be tested by adding a few drops of a fifteen-per-cent alcoholic solution of α-naphthol to a sample in a test tube and "underrunning" it with concentrated sulfuric acid; only an extremely faint reddish tinge should be formed at the junction of the liquids. The alcohol should be saturated with water by agitation with it.

Finally the aqueous liquid is evaporated to dryness with powdered glass in a Hofmeister capsule over the water-bath. Capsule and contents are then pulverized and extracted with hot chloroform. The residue of evaporation of the chloroform (X.) may contain curarin and muscarin.

The process may in many cases be greatly abbreviated. Thus if a sample of urine is to be examined for strychnin alone it may be acidulated with dilute hydrochloric acid (or tartaric), filtered, agitated with benzene two or three times, then again agitated with the same solvent after addition of ammonium hydroxid to alkaline reaction. Strychnin, if present, will be found in the residue of evaporation of benzene from alkaline solution.

The alkaloid is sometimes present in the residue in a degree of purity sufficient to permit of its identification directly, but in most instances a purification is required. This is usually effected by dissolving the residue in a small quantity of acidulated water, filtering, agitating the acid aqueous solution with the proper solvent, and then again (to extract the alkaloid) with the same solvent, after alkalization with ammonium hydroxid or with magnesia, and repeating the process if necessary. Or in some cases the alkaloid or the impurity may be removed by some other solvent. When the residue has been suitably purified, its quantity may in some cases be approximately determined at this stage. It is then dissolved in a small quantity of the proper solvent, and drops of the solution transferred either to a slip of glass which may be placed upon either a black or white background, or to small watch-glasses, and the identifying tests applied to the drops themselves or to the residues left upon their evaporation.

For the detection and identification of alkaloids *general* and *special reagents* are used. The former produce precipitates with a great number of alkaloids and glucosids, varying sometimes in their color and crystalline form with different individuals, and are consequently more frequently used to determine the presence of an alkaloid or glucosid than to determine its identity. The special reactions, on the other hand, are such as are peculiar to one or to a few substances, and are consequently of greater value for identification. The special reactions will be described in the division of special toxicology under each alkaloid or glucosid.

General Reagents for Alkaloids and Glucosids.

1. De Vry's (Sonnenschein's) Reagent—Phosphomolybdic Acid.[1]

This reagent is applied to an acid solution, in which it forms yellowish precipitates with most of the alkaloids. Acolyetin, aconitin, anagyrin, anthocerein, alpha-homo-chelidonin, arecolin, beta-homochelidonin, calamin, cannabin, carpain, ceanothin, cinchonin, cocain, colchicein, colchicin, curarin, cytisin, ecbolin, emetin, ergotin, ergotinin, laurotetanin, morphin, narcein, nicotin, oxyacanthin, pelletierin, physostigmin, protoveratrin, pseudojervin, sabadillin and sabatrin, solanin, strychnin, taxin, thebain, thein, veratralbin and veratrin give yellowish-white precipitates. The precipitates with aspidospermin, cannabinin, damascenin, scopolin, and spartein are white; those with ecgonin, hymenodictyonin, hyoscyamin, piliganin, pseudopelletierin, and theobromin are yellow; those with atherospermin and berberin are dirty yellow; that with delphinin is gray-yellow; those with narcotin, codein, and piperin are brown-yellow; that with brucin is orange; that with aconin is gray-bluish; that with erythrophlein is dirty green; that with calcitropin is yellow, turning to blue-green after twenty-four hours; those with atropin, bebeerin, coniin, and lobelin are yellowish and turn blue with ammonia; that with quinin is white and turns yellow with ammonia. The following alkaloids give no precipitates: arecain, chrysanthemin, eupatorin, lycoctonin, and papaverin.

The precipitates are not soluble in cold alcohol, ether, or dilute mineral acids (except phosphoric), but are soluble in concentrated hydrochloric acid, hot nitric acid, acetic, oxalic, and tartaric acids, and the hydroxids, carbonates, borates, and phosphates of the alkali metals. The precipitates are decomposed, with liberation of the alkaloid, by the oxids of calcium, barium, lead, and silver and by the hydroxids of potassium and sodium, and some are also decomposed by barium and sodium carbonates and by magnesia.[2] Ammonia and many of its derivatives, such as the monamins, anilin, quinolin, give precipitates resembling those formed by the alkaloids with this reagent, but non-

[1] De Vry: Jour. de pharm., etc., 1854, 3 s., xxvi., 220. Sonnenschein: "Ueber ein neues Reagens auf Alkaloide," Berlin, 1857. Also "Handb. d. ger. Med.," 1869, p. 317. To prepare the reagent, precipitate a solution of disodic phosphate, acidulated with nitric acid, with a similarly acidulated solution of ammonium molybdate. Wash the precipitate and dissolve it in the smallest quantity of sodium hydroxid solution. Evaporate the solution to dryness; ignite the residue so long as ammonia is given off; dissolve the residue in water (1 : 10), and add nitric acid until the precipitate formed is redissolved. The yellow solution must be protected from ammonia.

[2] See p. 150.

alkaloidal organic poisons are not as a rule precipitated by it. The following, however, are: euonymin, helleborein, and menyanthin. Acorin produces a blue color by reduction. Strophanthin gives a pale-green precipitate if present in quantity, and an emerald solution if dilute.

2. Scheibler's Reagent—Phosphotungstic Acid.[1]

This reagent forms precipitates in acid solutions of salts of the alkaloids, very similar to those produced by phosphomolybdic acid. They are, however, less permanent and somewhat more soluble.

3. Mayer's Reagent—Mercury-Potassium Iodid.[2]

This reagent, which is also used for quantitative determinations, gives with acid solutions of most of the alkaloids precipitates which are for the most part white or yellowish and crystalline, or become crystalline after standing for a time, when added to aqueous solutions of their sulfates or chlorids. According to Dragendorff the precipitates from dilute solutions of narcotin, thebain, narcein, emetin, aconitin, delphinoidin, and bebeerin do not become crystalline; and those with coniin and nicotin are at first white and amorphous, but afterward become resinous and adherent, and frequently after twenty-four to forty-eight hours beautifully crystalline. The precipitates with amaryllin, arecain, arecolin, artarin, aspidospermin, bellamarin, brucin, calcitropin, crysanthemin, corydalin, delphinin, emetin, granditlorin, lobeliin, piperin, rhœadin, sabadillin, sebatrin, and veratrin are yellow or yellowish. No precipitates are formed in dilute solutions of caffein, colchicin, solanin, and theobromin. The following non-alkaloidal substances are also precipitated: acorin, euonymin, menyanthin, sikimin, and sophorin.

4. Marme's Reagent—Cadmium-Potassium Iodid.[3]

This reagent forms precipitates in solutions of the salts of most of the alkaloids acidulated with dilute sulfuric acid. The precipitates are nearly all flocculent and white, but some soon become crystalline. They are soluble in excess of the reagent and in alcohol, insoluble in ether. The alkaloids may be recovered from the precipitates by decomposition with alkalies and extraction with the proper solvent. The

[1] Ber. d. Chem. Gesell., 1872, v., 801. Prepared by boiling sodium tungstate with phosphoric acid, neutralizing with hydrochloric acid, precipitating with barium chlorid, decomposing the precipitate with sulfuric acid, and evaporating to crystallization. The acid is dissolved in water.

[2] First suggested by Planta-Reichenau (Delffs): Dis., Heidelberg, 1846; perfected by Mayer: Ztsch. f. an. Chem., 1863, ii., 225. Obtained by dissolving 13.546 gm. mercuric chlorid and 49.8 gm. potassium iodid to the litre of water.

[3] Compt. rend. Ac. Sc., Paris, 1866, lxiii., 843. Ztsch. f. an. Chem., 1867, vi., 123. Prepared by saturating a boiling concentrated solution of potassium iodid with cadmium iodid and adding an equal volume of cold saturated solution of potassium iodid. Dilute solutions do not keep.

reagent does not precipitate with many glucosids and leucomains, nor with ammonia. Caffein forms no precipitate, and solanin and theobromin do so only in concentrated solutions.

5. Dragendorff's Reagent—Bismuth-Potassium Iodid.[1]

The reagent is applied to aqueous solutions of the alkaloids, acidulated with sulfuric acid (four drops of concentrated acid to ten cubic centimetres). The solution must contain no ether nor any trace of amyl alcohol. The precipitates formed with most of the alkaloids are orange. Of the glucosids menyanthin gives a yellow precipitate, and digitalin one in concentrated solution. Kraut[2] has studied the compounds formed by this reagent with amins, glycins, piperidin, sulfins, anilin, and toluidin.

6. Wagner's Reagent—Iodin in Potassium Iodid.[3]

Brown precipitates are formed in solutions of almost all of the alkaloids, acidulated with sulfuric acid, when the reagent is added in small amount. The precipitates are for the most part flocculent and amorphous, but in many instances become crystalline.

7. Tannic Acid.[4]

Gives white, grayish, or yellowish precipitates with almost all of the alkaloids, glucosids, and bitter principles. The following are not precipitated: arginin, aribin, arecolin, chrysanthemin, antiarin, chamelirin, chirettogenin, datiscin, frangulin, angelin, columbin, cascarillin, capsicin, cornin, marrubin, ononid, and picrotoxin; and the following only imperfectly: arecaïn, grandiflorin, theobromin; ericolin and strophanthin. In most cases the precipitates are soluble in acids, even in excess of the reagent, although some alkaloids: aconitin, physostigmin, and veratrin, give precipitates in solutions strongly acidulated

[1] Pharm. Ztsch. f. Russl., 1866, v., 81. Also "Ermitt. v. Giften," 4te Aufl., 155. Prepared like Marmé's reagent, using bismuth in place of cadmium iodid. Cannot be diluted (Dragendorff). Dissolve 80 gm. bismuth subnitrate in 200 c.c. nitric acid (sp. gr. 1.18), and 272 gm. potassium iodid in a little water; add the bismuth solution slowly and during agitation to the iodid solution, cool strongly, separate from the crystals and dilute to a litre. Must be kept in the dark (Kraut). See also Mangini: Gazz. ch. Ital., 1882, xii., 115.

[2] Ann. d. Ch., 1881, ccx., 310.

[3] Ztschr. f. an. Chem., 1865, iv., 387. A decinormal solution: 12.66 gm. iodin to the litre of potassium iodid solution. Wormley ("Micr.-Chem. Pois.," 2d ed., 444) recommends a stronger solution: 5 parts potassium iodid and 2 parts iodin in 100 of water. As the reagent is used in acid solution it is practically the same as the solution of iodin in hydriodic acid used by Selmi and others. Bouchardat (Wilder, "Tests," p. 13) uses a solution of 10 parts of iodin and 20 parts of potassium iodid in 500 of water. See also Hilger: "Verbind. d. Iod mit d. Pflanzenalkaloiden," Würzb., 1869, and abst. Pharm. Jahrb., 1869, 551. Jörgensen: "Berichte," Berlin, 1869, ii., 460.

[4] A freshly prepared solution in a mixture of 8 parts water and 1 part alcohol.

with sulfuric acid, but not in weakly acid solutions. The precipitates of the tannates of aconitin, berberin, cinchonin, colchicin, narcotin, papaverin, solanin, and thebain are not dissolved by cold dilute hydrochloric acid; and those of aconitin, physostigmin, quinin, solanin, and veratrin are not dissolved by cold dilute sulfuric acid. Dilute acetic acid dissolves the tannates of aconitin, brucin, caffein, colchicin, morphin, physostigmin, and veratrin; and the concentrated acid that of quinin.

8. Platinic Chlorid.

Forms double salts with the chlorids of most of the alkaloids, which are difficultly soluble and frequently crystalline. The precipitates are usually yellow; that of the hydrastin compound is yellowish-red; that of meconidin yellow, changing to red; and those of berbamin, emetin, papaverin, quinidin, and quinin are white or nearly so. The following are not precipitated: amaryllin, angelin, arecolin, chrysanthemin, ecgonin, jurubebin, lycoctonin, pelletierin, picraconitin, piliganin, protoveratridin, protoveratrin, and trigonellin; and the following only from concentrated solutions: aconitin, atropin, coniin, cytisin, hyoscyamin, narcotin, nicotin, physostigmin, piperin, piturin, pseudaconitin, and thein. Morphin gives a precipitate immediately in solutions of concentration greater than 1 : 100, or in more dilute solutions (to 1 : 3,000) after twenty-four hours; the deposit becoming crystalline and being insoluble in cold hydrochloric acid. Aspidospermin and acorin, aloin and catechin reduce the platinic salt with formation of a blue color.

The chloroplatinates of the alkaloids are definite compounds, whose analysis has been frequently utilized to determine the composition and molecular weight of the alkaloid.

9. Auric Chlorid.

Forms chloraurates with the alkaloids, which are for the most part yellow, insoluble or difficulty soluble and crystalline. The precipitates of the chloraurates of aricin, brucin, colloturin, delphinin, ecgonin, emetin, meconidin, quebrachin, quinicin, quinin, rhœadin, and strychnin are amorphous. Eupatorin and pilocarpidin form no precipitates: codein, narcotin, and nicotin only in concentrated solutions; and caffein, colchicin, and theobromin only slowly. Reduction of the gold compound occurs much more frequently than of the platinic, and is indicated by a purple color, either immediately or after a time. The following alkaloids cause reduction: aconin, apomorphin, concusconin, conquinamin, eupatorin, geissospermin, hydroquebrachin, paytamin, paytin, pelletierin (on warming), physostigmin and quinamin; as do many of the glucosids.

The crystalline chloraurates have served the same analytical purposes as the chloroplatinates.

10. Hager's Reagent—Picric Acid. [1]

Added to solutions of most of the alkaloids acidulated with sulfuric acid it causes precipitates which are in many cases crystalline. Glucosids, with the exception of adonin, euonymin, and limonin, are not precipitated. Morphin in concentrated solution forms a precipitate which disappears on dilution. From dilute acid solutions, anilin, caffein, morphin, pseudomorphin, solanin, and theobromin are not precipitated: and aconitin and atropin only incompletely even from concentrated solutions. From strongly acid solutions berberin, the cinchona alkaloids, colchicin, delphinin, emetin, the opium alkaloids other than morphin and pseudomorphin, the strychnos alkaloids and veratrin are completely precipitated.

The alkaloid may be liberated from its picrate by potassium hydroxid.[2]

11. Other General Reagents.

Besides various modifications of the above, a number of other general reagents have been used, of which the following are the more prominent:

Bromin,[3] in aqueous solution, causes in acid solutions of many alkaloids yellow precipitates, which sometimes (atropin) assume characteristic forms; and with some, such as brucin, strychnin, narcotin, and quinin, a rose-pink or red color, either immediately or on being boiled.

Phospho-antimonic acid[4] is said to cause white amorphous precipitates in solutions of the alkaloids in excess of dilute sulfuric acid. Color reactions are also produced with brucin, narcotin, and piperin. The delicacy of the reagent is less than that of Sonnenschein's, except with atropin. Digitalin is also precipitated.

Silicotungstic acid,[5] either in aqueous or alcoholic solution, causes precipitates with almost all of the alkaloids, from which the alkaloid may be liberated by decomposition with caustic alkalies.

Iodin chlorid[6] forms yellow, crystalline precipitates, which are

[1] Pharm. Centralhalle, 1869, 131. Ztschr. an. Ch., 1870, ix., 110. A saturated aqueous solution.

[2] See also Popoff: Ann. d'hyg., 1891, 3 s., xxvi., 81, and in Brouardel and Ogier: "Lab. d. Tox.," 203.

[3] Either bromin water, applied to the solution of the alkaloid in dilute hydrochloric acid (Bloxam: Chem. News, 1883, xlvii., 215); or a solution of bromin in potassium bromid; or aqueous hydrobromic acid saturated with bromin (Wormley: Prescott, "Organ. Anal.," p. 47).

[4] Schultze: Ann. Ch. u. Ph., 1859, cix., 177, obtained by dropping antimony pentachlorid into aqueous phosphoric acid; or by adding one volume antimony pentachlorid to four volumes of saturated solution of disodic phosphate.

[5] Godeffroy: Ztschr. f. an. Chem., 1877, xvi., 244. Prepared by boiling solution of sodium tungstate with freshly precipitated silicic acid, precipitating with mercurous nitrate, decomposing the washed precipitate with hydrochloric acid and evaporating to crystallization.

[6] Dittmar: Ber. deut. chem. Gesell., 1885, xviii., 1612. Pre-

blackened by ammonia, from hydrochloric acid solutions of pyridin and quinolin and of the alkaloids which are derivatives of those bases.

Potassium platinocyanid[1] forms crystalline deposits with some alkaloids, notably with morphin, strychnin, and the cinchona alkaloids.

Potassium argentocyanid[2] gives amorphous, colorless precipitates when added in excess to very faintly acid solutions of many of the alkaloids. Some of the precipitates become crystalline.

Potassium cupro-cyanid,[3] *potassium ferrocyanid*, *potassium ferricyanid*, and *potassium thiocyanate*[4] form precipitates in solutions of some of the alkaloids, which are frequently crystalline.

Potassium-zinc iodid[5] forms in not too dilute solutions of some of the alkaloids (strychnin, brucin, quinin, codein, papaverin, narcein) white or yellowish precipitates which are or become crystalline.

Lead acetate, neutral or basic, forms precipitates with only a few of the alkaloids (acolyctin, aconin, nicotin, spartein, and theobromin) but does with many glucosids and bitter principles.

Mercuric chlorid also gives white precipitates with many alkaloids, which are for the most part crystalline or gradually become so. Those of berberin, brucin (except concentrated), hyoscin, quinidin, and quinin remain amorphous. The following alkaloids give no precipitates: arecolin, chrysanthemin, ergotinin, lobeliin, lycoctonin, protoveratrin, taxin, and trigonellin. Many glucosids also form white precipitates.

Silver nitrate is reduced by some alkaloids: aconin, capsaicin, cupronin, dibromapophyllin, morphin, and pseudaconin; and precipitates with some others: anthocerein, capsicin (in strong alcoholic solution), capsicol, lobeliin, oxyacanthin, quinidin, and theobromin. It is also reduced by several glucosids and bitter principles.

Potassium dichromate in saturated aqueous solution forms precipitates which soon become crystalline with solutions of most alkaloidal salts. The following, however, form no precipitates: aconitin, amaryllin, and delphinin; the following only in concentrated solutions: cocain, morphin, sabadillin, sabatrin, and thein; and narcein only in acid solution. Alstonin and porphyrin form red solutions.

Potassium permanganate is reduced more or less rapidly by many alkaloids, with precipitation of manganic peroxid. A few alkaloids

pared either by agitating iodin chlorid with water; by passing chlorin through water holding iodin in suspension; or by mixing together 5 c.c. solution of potassium iodid (1:10) and 18 c.c. solution of potassium nitrite; adding 6.5 c.c. hydrochloric acid (thirty-three per cent.) and filtering after effervescence has ceased.

[1] Delffs: Chem. Centlbl., 1864, 607. Van der Burg: Ztschr. f. an. Chem., 1865, iv., 296.

[2] Dragendorff: "Ermitt. v. Giften." 4te Aufl., 157.

[3] Dragendorff: *L. c.*

[4] Dragendorff: *Op. cit.*, and Wormley: "Micro-Chem. Poisons," *passim*.

[5] Dragendorff: *L. c.* Prepared in the same manner as Marmé's reagent.

behave peculiarly with this reagent: apomorphin gives an intense green color; morphin forms a white, crystalline precipitate (oxydimorphin); cocain forms a stable violet precipitate; and narcein, narcotin, and papaverin similar, but less stable, precipitates.

Sodium thioantimonate forms yellow, flocculent precipitates in solutions, as nearly neutral as possible, of salts of quinin, cinchonin, quinidin, morphin, codein, narcotin, strychnin, brucin, atropin, and bebeerin.

Lead chlorid forms precipitates, usually finely crystalline, in solutions of the chlorids, acetates, or nitrates (not sulfates) of strychnin, quinin, cinchonin, morphin, codein, brucin, and probably of other alkaloids.

Ammonium diaminchromium thiocyanate[3] when added in saturated aqueous solution to solutions of the chlorids of the monamins, anilin, toluidins, pyridin, quinolin, quinin, strychnin, cocain, pilocarpin, atropin, and hyoscyamin, produces red or purple crystalline precipitates.

Benzoyl chlorid and *sodium hydroxid*[4] when agitated with aqueous solutions of cadaverin, putrescin, and other diamins causes the formation of insoluble crystalline precipitates of dibenzoyl compounds. Polyatomic alcohols, including glycerol and the carbohydrates, are similarly precipitated.

Color Tests.—Besides the above reagents, which act as precipitants, there are numerous substances or mixtures which produce with the alkaloids and glucosids changes of color more or less pronounced and characteristic. In applying them it is, however, essential that the alkaloid shall be separated in as pure a condition as the circumstances will permit, and shall be free from other substances capable of causing color changes which may mask those due to the alkaloid, prevent their formation, or themselves cause similar colorations.

Some of these reagents produce colors with a great number of alkaloids and glucosids and may therefore be considered as *general reagents*. Such are the following:

Sulfuric acid applied in the concentrated form to the solid alka-

[1] Palm: Ztsch. f. an. Ch., 1883, xxii., 224.

[2] Palm: *L. c.*, p. 227. Lead chlorid dissolved in boiling sodium chlorid solution.

[3] Reinecke's salt. Ann. d. Chem., cxxvi., 113. Christensen: Jour. pr. Chem. [2], xlv., 213, 356. J. Chem. Soc., 1892, lxii., 798, 1000 [abst.]. Ammonium thiocyanate (200 gm.) is fused in a porcelain crucible, powdered potassium dichromate (40 gm.) is gradually added, the melt is extracted with cold water, the solution crystallized, and the crystalline residue again extracted with cold water and recrystallized.

[4] Udransky and Baumann: Berichte, 1888, xxi., 2745.

loid causes colors which are *red*, *purple*, or *violet* with aconitin (only when impure), apomorphin, beta-homo-chelidonin, boldin, brucin (due to HNO_3 in the H_2SO_4), cocain (only when impure), delphinoidin, ditamin, echolin, echitamin, echitumin, fumarin, hydrastin (due to HNO_3 in the H_2SO_4), hypoquebrachin, laudanin, laurotetanin, narcein (if impure), pareirin, porphyrin, protoveratridin, rhœadin, rubijervin, sabadillin, sabadinin, sabatrin, staphisagrin, taxin, thebain; adonin, angelin, amygdalin, brucamarin, californin, calumbin, camellin, cascarillin, cerberin, cnicin, convallamarin, convolvulin, copalchin, cyclamin, digitonin, dulcamarin, elaterin, gratiosolin, helixin, helleborein, helleboretin, helleborin, hesperidin, hydrocarotin, jalapin, kosin, laserpitin, limonin, linin, panaquilon, phillygenin, phillyrnin, phloretin, physodin, pimpinellin, populin, quinovin (slowly), salicin, saligenin, sapogenin (slowly), saponin, sapotin, smilacin, sophorin, tampicin (slowly), and turpethin (slowly). With the following a red or violet color is preceded by *yellow:* atisin, corydalin, grandiflorin, heliotropin, phlorizin, sabadin, smilacin, veratralbin, veratrin; assamic acid, ononin, phlorrhizin, urechitin, and urechitoxin; by *brown* with menyanthin, scillitoxin, and vernonin; by *green* with asiminin, frangulin, and sparattospermin; and by *blue* with curarin and papaverin (if impure). The red tinge is succeeded by *blue* and *violet* with assamic acid; by *black* with chamælirin; by *green*, *blue*, and *violet* with podophyllotoxin; and by *brown* with scillitoxin. The color is *yellow*, *orange*, or *brown* with alpha-homo-chelidonin, amaryllin, anthocercin, berberin, calycanthin, ceanothin, chelerythrin, cinchonamin, colchicein, colchicin, delphinin, delphinoidin, emetin, gelsemin, gnoscopin, hydrocotarnin, imperialin, javanin, jervin, laudanosin, lobelin, narcein, oxyacanthin, solanin, veratroidin; andrometoxin, antiarin, capsicin, diosmin, ericolin, eupatorin, euphorbin, fraxetin, gentiopicrin, gratiolin, gratiosolin, guacin, helixin, helleborein, helleborin, junipicrin, liriodendrin, mangostin, picrotoxin, prophetin, rosaginin, scillain, scillin, solanin, strophanthin, syringopicrin, and villosin. A yellow or brown color is succeeded by *violet* and *brown-gray* with calcitropin, by *green* with chairamidin, concheiramidin, conchairamin, physostigmin, piperin (slowly), ustilagin, and agoniadin. With gelsemin and euonymin the color is yellow, changing to brown. The following give *blue* or *green colors:* aricin, aristin, bebeerin, codamin, concusconin, cryptopin, cuprin, cuscurin, geissospermin, glaucin, papaverin (if impure), paricin, protoveratrin, pseudomorphin, quebrachamin, rhœadin, thebaicin, thebenin, ustilagin; aristolochin, asclepiadin, coniferin, crocetin, crocin, digitalin (?), and syringin. Tylophorin and absinthin are at first *brown*, then *green*, then *blue*. Strophanthin is *pale yellow*, then *brown*, then *green*.

Erdmann's reagent,[1] **Froehde's reagent,**[2] **Buckingham's reagent,**[3] **Selmi's reagent,**[4] **Mandelin's reagent,**[5] **Brociner's reagents,**[6] **Guy's or Wenzell's reagent,**[7] **Luchini's reagent,**[8] **Sonnenschein's reagent,**[9] **and Lafon's reagent**[10]—

Are all solutions of reducible compounds in sulfuric acid which, although not general reagents, give color reactions with certain alkaloids. (See Strychnin, Morphin, Atropin.)

Arnold's Reactions.[11]

(1) Coniin, nicotin, and aconitin give color reactions when heated five to fifteen minutes with syrupy phosphoric acid. (2) Color reactions are obtained with brucin, codein, colchicin, delphinin, digitalin, emetin, morphin, narcein, narcotin, papaverin, solanin, strychnin, and veratrin when a particle of the alkaloid is moderately warmed with concentrated sulfuric acid and a strong (thirty to forty per cent.) aqueous or alcoholic solution of caustic potash is gradually added. (3) If the alkaloid be dissolved in concentrated sulfuric acid and a fragment of sodium nitrite added, and afterward alcoholic or aqueous potash solution, color reactions are obtained with aconitin, atropin, brucin, codein, colchicin, delphinin, digitalin, emetin, morphin, narcein, narcotin, papaverin, solanin, strychnin, and veratrin. Another reaction, particularly characteristic of narcein, suggested by Arnold,[12] consists in heating the alkaloid with sulfuric acid and phenol.

The Furfurol Reaction.[13]

When certain alkaloids (and many other substances) are mixed

[1] Ann. d. Ch., 1861, cxx., 120. Six drops of nitric acid (sp. gr. 1.25) added to 100 c.c. water. Of this 10 drops are added to 20 grams concentrated sulfuric acid.

[2] Ann. d. Ch., 1861, cxx., 188. 0.1 gm. sodium molybdate dissolved in 10 c.c. concentrated sulfuric acid. Must be freshly prepared.

[3] The same as Fröhde's except that ammonium molybdate is used.

[4] "Berichte," 1878, xi., 1692. A saturated solution of iodic acid in concentrated sulfuric acid.

[5] Ph. Jahrb., 1883–84, 766. A solution of 1 part of ammonium vanadate in 100 parts of concentrated sulfuric acid. See also Kundrat: Chem. Ztg., xiii., 265; Ztsch. f. an. Ch., 1889, xxviii., 709.

[6] J. d. ph. et de chim., 1888, 5 s., xviii., 204; 1889, xx., 390; 1890, xx., 468. Solutions of ammonium niobate, potassium ruthenate, ammonium uranate, and ammonium tellurate in concentrated sulfuric acid.

[7] Am. J. Ph., 1861, 517; 1870, 385. A solution of 1 part of potassium permanganate in 2,000 of concentrated sulfuric acid.

[8] L'Orosi: 1885, viii., 110; Ztsch. f. an. Ch., 1886, xxv., 565. A solution of potassium dichromate in concentrated sulfuric acid.

[9] Berl. kl. Wochenschr., 1873, 310. A solution of cerosoceric oxid in sulfuric acid.

[10] C. rend. Ac. Sc., Paris, c. 1543. Also da Silva: J. d. ph. et de ch., 1891, xxiv., 102. A solution of 1 gm. sodium selenite in 20 c.c. concentrated sulfuric acid.

[11] Arch. d. Pharm., 3 R., xx., 561; Ztsch. f. an. Ch., 1884, xxiii., 228.

[12] Repert. d. an. Ch., 1882, ii., 229.

[13] Schneider: Pogg. Ann., 1872, cxlvii., 128; Ztsch. an. Ch., 1873, xii., 218. Mylius: Ztsch. f. physiol. Ch., 1887, xi., 492. Udransky:

with cane sugar and moistened with concentrated sulfuric acid, a purple red, violet, or blue color is produced, apart from the brown or black due to the sugar itself. Wender has modified the test by using furfurol and sulfuric acid directly.

Concentrated nitric acid

also causes color reactions with many alkaloids. The following are colored *red, purple,* or *red-violet:* aconitin (if impure), apomorphin, asimmin, aspidospermin, boldin, brucin, capsaicin, corydalin (slowly), cotarnin, curarin, damascenin (slowly), geissospermin, granditlorin, igasurin, loxopterigin, meconidin, pareirin, paytamin, physostigmin, porphyrin, pseudomorphin, ratanhin (if impure), thebaicin, tylophorin, vicin (after evaporation); camellin, cascarillin, chicorin, colocynthin, daphnin, daphnetin, ecbolin, gardenin, laserpitin, sinalbin, and syringin. With echitamin, *purple* changing to *green;* with echitenin *red* to *purple,* to *green,* to *yellow;* with fraxetin, *dark violet* to *red,* to *yellow.* The following give *yellow, orange,* or *brown:* aconitin (impure), anthocercin, bebeerin, berberin, caenothin, cinchonamin, codein, cryptopin (slowly), cytisin, emetin, gelsemin, hydrastin, imperialin, lanthopin, laudanin, laurotetanin, morphin, narcein, narcotin, oxyacanthin, papaverin, physostigmin, piperin, ratanhin, rhoeadin, sabadillin, sabatrin, strychnin, taxin, thebain, veratrin; agoniadin, asamic acid, chamælirin, digitalin (?), euonymin, eupatorin, gratiolin, guacin, ononin, and strophanthin. Colchicin, *violet* to *brown,* to *yellow.* Ditamin, *yellow* to *dark green,* to *orange red.* Scillain, *yellow* to *green.* Scillitoxin, *yellowish red* to *orange,* to *yellowish green.* The following give *blue* or *green* colors: aricin, calycanthin, chairamidin, chairamin, codamin, colchicein, conchairamidin, conchairamin, concusconin, cusconin, ditamin, parabuxin, solanin, and adonin. Crocin, *blue* to *brown.* Scillain, *yellow* to *green.*

The Euchlorin Reaction.

According to Fraude,[1] an aqueous solution of perchloric acid, sp. gr. 1.13–1.14 when heated with the opium or cinchona alkaloids, veratrin, caffein, atropin, nicotin, or coniin gives no color, but with aspidospermin and the strychnos alkaloids produces yellow and red tints, which are permanent and show absorption spectra. Bloxam[2] obtained similar reactions by dissolving the alkaloid in dilute nitric or hydrochloric acid, heating and adding potassium chlorate. Fraude had previously used dilute sulfuric acid and potassium chlorate.

Liebermann's Reaction.[3]

Most of the alkaloids when cautiously heated to fusion with caustic

Ibid., 1888, xii., 355, 377. Wender: Ph. Jahrb., 1893, 482.

[1] Berichte, Berlin, 1879, xii., 1558.

[2] Chem. News, 1887, lv., 155.

[3] Berichte, Berlin, 1878, xi., 1606. See also Lenz: Ztsch. f. an. Ch., 1886, xxv., 29.

potash (purified by alcohol) give no characteristic reaction—aloetin, chrysophanic acid, the cinchona alkaloids, apomorphin, sabadillin, thebain, and cocain produce blue or green colors. Ptomaïns give no color.

Other color reactions, such as *Vitali's*, *Husemann's*, *Brouardel and Boutmy's*, the *thalleiochin*, *Chastaing and Barillot's*, *Gerrard's*, *Grandeau's*, *Pellagri's*, *Robinet's* and *Roussin's*, are rather special tests for certain alkaloids than general reagents, and will be referred to in discussing the alkaloids to which they particularly apply.

The spectroscopic appearances of alkaloidal color reactions have been studied by Brasche,[1] Hartley,[2] Hock,[3] and Masche.[4]

Other Methods of Extraction of Alkaloids.

The following processes are here briefly described, because, although not generally serviceable in toxicological analysis, they may under exceptional conditions be found of use.

By Dialysis.

This method of separating alkaloidal as well as mineral poisons from the colloid constituents of organic mixtures is of most limited utility, if indeed it can be considered as in any way advantageous. It is based upon the discovery of Graham, that crystalline substances pass through animal membranes from their solutions into pure water on the other side of the membrane, while albuminoid and other non-crystalline substances do so only imperfectly, if at all.

Fig. 5.—Dialyzer.

The solution (contents of stomach, etc.) is placed in the inner vessel of the *dialyzer* (Fig. 5), which is a ring or glass vessel whose bottom consists of bladder or parchment tightly tied on while moist, in such manner as to permit of no leakage. Pure water is put into the outer vessel and the inner is floated upon or suspended in the water. The water is renewed from time to time, and, after concentration, is examined for crystalline poisons. Little is gained by this treat-

[1] Diss., Dorpat, 1891, and Ph. Ztsch. f. Russland, 1891, xxx.
[2] Chem. News, l., 287.
[3] Diss., Bern., 1882.
[4] Chem. Centbl., 1891, 1091.

ment, however, as the poison is not separated in a condition approaching purity, and the water from the external vessel must be subsequently treated by the usual methods for the further purification and identification of either mineral or organic poisons.

Graham and Hofmann's method[1]

was first adopted for the separation of strychnin from beer, and is based upon the property of freshly burnt animal charcoal of absorbing strychnin salts from their aqueous solutions and of giving the same up to alcohol. Freshly burnt animal charcoal (bone black) in coarse powder is agitated with the solution to be examined, which is then filtered off. The charcoal is washed with water and then boiled with alcohol, which is then filtered off, the alcohol removed by distillation, and the alkaloid removed from the residue by a suitable solvent.

Precipitation methods

depend upon the formation of insoluble compounds of the alkaloids, which are subsequently decomposed by suitable agents, and the alkaloid removed by the proper solvent.

Sonnenschein's method[2] consists in extracting the materials with water acidulated with hydrochloric acid, concentrating over the water-bath, again diluting, filtering, and precipitating with phosphomolybdic solution.[3] The precipitate is washed with water containing a little phosphomolybdate and potassium nitrate, and decomposed by addition of barium hydroxid to alkaline reaction. The liberated volatile alkaloids are then separated by distillation, and the fixed alkaloids by treatment with carbon dioxid, evaporation and extraction of the residue with strong alcohol.

Palm, according to Kobert,[4] has modified this method in that he extracts with water slightly acidulated with sulfuric acid, and precipitates with neutral and with basic lead acetate before precipitating with molybdic acid.

Brieger's method[5] was devised for the extraction of ptomaïns, and is also essentially a modification of Sonnenschein's. The materials are extracted with water acidulated with hydrochloric acid, heated to boiling, filtered, and evaporated to a syrup. This is extracted with alcohol (ninety-six per cent.) and the filtered extract treated with alcoholic solution of lead acetate. The filtrate from the precipitate formed is evaporated to a syrup, again extracted with alcohol, the alcohol evaporated, the residue dissolved in water and freed from lead by hydrogen sulfid. After addition of a little hydrochloric acid the liquid is again concentrated to a syrup, which is extracted with alcohol and the solution precipitated with alcoholic solution of mercuric

[1] Ph. J. and Tr., 1851–52, xi., 504.
[2] Ann. d. Chem., 1857. civ., 45, and "Ger. Chem.," p. 329.
[3] See p. 139.
[4] "Intoxikationen," p. 95.
[5] "Untersuch. ü. Ptomaine," 1886, 3 Th., p. 19.

chlorid. The filtrate from the precipitate formed is freed from alcohol by evaporation after the acid has been nearly completely neutralized with soda, and from mercury by solution in water and treatment with hydrogen sulfid. This aqueous solution is concentrated, the residue extracted with alcohol, the alcohol expelled, the residue dissolved in water, the hydrochloric acid neutralized with soda, the solution acidulated with nitric acid and precipitated with phosphomolybdic acid.

The alkaloids contained in the mercuric and phosphomolybdic precipitates are recovered by decomposition of the compounds, and extraction of the liberated alkaloids by suitable solvents.

The precipitation methods, although they have rendered valuable service for purposes of investigation, are not adapted to forensic analysis, partly by reason of the number of reagents, some of which are poisonous, which are introduced into the materials under examination, partly because of the liability to decomposition of glucosids and certain alkaloids by the reagents used, and partly because they present no advantage over the extraction methods above described, either in facility or rapidity of manipulation, or of separation of alkaloids from ptomaïns.

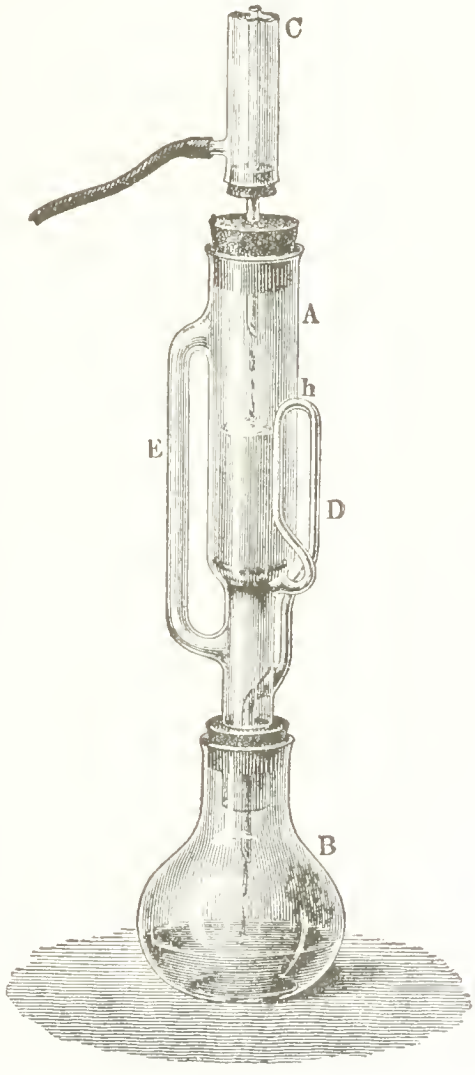

Fig. 6.—Soxhlet's Extraction Apparatus.

By Direct Extraction.

The direct extraction of alkaloids and other vegetable poisons by the action of a solvent, water, dilute acid, dilute alkalies, alcohol, ether, chloroform, petroleum ether, benzene, etc., upon the dried or semi-solid material can rarely be resorted to in forensic analysis. Occasionally, however, this method may be of service in the examination of an article of food or a medicine, particularly when the search is limited to one poison. The solvent selected should be that in which the materials with which the poison sought for is mixed are the least soluble, and which is yet capable of dissolving the substance to be extracted. With aqueous solvents, solution and separation are effected by tritura-

tion, agitation, and filtration. With solvents not miscible with water the materials must be previously dried at as low a temperature as possible. When solvents having a lower boiling point than water are used, the Soxhlet (Fig. 6), or other form of extraction apparatus permits of a complete extraction with a small quantity of solvent. The dried material is packed in a paper cylinder, which is placed in the tube A; the solvent is put into the flask B, which is heated on or in the water-bath. The solvent is volatilized, passes through E, is condensed in the condenser C, accumulates in A until it rises to the point *h*, when it flows through the siphon D back into the flask B, in which the substances extracted by it accumulate.

Micro-sublimation [1]

consists of applying heat to the substance in a "subliming cell," in such manner that the change in appearance and the temperature may be noted, and the sublimate collected and examined with the microscope. While some of the alkaloids, when examined in the crystalline form by this method, produce appearances which may serve to aid in their identification, indications of value cannot be obtained when the alkaloid is mixed with other substances.

Examination for Mineral Poisons.

The poisons included in this class are the compounds of arsenic, antimony, barium, bismuth, chromium, copper, lead, mercury, silver, tin, and zinc. The salts of other metals, such as those of cadmium, thallium, and uranium, are also poisonous, but by reason of their rarity have little practical interest, and if present, would attract the attention of the analyst in the course of the investigation.

The **first step** in the search for the mineral poisons is the destruction, as complete as possible, or the removal of the organic substances of which the tissues and food articles are largely composed.

The most rapid method of accomplishing this object is by simple incineration, but it is not available in searching for arsenic and mercury, which would be lost by volatilization. It may, however, be occasionally used when only a fixed metal is to be sought for. The dried and finely divided substance is gradually heated in a porcelain crucible, at first covered, after-

[1] Helwig: "Das Mikroskop in der Toxikologie," 1865, ii., 8. Guy: Ph. J. and Tr., 2 s., 1866–67, viii., 719; 1867–68: ix., 10, 58. "Forensic Med.," 6th ed., 1888, 398, 463, 509. Blyth: "Poisons," 1884, 240.

ward open, until converted into an ash which, after cooling, is extracted with dilute hydrochloric or nitric acid.

A modification of the method by incineration which permits also of the detection of the volatile metals has been suggested by **Verryken,**[1] but is only applicable to the treatment of small quantities of material. From 5 to 10 gm. of the perfectly dried material are very gradually heated in a current of oxygen in a combustion tube whose free end communicates with a Liebig bulb apparatus containing water, to arrest the volatilized arsenic or mercury.

Incineration in the ordinary method is facilitated by previously mixing the dried and powdered material with potassium nitrate, and adding the mixture in small portions to a red-hot porcelain crucible. The cooled melt is extracted with hot water.

The method of Wöhler and von Siebold[2] is an improvement upon the method by incineration, which permits of the detection of mineral poisons other than mercury, which is lost by volatilization. The materials are first partially oxidized by heating with strong nitric acid, neutralized with caustic potash or potassium carbonate, and dried at about 120° C. The dried mass, consisting of the partly oxidized organic matter and potassium nitrate, is then brought in small quantities at a time into a red-hot porcelain crucible, and the cooled melt is extracted with boiling water.

Method of Fresenius and von Babo.—This is the most frequently used and the best process hitherto devised, and effects the desired end by the oxidizing action of nascent chlorin generated in a liquid medium.

The substances to be examined are finely hashed if solid and freed from alcohol, if it be present, by evaporation on the water-bath after neutralization of any acid reaction with sodium carbonate. They are brought into a flask and hydrochloric acid and potassium chlorate added to the extent of about 25 c.c. of the former and 2 gm. of the latter for every 100 gm. of solid to be disintegrated. If the mass be not fluid, a sufficient amount of water is added. The mixture is al-

[1] J. de pharm. d'Anvers, 1872, 193, 241. Chem. Centlb., 1873, 823.

[2] Siebold: "Lehrb. d. ger. Med.," 1847, 494. Also Wöhler and v. Siebold: Reprint.

lowed to stand for twenty-four hours with occasional stirring, after which it is heated on the water-bath, with frequent agitation and the occasional addition of potassium chlorate, until the contents of the flask are yellow and fluid, except for fatty and white granular matter in suspension, and until the liquid does not darken on heating for half an hour without addition of chlorate. If, during the heating, the addition of chlorate produces little or no effect the quantity of hydrochloric acid is deficient, and more is to be added in the proportion of 10 c.c. to each 100 c.c. of original solid. The quantity of both acid and chlorate used should, however, be kept at the minimum required to accomplish the object. When the disorganization is completed the excess of chlorin is expelled by passing a rapid current of carbon dioxid through the warmed liquid. The solution is then filtered hot through a filter moistened with water, and the residue washed with hot water. Should a large quantity of fat remain, it is to be heated two or three times with water acidulated with hydrochloric acid, and the filtered extracts added to the main solution. The filtered liquid now contains in solution any of the mineral poisons which may be present except silver and a portion of the lead, if the latter have been present in large amount; these will remain in the undissolved residue.[1] (For the further examination of solution and residue see below, p. 157).

The objections to this method are: 1st. That a small and uncertain proportion of arsenic is lost by volatilization as arsenic trichlorid. 2d. That the destruction of organic matter is not complete, and that a portion of the mineral material, including arsenic and other poisons, is retained by the fat which remains. 3d. The large quantity of chlorate and hydrochloric acid introduced, and the difficulty of obtaining the latter free from arsenic.

The first objection may be overcome by operating in a retort or flask connected with a condenser and absorbing apparatus to retain any arsenic trichlorid which may be formed, and adding

[1] If mercury be present in the metallic form it is only very slowly dissolved and may remain as a metallic deposit if the decomposition have been rapidly effected. Lecco: Berichte, Berlin, 1891, xxiv., 928. Extraction of mercuric chlorid is also only satisfactorily effected if the disintegration is carried so far that no granular, yellow particles of undecomposed material remain. See Ludwig: Ztschr. f. an. Chem., 1891, xxx., 664.

the chlorate in saturated solution through a small separatory funnel. The second objection is only serious so far as the retention of poisons by the fat is concerned, and, if the amount of these be large, repeated washings do not effect complete extraction; yet the percentage retained is very small and only affects quantitative determinations of doubtful utility. The quantities of acid and chlorate used may be diminished by following Ogier's modification below described. Pure hydrochloric acid is, so far as we know, not purchasable, and must be manufactured or purified by the analyst himself.[1]

Ogier's[2] modification consists in making a mixture of the finely hashed viscera with one-tenth to one-eighth of their weight of potassium chlorate and enough water to form a rather thick liquid, and passing hydrochloric acid gas through the mixture in a specially constructed apparatus.

Method of Jeserich.[3]—This process is also based upon the decomposing action of nascent chlorin. The finely divided material is made into a thin paste with water, to which chloric acid is then added in small portions; after which the mixture is slowly and cautiously heated on the water-bath until the mass has assumed a spongy consistence, when hydrochloric acid is gradually added in small quantities. The attack is complete in two to three hours, and yields an almost clear yellowish liquid, upon which floats a layer of oil. The remaining treatment is the same as with the method of Fresenius and von Babo.

[1] The usual pharmacopœial tests for purity are not sufficiently delicate for the examination of hydrochloric acid intended for this use. It should be tested as follows: At least one litre of the acid is diluted with water to specific gravity 1.1; a small quantity of potassium chlorate is added, and the acid evaporated in a porcelain capsule on the water-bath with occasional addition of water. The residue is taken up with dilute sulfuric acid and heated until the hydrochloric acid is expelled, after which the liquid is introduced with the usual precautions into a Marsh apparatus. No stain whatever should be produced.

A sufficiently arsenic-free acid is obtained either by the action of arsenic-free sulfuric acid on fused sodium chlorid, which is tedious and costly, or by the purification of the commercial product by Bannow's method. The commercial acid (containing iron) is diluted to specific gravity 1.1 and treated with hydrogen sulfid at the ordinary temperature and during stirring, until it just smells of the gas, but no longer. It is then allowed to stand for several days, again treated with hydrogen sulfid, and again allowed to stand until clear. It is then carefully filtered and the filtrate submitted to fractional distillation, the first forty per cent. and the last ten per cent. being rejected.

[2] "Le Lab. de Tox.," Paris, 1891, 44.

[3] Repert. d. an. Chem., 1882, ii., 369.

Method of Gautier.[1]—This method depends upon the disintegrating action of sulfuric and nitric acids: 100 gm. of the hashed material are put into a capsule of a capacity of 600 c.c.; 30 c.c. of nitric acid are added, and the mixture is heated moderately. The substance liquefies gradually and assumes an orange tint; when this occurs the capsule is removed from the fire and 5 gm. of sulfuric acid are added, when the mass becomes brown and is strongly attacked. It is then heated until fumes of sulfuric acid are given off, when 10 to 12 gm. of nitric acid are added, drop by drop. The mass again becomes liquid and gives off abundant nitrous fumes. When all the acid has been added the mass is heated to beginning carbonization. Finally the carbonaceous residue is pulverized and extracted with boiling water.

Method of Chittenden and Donaldson.[2]—This is practically the same as the last described, except that the temperatures are lower and more carefully regulated.

It is claimed for the two methods last described that the extraction of arsenic is more complete than by that of Fresenius and von Babo; but they are open to the objections that, even with the most careful handling, the oxidation occasionally progresses with such rapidity as to cause deflagration and the loss of a large proportion of the material, and that any mercury present is lost.

The Method of Flandin and Danger,[3] by carbonization with sulfuric acid, and a small quantity of aqua regia subsequently added, is attended with loss of arsenic as chlorid.

Method of Pouchet.[4]—In this process the danger of deflagration is sought to be avoided by the use of monopotassic sulfate. The material to be decomposed is placed in a large porcelain dish with twenty per cent. of its weight of monopotassic sulfate, and its own weight of fuming nitric acid is added. The reaction, very violent at first, is subsequently aided by gentle elevation of temperature. The mass becomes

[1] Bull. Soc. de chim. de Paris, 1875, xxiv., 252. This method is a combination and perfection of that of Orfila (1839): "Tr. de Tox.," 5th ed., i., 494, and of the modification thereof suggested by Filhol in 1848 ("Études sur l'Arsenic," Th., Paris).

[2] Am. Chem. Jour., 1880–81, ii., 236.

[3] Flandin: "Tr. d. Poisons," i., 618.

[4] Comp. rend. Ac. Sc., Paris, 1881. Legrand du Saulle, Berryer, and Pouchet: "Méd. Lég.," 1424.

carbonized and porous. It is moistened with fuming nitric acid, and heated until nitrous fumes are given off. The carbonized mass is pulverized and extracted with water strongly acidulated with hydrochloric acid at a boiling temperature.

The **second stage** in the analysis for mineral poisons consists in their separation from the solution or residue obtained by some one of the foregoing methods.

If the disintegration has been effected by the method of Fresenius and von Babo, the portion remaining undissolved may contain silver, lead, or thallium as chlorids, mercury as sulfid or metal, and barium as sulfate.

If the liquid, clear when filtered, becomes turbid on cooling, the deposit so formed may contain lead, silver, or thallium as chlorids, or compounds of antimony or bismuth. The above insoluble portion (I.) and any deposit formed on cooling (II.), separated by filtration, are set aside for subsequent examination, and the clear, filtered liquid is treated as follows: Any great excess of acid is neutralized with sodium carbonate to such an extent that the reaction still remains distinctly acid, and the liquid is treated with sulfur dioxid until it smells strongly of the gas after standing in a closed vessel for some hours.[1] The excess of sulfur dioxid is then completely expelled by boiling the liquid. The solution is then treated at a temperature not exceeding 50° (120° F.), with hydrogen sulfid gas for about two hours, and subsequently at periods of about half an hour at intervals of three or four hours until the liquid becomes perfectly clear above the precipitate and smells strongly of hydrogen sulfid at the expiration of the interval. During the later treatment with hydrogen sulfid the liquid should be cold.[2]

[1] The treatment with SO_2, intended to reduce the arsenic compounds present to arsenous, prevents the separation of a large amount of sulfur in the subsequent treatment, and, while highly desirable if arsenic be present alone, is absolutely essential to avoid loss of mercury later if that metal be present along with arsenic.

[2] To obtain hydrogen sulfid free from hydrogen arsenid it should be prepared by the action of dilute (1 : 9) arsenic-free sulfuric acid upon fused ferric sulfid in a Kipp's generator (Fig. 2 *c*., p. 128) ; and purified from possible traces of arsenic derived from the ferric sulfid by Jacobsen's method (Berichte, Berlin, 1887, xx., 1999) : by passing the gas first over calcium chlorid, then through a tube about 40 cm. long loosely filled with solid iodin, and finally through a wash bottle containing a solution of potassium sulfid. A valve bottle, permitting entrance of air but not escape of gas, should be interposed between the generator and the calcium chlorid tube.

After complete precipitation the precipitate (III.), which may contain arsenic, tin, cadmium, antimony, gold, bismuth, mercury, silver, lead, or copper, is separated from the filtrate (IV.), which may contain zinc, nickel, cobalt, thallium, iron, manganese, chromium, or aluminum, by filtration through a weighed filter.

The precipitate (III.) on the filter is washed three or four times with water containing some hydrogen sulfid, and treated on the filter with hot ammonium monosulfid.[1] The filtrate, which in the presence of mercury is at first clear but afterwards deposits a sediment, is digested on the water-bath for ten minutes, and, should any precipitate form, is again passed through the filter originally used and containing any undissolved sulfids. The precipitate is washed once with hot, undiluted ammonium monosulfid, and the filtrate digested (if a precipitate was formed in the first extract) as above. When no further precipitation is observed in the washing, the sulfids on the filter are thoroughly washed with hot water.[2]

The extracts by ammonium sulfid, and the washings (IV.), which will contain all the arsenic, antimony, gold,[3] and tin, and a portion of the copper, are united and treated separately from the sulfids insoluble in ammonium sulfid (V.).

The liquid (IV.) is acidulated with hydrochloric acid, and the reprecipitated sulfids are, after subsidence, collected on a filter and washed with water containing hydrogen sulfid until a sample of the washings no longer gives any cloudiness when boiled and treated with nitric acid and silver nitrate. The point of the filter is then perforated and the precipitate washed into a porcelain capsule, the last adherent portions being dissolved with a small quantity of dilute ammonium sulfid solution. The contents of the capsule are dried, moistened with

[1] Prepared by saturating ammonium hydroxid solution (sp. gr. 0.96) with hydrogen sulfid until it no longer precipitates with magnesium sulfate, and adding an equal volume of ammonium hydroxid solution (sp. gr. 0.96). The use of ammonium sulfids otherwise prepared will entail loss of mercury if it be present.

[2] We have found that unless the operation be conducted precisely as described, there may be considerable loss of mercury, if it be present along with arsenic, by its passage into the solution and volatilization in the subsequent treatment. The entire operation must also be conducted without interruption.

[3] If gold is to be taken into account it should be searched for at this point by immersing a bar of pure zinc in the ammonium sulfid solution for about twelve hours. The zinc becomes plated with gold.

fuming nitric acid, and evaporated to dryness, and then moistened with water and evaporated to dryness two or three times. The residue is treated with a small quantity of water; sufficient sodium hydroxid (free from carbonate) to render the reaction alkaline is added, and then a mixture of dry sodium carbonate and nitrate (1 : 2), and the whole evaporated to dryness after thorough mixing.[1] When dry, the residue is gradually and cautiously heated to fusion and the heating continued until the mass is colorless or faintly greenish, or until the only colored portion consists of a heavy black granular powder (cupric oxid). If, after continued heating, the fused mass remains gray, the quantity of sodium nitrate is insufficient and more must be added. During this treatment arsenic is converted into the soluble sodium arsenate, antimony into the almost insoluble sodium pyroantimonate, and tin and copper into insoluble oxids. The fused mass is then extracted with warm water, carbon dioxid is bubbled through the solution, an equal volume of alcohol is added, and the liquid is filtered if not perfectly clear. The insoluble portion (VI.), collected on the filter, is examined for antimony, tin, and copper in the manner described under "Antimony." The solution (VII.) is evaporated to dryness, and the cold residue is treated with sufficient concentrated sulfuric acid and gradually heated until dense white fumes are given off. The cooled residue, diluted with water and again cooled, is now ready for the application of the Marsh test as described under "Arsenic."[2]

Returning now to the portion of the precipitate insoluble in ammonium monosulfid (VI.) upon the weighed filter: this, with the filter, is dried and weighed. The metallic poisons which may be present are cadmium, bismuth, mercury, lead, and a portion of the copper. They are treated with hot dilute nitric acid; mercury, if present, remains undissolved as the black mercuric sulfid, which is further examined as directed under "Mercury." The others, if present, pass into solution. The solution is filtered from precipitated sulfur, evaporated

[1] The sodium hydroxid used should be prepared from metallic sodium (that from other sources is most frequently arsenical), and the nitrate and carbonate used should be free from traces of chlorid.

[2] Experiments made with sodium peroxid to effect these separations have thus far proved unsatisfactory in our hands.

nearly to dryness, and diluted with water. A small portion of the solution is treated with dilute sulfuric acid: a white precipitate is produced if lead be present. If lead be present, dilute sulfuric acid is added to the whole of the solution, which is evaporated until nitric fumes are given off, diluted with water, and filtered immediately from the lead sulfate. The liquid, freed from lead, is rendered alkaline with ammonium hydroxid; if bismuth be present, it is precipitated, and is then separated by filtration. In the presence of copper the filtrate is usually blue, but very small quantities of copper should be tested for by acidulation with acetic acid and addition of potassium ferrocyanid. Cadmium is detected by the formation of a yellow precipitate in the solution, freed from copper and acidulated with hydrochloric acid, by hydrogen sulfid.

The metals not precipitated by hydrogen sulfid from acid solution (IV.) are rarely of forensic interest, but occasionally zinc, aluminium, iron, chromium, or barium are to be sought for. The filtrate and washings from the precipitate by hydrogen sulfid are concentrated by evaporation, treated with ammonium hydroxid and ammonium sulfid, and set aside in a well-corked flask. A more or less colored precipitate is produced, which may contain besides iron, aluminium, zinc, or chromium (VIII.). This is collected on a filter, washed with water, and examined as below. The filtrate (IX.) will contain any barium which may have been present.

The precipitate (VIII.) is dissolved in a small quantity of hydrochloric acid, diluted with water, the acid reaction nearly but not quite neutralized with sodium carbonate, and the liquid left in contact with finely divided barium carbonate for twenty-four hours. The liquid (VIII*a*), which will contain any zinc present, is filtered off and examined for that metal as described in the next division. The solid residue, which contains the oxids of the other metals named, is treated with caustic soda solution, boiled in a silver basin, and filtered. The filtrate (IX.) is examined for aluminium, and the residue (X.) for iron or chromium if necessary.

The material left undissolved after the attack by hydrochloric acid and potassium chlorate (I.) may still contain silver, thallium, and lead.[1] The presence of the last will have been

[1] See also under Mercury, Analysis.

detected, as above indicated, by the portion which has dissolved. Thallium is best detected by spectroscopic examination of the ash; and the method for the detection of silver will be discussed in the next division.

PHYSIOLOGICAL EXAMINATION—LIFE TESTS.

Observations of the effects of poisons upon lower organisms and upon man may be made for three distinct purposes: 1st. In the study of pharmacodynamics, with the object of determining the method of action of the drug or poison, and the nature of its effects upon the tissues, fluids, and organs of the body. 2d. In the investigations of forensic toxicology, for the purpose of identifying a poison by observation of effects which it is known to produce upon living organs, tissues, or fluids. 3d. In experimental toxicology, for the purpose of investigating such questions as the rate of absorption and elimination, post-mortem imbibition, etc.

Experimental observations of the effects of drugs and poisons upon all forms of life, from bacteria to the human subject, are very numerous and, combined with the observations of the clinician, constitute in great part the foundation of modern rational therapeutics, notwithstanding the well-known fact that a given drug or poison may, and frequently does, act differently or with varying degrees of intensity upon different forms of life. Consequently the conclusion can never be directly drawn that effects observed in a lower form of life will be produced by the same agent in man, whether taken in proportional dose or in any other quantity. Very valuable indications of the probable action on man have been, however, obtained by this means, which should never be omitted as a preliminary to the use of a newly discovered remedial agent upon the human subject.

Although the investigations of pharmacodynamics are necessarily the antecedents which furnish those data upon which life tests applied for forensic purposes depend, they do not come strictly within the limits of a work of this character.[1]

The application of life tests to the purposes of forensic toxi-

[1] For an extended account of the methods of experimentation see Kobert: "Intoxikationen," pp. 108–200. Also Hermann: "Experimental Pharmacology," Phila., 1883. Blyth: "Poisons," pp. 38–52.

cology is in its simplest form of great antiquity. Articles of food supposed to contain poison were administered to dogs and other animals to verify or disprove the suspicion,[1] rulers fearing poison caused their food and drink to be tasted by another before partaking of them themselves,[2] and instances are recorded in which suspected poisoners were tried and executed at the same time by being compelled to take their own preparations.[3]

The use of physiological reactions in forensic toxicology for the purpose of identifying a poison, although of the first importance in a few cases in which such tests afford distinct indications, is limited. The mineral poisons are separable and capable of positive identification by chemical reactions, even when present in much less quantity than that required to produce any visible effects upon a living organism. It is only to the identification of certain vegetable poisons, such as strychnin, atropin, digitalis, aconite, and curare, which produce characteristic symptoms in animals peculiarly sensitive to their action, that life tests are at present applicable. If the materials under examination have undergone putrefaction, physiological tests are only of value when corroborated by chemical reactions, as ptomains capable of causing mydriasis, tetanic spasms, etc., have been found to exist. On the other hand, physiological reactions may aid in some cases in differentiating a putrid alkaloid from one of vegetable origin similar in some other respects. With some vegetable poisons, such as aconite, physiological tests are more delicate and reliable than any chemical reactions at present known.

In applying physiological tests it is essential that the substance examined shall be previously separated in as pure a form as possible, and shall be in solution in an inert vehicle as nearly neutral as may be. Consequently life tests can only be used in searching for poisons in dead bodies, etc., after extraction by the chemical methods above described.

Collateral evidence is also sometimes accidentally furnished by animals, which is particularly valuable in cases of unsuccess-

[1] See Introduction, p. 6.

[2] Xenophon: "Cyropædia," i., 3, T. iii., p. 12, ed. Paris, 1842.

[3] Livius: "Hist.," viii., 18. An instance is reported to have occurred in Missouri in 1888, in which a negress was forced to drink coffee in which she had poisoned a family with strychnin, and died in half an hour (New York Times, June 4th, 1888).

ful attempts to poison. Thus in the case of Reg. *v.* Newton, reported by Taylor,[1] the intended victim vomited in the back yard, where some fowls were subsequently observed to be ill and two died. Arsenic found in the crops of the chickens furnished the only evidence of administration, as the person poisoned recovered, and the poisoner had thrown away the remainder of the food supposed to have been poisoned, and had cleaned out the vessel. In a somewhat similar case, reported by Maschka,[2] a woman recovered slowly from poisoning by arsenic, of which five grains were found in her vomit and one and a half grains in the body of a hen that was poisoned by eating of it. Numerous similar cases are reported.

FORENSIC QUESTIONS.

1. Was the death or disorder caused by poison? This, one of the three fundamental questions to be passed upon by the jury in cases of alleged homicide or injury by poison, is the main one to be answered by expert evidence, and to it all others are subsidiary.

Leaving out of consideration the moral evidence, which does not concern the expert, the proof of poisoning is complete when the symptoms known to be caused by the poison have been observed during life; when the post-mortem examination shows the presence of such lesions as it is capable of producing, and the absence of other causes of death; and when the toxic agent is demonstrated to be present in the cadaver, or in the vomit or dejecta of the person poisoned.

And even when other causes of death exist they do not of necessity negative the theory of poisoning. If poison be shown to be present in the cadaver, or to have been administered, and marks of violence or evidence of disease sufficient to cause death be found at the autopsy, it clearly remains to be determined which of the two or more causes of death known to have existed was the one finally operative; and it is equally incontestable that the existence of any possible cause of death cannot be invoked as conclusive proof that no other could have existed.[3]

[1] "Poisons," 3d Am. ed., 167.

[2] Gutacht. Prag. med. Fak., 1867, iii., 269-271.

[3] This apparently self-evident proposition is stated because we have known experts to invoke the

In such cases it is frequently most difficult to determine which was the preponderating or final cause of death. Did Mary Stannard die of the arsenic found in her stomach and tissues, or in consequence of having her head crushed with a stone? Was Jennie Cramer's death due to the arsenic found in her cadaver, or was she drowned in the water from which it was taken? Was the finally operative cause of Fisk's death a gunshot wound or an overdose of morphin? These were vital questions, to be determined in each case to the best of their ability by the jury in the light of the expert evidence offered.

Indeed, an anatomical lesion which is in itself sufficient explanation of the method of death may have been produced by poison as a cause at least concomitant, as in the case of a man who fell dead while retching violently—a large clot was found on the brain and oxalic acid in the stomach. The apoplexy of which the man died was provoked by the effects of the oxalic acid taken.[1]

A discussion of the relative force of the three factors of the proof would be idle, as no one of them, when taken alone and unsupported by other evidence, can demonstrate conclusively that poison was the cause of death. It is quite conceivable, however, that collateral evidence of fact may exist, sufficient to enable the jury to find that the deceased died from the action of poison, when but one of the factors of expert proof can be adduced. Thus in a case tried in Vermont in 1883 two defendants were convicted of murder in the first degree upon moral evidence, supported only by the detection of strychnin in the cadaver,[2] without any history of symptoms or the existence of characteristic lesions. In such cases, however, the expert

existence of mechanical causes of death as proof that the results of the chemical analysis must have been fallacious (see Morphin).

[1] Lancet, 1863, i., 47.

[2] State *v.* Emeline and Almon Meeker. This case, exceptional in many respects, has not been reported. The mother and son were convicted of poisoning a twelve-year-old girl, the half-sister of the former's husband, by strychnin, administered to the child while driving in a buggy during the night. The purchase of strychnin and the hiring of the buggy by the son were proved. The child's body was buried in a swamp. After some days the sheriff extorted a confession from the son, with whom he went to the swamp and recovered the body. The defendants were jointly indicted and the son pleaded guilty to murder in the first degree to prevent the introduction of his confession upon the trial. The mother was executed, the son's sentence commuted to life imprisonment. No motive for the crime was suggested by the prosecution during the trial.

should not be asked to express an opinion as to the cause of death, as the evidence is insufficient for the formation of a purely expert opinion, and such medical or chemical evidence as may be procurable should be simply submitted to the jury, to be by them weighed along with the moral evidence.

On the other hand, instances are numerous in which a positive expert opinion can be formed in the absence of one of the three factors. The symptomatology and lesions produced by the mineral acids and alkalies are sufficiently characteristic to indicate the cause of death without any chemical analysis beyond the mere determination of the reaction. The recognition of death by hydrocyanic acid may be reached, partly by exclusion, from the absence of all other causes of sudden death, and the presence of the poison in the cadaver, even when the body has been found dead and no symptoms were observed during life. Many poisons, such as strychnin, leave no physical evidence of their action, and in cases of poisoning by them the evidence from the autopsy is of negative value only, in excluding other possible causes of death. And in this connection also it must not be forgotten that persons in middle age are very rarely perfectly normal, and that a person suffering from even serious disease may die of poison.

When the absence of any portion of the facts upon which an expert opinion of the cause of death might be based is due to accidental failure, as when a person is found dead or when the body has been cremated, the failure to present such evidence on the part of the state should not count for or against the theory of poisoning. When it is due to interference with the cadaver, as when the body has been eviscerated and the organs made away with, or the body has been clandestinely burnt before a post-mortem could be made, the detection of poison in the unembalmed fragments that may remain is strongly indicative of criminal poisoning.[1] But if the circumstances be such that the lacking evidence should have been obtained if poisoning was the cause of death, as when a person dies within a short time and with the symptoms of strychnin poisoning, and a properly conducted analysis, made shortly after death, fails to show the presence of the poison, the theory of death from that partic-

[1] See the Shann case, previously referred to (p. 115, note), and the Pel case, under Arsenic.

ular poison becomes much more difficult of proof, if it be tenable at all.

It is hardly necessary to state that in the framing of the hypothetical question terminating in "What in your opinion was the cause of death," which is usually put to medical expert witnesses, the results of the autopsy, of the pathological examination, and of the chemical analysis should be included along with the history of the symptoms.

In non-fatal cases a positive diagnosis can only be reached by the observation of the symptoms and the proof of the presence of the poison in the excreta, vomit, or substances known to have been taken or administered. An adequate cause for the effects observed must be affirmatively proved to exist, either by lay or by expert evidence.

2. Could the poisoning have been simulated? Every toxicologist has been annoyed by persons having a fixed delusion that they are in danger of being poisoned. These persons can hardly be said to simulate poisoning, not only because the delusion is honestly entertained, but because, although they submit various articles of food or drink for analysis, and describe real or fancied symptoms, the analysis always leads to negative results, and the symptoms differ more or less widely from those of poisoning.

The symptomatology of poisoning very frequently closely resembles that of some so-called natural disease so closely that a differential diagnosis from the symptoms alone is difficult or impossible. These resemblances very frequently give rise to groundless suspicion of poisoning, which, if the person die, may either be immediately disproved by the discovery at the autopsy of some lesion adequate to cause the effects observed during life, or add to the list of "deaths from unknown causes," if neither lesion nor poison be found.

The possibility of the introduction of poison into a cadaver for the purpose of supporting a false accusation of criminal poisoning was recognized by Orfila as early as 1815.[1] Although the physical possibility of the commission of such a crime cannot be denied, we know of no instance in which it has been alleged either in accusation or as a defence. The non-criminal

[1] "Traité des poisons," Paris, 5ème ed., Paris, 1852, i., 61. 1814–15, II., ii., 293. "Tr. d. tox.,"

post-mortem introduction of poison is of frequent occurrence and will be discussed below.[1] The resemblance of the symptoms of poisoning to those of disease has been taken advantage of by poisoners to destroy their victims, with less probability of detection during the prevalence of an epidemic. Several such cases are referred to under arsenic, and Kobert[2] cites an instance in which a man was poisoned by tartar emetic during an epidemic of cholera, the poisoner thinking that the death would be attributed to the prevailing disease.

Cases of deliberate false accusations of attempted poisoning, made by persons still living, have also occurred; and it is quite conceivable that poison may be mixed with articles of food, medicine, urine, or even with vomited matters in support of the charge. Or an innocent person may be placed in serious jeopardy by the suicidal or accidental poisoning of relative, employer, etc., affected during life with the delusion of poisoning. An instance of this kind, in which the suspected person only escaped punishment in consequence of the expression by the medical experts of the opinion that arsenic cannot remain in the body for eight days without manifesting any of its usual effects and then cause death, is reported by Boutigny:[3] A woman accused her husband of attempting to poison her, and coarsely powdered arsenic was found in a dish of food alleged by her to have been prepared by her husband, who was then imprisoned. The woman at that time and for eight days after was perfectly well. She then had an attack of mania and died on the ninth day after the alleged administration by the husband, who had remained in prison. Arsenic in large quantity was found in the stomach and intestines.

3. Can a poisoning have occurred and the poison either be or have become undetectable? This question must receive an affirmative reply as to its first part for many poisons, and as to its second part for all. There are many vegetable poisons which leave no characteristic lesions, and whose chemical reactions and action upon animals, so far as known, are not sufficiently well marked to permit of their identification, even when they are separated in the minute quantities in which

[1] See Question 12 and Arsenic in Special Toxicology.

[2] "Intoxikationen," p. 83.

[3] Ann. d'hyg., etc., 1836, xvi., 391.

they may remain in the body after death from their action. And particularly when the body has undergone putrefaction, even in its earlier stages, their distinction from putrid alkaloids, which may have been formed is, in the present condition of our knowledge, impossible. Indeed, the number of alkaloidal poisons which can be distinguished from putrid products by physiological and chemical tests is quite small.[1]

And even poisons having distinctive chemical characters which ordinarily permit of their identification with certainty, may become undetectable from several causes. The person may die from secondary effects long after the first action has disappeared, as in corrosion by the mineral acids; or life may be prolonged for a sufficient length of time to permit the complete elimination of the poison and death still result from its action, by exhaustion or by a continuation of morbid processes which it established. This has been known to occur even with a poison usually so prompt in its action and of such certainty of detection as arsenic.[2]

Poisons are removed from the stomach and intestines by vomiting, purging, and absorption, as well as by washing out the stomach, if that method of treatment be resorted to; and it is quite conceivable that even an easily recognizable poison may have been thus entirely removed from these parts while it still remains in detectable quantities in the tissues into which it has passed by absorption and from which it is subsequently removed by elimination if life continue. The period of time required for absorption, elimination, etc., is different for different poisons, and varies with each according to the conditions under which it acts. This subject will be more particularly discussed in treating of the poisons individually. The possibility of washing out the stomach after death must also be recognized, although the probability of its suggesting itself to any other than a medical poisoner is remote. The operation would of course leave no traces except the presence, possibly, of an undue quantity of water, and the absence of all remains of food in the stomach, which would also tend to aid a simulation of death from drowning. Of course, poisons such as arsenic would remain in the tissue of the organ.[3]

[1] See in this connection Ptomains, Morphin, Strychnin, Atropin.

[2] See Arsenic—Elimination.

[3] An instance in which the

When a chemical antidote has been administered during life the poison or corrosive will be found in that form of combination produced by the reaction, possibly accompanied by excess of the antidote. Thus the contents of the stomach may be neutral or even alkaline when acids have been taken and alkalies administered.

Poison remaining in the body at death, if it be organic in nature, or volatile, or oxidizable, may either disappear or be converted into other products. Thus prussic acid in contact with the ammonia and hydrogen sulfid produced during putrefaction may be converted into ammonium thiocyanate, a normal constituent, in small amount, of the saliva. Prussic acid and chloroform, even when present in sufficient quantity to be recognizable by their characteristic odors at the autopsy, are rapidly dissipated by volatilization if the materials be left exposed to the air. Phosphorus is gradually oxidized, and is finally converted into phosphates, not distinguishable from those normally present in the body.

4. What poison produced the injury or death? As we have said above, the occurrence of symptoms which may be produced by a certain poison and the satisfactory demonstration of the fact that the kind of poison capable of producing such effects was taken by or administered to the injured person constitute, in the absence of other causes capable of producing such results, sufficient evidence that the agent shown to have been present was the cause of the effects observed. Yet a pernicious impression exists in the popular mind, and to some extent among toxicologists, that the poison must be actually isolated and produced in substance upon the trial. The poison so separated from the cadaver is even falsely designated as the *corpus delicti*, a term which applies to the crime and not to the material agent.[1] A shooting may be satisfactorily proved without the production upon the trial of either the pistol or the bullet, and a stabbing without the exhibition to the jury of the cutting instrument with which the wound was inflicted. Why in a

stomach was opened and sponged out in the interval between two autopsies is alleged to have occurred in Wisconsin in 1885. Arsenic was found in the tissue (New York Times, January 9th, 1886).

[1] "The *corpus delicti* in homicide has two components, namely, death as the result and the criminal agency of another as the cause." Peo. *v.* Bennett, 49 N. Y., 137.

case of poisoning should the exceptional demand for the actual isolation and production of the material agent which caused the injury be insisted upon? That such material proof is desirable, if it can be obtained, is unquestionably true in the one case as it is in the other, but it is frequently much more difficult to obtain in poisoning than in homicide by mechanical means, or is not obtainable.

Indeed, it is most exceptional that the poison is recoverable in the identical form in which it was administered. Phosphorus may be separated as such in some cases, and preserved by sealing in a glass tube, and arsenic trioxid may occasionally be found in its solid crystalline form in the stomach; but the demonstration of the presence of the lower oxids of phosphorus constitutes quite as satisfactory a factor in the proof of phosphorus poisoning as does the isolation of the element itself, and in the great majority of cases of arsenical poisoning arsenic is separated in the elementary form, and not as the trioxid or as Paris green. In a case of tartar emetic poisoning we have known the defence to lay great stress upon the fact that of the carbon, hydrogen, oxygen, potassium, and antimony of which that poison consists only the antimony was demonstrated to be present by the analysis, yet in view of the further facts of the purchase of tartar emetic by the defendants, and the manifestation of the symptoms of antimonial poisoning by the deceased, the jury very properly convicted.[1] Similarly in a case of morphin poisoning, in which the defendant was convicted, experts for the defence maintained that proof of the presence of the alkaloid was unsatisfactory because it had not been "isolated" in a state of purity, although its characteristic reactions had been observed.[2] Yet every chemist knows that in his processes of analysis, both qualitative and quantitative, it is only exceptionally that he "isolates" the elements or compounds whose presence and quantity he may determine with accuracy.[3]

The knowledge of the particular form of combination in which the poison was taken is frequently valuable as bearing upon collateral points, but is rarely obtainable. Thus if arsenic be proved to be present as Paris green, either by its separation in substance or by the presence of copper and arsenic in the

[1] State *v.* Fournier and Cox, Chittenden Co. Crt., Vt., 1894.

[2] Peo. *v.* Buchanan, Gen. Sessions, N. Y., 1893.

[3] See p. 123.

proper proportions, the theory of criminal administration in a colorless liquid is weakened, and that of suicide is strengthened thereby.

5. Was the substance found by the chemist really the poison? This question may be of importance in two ways: Either the methods of the analyst may be claimed to be defective or his conclusions unwarranted, or the substance demonstrated to be present may have found its way into the body by some means other than by administration in poisonous dose.

The former claim can be successfully advanced by the defence only when the analyst has been culpably ignorant or negligent. The Wharton trial in Maryland in 1872 is an instance in which the acquittal of the accused was probably due in part to this cause. The analysis of the stomach was so unsatisfactorily performed that the prosecution found a second exhumation and analysis of other parts to become necessary during the trial, which was adjourned from Friday noon to Tuesday noon to allow time for the supplementary analysis.

In all trials for poisoning by the vegetable alkaloids at the present time the defence advances the claim that the reactions observed were produced by ptomains and not by the vegetable alkaloid alleged to have caused the death. (See Ptomains, Morphin, Strychnin.)

When the poison, whether mineral or organic, is found only in minute quantities the question of its origin may be very properly raised. It may possibly have been administered medicinally, or may have found entrance to the living body as an impurity in some medicine or article of food, as arsenic in bismuth subnitrate, or copper in canned vegetables, or as a constituent of natural articles of diet, as oxalic acid in rhubarb, or as the result of addiction to a drug habit, as morphin, chloral, etc.; or it may have been imbibed by the cadaver, as arsenic from artificial flowers or from neighboring embalmed bodies in a water-soaked cemetery. Even when the poison is present in large amount, the possibility of post-mortem introduction, and, in some cases, of habituation must be considered. The question whether the doubt so raised is "reasonable" is for the jury to determine in the light of such expert evidence bearing upon the matter as the circumstances of the case permit of.

6. Could the substance administered have caused death? When the identity of the substance is proven or conceded, the question whether it is a poison or noxious thing may arise. (See Definition.) A substance may be administered with intent to kill, and yet, from its nature or form of administration, be incapable of causing death. Thus Tardieu[1] cites the case of a woman who tried to poison her husband with pure copper filings; and another of a man who sought to kill his wife by sulfuric acid administered in wine, in which the mineral acid was neutralized by the potassium tartrate.

The clinical history of the case may also demonstrate that the substance administered, although capable of causing death, could not have done so in the particular case at bar, either because the time between the administration and the death was such that no relation of cause and effect could exist, or because the symptoms observed were not those which the substance administered could have produced, or because the patient recovered from the effects of the poison, to die subsequently of some disease with which he was at the time afflicted, or which intercurred.

The question may also be raised on the part of the defence in cases of mechanical injury capable of causing death, followed by improper medical treatment. Thus in the case of Peo. *v.* Stokes, a compromise verdict was reached upon the second trial, in consequence of a grave doubt whether the victim, Fisk, died from the effects of a gunshot wound or from those of morphin administered in excessive doses.[2]

7. Is a poisonous substance, given in minute quantity, a poison? This question can only arise in cases in which the quantity administered is known to be small. It does not apply when the quantity of the dose is unknown and only minute quantities have been separated by analysis (see Question 8). When the poison or corrosive has been given in such minute quantity or extreme dilution that it is incapable of causing injury, it certainly does not come within the medical definition of poison or corrosive. Whether its administration in such quantity or form with criminal intent is punishable is a legal question, and one which does not concern the expert except in so

[1] "Empoisonnement," 2ème ed., p. 123.

[2] Peugnet: Papers Med.-Leg. Soc., N. Y., 1882, 2 s., 294.

far as he may be called upon to fix the limit of quantity or dilution below which the substance will be incapable of causing deleterious effects.[1]

8. Was the poison taken in a dose sufficient to cause death? This is a question a definite answer to which would be highly desirable were it possible. It is only rarely that it can be answered positively in the affirmative, and the analyst is not warranted in giving a negative answer if the presence of poison has been demonstrated.

The answer is necessarily based upon two data: (1st) The quantity of the poison in question which is capable of causing death; and (2d) the amount which was taken by the deceased. The first would be a fixed factor were it known, the second one to be determined in each case if possible. The first has not been determined within narrow limits, capable of numerical expression, even in the case of arsenic,[2] and varies with the circumstances of the case and the nature of the poison. The second can never be determined by analysis of the cadaver. The quantity present in certain organs may be ascertained with more or less accuracy by analysis, but from the results of such determination only one inference can be drawn, *i.e.*, that the quantity taken was greater than the amount found. How much greater it may have been may be guessed at, but not determined. The absolute amount present in the cadaver when it is analyzed could only be ascertained by one of three methods: 1st. By extracting all of the poison from the entire body. 2d. By extracting all from a properly equalized and weighed sample taken from a mass obtained by reducing the entire weighed body to a uniform and homogeneous pulp. 3d. By a similar process as that last mentioned, except that each organ, the different regions of the muscular system and included soft parts, and of the skeleton are treated separately. The first procedure is not possible; the second is neither possible nor desirable; the third is possible with mineral poisons, but would involve the entire destruction of the body and an expenditure of time warrantable only in very exceptional cases. With organic poisons the quantity separated is always a frac-

[1] Two cases in point are cited in Casper-Liman: "Handb. d. ger. Med.," 8te Aufl., ii., 362–367.

[2] See Arsenic—Lethal Dose.

tion of that actually present, as the methods available never effect complete extraction. What the magnitude of the fraction may be is not calculable. The quantity of poison separated from the body by the methods at present followed is therefore only a fraction, more or less large, of that which exists in the body when it is analyzed, and a still smaller fraction of that which was swallowed.

In attempting to calculate the total quantity in the cadaver it is never permissible to assume that the amount separated from a certain fractional part of the body bears the same relation to the entire amount present that the weight of the fraction examined bears to that of the body. The distribution of the poison in the different organs and tissues is uneven under all circumstances, and the quantity in one part is no indication of that in any other, except in so far as it is known that certain organs retain a greater quantity than others. And even when the quantity in a given organ is calculated from the amount found in a known fraction of that organ, the calculation is only reliable if the sample is a fair representative of the whole, taken from the finely divided and thoroughly mixed whole. It is not safe to assume, for instance, that because a given quantity of arsenic is found in one lateral half of the brain the entire organ contains twice that amount. The entire brain should be hashed, thoroughly mixed, weighed, and a weighed fraction taken for analysis.

But if the entire quantity present in the cadaver at death could be separated and weighed the result so obtained would have no greater significance. It would simply show that at least that quantity had been taken, and in all probability more. How much more will again depend upon the circumstances of the case and the nature of the poison. How much was expelled by vomiting and purging; how much was removed by absorption and elimination, or by medical treatment; how much was transformed by chemical reactions into other compounds? All these are questions which must be solved before the main question could be answered.

Yet the determination of the quantity of poison in the organs and parts analyzed should be made if possible, and with the greatest attainable accuracy. The amount thus actually separated may be greater than that which has been known in

accurately observed cases to have been the only cause of death, in which event, clearly, presuming the poison to have been in the body during life, the question under discussion may be answered in the affirmative. Or the quantity found may be such that, although not in itself poisonous, it is sufficient to negative the theory that the poison was legitimately administered in medicinal doses, particularly if allowance be made for expulsion and elimination known to have occurred during life.

When the quantity of poison separable from the body is small, all that can be argued from this fact is its mere presence. The question of its origin must be determined, if it be determinable, from the clinical history and from other elements of the case. But, assuming that the analysis has been properly performed and that the reagents used were pure, the fact of the presence of the poison in the cadaver must be accepted as proven, even when the amount must be designated as an unweighable trace. The reactions by which many of the poisons may be identified with certainty are of extreme delicacy, and are capable of certainly detecting the presence of amounts so small as to be unweighable by the most delicate balance. Thus 0.00001 gm. is practically an unweighable quantity, yet that amount of strychnin is sufficient to give the color and physiological tests and the bitter taste of the alkaloid.

In some cases an indication of the amount taken may be obtained from a quantitative analysis of vomited matters, or of the remains of articles of food or of medicine known to have been taken or administered.

9. Was the poisoning suicidal, accidental, or homicidal? Apart from the examination into the mental condition of the deceased or of the accused, the physician as well as the chemist may sometimes offer expert evidence which will aid in the determination of this important question.

The physical characters of the poison itself, or the quantity in which it is found to be present may raise a presumption in favor of suicide, or for or against the theory of the prosecution or of the defence. Presumably a poison which has a marked color, such as Paris green, or a strong taste, such as strychnin, would not be unwittingly swallowed by an adult in the full possession of his faculties. This is, however, only a presumption, and numerous cases of unquestionably homicidal poisoning by

both of these agents have occurred. Moreover, the color and the taste may be effectually concealed, the former by mixing with some green vegetable, the latter by enclosing the poison in a capsule administered to the victim as medicine.[1]

The finding of a large quantity of arsenic in the stomach was invoked in the case of Madeleine Smith in support of the theory of suicide, on the ground that such a quantity could not be administered without the knowledge of the deceased.[2] The degree of solubility of a poison is frequently a question of interest as bearing upon the form of administration and, indirectly, upon the question of responsibility.

The theory of accidental poisoning may also be in some cases set aside by chemical evidence. Thus in the case of Carlyle Harris the defence sought to show that the death might have been caused accidentally by an error of the druggist in dispensing morphin in place of quinin, although the defendant had himself supplied the proof that no such mistake had been made by reserving one of the capsules which, on analysis, was found to contain the proportions of quinin and morphin called for by the defendant's prescription.

The method of action of the poison may in some cases throw light upon this question. Thus in the case of Jean Humphreys, referred to by Taylor,[3] the theory of suicide was negatived by the facts that the deceased, who died from the effects of sulfuric acid, could not voluntarily have swallowed the acid unobserved during a period of more than twenty minutes before its action began, and that sulfuric acid produces the symptoms of corrosion immediately.

The presence of the poison in the clothing or in other articles in the possession of the defendant may also serve as a part of the proof of administration; thus in the case just cited the night dress of the defendant was stained by sulfuric acid; and in the Maybrick case arsenic was found in a handkerchief, the pocket of a dressing gown, and several other articles in the possession of the defendant.

In drawing conclusions from such facts, however, they should be carefully weighed, and the possibility of their expla-

[1] These capsules are readily opened, and substitution of poison for their legitimate contents is easy. Aconitin and morphin have been administered in capsules in homicidal cases.

[2] See Arsenic—Solubility.

[3] "Poisons," 3d Am. ed., p. 85.

nation in favor of the defendant fully recognized. Maschka[1] reports a case in which the unwarrantable conclusion that sulfuric acid, from the effects of which a man had died, must have been administered by another because the deceased had a knowledge of its action, because the burns produced by the acid were less marked at the lips than in the deeper parts of the mouth, and because no physician was called during the illness of the deceased. The first and last reasons do not concern the medical expert, and the second is insufficient.

The possibility of auto-intoxication, or food poisoning, must be held in mind. Auto-intoxications, due to the retention of poisons produced in the system, are for the most part accompanied by characteristic post-mortem lesions, such as the structural changes in the kidneys, which are present in so-called uræmic poisoning by urinary leucomains. Food poisonings, caused by eating food which has suffered decomposition resulting in the formation of poisonous products, are rarely fatal and rarely single; usually several persons eat of the same substance and suffer similar symptoms. An examination of the remains of the food may show the presence of putrid poisons.

10. Could the substance found have been administered as a medicine? In general terms irrespective of quantity, and as a mere question of possibility this question must be answered in the affirmative whatever be the kind of poison used. Of the poisons known to our predecessors all have been at some time used as medicines, and hardly is the poisonous character of a newly discovered compound recognized before its use as a medicine is suggested. And even quantity, when not excessive, does not permit an absolute negative, as medical literature is full of instances of the administration of medicines in heroic doses, which, it must be confessed, have in many instances caused the death of the patient; and this entirely irrespective of those cases of so-called accidental poisoning due to criminal negligence on the part of the prescriber or dispenser of medicines, or of both—unfortunately of much more frequent occurrence than they should be—in which either a much larger dose than was intended in the mind of the physician is given, or in which an actively poisonous drug is substituted for one of greatly inferior toxic power, by "mistake."

[1] Viertljschr. f. ger. Med., 1881, n. F., xxxiv., 197.

So far as the administration of medicines by the attending physician is concerned, it therefore behooves the prosecution to show affirmatively, by the evidence of the physician and copies of his prescriptions, what drugs he actually did prescribe or order, and by the druggist that the prescriptions were properly compounded. Without such evidence the question must remain a doubt, the reasonableness of which it remains for the jury to determine from all of the evidence; unless indeed the quantity found is so large that it could not have been medicinally given, or unless proof of some other method of administration is available, as, for instance, the existence of the same kind of poison in the remains of an article of food.

It may also be claimed that the poison found, if present in small quantity, was introduced as an impurity in a medicine legitimately administered, as arsenic in bismuth subnitrate. A case in which an incomplete analysis permitted the *possibility* of the presence of arsenic in bismuth subnitrate, administered to the deceased, to be considered by the court as an absolute bar to a conviction is reported by Rogers.[1] Arsenic was found in the stomach (which was nearly empty) and the intestines in "very decided amount." The liver was not sent for analysis. During the trial it was shown that bismuth subnitrate was among the medicines administered during the illness of the deceased. Ten samples of bismuth subnitrate (not including a specimen of that administered) were examined and eight were found to contain arsenic in quantities not determined. Upon this condition of facts the trial was cut short, and the defendant discharged, it being held that, in the absence of any proof of the absolute amount of arsenic adequate to destroy life, the mere fact of the discovery of a poisonous adulteration of the medicine administered was sufficient to invalidate the evidence against the prisoner and to justify her immediate discharge. Purchase of arsenic by the accused had been proved; and it was not shown, nor was it probable, that even if the subnitrate had been impure, it contained enough arsenic to cause the vomiting and purging proved to have occurred, much less a fatal result.

If this extreme view be accepted as correct, it must be admitted that evidence of the fact that bismuth subnitrate had

[1] Tr. Coll. Phys., Phila., 1857, n. s., iii., 197.

been administered in a case of alleged arsenical poisoning would be adequate ground for the discharge of the jury whatever the quantity of arsenic found. Yet in this case, had the liver and other organs been examined and the quantity of arsenic determined, it seems probable, from the presence of a "very decided amount" in the stomach and intestine after the patient had vomited and purged copiously for several days, that a distinctly weighable quantity of arsenic would have been found. If this were more than would have been contained in all of the bismuth called for by the prescriptions, even if the drug were in its most impure form, it seems to us clear that the case would have in nowise differed from one in which only that quantity of arsenic would have been found which would be represented by the excess found in this case over that accounted for as possibly attributable to the bismuth. And if the proportion of arsenic in a sample of the identical drug administered had been determined (as it might have been had such sample been available and it actually did contain arsenic), the excess of arsenic found, if there were such, over that which could by any possibility have been given with the bismuth could have been definitely stated. Yet even under these circumstances it would seem that under this ruling the prisoner would have been discharged. And as a logical consequence no conviction for arsenical poisoning could be had, or the case even submitted to the jury, unless more than fifteen grains of arsenic were actually separated from the cadaver and the patient had taken no bismuth—conditions which have obtained in only a very few of the large number of known arsenical poisonings.

On the other hand, if the quantity of arsenic found after death be a mere trace, or less than that which might have been present in bismuth or other drug or mineral water containing arsenic, the case must be considered as one in which no poison was found, and should be decided by the jury upon other evidence, including that showing the administration of drugs containing the poison.

It may also be claimed that the poison found, particularly if in small amount, was taken as a medicine without the prescription of a physician, either by the refilling of an old prescription [1]

[1] Section 405*a* of the N. Y. Crim. Code makes it a misdemeanor to refill more than once a prescription containing morphin or the prepara-

or as a patent or proprietary medicine, by the deceased himself or administered without criminal intent by another. Thus in the Maybrick case it was claimed that the deceased was in the habit of dosing himself with arsenic. Deaths of children from the effects of morphin in "soothing syrups" are of frequent occurrence; numerous poisonings by tartar emetic have resulted from the use of "*quietness*," to remove the effects of a debauch; and "arsenical wafers" are extensively advertised and, presumably, largely used. The possibility of such an origin of a small quantity of poison found must be admitted. Whether such possibility amounts to a reasonable doubt is for the jury to determine in the light of all the evidence in the case, as, even when the symptoms are well marked, the post-mortem appearances clearly defined so far as they go, negatively as well as affirmatively, and the poison is found in quantity which may be considered large for that particular poison, the possibility that an overdose was unintentionally taken still remains. The question is one of intent, and is not within the province of the expert evidence, except in so far as that may throw light upon the matter collaterally as shown in the discussion of these questions.

11. When and how was the poison taken? It is highly desirable in the investigation of criminal poisoning, as in that of homicide by other means, that the precise time of the infliction of the injury shall be fixed as closely as possible. Owing to the secrecy[1] with which poison is administered when given with criminal intent, it is but rarely that the time of the administration can be determined by other than circumstantial evidence, of which expert opinion may constitute an important part. The corrosives, which act upon the parts with which they are brought into immediate contact, begin their action at the instant of contact, consequently if the case has been observed during life or the patient can describe the attack, the time of administration can be fixed as immediately preceding the first effects. With the true poisons, however, action does not begin before the entrance of the toxic into the circulation, and consequently the interval between administration and the

tions of opium in which the dose exceeds one-fourth of a grain of opium or one-twentieth of a grain of morphin: but there is no provision against the refilling of other prescriptions containing poisons.

[1] In a few cases the administration was not secret but forcible.

first effects observed will vary with the conditions which favor or delay absorption (see p. 63). If the form in which the poison was taken be unknown, the duration of the interval preceding the manifestation of symptoms must be considered as having been between the extreme limits in observed cases, if these have been sufficiently numerous to afford a basis for comparison. Thus in the case of arsenic the duration of this interval has been from "immediately" to nine hours; and with phosphorus from "immediately" to two days.

A knowledge of the rapidity of elimination of the poison may also be of service in fixing the date since which it must have been taken if found to be present, and since which it is improbable that it was taken if it be not found in a properly conducted analysis, if it be a poison whose detection by this means is to be expected.

The clinical history of the case will frequently determine whether the poison was administered in a single dose or in repeated doses.

The question of *how* the administration was effected may relate to the channel of entrance or to the vehicle with which it was mixed. The great majority of criminal poisonings are by the mouth. It is only exceptionally that poison has been criminally given by the rectum, by hypodermic injection, by the vagina, by inhalation, or by the ear, yet such cases have occurred.[1] If the poison have any local action, as arsenic, or if a mineral acid have been used, the seat of application will be indicated by the local effects produced.

The vehicle of administration is best determined by analysis of remains of articles of food, drink, medicine, etc., and sometimes of the bodies of animals who have devoured such remains, when they have been thrown out. Frequently, also, valuable information can be obtained from analysis of even very minute quantities of solid or liquid residues remaining in cups, glasses, bottles, or cooking utensils.

12. Could the substance separated by the chemist have an origin other than administration during the life of the deceased ? Admitting the identity of the poison to have been satisfactorily proven by the results of the analysis,

[1] See under Sulfuric Acid, Morphin, Carbon monoxid, and Arsenic, Mercury, Phosphorus, Chloroform.

the answer to the first question discussed above will depend in some measure upon the exclusion of a possible fortuitous origin of the substance found. The chemist must be depended upon solely to exclude some of the possible sources of contamination, as, for instance, by impurity of chemicals, or introduction into the materials under analysis during the examination; while the exclusion of others, such as possible post-mortem imbibition, can at present only be reached from a consideration of all of the evidence in the case, and sometimes not then.

The **purity of the chemicals** used must be beyond question. This can only be assured by a careful examination of the reagents by the chemist himself, and a purification, if necessary; by the preparation of the chemicals in the laboratory from materials known to be pure; or by a "blank testing." No reliance, for toxicological purposes, is to be placed upon the labels of even the most reputable manufacturers; the chemist must *himself know* that he has not unwittingly introduced the substance which he has found. The methods of testing chemicals, their purification or manufacture, have been referred to previously, or will be in the division of special toxicology in discussing their use.

A "blank testing" consists of a performance of the same operations as those conducted with the organs examined, and with the same reagents in equal or greater quantity, upon tissue or materials known to be free from poison. Its negative result demonstrates the absence of the poison found from the reagents used.

An unrecognized accidental or intentional **addition of poison** to the substances analyzed, at the autopsy or subsequently, should be rendered a physical impossiblity by attention to the directions already laid down (see pp. 112, 114, 123).

The occurrence of poisons such as arsenic and copper as **normal constituents of the human body** has been, after considerable discussion by the older toxicologists, definitely negatived. It must not be forgotten, however, that both of these poisons, by constant presence in articles of food or drink and relatively slow elimination, may be present as quasi-normal constitutents of the bodies of members of certain communities, being taken by them unknowingly and not in consequence of the contraction of drug habits. Thus, although we know of no

specific observation of the fact, it would seem probable that if the cadaver of an inhabitant of the village of Whitbeck (where a stream whose water is impregnated with arsenic is used as the source of drinking-water[1]) were analyzed it would be found to contain small quantities of arsenic; and owing to the very general use of copper utensils and of the salts of copper in "greening" canned vegetables, pickles, etc., copper in small amount is almost invariably found, particularly in the liver.

The possibility of the **post-mortem introduction** of poison into the cadaver has been recognized by toxicologists from the time of Orfila, and the possibility of distinguishing poison so introduced from that taken during life has been the subject of much careful study in recent years. The post-mortem introduction of poison into the cadaver may be either (1st) By malicious injection; (2d) for the prevention of putrefaction, in embalming, so-called; (3d) by imbibition from the soil or from surrounding objects.

It must be admitted as a physical possibility that a properly selected poison might be introduced into a cadaver in such manner as to lend color to a false accusation of poisoning. But the success of such a crime would require the fulfilment of conditions which are hardly within the range of possibility: great toxicological knowledge, combined with diabolical intent on the part of the perpetrator, and a series of occurrences favorable to his design and more or less beyond his control, both preceding and succeeding the death of the alleged victim.

The post-mortem impregnation of bodies with poisons for purposes of preservation and by accidental imbibition is of much greater practical interest, particularly in the United States, where the practice of undertakers is entirely unbridled by law. Although any poison of alleged antiseptic power may be a constituent of a so-called embalming liquid, and although many poisons may find their entrance into a cadaver by imbibition from without if brought in contact with it, it is chiefly with reference to arsenic that the question is of forensic interest; and we therefore defer its consideration to the division of special toxicology under arsenic.

13. Is the flesh of poisoned animals poisonous to man? Apart from the frequently occurring poisonings by

[1] Ph. J. and Tr., 1860–61, n. s., ii., 286.

tainted meat, it must be recognized as possible that, under favorable conditions, a sufficient amount of mineral or vegetable poison may remain in the tissues of an animal whose death resulted from its action to cause poisoning in a human being who may eat of them. No authenticated case of poisoning by this means is, however, recorded so far as we know.[1]

14. Can a case of homicide by poison be made out if the body has been cremated? If there have been neither autopsy nor chemical analysis previous to the cremation this question is practically, Can a case be made out without autopsy or proof of the presence of poison in the cadaver? because lead and copper are the only poisons of which traces would remain in the ash, and these are very rarely used in criminal poisoning, and might be readily disposed of by substituting other ashes for those of the cadaver. The answer to the question in its latter form has already been given in the discussion of the first question.

As the proof of poisoning, difficult in any case, is rendered well-nigh impossible by cremation, and as in a very large proportion of cases of poisonings which have been proved, the body has been exhumed after burial, it would seem desirable that the cremation of a body should not be permitted except after an investigation by the proper medical officer, sufficient to establish a reasonable certainty that death was due to natural causes.[2] The fact that the practice of cremation is by no means general is no argument against the advisability of this supervision, as while cremation without an autopsy is possible, it may be resorted to by the criminal to remove the evidences of the crime.

For other "Forensic Questions" bearing particularly upon individual poisons, the reader is referred to the division of Special Toxicology.

[1] Taylor: "Poisons," 3d Am. ed., 170–174, discusses this question at length. The only case of poisoning in the human subject, however, which he cites (from Galtier) was more probably one of trichinosis or of putrid intoxication than one of poisoning by copper. See, however, Seeley: Med. Rec., N. Y., 1894, xlv., 14 and Reg. v. Sprague, Ph. J. and Tr., 1865, n. s., vii., 72.

[2] The destruction of the body removes evidences of violent death or injury from other causes as well as those of poisoning. That the mere certificate of death by the attending physician is sufficient is disproved by the instances given on p. 105. See also Chapuis: "Précis d. Tox.," 2d ed., 1889, p. 98.

CLASSIFICATION OF POISONS.

Poisons and corrosives require classification for two distinct purposes: 1st. Into analytical groups to facilitate their extraction and identification. 2d. Into natural groups to render the study of all of their characters more logical and consecutive.

The **analytical classification** is both possible and necessary, and follows the analytical methods used:

I. GASEOUS POISONS: Carbon monoxid, hydrogen sulfid, sulfur dioxid.

II. VOLATILE POISONS, which are separable from mixtures by mere distillation, with or without vapor of water; and from acid, neutral or alkaline liquids: Alcohol, chloroform, hydrocyanic acid, ammonia and its derivatives, phosphorus, etc.

III. ACIDS, ALKALIES, AND SALTS: Mineral poisons and corrosives, which are best separated by extraction with water. Mineral acids and alkalies and certain soluble metallic salts.

IV. ORGANIC POISONS: Substances which do not withstand the action of powerful reagents, and which are extracted from the mixtures in which they exist by neutral solvents or by dilute acids, either applied directly or in agitation methods with immiscible solvents—vegetable acids, glucosids, alkaloids, and bitter principles, and animal poisons.

V. MINERAL POISONS: Substances of sufficient stability to permit of their separation by the decomposition and removal of the organic substances with which they may be mixed, followed by the usual methods of mineral analysis, somewhat modified to meet the requirements of the case.

A **systematic classification** is more difficult to arrange and, in its minuter ramifications, not practicable at present. Different systems have been suggested, based either upon the origin of the poisons and corrosives, or upon their method of action upon the economy. Both arrangements have advantages and disadvantages, but of the two we believe that based upon origin to be the preferable one for the present, although the other would be the more natural and the more useful: because the origin is determinable in every case, while the method of action of many poisons is still imperfectly understood.

The earliest classification, suggested by Plenck in 1785,[1] was based upon origin, and consisted of four groups: (I.) Animal poisons; (II.) vegetable poisons; (III.) mineral poisons; and (IV.) poisonous gases, vapors, and dust.

The first to suggest a classification based upon the method of action of toxic substances was Fodéré,[2] who divided poisons into six classes: (I.) Septic poisons; (II.) stupefying or narcotic poisons; (III.) narcotico-acrid poisons; (IV.) acrid or rubefacient poisons; (V.) corrosives, or escharotic poisons; and (VI.) astringent poisons.

The classifications of writers following Plenck and Fodéré: Mahon,[3] Orfila,[4] Christison,[5] Flandin,[6] Casper,[7] Taylor,[8] Tardieu[9] and others, are merely modifications of these either by extension or by contraction; if we except the division of Anglada[10] into the two classes of solid and liquid, and of gaseous poisons.

Of the classifications based upon physiological action, probably the best developed hitherto suggested, is that of Rabuteau,[11] who divides poisons into six classes:

I. Hæmatic Poisons.—A. *Globular poisons*, acting chiefly on the red corpuscles: Carbon monoxid, hydrocyanic acid, hydrogen sulfid, and ammonium sulfhydrate, compounds of selenium and tellurium, phosphorus, arsenic, alcohols.

B. *Plasmic poisons*, acting on corpuscles and plasma. Nitrates and nitrous fumes, salts of silver injected into the veins, the greater part of the metallic salts, when given in small and repeated doses.

II. Neurotic Poisons.—A. *Paralyso-motors;* which abolish the functions of motor nerves: Curare, calabar bean, aconitin, coniin.

B. *Spinals*, which exaggerate reflex sensibility: Strychnin, m'boundon, compressed oxygen, cantharides, etc.

[1] "Toxikologie," Wien, 1785.
[2] Traité de médecine légale, Paris, 1813, iv., 6.
[3] "Œuvres posthumes," Paris, An x. (1801), ii., 320.
[4] "Traité des poisons," Paris, 1814, i., 5–14.
[5] "Treatise on Poisons," Edinb., 1829, 81–83.
[6] "Traité des poisons," Paris, 1846, i., 225.
[7] "Handb. d. ger. Med.," Berlin, 1857, ii., 383.
[8] "Med. Jur.," 3d ed., London, 1849, p. 7.
[9] "Étude s. l'empois.," Paris, 1867, p. 167.
[10] "Traité de toxicologie," Brux., 1837, p. 252.
[11] "Elements de toxicologie," 2ème ed., Paris, 1887, pp. 29–34.

C. *Cerebro-spinals*, which act upon the elements of the brain and spinal cord: Chloroform, ether, opium.

III. NEURO-MUSCULAR POISONS: Solanaceæ, digitalis, antimonials, carbon dioxid.

IV. MUSCULAR POISONS: Strophanthus, veratrin, salts of potassium and of barium, copper, zinc, cadmium, tin, lead, mercury, etc.

V. IRRITANTS OR CORROSIVES: Sulfuric, nitric, hydrochloric, hydrofluoric, oxalic acids, potash, soda, ammonia, alkaline sulfids, bromin, chlorin, etc.

The classification which will be followed in this work, after the primary division into corrosives and poisons,[1] is based upon origin. Within the classes a further subdivision is desirable, but, we regret to believe, impracticable at present, consequently an alphabetical arrangement is as good as any. We will divide toxic agents into:

I. **Corrosives**: Substances which act chemically upon tissues with which they are brought into immediate contact—mineral acids, alkalies, halogens, etc.

II. **Poisons**: Substances which act after entrance into the circulation, followed by solution in the blood or chemical action upon the blood itself.

A. MINERAL POISONS: Arsenic, antimony, the salts of copper, lead, mercury, etc., phosphorus.

B. VEGETABLE POISONS: Vegetable acids, alkaloids, bitter principles, glucosids, etc.

C. ANIMAL POISONS: Leucomaïns, ptomaïns, toxins, toxalbumins.

D. SYNTHETIC POISONS: Chloroform, alcohol, chloral, phenol, antipyrin, etc.[2]

[1] See Definition, p. 43.

[2] The synthetic poisons may be classified according to their positions in the purely chemical classification, first, into *open-chain* and *closed-chain* derivatives, and within these classes into hydrocarbons, alcohols, aldehyds, acids, etc. See Witthaus: "Manual of Chemistry," 4th ed., 226, *seq.*, or any modern organic chemistry.

SPECIAL TOXICOLOGY.

CORROSIVES.

MINERAL ACIDS.

All acids are absorbed and act as true poisons when they are administered in such dilution that intense local action is avoided, but yet in sufficient absolute quantity and for a sufficient period of time to cause diminution of the normal alkalinity of the blood and other parts.[1] The symptoms of poisoning—such as somnolence, sopor, coma, and convulsions, referable to the action upon the nervous system; and the appearance of albumin, globulin, blood, and casts in the urine, resulting from the action of the diminished alkalinity upon the blood and kidneys—and death occur before the quantity of acid absorbed is sufficient to entirely neutralize the alkaline reaction. Indeed, the same effects have been produced in rabbits without the administration of acids, by withholding food capable of producing alkalies, when the formation of sulfuric acid resulting from the oxidation of the sulfur existing in albuminoid substances,[2] and of phosphoric acid similarly produced from the lecithins and other phosphorized constituents of the body [3] causes sufficient diminution of alkalinity. It has also been shown by Walter [4] that acid poisoning is much more rapidly produced in herbivorous animals than in the carnivora; which is accounted for by the neutralization of absorbed acid by ammonia to a greater amount in the latter than in the former.

Instances of such "acidism" or acid poisoning, caused by dilute weak acids—such as oxalic and acetic—have been observed in the human subject (see Oxalic Acid), and are by no means uncommon after corrosion by the more powerful acids, if life be sufficiently prolonged. But the powerful mineral acids—sul-

[1] The gastric juice and urine only are acid normally.

[2] Salkowski: Arch. f. path. Anat., etc., 1873, lviii., 1.

[3] Jitta: Diss., Amsterdam, 1885. Kraus: Arch. f. exper. Path. u. Pharm., 1890, xxvi., 186.

[4] Arch. f. exper. Path. u. Pharm., 1877, vii., 148.

furic, nitric, and hydrochloric—as well as chromic anhydrid, when taken in concentrated form, act immediately and energetically upon the tissues with which they come in contact, causing disintegration and destruction of the part by their chemical action, and producing symptoms of such intensity and violence, due to this corrosion, that the phenomena of true poisoning fall into the background, or are entirely omitted in rapidly fatal cases. Moreover, in the frequently recurring instances in which the patient recovers from the first effects of the acid, from the acidism as well as from the primary local action, only to die weeks or months later from interference with the processes of nutrition, the lesions which are the cause of death are referable entirely to the corrosive action of the acid and in no way to its transitory poisonous effects.

Therefore, while the occurrence of acidism is of scientific and medical interest, and while it offers an explanation of the cause of certain of the symptoms observed in some cases of corrosion by the powerful acids, it is of minor importance in the forensic toxicology of these substances, which are the most energetic of corrosives.

Statistics and Causation.

Although no statistics serving to indicate the absolute number of instances in which sulfuric, hydrochloric, and nitric acids have caused death or injury in the human subject are available, the following table of cases reported in medical journals accessible to us will show the relative frequency, mortality, and origin of corrosions by those acids:

	Cases.	Deaths.	Recoveries.	Homicides.	Suicides.	Accidents.
Sulfuric acid [1]	303	207	84	45	151	75
Hydrochloric acid	69	55	14	5	39	24
Nitric acid	65	54	11	13	34	9

It may be noted that the proportion of recoveries indicated is certainly too high, as a large number of the cases set down as having recovered have been reported shortly after their occurrence, and before sufficient time has elapsed for the development

[1] Cases of external application (vitriol-throwing) not included.

of the secondary effects which have proved fatal in many instances.

Of the 63 **homicidal cases**, 53 were fatal, including all of those by nitric and hydrochloric acids, save one by the latter. Ten occurred in Great Britain (1, HCl; 9, H_2SO_4); 26 (1, HCl; 7, HNO_3; 18, H_2SO_4) in Germany; 4 (2, HNO_3; 2, H_2SO_4) in Finland; 9 (1, HNO_3; 8, H_2SO_4) in Austro-Hungary; 12 (3, HNO_3; 2, HCl; 7, H_2SO_4) in France; 1 (H_2SO_4) in Holland; and 1 (HCl) in Belgium. We have been unable to find record of a case of criminal internal administration of a mineral acid to another in the United States; although these agents have been frequently resorted to by suicides, and the crime of "vitriol-throwing" is a common one in this country.[1]

In 38 of the 63 cases the victim was an infant or young child, to whom the acid was given more or less forcibly. In one instance a father, after conviction for having destroyed his child shortly after its birth by nitric acid, confessed to having previously disposed of five others in a similar manner.[2]

It is difficult to imagine a manner in which a strong mineral acid could be made the agent of *secret* poisoning of an adult in the possession of his faculties. The corrosive action upon the lips and tongue is immediate upon contact, and so severe that it would cause the instant expectoration of any small quantity which might have entered the mouth before being swallowed. Indeed, the Appellate Court in a Dutch case held the extreme opinion that the mixing of sulfuric acid with coffee, a portion of which was subsequently taken into the mouth of the intended victim and by him immediately expectorated, was not an attempt at administration of a deleterious thing, because "any person who did not wish to take it would be prevented by the repulsive taste from taking a second portion into the mouth, and would immediately expel that already taken, so that it would not be possible for the sulfuric acid to reach the stomach."[3] In a somewhat similar case, tried at Cassel in 1853, in which the injuries were limited to the mouth and tongue, beyond which

[1] Beck: "Med. Jur.," 12th ed., ii., 464, states that: "In 1817 a female was tried in this State [New York] for poisoning an illegitimate child" [by sulfuric acid]; but he gives no particulars.

[2] Osenbrüggen: Allg. deut. Strafrechtsztg., 1865, v., 273, *ex* Friedr. Bl. f. ger. Med., 1867, xviii., 75.

[3] Friedr. Bl. f. ger. Med., 1856, vii., Heft 3, p. 73.

the acid did not penetrate, the defendant was more properly convicted of attempted murder, and sentenced to fourteen years' imprisonment.[1] A case is, however, reported by Mignot,[2] of a man of forty years who died in three months from the secondary effects of sulfuric acid, which had been adroitly substituted for brandy which he was in the habit of drinking. The powerful taste of the acid has also been the means, indirectly, of preventing the murder of an infant; as in an early case in which the mother tasted the food prepared by a servant who for revenge had added sulfuric acid to it. The soup was not given to the infant, but was sent to the authorities for examination.[3]

Of the other twenty-three cases in adults classed as homicidal, three were declared after investigation to have been more probably accidental or suicidal than homicidal.[4] In one fatal case a woman poured sulfuric acid into the open mouth of her sleeping husband.[5] In another instance, also fatal, a weak-minded woman was induced to swallow the same acid.[6] A woman died from the effects of nitric acid which her husband poured into her ear while she was intoxicated, "to correct her," as he alleged.[7] In three instances (one each of sulfuric, hydrochloric, and nitric acid) the corrosive was taken by a pregnant female upon the advice of her lover, and under the belief, on the part of one or both, that it would cause abortion.[8] Sulfuric acid, mixed with kerosene, was given to a pregnant woman with the same object by a professional abortionist. She died on the third day, after having aborted the day before she died.[9] In an early case nitric acid mixed with wine was poured down the throat of a woman during a debauch, and caused her death.[10] A man was induced to take sulfuric acid upon sugar, which was blackened by the

[1] Friedr. Bl. f. ger. Med., 1854, v., Heft 5, p. 63.

[2] Bull. et mém. Soc. méd. d. hôp. de Paris, 1878, 2 s., xiv., 89.

[3] Jahrb. d. ges. Staatsarznk., 1837, iii., 146.

[4] Maschka: Vierteljschr. f. ger. Med., 1881, n. F., xxxiv., 197. Chevallier and Ollivier: Ann. d'hyg., etc., 1845, xxxiii., 179. Maschka: Allg. Wien. med. Ztg., 1880, xxv., 315.

[5] Christison: Edinb. M. and S. Jour., 1831, xxxv., 296.

[6] Fagerlund: Vierteljschr. f. ger. Med., 1894, 3 F., viii., Supplhft. 54.

[7] Arch. gén. d. M., 1826, xi., 104, *ex* Orfila: "Tox.," 5ème ed., i., 172.

[8] Van den Broeck: Gaz. méd. Belge, 1847, v., 94 (Affaire Denisty; HCl). Buchner: Friedr. Bl. f. ger. Med., 1866, xvii., 192 (HNO_3). Riecker: Zeitschr. f. Staatsarznk., 1843, 32 Erghft., 284 (H_2SO_4).

[9] Casper-Liman: "Handb. d. ger. Med.," 8te Aufl., ii., 460.

[10] Tartra: "Traité de l'emp. p. l'ac. nitrique," Paris, An x. (1802), p. 87.

action of the acid, under the representation that it was a medicine. Although the injuries were severe, the man recovered.[1] In 1823 a man in Strasbourg attempted to kill his wife, first by tartar emetic and afterward by sulfuric acid in syrup, administered under the pretense that they were medicines.[2] In two instances concentrated sulfuric acid was administered unmixed with other substances to adults in the full possession of their faculties, they, however, believing that they were about to receive medicine. Both were successful husband murders. In one the acid was given in a spoon, which was passed well back into the mouth before its contents were discharged.[3] In the other the acid was injected into the rectum.[4] An unsuccessful attempt at administration of sulfuric acid to a woman is related by Taylor:[5] A woman gave, "while acting as nurse to the wife of a man with whom she was cohabiting, some oil of vitriol in a wineglass, representing it to be castor oil. The woman requested that some water might be put in it. This was done, but the glass became suddenly so hot that the woman could not hold it. She put it to her lips, which were burnt by it, but she did not swallow any. The glass was handed back to the accused, who threw the contents away and washed out the glass. But for the clear description of the effect of adding water to the liquid, the nature of the poison would not have been known, as after the occurrence no oil of vitriol was found. The poisoner was acquitted on the ground that she might have made a mistake. She was subsequently convicted and executed for poisoning" (Reg. *v.* Catharine Wilson, 1862). Three attempts at murder by mineral acids mixed with beverages proved unsuccessful: One was the Dutch case already referred to.[6] The other two are referred to by Taylor.[7] In Reg. *v.* Hartley (1850) the prisoner, a girl, was charged with attempting to administer oil of vitriol to her father in coffee. In Reg. *v.* Somers (1866) a girl of twelve years was charged with administering hydrochloric acid to her mistress in beer. Two instances of attempted wife murder by repeated administration of small doses of sulfuric

[1] Maschka: "Gutacht. Prag. m. Fak.," 1853, i., 38.

[2] Bull. Soc. de méd., Jan., 1830; *ex* Leconte: Thèse, Strasb., 1855, p. 26.

[3] Hager, in Gross: "Die Strafrechtspflege in Deutschland," 1861, Heft i., p. 181; *ex* Maschka: "Handb. d. ger. M.," ii., 86.

[4] Leconte: *Loc. cit.*, p. 27.

[5] "Poisons," 3d Am. ed., 164.

[6] See p. 190.

[7] "Poisons," 3d Am. ed., 163, 220.

acid are recorded. One is an early case (1701) reported by Valentine,[1] in which two ounces of the acid were given in brandy, under the disguise of medicine, during three weeks, from the effects of which the woman died. The second, an unsuccessful attempt, was the subject of a criminal trial at Belfast in 1883.[2] The acid was given in port wine, a sample of which was found on analysis to contain 12.5 per cent. of the acid. Possibly arsenic was also given.

Notwithstanding the extreme pain and the sometimes lingering death caused by the mineral acids, **suicides** apparently resort to them quite frequently in European countries. In England and Wales (1871–80) 17 per cent. of the suicides by poison were caused by mineral acids; in Vienna (1874–75), 14.17 per cent.; in Prussia (1869) 17.47 per cent. According to Schmiedel, of 111 poisonings treated at the Charité in Berlin, (1874–80), 65 were by sulfuric acid.[3] Suicides by mineral acids are also of frequent occurrence in Denmark[4] and in Finland.[5] In the United States these agents seem to be less frequently resorted to by suicides. Thus only 1.82 per cent. of the suicides by poison in New York City (1866–80) were by mineral acids. Sulfuric acid is more frequently taken by women than by men (men 38.8 per cent., women 61.2 per cent.), while the reverse is the case with nitric acid (men 60.7 per cent., women 39.3 per cent.) and hydrochloric acid (men 53.1 per cent., women 46.9 per cent.).

Accidental cases have resulted usually from the careless exposure of the acid within the reach of children, or by the acid being taken in mistake for spirits, beer, medicine, or even water. In two cases sulfuric acid was taken by pregnant females to cause abortion, once by the mouth[6] and once by injection into the vagina.[7] In neither case did the woman abort. In four instances sulfuric acid was administered in enemata by mistake.[8]

[1] "Novellæ Med.-leg.," Francof., 1711, p. 588, Cas. 29.

[2] Reg. *v.* Sorroghan, Ulster Winter Assizes, 1883, Lancet, 1884, i., 226.

[3] Friedreich's Bl. f. ger. Med., 1882, xxxiii., 121: oxalic acid, 14; phosphorus, 32.

[4] Trier: "Hosp.-Meddelelser," Kjöb., 1851, iv., 1–28; 1852, v.

[5] Fagerlund: Vrtljschr. f. ger. Med., 1894, 3 F., viii., Splheft. 48.

[6] Fagerlund: *Ibid.*, 54.

[7] Jour. de chim. méd., etc., 1831, vii., 312.

[8] Jour. d. chim. méd., etc., 1835, 2 s., i., 425. Deutsch. preuss. med. Ver. Ztg., 1848, No. 13. Hofmann: "Lehrb. d. ger. M.," 5te Aufl., 648.

And in an early case the same acid was even given by a medical student as a cure for toothache.[1]

Lethal Dose—Concentration.

The absolute quantity of any of the mineral acids capable of causing death cannot be stated. The violence of the action depends more upon the degree of concentration, the age and condition of the person affected, and the part most seriously attacked, than upon the absolute quantity taken. Cases are not wanting, however, in which diluted acids have caused death. Indeed, the absolute degree of concentration of the acid is stated in but few cases, and it is probable that, as generally sold by apothecaries, it is more dilute than that met with in chemical laboratories and factories. The "oleum," or commercial oil of vitriol referred to in German cases, is a dilute acid (6 : 1); and in that country sulfuric acid sold at retail must be diluted with five parts of water, the purpose of the regulation being to avoid accidents.[2] An instance is reported by Heginbotham[3] in which a man of fifty years died in two and one-half hours after swallowing 22 c.c. (℥ vi.) of sulfuric acid (specific gravity 1.843), diluted with 66 c.c. (℥ xviij.) of water. A woman died in two months from the effects of two swallows of a mixture of one volume of sulfuric acid and four volumes of water.[4] Fernbacher[5] refers to the case of a man of nineteen years who died in eleven days from swallowing 100 c.c. (℥ 3¾) of the same acid diluted with water in the proportion of one to five; and in one of Mannkopf's cases[6] a woman of twenty-four years died in eight weeks after taking from 15 to 20 c.c. (℥ ½–¾) of the same acid, also diluted with five volumes of water.

Sulfuric acid has caused the death of adults in doses of 3.7 gm. (℥ i.) in two instances,[7] and hydrochloric acid in the

[1] Tulpius: "Obs. Med.," L. iii., c. 43, p. 254.

[2] Casper-Liman: "Handb. d. ger. Med.," 8te Aufl., 461, 467.

[3] Med. Times and Gaz., 1863, i., 183.

[4] Wyss: Arch. d. Heilk., 1869, x., 184.

[5] Diss., Munich, 1890, p. 8.

[6] Wien. med. Wochenschr., 1862, 564.

[7] Christison: Am. ed., 131. Whitton and Turner: Austral. M. Gaz., 1890–91, x., 299. In the early case of Consbruch (J. d. pr. Arznk. u. Wundarznk., 1798, vii., 2 st., 18) the accuracy of the dose given (twenty drops) is questionable.

same quantity in one case.[1] The smallest quantity of nitric acid known to have caused the death of an adult was in a case reported by Warren,[2] in which a woman of thirty-seven years accidentally took 12 gm. (ʒ iij.), of which some was spit out, and died in thirteen days. Children have been destroyed by less quantities of sulfuric and nitric acids. In a case reported by Liman[3] a child of two months died in twenty-eight and one-half hours after the administation of half a teaspoonful (1.8 c.c.) of sulfuric acid; and in a case of Maschka's a two-year-old boy died in seventeen days from the effects of the same quantity of the same acid.[4] Beyerlein[5] reports the case of a girl of two and one-half years who died in eighteen hours from the effects of 3.7 c.c. (ʒ i.) of hydrochloric acid. We find no record of a child having been destroyed by less than 7.5 c.c. (ʒ ij.) of nitric acid, which quantity caused the death of a boy of six months in twenty-four hours in a case reported by Buchner.[6]

On the other hand, both adults and children have taken notably larger quantities of sulfuric acid and have not died within the period covered by the reports. Brettner[7] relates the case of a young woman who took 250 c.c. (ℨ viiiss.) of sulfuric acid with suicidal intent. In seven days she could swallow and retain liquids. In one of Mendelsohn's cases[8] a woman of twenty-four years attempted suicide by swallowing a cupful (ℨ iv.=118 c.c.) of sulfuric acid. At the time of the report, eight days after the injury, she was in a fair way to recovery. In a case reported by Correa de Serra[9] a woman took by mistake 75–90 c.c. (ℨ iiss.–iij.) of the acid on June 5th, and was under observation to the end of October, after which she was not seen by the reporter. In these cases, however, there is no evidence that death did not occur later from the secondary effects, an event the probability of which is not remote. Several cases of recovery after taking 60 c.c. (ℨ ij., or a "wineglassful") of the acid have been

[1] Johnson: Brit. Med. Jour., 1871, i., 221.

[2] Extr. Rec. Bost. Soc. M. Impr., 1853, i., 78.

[3] Vrtljschr. f. ger. M., 1865, n. F., iii., 89.

[4] Samml. Gutacht. Prag. med. Fak., 1858, 2 F., 224. In another of Maschka's cases a woman stated that she had destroyed her child with two drops of sulfuric acid, but the quantity given, although small, was certainly greater.

[5] Friedreich's Bl. f. ger. Med., 1890, xli., 31.

[6] Friedreich's Bl. f. ger. Med., 1866, xvii., 187.

[7] Méd. mod., 1890, i., 261.

[8] Charité Ann., (1885-86), 1887, xii., 183 (Case VII.).

[9] Jour. d. chim. méd., etc., 1826, ii., 209.

reported,[1] in one of which[2] a woman of twenty-six years attempted suicide by swallowing a glassful of sulfuric acid. Two years later she was still living as a nurse at the Maternity, and although not well nourished, was capable of performing her duties. She had three œsophageal constrictions which were held in check by daily passage of the sound. Letheby has reported[3] an instance in which a boy of nine years swallowed 30 c.c. (℥ i.) of sulfuric acid, and, after suffering severely for five days, gradually recovered. The same author has also recorded[4] the case of a boy of six years who took 15 c.c. (℥ ss.) of the acid, although it is believed that not much reached the stomach, and slowly recovered. Galtier[5] cites an observation of Leuret of a man of eighteen years who recovered after an attempt at suicide by 60 gm. (℥ ij.) of commercial nitric acid. We can find record of but one case in which a child has recovered from the effects of nitric acid, and in that the amount of the dose is not stated;[6] and but one case in which a child recovered after taking hydrochloric acid. In this the dose was only "a drop or two."[7] Atkinson[8] has reported a recovery of a woman of forty-five years, from the primary effects of a dose of about 60 c.c. (℥ ij.) of hydrochloric acid.

Duration.

The mineral acids may cause death within a few hours by the intensity of their local action from peritonitis, shock, hemorrhage, or suffocation, due to destruction of tissue; or death may result in several days from a combination of the local action, less destructive than in rapidly fatal cases, with true poisoning consequent upon the absorption of the acid; or the fatal result may be delayed for months, and finally result from inanition due to interference with digestion and absorption by the lesions caused or provoked by the local action of the corrosive.

[1] Johnson: Lond. Med. Gaz., 1828–29, iii., 253, F. 19. S. Crawford: M. T. and Gaz., 1867, i., 182, F. 38, S. Desgranges: Rec. per. d. l. Soc. d. méd., Paris, An. vii., vi., 3, M. ad. A. Tott: Jour. d. pr. Hlk., 1837, lxxiv., 1 st., 116, F. ad. A.

[2] Maurel-Lavallée, Mausière: Sur les retrécissements intrinsiques de l'œsophage, Th. Paris, 1865, p. 94.

[3] Lancet, 1847, i., 43.

[4] Med. Times, Lond., 1850, n. s., i., 58.

[5] "Toxicologie," i., 154.

[6] Huber: Ztschr. f. kl. Med., 1888, xiv., 502, F. 14m, A.

[7] Austral. Med. Jour., 1880, ii., 110.

[8] San Francisco Med. Press, 1860, i., 19.

The relative frequency of different periods of duration of fatal cases is shown in the following table, computed from cases reported in medical literature:

	Number of cases.	Less than 3 hours.	3 to 24 hours.	1 to 7 days.	1 to 4 weeks.	1 to 3 months.	More than 3 months.
Sulfuric acid.....	124	13	42	28	17	16	8
Hydrochloric acid..	40	2	18	6	4	6	4
Nitric acid.....	27	2	10	6	3	3	3

The shortest duration was in a case referred to by Thomson,[1] in which a child, while attempting to swallow strong sulfuric acid in mistake for water, died almost immediately, to all appearances from suffocation due to the action of the acid upon the glottis. None of the acid had reached the stomach. In a case of corrosion by nitric acid, a new-born infant died in about half an hour.[2] Sulfuric acid in the quantity of 104 c.c. (℥ iiiss.) caused the death of a man of fifty years in three-quarters of an hour.[3] The same acid has caused death in one hour in four cases: in one a man committed suicide by swallowing about 60 c.c. (℥ ij.) of the acid;[4] in another a woman died in great agony in one hour after accidentally taking a wineglassful of acid, of specific gravity 1.833;[5] and in still another a man of forty-eight years died in collapse in an hour after taking an unknown quantity.[6] A woman destroyed her husband in one hour by an injection of sulfuric acid into the rectum.[7] The shortest duration of fatal corrosion by hydrochloric acid was two hours in two cases: both of women who took it with suicidal intent.[8] Fauvel reports the case of a man of thirty years who suicided by taking about a glassful of a highly acid solution of mercuric nitrate containing hydrochloric acid, and died in two and three-quarters hours.[9]

When death results from the primary action of the acid it

[1] Lancet, 1836–37, ii., 835.
[2] Buchner: Friedreich's Bl. f. ger. Med., 1886, xxxvii., 9.
[3] Rapp: Gaz. méd. de Paris, 1850, 3 s., v., 926.
[4] Bell: Canada M. Jour., 1868, iv., 53.
[5] Traill: Monthly J. M. Sc., Edinb., 1854, xix., 138.
[6] White: Tr. Path. Soc. Lond., 1882–83, xxxiv., 96.
[7] Leconte: Thèse, Strasb., 1855.
[8] M. J., Friedreich's Bl. f. ger. Med., 1858, ix., 6 Heft. 70. Deutsch. Med. Ztg., Berlin, 1855, xxiv., 227.
[9] Tardieu: "Emp.," 2ème ed., 236.

usually occurs within forty-eight hours after the beginning of the action. But a longer duration has been observed in some cases. Thus a man of fifty-six years died suddenly on the fourth day after having taken a dessertspoonful of sulfuric acid.[1] Legg and Ormerod report the case of a man of twenty-one years who died in great agony five days after taking an unknown quantity of the same acid.[2] Huber gives a case in which death occurred on the seventh day;[3] one in which the patient died in sixteen days from the primary effects of 60 c.c. (℥ ij.) of sulfuric acid occurred in the service of Dr. Jenner;[4] and Mascarel reports a case whose duration was twenty-one days.[5] In some instances sulfuric acid has also caused death by pleuro-pneumonia in from ten to twenty days.[6] Cases of death from the primary action of hydrochloric acid in four and seven days,[7] and similarly from nitric acid in eight and thirteen days,[8] are also reported. When death is due to starvation from the secondary effects of the action of the acid the duration may be much longer. Thus cases of eleven, fifteen, and eighteen months' duration in which death was caused by sulfuric acid are recorded.[9] In one case of death from hydrochloric acid the victim survived for more than four months.[10] A man died in four months and twenty-two days after taking a mouthful of the same acid;[11] and a woman only succumbed to the effects of nitric acid in two years. It is highly probable that instances of still longer duration, terminating finally in death attributable to the action of each of the three acids, have escaped observation.[12]

Treatment.

The treatment indicated is the same whether sulfuric, nitric, or hydrochloric acid has been taken. As the action of the acid

[1] Guy's Hosp. Rep., 1859, 134.
[2] St. Barth. Hosp. Rep., 1876, xii., 261.
[3] Ztsch. f. kl. M., 1888, xiv., 490.
[4] Med. T. and Gaz., 1857, xv., 629.
[5] Bull. Soc. d'anat., Paris, 1840, xv., 299.
[6] Smoler: Wien. med. Halle, 1861, ii., 434. Maschka: Samml. Gutacht. Prag. med. Fak., 1858, 2 F., 224. Barth: Bull. Soc. d'anat., Paris, 1853, xxviii., 103.
[7] St. Geo. Hosp. Rep., 1877–78, ix., 18. v. Wundschheim: Prag. m. Wchnschr., 1891, xvi., 605.
[8] Erichsen: St. Pet. m. Ztschr., 1867, xii., 225. Warren: Extr. Rec. Bost. Soc. M. Impr., 1853, i., 78.
[9] Peters: Bull. Soc. d'anat., Paris, 1855, xxx., 153. Kühn: Diss., Berlin, 1838, p. 9. Katz: Diss., Berlin, 1872, p. 25.
[10] Ewart: New Zeal. M. Jour., 1888–89, ii., 241.
[11] Gehle: Berl. kl. Wochenschr., 1884, xxi., 337.
[12] See case cited by Tardieu (obs. iv.), "Emp.," 2ème ed., 239.

begins at once, and is the most energetic when it first comes in contact with the tissues, treatment must be as expeditious as possible, and although cases have occurred in which the patient has recovered without any treatment, many have died from the effects of relatively small doses who might have been saved by the timely administration of antidotes. The immediate indications are two: viz., to neutralize and to dilute the acid. The latter object is best attained by giving milk in large quantity, which also aids in neutralizing the acid by its alkalinity. The safest, although not the most rapidly acting, agent to fulfil the former indication is magnesium oxid (magnesia usta) or preferably precipitated magnesium hydroxid (milk of magnesia). Soap may also be used, and acts by virtue of the alkaline base which it contains. The carbonates and bicarbonates of sodium, potassium, calcium (chalk), and magnesium have also been frequently used and are generally recommended. Their introduction, however, into a stomach whose walls are thinned in places by corrosion cannot be unattended with the danger that the sudden generation of a large volume of gaseous carbon dioxid from the decomposition of the carbonate may cause perforation. Although we know of no reported case in which the death has been directly attributed to this cause, there are some in which perforation of the stomach would seem to have been aided at least by the exhibition of a carbonate.[1] It is true that the carbonates are more prompt in their action than the dry powdered magnesia, and that the latter has been found in the vomited matters, while these still retained a strongly acid reaction,[2] but in Walker's case above cited the contents of the intestine were found to be strongly acid although magnesium carbonate had been given. Swallowing is frequently difficult or impossible. When this is the case cocaine may control the pain sufficiently to permit deglutition. The administration of emetics is distinctly contraindicated. The introduction of the stomach tube is always attended with great danger of perforation of the œsophagus or stomach, and should not be attempted except as a last resort, when swallowing is impossible, for the purpose of introducing the antidote. If the pipe has been once safely

[1] Thomas: Austral. M. J., 1891, n. s., xiii., 542. Fagerlund: Vierteljschr. f. ger. M., 1894, 3 F., viii., supplhft., 56. Walker: Mthly. J. M. S., 1850, x., 538.

[2] Taylor: "Poisons," 3d Am. ed., 195.

introduced the stomach should be washed out with an abundance of milk alkalized with magnesia until the washings come away alkaline. Cases in which this method of treatment was successfully followed, even when a large quantity of the acid had been taken, have been reported by Mendelsohn [1] and by Brettner.[2] Even when the stomach was not wounded, death has been caused by the introduction of the stomach tube in a patient having a fatty heart.[3] The destructive effects of the corrosive are less violent when the acid has been mixed with food articles, or taken into a stomach containing food, than when it is taken in its own form into an empty stomach. The acid is also similarly partly neutralized and diluted when gruel, milk, or other liquid or semi-liquid foods are given after the corrosive has been swallowed. Gutman [4] observed that a gargle of salicylic acid diminished the pain in the mouth and throat and caused speedy cicatrization of the wounds there produced by the acid. He also recommends its internal administration to the same end. Narcotics are indicated to control the pain, and ice in fragments for the same purpose, as well as to relieve the intense thirst. In collapse stimulants should be given, and the body temperature should be raised by the hot pack or by hot-water bags. When death from suffocation is threatened tracheotomy should be performed. In a case reported by Patterson[5] the action of a small quantity of sulfuric acid did not extend to the stomach, and after the relief of serious interference with respiration by tracheotomy the case progressed favorably. Those in whom the secondary effects of the strong acids are manifested are subjects for surgical treatment, although life may be maintained for a time by nutritive pancreatized enemata, or, if the strictures be limited to the œsophagus, by daily dilatation with the sound. The operations of gastrostomy for relief of cardiac or œsophageal stricture, or of resection of the pylorus for pyloric stricture due to the action of mineral acids or alkalies, have been made in several cases.[6] In three instances after the operation

[1] Charité Ann. (1885-86), 1887, xii., 183.

[2] Méd. moderne, 1890, i., 261.

[3] Bundy: N. Y. Med. Rec., 1884, xxvi., 504.

[4] Wien. med. Presse, 1878, xix., 153.

[5] Glasg. M. J., 1879, xii., 390.

[6] Foster: Guy's Hosp. Rep., 1859, 3 s., v. 1. Weinlechner: Ber. . . . Rudolph Stift. Wien, 1879, 396. Masing: St. Pet. med. Wchnschr., 1885, n. F., ii., 310. Koehler: Charité Ann. (1886), 1888, xiii., 538, and Mendelsohn: *ibid.* (1885-86), 1887, xii., 183. White and Lane:

of gastrostomy the patient recovered,[1] and once after resection of the pylorus.[2]

SULFURIC ACID.

This, the most powerful of the mineral acids, is used in some stage of almost every manufacturing process in which chemical methods are utilized. It is known popularly as *oil of vitriol*, and the commercial article is an oily, heavy liquid, sometimes colorless, but usually having more or less of a brown color. Sometimes it is almost black. Its chemical formula is H_2SO_4, and its molecular weight is 98. When pure it crystallizes at 10°.5 (50°.9 F.) and boils at 338° (640°.4 F.). It is odorless. The specific gravity of the pure acid is 1.848 at 12° (53°.6 F.), and its mixtures with water have, according to Ure, the following gravities:

Specific gravity.....	1.848	1.837	1.811	1.767	1.712	1.652	1.597	1.539
Per cent. H_2SO_4.....	100	95	90	85	80	75	70	65
Specific gravity.....	1.486	1.436	1.388	1.344	1.299	1.257	1.218	1.179
Per cent. H_2SO_4.....	60	55	50	45	40	35	30	25
Specific gravity.....	1.141	1.101	1.068	1.033				
Per cent. H_2SO_4.....	20	15	10	5				

The commercial and C. P. (chemically pure, so-called) acids usually have a specific gravity of 1.835 to 1.845, and contain from 92.5 to 98.8 per cent. of the pure acid.

When sulfuric acid is added to water it produces a hissing sound and a marked elevation of temperature; 100° (212° F.) is reached by mixing four volumes of the strong acid with one of water, both ice-cold. This elevation of temperature may serve to attract attention and thus prevent an accidental or even homicidal administration if the dilution is made at the time.[3] Sulfuric acid absorbs water with great eagerness, and if exposed to the air rapidly increases in volume and diminishes in concentration by absorption of atmospheric moisture. The action of the acid upon organic substances, including animal tissues, is

Brit. M. J., 1891, i., 409. Duncan: Lancet, London, 1890, i., 797. Hadden: Tr. Path. Soc., London, 1889-90, xli., 84. Koehler: Deut. med. Wochnschr., 1890, xvi., 783. Postempski: Bull. Ac. roy. med., Roma, 1889-90, xvi., 436. Brug: Bost. M. and S. J., 1876, xcv., 481.

[1] Aigre: La France méd., 1878, xxv., 577. Haff: N. Orl. M. and S. Jour., 1880, n. s., viii., 16. Bryant: Lancet, Lond., 1881, i., 572.

[2] v. Eiselberg: Wien. med. Bl., 1889, xii., 360.

[3] See case of Catharine Wilson, p. 192.

largely due to removal of water or of its elements from the substance acted upon, which in some cases is thus carbonized.[1] Sulfuric acid, in the presence of water, is decomposed by some metals—such as zinc—with evolution of hydrogen and formation of the sulfate of the metal. At elevated temperatures it is decomposed by certain other metals—such as copper—and by carbon and organic substances, with evolution of sulfur dioxid, SO_2. The salts of sulfuric acid, called sulfates, are soluble in water, with the exception of those of barium, strontium, calcium (sparingly soluble), and lead. They are not soluble in alcohol. When mixed with charcoal and heated to redness the sulfates are reduced to sulfids.

An acid, known as *fuming* or *Nordhausen sulfuric acid*, is extensively manufactured in Bohemia for use principally as a solvent of indigo and of alizarin. Such solutions of indigo, used as *blueing* in laundry operations, have caused a number of deaths, suicidal and accidental, from the acid contained in them. The Nordhausen acid is a mixture or a combination of the ordinary sulfuric acid, H_2SO_4, and sulfur trioxid, SO_3; and is also known as *pyrosulfuric acid*, $H_2S_2O_7$ ($=H_2SO_4+SO_3$).

Sulfuric acid produces little alteration in the appearance of such liquid articles of diet as contain only small quantities of solid organic materials. Substances rich in albuminoid materials are modified by coagulation; and those containing sugar or starch are darkened or blackened.

Free sulfuric acid is not normally present in any part of the human body, although it has been found in the acrid secretions of certain gasteropods, notably in that of *Dolium galea*.[2] The sulfates are, however, constant constituents of most of the tissues and fluids of the body, being partly introduced from without, but principally produced by oxidation of the sulfur contained in the albuminoids. They are absent from the milk, bile, and gastric juice.

Symptoms.

The symptomatology of corrosion by sulfuric acid may be divided, if the patient survive for a sufficient time, into two pe-

[1] See "Post-mortem Appearances," p. 215.

[2] Gorup Besanez: "Physiolog. Chem.," 4te Aufl., 104. Maly: Monatsh. f. Chem., i., 205.

riods: First, the effects due to the *primary action* of the corrosive, more or less complicated with its action as a poison, sometimes designated as *acute* phenomena; and second, *secondary effects* resulting from the local action of the acid, improperly designated as *chronic*.

Primary Symptoms.—When the acid is taken by mistake for some other liquid by a person in the possession of his faculties, or when from any cause it is not swallowed, its effects are limited to those parts of the lips, mouth, and pharynx with which alone it comes into contact. Such parts are then cauterized, and covered with a grayish-white coating, resembling an application of white paint or wet parchment, which subsequently becomes darker or brown in color. The parts attacked are the seat of severe pain. The teeth where touched by the acid are of a chalky whiteness and are deprived of their polish. The acid may also in such cases, as well as in bungling attempts at administration to a child or unconscious adult, be scattered upon the chin, face, and breast, producing spots or blotches of a whitish or gray color, which subsequently become brown or even black; and similar stains, linear in character, may be caused by the acid flowing from the angles of the mouth. Death may occur rapidly in such cases from penetration of the acid into the larynx, and consequent œdema of the glottis or suffocation. These cases are, however, usually light, and terminate in complete recovery, except for the cicatrices which remain.

If the acid be swallowed, the act is attended or immediately followed by intense burning pain, extending from the lips to the epigastrium, so severe that the person usually cries out at the time of taking the acid. Subsequently the pain extends over the abdomen and chest, and the patient suffers excruciating agony. The abdomen is extremely sensitive to pressure, and later becomes distended. A few cases have, however, been reported in which the pain was slight, or ceased in a short time. A man of fifty-six years took by accident a dessertspoonful of oil of vitriol, after which he walked up-stairs and went to bed; he vomited slightly, and for the two following days seemed depressed, but presented no urgent symptoms. The case was considered to be a slight one and recovery was expected, but he died suddenly on the fourth day.[1] Chowne reports the case of a man

[1] Guy's Hosp. Rep., 1859, 134.

of fifty-two who took 15 c.c. (℥ ss.) of the concentrated acid with suicidal intent. He felt as if strangled, fell, and suffered burning pain in the pit of the stomach. The pain, however, subsided in an hour, the abdomen not being sensitive to pressure. After death, which occurred in forty hours, the stomach and duodenum were found to be extensively corroded, and the former thickened in places, although not perforated.[1] Scholtz has described the still more exceptional instance of a woman of twenty-nine years who took about 125 c.c. (℥ ivss.) of commercial oil of vitriol, and died in twenty-four hours in collapse. Although there was excessive burning pain in the throat, the abdomen was not distended, nor was it sensitive to pressure; yet the autopsy showed the fundus of the stomach to be corroded, the pylorus softened, the walls perforated in four places, and the liver and spleen reduced to a soft paste.[2]

The case above quoted from Guy's Hospital Reports bears upon the medico-legal question of whether in a fatal corrosion by a mineral acid a person is capable of any considerable voluntary movement after taking the acid. That this question must be answered in the affirmative is shown not only by this case but by others: A man of thirty years, who died from the effects of 57.3 c.c. (℥ xvss.) of sulfuric acid of specific gravity 1.842, was able to get out of bed and sit on the night stool twenty hours after taking the acid and five hours before his death.[3] A woman of sixty-seven years, who died in twenty-four hours, walked some distance to the place where she was found unconscious two hours later.[4] A man, who died in twenty-four hours from the effects of 22 c.c. (℥ vi.) of the acid, called a cab and was driven home after taking the acid. The only peculiarities observed by the driver were that the man was very pale and held a handkerchief to his mouth.[5] A man of fifty-two years took about 90 gm. (℥ iij.) of commercial sulfuric acid at nine o'clock, after having eaten some soup; he remained in bed until noon, when pain obliged him to call out. At one o'clock he got up, dressed himself unaided, and was transferred to the hospital, where he died five and one-half hours after taking the acid.[6]

[1] Lancet, 1847, ii., 35.
[2] Aerztl. Ber. allg. k.k. Krankh., Wien (1891), 1893, 70.
[3] Walker: Mthly. Jour. M. Sc., 1850, x., 538.
[4] Schad: Aerztl. Int. Bl., 1885, xxxii., 388, Case i.
[5] Lond. M. Gaz., 1845, xxxvi., 826.
[6] Stansky: Gaz. méd. de Par., 1837, v., 188.

Vomiting follows soon, without, however, bringing relief from the pain, and continues with more or less violence throughout the duration of the more active period of primary corrosion. It has, however, been known to cease early, as in a case reported by Geoghegan,[1] in which it ceased after three or four hours and did not recur, the woman dying in thirty-one hours. The vomited matters may be at first thick and slimy, but they are usually from the first and always soon become dark-brown or even black, thick, and grumous in consistence, the so-called "coffee-ground vomit," whose appearance is due to blood pigment acted upon by the acid. The vomit is strongly acid in reaction, a quality which is of great importance in establishing a diagnosis between corrosion by acids and that by alkalies, the symptoms of which resemble each other in many respects. But they may become alkaline from the administration of an excess of alkali as an antidote. They may also be effervescent when ejected shortly after administration of a carbonate,[2] and, if acid, they effervesce on contact with a carbonate (chalk or a limestone soil or floor). The vomit contains, beside blood pigment, abundance of epithelial cells and frequently patches or shreds of detached mucous membrane, to which parts of the muscular coat of the stomach or œsophagus, which may be brown or black in color, are attached. When the acid vomit is discharged or spattered upon the clothing or other textile fabrics it produces stains similar to those caused by the acid (see Stains, below). If "liquid blueing" have been taken the vomited matters and sometimes the alvine dejections are colored by the indigo. There is severe thirst which is difficult to relieve, owing to the intense pain caused by deglutition, which act is, indeed, frequently impossible, and the intense vomiting provoked by the entrance of any substance into the stomach. There is great difficulty in breathing, caused partly by œdema and swelling of the tongue and throat in some cases, and by the pain caused by contraction of the abdominal muscles in almost all. Later in the case, if the patient survive, respiration is further interfered with by pleuro-pneumonia, developed by the toxic action of the absorbed acid, or in exceptional cases by perforation of the stomach and diaphragm and direct entrance of the acid into the pleural cavity.

[1] Lond. M. Gaz., 1851, xlviii., 328. [2] Walker: Mthly. Jour. M. S., 1850, x., 538.

The swelling of the tongue and glottis also causes more or less modification of the voice and sometimes entire loss of speech. If death does not result rapidly from œdema of the glottis, it may still occur early and suddenly from collapse or shock, due to perforation and acute peritonitis or extensive hemorrhage from corrosion of the walls of large vessels.

If the patient escape a rapidly fatal conclusion, the epigastric pain persists or increases in intensity and extends over the abdomen and into the chest. Deglutition is more and more painful and difficult, or continues to be impossible, and every attempt to swallow provokes a violent fit of vomiting. There is abundant salivation of a ropy character, sometimes tinged with blood, and sometimes forming a brown or even black froth, discharged from the mouth and nostrils. The breath is often very fœtid. There is obstinate constipation, and such evacuations as are passed are dark brown or even black. Exceptionally there is diarrhœa, the stools containing altered or unaltered blood and shreds of mucous membrane. A condition of collapse supervenes, with an almost imperceptible or thready, frequent, and small pulse, diminished temperature, cold surface, sunken eyes, an anxious countenance, dilated pupils, and a cold, clammy perspiration bathing the skin. This condition may terminate in death within twenty-four hours, the patient being usually unconscious, or sometimes having violent convulsions or a severe attack of suffocation. In one case death was preceded by furious delirium.[1]

After the first day in severe cases the foregoing symptoms persist or even increase in intensity, the respiration becomes more seriously affected, and râles are heard on auscultation. The parts burnt by the acid become ulcerated and the temperature rises. The patient may die during this period, in from three to eleven days, either from exhaustion or in sudden collapse. When the course of the corrosion tends to recovery the ulcerated wounds heal more or less rapidly, and sometimes very slowly, leaving cicatrices, thickening, and vegetations which are the causes of the secondary or "chronic" phenomena.

The condition of the urine during the course of a corrosion by sulfuric acid indicates that, in many instances at least, the acid acts upon the kidneys. The urine is retained from the first,

[1] Kast and Rumpel: "Illustr. Path. Anat.," 1893, Case i.

and must be drawn with the catheter. It is diminished in quantity, at first of high specific gravity, 1.035–1.045, falling gradually to 1.015–1.010. It is at first hyperacid. The elimination of sulfates is relatively high, if the small quantity of urine passed be considered; but the absolute amount in twenty-four hours, which affords the only true indication of the elimination, is rather below than above the normal of 2.5 to 3.5 gm. (38.6 to 54 grains) in twenty-four hours, except upon the first day or two. We know of no case in which the saliva, which is abundantly discharged after the first or second day, has been analyzed. It seems probable, however, that a part at least of the elimination is in this secretion and by the stomach, as when hydrochloric acid has been taken.[1] In a case of Letheby's, a boy of nine years during the first four days eliminated sulfates corresponding to 4.018, 2.592, 1.185, 0.823 gm. (62, 40, 18.3, 12.7 grains).[2] At a later period in cases tending to recovery the absolute elimination of sulfates is diminished. Thus in the case of a woman of twenty-one years who recovered, reported by Hoppe-Seyler,[3] the total amounts of H_2SO_4 eliminated on the sixth to ninth days were: 1.6865, 1.8901, 1.6209, 0.936, 1.1817 gm. (26, 29, 25, 14, 18 grains). On the fifth and sixth days the amount in the form of sulfo-conjugate compounds was 0.2537 and 0.2583, or 5 : 1 and 6.3 : 1. As the normal elimination of sulfo-conjugate compounds is on an average 0.25 gm., but varies between 0.617 and 0.094, and is in proportion to simple sulfates as from 6 : 1 to 15 : 1,[4] it appears that the elimination of sulfo-conjugate compounds remains unaltered, while that of the simple sulfates is diminished—a result the opposite to that which would be expected if elimination of sulfuric acid continues to take place by the urine[5] later than the first two or three days. In the same case the urine was found to contain acetone on the fifth to eighth days in progressively diminishing amount, and during the period of inanition due to the pain incident upon deglutition. The urine also contains excess of earthy phosphates, and has also been observed to contain excess of calcium oxalate[6] and indican.[7] The color of the urine is sometimes yellow, more

[1] See p. 236.
[2] Lancet, 1847, i., 43.
[3] Ztsch. f. kl. Med., 1883, vi., 478.
[4] Hammarsten: "Physiol. Chem.," Mandel's transl., p. 363.
[5] See Schuchardt in Maschka's "Handb. d. ger. Med.," ii., 70.
[6] Smoler: Wien. med. Halle, 1861, ii., 434.
[7] Wyss: Arch. d. Hlk., 1869, x., 184.

frequently reddish or reddish-brown. The urine of a boy of two years who died in seven and one-half hours after swallowing a quantity of liquid blueing was distinctly blue.[1] Frequently the urine contains albumin, sometimes in large amount.[2] But in mild cases, or in the later days of those which progress favorably, it is absent.[3] In a case of suicide, terminating in recovery, in which the acid was taken diluted with alcohol, no urine was passed in thirty-six hours, when 700 c.c. free from albumin were drawn off.[4] The urinary sediment, when albumin is present, contains casts, hyaline, faintly granular or brown; and sometimes much granular matter colored with hæmatin,[5] and occasionally, though rarely, pus and blood.[6] The condition of the urine during a non-fatal case is well shown in one of Mannkopf's cases,[7] a boy of sixteen years who was under observation for about five weeks; the quantity in twenty-four hours only exceeded 1,000 c.c. (about ℥ xxxiv.) on two days, the twenty-eighth and twenty-ninth. The specific gravity was 1.038 at the outset, slowly fell to 1.015 during the first four weeks, and then again rose to 1.027. On the first day there was a little albumin, less until the end of the third week, when it became abundant, and again gradually diminished to disappearance. The quantity of casts kept pace with that of albumin. The elimination of sulfuric acid during the first three days was 2.51, 1.81, 1.49 gm. In some instances there is diuresis accompanied by constipation during convalescence.

The temperature is not similarly affected in all cases. In one fatal case in which death occurred in twenty-four hours it was subnormal, 36°.2 (97°.1 F.);[8] while in another, fatal in the same period, it reached 38°.9 (102° F.).[9] In one protracted case in which death occurred in one month, the temperature on the second day was 37°.8 (100° F.), and fell progressively with some oscillations to 35°.9 (96°.6 F.) on the day preceding death.[10] In another case, in which death occurred in thirty-seven days, the

[1] Deslandes: Nouv. bibl. méd., 1825; *ex* Galtier: "Tox.," i., 206.

[2] Katz: Diss., Berl., 1872, p. 9.

[3] Smoler: *Loc. cit.* Hoppe-Seyler: *Loc. cit.* Wyss: *Loc. cit.* Mendelsohn: Charité Ann. (1885–86), 1887, xii., 183, Case ii.

[4] Gottschalk: Diss., Berl., 1869, p. 22.

[5] Bamberger: Wien. med. Halle, 1864, v., 301, 309.

[6] Med. T. and Gaz., 1857, xv., 629.

[7] Wien. med. Wchnschr., 1862, xii., 25, Case iv.

[8] Scholtz: Aerztl. Ber. allg. k. k. Krankenh., Wien (1891), 1893, 70.

[9] Katz: Diss., Berl., 1872, Case iv., p. 21.

[10] Graeffner: Diss., Breslau, 1875.

temperature on admission was 36°.5 (97°.7 F.), rose the next day to 37°.5 (99°.5 F.), and oscillated during the entire time between 37° (98°.6 F.) and 40°.5 (104°.9 F.), reaching the maximum shortly before death.[1] In a severe case referred to by Gruhn,[2] during the fourteen days in which the patient was under observation, the temperature varied but slightly from the normal, the extremes having been 36°.8 (98°.2 F.) and 38° (100°.5 F.).

One of the five instances in which sulfuric acid has been injected into the **rectum** is quoted by Schuchardt, who comments that but little of the acid could have been actually injected, as both patients, mother and child, to whom it was given by mistake, recovered in a few days.[3] Hofmann's two cases are merely mentioned in a note, without any particulars.[4] In the two remaining cases there was immediate, intense pain, causing the person to cry out loudly. A physician gave sulfuric acid in an enema in mistake for linseed oil; the administration was made at 11 P.M., the patient passed the night in great agony, and in the morning a part of the corroded intestine was found to have been discharged, and the bed clothing to have been burnt by the discharges.[5] The other case was one in which a woman murdered her husband by injecting the acid in place of a medicinal enema. The man, who was on his knees, withdrew, and his back and buttocks and the bed clothing were extensively burnt by the acid. He died in one hour from acute peritonitis, the acid having perforated the gut and entered the peritoneal cavity.[6]

In the one case in which sulfuric acid was injected into the vagina, a woman, the mother of four children, sought to abort her fifth pregnancy by injecting half a litre of the acid. There were severe pain and inflammation of the parts, followed by a bloody, purulent discharge and subsequent cicatrization, which so constricted the passage that delivery was impossible. A dead child was removed by Cæsarean section and the woman

[1] Gruhn: Diss., Berl., 1870, Case iii., p. 19.

[2] *Loc. cit.*, Case iv., p. 28.

[3] Schuchardt, in Maschka's "Handb. d. ger. Med.," ii., 67; *ex* Deutsch. Preuss. Med. Ver. Ztg., 1848, No. 13.

[4] "Lehrb. d. ger. M.," 1891, 5te Aufl., 648.

[5] Jour. d. chim. méd., etc., 1835, 2 s., i., 425.

[6] Leconte: Thèse, Strasb., 1855, p. 27.

died also. The vaginal canal was found to be completely occluded, and the os uteri also closed by adhesion to the vaginal wall.[1]

Secondary Symptoms.—When the violent primary effects of the acid are withstood, the corroded parts slowly undergo repair, and the patient may be discharged in a few days or weeks as cured. The internal burns have, however, been so severe that from destruction of the gastric mucous membrane with its secreting glands, from the thickening and contraction due to cicatrization, from continuation of ulcerative processes, and even from persistence of effects upon remote organs, the individual subsequently becomes a subject for medical or surgical treatment, and usually finally succumbs to the remote effects of the corrosive.

The condition usually met with is one of starvation and inanition, caused partly by absence of the normal gastric secretion, and partly by strictures of the œsophagus or stenosis of the openings of the stomach, which mechanically prevent the passage of the food or chyme. In cases in which the vomit in the secondary stage has been analyzed, it has been found to contain no hydrochloric acid,[2] and lactic acid is only present when food has been introduced into the stomach.[3] Mendelsohn's Case II. recovered, however, under prolonged treatment, the quantity of acid taken having been 7 c.c. (about ʒ ij.), and hydrochloric acid, absent from the stomach during the previous history of the case, began to make its appearance about four months after the first attack, and increased in amount thereafter. Strictures of the œsophagus are frequently recognized during life and observed at the autopsy; and Maurel-Lavallée has reported the case of a woman, living two years after the primary attack, who was still throwing out shreds of necrosed tissue from the pharynx, œsophagus, and stomach, while her œsophagus was contracted in three places.[4] In the stomach sometimes the cardiac orifice and sometimes the pyloric is almost closed by cicatricial thickening. Instances are also reported in which death has been

[1] Lombard: Jour. d. chim. méd., etc., 1831, vii., 312.

[2] Kast u. Rumpel: "Ill. path. Anat.," 1893, Case ii. Masing: St. Pet. med. Wchnschr., 1885, n. F., ii., 310. Mendelsohn: Charité Ann. (1885–86), 1887, xii., 183, Cases ii., vii.

[3] Mendelsohn: *Loc. cit.*, Case ii.

[4] Mansière: Thèse, No. 94, Paris, 1865.

caused after long intervals by ulceration and perforation. A man, six months after an attempt at suicide by sulfuric acid, was attacked suddenly with hæmatemesis, and died in a short time. At the autopsy an artery capable of admitting a crow's quill was found to have been perforated, and the stomach was full of black, coagulated blood.[1] In another instance a woman died, eleven months after the primary attack, of peritonitis caused by a perforation of the intestine at the junction of the cæcum and ascending colon.[2] That death may occur suddenly is shown by a case reported by Lesser,[3] of a woman of thirty-five years who fell dead while at the wash-tub. During the morning her appearance and actions had not differed from those of the week preceding. At the autopsy a circular gastric ulcer and total peritonitis were found to exist.

Among the remote effects which have been observed in the course of the secondary action are: Intercostal neuralgia, noted in one case by Mannkopf;[4] eruption of red blotches on the skin of the forearm, also once observed on the twentieth day;[5] and frequently coughing, expectoration, pains in the chest, dyspnœa, and modifications of the normal respiratory sounds and resonance, which are, however, more commonly met with as symptoms of protracted primary action.

Diagnosis.

The diagnosis of corrosion by a mineral acid is usually plain from the character of the violent symptoms, from the history of the attack, and from the appearance of the stains upon the skin, lips, and clothing, if such exist. The only question which remains to be determined is whether the corrosive taken was a mineral acid or a mineral alkali. The settlement of this point only requires a testing with litmus of the matters first vomited or any remains of the liquid taken as to its reaction, which will be strongly acid in the one case and strongly alkaline in the other. It is not necessary to distinguish between free acids and acid salts for the purposes of treatment in most cases,

[1] Charcellay: Bull. soc. d'anat., Paris, 1836, xi., 171.
[2] "Atl. d. ger. M.," i., 16.
[3] Peters: Bull. soc. d'anat., Paris, 1855, xxx., 153.
[4] Wien. med. Wochenschr., 162, xii., 25, Case ii.
[5] Galtier: "Tox.," i., 176.

but if it be desirable to do so, paper charged with Congo-red or benzo-purpurin may be used for the purpose.

It is of no clinical significance whether the acid is sulfuric, nitric, or hydrochloric, as the treatment is the same for the three acids. But the question is sometimes one of forensic importance, and may be subsequently solved by an examination of the vomited matters by the method to be described below.

Prognosis.

The prognosis depends upon the amount of the acid taken and upon whether it has reached the œsophagus or stomach or not. If the quantity taken have been small, or if it have been expectorated and none swallowed, recovery is assured after the danger of death by suffocation has passed, although more or less deformity from cicatrization may remain. But if the acid have been swallowed the prognosis is unfavorable. Of the 303 cases referred to on page 189, including those in which the acid was not swallowed, and light as well as severe cases, the mortality was 68.3 per cent., of which 10.5 per cent. died in less than three hours, 44.4 per cent. in the first twenty-four hours, 66.9 per cent. within a week, 80.6 per cent. within a month, and 19.4 per cent. in periods longer than one month.

Even when the primary effects have been recovered from, if the acid have penetrated beyond the mouth, the prognosis must be guarded, as there can be no certainty that strictures will not be developed in the stomach or œsophagus. In any event complete recovery is most exceptional, as the destruction of gastric mucous membrane will entail malnutrition, even if the cicatrization be not sufficient to produce dangerous stricture. For the same reason the ultimate benefit of surgical interference for the relief of closure of the orifices of the stomach is highly problematic (see page 200).

External Application—Vitriol Throwing.

Accidental burns of the skin of the face, hands, and even of parts usually covered by clothing, have occurred in chemical laboratories and in manufacturing establishments in which the acid is used; but it is chiefly in connection with the crime of *vitriol throwing* that this branch of the subject is of forensic

interest. According to Christison,[1] this crime originated in Glasgow during the quarrels in 1820 between masters and workmen regarding the rate of wages, and became at last so frequent that it was made a capital offence by a special enactment. Vitriol throwing was not unknown in Germany at about the same time, as Bopp[2] relates the case of a woman who, in 1825, attacked another woman and her child with vitriol, and was convicted of attempt at murder in 1828. The offence is usually prompted by jealousy, and is one of quite frequent occurrence in the United States. The liquid, which is generally thrown into the face of the victim from a bottle, but sometimes in mere malice upon the clothing only, is in most cases sulfuric acid. Exceptionally nitric or hydrochloric acid has been used.[3]

Christison[4] has recorded the case of Mr. Campbell, upon whom a large quantity of acid was thrown. The local effects were severe, involving the loss of one eye, and he died in twelve days, apparently from pleurisy. Another fatal case is reported by Zwicke.[5] The man upon whom the acid was thrown sank down immediately, overwhelmed with pain. At the hospital he vomited greenish masses, complained of pain in his chest, but not in the stomach. The skin of the face was covered with whitish eschars from the forehead to the throat; the cutis of the left ear was reduced to a slimy pulp; the mucous membrane of the nose and lips was corroded; both corneæ were opaque; and there were corroded spots on the tongue and the ulnar side of the forearm. Pulse 110, respiration normal. Small râles in the lungs. The urine formed a brick-dust deposit and contained a large quantity of albumin. The next day he complained of apnœa; the respiration 20; the voice hoarse; and the tongue œdematous. Temperature 37°.2 (99° F.), pulse 108. At 10 P.M. he was very uneasy. Respiration 28; pulse 140, small. The respiration was very labored, with tracheal râles, but no cyanosis. The collapse increased, and he died in forty-eight hours after the injury. The autopsy showed the existence of chronic catarrhal enteritis, parenchymatous nephritis, multiple fibrinous pneumonia, multiple pleuritis, myocarditis, and partial corrosion of the posterior wall of the larynx and trachea, besides the

[1] "Poisons," Am. ed., 122.
[2] Ztschr. f. Staatsarznk., 1836, 22 Erghft., 256.
[3] See Stains, p. 227.
[4] Edinb. M. and S. Jour., 1829, xxxi., 230.
[5] Charité Ann., 1882, ix., 367.

external injuries. Still another fatal case, in which the acid had not only attacked the face, but had also been swallowed and respired, is reported by Liman.[1] In 1894 the secretary of the Danish Legation in London died the next day after sulfuric acid had been thrown in his face by an unknown man to facilitate robbery. In most instances, although the local injuries are severe, and may involve the loss of one or both eyes and serious disfigurement, the victim recovers.

As the action of sulfuric acid upon the skin is not absolutely instantaneous, its effects are greatly mitigated if the parts be immediately washed with a large quantity of water or a weak alkaline solution, as of sodium bicarbonate.

It is sometimes necessary to determine whether an external burn was caused by fire or by sulfuric acid. Thus in a case investigated by Maschka,[2] a man's assertion that the injuries upon a child were not produced by acid but by fire, was verified. For the points of distinction, see Vol. I., p. 641,[3] and below under "Examination of Stains," p. 227.[4]

Post-Mortem Appearances.

The appearances observed at the autopsy after death from sulfuric acid will differ markedly, according as the death was caused by the primary or by the secondary action of the acid.

Primary.—After death from the primary action of any of the mineral acids, it is difficult to estimate what portion of the changes observed were produced during life, and how far they resulted from the continued action of the corrosive after death. Thus in the case of a person found dead, in which there is no history of symptoms observed during life, and in which an unknown interval has elapsed between the death and the autopsy, extensive destruction of tissue may have been in great part the result of post-mortem action.[5] Usually, however, the nature of

[1] Casper-Liman: "Handb. d. ger. Med.," 8te Aufl., ii., 469. Other cases are reported by Orfila: "Tox.," 5ème ed., i., 156. Leconte: Thèse, Strasb., 1855, p. 3. Letheby: Med. Times, Lond., 1850, n. s., i., 58.

[2] Gutacht. Prag. med. Fak., 1853, i., 121.

[3] In the case there quoted from Taylor the acid used was nitric, not sulfuric.

[4] See also Schuchardt in Maschka's "Handb. d. ger. Med.," ii., 77, and the experiments of Samuel: Arch. f. path. Anat., etc., 1870, li., 41.

[5] For a case in illustration see Gull: Lond. Med. Gaz., 1850, xlv., 1102.

the symptoms will throw sufficient light upon the question whether perforation, etc., were ante-mortem or post-mortem.

Putrefaction is frequently delayed, possibly by reason of the diminished alkalinity.[1] The skin, if the acid have touched it, is marked with brownish, rust-colored, or black-brown stains, most frequently seen running linearly from the angles of the mouth and on the chin. At these points the epidermis is wanting, and the derma is parchmentized, the cells of the rete Malpighii have a brown-red color, and in the corium there are transudations from the blood-vessels, causing a diffuse dirty-reddish color. In the lips, mouth, and tongue the appearances noted during life are observed. The lips may be black; the tongue is sometimes softened, and has the appearance of having been boiled. The mucous membrane of the pharynx and tongue are in some cases gray in color. The œsophagus is corrugated, has a worm-eaten appearance, is more or less extensively deprived of mucous membrane, and dark-gray or even black in color. Where it still exists, the mucous membrane is easily detached in shreds. Contraction of the œsophagus has also been observed. Thus in a case of death in twenty-four hours there was extreme narrowing of the œsophagus, whose walls were greatly thickened and deprived of mucous membrane.[2]

The stomach presents the most characteristic lesions. If the organ be entire its external surface appears injected, blue-black, gray, or violet, in places where it feels to the touch thinned and filled with liquid. The contents consist of a brown, highly acid liquid, having the appearance of coffee containing a considerable amount of grounds; or are even black. The mucous surface is pretty evenly brown-black or black in color, very much thinned in places, sometimes to the verge of perforation, and frequently spotted here and there with islands of less altered or unaltered patches.[3] The black appearance of the stomach and other parts has been considered as produced by a carbonizing action of sulfuric acid similar to that which it exerts upon wood, sugar, etc. But Lesser has shown that it is

[1] See Casper-Liman: "Handb. d. ger. Med.," 8te Aufl., p. 472, Case 193.

[2] Virchow: Charité Ann., 1879, iv., 788.

[3] Good colored plates of post-mortem appearances after death from the primary effects of sulfuric acid will be found in Kast and Rumpell: "Illustr. path. Anat.," 1893, F., i., and in Lesser. "Atl. d. ger. Med.," 1883, Taf. iii.; iv., Figs. 1, 2; xv., Figs. 1, 4, 5.

due to decomposition of hæmoglobin, with formation of hæmatin.[1] The pylorus is frequently thickened and parchment-like, and sometimes has a shredded appearance. Partially healed ulcerations are sometimes met with. In an early case in which death occurred in five hours, Desterne[2] found the stomach exteriorly of a brilliant black color and red in spots. The mucous membrane was converted into a thick layer, split in all directions, dry, and of a brick-red color in places, which contrasted strangely with the brilliant black of the remainder. In about one-third of the cases the stomach is found perforated. But, unless the autopsy is made very early, it is not possible to determine from the appearances to what extent the acid has continued to act after death. Perforation from ulceration, which may occur when the duration of primary action is sufficiently prolonged, is, of course, produced during life. The disorganization of the stomach in severe and rapidly fatal cases may even extend to destruction of large areas. Thus in the case of a woman of twenty-three years who died in three hours in a comatose and collapsed condition, the autopsy, made twenty hours after death, showed the great curvature of the stomach to have been completely dissolved, and a dark, grumous liquid discharged upon the intestines and omentum.[3]

The acid which escapes from the perforated stomach has in several cases produced disorganization of the surrounding parts. In a case reported by Otto,[4] of six hours' duration, the stomach was found extensively disorganized, the posterior part of the spleen black and corroded, the left lobe of the liver black-brown, the duodenum externally and internally gray-black and corroded, the left kidney greenish-black and deeply corroded in lines, and the mesentery and left side of the peritoneum to the pelvis blue-black. Scholz[5] has reported the case of a woman of twenty-nine years who died in twenty-four hours after taking 125 c.c. (about ℥ ivss.) of the undiluted acid, and the autopsy was made twelve hours after death. The stomach, which was blackened and softened, was perforated in four places, the adjoining perito-

[1] Arch. f. path. Anat., etc., 1881, lxxxiii., 198.

[2] Bull. soc. d'anat., Paris, 1848, xxiii., 223.

[3] Sewell: Canada M. Jour., 1852–53, i., 131.

[4] "Memorabil.," Heilbronn, 1870, xv., 295.

[5] Aerztl. Ber. allg. k. Krankenh., Wien (1891), 1893, 70.

neum was softened, and the omentum in places glued to the abdominal wall by fibrinous exudation. Between the abdominal wall, the liver, and lesser omentum was a sero-purulent liquid containing gas; and the liver and spleen were a soft pulp. Or the diaphragm may even be perforated. In an early case reported by Willndovius,[1] the suicide died in two hours. The stomach, in spite of the most careful handling, tore into pieces, and its coats could not be distinguished; only a few tatters of the omentum remained; the diaphragm was soft and corroded in places; the lungs were adherent to the pleuræ, and their exterior brown and leathery to the depth of two lines. In another instance, reported by Moore,[2] in which death occurred in an hour and a half, the œsophagus and diaphragm were both involved; the œsophagus was saturated with the acid, which had soaked through and saturated the pericardium and back part of the heart. At the base of the lung one-eighth of the lung tissue was hardened. The acid may also attack the blood in the vessels, which it coagulates, hardens, and blackens, producing an appearance of black arborescence in the smaller vessels, and the formation of hard black casts, like sticks of licorice, in the larger vessels.[3] The cavities of the heart have also been found distended with dark clots, which were acid in reaction, the post-mortem having been made thirteen hours after death.[4]

In most instances the changes do not extend beyond the stomach, but congestion, ecchymoses, swelling, and œdema of the mucous membrane of the duodenum and upper small intestine, and ulcerations of the upper and lower small intestine, as far as the ileo-cæcal valve, are frequently observed. Indeed, in some cases the duodenum has been blackened and corroded in the same manner as the stomach,[5] while in other instances the corrosion of the duodenum has advanced to perforation.[6] In one case intestinal stricture was observed. Death had followed the swallowing of the acid in two hours. The mucous coat of

[1] Jour. d. prakt. Heilk., 119, xlix., 3 St., 56.

[2] Brit. M. Jour., 1879, i., 430.

[3] Haldane: Edinb. M. Jour., 1862, vii., 739. Stausky: Bull. soc. d'anat., Paris, 1836, xi., 298. Grizolle: *Ibid.*, 1835, x., 132. Moore: Tr. Path. Soc., Lond., 1879, xxx., 297.

[4] Walker: Mthly. Jour. M. Sc., 1850, x., 538. Leyden and Munk: Arch. f. path. Anat., etc., 1861, xxii., 237, Case i.

[5] Corfe: M. Times and Gaz., 1848, xvii., 258. Leyden and Munk: *Loc. cit.*, Case ii.

[6] Thomas: M. Times and Gaz., 1873, ii., 92. Walker: Mthly. Jour. M. Sc., 1850, x., 538. Maschka: Vierteljschr. f. ger. Med., 1881, n. F., xxxiv., 197.

the small intestine was much shrivelled, the valvulæ conniventes standing out like thick cords, so as to entirely obliterate the canal.[1]

The larynx and trachea are the seat of serious lesions if the acid have passed the epiglottis. In Walker's case, above mentioned, the larynx and trachea, to within an inch of the bifurcation, were swollen and congested, of a coffee color, and deprived of mucous membrane. The epiglottis was bleached and shrunken to half its natural size. In a woman who died in thirty hours, the larynx and trachea contained a gray, rather abundant liquid, and a gray adherent membrane which extended into the larger bronchi. The smaller bronchi contained much thick, opaque, light-greenish secretion.[2] A woman was found dead in her chair, the face black, with a bottle of sulfuric acid in her hand. She had probably been dead about two hours. The acid had not reached the stomach, but had corroded the epiglottis. The rings of the trachea were quite dissected out and the lungs charred. The acid had escaped into the pleura, had dissolved a part of the ribs on the right side, and had formed a crust of calcium sulfate on the lungs. The thoracic vessels contained a solid magma of decomposed blood, looking like dried blacking.[3] In one case the corrosive action of the acid had established communication between the œsophagus and the left bronchus.[4]

Among the remote effects are evidences of pleuro-pneumonia, emphysema, and hyperæmia in the lungs, of parenchymatous nephritis in the kidneys, and of fatty degeneration in the liver.[5]

Secondary.—The usual post-mortem appearances after death from the secondary action of sulfuric acid are in marked contrast with those found when death has been due to its primary effects. They more closely resemble those found when the fatal result has followed the secondary action of the other corrosives. The body is greatly emaciated. The subcutaneous panniculus is almost free from fat, which has also in great part dis-

[1] Willndovius: *Loc. cit.*

[2] Katz: Diss., Berl., 1872, p. 29.

[3] Letheby: M. Times and Gaz., 1850, n. s., i., 58, Case iii.

[4] Nagel: Ungar. Ztschr., 1851, i., 48.

[5] For microscopic appearances after death from primary and secondary action of sulfuric acid see Fernbacher: "Ein Fall von Schwefelsäure-Vergiftung," Diss., München, 1890. Bamberger: Wien. med. Halle, 1864, v., 301, 309. Löwer: Ber. kl. Wchnschr., 1864, i., 385. Lesser: "Atl. d. ger. Med.," Pl. xv., Figs. 7, 8; Pl. xvi., Fig. 6. Fraenkel u. Reiche: Arch. f. path. Anat., etc., 1893, cxxxi., 130.

appeared from or suffered myxomatous degeneration in internal parts where it is usually present even in very thin subjects, as in the omentum and about the kidneys. The eyes appear sunken from removal of fat from the orbit. This emaciated condition has also been observed in cases of as comparatively short duration as five days.[1] If the local action of the acid upon the skin, particularly at the angles of the mouth, lips, tongue, and buccal and pharyngeal mucous membrane have been severe, the points of its action may be marked by eschars, or by whitish, hardened, contracted cicatrices. But if the acid have not come in contact with any one of the parts mentioned, or even if it have there occasioned less severe injuries, no changes in the skin, lips, mouth, or tongue may be observed, even in cases of comparatively rapid course.[2] The œsophagus is more or less extensively ulcerated, cicatrized, deprived of mucous membrane, contracted, and its walls thickened. In a man who died in three months from the effects of a mouthful of the acid the mucous membrane of the œsophagus, with the exception of the upper six centimetres, was one diffuse ulcer, increasing in depth toward the cardia, with two small necrotic spots in the upper part. The œsophagus was not contracted, but somewhat dilated throughout its length.[3] The strictures of the œsophagus are sometimes single and located at the lower part, but more frequently multiple, and sometimes alternated with dilatations.[4] The color of the œsophageal mucous membrane is gray, dark gray, or red-brown, but has been found blackened in places in a case of three months' duration.[5] The cicatrized appearances due to the action of the corrosive may also be accompanied by more recent lesions, linear ecchymoses, or hemorrhages, caused by the too forcible introduction of the sound in attempts at dilatation.[6] The œsophagus has also been found perforated by late ulceration in a child which died in forty-five days.[7] The stomach is contracted, its walls thickened, its mucous membrane either wanting and replaced by more or less extensive cicatrices, or the seat of catarrhal in-

[1] Legg and Ormerod: St. Barth. Hosp. Rep., 1876, xii., 261.

[2] Casper-Liman: "Handb. d. ger. Med.," 8te Aufl., ii., 472, Case 193. Niemann: Ztsch. f. Staatsarznk., 1862. lxxxiii., 179.

[3] Kast and Rumpell: "Illustr. Path. Anat.," Lond., 1893, F. ii.

[4] Wyss: Arch. d. Heilk., 1869, x., 184. Katz: Diss., Berl., 1872 p. 28.

[5] Niemann: *Loc. cit.*

[6] Lesser: "Atl. d. ger. Med.," i., 30.

[7] Husson: Bull. soc. d'anat., Paris, 1836, xi., 103.

flammation, and of a yellow-buff color.[1] Very frequently thickening at the pylorus causes more or less complete stenosis. The stomach has also been found attached to the liver, pancreas, omentum, or abdominal wall by old adhesions.[2] The duodenum and upper small intestine present changes similar to those observed in the stomach.

Analytical.

The materials to be analyzed may be either the viscera or vomit of the victim; the remains of articles of food or other material supposed to have been the vehicle of administration; or stains upon the clothing or elsewhere.

The Contents of the Stomach or Vomit.—These are usually strongly acid in reaction; but neither does a very distinct acid reaction indicate conclusively the presence of abnormal free mineral acid, nor does its absence prove that none has been present. Not only is the reaction of the stomach contents normally acid during life, from the presence of free hydrochloric or lactic acid, or both, but very soon after death the alkaline reaction, existing in all parts of the body other than the stomach and urine during life, rapidly gives place to an acid reaction, caused by the formation of volatile fatty acids, lactic acid, and, exceptionally, oxybutyric acid, as products of post-mortem decomposition. As these acids are organic, and as the contents of the stomach may normally contain the salts of hydrochloric, sulfuric, and even nitric acid, the first point necessary in the analysis, after suitable extraction, is the proof of the presence of a **free mineral acid**.

The existence of an alkaline reaction, or the absence of acidity, while it is not evidence that corrosion by a mineral acid has not occurred,[3] indicates the impossibility of direct chemical evidence of the presence of a free acid, and limits the value of the analysis to such confirmatory proof as is afforded by the presence of an undue quantity of the salts of the acid, or an un-

[1] For good colored plates see: Lesser: "Atl. d. ger. Med.," Pl. v., Fig. 2. Kast and Rumpell: "Ill. Path. Anat.," F. ii.

[2] Ackermann: Deut. m. Wochenschrift, 1894, xx., 835. Masing: St. Pet. m. Wchnschr., 1885, n. F., ii., 310. Graeffner: Diss., Breslau, 1875, p. 15. Wyss: *Loc. cit.*

[3] For cases in illustration see Maschka: Gutacht. Prag. med. Fak., 1867, 3 F., 284. Buchner: Friedreich's Bl. f. ger. Med., 1886, xxxvii., 9.

usual form of its combination. Such a condition may arise from one of two causes: 1. The administration of antidotes in excess, in which event the stomach contents or vomit will be found to contain a quantity of the combined acid much in excess of that which can be normally present, particularly if the acid be nitric or sulfuric, accompanied or not by an excess of the basic substance (magnesium oxid, etc.) which was given as an antidote. 2. In many cases the corrosive is so completely expelled by vomiting, even within a few hours, that none remains. In Schauenburg's case,[1] in which the child died in about five hours, during which there was violent and copious vomiting, the analysis failed to give satisfactory evidence of the presence of the acid.[2] Moreover, the acid acts chemically upon the tissues, being itself thereby modified by combination and dilution, and the products so formed are rapidly absorbed and as rapidly eliminated. It goes without saying that none of the acid can be found in the cadavers of those who have died from its secondary effects. Indeed, failures to detect the free acid in the stomach contents after death constitute the rule rather than the exception, and Buchner's statement that "the chemical detection of a poisoning by nitric or sulfuric acid is as a rule impossible"[3] is quite true. Nevertheless an analysis, including a quantitative determination of sulfates, as well as of free sulfuric acid, should never be omitted if death have occurred within forty-eight hours after the acid was taken, and the history and post-mortem appearances (which are generally clearly defined) point to the probability of sulfuric acid corrosion, as both free acid and excess of sulfates have been repeatedly detected, not only in the vomit but in the stomach contents, after death. On the other hand, in the absence of such indications pointing to the presence of a corrosive, it is inadvisable to jeopardize the detection of true poisons by a search for the corrosives. In one instance, 0.031 gm. of free sulfuric acid was found in 40 gm. (about half a grain in one and a half ounces) of the stomach, œsophagus, and intestine of a child thirteen days old, that had died in twenty-four hours

[1] Vierteljschr. f. ger. Med., 1872, n. F., xvi., 54.

[2] The analyst in this case, however, seems to have detected traces of the acid, but to have been unwilling to recognize them under the mistaken idea that "free sulfuric acid can occur in the stomach contents of normal cadavers," *loc. cit.*, p. 60.

[3] Neues Repert. f. Pharm., 1866, xv., 241.

after its mother had poured the acid down its throat.[1] If, however, the victim survive more than a few hours (eight to ten), the probabilities of detection of the free acid are quite remote; although an excess of sulfates may still be found, as in another case, in Casper-Liman,[2] also of twenty-four hours' duration, in which no evidence of free acid was obtained from the stomach and œsophagus, which, however, yielded 0.0257 gm. (0.4125 "gran") of the acid in combination. As several sulfates (such as those of sodium, magnesium, quinin, morphin, etc.) are in common use as medicines, and as many articles of food and nearly all natural waters contain notable quantities of the sulfates of sodium, potassium, calcium, and magnesium, the detection of the acid in the stomach or vomit, in any form other than uncombined, can only be considered as confirmatory evidence, the weight of which will vary with the circumstances of the case. If, for instance, a large quantity of magnesium sulfate, accompanied by excess of magnesium oxid or hydroxid, be found in the vomit discharged from the stomach of a person to whom magnesium oxid had been administered, the inference that the magnesium sulfate was produced in the stomach contents by neutralization of the acid by the base would be strong in the absence of positive evidence of the administration medicinally of a mixture of magnesium sulfate and oxid, a combination which, although sometimes used, is exceptional.

To extract mineral acids from organic mixtures, stomach contents, vomit, etc., these are treated with absolute alcohol in such quantity that the material is in contact with alcohol of at least seventy-five per cent. The mixture is warmed to 50°–60° (122°–140° F.) for about ten hours, filtered, and the unsoluble residue washed with strong alcohol so long as the washings have an acid reaction. The filtrate and washings are received in a gauged flask, made up to a known volume, thoroughly mixed, and aliquot portions taken for qualitative and quantitative examination. In a portion the presence or absence of free mineral acid is determined; another part is neutralized exactly with pure caustic potash in alcoholic solution, evaporated to dryness, and the residue examined qualitatively to determine the nature of the acid. The material left undissolved by alcohol is to be ex-

[1] Casper-Liman: "Handb. d. ger. Med.," 8te Aufl., 468.

[2] *Loc. cit.*, p. 465.

tracted in like manner with water, and the aqueous extract examined as to the presence and quantity of soluble salts of such acid as may have been found. Finally, the material left undissolved by alcohol and water is treated with excess of pure disodic carbonate, dried, fused, and the residual mass extracted with water or dilute hydrochloric or nitric acid. In this solution the bases and acids present in insoluble combination are identified and determined quantitatively.[1]

The presence of a **free mineral acid** is indicated by the following reactions:

1. A few drops of an alcoholic solution of methyl violet (1 : 100) colors the liquid blue.

2. An aqueous solution of Congo red forms a blue color or a blue precipitate. With acetic acid the color is violet.

3. A few drops of the liquid added to three or four drops of an alcoholic solution of tropæolin 00 (1 : 1,000) and warmed, produce a violet color.

4. Add to solution of ferric acetate (Liq. ferri acetatis, U. S. P.), diluted with water until it has only a faint yellow color, a few drops of a solution of potassium thiocyanate. The solution remains yellow, but on addition of a trace of a free mineral acid is colored blood red.

5. Add to a similarly diluted solution of ferric acetate a little potassium iodid solution and some starch paste. The blue starch-iodin reaction appears on the addition of a trace of free mineral acid.

6. A test paper, made by dipping ash-free filter paper into a freshly prepared solution of 1 gm. phloroglucin and 2 gm. of vanillin in 30 gm. of alcohol, assumes a red color or a red border to the carbonized portion, when dipped into a liquid containing free mineral acid and heated in a small capsule.

Free sulfuric acid is distinguished from sulfates by the following tests:

1. Add a few crystals of granulated cane sugar, and evaporate to near dryness; the residue becomes blackened by free sulfuric acid as it approaches dryness.

2. Concentrate the liquid, add some strips of metallic copper, and heat. In the presence of free sulfuric acid the odor of sulfur dioxid (burning sulfur matches) is produced. If the con-

[1] See also pp. 242, 253.

centrated liquid and copper be heated together in a small flask, fitted with a cork through which passes a tube twice bent at right angles, whose free end dips into a small quantity of water in a test tube, the water will contain sulfurous acid, recognizable by its odor; or by addition of a crystal of iodic acid and agitation with chloroform, when the aqueous liquid becomes yellow and the chloroform violet.

3. A crystal of veratrin, moistened with concentrated sulfuric acid, is colored slowly yellow, and after a time dissolves, forming a crimson liquid. The color is not produced by the dilute acid.

4. By the quantitative method given below.

Test for free sulfuric acid or soluble sulfates. Solution of barium chlorid (or nitrate) produces in solutions of sulfuric acid or of sulfates, acidulated with hydrochloric or nitric acid, a white precipitate of barium sulfate. Nitric acid and barium nitrate are used in place of the corresponding chlorin compounds in the presence of silver, lead, or mercurous compounds, which precipitate with hydrochloric acid. The only other mineral compounds of barium insoluble in acid liquids are the selenate and silico-fluorid, both of which are compounds rarely met with and not likely to be present in mixtures examined by the toxicological chemist. Even this remote doubt may be removed by drying the precipitate of barium sulfate, mixing it with a mixture of sodium carbonate and potassium cyanid, and heating to redness on the lid of a platinum crucible, by which treatment the sulfate is reduced to sulfid. The presence of the latter is recognized by placing a fragment of the cooled melt on a piece of paper which has been previously moistened with dilute solution of lead acetate and dried, and adding a drop of dilute hydrochloric acid. If a sulfid have been formed, the fœtid odor of hydrogen sulfid is observed, and the paper is turned brown or black.

Other reactions common to free sulfuric acid and the sulfates, but much less delicate than the barium test, and by no means as characteristic, are: 1. Strontium nitrate forms a white precipitate, which is soluble to a slight extent in hydrochloric and nitric acids. 2. Lead acetate forms an amorphous white precipitate, which is somewhat soluble in solutions of the caustic alkalies and of ammoniacal salts.

The **quantity** of sulfuric acid, whether present as free sulfuric acid or as sulfate, is estimated, by precipitation as barium sulfate, with the usual analytical precautions, and weighing the barium salt after ignition. The weight of barium sulfate found, multiplied by 0.4205, gives the weight of sulfuric acid (H_2SO_4) in the quantity of liquid examined.

To estimate the quantity of *free sulfuric acid* in the presence of sulfates, a measured volume of the solution is evaporated to as near dryness as is possible on the water-bath, and the residue extracted with a mixture of equal parts of absolute alcohol and ether. The solution is mixed with water, and warmed on the water-bath to expel the ether and a portion of the alcohol. The cooled liquid is made up to a known volume with water, and in a known fraction the degree of acidity is determined by titration with one-tenth normal caustic potash solution, using litmus or methyl orange as an indicator. The remainder of the solution is evaporated to one-third of its bulk, diluted if necessary with water, and treated with nitric acid and barium nitrate solution. The precipitated barium sulfate is collected and weighed. The filtrate and washings, concentrated if necessary, are treated with nitric acid and ammonium molybdate solution, and the quantity of phosphoric acid in the yellow precipitate determined by the usual method [1] (see below). The results obtained by this process are not absolutely accurate, but they are more nearly so than with other processes, such as by neutralization with barium carbonate; by mixing with alcohol without evaporation; by extraction with ether alone; by extraction of the residue of evaporation with water, either after or without ignition; or by calculation from the total quantity of bases and acids.[2] The sources of error are the following: There may be more or less action between the dissolved sulfuric acid and the alcohol, by which the dibasic mineral acid is converted into the monobasic sulfovinic acid ($C_2H_5.HSO_4$), which only requires half the quantity of caustic potash for its neutralization. This action is the less the smaller the quantity of sulfuric acid present and the lower the temperature of evaporation; hence, if the extract be strongly acid, it should be

[1] Fresenius: "Quant. Anal.," 6te Aufl., i., 404.

[2] See Beilstein and Grosset: Ztschr. f. an. Chem., 1890, xxix., 73. Garnier and Schlagdenhauffen: Ann. d'hyg., etc., 1884, 3 S., xi., 227; and Fresenius: "Quant. Anal.," 6te Aufl., i., 399.

diluted with water correspondingly, and during the heating to expel ether the water in the bath should not be heated to boiling. Although the sulfates are as a rule insoluble in alcohol-ether, the ferric sulfate is somewhat soluble. If present, however, it colors the liquid yellow. Free lactic acid is soluble in alcohol-ether, as well as free sulfuric and phosphoric acids. If it be present the total acidity will exceed that accounted for by the sum of the amounts of sulfuric and phosphoric acids found.

If a notable quantity of combined sulfuric acid have been found, the bases present should be quantitatively determined, notably the sodium and magnesium.[1]

The significance of the presence of free phosphoric acid was first recognized by Garnier,[2] in the case referred to in the note above, in which the alcohol-ether extract from the alimentary canal was strongly acid from the presence of a fixed acid, yet contained no sulfuric acid. The acidity was ascribed to the presence of free phosphoric acid, liberated from the phosphates by the stronger sulfuric acid, which had itself combined with the bases. Consequently the existence of free phosphoric acid in viscera (unless it have been medicinally administered) presenting the lesions caused by the corrosives, is strong confirmatory evidence that sulfuric acid has been present. This view is supported by the fact that, as Garnier has shown, the other free acids likely to be present—hydrochloric, butyric, and lactic—are not competent to liberate phosphoric acid from the phosphates.

In the case referred to (Affaire de Lichtemberg, Assises de Saint Michel, 1883) Garnier and Schlagdenhauffen also noted the presence of minute quantities of arsenic in the cadaver. The quantity was such as would be accounted for by the ingestion of seven to eight grammes, or about a teaspoonful of the commercial acid, which, notoriously, is always contaminated with arsenic. The viscera should also be examined for lead and its quantity determined if found, as that metal is a frequent impurity of commercial oil of vitriol.

Examination of Suspected Materials.—This may be limited to the mere identification and the determination of the

[1] For methods see Fresenius: "Quant. Anal.," 6te Aufl., or Johnson's translation.

[2] Ann. d'hyg., etc., 1887, xvii., 148.

degree of concentration of a sample of commercial oil of vitriol, or it may extend to an examination of articles of food, drink, or medicine, not only for sulfuric acid, but for other substances as well.

The acid is readily identified by its giving off dense white fumes, and being volatilized with little or no residue when sufficiently heated; by its blackening sugar; by the development of heat on dilution with water, and by the barium chlorid test above given. Its concentration is determined by its specific gravity, by dilution and titration with standard alkaline solution, or gravimetrically by precipitation as barium sulfate.

As an instance of evidence upon collateral points obtainable from analyses of food articles, we may quote a case from Taylor:[1] "A girl was charged with attempting to administer oil of vitriol in coffee to her father. The prisoner usually made the coffee for breakfast, and would then have had an opportunity of adding it to the liquid. The acid might, however, have been mixed with the coffee in the cup after it had been poured out; and in this case other persons would have had the opportunity of poisoning the coffee. This question was solved by the aid of chemistry. I procured the coffee pot, and found that it was old and rusty. The poisoned coffee was tested, and it contained no trace of iron; but on warming a small quantity of the acid coffee in the pot, it was immediately and strongly impregnated with sulphate of iron. It was therefore clear that the acid had not been mixed with the coffee in the pot, and that it might have been afterward put into the cup without the knowledge of the prisoner." This case also serves to indicate the necessity of caution in drawing conclusions from the presence of poison in articles of food, etc., which *may* have been tampered with by persons other than the accused.

Examination of Stains.—The identification of sulfuric or other acid as the cause of stains upon garments, bed clothing, etc., may afford the most reliable evidence of the nature of the corrosive administered, and may occasionally aid in pointing out the person by whom it was administered.

The mineral acids dropped upon earth cause effervescence from evolution of carbon dioxid. At the same time the acid is neutralized by conversion in the corresponding salt, and an acid

[1] "Poisons," 3d Am. ed., 163.

reaction only remains if the quantity of acid has been greater than is necessary to decompose all of the carbonates present. Wood is almost immediately blackened by strong sulfuric acid, is colored yellow by nitric acid, and is hardly modified by hydrochloric acid. Iron is attacked by sulfuric and hydrochloric acids, with formation of sulfate or chlorid, which is dissolved readily by water, leaving a roughened appearance of the metal, as if it had been rusted and cleaned. Nitric acid attacks iron similarly if it be dilute, but not if concentrated. All of the acids attack the zinc coating of "galvanized iron." The tin coating of sheet tin is dissolved by hydrochloric acid, is not attacked by cold sulfuric acid, and is turned white by nitric acid. Copper is colored green by cold sulfuric or hydrochloric acid, and blue by nitric acid, which also evolves copious brown fumes. Silver is similarly acted upon by nitric acid, but cold hydrochloric or sulfuric acid does not affect it except after long contact. Gold is not acted upon by any single acid; but is corroded and dissolved by a mixture of nitric and hydrochloric (aqua regia).

On cotton, paper, or other white or light-colored vegetable or animal tissue, sulfuric acid produces a stain which is brown or black; the tissue is rendered friable, breaks down easily, and remains moist for a long time. Even by short contact the fabric is perforated if the acid be concentrated. The moistness of the stain or of the margins of the perforations is the principal difference in mere appearance between burns in paper or other fabric produced by sulfuric acid and those produced by heat, the latter being quite dry. On similar fabrics nitric acid produces a yellow stain, and at the time of contact brown fumes may be evolved. Hydrochloric acid does not corrode such tissues. Stephenson[1] relates an exceptional instance (Reg. *v.* Lipski, C.C.C., July, 1888), in which the cotton night-dress of a woman whose life was taken by pouring a mixture of sulfuric and nitric acids down the throat, as well as the deal floor on which the acids were spilled, were partially converted into nitro-cellulose, which partially exploded when ignited in a closed tube. The stains produced upon dark-colored fabrics by sulfuric or hydrochloric acid are usually red, sometimes green in color, and, if not too old, disappear permanently when moistened with am-

[1] Taylor: "Manual of Med. Jur.," 11th Am. ed., 1892, 373.

monium hydroxid solution. Those formed by nitric acid are dirty yellow or brownish, and, although they may disappear temporarily when moistened with ammonium hydroxid solution, they return when the spot dries. The nature of the acid may also be sometimes ascertained by chemical means by boiling the stained fabric for a short time with pure water, filtering the solution, and testing the filtrate not only for the free acid but for the corresponding salts also, by the methods already described. Sulfuric acid remains in the tissue for a long time, months or even years; but nitric and hydrochloric acids, if not decomposed or neutralized, soon disappear by evaporation. The detection of a small quantity of free sulfuric acid in the margins of a burnt perforation is not conclusive proof that the burn was caused by the acid. In a case already referred to, Maschka[1] showed that the margins of burns thus produced by incandescent coals may contain sulfuric acid, which is produced by the oxidation of the sulfur which exists in many kinds of coal.

Acid Sulfates—Alum, etc.

Sulfates which have a markedly acid reaction behave both as true poisons and as mild corrosives, or at least as intense local irritants. Such are alum (double sulfate of aluminium and ammonium, or potassium, $Al_2(SO_4)_3,(NH_4)_2SO_4 + 24$ Aq, or $Al_2(SO_4)_3,K_2SO_4 + 24$ Aq), the monometallic sulfates (sodium or potassium bisulfates, $NaHSO_4$ or $KHSO_4$), white vitriol (zinc sulfate, $ZnSO_4$), and blue vitriol (cupric sulfate, $CuSO_4$).

Alum.

The salt usually met with is the ammonium compound, which, by reason of its less cost, has practically displaced the "potash alum" formerly in use. It is largely used in dyeing and for numerous other uses in the arts, and is sold without restriction. It has been used by bakers to whiten bread made from inferior flour, a practice which has met with the almost unanimous condemnation of hygienists. The same cannot be said of its use in the preparation of baking powders, if it be combined with sodium bicarbonate in the proper proportion, for then, during the raising and baking of the bread, it is completely decomposed, and the product contains no alum, but aluminium hydroxid or phosphate. Alum is now also added to water in minute quantity in certain processes of purification as a preliminary to filtration. Here also the alum is decomposed and precipitated as aluminium hydroxid.

[1] Gutacht. Prag. med. Fak., 1853, i., 121.

Up to the present time six instances of fatal poisoning by alum have been reported. Taylor[1] states that a case of death from alum appeared in the registration returns for 1838-39. Van Hasselt[2] refers to an accidental case as having occurred at Hoogeveen, in Holland, in 1851, in consequence of the use of burnt alum as a popular remedy for gastric pain. Tardieu[3] relates the case of a girl who died at Bayonne in 1873 of gangrenous angina, as it was supposed, but in whose stomach a large quantity of alum was found. Hicquet[4] gives a circumstantial account of the accidental death in twenty-four hours of a man of fifty-seven years from the effects of 30 gm. (℥ i.) of alum. Fagerlund[5] notes the case of an infant killed by alum in Finland in 1886. A large but not accurately determined quantity of alum was found in the alimentary canal. Stevenson[6] refers to the death of a child of three years from the effects of a teaspoonful of alum given in syrup as an emetic. A non-fatal case is discussed by Orfila.[7]

The SYMPTOMS produced by alum are burning pain in the mouth, which looks as if tanned, and in the throat and stomach; persistent vomiting of bloody material;[8] accelerated pulse and respiration, disturbances of coördination; clonic spasms of the muscles; diminution of temperature; and hyperæmia of the intestines and kidneys.

The POST-MORTEM APPEARANCES are delayed putrefaction, corrosion and œdema of the tongue, mouth, and œsophagus. The stomach contracted, grayish or velvety red, its veins filled with almost coagulated blood; the mucous membrane corrugated, loosened, or hardened. The intestines are inflamed. There are evidences of peritonitis, including effusion of red serum into the peritoneal sac, and congestion of the vessels of the omentum. The liver is the seat of fatty degeneration, and the kidneys are highly injected, with parenchymatous degeneration.

DETECTION AND DETERMINATION.—In the method of Fresenius and von Babo any aluminium present will be contained in the solution designated as IX. in the description of the process given in the first section (see page 160). The aluminium is separated as hydroxid by addition first of hydrochloric acid to acid reaction, and then of ammonium hydroxid to alkaline reaction, and boiling until the liquid is neutral or only faintly alkaline. This is best done in a platinum vessel, or, failing that, less well in one of porcelain. Glass vessels should not be used. The precipitated aluminium hydroxid is washed with boiling water, first two or three times by decantation and after-

[1] "Poisons," 3d Am. ed., p. 266.
[2] "Handb. d. Giftlehre," 1862, ii., 207.
[3] "Empoisonnements," 2ème ed., 1875, 219.
[4] Bull. Soc. méd.-lég., Paris, 1871, ii., 428.
[5] Vrtljschr. f. ger. Med., 1894, 3 F., viii., Supplehft., 74.
[6] Taylor: "Man. Med. Jur.," 11th Am. ed., 98.
[7] "Tr. d. Tox.," 5ème ed., i., 370.
[8] In Stevenson's case the child did not vomit, but died shortly after the administration.

ward in a filter or Gooch crucible with the aid of a filter pump, until the washings no longer precipitate with silver nitrate. For quantitative estimation the aluminium hydroxid is ignited in a platinum crucible and weighed as aluminium oxid (Al_2O_3). Of this one part represents 17.0777 parts of crystallized ammonia alum [$Al_2(SO_4)_3$, $(NH_4)_2SO_3 + 24Aq$], or 8.6893 of the anhydrous alum [$Al_2(SO_4)_3$, $(NH_4)_2SO_4$]; or 6.1262 of aluminium sulfate [$Al_2(SO_4)_3$]. After weighing, the oxid is heated in the platinum crucible with monopotassic sulfate ($KHSO_4$) to quiet fusion, and the cooled mass treated with water, in which it should dissolve completely. If any insoluble residue remain it is silicic anhydrid, and should be dried, ignited, and weighed, and its weight subtracted from that of the aluminium oxid found. The solution will serve for further identification reactions.

Any shorter method of separation of dissolved alum from the viscera is not to be recommended. If the question be whether a given white solid is or is not alum, it should be dissolved in water, evaporated to crystallization, and the proportions of aluminium and of sulfuric acid determined in a portion of the crystals. When aluminium has been found in the viscera a quantitative determination of the sulfuric acid is desirable, and should be made if any material remain available.

Aluminium is identified: 1. By its behavior as above described in the process of separation.

2. Potassium and sodium hydroxids produce voluminous, white, flocculent precipitates of the hydroxid, easily soluble in excess of the precipitant.

3. Ammonium hydroxid and hydrosulfid also cause similar precipitates, only soluble in a great excess of the hydroxid.

4. Alkaline carbonates produce white, flocculent precipitates of a basic carbonate, sparingly soluble in excess of the precipitant.

5. Sodium phosphate produces a voluminous white precipitate of the phosphate, which is easily soluble in soda or potash solution, but almost insoluble in ammonium hydroxid, particularly in presence of ammonium chlorid. The precipitate is also soluble in nitric or hydrochloric acid, but not in acetic acid. The precipitation is hindered by the presence of citric acid.

6. If a solid aluminium compound be strongly heated on charcoal before the blowpipe, moistened with cobalt nitrate solution, and again heated, a deep sky-blue, unfused mass remains, the color becoming more pronounced as the mass cools. The reaction is best performed with the oxid obtained by heating the precipitated hydroxid. A similar blue mass is also formed in the absence of aluminium by the phosphates of the alkaline earths.

Cupric Sulfate.—See under **Copper.**

Zinc Sulfate.—See under **Zinc.**

Acid Potassium Sulfate.

MONOPOTASSIC SULFATE; *Potassium Bisulfate*, $KHSO_4$.—This is a salt of strongly acid reaction, which is used in chemical laboratories for the decomposition of refractory oxids, such as that of aluminium, the acid sulfate at elevated temperatures being decomposed into the dimetallic salt and free sulfuric acid—($2KHSO_4 = K_2SO_4 + H_2SO_4$). Although it would probably act even more energetically than alum if swallowed, we know of no instance of its having been taken by the human subject. It may be generated, however, under certain conditions, and give rise to a form of chronic sulfuric poisoning. In the "plastering" of wine, the added plaster-of-Paris produces free tartaric acid and neutral potassium tartrate from the cream of tartar naturally present, and subsequently, particularly in the presence of alcohol, cream of tartar and acid potassium sulfate are formed. The reactions are $2(KH.C_4O_6H_4) + CaSO_4 = CaC_4O_6H_4 + K_2SO_4 + C_4H_6O_6$, and $K_2SO_4 + C_4H_6O_6 = KH.C_4O_6H_4 + KHSO_4$. The continued use of plastered wines produces disorders of digestion, and the practice of plastering is ranked as an adulteration.[1] According to Kobert,[2] acid potassium sulfate is also formed in the economy during starvation and with extensively albuminous diet.

The NEUTRAL POTASSIUM SULFATE; *Dipotassic sulfate*, K_2SO_4 when taken in large dose and in concentrated form behaves as an intense local irritant and also as a true poison, as do all of the potassium salts. It has caused death in a few instances, and in two English cases men were tried for murder for having caused the death of their wives by this salt given as an abortive with the consent of the deceased.[3]

HYDROCHLORIC ACID.

Hydrochloric acid is a colorless gas, intensely irritating when inspired even in a highly diluted form, very soluble in water, one volume of which dissolves four hundred and eighty volumes at 0° (32° F.), acid in taste and in reaction, heavier than air, specific gravity 1.259. Its molecular weight is 36.5, and its formula is HCl. It exists in volcanic gases, and is produced whenever a chlorid is decomposed by a stronger acid. The reaction utilized in its manufacture is the decomposition of sodium chlorid by sulfuric acid, which takes place according to

[1] See Gaultier: "Sophistication des Vins," Paris, 1884, 222. Viard: "Les Vins," etc., Paris, 1884, 299, 346. Bussy et Buignet: J. d. Pharm. et de Chim., 1865, 4 s., ii., 1.

[2] "Intoxikationen," p. 211.

[3] Reg. *v.* Haynes, 1843; Ph. J. and Tr., 1843–44, iii., 243, 256. Reg. *v.* Gaylor, 1856; *ibid.*, 1856–57, xvi., 295. See also Bayard: Ann. d'hyg., etc., 1842, xxvii., 397; Bonnassies: Gaz. d. hôp., 1843, Du Merat et Delens: Dict. univ. d. mat. méd. 485.

the equation: $NaCl + H_2SO_4 = NaHSO_4 + HCl$ at low temperatures; or $2NaCl + H_2SO_4 = Na_2SO_4 + 2HCl$ under the influence of heat.

The material so extensively used in chemical industries, and the one which is of toxicological interest, known as *hydrochloric* or *muriatic acid*, *spirit of salt*, is a solution of this gas in water of varying degrees of purity and of strength.

The *commercial* acid is a yellow liquid, containing about thirty-two per cent. HCl (specific gravity 1.16), and is contaminated with ferric salts, to which its color is due, sodium chlorid, arsenic trichlorid, free chlorin, sulfur dioxid, and occasionally a minute quantity of thallous chlorid and of selenium.

Acidum hydrochloricum, *U. S. P.*, *Br. P.*, is a colorless liquid, freed from all but traces of impurities, specific gravity 1.16 (= 31.9 per cent. HCl). The *acidum hydrochloricum dilutum* is the above, diluted with water to specific gravity 1.049 (= ten per cent. HCl) U. S. P., or specific gravity 1.052 (= 10.5 per cent. HCl) Br. P.

C. P. (*chemically pure*) *hydrochloric acid* is usually the same as the pharmacopœial acid, and is far from pure. "*Absolutely*" or "*strictly*" C. P. acid more nearly approaches but does not attain the degree of purity requisite for toxicological uses.[1]

The strength of the acid, in the absence of dissolved solids, is indicated by its specific gravity. There is some divergence between the older tables of Ure and of Davy. The following, by Kolb,[2] is probably the most accurate:

Specific gravity at 15°.[3]	HCl in 100 parts.	Specific gravity at 15°.	HCl in 100 parts.	Specific gravity at 15°.	HCl in 100 parts.	Specific gravity at 15°.	HCl in 100 parts.	Specific gravity at 15°.	HCl in 100 parts.	Specific gravity at 15°.	HCl in 100 parts.
1.007	1.5	1.052	10.4	1.091	18.1	1.134	26.6	1.166	33.0	1.190	37.9
1.014	2.9	1.060	12.0	1.100	19.9	1.143	28.4	1.171	33.9	1.195	39.0
1.022	4.5	1.067	13.4	1.108	21.5	1.152	30.2	1.175	34.7	1.199	39.8
1.029	5.8	1.075	15.0	1.116	23.1	1.157	31.2	1.180	35.7	1.205	41.2
1.036	7.3	1.083	16.5	1.125	24.8	1.161	32.0	1.185	36.8	1.210	42.4
1.044	8.9	....	....	....	....	....	....	....	....	1.212	42.9

Hydrochloric acid reacts readily with iron, zinc, and most

[1] See note, p. 155.

[2] Bull. Soc. Chim., Paris, 1872, xvii., 281.

[3] =59° F.

of the more distinctly electro-positive metals, except copper, with formation of a soluble chlorid and evolution of hydrogen. A few of the chlorids, such as stannic chlorid—$SnCl_4$—and antimony pentachlorid—$SbCl_5$—are liquid; the remainder are solid, crystalline, and more or less volatile. The chlorids of the non-metals are decomposed by water. Those of the metals are soluble in water, except argentic and mercurous chlorids—AgCl and Hg_2Cl_2—which are insoluble, cuprous chlorid—Cu_2Cl_2—which is very sparingly soluble, and lead chlorid—$PbCl_2$—which is only sparingly soluble in cold and more readily in hot water. Hydrochloric acid also dissolves most of the basic oxids and hydroxids with formation of chlorids, and decomposes the carbonates with evolution of carbon dioxid.

Hydrochloric acid is first referred to in the fifteenth century by Basil Valentine, who obtained it in solution by distilling vitriol and common salt. The gas was first collected and studied by Priestley in 1772. It was not, however, until the early years of the nineteenth century that the development of the Leblanc soda industry and the recovery of the enormous volumes of hydrochloric acid which it produced, as an incidental process, furnished the acid on a commercial scale, and caused its extensive introduction into the arts. It is at this period also that its toxicological history begins. Although Sproegel[1] had, in the middle of the eighteenth century, experimented to determine the effects produced by hydrochloric acid when injected into the veins of animals and similar experiments were made by Courton, Viborg, and Orfila early in the nineteenth,[2] the first death of a human being following the swallowing of the acid was in Serres's case in 1805, in which, however, it may be questioned whether the acid, which was given the patient by the hospital interne by mistake, was the cause of death, or whether he succumbed to the injuries he had received. Certainly the dose, 45 gm. (℥ iss.), was sufficient to cause death.[3] Death from hydrochloric acid is of exceptional occurrence, and but a very small proportion of the cases have been homicidal. We find mention of but five alleged homicides by this corrosive, in two of which the victims were adults: Affaire Quenardel,

[1] "Experimenta circa varia venena," etc., Diss., Göttingen, 1753, pp. 84–86.

[2] Wibmer: "Wirkung der Arzneimittel," etc., iii., 97.

[3] Orfila: "Tox.," 5ème ed., i., 195.

France, 1839;[1] Affaire Denisty, Belgium, 1846;[2] Affaire Poindron, France, 1847;[3] Reg. *v.* Somers, England, 1866;[4] and a case in Germany in 1873.[5] But deaths from hydrochloric acid are of more frequent occurrence at the present time than formerly. Of 69 cases 3 occurred before 1840, 3 in 1840–49, 7 in 1850–59, 9 in 1860–69, 15 in 1870–79, 23 in 1880–89, and 10 in the five years 1890–94.

In suicidal and accidental cases the acid has been either taken in its own form or in that of "soldering-liquid," used by tinsmiths and metal workers, which is a solution of zinc chlorid containing excess of acid.

Symptoms.

The deleterious effects produced by the inhalation of gaseous hydrochloric acid have at present little forensic interest, although they may become the subject of discussion in connection with the public health, should the gas be discharged into the air as it was in the early days of the soda industry.

The liquid acid, when swallowed, produces symptoms very similar to those caused by sulfuric acid. The main points of difference are the following: The action upon the skin is much less intense. The hands may be washed in a moderately dilute acid with perfect impunity; and Lesser[6] has demonstrated that even the most concentrated acid leaves no permanent marks when applied to the skin. White vapors are sometimes exhaled from the mouth and nose.[7] The mucous membrane of the mouth, lips, and tongue are never blackened, but are usually whitish or ash-gray. The inhalation of the gaseous acid, which is given off from the solution, produces irritation of the respiratory passages, whether the liquid penetrates them or not. Respiration is accelerated, there is pain in the throat and behind the sternum, cyanosis of the face, and almost complete extinc-

[1] Orfila: *Loc. cit.*, i., 216.

[2] Orfila: *Loc. cit.*, i., 221. Van den Broeck: Gaz. méd. belge, 1847, v., 94, 99, 102, 105, 169, 175. Pierard: *Ibid.*, p. 113. Orfila: Ann. d'hyg., etc., 1848, xl., 137. Flandin: "Poisons," ii., 491.

[3] Orfila: "Tox.," 5ème ed., i., 224. Ann. d'hyg., etc., 1847, 178. Flandin: "Poisons," ii., 482.

[4] Taylor: "Poisons," 3d Am. ed., 220.

[5] Zimmermann: Cor. - Bl. d. aerztl. Ver. d. rhein. Prov., 1873, No. xii., 21.

[6] Arch. f. path. Anat., etc., 1881, lxxxiii., 215.

[7] Viscarro: Siglo med., Madrid, 1878, xxv., 667.

tion of the voice, if the acid have been taken in concentrated solution. Indeed, the action upon the respiratory organs may be so intense as to mask almost completely the symptoms of corrosion. This was well marked in a man of fifty-eight years who swallowed about 200 gm. (℥ vij.) of the commercial acid. He immediately experienced severe epigastric pain and a sense of suffocation. A physician who was called immediately provoked several acts of vomiting. During the night and the following day the oppression and suffocation increased. When admitted to the hospital on the second day there was no vomiting and no violent epigastric pain, even on pressure; but the oppression and dyspnœa were intense. The respiratory troubles were so predominant and the lesions of the stomach so silent, that but for positive information, the poisoning would have been doubted, and the case would have been considered as one of acute broncho-pneumonia, developed in a somewhat delirious alcoholic. A few hours after admission, after having eaten and drank without difficulty, he died almost suddenly, in full consciousness, without having vomited once.[1]

The kidneys are but rarely affected, probably only when they are already diseased. In a careful study of the urine of a man of twenty-eight years who died in three months from the effects of 100 c.c. (℥ iiiss.) of the commercial acid, Bourget[2] found that while the acidity was greatly increased during the first day or two, the urine contained no free hydrochloric acid, but a great excess of phosphates; and that the quantity of chlorids eliminated, far from being increased, was notably less than the normal (about 10 to 15 gm. in twenty-four hours). During the first few days albumin was present in small and progressively diminishing amount. The urine of the first six days gave the following quantitative results:

	1st.	2d	3d.	4th.	5th.	6th.
Quantity in 24 hours	1,100 c.c.	1,000	750	400	400	450
Sodium chlorid.....	6.6	2.75	0.60	0.08	0.08	0.18
Phosphates.....	9.75	5.	3.24	1.52	1.36	1.70
Reaction[3].....	6.	1.8	1.4	1.	normal.	
Albumin .....small quant.		dim.	slight.	none.	none.	

The elimination of the acid did not therefore take place by

[1] Letulle and Vaquez: Arch. d. phys. norm. et path., Paris, 1889, 5 s., i., 101.

[2] Rev. méd. de la Suisse Rom., 1889, ix., 210.

[3] Acidity in c.c. normal KHO.

the kidneys. But the patient on the second day and for several days thereafter, vomited and regurgitated large quantities of mucous liquid, which was found on analysis to contain an average of about ten per thousand of sodium chlorid. Although in this case and in another in which the dose taken was still larger (200 gm.) the acid was without action on the kidneys, other cases have occurred in which the urine contained albumin, blood corpuscles, and casts,[1] and, according to Demieville,[2] even medicinal doses of dilute hydrochloric acid may produce acute nephritis and hæmaturia in those having latent kidney disease.

In rapidly fatal cases, consequent upon the taking of large doses, the action of the acid upon the central nervous system is frequently well marked. In such cases death occurs either in violent convulsions[3] or in collapse.[4] Instances of cicatricial pyloric stenosis and of death from inanition due to hydrochloric acid are by no means exceptional. In eleven of sixty-nine cases (15.9 per cent.) death occurred in periods greater than one month after the ingestion of the acid, from its secondary effects. In Gehle's case, above referred to, the patient, a man of forty-four years, died in four months and twenty-two days, having suffered greatly during the last two months from enormous dilatation of the stomach, consequent upon pyloric stenosis. In another instance a woman of fifty-five years, who died in about five weeks, became so emaciated that the abdominal aorta could be felt through the abdominal walls.[5] Nor is death from perforation of the alimentary canal and peritonitis of as rare occurrence as has been supposed.[6] A girl took 177 c.c. (℥ vi.) of the acid in the night. The next morning at eight o'clock vomiting had ceased and she said she "felt fine, except for the pain," which was intense. She died suddenly at 3 P.M. The stomach was found perforated in three places.[7] A woman of thirty years, about twenty-four hours after taking a glassful of the acid, was in great pain, the abdomen distended, the vomit

[1] Gehle: Berl. kl. Wchnschr., 1884, xxi., 337.

[2] Rev. méd. de la Suisse Rom., 1889. ix., 214.

[3] Friedreich's Bl. f. ger. Med., 1858, ix., 6 Heft, 70.

[4] Bloomfield: Med. Times and Gaz., 1883, i., 471.

[5] Ber. . . . Rudolph Stiftung, Wien, 1865, 153. See also under "Post mortem Appearances."

[6] von Jaksch in Nothnagel's "Spec. Path. u. Ther.," 1894, i., 22.

[7] Thomas: Australas. Med. Jour., 1891, n. s., xiii., 542.

greenish, not bloody, pulse 130, filiform. She died soon after of acute peritonitis, the evidences of which and a perforation of the duodenum were found at the autopsy.[1] A man of thirty-five years developed the symptoms of peritonitis on the fourth day, became greatly prostrated, and died on the seventh.[2] A man of fifty-four years died in collapse seventeen hours after swallowing two tablespoonfuls of the acid. Not only was the stomach found to be perforated, but the œsophagus and the duodenum as well.[3] Perforations of the stomach or intestine were found to exist in twelve out of forty-five autopsies after death from hydrochloric acid (or in 26.7 per cent. of the cases observed).[4]

Diagnosis.

The diagnosis between corrosion by hydrochloric acid and that by sulfuric or nitric acid is not always easy. Fortunately for purposes of treatment it is not necessary. Occasionally the white fumes and the odor given off with the expired air or from the vomit may serve to distinguish hydrochloric acid. Positive reliance is not to be placed upon the existence of yellow stains upon the mucous membranes as indicating nitric acid, as stains of that color are sometimes produced by hydrochloric acid and even by sulfuric acid. The detection of the mere presence of the acid in the vomit affords no indication as, except under unusual circumstances, it is a constituent of the gastric juice. Probably the only means of positive diagnosis, except when a residue of the liquid taken is available and may be identified, is by a quantitative determination resulting in the finding of an amount of hydrochloric acid distinctly greater than that normally present in the stomach contents; combined with the existence of the usual symptoms of acid corrosion.[5]

Prognosis.

According to von Jaksch[6] and others the prognosis is more favorable with hydrochloric acid than with the other mineral acids. This view is not borne out by the statistics of the 69

[1] Le Gendre: Progrès méd., 1883, xi., 1.057.

[2] von Wundscheim: Prag. med. Wchnschr., 1891, xvi., 605.

[3] Dyson: Lancet, 1884, i., 65.

[4] See also "Post-mortem Appearances."

[5] See below, under "Analytical."

[6] Nothnagel: "Specielle Path.," etc., 1894, i., 22.

cases which we have collected. In these death occurred in 82.2 per cent. Of the 44 cases in which the duration is stated, it was less than three hours in 2 (4.5 per cent.); during the first twenty-four hours in 22 (50 per cent.); during the first week in 29 (65.9) per cent.; during the first month in 33 (75 per cent.) and in periods of from one month to four months and twenty-two days in 11 (25 per cent.).

Post-Mortem Appearances.

Primary.—The external burns, stains, and scars which are so generally observed on the lips and face when sulfuric or nitric acid has been taken, are almost invariably absent. Indeed Lesser[1] considers the absence of such marks of corrosion on the skin as a characteristic mark of distinction. The case of a child of two years which died in a few hours from the effects of sixty-two per cent. hydrochloric acid is, however, reported,[2] in which the skin of both cheeks, as well as of the chin, was in many places colored light yellow, hardened, and reddened blue litmus paper applied to it. Sections through the skin showed a uniform yellow coloration of its tissue. In the same place another instance is related in which a man of twenty-eight years drank a quantity of hydrochloric acid and then hanged himself. The lips at their posterior half were whitish, cloudy, and partly reddened from loss of epithelium. In a child which died in nine hours from the effects of hydrochloric acid, the lips were brownish-red externally, gray internally, and the gums and tongue also gray.[3]

The mouth, pharynx, and œsophagus are usually not the seat of the severe lesions observed after death from sulfuric or nitric acid. Indeed, even when a large dose had been taken they have been found to be absolutely intact, save for a slight reddening of the mucous membrane of the œsophagus near the cardia.[4] More frequently they are whitish or slate-gray in color. That this is not always the case is evidenced by the following observations: In a child which died in about twenty-four hours the tongue was found corroded, the œsophagus con-

[1] Arch. f. path. Anat., etc., 1881, lxxxiii., 215.

[2] Casper-Liman: "Handb. d. ger. Med.," 8te Aufl., ii., 547.

[3] Zimmermann: Cor.-Bl. d. aerztl. Ver. d. rhein. Prov., 1873, No. xii., 21.

[4] Letulle and Vaquez: Arch. d. phys. norm. et path., Par., 1889, 5 s., i., 102.

gested and corroded; the parts about the upper opening of the larynx were in part hyperæmic, in part corroded, the latter of the color of fat.[1] In a man who died in seven hours the inner surface of both lips were gray-black; the upper part of the epiglottis black; the walls of the œsophagus swollen and gray-black in color.[2] In a young woman who died in a few hours, the mucous membrane was black and looked as if burnt.[3] In a woman of thirty years, who died in twenty-four hours, the tongue was greatly congested, the isthmus of the fauces blackish, as well as the upper surface of the epiglottis. The œsophagus presented an annular ecchymosis 2 cm. wide, and the remainder of the mucous membrane was of a dark yellow-brown. At about 5 cm. from the cardia the œsophageal mucous membrane was black; the coloration, which resembled a coating of pitch, extending over portions of the stomach.[4] In a man who died in twenty-six hours, the buccopharyngeal mucous membrane was dead white and the tongue and fauces were moderately swollen. In front of the lingual V the tongue was whitish, behind distinctly arborescent, with prominent papillæ; on both sides, between the amygdalæ and epiglottis, the exposed submucous tissue was bright red. The œsophagus was brown, darker above.[5] In a man who died in four and a half hours the mucous membrane of the œsophagus was blackish gray, and the submucous vessels thrombosed.[6]

The œsophagus has been found dilated in two instances,[7] and once perforated. In this case the appearances closely resembled those produced by sulfuric acid. The mucous membrane of the œsophagus was congested in the upper half. In the lower half it was black, thickened, and contracted; the veins were filled with black blood. Just above its passage through the diaphragm it was perforated in the posterior wall, and the tissue around the opening was black, pulpy, and infiltrated with grumous matter.[8]

The stomach is usually of normal size or contracted, al-

[1] Hunt: Med. Times and Gaz., 1883, i., 609.
[2] Fagerlund: Vrtljschr. f. ger. Med., 1894, 3 F., viii., Supplhft., 56.
[3] *Ibid.*
[4] Le Gendre: Progrès méd., 1883, xi., 1057.
[5] Fortunet: Lyon méd., 1885, xli., 587.
[6] Legg: St. Barth. Hosp. Rep., 1874, x., 230.
[7] Hadden: Tr. Path. Soc., London, 1889–90, xli., 84. Gehle: Berlin. kl. Wchnschr., 1884, xxi., 337.
[8] Dyson: Lancet, 1884, i., 65.

though in two instances of death from primary action it was found distended.[1] Its mucous membrane is for the greater part corrugated, hardened, and of a brownish or slate-gray color.[2] Although in most cases the gastric mucous membrane is much lighter in color than when corroded by sulfuric acid, several cases are reported in which it was found blackened in whole or in part.[3] Among the small number of early cases we find mention of perforation of the stomach as having been met with in one case only.[4] Since 1872 it has, however, been observed in seven autopsies;[5] while in two cases the gastric wall had become thinned to such a degree that perforation was imminent.[6]

The intestines have been found perforated in two instances, in both cases near the pylorus.[7] When there has been perforation of the alimentary canal, more or less extensive evidences of peritonitis are observed. In two instances the escaped acid liquid had also corroded the liver.[8] In one of Fagerlund's cases,[9] although there was no perforation, there were subserous ecchymoses in the stomach, the peritoneal cavity contained one and a half litres of reddish, serous liquid, and the visceral peritoneum was dull.

Even in rapidly fatal cases the liver has been found fatty.[10]

The kidneys are usually normal, or, if abnormal, more probably so from disease than from the action of the acid.[11]

Marked changes are met with in the respiratory organs. The larynx and trachea are reddened and congested, occasionally superficially ulcerated in places, and covered with a grayish-white friable false membrane. The bronchi also are inflamed, and evidences of broncho-pneumonia are found in the lungs.[12]

[1] Johnson: Brit. Med. Jour., 1871, i., 221. Legg: *Loc. cit.*

[2] For colored plate of stomach in a case of primary hydrochloric acid intoxication see Letulle and Vaquez, *loc. cit.*

[3] Hunt: *Loc. cit.* Legg: *Loc. cit.* Fagerlund: *Loc. cit.* (2 cases). Le Gendre: *Loc. cit.* Thomas: Austral. Med. Jour., 1891, n. s., xiii., 542. Beyerlein: Friedreich's Bl. f. ger. Med., 1890, xli., 31.

[4] Aff. Poindron, 1847, Ann. d'hyg., etc., 1847, xi., 178.

[5] Dyson, *Loc. cit.* Legg: *Loc. cit.* Fagerlund: *Loc. cit.* Thomas: *Loc. cit.* Nager: Arch. d. Heilk., 1872, iii., 213. Casper-Liman: *Loc. cit.*, case 232. Beyerlein: *Loc. cit.*

[6] Fortunet: *Loc. cit.* Casper-Liman: *Loc. cit.*, case 223.

[7] Dyson: *Loc. cit.* Le Gendre: *Loc. cit.*

[8] Dyson: *Loc. cit.* Schad: Aerztl. Int. Bl., 1885, xxxii., 406.

[9] *Loc. cit.*, p. 58.

[10] Beyerlein: *Loc. cit.* Legg: *Loc. cit.*

[11] Le Gendre: *Loc. cit.* Bourget: Rev. méd. de la Suisse Rom., 1889, ix., 210. Fortunet: *Loc. cit.* Schad: *Loc. cit.*

[12] Nager: *Loc. cit.* Letulle and Vaquez: *Loc. cit.*

In other cases, however, the respiratory organs are found entirely normal.

Secondary. — The post-mortem appearances after death from the secondary effects of hydrochloric acid do not differ from those observed in similarly fatal cases caused by other corrosives.

Analytical.[1]

The chemical detection of abnormal free hydrochloric acid in the stomach is even more difficult than that of sulfuric or nitric acid, and absolutely requires a determination of quantity. Not only are the same difficulties met with as have been referred to in considering sulfuric acid, but two sources of error peculiar to this acid are to be avoided. The normal gastric secretion contains free hydrochloric acid in the average proportion of about 1.7 in 1,000 parts in the human gastric juice. The extremes found by Richet in eighty determinations were 0.5 and 3.2 per cent.[2] Moreover, the stomach contents always contain chlorids in widely varying amount, and these may be decomposed at elevated temperature even by lactic acid, which exists in the stomach during the digestion of carbohydrates, with liberation of free hydrochloric acid. The mere detection of free hydrochloric acid in the stomach contents or vomit is, therefore, entirely without significance, and it is only when it is found present in large amount that the results of a chemical examination of these materials are of value when taken in conjunction with the clinical history and the post-mortem appearances. It is only when death occurs quite early, or when the matters first vomited (before the administration of an antidote) are available, that any information is to be expected from a chemical analysis.[3] We can find no record of any such quantitative determination having been made in a case of hydrochloric acid intoxication, although in the description of one of Fagerlund's cases it is said that the highly acid stomach contents gave distinct reactions for hydrochloric acid, and dissolved a bar of zinc with evolution of gas.[4]

[1] See also p. 220.

[2] Compt. rend. Ac. Sc., Paris, 1877, lxxxiv., 1514; lxxxv., 155.

[3] In the Aff. Denisty an analysis served rather to befog than to elucidate the case by reason of the untenable ground taken by the analyst. See Van den Broeck: Gaz. méd. Belge, 1847, v., 94, 99, 102, 105, 169, 175.

[4] *Loc. cit.*, p. 57.

Separation from Stomach Contents, etc. — Several methods for the separation and determination of free hydrochloric acid in organic mixtures have been suggested:

1. By SIMPLE DISTILLATION of the material (after dilution with water and division of solid particles if necessary) to dryness, and collection of the distillate, in which the acid is identified and determined by the silver method (see below).

Although this method may yield fairly good results if the quantity of acid present be very great, it is open to several objections, and requires the exercise of certain precautions. During the distillation small quantities of sodium chlorid may be mechanically carried over with the distillate. This may be avoided by passing the vapor through a wide glass tube, loosely packed with glass wool and maintained at a temperature of about 110° (230° F.). The materials may contain ammonium chlorid (particularly if they have undergone putrefaction), which also passes over with vapor of water and is not completely arrested by the glass wool. The most serious objection is that organic substances strongly retain hydrochloric acid, which, even if present in notable quantities, may be entirely absent in the distillate if the heat be raised only sufficiently to cause evaporation to dryness. The remainder of the acid is only driven off by heating the organic matter to carbonization, when empyreumatic products are given off which interfere with the subsequent determination.

It must not be forgotten that in this, as well as in all distillation methods, if the materials contain a free fixed acid (sulfuric, phosphoric, lactic) it will decompose the chlorids present and yield free hydrochloric acid. Consequently in all such methods the presence or absence of these acids must be ascertained, and if present their quantity determined in a separate portion of the material.

2. VITALI'S METHOD,[1] which is one of the best, is a combination of extraction by alcohol and by water and distillation The materials, suitably comminuted if necessary, are macerated in eight times their weight of absolute alcohol for twenty-four hours, after which the alcohol (*A*) is filtered off and the residue (*B*) washed with alcohol until a few drops of the washings no longer become cloudy with silver nitrate.

[1] L'Orosi, 1886, ix., 361; "Manuale di Chim. Toss.," 1893, p. 159.

A, the alcohol extract, is divided, by distillation until the amount of alcohol added has passed over, into an alcoholic distillate, *Aa*, and an aqueous residue, *Ab*. *Aa* is fractionally distilled at very low temperature, using well-cooled condenser and receivers. Two fractions are collected, the receiver being changed when the distillate begins to have an acid reaction.

The first fraction is tested for ethyl chlorid (produced by the action of the acid on the alcohol) as follows: It is placed in a small flask fitted with a cork carrying two tubes, one of which, communicating with the air, dips to near the bottom of the liquid; the other, terminating just below the cork and bent at right angles, communicating with a calcium chlorid tube, which in turn communicates with a wider tube 30 cm. long, filled with purified asbestos and supported in a furnace. The other end of the asbestos tube is fitted to a Liebig's bulb, filled with silver nitrate solution, whose other opening is attached to an aspirator. The asbestos tube is first heated to redness when a slow stream of air is drawn through the apparatus, while the flask is gently heated on a water-bath. The volatilized ethyl chlorid is decomposed in the hot tube, and the hydrochloric acid regenerated is precipitated as silver chlorid (see below).

In the second fraction hydrochloric acid is precipitated by silver nitrate (see below).

Ab, the aqueous residue of the first distillation, is further distilled to dryness, the distillation being continued until that which passes over is no longer acid. A known fraction of the distillate is removed and used for the application of tests 3, 4, and 6 (see p. 223) for mineral acids, and of tests (see below) for the identification of hydrochloric acid. The remainder is precipitated with silver nitrate and the silver chlorid collected.

Finally the temperature of the distilling vessel is gradually raised to redness. Any ammonium chlorid present partly distils and partly sublimes. The distillate and sublimate are united, treated with acetic acid, evaporated to dryness, and the residue washed with absolute alcohol and dissolved in water. In this solution hydrochloric acid is tested for by silver nitrate, and ammonium by platinic chlorid.

B. The material insoluble in alcohol is macerated in water for twenty-four hours. The solution *Ba* is filtered from the undissolved portion (*C*), which is washed with water until the

washings are no longer acid. The filtrate and washings are evaporated on the water-bath to a syrup, which is treated with eight times its weight of absolute alcohol. The precipitate which forms contains any hydrochloric acid which was present in the form of an acid albumin soluble in water, but insoluble in alcohol. It is treated with disodic carbonate (free from chlorid), evaporated, fused, and the fused mass dissolved in water, and precipitated with silver nitrate after addition of nitric acid to acid reaction. The undissolved portion *C* may still contain acid albumin insoluble in both water and alcohol. This is digested with dilute disodic carbonate solution, filtered, washed, and the united liquids evaporated to dryness, and the residue fused and treated as above described.

The quantitative determination of free hydrochloric acid is made with the acid second fraction above referred to. To an aliquot part of this nitric acid is added, then silver nitrate solution in slight excess. The precipitate formed is collected on a filter after it has been rendered flocculent by agitation and slight warming, dissolved off with ammonium hydroxid solution, reprecipitated with nitric acid in excess, collected upon an ash-free filter, washed, and dried. The silver chlorid is separated from the filter as much as possible, the filter burned in a small, weighed porcelain crucible, the ash treated with nitric acid and a few drops of hydrochloric acid, and evaporated to dryness. The silver chlorid is then added to the contents of the crucible and cautiously ignited until it just begins to fuse at the edges, cooled and weighed. The weight obtained by subtracting the weight of the empty crucible from that last obtained, and multiplying the difference by 0.2543 is that of the hydrochloric acid in the quantity of solution operated upon.

3. Roussin's Method.[1]—The material, finely hashed and diluted to a thin paste if necessary, is intimately mixed and divided into two perfectly equal parts. To one of these an excess of pure disodic carbonate is added. Each half is then separately treated as follows: They are evaporated to dryness, incinerated in porcelain crucibles to complete carbonization, the carbonized residues extracted with equal volumes of water, and the quantity of hydrochloric acid in each is determined by precipitation with silver nitrate as above described. The difference

[1] Tardieu: "Empoisonnement," 2ème ed., 1875, 243.

between the two amounts so found is the quantity of free hydrochloric acid present in one-half of the material.

A modification of this process, which permits of the determination of that portion of the acid present as acid albumin, has been suggested by Winter-Hayem.[1] Three equal portions are taken in place of two: to one (*a*) excess of disodic carbonate is added, the liquid evaporated on the water-bath, the residue moderately ignited, extracted with water containing nitric acid in excess, boiled, and the total chlorin determined as silver chlorid (gravimetrically or volumetrically). The second portion (*b*) is evaporated, dried, moderately ignited, extracted with water and nitric acid, and the quantity of chlorin, existing as chlorids, similarly determined. The third portion (*c*) is evaporated to dryness, the residue warmed on the water-bath for an hour, after which excess of disodic carbonate is added and the process continued as with *a*. The free hydrochloric acid= $a-c$; that existing as acid albumin $= c-b$.

4. Cohn and v. Mering's Method.[2]—The filtered stomach contents are distilled until two-thirds of the liquid have gone over, water is added to the residue, and the distillation continued. The quantity of volatile acid in the distillate is determined by titration of a portion. The residue is agitated with much ether. In the residue of evaporation of the ether the quantity of lactic acid is determined by titration. The free hydrochloric acid is determined in the distilled liquid, after having agitated it with ether, as follows: Excess of freshly precipitated cinchonin is added, and the mixture digested some time at a moderate temperature. It is then extracted by several agitations with chloroform, a further quantity of cinchonin being added after the third agitation. The residue of evaporation of the chloroform is dissolved in water containing acetic acid, nitric acid is added, and the hydrochloric acid determined as described above as silver chlorid.

5. Sjögvist's Method.[3]—(v. Jaksch's Modification[4]).—Neutral litmus solution is added, and then pure barium carbon-

[1] Corr.-Bl. f. Schweiz. Aertze, 1892, 735.

[2] Deut. Arch. f. kl. Med., 1887, 233; Ber., Berl., 1887, xx., c. 226. This is a modification of the Tardieu-Rabuteau method (Compt. rend. Ac. Sc., Par., lxxx.), cinchonin being substituted for quinin, as the latter decomposes chlorids.

[3] Ztschr. f. physiol. Chem., 1889, xiii., 1.

[4] Monatsheft f. Chem., 1889, x., 464.

ate in slight excess until the red color has disappeared. The mixture is then evaporated to dryness, and the residue moderately ignited. The carbonaceous mass is repeatedly extracted with water, and the barium contained in the solution determined by precipitation and weighing as barium sulfate. The weight of barium sulfate, multiplied by 0.6259, gives the weight of hydrochloric acid present in the material operated upon.[1]

Tests for Hydrochloric Acid and Chlorids.—1. Silver nitrate produces a white, cheesy precipitate of silver chlorid, which is insoluble in nitric acid, soluble in ammonium hydroxid or potassium cyanid solution.

2. Mercurous nitrate produces a white precipitate of mercurous chlorid, which turns black on addition of ammonium hydroxid.

3. Lead acetate produces a white precipitate in solutions which are not too dilute. The precipitate dissolves in hot water, from which it crystallizes on cooling.

4. Free hydrochloric acid, when warmed with manganese dioxid or with lead peroxid, evolves chlorin, which may be recognized by its odor, color, and bleaching action on moist vegetable pigments, if the quantity be sufficient. Bouis[2] applies this reaction to organic mixtures as follows: The material is acidulated with pure acetic acid, filtered through muslin under pressure, and then through paper, a few fragments of potassium chlorate and some gold leaf are added, and the mixture is warmed for an hour or two on the water-bath. If the liquid be very dilute it is concentrated by evaporation on the water-bath. The liquid is then decanted and the dissolved gold tested for by stannous chlorid solution, which produces a purple-red or vio-

[1] For other methods see Bidder u. Schmidt: "Die Verdauungssäfte," Leipzig, 1852. Leo: Centbl. f. d. med. Wissensch., 1889, 481. F. A. Hoffmann: Centlb. f. klin. Med., 1890, No. 40. Salkowski: Jahresber. d. Pharm., 1891, 220. Jolles u. Wallenstein: Pharm. Post, 1891, 445. Graffenberger: Pharm. Ztg., 1891, 392. Katz: Wien. med. Wchnschr., 1890, 2193. Lüttke: Jahresb. d. Pharm., 1891, 226. Rosenheim: Deutsch. med. Wchnschr., 1891, 1155; 1892, 280, 309. Wittmann: Jahresb. f. Kinderhlk., 1892, xxxiv., 1. Marino and Dotto: Deut. med. Wchnschr., 1892, 126. Wagner: Centlb. f. d. m. Wissensch., 1892, 192. v. Pfungen: *Ibid.*, 262. Dmochowski: Deut. m. Ztg., 1892, 388. Maurice: J. d. pharm. et de chim., 1892, 6 s., v., 350, 450. Kossler: Ztschr. f. physiol. Chem., 1893, xvii., 91. Friedländer: Apoth. Ztg., 1892, 665. Martins u. Lüttke: Centlb. f. d. m. Wissensch., 1893, 218.

[2] Ann. d'hyg., etc., 1874, 2 s., xli., 457

let precipitate. Chlorids do not produce this reaction except in the presence of free sulfuric acid.

Acid Chlorids.

Solutions of certain chlorids, acid in reaction or containing notable excess of hydrochloric acid, exert an action upon the economy which is a combination of the corrosive effects due to the acid and the truly poisonous action of the metal. The most important of these are antimony trichlorid, which will be considered along with the toxicology of the other antimonials; zinc chlorid, which has already been referred to as the "soldering liquid" used by tinsmiths, and which will be further discussed among the zinc compounds, and mercuric chlorid, or "corrosive sublimate," which will be treated of under "Mercury."

NITRIC ACID.

Pure nitric acid is a colorless liquid, which boils at 86° (186°.8 F.) and solidifies at −40° (−40° F.), gives off white fumes when exposed to air, and is strongly acid in reaction. The specific gravity of the concentrated acid is 1.522. The specific gravity and boiling-point of the diluted acid vary with the concentration:

Specific gravity.	Per cent. HNO_3.	Boiling-point.	Specific gravity.	Per cent. HNO_3.	Boiling-point.	Specific gravity.	Per cent. HNO_3.	Boiling-point.	Specific gravity.	Per cent. HNO_3.	Boiling-point.
1.522	100.0	86	1.315	50.00	113°	1.207	33.33	108°	1.155	25.00	104°
1.486	88.75	99	1.297	46.67		1.197	31.82		1.138	23.00	
1.452	77.78	115	1.277	43.75		1.188	30.44		1.120	20.00	
1.420	70.00	120	1.260	41.18		1.180	29.17		1.101	17.44	
1.390	63.64	119	1.245	38.89		1.173	28.00		1.089	15.00	
1.361	58.33	117	1.232	36.84		1.166	26.92		1.068	11.43	
1.338	53.84		1.219	35.00		1.160	25.93		1.022	4.00	

If a strong acid be distilled, the boiling-point gradually rises from 86° until it reaches 120° (248° F.), when it remains constant, the specific gravity of both distillate and distilled being 1.42. If a weak acid be distilled the boiling-point rises until it becomes stationary at the same temperature.

The formula of nitric acid is HNO_3, and its molecular weight 62.98. When exposed to air or to light, or when heated, it is decomposed into nitrogen tetroxid, NO_2, water, and oxygen.

Hence it is useful as an oxidizing agent. Most of the metals dissolve in nitric acid as nitrates, a portion of the acid being at the same time decomposed into nitrogen dioxid (NO) and water. Gold and platinum are not attacked by nitric acid. The salts of nitric acid (nitrates) are for the most part colorless and, with the exception of a few "basic salts," soluble in water. When fused they act as powerful oxidants. They are decomposed by sulfuric acid at more or less elevated temperatures, with liberation of the acid.

The commercial acid, *aqua fortis*, is a yellow liquid, met with in two degrees of concentration: *single aqua fortis*, specific gravity about 1.25 = thirty-nine per cent. HNO_3; and *double aqua fortis*, specific gravity about 1.4 = sixty-four per cent. HNO_3. It contains as impurities the oxids of nitrogen, sulfuric acid, iron, fixed salts, and sometimes chlorin and iodin.

Acidum nitricum, U. S., Br., is a colorless acid, free from the impurities above mentioned, and of specific gravity, 1.42 = seventy per cent. HNO_3. Like the C. P. acid, specific gravity 1.522 = one hundred per cent. HNO_3, used in chemical laboratories, it must be kept in bottles completely filled and protected from light. The dilute acid of the U. S. P. is of specific gravity 1.059, and contains ten per cent. HNO_3.

Fuming nitric acid is a yellow liquid, fuming when exposed to the air, made by saturating strong nitric acid with nitrogen tetroxid. A similar acid, though more dilute, is known as *nitroso-nitric acid*.

Although nitric acid was known certainly in the thirteenth century, probably to the Arabians of the eighth century, and possibly to the ancient Egyptians, we know of no earlier reference to its toxic action than that of Bruno Seidel, who relates the case of a girl who was killed by drinking the acid.[1] During the seventeenth and eighteenth centuries deaths from nitric acid occurred occasionally, and Tartra, whose thesis[2] was for that period an exhaustive treatise upon the toxicology of the mineral acids, relates 55 cases, 29 of which were first described in this thesis. Of these, 26 died—19 from the primary action, and 7 from the secondary effects of the corrosive. Considering

[1] "De morbis incurabilis," Francof., 1593, p. 13.

[2] "Essai sur l'empoisonnement par l'acide nitrique," Paris, An x. [1802].

the much more extended use of nitric acid at the present time, the frequency of intoxications by it has not materially increased since the time Tartra. We find reference to 73 cases since 1802, distributed as follows: 1802–9, 1; 1810–19, 3; 1820–29, 6; 1830–39, 8; 1840–49, 5; 1850–59, 9; 1860–69, 16; 1870–79, 6; 1880–89, 13; 1890–95, 6. At the present time nitric acid is apparently less frequently the cause of death directly than either hydrochloric or sulfuric acid.

We find mention of thirteen criminal administrations of nitric acid, all of which occurred on the continent of Europe.[1] The earliest was one of Tartra's cases.[2] In 1801 a woman of thirty-five died, it was alleged, from the effects of nitric acid mixed with white wine which was poured down her throat while she was intoxicated. Two other French cases are reported. One that of a woman who died from the effects of nitric acid poured into her ear by her husband while she was intoxicated, "to punish her";[3] the other the case of a woman who killed her infant shortly after its birth by pouring nitric acid down its throat.[4] Seven cases are reported in German literature. One was in an adult female who took the acid under the advice of her lover in the belief that it would provoke abortion;[5] the others were in infants or young children.[6] One case, also in an infant, is reported from Hungary,[7] and two cases in young children from Finland.[8]

Symptoms and Diagnosis.

The effects and manifestations produced by nitric acid are similar to those caused by sulfuric or hydrochloric acid. The

[1] Gribble and Hehir (Med. Jurispr. for India," 3d ed., 1892, p. 435) state, on the authority of Dr. Rogers, that two cases of intoxication occurred in India during 1883, in which nitric acid was found mixed with coffee.

A man was convicted of murder by a mixture of sulfuric and nitric acids in 1889. (See Mixed Acids.)

[2] Tartra: *Loc. cit.* Orfila: "Tr. de. tox.," 5ème ed., i., 166.

[3] Morisson: Arch. gén. de. méd., 1826, xi., 104. Orfila: *Op. cit.*, 172.

[4] Taylor: "Poisons," 3d Am. ed., 210, from Cazauvieilh: "Du suicide, de l'aliénation mentale," etc., Paris, 1840.

[5] Buchner: Friedreich's Bl. f. ger. Med., 1866, xvii., 192.

[6] Osenbrüggen: Friedreich's Bl. f. ger. Med., 1867, xviii., 75. Buchner: *Loc. cit.* (2 cases). Casper-Liman: "Handb. d. ger. Med.," 8te Aufl., ii., 549; and Lesser: "Atlas d. ger. Med.," Pl. i., Fig. 2; Buchner: *Loc. cit.*, 1886, xxxvii., 9 and 14.

[7] Maschka: Samml. Gutacht. Prag. m. Fak., 1867, 3 F., 284.

[8] Fagerlund: Vrtljschr. f. ger. Med., 1894, 3 F., viii., Supplhft., 59.

principal point of difference is in the color of the stains formed upon those portions of the skin and visible mucous membrane touched by the acid. Owing to the *xanthoproteic* reaction produced by nitric acid upon the albuminoids, these are colored a bright yellow, which turns to orange on the addition of ammonia. Consequently the stains produced by nitric acid are usually distinctly yellow, and subsequently become dirty light-brown, and the superficial layer readily peels off in a short time. Yellow stains are, however, neither constantly present nor certainly characteristic. The acid may be taken from a spoon or other vessel passed well back into the mouth, when no visible stains are formed, as in a case reported by Berselli.[1] The lips and skin of the chin have been seen to be excoriated, and the tongue and buccal mucous membrane white or red in place of yellow,[2] even when they have been corroded by nitric acid; but such cases are unusual and exceptional. If the patient be seen some time after the acid has been taken, the yellow stain may be concealed by an eschar—as in a man who was seen on the day after taking a teaspoonful of the acid. The lips and tongue were covered with a thick brown scab, which peeled off the next day, exposing the brightly yellow stained mouth and tongue.[3] On the other hand, yellow stains may be produced by picric acid or by any one of a number of other yellow dyes, and have even been met with in corrosion by sulfuric acid.[4]

If the patient be seen during the first twenty-four hours the most certain means of establishing a diagnosis in doubtful cases is by the detection of nitric acid in the urine, which, moreover, is red or dark in color.[5]

Prognosis.

The prognosis in nitric acid intoxication is even more unfavorable than in that by hydrochloric acid. Of 64 persons affected 54, or 84.4 per cent., died, and 10, or 15.6 per cent., recovered from the primary effects. Of 44 fatal cases in which

[1] Gaz. med. ital. Prov. Venet., 1860, iii., 201.

[2] Duckworth: Tr. Clin. Soc., London, 1886, xix., 53. Richardière: Ann. d'hyg., etc., 1886, 3 s., xv., 88. St. Geo. Hosp. Rep., 1877–78, ix., 18.

[3] Guy's Hosp. Rep., 1859, 140.

[4] Taylor: Guy's Hosp. Rep., 1846, 2 s., iv., 396.

[5] Richardière: *Loc. cit.*; Schulzen: Arch. f. Anat., Phys., etc. 1864, 500. Ipsen: Vrtljschr. f. ger. Med., 1893, 3 F., vi., 11.

the duration is reported death occurred in less than three hours in 5 (11.3 per cent.), during the first twenty-four hours in 19 (44.2 per cent.), during the first week in 28 (63.6 per cent.), within a month in 33 (75 per cent.), and in periods longer than a month in 11 (25 per cent.). The extremes of duration were "in a few minutes" in a new-born infant,[1] and in three hours in a woman of fifty-five years,[2] as minima; and as maxima, two years in one case and "several years" in another.[3]

Post-Mortem Appearances.

These are very similar to those observed after death from sulfuric acid, and under like conditions the destruction of tissue is quite as extensive. In place, however, of the blackening of the gastric mucous membrane so frequently met with after death by sulfuric acid, the stomach, as well as the œsophagus, pharynx, and tongue, are usually yellow in color. The color varies from a bright yellow to a dirty olive yellow, and frequently alternates with black or red.[4] While the yellow color is only absent in the œsophagus and stomach after death from the primary action of nitric acid in those exceptional cases in which the acid has not been swallowed,[5] its presence may be due to causes other than nitric acid. Not only is the gastric mucous membrane frequently tinged yellow by biliary pigment and by the formation of arsenic trisulfid, but in a few instances a more or less extensive yellow coloration has been observed after death caused by sulfuric[6] or hydrochloric[7] acid, or by the mineral alkalies.[8]

[1] Taylor: "Poisons," 3d Am. ed., 210.

[2] Ipsen: Vrtjschr. f. ger. Med., 1893, 3 F., vi., 11. In the notice of Bernt's case, of death in one and three-quarter hours, quoted by Taylor from Sobernheim ("Handb. d. pract. Tox.," Berlin, 1838, 402) it is not stated whether the deceased was a child or an adult.

[3] Tardieu: "Empoisonnement," 2ème ed., 1875, 238, 239, quoted from Moutard-Martin and Vernois.

[4] Colored plates are to be found in Lesser's "Atl. der ger. Med.," Pl. ii., Fig. 2 and in Vrtljschr. f. ger. Med., 1893, 3 F., vi., T. 1 and 2 (Ipsen).

[5] Buchner: Friedreich's Bl. f. ger. Med., 1866, xvii., 187; 1886, xxxvii., 9.

[6] Habershon: Med. Times and Gaz., 1855, n. s., xi., 470. Jenner: *Ibid.*, 1857, xv., 629. Falk: Vierteljschr. f. ger. Med., 1875, n. F., xxiii., 14. Maschka: *Ibid.*, 1881, xxxiv., 197.

[7] Kryspen: Arch. d. Pharm., 1861, clvii., 23. v. Wunschheim: Prag. med. Wchnshr., 1891, xvi., 605.

[8] See p. 269.

Analytical.[1]

If the acid have been swallowed in notable quantity and have caused death within a short time, nitric acid, both free and in combination, should be found not only in the stomach contents, but in other tissues as well. In the body of a suicide who died in three hours from the effects of a large but undetermined dose of nitric acid, Ipsen[2] found free nitric acid in the stomach, œsophagus, left pleural cavity, duodenum, pancreas, liver, and spleen, and nitrates in the urine, 50 c.c. of which contained 1.14975 gm. HNO_3, as well as in the brain, and in the blood from the heart and lungs. As in fatal cases of acidism the circulating liquids and the tissues do not become acid before death, although the degree of their alkalinity is diminished, the presence of free nitric acid outside of the alimentary canal was due to post-mortem diffusion of the acid through the non-perforated wall of the alimentary canal, although only nineteen hours elapsed between the death and the autopsy. That such diffusion takes place rapidly was shown by Ipsen by experiment upon a cadaver.

For the separation of nitric acid and of nitrates from portions of the body, the process already described (see p. 222) is to be followed. The extraction with alcohol should, however, be made at the ordinary temperature, and should not be unduly prolonged, because of the tendency of the acid to act upon the alcohol.

Or Vitali's modification of the Tardieu-Rabuteau sulfuric acid method[3] may be followed for the extraction of free nitric acid. The liquid part of the material, or an aqueous extract, filtered and expressed, is treated with alcohol, filtered, and evaporated to expulsion of the alcohol. Freshly precipitated quinidin is added and the mixture warmed slightly, after which it is filtered and concentrated. To three volumes of this liquid two volumes of chloroform are added and enough absolute alcohol to dissolve the chloroform in the aqueous liquid and to hold it in solution; and, finally, without agitation, half the original volume of water. In this way the chloroform, holding in solu-

[1] See also p. 220.

[2] Vrltjschr. f. ger. Med., 1893, 3, vi., F., 11.

[3] "Man. di Chim. Toss.," Milano, 1893, p. 239.

tion the alcohol and quinidin nitrate, is separated, while alkaline nitrates remain in the aqueous alcoholic layer. The chloroform stratum is drawn off and evaporated to dryness; the residue is extracted with a mixture of absolute alcohol and anhydrous chloroform; and the extract is filtered through a double or triple filter, and again evaporated to dryness. The nitric acid in the residue is converted into potassium nitrate by the action of dipotassic carbonate, and known fractions of the solution are used for identification and quantitative determination.

Tests for Nitric Acid and Nitrates.—1. The free acid when warmed in contact with copper filings is reduced and gives off brown fumes, which, if present in small amount, are best seen by looking into the mouth of the test tube. With nitrates only in presence of free sulfuric acid.

2. If a solution of a nitrate or of nitric acid be mixed with an equal volume of concentrated pure sulfuric acid, cooled, and a concentrated solution of ferrous sulfate is floated on the mixture, a red-brown band is found at the junction of the two strata. A similar reaction is produced by selenious acid, but if the layers be mixed and allowed to stand selenium separates as a red deposit.

3. Add to some hydrochloric acid in a test tube enough indigo-carmine solution to give it a pale blue color, and boil; the blue color should remain. If now a nitrate or nitric acid be added and the liquid boiled, the color is discharged. A like effect is produced by chlorin and by other oxidizing agents.

4. A crystal of brucin dissolves in *pure* sulfuric acid, forming a colorless solution. If to this a nitrate or nitric acid be added, a brilliant red-orange color is developed, which gradually fades to yellow. Chloric acid and its salts act like nitric acid, but with sulfuric acid alone they produce a yellow or orange color, and evolve a greenish, suffocating gas. This reaction is only exceeded in delicacy by No. 7 (Berthemont).

5. Narcotin is dissolved by pure concentrated sulfuric acid, forming a light-yellow solution, which turns purplish when heated. But if nitric acid or a nitrate be present, the cold solution is reddish-brown and turns dark red when warmed (Wormley).

6. If to a few drops of a solution of paratoluidin sulfate (or

a sulfuric acid solution of commercial anilin oil) a solution of a nitrate be added, and then an equal volume of concentrated sulfuric acid in such manner that the liquids do not mix, a red zone, gradually changing to dark yellow, is formed at the junction of the liquids. Nitrites produce a yellowish or brownish color, which only becomes red after a time. Chlorates give the same reaction as nitrates (Braun, Longi).

7. A drop of a freshly prepared solution of 0.01 gm. diphenylamin in 100 c.c. of pure concentrated sulfuric acid is placed in a porcelain dish, and a minute drop of a solution of a nitrate or of nitric acid brought into it with a pointed glass rod; a fine blue zone is formed around the lesser drop, which becomes more intense as the two liquids mix, gradually assumes a green tinge, and finally fades out. Although this is the most delicate of the nitric acid reactions, it is not so characteristic as some of the others; a blue color is also produced by nitrites, chlorates, hypochlorites, bromates, iodates, vanadates, chromates, permanganates, molybdates, salts of iron, barium peroxid, and hydrogen peroxid (Kopp, Laar).

8. One part of phenol is dissolved in four parts of concentrated sulfuric acid, and two parts of water are added. If a drop or two of this reagent be added to a solid nitrate, a reddish-brown color is produced, which, on addition of a drop of two of strong ammonium hydroxid solution, turns yellow or greenish (Sprengel).

Quantitative Determination.—The quantity of nitric acid is best determined by v. Dumreicher's method:[1] To the solution containing nitric acid or nitrate contained in a flask a freshly prepared solution of stannous chlorid[2] is added, and the mixture boiled for an hour, after which it is transferred to a porcelain capsule and evaporated on the water-bath to the formation of a crystalline crust. The capsule is left on the water-bath for another half-hour and then allowed to cool. By this treatment nitric acid is converted into ammonia, which is retained by the acid. The cooled solution is distilled with caustic potash, and the distillate collected in normal acid solution.

[1] Sitzber. d. k. Acad. d. Wissensch. z. Wien, 1880, lxxxii., 2, p. 583.

[2] Made by dissolving 16 gm. of granulated and washed tin, in 60 gm. of forty-per-cent. *pure* hydrochloric acid, and used in such amount that the above quantity is present for each gramme of nitric acid.

The quantity of ammonia is determined by titration. The quantity of ammonia (NH_3) found, multiplied by 3.683, gives the quantity of nitric acid (HNO_3) present.

Nitrous Fumes.

Nitric acid has in several instances been indirectly the cause of death. When the acid is exposed to the air, or, more rapidly, when it is in contact with organic matter or with metals, it is partly volatilized and partly decomposed with formation of the oxids of nitrogen, particularly of nitrogen tetroxid, NO_2. The *fumes* or *vapors* thus given off are brown in color and are actively poisonous, being particularly dangerous because of the comparatively slight discomfort which they produce *at once* when inhaled. We have met with the records of twenty-two cases of poisoning by the inhalation of such fumes, of which fifteen caused death in from ten to forty hours, the average duration being twenty-two hours. In fourteen of the cases, eleven of which were fatal, the poisoning was due to an attempt on the part of the deceased to repair the damages caused by the breaking of a vessel containing the acid, or by its having been otherwise spilled out.[1] In one case a morocco worker was exposed to the fumes,[2] in another a chemist,[3] in another an operative in a sulfuric acid works inhaled the fumes escaping from the leaden chambers and was found dead.[4] A servant girl was severely poisoned by inhaling the fumes given off by strong nitric acid, which she was carrying in a tin box (!).[5] In preparing a fertilizer containing superphosphate (probably containing excess of sulfuric acid) and Chili saltpetre among its ingredients, the mass took fire and evolved thick clouds of orange-yellow vapors. Some thirty persons were poisoned, of whom two died.[6]

Symptoms.

No effects, save a trifling irritation of the air passages, are felt for from two to four hours after the inhalation, if we except the temporary sense of suffocation sometimes experienced at the time, when the vapors are inhaled in a condition but slightly diluted with air. There is then a sense of uneasiness and loss of muscular power, accompanied by sense of constriction of the chest, pain on inspiration and coughing. The face is pale, the lips cyanosed, the forehead bathed in cold perspiration, the extremities cold, and the pulse weak. The respiratory organs are the seat of very acute hyperæmic inflammation of the bron-

[1] In one of these the evolution of the fumes was favored by sawdust being thrown upon the spilled acid.

[2] Wharton and Stillé: "Med. Jur.," 4th ed., ii., 529.

[3] Schmitz: Berl. kl. Wchnschr., 1884, xxi., 428.

[4] Enlenberg: Vrtljschr. f. ger. Med., 1876, xxv., 209.

[5] Schmitz: *Loc. cit.*

[6] Pott: Deutsch. m. Wchnschr., 1884, x., 451.

chial mucous membrane, which at first is limited to the larger tubes, but rapidly extends to the finest bronchi. The sputa are lemon yellow to dirty gray in color, tenacious, adherent to glass, alkaline in reaction,[1] and contain a few pavement and columnar epithelial cells, innumerable nuclei, large numbers of red and white blood corpuscles, and groups of yellow non-refracting pigment globules, which exhibit a lively Brownian movement. Vomiting occurs but rarely, and pain, which is in some cases intense, is not epigastric but thoracic. The urine is normal. In fatal cases the dyspnœa increases, sometimes after marked remissions, coarse râles are heard all over the chest, and death occurs with all the signs of acute pulmonary œdema. Recovery is slow, and occupies one to two weeks.

Post-mortem Appearances.

The most marked changes are found in the thoracic organs. The pleuræ are adherent over more or less of their extent. The lungs are extremely hyperæmic, and their tissue is extensively disorganized, saturated with black, fluid blood, presenting a spleen-like appearance on section, and non-crepitant except in a few places. The cavities of the heart, particularly those of the right side, are distended with black blood, partly fluid and partly imperfectly coagulated, and a similar material fills the great vessels.[2] The mucous membrane of the trachea and bronchi is more or less reddened. The stomach is distended with gas, contains a small quantity of fluid, which may be strongly acid in reaction, and its mucous membrane is inflamed, thickened, and stained yellow in places. The veins of the pia mater and the arteries at the base of the brain are distended with dark-colored, tarry, or fluid blood.

Acid Nitrates.

Nitrates which are used in solution containing excess of acid, or which are readily reduced by contact with organic substances, produce effects which are similar to those of nitric acid itself, more or less complicated by the toxic effects of the metallic element. Examples of such nitrates are the acid mercuric nitrate and silver nitrate.

Acid Mercuric Nitrate.—See Mercury.

Silver Nitrate—*Lunar Caustic, Lapis infernalis.*

Acute intoxications by this substance are of very rare occurrence. Of seven cases reported five were the results of accident in children, but one of which died, a child of fifteen months, into whose throat a stick of lunar caustic slipped during its medicinal application. Death occurred in six hours in violent convulsions.[3] Of the two cases in adults, one

[1] Pott: Deutsch. m. Wchnschr., 1884, x., 451.

[2] In Eulenberg's case (*loc. cit.*) in which the gas inhaled contained other ingredients than nitrous fumes, the heart and great vessels were found empty.

[3] Scattergood: Brit. Med. Jour., 1871, i., 527.

was that of a woman of fifty-one years who died in three days from the effects of a six-ounce mixture containing fifty grains of silver nitrate, taken in divided doses;[1] the other was an attempted suicide, who recovered after swallowing about 30 gm. (one ounce) of silver nitrate.[2]

The symptoms are those of intense gastro-enteritis.

The antidote indicated is common salt, followed by an emetic. Silver chlorid and sodium nitrate are produced. The former, although insoluble in water, is soluble to some extent in sodium chlorid solution, and also forms with albuminous substances a compound which is soluble in sodium chlorid solution and in the digestive liquids.

Mixed Acids.

Mixtures of mineral acids are used in the laboratory and in the arts, and have been swallowed by man in a few instances, producing effects similar to, but even more intense than, those caused by either acid alone.

A mixture of nitric and hydrochloric acid, in the proportion of one molecule of the former to three of the latter (one part by weight HNO_3, specific gravity 1.42, to four parts HCl, specific gravity 1.16), is used as a solvent for gold and platinum, under the names *aqua regia* and *nitromuriatic acid*, and in a diluted form as a medicine, *acidum nitrohydrochloricum dilutum*, *U. S. P.* In the concentrated form it is a colorless liquid when first prepared, but soon becomes orange yellow, which when warmed is decomposed, with liberation of chlorin and formation of water and compounds of chlorin, nitrogen, and oxygen, NOCl and $NOCl_2$. Its solvent action upon gold and platinum is due to the nascent chlorin, which unites with the metals to form auric or platinic chlorid.

We find but two references to intoxication by aqua regia in the human subject: one a fatal case reported by Liouville;[3] the other a non-fatal case in a man who took four or five swallows of the dilute acid and in whose urine nitric acid was detected on analysis.[4]

A mixture of sulfuric and nitric acids is extensively used in the preparation of dynamite, gun-cotton, and other nitro-deriv-

[1] Taylor: "Poisons," 3d Am. ed., 475.

[2] Orfila: "Tox.," 5ème ed., ii., 22.

[3] Bull. Soc. d'anat., Paris, 1872, 2, s., xvii., 274.

[4] Schultzen, *ex* Kobert: "Intoxikationen," p. 212.

atives. In 1889 a man was convicted in England of the murder of a woman by pouring such a mixture of acids down her throat while she was in bed.[1]

OTHER MINERAL ACIDS.

Hydrofluoric Acid—HF, 20.06.

This intensely acid gas is usually produced by the action of sulfuric acid upon calcium fluorid (fluorspar), and is used principally for etching upon glass, which is attacked by it whether in the gaseous form or in solution. In this industry the sand-blast has to a great extent, although not entirely, replaced the use of the acid. In solution it has also been used to a limited extent as a germicide.

Its action upon the animal economy is most intense, yet but few deaths have been caused by it, owing to its limited uses.

There is some difference in opinion as to the effects of inhalation of gaseous hydrofluoric acid. Most writers upon professional hygiene assert that those exposed to an atmosphere contaminated with the gas suffer from ulcerations of the conjunctiva, nose, mouth, and gums, and from severe laryngitis and bronchitis. According to Kobert,[2] Bergeron makes the astounding statement that 400 gm. of hydrofluoric acid may be inhaled daily. The inhalation of so powerfully acid a gas cannot but be deleterious. Indeed, the deaths of two chemists were due in whole or in part to inhalation of this gas: Nicklès, of Nancy, who died in 1869, and whose death was considered as accelerated by exposure to the fumes of phosphorus and fluorin compounds;[3] and the Belgian chemist, Louyet, whose death resulted from the inhalation of hydrofluoric acid during his endeavors to isolate the element.[4]

Silicon fluorid—SiF_4,

is another gaseous fluorin compound, possessed of poisonous qualities which were recognized by Eulenberg from experiments upon animals,[5] and observed in the human subject in two fatal cases by Cameron.[6] This gas is produced by the action of sulfuric acid upon calcium fluorid in the presence of silicic acid or a silicate, and is decomposed by contact with moisture, with formation of gelatinous silicic acid and of hydrofluoric acid. In Cameron's cases the victims were operatives in a fertilizer works in which phosphates containing notable quantities of fluorid and silicate were treated with sulfuric acid. They complained

[1] Reg. *v.* Lipski: C.C.C., July, 1888. Taylor: "Med. Jur.," 11th Am. ed., 102.

[2] "Intoxikationen," 377.

[3] Jour. d ph. et de chim., 1869, 4 s., ix., 446.

[4] Rabuteau: "Toxicologie," 2ème ed., 710.

[5] "Schädlichen und giftigen Gasen," Braunschweig, 1865, p. 465.

[6] Dublin Jour. M. Sc., 1887, 3 s., lxxxiii., 20.

of a sense of suffocation, and expectorated frothy material. Death followed in a few hours, after increased dyspnœa, and in full consciousness. Cameron considers that on contact with the moist bronchial mucous membrane the gas is decomposed, the gelatinous silicic acid forming a varnish-like coating upon the bronchi to their most minute ramifications, and the irritating hydrofluoric acid causing reflex spasmodic contraction. From the lungs of one man 0.75 gm. (11.6 grains) of silicic acid were obtained.

We know of but two cases of death from swallowing the acid in solution, both suicides. The earliest case is that reported by King:[1] A man of forty-six years, of intemperate habits, swallowed 15 c.c. (℥ ss.) of the solution, was immediately seized with severe pain and vomiting, and died in thirty-five minutes from cardiac paralysis, the respiration continuing for some time after disappearance of the radial pulse and heart beat. The buccal mucous membrane was white, the tongue, gums, epiglottis, and œsophagus denuded of epithelium; the mucous membrane of the stomach softened, corroded in places, black on the prominences of the rugæ, and intensely red and ecchymosed in the depressions. The duodenal mucous membrane was somewhat injected. In the opening of the glottis was a small quantity of dark-brown mucus, insufficient, however, to account for the death, which was more probably due to true poisoning following absorption of the acid.

In 1885 a manufacturer of glass signs died in the Chambers Street Hospital, New York, in about two hours after swallowing about a gill (118 c.c.) of the acid used in his trade.

Solutions of the acid in contact with the skin produce severe and painful burns which heal very slowly.

The toxic effects of the fluorids have been studied experimentally by Rabuteau,[2] Muller,[3] Tappeiner,[4] Schulz,[5] and Hewelke.[6]

For the DETECTION of fluorin in organic mixtures, these may be incinerated with the addition of a little soda, and the ash warmed with concentrated sulfuric acid in a platinum crucible covered with a watch glass partly coated with wax or paraffin. The uncoated parts of glass are roughened. This method is not available if the materials contain silica, in which case the non-etching silicon fluorid is formed. Tammann's method[7] avoids this source of error: the substance, intimately mixed with quartz powder, is placed in a flask provided with a cork having three holes; sulfuric acid is added through a funnel tube, and the flask heated; a stream of dry air is caused to carry the silicon

[1] Tr. Path. Soc., London, 1873, xxiv., 98.

[2] Thèse de Paris, 1867, "Toxicologie," 2ème ed., 709.

[3] Diss., Greifswald, 1889.

[4] Arch. f. exp. Path. u. Ph., 1889, xxv., 203; 1890, xxvii., 108.

[5] *Ibid.*, 1889, xxv., 326.

[6] Deut. med. Wchnschr., 1890, xxii., 477.

[7] Ztschr. f. physiol. Chem., 1888, xii., 322, and Ztschr. f. anal. Chem., 1885, xxiv., 328.

fluorid formed into a narrow vessel containing water, where it is decomposed with separation of gelatinous silicic acid.

The mere detection of traces of fluorin in organic mixtures is not evidence that a toxic fluorin compound has been taken, as fluorin is a normal constituent of many animal tissues. It is most abundant in the enamel of the teeth, which contains three to four per cent., calculated as calcium fluorid, and in bones, whose ash contains about the same percentage. It has also been found in less amount in the blood, milk, brain, and in hen's eggs.

Boric Acid—*Boracic Acid*—H_3BO_3—61.78.

Is a crystalline solid, unctuous to the touch, slightly bitter, odorless, soluble in twenty-five parts of water at 10° (50° F.), which is used as an antiseptic. In recent medical literature several cases have been recorded in which more or less severe symptoms followed its use either in the form of powder dusted on abraded surfaces, or in aqueous solution for washing out cavities in surgical practice.[1]

The symptoms consist of loss of appetite, nausea and persistent vomiting, hiccough, marked cardiac weakness, emaciation, diarrhœa, great depression, coryza, conjunctivitis, bronchitis and pharyngitis, and a papular eruption on the skin near the seat of application and on the neck, arms, and chest. The urine is suppressed or diminished in amount. In Bruzelius' case, in which enemata of 1,400 to 1,500 c.c. of tepid saturated aqueous solution of boric acid were given twice a day for five days, and once a day for three days thereafter for chronic diarrhœa, the urine was tested from the start and found to contain boric acid, which disappeared with the cessation of the treatment.

Three fatal cases have been reported: two by Molodenkow,[2] one that of a man of twenty-five years who died in four days after his pleural cavity had been washed out with a large quantity of the solution, the other that of a boy of sixteen years who died in three days after a lumbar abscess had been opened and washed out. In Brose's case, in which the powdered acid had been applied to an open wound, there was found a large clot in the right ventricle, partly ante-mortem. The stomach was congested and there were several spots of erosion of the mucous membrane. The intestines were greatly congested and inflamed. The liver was enlarged, and the right lobe congested and softened. The spleen was enlarged and much inflamed.

Methods for the detection of boric acid or of borates in organic mixtures are not very satisfactory, owing to their volatility with vapor of water or when heated, and the sparing solubility of even the least soluble borates. Probably the best method consists in drying the ma-

[1] Spencer: Northwestern Lancet, 1888, viii., 22. Brose: M. News, Phila., 1883, xliii., 199. Huse: *Ibid.*, 1882, xl., 704. Welch: Med. Rec., New York, 1888, xxxiv., 531 (3 cases). Bruzelius: "Hygiea," Stockh., 1882, xliv., 548. Lemoine: Gaz. méd. d. Par., 1890, xviii., 205 (3 cases).

[2] Med. News, Phila., 1882, xl., 571.

terial, fusing the residue with sodium carbonate and nitrate, and testing the residue for borates: 1. By adding alcohol and sulfuric acid, and igniting the alcohol; the flame is colored green, and shows four lines in the spectrum, in the yellow, green, and blue (?, 581, 548, 544, 519 Boisbaudran). 2. A solution in dilute hydrochloric acid, when dried upon turmeric paper, stains it red-brown, and the color is changed to black-blue by caustic potash. The following reagents cause precipitates in solutions of borates: 3. Calcium chlorid, white, soluble in ammonium chlorid. 4. Ferric chlorid, yellow. 5. Silver nitrate, white. 6. Solutions of barium chlorid, magnesium chlorid, and lead nitrate also form white precipitates in solutions which are not too dilute.[1]

Chromic Anhydrid—*Chromic Acid*—CrO_3—100.33.

Crystallizes in brilliant crimson prisms, very soluble in water and in alcohol, forming yellow or orange solutions. It is produced by the action of sulfuric acid upon potassium dichromate solution, and consequently exists in "battery fluids" such as "electropoion." It is a powerful escharotic, for which purpose it is used medicinally.

In two[2] of the thirteen reported cases the patient swallowed some of the solution which was being applied to the tonsils with a brush, Six cases[3] of which three were fatal, were in females to whom the anhydrid or its solution was medicinally applied. In one instance a man made an unsuccessful attempt at suicide by taking 1 gm. (15 grains) of the acid in aqueous solution,[4] and in the remaining cases a battery fluid was swallowed with suicidal intent in one instance, and with fatal results in two.[5]

In Fowler's case there were severe epigastric pain, agonizing vomiting of green ropy matter, collapse, scarcely perceptible pulse, fear of impending death, contracted pupils, deafness, purging, and copious urination, with recovery in three hours. In Tisné's case there were severe gastritis and vomiting, great pain in the back of the neck, and vertigo. After four hours the symptoms abated, but there remained constipation and suppression of urine. The urine of the next day gave no reaction with silver nitrate. In von Limbeck's case the symptoms caused by chromic acid were complicated with those due to sulfuric acid. In White's case the intellect remained clear until death.

[1] See also Villiers and Fayolle: Ann. d'hyg., etc., 1895, 3 s. xxxiv., 272.

[2] Fowler: Brit. M. Jour., 1889, i., 1113. Tisné: J. d. méd. de Paris, July 10th, 1887, xiv.

[3] White: Univ. M. Mag., 1889–90. ii., 54 (D.). Mosetig, Falck: "Toxikologie," p. 144 (D.). Bruck: *Ibid.* Jacob: Bay. aerztl. Int. Bl., 1878, xxv. (2 cases). N. Schmidt's Jahrb., 1884, cci., 129 (D.).

[4] Wardner: M. and S. Reptr., Phila., 1869, xx., 362.

[5] v. Limbeck: Prag. med. Wochenschrift, 1887, xv., 25 (S.). Bernasconi: Thèse, Lyon, 1883 (2 D.). Whitney: "Sei-i-Kwai," Tôkyô, 1887, vi., 227.

No post-mortem after death from chromic acid taken internally is reported.

In White's case chromium, probably in the form of sodium chromate, was found by analysis in the kidneys and liver, but none in the rectus muscle. In von Limbeck's case chromium was found to be present in the urine, vomit, and fæces.

For the detection of chromium in the urine, fæces, and vomit these are dried, ignited with potassium nitrate, and the residue is dissolved in water. In this solution: 1. Silver nitrate forms a dark brownish-red precipitate, readily soluble in nitric acid or in ammonium hydroxid. 2. When heated with dilute hydrochloric acid and alcohol, or with the same dilute acid and stannous chlorid or metallic zinc, the yellow or orange color changes to green. 3. Lead acetate forms a yellow precipitate, insoluble in ammonium hydroxid, soluble in caustic potash or soda, and insoluble in acetic acid.[1]

FIXED MINERAL ALKALIES.

The most important of these are the hydroxids and dimetallic carbonates of the alkaline metals.

Potassium hydroxid — *caustic potash, potassa, potash*—**KHO**—and **sodium hydroxid**—*caustic soda, soda*—**NaHO**—these are hard, white, amorphous solids, usually met with in irregular masses or in cylindrical sticks, very soluble in water, and forming intensely alkaline and corrosive solutions. They are used in medicine in the solid form as escharotics, either alone or combined with quicklime (*potassa cum calce, U. S. P.*), or in solution in the proportion of fifty-six parts in one thousand, as *liquor potassæ* and *liquor sodæ, U. S. P.* In the arts and in the household they are used in soap-making and in cleansing and bleaching. *Soap lye, caustic lye, soap lees, laugenstein, laugenessenz*, consist of sodium or potassium hydroxid with varying quantities of the carbonate, formed by absorption of atmospheric carbon dioxid.

Dipotassic carbonate — *carbonate of potash, pearlash, salt of tartar, potassii carbonas, U. S. P.*—K_2CO_3 or $K_2CO_3 + 3$ **Aq**, and **disodic carbonate**—*carbonate of soda, washing soda, sal soda, soda crystals, sodii carbonas, U. S. P.*—Na_2CO_3 or $Na_2CO_3 + 10$**Aq** — are amorphous powders or crystalline solids, soluble in water, forming solutions

[1] See also *ante*, p. 160; and Fresenius: "Qual. Anal.," 16te Aufl., 1895, 263, 484, 507, 508.

which are strongly alkaline, although less so than those of the hydroxids.

Sodium peroxid—Na_2O_2—is a yellowish powder, recently introduced in chemical technology, and possessed of even more energetic powers than the hydroxid. Its violent reaction with water will probably prevent its becoming of practical toxicological interest.

The monometallic alkaline carbonates — **monosodic carbonate** — *bicarbonate of soda, cooking soda, saleratus, sodii bicarbonas, U. S. P.*—$NaHCO_3$—and **monopotassic carbonate**—*bicarbonate of potash, sal aeratus, potassii bicarbonas, U. S. P.*—$KHCO_3$—exert but little corrosive action, owing to their slight alkalinity.

Calcium monoxid — *quicklime, calx, U. S. P.*—CaO—would, were it swallowed dry, produce violent symptoms, not only from its water-absorbing and caustic action, but also by the heat which it develops on contact with moisture. There is but slight probability of its proving of toxicological interest in this manner, and if brought in contact with water it becomes "slaked," and converted into the comparatively harmless *calcium hydroxid*, CaH_2O_2. The corresponding barium compounds are of more interest as true poisons than as corrosives.

Statistics—Causation.

Corrosion by the fixed mineral alkalies is of much less frequent occurrence than by the mineral acids. We have found record of 81 cases, of which 9 were by impure sodium hydroxid, 3 by disodic carbonate, 1 by monosodic carbonate, 21 by potassium hydroxid, 12 by monopotassic carbonate, 2 by a mixture of potassium hydroxid and carbonate, and 33 by "lye," etc. Twenty-four of the cases were in young children, 55 in persons over ten years of age, and in 2 both adults and children were affected. In 41 cases the alkali was taken inadvertently by children or by adults in mistake for beer, medicine, etc.; in 1 case by aspiration through a pipette; in 17 cases it was taken with suicidal intent; and in only 6 instances was there allegation of homicide. Suicide by alkalies appears to be quite prevalent in Vienna.[1]

[1] See reports of Viennese hospitals, and Hofmann: "Lehrb. d. ger. Med.," 5te Aufl., Wien, 1891, 655.

Of the six homicides two were Austrian cases in which women destroyed their children and themselves.[1] Two occurred in Germany: In one, reported by Casper,[2] a woman attempted to murder her three-year-old illegitimate child. In the other, reported by Liman,[3] the victim was also a child (five months old), which died in eighteen days. The other two cases are referred to by Taylor,[4] and appear to have been the result of negligence rather than of intentional poisoning.

Symptoms—Duration—Diagnosis—Prognosis.

When the alkali comes in contact with the lips or mouth the patient experiences an intense soapy taste and sensation, immediately followed or accompanied by violent burning pain. Even if the lye be immediately expelled, as is frequently the case, even with suicides, the short contact has been sufficient to cause corrosion of the parts touched, which are colored at first white, afterward becoming red or dark brown, are more or less eroded, and subsequently become greatly swollen. The symptoms referable to the local action upon the parts touched by the alkali follow, as in corrosion by the mineral acids, with the following differences: The vomited matters are alkaline in place of being strongly acid, are at first thick and slimy, and only become tinged with blood or dark in color later. The vomited matters contain shreds of necrosed tissue from the œsophagus and stomach. In one case, a week after an attempt at suicide, a portion of a cast of the œsophagus was found protruding from the mouth. Having been carefully withdrawn it was found to consist of the mucous, submucous, and part of the muscular coats of the entire œsophagus, including the lowest part of the pharynx and a part of the necrosed epiglottis.[5] Whether the alkali taken was a hydroxid or a carbonate, the vomit, if alkaline, effervesces on addition of an acid. This is partly due to the carbonate which almost invariably exists in the hydroxid, and partly to that formed by the carbon dioxid of the expired air. The urine rapidly becomes strongly alkaline,

[1] Hofmann: "Lehrb. d. ger. Med.," 5te Aufl., Wien, 1891, 655.
[2] "Handb. d. ger. Med.," 3te Aufl., ii., 475.
[3] *Ibid.*, 8te Aufl., ii., 530.
[4] "Med. Jur.," 11th Am. ed., 109, and (Reg. *v.* Boothman, 1867) "Princ. and Pract. Med. Jur.," 3d ed., i., 234.
[5] Hadden: Tr. Path. Soc., London, 1889–90, xli., 86.

and may continue to be alkaline or subacid for several days. It is cloudy, and deposits amorphous phosphates and crystals of ammonio-magnesian phosphate. In some cases the urine is retained, causing painful distention of the bladder.

The symptoms of perforation, shock, and peritonitis, so commonly observed as effects of the primary action of the mineral acids, have not, so far as we know, been caused by the mineral alkalies. Indeed, death is rarely a primary result of the fixed alkalies. In only twenty-two fatal cases out of fifty in which the duration has been reported did death occur in less than one month; in fifteen in a week or less; and in ten within forty-eight hours. The shortest alleged duration is in a mere mention by Taylor[1] of the case of a boy who is said to have died in three hours. Excluding this case as insufficiently reported, the most rapidly fatal case on record is Dewar's case, quoted by Christison,[2] of a boy who died in twelve hours after taking three ounces of a strong solution of potassium carbonate. "Here," it is said, "death was owing to the general system or some vital organ being affected through sympathy by the injury sustained by the alimentary canal." In a suicidal case reported by Liman[3] a woman died in thirteen hours, but, although the alkali swallowed was the cause of death, the fatal termination was probably accelerated by severe injuries to the head, not, however, involving fracture of the skull, which she had inflicted upon herself. The most rapidly fatal uncomplicated case reported in the more modern literature is that of a boy of four years who died in collapse in fifteen hours. The stomach, although seriously attacked, was not perforated.[4] A somewhat similar case, in which a girl of nineteen years died suddenly in collapse in eighteen hours, is reported by Kiemann, but in this instance the autopsy showed that the alkali had entered the lungs by inspiration.[5] In two rapidly fatal cases death was due to suffocation caused by the action of the alkali upon the upper air passages. In one of these a child of two years died in twenty-

[1] Taylor does not give this as his own observation, and quotes it without reference. We find no mention of it in Christison, or elsewhere anterior to Taylor.

[2] Edinb. M. and S. Jour., 1828, xxx., 309. Christison: "Poisons," Am. ed., 185.

[3] Casper-Liman: "Handb. d. ger. Med.," 8te Aufl., ii., 532.

[4] Lesser: "Atlas d. ger. Med.," i., 9. See also Post-mortem Appearances.

[5] Ber. . . . Rudolph-Stift., Wien, 1881, 305.

two hours;[1] in the other a child of three years died in twenty-four hours.[2] Another child of three years died in twenty-four hours without coma or convulsions and only slight difficulty in breathing, with some moist rattles in the throat, the sensorium remaining clear to the end.[3] Still another child of two and a half years died in about twenty-four hours, apparently in collapse, and the appearances observed at the autopsy were very similar to those described by Lesser.[4] Liman[5] mentions two cases of deaths of young children in twenty and twenty-four hours respectively.

Although we find no record of perforation as a primary effect of the caustic alkalies, late deaths from perforation and peritonitis have been reported. The perforation is either spontaneous—as in the case of a man of forty-nine years who died over two months after an attempt at suicide with caustic lye[6]—or it may be produced by attempts to dilate an œsophageal stricture by means of bougies, as in the case of a child of two years which died in thirty-six days[7]; and in the case of a girl of twenty years whose death—eight months after she had taken two spoonfuls of caustic potash solution—was caused by pulmonary disorder which had resulted from the passage of a bougie through the œsophageal walls.[8]

In the majority of fatal cases death is the result of inanition, caused by cardiac or œsophageal, less frequently pyloric, stenosis, or after unsuccessful attempts at surgical relief of such conditions. The final result from this cause may be delayed for a long period. Barham[9] has reported the case of a woman of twenty-two years who died from inanition two years and three months after swallowing a quantity of soap-lees by mistake. Weinlechner[10] describes the case of a boy of seventeen years who died in thirty hours after an operation for gastrostomy, performed to relieve œsophageal stricture, which was caused

[1] Nager: Arch. d. Heilk., 1872, xiii., 221.

[2] Cox: Lancet, London, n. s., xvi., 660. Beck: "Med. Jur.," 7th ed., 813.

[3] Brannock: Nashville J. M. and S., 1857, xii., 361.

[4] Voss: "Ueber Laugenvergiftung," Diss., Berlin, 1892, p. 26.

[5] Casper-Liman: "Handb. d. ger. Med.," 8te Aufl., ii., 529, 532.

[6] Ber. . . . Rudolph-Stift., Wien, (1875), 1876. 373.

[7] Schuberg: "Ein Fall von Laugenvergiftung," Diss., München, 1888.

[8] Weinlechner: *Loc. cit.*, 1879, 398.

[9] Lancet, 1850, i., 275.

[10] Ber. . . . Rudolph-Stift., Wien, 1879, 396.

by "*laugenessenz*" with which he had attempted suicide two and a half years previously. Sir Charles Bell[1] describes the stenosed œsophagus of a woman who died literally from starvation, having taken soap-lees twenty years previously.

That œsophageal stricture may result from the action of a very small quantity of alkali is evidenced by a case reported by Brug.[2] A man drank from a bottle of caustic potash by mistake, but discovered his error in time to prevent much of the liquid passing into the œsophagus, and rinsed out his mouth with water. He did not consult a physician at the time, but two months later noticed a difficulty in swallowing. Examination showed a stricture near the cardia, which was dilated, and the patient taught to pass the sound himself. He was readmitted later, the stricture having narrowed until permeable only to the smallest catheter with great difficulty, while above the stenosis was a sac-like dilatation of the œsophagus. The third day after readmission the stenosis became impassable. He died twenty-four hours after an operation for gastrostomy.

The physiological action of the potassium compounds differs notably from that of the sodium compounds, in that, while the neutral salts of latter metal are practically inert unless taken in enormous quantity, those of the former exert a distinctly toxic action upon the heart, causing at first increase and subsequently diminution of blood pressure, and diminution and even arrest of the heart's action. This is, however, of little interest when the caustic alkalies are taken, as the pulse and heart's action are similarly affected in the exhaustion and collapse caused by either alkali.

Among exceptional cases we may note the following: A woman of forty-five years died on the third day after an application of monosodic carbonate to a wounded surface.[3] A man of fifty-three years died suddenly while upon a journey. The remote cause of death was in the condition of his alimentary canal and general nutrition, caused by his having taken two ounces of sodium bicarbonate daily for a period of sixteen years.[4]

The **prognosis** is far from favorable. In 56 out of 77 cases

[1] "Surgical Observations," 1816, Pt. i., p. 82.

[2] Bost. M. and S. Jour., 1876, xcv., 481.

[3] Hancock: Lancet, 1854, ii., 435.

[4] Tunstall: Med. Times, London, 1850, 564. See Post-mortem.

(72.7 per cent.) death is known to have resulted, and in 21 (27.3 per cent.) the patients did not die while under observation.

Treatment.

As in acidism, the indications are to dilute and neutralize the corrosive as expeditiously as possible. All that has been said regarding the treatment of acid intoxication[1] applies here also, save that the neutralizing agent must be an acid and not a base. The most available acids for the purpose are acetic (vinegar) and citric (lemon juice). Albuminoid substances and oils may be subsequently given, more, however, as mechanical antidotes than with the hope of neutralizing the alkali by formation of albuminate or of soap.

Post-Mortem Appearances.

We find but few observations of the post-mortem appearances after early death from fixed alkalies. The most complete are those of Lesser,[2] Voss,[3] and Nager,[4] from the first mentioned of which the following description is taken:

Stains of a dirty-yellow color upon the skin of the chin[5]; outer portion of lips cyanotic and deprived of epithelium; the visible mucous membrane of a dirty red-brownish to greenish color; tongue swollen; epithelium where present opaque, whitish, or greenish-gray; where denuded, subepithelial tissue uniformly intensely red. Gums and pharynx injected and swollen, most markedly so at the opening of the larynx, which is markedly narrowed, and where there is considerable submucous œdema. Brown, linear, opaque stains on anterior surface of the uvula and on the soft palate. Œsophagus corroded in its upper part; lower part normal in consistence. Stomach of medium size; peritoneal surface pale; only the large vessels of the subserous tissue moderately injected with dark fluid blood. It contains about 80 c.c. (℥ iij.) of cloudy, thick fluid tinged

[1] See p. 198.

[2] "Atlas d. ger. Med.," i., 9, 13, Pl. ii., Fig. 1, 5 (good colored plates).

[3] "Ueber Laugenvergiftung," Diss., Berlin, 1892, p. 26.

[4] Arch. d. Heilk., 1872, xiii., 221. See also Casper-Liman: "Handb. d. ger. Med.," 8te Aufl., 529, 532.

[5] In one of Liman's cases yellow stains similar in character were observed in a child that had survived for eighteen days.

with bile, and *acid*[5] in odor and in reaction. On the inner surface the cardia, the lesser curvature in its left two-thirds, and the neighboring parts of the anterior and posterior surfaces are in marked contrast with the remainder. They form an irregular triangle, whose apex is directed toward the pylorus, about 10 × 12 cm. (4 × 4¾ inches), with sharply defined borders, brown-red in color, uneven, and nodulated and leathery in consistence. The remaining mucous membrane is catarrhal, flat, and pale reddish-yellow or greenish, with a few small spots, altered as in the principal lesion. A section through the eschar shows that it penetrates the entire mucous layer, but the submucous and part of the muscular layers only in the immediate vicinity of the cardia. In other parts of the stomach wall adjacent to the eschar there is only a considerable œdematous and cellular infiltration. The section of the eschar is thicker than normal. The entire small intestine is catarrhal, less so toward the ileo-cæcal valve, and in its upper part also colored by biliary imbibition. The abdominal glands are normal but pale. Heart normal; the blood contained in it partly coagulated. The mucous membrane of the larynx, trachea, and bronchi bright-red; in the bronchi a notable quantity of viscid, purulent mucus. In the posterior part of the lower lobes of the lungs are numerous, circumscribed, brown-red, pneumonic spots, of the size of peas, caused by inhalation of ingesta.

The appearances in Voss' case were similar, but the stomach contained coagulated blood, and its contents were neutral in reaction.

In Kiemann's case of death in eighteen hours, the mucous membrane of mouth, pharynx, larynx, trachea, large bronchi, œsophagus, stomach, and duodenum was brown-yellow and corroded. There was much blood in the small intestine. In places in the lungs there was gray-red hepatization, due to inspiration of caustic potash.

In Nager's case of death in twenty-two hours the stomach was perfectly normal, the fauces were ulcerated, the follicular gland tissue protruding in the form of yellow ridges. The intestines were normal. The lungs were affected with acute œdema, and to the right below with lobular pneumonia. The cortex of the kidneys was hyperæmic, black-red; the pyramids

[5] See p. 272

anæmic, pale-yellow. Nager gives a description of the microscopic changes, with illustrations, for which we refer to the original.

Autopsies after death from the secondary effects of alkalies have been made much more frequently. The most marked changes are found in the œsophagus, stomach, and lungs. If death have resulted from inanition, great emaciation and other evidences of starvation are observed. The œsophagus is affected in most cases, being deprived of epithelium, thickened, and cicatrized, and thereby diminished in calibre, most frequently toward the middle or lower part, frequently to the extent of complete or almost complete closure. Sometimes it is greatly dilated above the stenosed portion.[1] Or the œsophageal wall may be softened in places, as in one of Weinlechner's cases,[2] in which, two months after the injury, the lower third of the œsophagus was found inflamed, purulent, of a punk-like consistence, and distended above the cardia to a pocket, the wall of which had been perforated by the sound, causing purulent peritonitis. The stomach is frequently contracted, pale, buff color, more or less deprived of epithelium, and cicatrized, with thickened walls. The pylorus is often stenosed by cicatrization, but less frequently than the œsophagus or cardia. When it is affected the cardia or œsophagus is also contracted, and the stomach more or less cicatrized. In one case, however, the cardiac opening of the stomach hardly admitted a director, while at the pylorus there was an extensive cicatrix, involving the valve, and completely shutting off the duodenum. Yet the stomach intervening between the pylorus and cardia was healthy.[3]

Evidences of pneumonia or of tuberculosis or of œdema are frequently found in the lungs. In one case the fatal termination was caused by secondary empyema.[4]

In Tunstall's case of death from long-continued doses of the bicarbonate, the tissues were very pale, the stomach twice its natural size, its peritoneal surface covered with reticulated blood-vessels, with a bright brick-red blush spreading from the

[1] Barham: Lancet, 1850, i., 275. Lesser: "Atl. d. ger. Med.," Pl. v., Fig. 3.

[2] Ber. . . . Rudolph-Stift., Wien, 1875 (1876), 373.

[3] Barclay: Med. Times and Gaz., 1853, vii., 553.

[4] Shuberg: "Ein Fall von Laugenvergiftung," Diss., München, 1888.

cardia over one-third of its surface; the entire mucous membrane was disintegrated, pulpy, with numerous ulcers from the size of a pea to that of a bean. The stomach had the appearance of polished tortoise shell. It contained half a pint of strongly acid liquid.[1] The small intestines were in the same condition as the stomach. There was no fat in the omentum, and the liver and spleen were small and consolidated.

Analytical.

Very little is to be expected of the chemist in cases of alkaline intoxication. Although the identification and quantitative determination of potassium and sodium present no difficulties, salts of these metals are normal constituents in quite notable quantity of animal tissues and fluids. Hence to argue the presence of the hydroxid or carbonate it must be detected in its own form. This, so far as we are aware, has never been accomplished in portions of the cadaver, nor is it probable that it will be, as during the survival of the victim it will be expelled by vomiting or converted into other forms of combination. Indeed, we know of no instance in which the stomach contents have been found to be alkaline.[2] It is not impossible, however, that under exceptional circumstances the stomach may be found to contain free alkali, and it is to be regretted that Liman, in his description of the exceptional instance of death in thirteen hours,[3] does not state what the reaction of the 500 c.c. of liquid found in the stomach was. If the matters first vomited have been preserved, it may be possible to detect and determine the amount of alkali in them, but it must not be forgotten that a considerable quantity of alkali may be neutralized by the acid gastric contents. The materials should be dried and extracted with warm absolute alcohol, which dissolves the hydroxids but not the carbonates. A quantitative analysis of all the bases and acids present is also desirable. Usually the function of the chemist will be limited to a determination of the nature and quantitative composition of a sample of the substance which caused the intoxication. The urine should be carefully ana-

[1] He had taken several doses of sodium bicarbonate during the day, and about a pound was found in his pockets.

[2] See p. 270 and above.

[3] Casper-Liman: "Handb. d. ger. Med.," 8te Aufl., ii., 532.

lyzed, particularly as to the degree of its alkalinity or acidity. If stains upon clothing, etc., have been produced by the alkali, they will be white, and will contain the carbonate whether the substance taken was carbonate or hydroxid. Confirmatory evidence may be obtained from the presence or absence of arsenic in small quantity in the cadaver, as it is an almost constant impurity of commercial caustic soda.[1]

AMMONIUM HYDROXID AND CARBONATE.

Ammonia—NH_3—is a colorless, pungent, intensely suffocating and irritating gas, lighter than air (specific gravity 0.589 A), which is formed during decomposition of organic nitrogenous matter either spontaneously or by destructive distillation. It is most readily obtained by decomposing the ordinary aqua ammoniæ by heat, or an ammonium compound by a stronger base. The gas is extremely soluble in water (1,050 volumes in 1 at 0°), to which it communicates an intensely alkaline reaction. When subjected to a pressure of about seven atmospheres it is reduced to a colorless liquid, which boils at −33°.7 (−28°.6 F.). In many forms of ice machines this liquid is produced along with a certain amount of frozen water by pressure, and the lowering of temperature is attained by its volatilization.

Ammonia is not only irrespirable, but causes immediate suffocation when it is inhaled, even if largely diluted with air. Death from inhalation of ammonia has occurred either from the injudicious use of aqua ammoniæ or ammonium carbonate as a remedy, or by its escape from ice machines. Falck[2] refers to two cases of ammonia intoxication by the careless administration of the fumes of aqua ammoniæ to epileptics,[3] one caused by a similar use of the same substance as an antidote for hydrocyanic acid poisoning, one from the breaking of a bottle containing strong aqua ammoniæ, and one in which a work-

[1] See p. 226. In one of Liman's cases ("Handb.," ii., 532) the presence of arsenic in the liquid which caused the intoxication, and the existence of a large quantity of sodium compounds in the "contents of the cadaver" are noticed, but nothing is said of the presence of arsenic in the body, nor is its significance there recognized.

[2] "Lehrb. d. Tox.," Stuttg., 1880, p. 107.

[3] In one of these, the earliest recorded case of ammoniacal intoxication in the human subject (Gaz. de santé, May 21st, 1816), about two drachms of the liquid ran into the mouth and nose.

man employed about a Carré ice machine almost perished. An accident, fatal to three men, is reported by Fairbrother.[1] While some workmen were employed in the construction of a freezing-machine the floor gave way. A tank containing anhydrous ammonia was broken open, and four men were exposed to the fumes during three minutes. The body of one was drenched with the liquid, the exposed portions of the skin were severely blistered, and the tongue and pharynx denuded of epithelium. He died comatose in fifteen minutes. A second was found in a condition resembling chloroform delirium, and remained in the same state for two hours, when he suddenly expired. The third was conscious and able to walk, but complained of occasional difficulty in breathing. He swallowed and conversed readily. In about five hours the dyspnœa suddenly increased, and he died after a few gasps. The fourth man sustained a compound fracture of the leg, which was amputated. For three months he suffered from bronchial irritation and constant hæmoptysis, but recovered except for partial paralysis of the right side.[2]

We find mention in medical literature of 53 cases of intoxication caused by swallowing **aqua ammoniæ**—*spirits of hartshorn*, *volatile alkali*—**ammonium hydroxid solution**—**NH_4HO**—or **ammonium carbonate**—or some preparation containing one or the other, such as "Household ammonia," ammonia liniment, aromatic spirits of ammonia, eau sedative, Preston salts. Of the 50 cases in which the causation is given, 29 were accidental, 18 suicidal, and 3 homicidal.

It is difficult to imagine how a substance possessed of so pungent an odor could be taken accidentally and unknowingly by a person in the full possession of his faculties, yet in only 3 of the "accidental" cases were the victims young children. Six of the 25 accidental (?) cases in adults were caused by druggists' errors, and 12 by errors of the patients themselves or their attendants in taking or administering medicine; in 3 cases aqua ammoniæ was taken in mistake for spirits, in 1 for water in the dark, in 1 instance while the man was drunk, and in 2 cases "by mistake."

[1] St. Louis M. and S. Jour., 1887, lii., 272.

[2] Another case of poisoning by ammonia vapor is reported by Harris: Ind. M. Jour., Calcutta, 1885, iv., 673, to which we have not had access.

Of the three homicidal (?) cases, one is referred to by Husemann[1] as reported by Souchard, of a girl of six years who intentionally poured aqua ammoniæ into the mouth of her younger sister. The other two are mentioned by Taylor: One, the death of an infant of four and a half days from the effects of an ammoniacal embrocation mixed with its food, as it would seem either by the mother or by a two-year-old child[2]; the other (Reg. *v.* Haydon, 1845) in which a man was tried for the murder of a child by administering to it spirits of hartshorn.[3]

One case is reported of the malicious external application of ammonia. A liniment containing a strong solution of ammonia was thrown by a man into a woman's face, with intent to injure her. A portion reached the eyes. She recovered.[4]

Prognosis.—The prognosis when aqua ammoniæ has been swallowed appears to be more favorable than when the fixed alkalies or mineral acids are similarly taken. Of 47 cases, 27 (57.4 per cent.) died, and 20 (42.6 per cent.) recovered. Nor is death from secondary effects to be anticipated. It has only occurred twice among the cases of which we find record.[5]

Duration.—The lethal action of volatile alkali is as a rule more rapid than that of the fixed alkalies. Of 23 cases in which the duration is given, 13 died within thirty-six hours. The most rapidly fatal case is one reported by Stevenson,[6] in which a man, a hard drinker, took a teaspoonful of liquor ammoniæ of specific gravity 0.88. After complaining of some pain in the abdomen, he turned over on his side, turned blue in the face, and expired at once without any struggle for breath. A man of sixty-eight years, having defective sense of smell, swallowed 60 gm. (about ℥ ij.) of aqua ammoniæ in two doses, and died of suffocation three and one-quarter hours later.[7] A man of seventy years took two mouthfuls of aqua ammoniæ while drunk, and died in convulsions in four hours.[8]

The most prolonged case in which death resulted from the

[1] "Toxikologie," i., 948.

[2] Guy's Hosp. Rept., 1864, 3 s., x., 190, "Poisons," 3d Am. ed., 255.

[3] In the table of criminal poisonings in France, 1851-63, given by Tardieu (p. 164) are included four by ammonia.

[4] Reg. *v.* Gavan, 1873, Stevenson, Taylor: "Med. Jur.," 11th Am. ed., 111.

[5] Barclay: Med. Times and Gaz., 1853, ii., 554. Mann: "Forensic Med.," p. 421.

[6] Guy's Hosp. Repts., 1871, 3 s., xvii., 225.

[7] Christen: J. d. chim. méd., etc., 1869, 5 s., v., 309.

[8] Kern: Med. Corr.-Bl., Würt. Aerztl. Ver., 1868, xxxviii., 304.

primary action of the alkali of which we find record is that of a man of forty years who died of laryngismus stridulus in nineteen days after swallowing one ounce of liquor ammoniae.[1] A man died in ten days after taking 100 gm. (℥ iij.) of liquor ammoniae[2]; another in a like period from the effects of an equal quantity[3]; another in eight days from the effects of 250 gm. (℥ viij.) of eau sedative[4]; and a woman expired eight days after taking 90 gm.[5] In the single case of death from the secondary effects of volatile alkali above referred to, death occurred in three months.

Symptoms.—Immediately on swallowing the alkali the person experiences a sensation as if he were being mechanically suffocated, he gasps for breath, sometimes utters a low cry, and sometimes falls back insensible. Burning pain in the lips, mouth, and fauces practically accompanies the act of swallowing or is complained of immediately the patient regains consciousness. The pain soon extends to the stomach, and later the abdomen becomes distended, tympanitic, and tender to the touch. Retching and vomiting also soon follow the ingestion, the vomited matters having the odor of ammonia, and containing blood, frequently in large amount. The lips, tongue, and mucous membrane of the mouth and pharynx are white or bright red, with the epithelium peeling off in shreds, and become greatly swollen and œdematous. The œdema may involve the glottis, and thus cause death suddenly.[6] Respiration is interfered with; deglutition is difficult or impossible; there are severe thirst and dryness of the mouth and pharynx, and the voice is husky, reduced to a whisper, or extinguished. The breath smells of ammonia. The lachrymal and salivary secretions are greatly augmented, the saliva is frequently streaked with blood, and bloody or purulent sputa are discharged from the air passages. The urine is scanty, high colored or dark, subacid or alkaline, and contains albumin, casts, and blood cor-

[1] Patterson: Edinb. M. J., 1857, iii., 236.
[2] Potain: Union méd., 1862, xiii., 119.
[3] Gaz. d. Hôp., 1862, xxxv., 39.
[4] Rulie: Union méd., 1857, viii., 522.
[5] François: Ann. d'hyg., etc., 1877, 2 s., xlvii., 556.
[6] Tracheotomy has been performed for the relief of this condition three times, once successfully (Dickinson: Lancet, 1890, ii., 1 214), and twice unsuccessfully (Hancock: Med. Chron., Manchester, 1891, xiv., 107. Benn: Pac. M. and S. J., 1867–68, n. s., i., 13).

puscles. The bowels in some cases remain constipated during the entire attack; in others there are copious evacuations, containing blood or consisting entirely of blood and clots. Death most frequently occurs suddenly from suffocation. Others have died from exhaustion, comatose, or in convulsions. In non-fatal cases aphonia may continue for weeks or months.

Treatment.—The indications for neutralization are the same as when the fixed alkalies have been taken. Impending death from suffocation may be averted by tracheotomy or artificial respiration; anodynes for the pain; ice pills.

Post-Mortem Appearances.—The mucous membrane of the mouth and pharynx is deprived of epithelium, red, glazed, much inflamed, or in parts softened or destroyed. The œsophageal mucous membrane has been found detached in shreds at its upper part or intensely inflamed at its lower part. In one instance, in which death occurred in thirty-six hours, the œsophageal mucous membrane was yellow, thickened, partly destroyed, with almost complete stenosis in some places.[1] The stomach contains dark, altered blood, which has the odor of ammonia, and may also be distended with gas. In a woman who died the same day from the effects of 10 c.c. (℥ iij.) of ten per cent. aqua ammoniæ, the stomach was found to contain 500 c.c. (about a pint) of tenacious, mucous, acid fluid.[2] Its mucous membrane is thinned or destroyed, particularly at the points where the ammonia would first come in contact. The erosion has been observed to extend into the muscular coat, but we find no case in which it has proceeded to perforation. The remainder of the gastric mucous surface is intensely injected, thickened, and reddened, or white and covered with mucus. The duodenum and jejunum have been found intensely injected or filled with blood, but in other instances the intestines were normal. The epiglottis is eroded and swollen. The submucous tissue of the glottis and larynx are highly œdematous, sometimes to almost complete closure of the opening of the glottis. The œdema has been found to cease at the vocal cords, the trachea and lungs being normal, but usually the trachea and lungs are also œdematous or congested. The lungs have even

[1] Gillam: Med. Times and Gaz., 1878, ii., 706.

[2] Kanders: Wien. med. Bl., 1881, iv., 513.

been found affected with pneumonic consolidation.[1] The blood is fluid and bright red, or if dark becomes light on exposure to air. The kidneys in some cases are unaffected, in others congested and affected with glomerulo-nephritis and fatty degeneration. The liver is usually healthy, but may be the seat of fatty degeneration.

In Barclay's case of secondary death after three months[2] the œsophagus was healthy, the cardiac orifice of the stomach contracted, and the pyloric opening reduced to the size of a crow quill.

Analytical.—The detection of an alkaline ammoniacal compound in the stomach contents is only possible or significant if the case has been of short duration, and if the examination be made soon after death. Ammonia and ammonium hydroxid and carbonate are very rapidly diffusible, and the two former gaseous or extremely volatile; hence they are not only rapidly absorbed, but soon escape from the living or dead body. Moreover, ammonium compounds are among the products of putrefaction. Under favorable conditions, however, the stomach contents may have a marked odor of ammonia, be alkaline in reaction, and yield ammonia on analysis.[3] Probably the best method of separation is that proposed by Vitali[4]: The viscera are treated with absolute alcohol, ether is added, and the mixture distilled at a temperature not exceeding 40° (104° F.). Free ammonia (or that present as hydroxid) passes over with the ether, but ammoniacal salts are not decomposed at that temperature. The ammonia is extracted from the ether by agitation with dilute sulfuric acid. If a decinormal acid be used the quantity of ammonia may be determined from the loss of acidity of the acid solution. To extract ammonium carbonate the viscera are mixed with alcohol and distilled, and the carbonate in the distillate precipitated with lime water or barium chlorid.

Ammonia may be recovered from the solution of the sulfate obtained as above by evaporation to dryness, washing with absolute alcohol, dissolving in water, and distilling the solution

[1] Dyson: Med. Times and Gaz., 1878, i., 35.

[2] Med. Times and Gaz., 1853, ii., 554.

[3] We know, however, of no instance in which this has been actually found.

[4] "Manuale d. Chim. toss.," Milano, 1893, p. 165.

with caustic soda in a small retort connected with a bulb tube charged with water and cooled by ice. The presence of ammonia in the distillate is recognized by : (1) The odor; (2) the formation of white fumes when a glass rod moistened with moderately concentrated hydrochloric acid is approached to it; (3) freshly made hæmatoxylin paper is colored blue-violet by the vapor; (4) phenolphthalein paper is colored red; (5) rosolic acid paper is colored purple; (6) Nessler's reagent colors the liquid yellow or brown; (7) a solution of mercuric chlorid forms a white precipitate; (8) a solution of mercurous nitrate forms a black precipitate; (9) neutralized with slight excess of hydrochloric acid, the solution treated with excess of platinic chlorid solution, evaporated to dryness and extracted with alcohol-ether, leaves a yellow residue consisting of microscopic octahedral crystals of ammonium-platinic chlorid—NH_4Cl,-$PtCl_4$. The formation of this compound may be utilized to determine the quantity of ammonia. To that end it is dried at about 100° and weighed; 100 parts correspond to 7.61 parts of ammonia (NH_3).

If caustic alkalies be present in the objects examined along with ammoniacal salts, the latter are decomposed at slightly elevated temperatures, and ammonia is liberated.

The detection of ammonia (as carbonate) in the urine is of no toxicological significance, as the urine becomes ammoniacal from decomposition of urea, always on exposure to air, and frequently in the bladder.

THE HALOGENS.

Chlorin—Bromin—Iodin.

The halogens (ἁλς = salt, γενεά = birth) are the four elements fluorin, chlorin, bromin, and iodin, which exist in compounds more or less closely resembling sea salt, sodium chlorid. Fluorin, although probably a most intense corrosive, has no practical toxicological interest, owing to the exceptional conditions necessary for its liberation.

Chlorin.

Chlorin is liberated by the action of manganese dioxid, MnO_2, upon hydrochloric acid, either alone or in the presence of sulfuric acid, or upon a mixture of sulfuric acid and common salt; or electrolytically by the decomposition of a chlorid. It is also given off when a hypo-

chlorite (bleaching powder, etc.) is exposed to the air or decomposed by an acid.

It is a yellowish-green gas; has a very penetrating, pungent odor; is very soluble in water; and is reduced to a liquid under a pressure of eight and a half atmospheres at 12° (53.6° F.). Liquid chlorin, in steel cylinders, is now an article of commerce. Its chief use is as a bleaching and disinfecting agent.

We find reference in medical literature to seven cases of accidental acute poisoning by inhalation of chlorin among operatives in factories where the gas is used, or from generation of the gas from chloride of lime.[1] A quasi criminal, fatal poisoning by chlorin occurred in New York State in 1894, in which an aged negress lost her life through the pranks of some college students.

Probably the most accurate description of the symptoms is that given by Sury-Bienz: The man had inhaled a small quantity of the pure gas. He immediately experienced an inclination to cough, dyspnœa, and a stabbing pain in the chest. He was seen by the physician the morning after the accident (which had occurred during the night) suffering from severe dyspnœa and coughing constantly. In the evening he was worse, and was transferred to the hospital. The next morning the dyspnœa was intense, and coughing, with little expectoration, was almost uninterrupted. The patient was greatly excited, and spoke with great difficulty. The heart impulse was not detectable, but the heart sounds were clear. Sibilant râles were heard, particularly to the right and above. The liver dulness was normal. The urine was brown-yellow, clear, and without albumin. The face was cyanosed. The sputum was frothy and bloodless. There was nothing abnormal in the pharynx, and the voice was clear and loud. Towards evening the dyspnœa increased markedly; every respiratory muscle was called into play; the movements of respiration were short, particularly the expiration. There was slight cyanosis and the pulse was very frequent and small. At 10 P.M. the patient slept quietly. At 10:30 he awoke and threw himself about; the pulse became imperceptible; there were râles in the throat; and he died at 11 P.M., about forty-eight hours after the inhalation.

The prominent post-mortem appearances noted in the above case were: rather intense inflammation of the mucous membrane of the upper as well as the more minute air passages; marked œdema of the lungs; slight catarrh of the right kidney; severe, acute catarrh of the stomach and of part of the duodenum. There were also found (of an-

[1] Kostner: Arch. d. Apoth. Ver., 1826, xviii., 101. A. B.: Lancet, 1839, ii., 194. Meissner: Ztschr. f. Med. Chir. u. Gebh., 1862, n. F. i., 347. Cameron: Dublin Q. J. M. Sc., 1870, xlix., 116. Baylon: Bull. Soc. méd. Suisse Rom., 1876, x., 177. Med. Pr. and Circ., Dublin, 1880, n. s., xxix., 245. Sury-Bienz: Vrtljschr. f. ger. Med., etc., 1888, n. F., xlix., 345.

terior origin) marked emphysema, enlargement of the heart, with some thickening of the valves of the right side, complete destruction of the right kidney, and chronic catarrh of the bladder. The death was ascribed to cardiac paralysis.

In Cameron's case, on cutting into the ventricles of the brain, a very strong, unmistakable odor of chlorin was observed.

HYPOCHLORITES.—Mixtures and solutions containing hypochlorites are extensively used in bleaching and laundrying. Those most frequently met with are: BLEACHING POWDER=*chloride of lime*, a white, rather moist powder, made by treating slaked lime with chlorin, and containing calcium hypochlorite, $Ca(ClO)_2$, with some calcium chlorid and excess of slaked lime. LABARRAQUE'S SOLUTION = *liquor sodæ chlorinatæ*, *Liquor sodæ chloratæ*, *U. S. P.*—a faintly greenish liquid, having the odor of chlorin, made by adding a strong solution of disodic carbonate to bleaching powder suspended in water. It contains sodium hypochlorite, NaClO. Sodium hypochlorite may also be produced from the chlorid by electrolysis, if the temperature be not allowed to rise. EAU DE JAVELLE = *Javelle water*, is properly the potassium compound, corresponding to that of sodium contained in Labarraque's solution. Usually, however, the two are practically identical.

A few cases of intoxication caused by swallowing Javelle water have been reported in France,[1] one a homicide in which a child of six months was destroyed by eau de Javelle forcibly administered by its father.[2] The toxic power of these solutions is largely due to the excess of alkali which they contain, and the symptoms and lesions which they cause resemble those produced by the fixed alkalies.

A girl made an unsuccessful attempt to poison a family by chloride of lime in Kings County, N. Y., in 1888.

BROMIN.

Bromin is a dark reddish-brown liquid, volatile at all temperatures, giving off brown-red vapors, which have an odor similar to that of chlorin, and a like effect upon the air passages. It is soluble in water to the extent of 3.2 parts in one hundred at the ordinary temperature, the solution being known as "bromin water." It is more soluble in alcohol and ether. Its chemical characters are similar to those of chlorin, but in general somewhat less energetic.

We find in medical literature but three instances of bromin intoxication.[3] Two of these were suicidal: one that of a man of twenty-four

[1] Tardieu: "Empoisonnement," 2ème ed., 1875, 285–292 (cases iv., v., vi.). Barbet and Brulatour: J. d. chim. méd., etc., 1844, 2 s., x., 249. Carles: Ann. d'hyg., etc., 1876, 2 s., xlv, 550.

[2] Case vi., Tardieu.

[3] We have not seen Hering: "Ein Fall von Bromvergiftung," Ztschr. f. Med.-Beamt., Berlin, 1889, ii., 217; possibly it is the same case as that reported by Schmalfuss.

years, a daguerrotyper, who swallowed an ounce of undiluted bromin, and died in seven and one-half hours;[1] the other that of a teacher, who was found dead under circumstances which left no doubt that he had swallowed bromin.[2] The third case was either accidental or homicidal. It was that of a child of one and three-quarter years, which died in six days from the inhalation of bromin vapor. The question whether the accused, a photographer, had intentionally caused the inhalation was not determined, as he died in prison during the inquiry.[3]

In Snell's case there were immediate spasms of the pharyngeal and laryngeal muscles and difficult respiration, and soon afterwards great anxiety, restlessness, pain in the stomach, and tremors of the hands. The respiration was hurried and the pulse tense and corded. The symptoms increased in intensity; the extremities became cold, the pulse failed, and death followed. In Kornfeld's case the skin below the mouth was inflamed and yellow brown. The voice became husky an hour after the inhalation, and remained so until death. Respiration was difficult and abdominal. Three days later there were gastric pain and painful deglutition; and in the evening transitory gnashing of the teeth and slight contractions of the fingers. On the sixth day a severe attack of dyspnœa occurred and was repeated in the evening, accompanied by cardiac palpitation. During the night spasms and general convulsions were followed by death in complete unconsciousness.

In Snell's case, in which the autopsy was made seventeen hours after death, the external surface of the stomach was found vividly injected, most markedly so at the lesser curvature; near the middle a soft ecchymosed spot and several smaller ones posteriorly. The stomach contents consisted of 120 c.c. (℥ iv.) of a port-wine colored fluid, having a faint odor of bromin. The mucous surface was covered with a thick black layer, resembling coarse tanned leather, or a mixture of lampblack and gum arabic. The submucous tissue was intensely injected. The duodenum was vividly injected. Those organs nearest the stomach were stained yellow. In Schmalfuss' case, in which from ninety to one hundred hours intervened between the death and the autopsy, the odor of bromin was perceptible on opening the abdomen. The external surface of the intestines and peritoneum were coated with a brown-black, smeary mass. The posterior wall of the stomach was entirely absent, only shreds of the pylorus remained, and what was left of the stomach and contents appeared as if burnt. The contents of the small intestine were yellow or orange, and had a distinct odor of bromin. The small intestine had the appearance of having beencooked. At about a half a metre from the pylorus the intestine was perforated, and near the opening in the peritoneum was a yellowish

[1] Snell: N. Y. M. Jour., 1850, v., 179. Schapps: *Ibid.*, p. 340.

[2] Schmalfuss: Vrtljschr. f. ger. Med., etc., 1889, n. F., l., Supplh., 37.

[3] Friedr. Bl. f. ger. Med., etc., 1883, xxxiv., 228.

mass weighing about 50 gm. and smelling strongly of bromin. As the case was clearly one of suicide no analysis was ordered, but bromin was obtained from the contents of the intestine by distillation.

The analytical method which should be followed for the detection of bromin is the same as that recommended for iodin.[1] Free bromin may be recognized by (1) its odor; (2) its color and appearance; (3) the orange and yellow color of its solution in chloroform or carbon bisulfid; (4) the brown color of its vapor; (5) it colors starch paste yellow or orange; (6) with phenol it forms a crystalline precipitate of tribromophenol, insoluble in water.

Bromids give the following reactions: 1. Silver nitrate produces a faintly yellowish-white precipitate of silver bromid, which is insoluble in nitric acid, sparingly soluble in ammonium hydroxid solution, and almost insoluble in boiling solution of ammonium sesquicarbonate. 2. Solution of palladious nitrate (but not that of the chlorid) forms in concentrated solutions a red-brown precipitate of palladious bromid ($PdBr_2$). 3. If chlorin water be added to a solution of a bromid, the bromin is liberated and colors the liquid darker yellow or orange. If it be then shaken with carbon bisulfid or chloroform, these dissolve the bromin, forming reddish-yellow solutions. 4. Bromin similarly liberated by chlorin water from bromids responds to the other tests for free bromin given above.

Iodin.

Iodin is a blue-gray solid, forming crystalline scales which have something of a metallic lustre. Specific gravity 4.948. It is volatile at all temperatures, and if heated gives off a dark-violet-colored vapor, whose odor somewhat resembles that of bromin. It is but sparingly soluble in water, but water standing upon excess of iodin continues to dissolve it by formation by hydriodic acid. Its solubility in water is greatly increased by the presence of hydriodic acid or of certain salts, notably potassium iodid. It dissolves readily in alcohol or ether, forming brown solutions; or in chloroform or carbon bisulfid, forming violet-colored solutions. Its chemical characters are similar to those of chlorin and bromin, but less pronounced. Its atomic weight is 126.54.

Elementary iodin is used medicinally in solution as *Tinctura iodi*, *U. S. P.*, an alcoholic solution containing eight parts of iodin in one hundred parts; and as *Liquor iodi comp.*, *U. S. P.*, or *Lugol's solution*, an aqueous solution containing five parts of iodin and ten of potassium iodid in one hundred parts. The so-called "colorless tincture of iodin" contains no free iodin, but triethylamin, ethyl iodid, and ammonium iodid, along with alcohol and some free ammonia. Iodin in combination is also extensively used in medicine: in metallic com-

[1] See p. 286. [2] See p. 288, Note 1.

bination in the iodids of the alkalies, and of zinc, iron, and mercury, and in more recent times as iodin trichlorid and tribromid, and sulfur iodid; and in organic combination as iodoform, CHI_3, iodol (tetriodopyrrol, C_4I_4NH), iodal (monoiodaldehyde, C_2H_3IO), diiodoform tethylene periodid, carbon tetriodid, C_2I_4), iodoglycerol, iodalbuminate, and iodocasein, in all of which the therapeutic action depends largely if not entirely upon the iodin present. Iodin exists in minute quantity in the tissues of all plants and vegetables inhabiting the sea.

Acute iodin intoxication, caused by free iodin or its solutions is of comparatively rare occurrence. We have met with 27 cases in medical literature. Of these the majority, 18, were accidental; 5 were children who drank from carelessly exposed bottles of the tincture; 2 were adults who took the tincture in mistake for internal remedies; and the remaining 11 were cases of medicinal poisoning by solutions of iodin, either given internally, applied to the surface, or injected into cavities, abscesses, or tumors. Seven were attempts at suicide, 3 of which were successful. Two were unsuccessful attempts at homicide. One of these is mentioned by Taylor[1]: an attempt by a woman to poison a fellow-servant by tincture of iodin added to food in a plate. The deep blue color produced by the iodin with the farinaceous food attracted attention and frustrated the attempt. The other was an attempt by a girl to destroy her illegitimate child by administration of the tincture.[2]

The action of elementary iodin is rather that of a true poison than that of a corrosive, although it combines with albuminoid substances, and thus acts locally upon tissues with which it is brought in contact. The gastric symptoms which it produces appear to be due quite as much to its action through the blood as to its action from the stomach directly. Rose[3] found in a woman who received 10 gm. of iodin (℥ iiss.) daily, injected into an ovarian cyst in the form of Lugol's solution, that during the first days about 4 gm. (℥ i.) was daily expelled in the vomit, which contained free iodin, while at no time did the urine contain iodin, except in combination. Iodin also exerts a marked influence upon the circulation and upon the blood. Pellacani[4] has shown that iodin acts upon the blood corpuscles and enters into combination with the liberated hæmoglobin.

On contact with the skin iodin produces a brown stain, and, if the action be more intense, a sluggish dermatitis which may become purulent. Death has been caused by the systemic disturbance resulting from too extensive an application of the tincture to the skin in at least

[1] "Poisons," 3d Amer. ed., 285.

[2] Laennec: Ann. d'hyg., etc., 1883, 3 s., ix., 534. In Tardieu's table of criminal poisonings in France during 1851–71 one by tincture of iodin is included. No particulars.

[3] Arch. f. path. Anat., etc., 1866, xxxv., 12.

[4] Sulla tossicologia del iodio, Milano, 1884.

two instances: one a boy of eleven years,[1] the other a boy of seventeen years.[2]

As an example of the symptoms in a fatal case of acute poisoning by iodin we may cite the suicidal case reported by Hermann[3] of a man of thirty-eight years, who died in thirty-three hours from the effects of 60 gm. (℥ ij.) of tincture of iodin. He immediately experienced burning pain in the gullet, which extended downward and became most severe in the stomach. He vomited and had liquid stools. In two and a half hours he was admitted into the hospital: face pale, pupils moderately dilated, sensitive to light, surface cool; severe abdominal pain, not increased by pressure; mouth pale and dry; lips not cyanosed; posterior wall of pharynx dry, slightly reddened; expired air had strong odor of iodin, particularly during and after vomiting. He was perfectly conscious; no stupor, no loss of perception, no incoherence of ideas, no sensory disturbance, except buzzing in the ears and a sense of heaviness in the head. Complained only of great præcordial anxiety, which caused him to change his position constantly. Collapse and prostration, but not to the extent of fainting. Respiration somewhat accelerated. Radial artery seemed like a hard, thin cord without pulsation; carotid pulse weak, small, 120; heart's action irregular and tempestuous. In first few hours quick vomiting, preceded by retching every ten to fifteen minutes; later at longer intervals. Vomit at first dark; after administration of starch and magnesia faintly blue, rather white, thin and fluid. Vomiting provoked by drinking or by slight movement. Stools after colic and straining thin and brownish, smelling of iodin. After a sleepless night, the præcordial anxiety and derangement of respiration had increased; radial pulse very small, 132; marked collapse; speech stuttering and voice hoarse; thirst intense; deglutition difficult; epigastric pain severe, and extended to the distended abdomen, back, and œsophagus. Vomiting less frequent, but vomit contained considerable pure blood, as did the fæces. Of urine, barely three tablespoonfuls in twenty-four hours, dark red-brown, had strong odor of iodin, and resembled tincture of iodin in appearance. Patient very uneasy but conscious, and died almost without a struggle.

The presence of free iodin in the urine in this case (verified by analysis of that from the cadaver) is a condition different from that observed in Rose's case, in which the urine, while containing iodids, was free from elementary iodin.

In more prolonged cases death is the result of changes caused by the truly poisonous action, resulting in part from the destruction of gastric follicles as a result of their eliminatory action and the disturbance of the circulation. Thus in a woman who took thirty drops of tincture

[1] Culpepper: Therap. Gaz., Detroit, 1888, 3 s., iv., 225.

[2] Gillespie: Med. Times and Gaz., 1864, ii., 488.

[3] St. Petersb. med. Wchnschr., 1868, xv., 336.

of iodin three times daily (in all 60 gm. of the tincture) and who became greatly emaciated, large furuncles appeared on the breast and shoulder blades, with great inflammation in their neighborhood. Warm compresses caused separation of the furuncles as hard, nodular bodies; leaving deep, painless, but non-granulating ulcers. Soon after gangrenous inflammation began at one great toe and extended upward rapidly, whereof she died.[1]

The smallest dose taken internally which has been known to cause the death of an adult was one drachm (3.9 gm.),[2] and a child of four years died from the effects of twenty grains (1.3 gm.).[3] Probably less quantities might prove fatal.

When a solution of iodin has been swallowed, solution of sodium thiosulfate should be given and the stomach washed out with water containing albumin and starch paste, or water holding sodium thiosulfate in solution. In one instance threatened death from œdema of the glottis was averted by tracheotomy.[4]

After death from iodin, the skin and mucous membranes which have been touched by the solution may be stained brown. In Hermann's case, however, there were only orange-yellow spots on the tonsils, consisting of a detachable membrane which extended to the pharynx and œsophagus. The tracheal mucous membrane is eroded, and the submucous tissue red and œdematous, the bronchi to the finest capillaries are inflamed and filled with thick mucus; intense emphysema is present; the parenchyma of the lungs is bloodless. The œsophagus throughout is covered with orange-yellow, mucoid liquid. The stomach contains an orange-colored mucoid liquid; its mucous membrane is of the same color, finely punctate and swollen. The same appearance presents in the duodenum. The liver is large, cirrhotic, and icteric. Kidneys large; substance of parenchyma hard, hyperæmic. Bladder contracted, contains a small quantity of liquid having the color and odor of iodin.

When free iodin has been absorbed it may be detected, either free or in combination, in the contents of the stomach and intestines, in small quantity in the fæces, in all secretions, urine, perspiration, tears, saliva, etc., in pathological exudations if present, and in the expired air. The bile only contains iodin in detectable quantity when it has entered the circulation in large amount at one time.[5] Its excretion does not take place with great rapidity, probably because iodin is eliminated by the gastric mucous membrane and again absorbed from the intestine, very little being contained in the fæces. The duration of its sojourn in the

[1] Lewin: "Nebenwirkung der Arzneimittel," 2te Aufl., 1893, p. 383, which also contains an extensive account of the varied symptoms observed in toxic but non-fatal cases.

[2] Jackson: Prov. M. J., 1847, p. 356.

[3] Gairdner: "Essay on the Effects of Iodin," 1824, p. 20.

[4] Kobert: "Intoxikationen," 375.

[5] Rózsahegyi: Ph. Jahrbr., 1878, 568. Hermann: *Loc. cit.*

body has been found to be from forty-five to one hundred and forty-nine hours.[1]

Iodin has been detected and determined in several cases of poisoning in the human subject. In Hermann's case[2] (death thirty-three hours after two ounces of tincture of iodin) 91.3 gm. of stomach contents yielded 0.0182 gm. iodin, and a like quantity of blood from the liver, 0.0209 gm. The large intestine contained about as much as the stomach contents; the small intestine a decidedly smaller quantity; the urine gave a distinct iodin reaction. The quantity could not be determined in the small amount of urine from the cadaver, and that passed during life was lost. The bile contained no trace of iodin. The urine passed by a woman on the second day of poisoning by tincture of iodin was found by Huber[3] to contain 0.2781 gm. of iodin in 500 c.c., while 1,300 c.c. of vomit contained 0.013 gm. In a woman who attempted suicide with tincture of iodin, the breath had the odor of iodin in half an hour. Iodin was detected in the vomit, and in the urine of the second day, but none in the urine of the third day. There was no trace of iodin in the fæces passed on the fourth day. The quantity taken was about 1 gm. of iodin.[4] In Rose's case, already referred to, iodin was never found free in the urine, but was found in the vomit of the first few days. The urine passed by a child while suffering from the effects of an overdose of tincture of iodin was found by Stewart and Gulliver[5] to contain iodin in combination, but not free. It also contained indican, a large excess of urea, and a large quantity of mucin, but no albumin. Its reaction was strongly acid.

If present in notable amount, iodin may be directly recognized in the urine or other liquid by agitation with chloroform, or preferably, with carbon bisulfid, which is colored violet by free iodin, but not by iodids. The color is only produced by the latter if the liquid be agitated with chloroform or bisulfid after addition of yellow nitric acid or of chlorin water.

The materials are to be distilled with water, the receiver being cooled with ice. If iodin be present in sufficient amount the vapor may have a violet color, and a paper moistened with cold starch paste and exposed to the vapor will be colored blue. After distillation of the free iodin the materials are to be dried with potassium hydroxid and sodium nitrate in a silver crucible, and incinerated at as low a temperature as possible. The ash is extracted with alcohol, the solution evaporated, the residue dissolved in water, the solution acidulated strongly with sulfuric acid, and potassium dichromate or manganese dioxid added, and the mixture again distilled. Iodin which existed in metallic combination (iodids) will pass over in this distillate.

[1] Rózsahegyi: *Loc. cit.*

[2] *Loc. cit.*

[3] Ztschr. f. kl. Med., 1888, xiv., 471.

[4] Malmsten: Hygiea, Stockh., 1885, xlvii., 119.

[5] Ord: Brit. M. Jour., 1877, i., 671.

Free iodin may be recognized by: (1) its odor; (2) the violet color of its vapor; (3) the violet color of its solution in carbon bisulfid or chloroform; (4) it colors cold starch paste dark blue, the color disappearing on the application of heat and reappearing on cooling; (5) if present in sufficient amount it separates in the distillate in its characteristic solid form. If too large a quantity of chlorin has been generated in the second stage of the process above described, iodin trichlorid will be produced, which does not give the above reactions. In that event enough caustic potash is to be added to the distillate to decolorize it, the liquid evaporated, the residue ignited, and dissolved in water, and the solution tested for iodids.

The iodids are recognized by 1. Silver nitrate produces a flocculent, yellowish-white precipitate of silver iodid (AgI), insoluble in nitric acid, very sparingly soluble in ammonium hydroxid, and insoluble in a boiling solution of ammonium sesquicarbonate.[1] 2. A solution of palladious chlorid or nitrate produces a brown precipitate of palladious iodid (PdI_2), which is sparingly soluble in solutions of chlorids, but almost insoluble in cold dilute hydrochloric or nitric acid. 3. A solution of one part of cupric sulfate and one and a half parts of ferrous sulfate produces a dirty white precipitate of cuprous iodid (Cu_2I_2) in neutral, watery solutions. 4. Add to the liquid a little dilute starch paste and a few drops of dilute sulfuric acid and a little yellow nitric acid; the liberated iodin colors the liquid blue or black, the color disappearing on the application of heat. 5. Add a little sulfuric acid and some yellow nitric acid and agitate with carbon bisulfid, the latter is colored violet.

The quantity of iodin may be determined from that of the palladious iodid. This is collected on a weighed filter after standing twenty-four hours, washed in succession with water, alcohol, and ether, dried at a temperature of 80° (187° F.), and weighed. Afterward the filter is burnt in a porcelain crucible, ignited, and the remaining palladium weighed: one hundred parts of palladious iodid correspond to 68.67 parts of iodin, and one hundred parts of palladium to 259 parts of iodin. Or the iodin or iodid may be determined volumetrically by the methods of Harnack,[2] Hilger,[3] Rose,[4] or Jaworowski.[5]

[1] This solution, which dissolves silver chlorid, is prepared by dissolving one part of transparent commercial ammonium carbonate in nine parts of water at the ordinary temperature, and adding five drops of ammonium hydroxid solution of sp. gr. 0.96 to each 10 c.c. of the liquid.

[2] Ztschr. f. physiol. Chem., 1884, viii., 158; Berlin. kl. Wchnschr., 1882, xix., 788.

[3] N. Rep. of Ph., 1874, xxiii., 298; Ann. d. Ch. u. Ph., 1874, clxxi., 212.

[4] Arch. f. path. Anat., etc., 1866, xxxv., 12.

[5] Ph. Ztschr. f. Russl., 1893, 819.

MINERAL POISONS.

ANTIMONIALS.

THE native black sulfid of antimony was known to the ancients, and was used by them as a cosmetic for staining the eyebrows and eyelashes, and also in the form of ointment for external application. Its Latin name *stibium* is derived from the Greek στίμμι, or στίβι.[1] In the latter part of the sixteenth century the first description of elementary antimony and of many of its preparations, including the trioxid and oxychlorid, was published in a work attributed to Basil Valentine, in which, however, the element is constantly referred to as a substance long known.[2] Tartar emetic was discovered and first used as a medicine by Adrian van Mynsicht in 1631.

The internal administration of antimonials by physicians dates from the latter part of the sixteenth century. It was chiefly to the medical use of antimony and mercury that the acrid discussions between the iatrochemists and Galenists of that time related. The former adopted the view of Paracelsus, that a poison might be beneficially used as a medicine, while the latter insisted that a poisonous substance was such under all circumstances, and inadmissible in medical practice. In many European countries for over a century antimonials were only administered in violation of the laws.[3]

[1] Ezekiel xxiii. 40. Dioscorides: "Materia medica," v. 99; Ed. Frankof., 1598, p. 361. Pliny: "Hist. nat.," xxxiii., 33, 34; xxix., 37. Dioscorides also designates it πλατυόφθαλμον and γυναικεῖον.

[2] "Triumph Wagen Antimonii," Fratris Basilii Valentini, Benedicter Ordens, Ed. Johan Thölden, Leipzig, 1624, pp. 116–136, 172, *et passim*. For a discussion concerning the authorship of this work see Kopp: "Beitr. z. Gesch. d. Chem.," iii., 110–129.

[3] The parliament of Paris in 1566, following a decree of the medical faculty, passed an enactment declaring antimony to be a poison and forbidding its use. This remained in force until 1666, and a half-century later, in 1719, the French Government paid a goodly sum to de la Ligerie for the secret of the preparation of Kermes (a preparation of antimony) that it might be used as a medicine. From 1580 to 1655 every graduate in medicine at Heidelberg took oath that he would not administer antimony or mercury.

Although Grevin in 1568 wrote that "there is no poison by which one might more secretly poison a man,"[1] he cites no case. The first investigation of a death supposed to have been caused by antimony is given by Zacchias,[2] who, however, declares that antimony, borax, oleander, and savin, administered by the accused to produce abortion, are not poisons. The earliest recorded case of death from tartar emetic was that of an epileptic child in 1682.[3]

If we exclude poisonings by antimony among operatives exposed to inhalation of antimonial fumes,[4] all modern instances of antimonial poisoning of which we have knowledge, save one, have been caused either by the trichlorid or by tartar emetic.[5] The exception referred to is the non-fatal poisoning of a young girl and two children, reported by Page,[6] caused by lozenges purchased of an itinerant confectioner, and found on analysis to contain 0.015 gm. (half grain) of antimony trioxid each. The antimony appears to have been present as trioxid and not as tartar emetic. In what manner it got into the lozenges is unknown.

CHEMICAL.

Antimony—Sb—*atomic weight*, 120.193—*specific gravity*, 6.175—*fuses at* 450° (842° *F.*).

Elementary antimony is a bluish-gray, brittle solid, having a metallic lustre, readily crystallizable, tasteless and odorless; volatilizes at a red heat, and may be distilled in an atmosphere of hydrogen. It is prepared by roasting the native trisulfid, and reducing the product by heating with charcoal. Commercial antimony (regulus of antimony) has the appearance of a silvery metal, with a crystalline fracture, and having fern-like marks upon the surface. When deposited from decomposition of its hydrogen compound, as in the Marsh test, antimony is amorphous and has a dull silvery lustre.

Antimony is not altered in dry or moist air at the ordinary tem-

[1] "Deux livres des Venins," Anvers, 1568, p. 322.

[2] "Quæst. med.-leg.," ed. Venet., 1737, iii., 19.

[3] "Zodiacus med.-gall.," Genevæ (1682), 1685, iv., 40–42.

[4] See Lohmeier: Wchnschr. f. d. ges. Heilk., 1840, 265, 286.

[5] Early poisonings by other antimonials are mentioned by Wepfer: "Hist. Cicut. Aquat.," 1716, p. 254 (glass of antimony). Zacchias: "Quæst. Med.-leg.," ed. Venet., 1737, iii., 19, 28 (native sulfid). Hasenest: "Medicin. Richter," 1755, p. 151 (glass of antimony), and others. See Wibmer: "Wirkung d. Arznm.," v., 200, 202, 208.

[6] Lancet, 1879, i., 699.

perature. When sufficiently heated in air it burns, with formation of the trioxid. It also burns in chlorin, and combines directly with bromin, iodin, and sulfur, and with many metallic elements. It unites with hydrogen under the same conditions as arsenic does. Cold, dilute sulfuric acid does not affect it, but the hot, concentrated acid forms with it antimonyl sulfate $(SbO)_2SO_4$, and sulfur dioxid. Hot concentrated hydrochloric acid dissolves it very slowly when it is finely divided. Nitric acid oxidizes it with formation of an oxid or of antimonic acid. It dissolves readily in aqua regia, as the trichlorid or pentachlorid. It is not affected by solutions of the alkaline hydroxids, but is dissolved by solutions of potassium or sodium sulfid. The element itself does not form oxysalts. In the so-called antimony salts, such as the sulfate, tartar emetic, etc., the hydrogen of the acid is displaced by the group *antimonyl* $(SbO)'$.

Elementary antimony enters into the composition of type metal (twenty to twenty-five per cent.), Britannia metal (ten to sixteen per cent.); pewter (seven per cent.); anti-friction alloys (ten to nineteen and a half per cent.), and in small amount in brass, bell-metal, and speculum metal.

Hydrogen Antimonid—*stibin, antimoniuretted hydrogen, stibamin, stibonia*—$\mathbf{SbH_3}$—123.193.

Is produced (along with hydrogen) when a compound of antimony is in presence of nascent hydrogen, under the same conditions as the corresponding compound of arsenic, and may be obtained in larger quantity by acting upon an alloy of four hundred parts of two per cent. sodium amalgam and eight parts of freshly reduced and dried antimony with water in a current of carbon dioxid.

It is a colorless, odorless, tasteless, combustible gas, subject to the same decompositions as hydrogen arsenid, from which it differs in being by no means so poisonous,[1] in not possessing the garlic odor of the arsenical gas, and in its action upon silver nitrate solution. Hydrogen antimonid, arsenid, phosphid, and sulfid, all color dry silver nitrate yellow, from the formation of double compounds of silver nitrate with silver antimonid, arsenid, phosphid, or sulfid. All of these compounds are decomposed and turned black by water, those containing arsenic or phosphorus instantly, those of sulfur and antimony slowly. These gases also cause the formation of black deposits in solution of silver nitrate. In the case of the arsenical gas all of the arsenic remains in the solution in the form of silver arsenite, which may be precipitated by neutralizing with ammonium hydroxid, and the deposit consists of metallic silver; while with the other gases the antimony, phosphorus, or sulfur is entirely contained in combination with silver in the deposit. (See Analysis, p. 320 and Arsenic, pp. 491–505.)

[1] See Hanon: Gaz. d. hôp., Paris, 1860, xxxiii., 46. Kubeler: Arch. f. exp. Path. u. Ph., 1890, xxvii., 451.

Compounds of Antimony and Oxygen.

Three are known, Sb_2O_3, Sb_2O_4, and Sb_2O_5.

Antimony Trioxid—*antimonous anhydrid, or oxid*—**Antimonii Oxidum (U. S. Ph., Br. Ph.)**—Sb_2O_3—288.276.

Occurs in nature and is prepared by the decomposition of the oxychlorid (*q. v.*) by heat, or by heating the element in air or oxygen.

It is an insoluble, tasteless, odorless powder; white at ordinary temperatures, yellow when heated. Crystallizes in rhombic prisms, transparent and having a pearly lustre, and sometimes in octahedra. It is therefore isodimorphous with arsenic trioxid (see Reinsch test, p. 493); specific gravity, 5.2 to 5.778. At a dull-red heat it fuses to a yellowish liquid, which, on cooling, forms a crystalline mass having a silky lustre. At a higher temperature it volatilizes and condenses, in the absence of air, in prismatic needles. Strongly heated in air it burns like tinder, and is converted into the tetroxid. It is almost insoluble in water, insoluble in alcohol. Nitric acid does not dissolve it, but oxidizes it to the tetroxid. It dissolves in hydrochloric acid, with formation of the trichlorid; in Nordhausen sulfuric acid, from which solution brilliant crystalline plates of antimonyl pyrosulfate $(SbO)_2S_2O_7$, separate; and in solutions of tartaric acid or of monopotassic tartrate, with formation of tartar emetic (*q. v.*). Boiling solutions of the alkaline hydroxids convert it into antimonic acid. It is reduced, with separation of elementary antimony, when heated in a current of hydrogen.

Antimony Tetroxid—*intermediate oxid of antimony—antimony antimonate*—Sb_2O_4—304.24.

Is formed when the oxids or hydroxids of antimony are strongly heated, or when the lower oxid or the sulfids are oxidized with nitric acid or by fusion with sodium nitrate. It is a white powder, very sparingly soluble in water, with which it forms a solution acid in reaction.

Antimony Pentoxid—*antimonic anhydrid or oxid*—Sb_2O_5—320.263.

Is obtained by heating metantimonic acid to dull redness. It is an amorphous, tasteless, odorless, pale lemon-yellow powder, very sparingly soluble in water and in acids. At a red heat it is decomposed into antimony tetroxid and antimony.

Antimony Acids.

The normal *antimonous acid*, H_3SbO_3, is unknown, but the series of *antimonic acids:* ortho- H_3SbO_4, pyro- $H_4Sb_2O_7$, and meta- $HSbO_3$, is complete, either in the form of salts or in that of the free acids. There also exists, in its sodium salt, a derivation of the lacking *antimonous acid: metantimonous acid*, $HSbO_2$. The compound sometimes used in medicine under the name *washed diaphoretic antimony*

is potassium metantimonate, united with an excess of the pentoxid: $2KSbO_3,Sb_2O_5$. The *monopotassic pyroantimonate*, $K_2H_2Sb_2O_7$, 6 Aq, is a valuable reagent for the sodium compounds, the corresponding sodium salt being insoluble. The insolubility of *monosodic pyroantimonate* $Na_2H_2Sb_2O_7$, 6 Aq, is also taken advantage of in Meyer's method for the separation of antimony and arsenic (see p. 316).

Chlorids of Antimony.

Two chlorids, $SbCl_3$ and $SbCl_5$, and three oxychlorids, SbOCl, $SbOCl_3$, and $Sb_4O_5Cl_2$, are known.

Antimony Trichlorid—*protochlorid of antimony*—*butter of antimony*—**$SbCl_3$**—226.564.

Is obtained by passing dry chlorin over excess of antimony trisulfid; by dissolving antimony trisulfid in hydrochloric acid, or by distilling mixtures, either of antimony trisulfid or of antimony and mercuric chlorid, or of antimonyl pyrosulfate and sodium chlorid.

At low temperatures it is a solid, crystalline body; at the ordinary temperature a yellow, semi-solid mass, resembling butter; at 73°.2 (164° F.) it fuses to a yellow, oily liquid, which boils at 223° (433 .4 F.), forming a colorless vapor. Obtained by solution of antimony or its trisulfid in hydrochloric acid of the usual strength, it forms a dark-yellow solution, which, when concentrated to specific gravity 1.47, constitutes the *Liq. antimonii chloridi* (*Br. Ph.*).

It absorbs moisture from the air, and is soluble in a small quantity of water. With a larger quantity of water it is decomposed, with precipitation of a white powder, *powder of Algaroth*, whose composition is SbOCl if cold water be used, and $Sb_4O_5Cl_2$ if the water be boiling. In water containing fifteen per cent. or more of hydrochloric acid, antimony trichlorid is soluble without decomposition.

Antimony Pentachlorid—$SbCl_5$—297.478.

Is formed by the action of chlorin in excess upon antimony or its trisulfid or trichlorid, and is purified by distillation in a current of chlorin.

It is a fuming, colorless liquid, which solidifies at −20° (−4° F.), forming a crystalline mass which fuses at −6° (21°.2 F.). It absorbs moisture from the air. With a small quantity of water it forms a crystalline hydrate, $SbCl_5,4H_2O$; with more water a crystalline oxychlorid, $SbOCl_3$; and with a still greater quantity a white precipitate of antimonic acid, H_3SbO_4.

Sulfids of Antimony—Antimony Trisulfid—*sesquisulfid of antimony*—*black antimony*—**Antimonii Sulfidum (U. S. Ph.)—Antimonium Nigrum (Br. Ph.)—Sb_2S_3**—336.338.

Is the chief ore of antimony, and is formed when hydrogen sulfid is passed through a solution of tartar emetic. The native sulfid is steel

gray; the artificial product, an orange-red or brownish-red amorphous powder.

Heated in air it is decomposed into sulfur dioxid, and a brown, vitreous, more or less transparent mass, composed of varying proportions of oxid and oxysulfids, known as *crocus*, or *liver*, or *glass of antimony*. Corresponding to antimony trisulfid are a number of salts known as *sulfantimonites* or *thioantimonites*, having the general formula M_3HSbS_3. If an excess of antimony trisulfid be boiled with a solution of caustic potash or soda, a liquid is obtained which contains an alkaline thioantimonite, and an excess of antimony trisulfid. If this solution be filtered and decomposed by an acid while still hot, an orange-colored amorphous precipitate is produced, which is the *antimonium sulfuratum* (*U. S. Ph.*, *Br. Ph.*) and consists of a mixture, in varying proportions, of the trioxid and trisulfid. If, however, the solution be allowed to cool, a brown, voluminous, amorphous precipitate separates, which consists of antimony trisulfid and trioxid, potassium or sodium sulfid, and alkaline thioantimonite in varying proportions, and is known as *kermes mineral*. If now the solution from which the kermes has been separated be decomposed with sulfuric acid, a reddish-yellow substance separates, which is the *golden sulfuret of antimony*, and consists of a mixture of antimony trisulfid and pentasulfid. The precipitate obtained when hydrogen sulfid acts upon a solution of an antimonial compound is, according to circumstances, the trisulfid or pentasulfid, mixed with free sulfur. Hydrochloric acid decomposes antimony trisulfid, with formation of hydrogen sulfid.

Antimony Pentasulfid—Sb_2S_5—400.306.

Is obtained by decomposing an alkaline thioantimonate by an acid. It is a dark orange-red, amorphous powder, readily soluble in solutions of the alkalies and of alkaline sulfids, with which it forms *thioantimonates—sodium thioantimonate* is known as *Schlippe's salt*. An oxysulfid, $Sb_6S_6O_3$, is obtained by the action of a solution of sodium thiosulfate upon antimony trichlorid, or upon tartar emetic. It is a red powder, used as a pigment, and called *antimony cinnabar* or *antimony vermilion*.

Potassium-Antimonyl Tartrate—*tartarated antimony*—Tartar Emetic—Antimonii et Potassii Tartras (U. S. Ph.)—Antimonium Tartaratum (Br. Ph.)—$K(SbO)C_4H_4O_6$—322.969.

Is prepared by boiling a mixture of three parts antimony trioxid and four parts cream of tartar in water for an hour, filtering and crystallizing. When required pure it must be made from pure materials.

It crystallizes in right rhombic octahedra, containing one-half Aq., which turn white and lose a part of the Aq. in air, the remaining Aq. being expelled entirely at 100° (212° F.). It is soluble in about two parts of boiling water and in about fifteen parts of cold water. Its

solutions are acid in reaction, have a nauseating, metallic taste, are lævogyrous, $[\alpha]_D = -156°.2$, are precipitated by alcohol, and decompose rapidly from the formation of algæ, when exposed to the air. It is decomposed by the alkalies, alkaline earths, and alkaline carbonates, with precipitation of antimony trioxid. The precipitate is redissolved by excess of soda or potash, or by tartaric acid. Hydrochloric, sulfuric, and nitric acids precipitate corresponding antimonyl (SbO) compounds from solutions of tartar emetic. It converts mercuric into mercurous chlorid. It forms double tartrates with the tartrates of the alkaloids.

Pharmaceutic and Other Preparations Containing Antimony.

Officinal in United States and British Pharmacopœias:

Antimonii et potassii tartras, *U. S.*; tartar emetic. *Antimonii oxidum*, *U. S.*, *Br.*; antimony trioxid. *Antimonii sulphidum*, *U. S.* (*Antimonii sulphuretum*, 1870); the native trisulfid purified by fusion. *Antimonii sulphidum purificatum*, *U. S.*; the last further purified by extraction with ammonium hydroxid. *Antimonium nigrum*, *Br.*; the same as *Antimonii sulphidum*, U. S. *Antimonium sulphuratum*, *U. S.*, *Br.*; chiefly the trisulfid, with a small amount of trioxid. *Antimonium tartaratum*, *Br.*; tartar emetic. *Liquor antimonii chloridi*, *Br.*; solution of antimony trichlorid, with excess of hydrochloric acid. *Pilulæ antimonii compositæ*, *U. S.* (*Plummer's pills*): sulphurated antimony 3.25 gm., calomel 3.25 gm., guaiac 6.5 gm., mucilage of tragacanth q.s., in 100 pills. *Pilulæ hydrargyri subchloridi compositæ*, *Br.*; the same as Pil. ant. comp., U. S., except that castor oil is used in place of the mucilage. *Pulvis antimonialis*, *U. S.*, *Br.* (*James' powder*), antimony trioxid 33, precipitated calcium phosphate 67. *Syrupus scillæ compositus*, *U. S.*, contains 3 pts. of tartar emetic in 2,000. *Unguentum antimonii tartarati*, *Br.*, contains 20 per cent. of tartar emetic. *Vinum antimonii*, *U. S.*, *Br.*, the U. S. preparation contains 4 pts. of tartar emetic in 1,000; the Br., 4 pts. in 875.

Non-officinal medicines said to contain antimony:

Ayer's cherry pectoral (Hoffmann): 93.3 syrup. prun. virgin., 11.7 vin. ipecac., 11.7 vin. antim., 7.8 Tr. sanguinar., 0.2 morph. hydrochlor. *Balsam of wild cherry* (H. & H. 169[1])—contains 1 fl. ʒ extr. prun. virgin., 2 fl. ʒ extr. ipecac., 2 fl. ʒ extr. scillæ, 1 ʒ Tr. opii, 1.0 tartar emetic, 3 drops ol. anisi, 1 ʒ alcohol, ½ ʒ syrup, ½ ʒ Tinct. per-

[1] Hahn und Holfert: "Specialitäten und Geheimmittel," Berlin, 1893.

sionis comp., water to 8 ℥. *Brechzucker* (Richter) lozenges, each of which contains about 0.2 gm. tartar emetic. *Chemisches Präparat des Buchhalters J. D. Molfenter in Ulm* (Hager) is said by the manufacturer to be antimony chlorid. It is in fact commercial fuming muriatic acid. *Coxe's hive syrup* (H. & H. 801)—an infusion of 10 pts. each of serpentaria and squills in 100 pts. water, made up to a syrup with 50 pts. each of sugar and honey, in which 0.025 pt. of tartar emetic is dissolved. *Cuff's cattle medicine* (veterinary) (Geissler)—contains potassium iodid, tartar emetic, arsenic trioxid, and arsenic trisulfid. *Derby's condition powders* (veterinary) (Schädler)—2.0 tartar emetic, 20.0 antimony trisulfid (black), 10.0 sulfur, 10.0 saltpetre, 40.0 fenugreek seeds, 20.0 juniper berries. *Diaphoretic antimony*—is potassium metantimonate (see p. 293). *Dixon's pills* (Blyth)—each pill contains comp. extr. colocynth 0.1296 gm., rhubarb 0.0648, tartar emetic 0.0038. *Dr. J. Johnson's pills* (Blyth)—each pill contains: comp. extr. colocynth 0.162 gm., calomel 0.039, tartar emetic 0.002, oil of cassia 0.007. *Elixir tonique antiglaireux de Guillé* (H. & H. 483), a complex elixir containing 1 gm. of tartar emetic in 11 litres. *Ethiops of Antimony*—is a mixture of antimony trisulfid 3 pts. and mercuric sulfid 2 pts. *Flechten*[1] *pulver aus St. Lubes* (Wittstein)—contains saltpetre 10 pts., antimony trichlorid 1 pt., antimony trioxid 20 pts. *Flowers of antimony*—is an impure oxysulfid, with varying quantities of the trioxid and trisulfid. *Glass of antimony*—is a mixture of antimony trioxid and trisulfid. *Hind's sweating ball* (veterinary) (Blyth), 3.888 gm. each of tartar emetic and asafœtida made into a ball with liquorice powder and syrup. *James' powder*—see *Pulv. antimonialis* among the officinals. *James' pills* (Wittstein)—consist principally of mercuric oxid 1 pt., James' powder 30 pts. *Kermes mineral*[2] was formerly officinal under the name *Antimonii oxysulphuretum, U. S. Ph. Liver of antimony*—is a mixture of antimony trioxid and trisulfid, potassium sulfid and potassium carbonate; used in veterinary practice. *Mitchell's pills* (Blyth)—each pill contains: aloes 0.070 gm., rhubarb 0.103, calomel 0.01, tartar emetic 0.003. *Poudre merveilleuse de Virier* (H. Bürchner) consists of iodin 48 pts., arsenic 8 pts., tartar emetic 8 pts., phosphorus 1 pt. *Poudre unique de Godemaux*, for epilepsy, is precipitated calomel (Braconnot). Gray oxid of antimony (Alcyon). Calomel and metallic mercury (Planche). *Quietness* is tartar emetic, used in England as a popular cure for dipsomania. *Rotzkrankheit*[3] *Mittel* (veterinary) (Bürchner)—1. Each bolus weighs about 35 gm. and contains about 1 gm. of golden sulfuret of antimony (see p. 294), and 7 gm. of liver of antimony (see above). 2. Each contains 7 gm. of antimony trisulfid (black). *Schnellmast pulver von R. Hübner* (veterinary; condition powder) (Karmrodt), contains 15.56 per cent. of antimony trisulfid. *Schweine pulver von Dr.*

[1] Flechte=herpes.
[2] See p. 294.
[3] Glanders.

Gustav Swoboda (veterinary) (Hager) contains about 34 per cent. of antimony trisulfid. *Sel desopilant d'Audin-Rouviere* (H. & H. 1539) contains 0.2 per cent. of tartar emetic. *Trunksuchtsmittel*[1] *von Karrer-Gallati* (Karlsr. Ortsges.-Rath) consists of two liquids, one an alcoholic extract of gentian, the other a 2.6-per-cent. solution of tartar emetic. *Trunksuchtsmittel von J. H. Rungel* (E. Harms) is a 3.5-per-cent., aqueous solution of tartar emetic. *Trunksuchtsmittel von Fr. Schumacher* (H. & H. 1705) is a solution of tartar emetic. *Vomi-purgatif de Leroy* (H. & H. 1770) contains 0.8 per cent. of tartar emetic. *Ward's red pill* (Blyth)—glass of antimony and dragon's blood. *Washed saffron of antimony* is liver of antimony (see above) from which the potassium sulfid and carbonate have been removed. *Weikard's Hauspillen* (H. & H. 761) contain 7.2 per cent. of "golden sulfuret of antimony," and also about 15 per cent. of calomel.

Antimonials used in the Arts.

Alloys of antimony, see p. 291. *Antimony cinnabar*, or *vermilion* is an oxysulfid.[2] *Antimony yellow* is a mixture of lead antimonate and basic lead chlorid. *Naples yellow* is basic lead antimonate. *Antimony trisulfid* is used in the native, crystalline form in the friction tube used in firing cannon, in percussion caps, in red and blue fires, and in the heads of Swedish matches; and in the amorphous, orange form in the vulcanization of caoutchouc, which it colors red-brown. *Glass of antimony*[3] is used to color glass and porcelain yellow. *Liquid butter of antimony* is a concentrated solution of the trichlorid, specific gravity 1.35, used to color iron and steel brown (gun barrels). *Schlippe's salt*[4] is used in photography.

ANTIMONY TRICHLORID.

Of the eight intoxications by antimony trichlorid of which we find record[5] three were accidental, three suicidal, and in two it was "taken by" the patient. Four cases were fatal and four recovered. In two of the non-fatal cases the dose taken was one ounce (31 gm.). In all of the fatal cases the dose was two ounces (62 gm.) or more. The shortest duration was in Cooke's (R. B.) case,[6] in which a woman of forty years died in less than two hours; and the most prolonged in Crisp's case[7] in which a girl of nineteen years died in twenty-four hours.

[1] Trunksucht=dipsomania.
[2] See p. 294.
[3] See p. 294.
[4] See p. 294.
[5] Cooke (W.): Lancet, 1848, 230. Houghton: Lancet, 1841, i., 324. Crisp: Tr. Path. Soc., London, 1865, xvi., 125. Taylor: "Poisons," 3d Am. ed., 456–458. Banks: Prov. M. and S. Jour., 1846, December 23d. Cooke (R. B.): Lancet, 1883, i., 860.
[6] *Loc. cit.*
[7] *Loc. cit.*

Symptoms.—Antimony trichlorid acts mainly as a corrosive, and the symptoms observed are those of intoxication by the mineral acids (*q. v.*). The violent symptoms may, however, be slightly pronounced and the case present a narcotic type, as in one of Taylor's cases (attributed to Mann),[1] in which a man swallowed from two to three ounces (62–93 gm.). When seen about an hour later there was entire prostration of strength, with coldness of the skin, and incessant attempts to vomit. Severe 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 and a half hours after he had swallowed the poison. In Crisp's case there was no urine passed.

Post-mortem Appearances.—The most noteworthy appearance is a blackening and apparent charring of those parts with which the corrosive liquid has come into contact, similar to that produced by sulfuric acid. The lips and mouth may be black and excoriated, also the œsophagus, stomach, and upper intestine. In Crisp's case the stomach appeared as if uniformly coated with the antimonial. When this coating was removed the surface appeared black and charred. In Evans' case (reported by Taylor) the stomach was vivid red in places, in others blackened. In Cooke's (R. B.) case the mucous membrane of the stomach was intensely congested and of a bluish-black color. In this case, as well as in Evans', corrosion and blackening of the lips, mouth, and œsophagus were not observed. Perforation of the œsophagus, stomach, or intestine was not met with in any of the cases. From the intensity of the corrosive action, however, it is quite probable that it might occur.

Analysis.—In Evans' case antimony was found in the putty-like masses of altered mucous membrane, etc., from the stomach. In Cooke's (R. B.) case the stomach was found to contain compounds of antimony and of arsenic to the amount of eight grains (0.5 gm.) of the former, and 0.1 grain (0.0065 gm.) of the latter. (For methods see under Tartar Emetic and Arsenic.)

[1] *Loc. cit.*

TARTAR EMETIC.

Statistics and Administration.

Of 119 cases of tartar emetic poisoning of which we find record, 87 were "accidental," 6 suicidal, and 17 homicidal. In comparing tartar emetic poisonings with those by other poisons, except arsenic, the very small proportion of suicidal cases (5 per cent.), and the large proportion of homicides (14.3 per cent.)[1] are noteworthy.

Of the **homicidal cases** two occurred in the seventeenth century,[2] the remainder since 1850. The following are the homicidal cases since 1850 of which we have knowledge:

1854. *Case of Ann Palmer.*—The symptoms of her last illness were those of antimonial poisoning. The body was exhumed one year after death and found to contain antimony. Her son, suspected of having poisoned her with repeated doses of tartar emetic, was executed for another poisoning by strychnin.[3]

1856. *Reg. v. McMullen.*—Woman gave her husband "*quietness*" (a mixture of cream of tartar and tartar emetic) to counteract the effects of a debauch, whereof he died. Convicted of manslaughter.[4]

1857. *Reg. v. Freeman.*—Mentioned by Taylor. No particulars.[5]

1857. *Reg. v. Hardmann.*—Mentioned by Taylor. Tried for the murder of his wife by repeated administration of small doses of tartar emetic.[6]

1858. *Affaire Ramier.*—Conspiracy by R., his mistress (Zélie), and Porel to poison the wife of R. First attempt with phosphorus, second with tartar emetic. Both unsuccessful. Porel confessed; Ramier convicted; Zélie acquited.[7]

1859. *Reg. v. Smethurst.*—Surgeon, indicted for the murder of his mistress by repeated small doses of antimony and arsenic. Tried twice. Convicted on second trial. Subsequently pardoned.[8]

[1] If we add seven cases of gross negligence the percentage is increased to 20.1.

[2] Zacchias: "Quæst. Med.-leg.," Consil. iii., 12–14, ed. Venet., 1737, iii., 19–24. Zittmann: "Med. For.," 1706, p. 1455.

[3] Browne and Stewart: "Trials for Murder by Poison," pp. 87, 88. Taylor: "Poisons," 3d Am. ed., 96, 121; Guy's Hosp. Reps., 1857, 3 s., iii., 369–481.

[4] Ph. J. and Tr., 1856–57, xvi., 147, 197. Taylor: *Loc. cit.*, 58, 121; and "Med. Jur.," 11th Engl. ed., 47; Lancet, 1856, ii., 259.

[5] "Poisons," 3d Am. ed., 145.

[6] *Ibid.*, 2d Engl. ed., 97.

[7] J. d. chim. méd., etc., 1858, 4. s., iv., 32.

[8] Browne and Stewart: *Op. cit.*, pp. 397, 448–479; Sessions Papers, Central Crim. Crt., 1859. Stephen: "Hist. of the Crim. Law of England," iii., 438, 465; Brit. M. Jour., 1859, ii., 707–711. Taylor: "Princ. and Pr. of Med. Jur.," 3d ed., 1883, 196.

1860. *Reg. v. Winslow.*—Defendant charged with murder of Ann James by aggravating a disease of the cæcum from which she was suffering by the repeated administration of small doses of antimony. Antimony was found in the body of the deceased. Prisoner acquitted.[1]

1865. *Reg. v. Pritchard.*—Tried at Edinburgh. Defendant was a physician. Poisoned his wife with repeated doses of tartar emetic, and his mother-in-law with antimony and aconite. Convicted. Confessed before execution.[2]

1866. *Reg. v. Bellemey, alias Barnett.*—An apothecary was tried at Melbourne, Australia, for the murder of his paramour by repeated doses of antimony. Acquitted.[3]

1871. *State v. Eliz. G. Wharton.*—Tried at Annapolis, Md., December, 1871, and January, 1872. Defendant was accused of the murder of Gen. W. S. Ketchum by tartar emetic. Another visitor at defendant's house, Mr. Eugene Van Ness, suffered violent symptoms of antimonial poisoning. Acquitted.[4]

1871. A woman was held for trial in England (Staffordshire assizes). Antimony had been found in the cadavers of three of her children. She had buried ten out of twelve children at different times, some having manifested symptoms of antimonial poisoning. We are unable to find further reference to this case.[5]

[1876. *The Balham Mystery*, or *Bravo Case.*—Bravo, a barrister, probably died from the effects of antimony and laudanum. The coroner's jury found a verdict of wilful murder by some unknown person. Subsequently it was shown that the deceased had purchased a large number of quack powders, advertised for the cure of dipsomania, which contained tartar emetic.[6] (Classified above as suicide.)]

1878. Prof. H. Ranke reports the case of a man who was supposed to have died from tartar emetic administered by a barber-surgeon (bader).[7]

[1] Browne and Stewart: *Op. cit.*, 479–489. Taylor: "Poisons," 3d Am. ed., 121.

[2] Browne and Stewart: *Op. cit.*, 397–448. Reports of trial: William Kay, Edinb., 1865; Edinb. M. and S. Jour., 1865, xi., 163–200; Wien. med. Wchnschr., 1865, xv., 1072, 1105. Felizet: Arch. gén. d. méd., Paris, 1865, ii., 267–277.

[3] Ph. J. and Tr., 1868–69, 2 s., x., 325.

[4] Report of trial. Baltimore Gazette, Balt., 1872. Aiken: "A Review of Professor Reese's Review of the Wharton Trial," N. Y., 1873; also, on "Some Supposed Fallacies," etc., Richmond and Louisville M. Jour., 1873, xv., 7–13. Chew: *Ibid.*, 1872, xiv., 93–116; Med. Rec., N. Y., 1873, viii., 332–338. Morse: Am. Law Rev., Boston, 1874, 647–668; also in his "Famous Trials," 274–309. Reese: Am. J. M. Sc., Phila., 1872, n. s., lxiii., 329–355. Williams: Richm. and Louisv. M. Jour., 1873, xv., 721–747; Med. and Surg. Reptr.; Phila., 1872, xxvi., 475, 503, 523. Wood, H. C., Jr.: Med. Rec., N. Y., 1873, viii., 169. Taylor: "Med. Jur.," 11th Am. ed. (Bell), 23.

[5] Ph. J. and Tr., London, 1871–72, 3 s., ii., 397, 458, 516.

[6] Taylor: "Med. Jur.," 11th ed., 136. Johnson, Payne, and Redwood: Lancet, 1876, i., 755. Wade: Brit. M. Jour., 1876, ii., 264.

[7] Friedreich's Bl. f. ger. Med., 1879, xxx., 241. See also Schelle: *Ibid.*, 1882, xxxiii., 8.

1878. *State v. Vosburgh.*—A preacher tried in Hudson County (N. J.), for an attempt to murder his wife by tartar emetic. Case unpublished.

1887. *Reg. v. Th. Hall.*—New Zealand. A man of seventy years, poisoned, as alleged, by his son in law.[1]

1893. *Peo. v. Henry Meyer, etc.* Defendant tried twice for the murder of one Baum or Brandt, to obtain an insurance upon his life. Convicted of murder in the second degree.[2]

1894. *State v. Effie Cox, alias Whalen, and Rose Fournier.*—Tried in Chittenden County Court, Vermont, 1894. Defendants indicted for the murder of Fournier by repeated administration of tartar emetic; convicted of manslaughter, first degree. "Reasonable doubt" that the purpose of the administration was to correct an alcoholic habit. Case not published.

Suicide by tartar emetic is of most exceptional occurrence. Excluding the Bravo case above mentioned (which was to some extent at least an opium poisoning, and possibly accidental), there are but 5 cases, all anterior to 1860. Of these 3 were in females, of which 1 was fatal.[3] Of the 2 men 1 died.[4] (See Lethal Dose, below.)

Of the **accidental** cases 28 were in children less than ten years of age (for the most part medicinal poisonings); and 56 in persons over ten years of age, who took the poison either medicinally in overdose or in mistake for other drugs—*i.e.*, Glauber's salt, nitre, cream of tartar, bicarbonate of soda, Epsom salt, potassium acetate, oxid of antimony, sugar, or as an external application.

The use of tartar emetic as a medicine is now much less general than it formerly was, and consequently accidental poisonings by it are now very infrequent. During the past twenty-five years but 18 tartar-emetic poisonings have been reported. Of these 6 were homicidal, 10 due to negligence or mistake, 1 to absorption from a fabric, and 1 medicinal.

The **administration** in all but 8 cases was by the mouth. In 4 instances it was by external application, either as powder

[1] Brit. Med. Journ., 1887, i., 853.

[2] Doremus: J. Am. Ch. Soc., 1895, xvii., 667.

[3] A trance medium who apparently poisoned herself to verify her own prediction of her death (Ellis: Bost. M. and S. Jour., 1856–57, lv., 400). The other two are reported in Lancet, i., 1844, 444 (Lefevre); and Galtier: "Toxicologie," i., 505 (in 1835).

[4] Récamier's case. See Galtier: *Op. cit.*, 507. The non-fatal case is reported by Serres. See Galtier: *Op. cit.*, 506.

or ointment;[1] in 2 cases in enemata;[2] in 1 case by absorption from a fabric mordanced with tartar emetic,[3] and in 1 case in the milk of the mother.[4]

In 28 instances tartar emetic was taken in aqueous solution; in 25 as a powder; in 5 as antimonial wine, in 1 as "horse medicine," and in 1 as a patent medicine.

Lethal Dose.

It is only under exceptional conditions that a single dose, however large, of tartar emetic may cause the death of an adult. An analysis of the 37 deaths of adults attributed to tartar emetic shows that 13 were cases of alleged homicide, in most, if not all, of which the theory of the prosecution implied repeated administrations; 7 were accidental poisonings caused by more than one dose, 5 were cases in which antimonial poisoning was probably not the proximate cause of death; in 5 the method of administration is doubtful, and in 1 it was by external application. The 6 remaining cases may be summarized as follows:

1. *Hoffmann*, 1760.[5]—A mere statement that a person (a woman) died in a short time after an improper administration of tartar emetic.

2. *Orfila*.[6]—An incidental statement that a woman of eighty-two years died in fifteen hours after having taken 0.5 gm. of tartar emetic, which caused some purging but no vomiting.

3. *Skae*, 1844.[7]—In this case a man of forty-five years certainly took one dose of about fifty-five grains of tartar emetic, while suffering from delirium tremens, and vomited in ten minutes. About sixteen hours later he got out of bed and in so doing fell insensible and died.

4. *Beale*, 1854.[8]—A girl of sixteen years took a quantity of tartar emetic estimated at forty to sixty grains at one time. In fifteen minutes she vomited, and continued to vomit and purge continuously for about three hours. The next morning she did not seem ill, but in the afternoon she felt as if dying. A physician was then called, who found her continually throwing the head back and screaming, with dilated pu-

[1] Lancet, 1838, i., 250 (two cases); Beitr. z. prk. Heilk., 1834, i., 670 (two cases).

[2] Charier: J. d. chim. méd., 1847, 3 s., iii., 472.

[3] Gillet: Ann. d. l. Soc. méd.-chir. d. Liége, 1886, xxv., 83.

[4] Wibmer: "Wirkung d. Arzneimitt.," v., 179.

[5] "Op. omn.," v. i., pt. ii., ch. v., ed. Genev., 1760, i., 213.

[6] "Tox. gén.," 5ème ed., i., 620.

[7] North. J. Med., Edinb., 1844, i., 289.

[8] Lancet, 1854, i., 68.

pils, knees drawn up, and a thin pulse. She was delirious for six hours before death, which occurred thirty-six hours after she had taken the poison. The autopsy revealed the existence of extensive incipient ovarian disease.

5. *Gabb*, 1866.[1]—A man of forty-three years took about two drachms of tartar emetic at one time by mistake, and very shortly thereafter "a draft of vinegar." He walked a mile to a physician's office, where he appeared so well, so free from any of the symptoms of having taken tartar emetic, that it was believed that he was mistaken. Soon after he vomited for the first time, about an hour after having taken the emetic. After a severe attack of vomiting, purging, cramps, etc., he passed a comfortable night and the next day was much better. Early the following morning he was pulseless and in a condition of collapse resembling that of cholera. After administration of brandy his condition was much improved, but, sitting up in bed contrary to advice, he had an attack of syncope, from which he rallied a little, but sank again and died about forty hours after the accident, retaining his consciousness nearly to the last moment.

6. *Dobie*, 1887.[2]—A boy of fifteen years took forty grains of tartar emetic in mistake for Epsom salt. He vomited in ten minutes and suffered the usual symptoms of antimonial poisoning, from which he seemed to be recovering on the third day. On the third day he became delirious and died comatose on the sixth day. In the short report of this case the duration is stated in one place as six days and four lines below as twelve days.

Cases 1, 2, and 6 are incompletely reported. In Case 3 acute alcoholism was at least a contributing cause of death. In Case 5 the long delay of the emetic action of the poison (caused possibly by the vinegar taken) favored absorption, prevented that prompt removal of the poison which is the chief factor of safety when single doses are taken, and established very much the same conditions as obtain in cases of repeated administration. Beale's case may therefore be said to be the only uncomplicated fatal poisoning of an adult by a single dose of tartar emetic. And in this the history is by no means sufficiently explicit (the reporter having only seen the case on the second day, and the hearsay evidence having been given in a case was which the subject of a coroner's inquiry) to fully prove the method of administration and the absence of other causes of death.

Of the five cases above referred to as instances in which the

[1] Med. Times and Gaz., 1866, ii., 379.

[2] Lancet, 1887, i., 773.

causative relation of antimonial poisoning to the fatal result is questionable, two were clearly deaths solely from other causes.

These are: (7) Deutsch's case, cited by Wharton and Stillé,[1] of a woman who is alleged to have died "in the course of a year" from the effects of a single dose of a scruple (1.3 gm.) of tartar emetic. The other (8) is Wakeley's case, cited by Taylor,[2] of a man said to have been fatally poisoned by three grains, and concerning which Taylor says that "death could not be reasonably attributed to the medicine."

In the other three cases tartar emetic administered to persons already seriously ill can be considered at the most only as a contributary cause of death.

9. *Récamier.*[3]—A man of about forty years took with suicidal intent 2 gm. (thirty-one grains) of tartar emetic. After exhibiting the usual antimonial symptoms he became furiously delirious and died in convulsions on the fourth day. At the autopsy excessive cerebral lesions were discovered. In commenting upon this case Galtier[4] remarks that death should be attributed to the recent lesions of the brain and meninges, of which the emetic, or rather the inflammation of the intestinal canal and the efforts of vomiting, were only the provoking cause.

10. *Constant*, 1831.[5]—The case of a medical student under treatment by Professor Andral for "gastric trouble due to insufficient nutrition and overstudy," received two grains of tartar emetic in three half-glasses of water, which immediately provoked vomiting, and severe pain, which persisted the remainder of the day, with abundant diarrhœa. He died on the second day.[6]

11. An extremely feeble woman of fifty-five years, suffering from broncho-pneumonia, was bled 500 gms. and was given 0.15 gm. (about gr. iiss.) of tartar emetic. In the following night and without any symptoms referable to the alimentary canal, she was suddenly attacked with copious and continued intestinal hemorrhages which caused her death.[7]

In several instances persons have taken quantities much greater than that which is probably the lethal dose of tartar

[1] "Med. Jur.," 4 ed., ii., 193, *ex* Canstatt's Jahresb., 1851, iv., 270.

[2] "Poisons," 3d Am. ed., 448.

[3] Magendie: "De l'influence de l'émétique," etc., Paris, 1813, and quoted by most authors from Orfila down.

[4] "Toxicologie," i., 508.

[5] Arch. gén. de méd., 1831, 1 s., xxiv., 262.

[6] This is the case referred to by Wharton and Stillé, ("Med. Jur.," 4th ed., ii., 193), in the brief statement: 'Two grains have proved fatal to an adult." It is also mentioned by Taylor ("Poisons," 3d Am. ed., 448), who, however, adds: "But in this case there were circumstances which favored the fatal operation of the poison."

[7] Piorry: Gaz. d. hôp., 1853, xxvi., 147.

emetic, and although rendered severely ill thereby, have recovered. Thus Taylor[1] quotes a case of Couling's, of a man who took two hundred grains (13 gm.) in mistake for carbonate of soda. McCreery[2] reported that of a man who took half an ounce (15.6 gm.) in mistake for Glauber's salt, and Gleaves[3] that of a man who took a tablespoonful (about an ounce, 31 gm.). Orfila[4] also quotes Lebreton's observation of a young woman who recovered from the effects of 24 gm. of tartar emetic.

Very much smaller quantities have been known to cause death when taken by adults in more than one dose. That even two administrations upon two consecutive days of as small a total quantity as 0.2 gm. (three grains) may cause death when the first dose fails to provoke emesis is proved by the fatal poisoning of a woman who died in thirty-six hours after taking the first dose.[5] In Laveran's case, reported by Tardieu,[6] a man suffering from erysipelas of the face was given 0.4 gm. (6.17 grains) of tartar emetic in doses of 0.1 gm. (one and one-half grains) during four days. He was attacked with nausea, vomiting, epigastric pain; passed liquid stools, and died in collapse in nine days. Antimony was found in the liver.

The susceptibility of young children to the poisonous action of tartar emetic is such as to render its administration to them as a medicine dangerous. Beck[7] has collected a number of observations of serious and fatal poisoning in young children by tartar emetic. Among these are two by Charier[8] of children recovering from the measles who were rapidly destroyed by 0.05 gm. (three-quarter grain) given in enemata.

Duration and Prognosis.

In children the duration of fatal tartar-emetic poisoning is shorter than in adults. The average of 8 cases in children less than ten years of age, in which the duration is definitely stated, was 15.9 hours, the extremes being 1 and 48. In adults the average of 18 cases was 88.9 hours, or, if 5 exceptionally protracted cases are omitted, 37.4 hours, with 6 and 96 as the extremes.

[1] "Poisons." 3d Am. ed., 443.
[2] Am. Jour. M. Sc., 1853, n. s., xxv., 131.
[3] West. J. M. and S., 1848, 3 s., i., 23.
[4] "Tox. gén.," 5ème ed., i., 620.
[5] Beau: Bull. gén. de thérap., 1856, li., 231.
[6] "Empoissonement," 2ème ed., 744.
[7] "Infant Therapeutics," 2d ed., N. Y., 1855, 30–47.
[8] J. d. chim. méd., etc., 1847, 3 s., iii., 472.

The most rapidly fatal poisoning was in a child to which, after an attack of benignant measles, 0.05 gm. (three-quarter grain) of tartar emetic was given in an enema, and an hour after he was dead.[1] One of Wilton's[2] cases was that of a child nearly a year old whose mother gave it repeated small doses of antimonial wine for a cold. Very suddenly thereafter it had slight but frequent convulsions, with sickness and severe diarrhœa, and died in about two hours. Hartley[3] has reported the cases of two children of five and three years which died in eight and twelve hours respectively from the effects of ten grains (0.65 gm.) of tartar emetic. A more recent and somewhat more reliable case is that published by Chabon,[4] in which a vigorous child of two and a quarter years received two doses of 0.05 gm. (three-quarter grain) each, ten minutes apart, and died of syncope eight hours later. The most protracted duration of a fatal case in a young child of which we find notice is that merely mentioned by Sigmond[5] of a child of two years which died forty-eight hours after an external application of tartar emetic to the spine. Two fatal cases of twenty-four hours' duration are reported, in one[6] of which, however, the child died most probably from disease; while in the other,[7] in which tartar-emetic ointment was applied to a child of three years, which died in eclampsia, the patient was suffering from whooping-cough.

In adults only the duration of poisonings in which more than one dose was certainly or probably administered can be given. The shortest duration was in a case involving an accusation of criminal negligence, in which a man of twenty-eight years died six hours after he had received 1 gm. (15.4 grains) of tartar emetic in two doses, half an hour apart. It is questionable whether in this case the antimonial was more than a contributory cause of death.[8] The fatal termination followed the last administration in ten hours in two instances—a trance medium, aged twenty-one, who appears to have taken an unknown quantity, after having predicted her own death. She

[1] Charier: J. d. chim. méd., etc., 1847, 3 s., iii., 472.
[2] Prov. M. and S. J., 1844, 204.
[3] Lancet, 1846, i., 460.
[4] Union méd. d. l. Seine inf., 1881, xx., 114.
[5] Lancet, 1838, i., 250.
[6] Taylor: "Poisons," 3d Am. ed., 447.
[7] Krebs: Beitr. z. prkt. Heilk., 1834, i., 670.
[8] Ranke: Friedreich's Bl. f. ger. Med., 1879, xxx., 241. Schelle: *Ibid.*, 1882, xxxiii., 8.

had had a similar attack previously.[1] A man of thirty years died in ten hours after the outbreak of symptoms supposed to have been caused by tartar emetic which he had had several weeks in his possession.[2] In four instances the duration has exceeded a week. A woman of eighty-three years, a patient in the Salpêtrière, received 4 gm. (61¾ grains) of tartar emetic during three days, and having exhibited the symptoms of antimonial poisoning, died upon the seventh day.[3] A man received 0.4 gm. (6.2 grains) of tartar emetic during four days. He suffered from nausea, vomiting, and epigastric pains, passed liquid stools, and died in collapse nine days after taking the last dose.[4] A man of thirty-four years is reported to have died in twelve days after a "treatment" at the hands of an empiric, which is said to have consisted in part in the administration of daily doses of twenty to thirty grains (1.3 to 2 gm.) of tartarized antimony almost continuously for three weeks.[5]

In all of the homicidal cases of which we find published accounts the poisoning extended over several days. In the Ann Palmer, McMullen, and Hardman cases death occurred after symptoms referable to the administration of repeated doses. In the Wharton case the deceased, a healthy man of about sixty years, was first attacked on June 24th, and died on the 28th, having had two distinct outbreaks of symptoms. Another gentleman who spent some days at the defendant's house had a violent attack of symptoms such as are produced by antimony, from which he recovered; and some days afterward the accused offered him a glass of beer, which was analyzed, and found to contain fifteen grains (1 gm.) of tartar emetic. In the Pritchard case one of the victims (Mrs. Pritchard) was first taken ill about January 1st, 1865, and died March 18th, having had several attacks, each probably succeeding an administration, while the other victim (Mrs. Taylor) had two distinct attacks, February 13th and 22d, of the last of which she died February 24th. In Smethurst's case the first symptoms were observed

[1] Ellis: Bost. M. and S. Jour., 1856–57, lv., 400.

[2] Pollock: London M. Gaz., 1850, xlv., 801.

[3] Durand-Fardel: Bull. gén. d thérap., 1843, xxv., 370.

[4] Laveran, cited by Tardieu: "Empois.," 2ème ed., 744.

[5] Le Boutillier: N.-West. M. and S. J., Chicago, 1855, xii., 353. Beck ("Infant Therap.," 2d ed., 141) makes a mere mention of the death of a woman of twenty-six years, twelve days after she had taken ʒ ss. of tartar emetic. See also Dobie's case above referred to.

March 29th, and the woman died May 3d. In the Winslow case the poisoning began early in February, and death only occurred June 24th. In the Cox-Fournier case there were repeated administrations, the last illness lasted four days, and the last dose was administered six hours before death. In the Meyer, Bellemey, and Hall cases there was also allegation of repeated administration on the part of the prosecution.

The **prognosis** is probably more favorable than is indicated by the cases reported. Of 118 such 56 (47.4 per cent.) died, and 62 (52.6 per cent.) recovered.

Symptoms and Elimination.

The taste of tartar emetic, which is metallic in character, although not very pronounced, may be noticed at the time of swallowing it; but more frequently it escapes observation.

The **interval** preceding the manifestation of the symptoms proper is usually from a quarter to half an hour. In one instance vomiting is said to have immediately followed the taking of forty grains (2.6 gm.) of powdered tartar emetic.[1] In another case in which 1.8 gm. (ʒ ij.) were taken in aqueous solution, nausea, trembling, and cold sweat followed in seven minutes.[2] In three instances the duration of the interval was ten minutes.[3] In three instances no symptoms appeared for an hour.[4]

Soon there is nausea, which is followed by vomiting, which is frequently repeated, sometimes as often as every three or four minutes, and after a time without nausea. The vomited matters at first consist of the stomach contents, and later become fluid and tinged with bile or blood. At the same time there is a burning sensation in the mouth and œsophagus, with a sense of constriction at the base of the throat. Deglutition is difficult, and thirst severe. The lips and buccal mucous membrane sometimes become swollen, and there is salivation in some cases. The acts of vomiting are early accompanied by violent epigastric and abdominal pain and by purging, the stools being watery or

[1] Durrant: Prov. M. and S. Jour., 1850, 369.

[2] Ornellas: Gac. med. d. Lima, 1856, i., No. 10.

[3] Galtier: "Toxicologie," i., 504. Dobie: Lancet, 1887, i., 773. Skae: North. J. of M., Edinb., 1844, i., 289.

[4] Freer: Lancet, 1847, i., 535. Gabb: Med. Times and Gaz., 1866, ii., 379. Mason: Brit. M. Jour., 1877, i., 674.

"rice water" in character, and suddenly and involuntarily expelled. The pulse is at first accelerated, but the arterial tension is rapidly lowered, and the pulse and respiration sink. There are cramps in the extremities, sometimes accompanied by spasmodic contractions, which may even become general and distinctly tetanic in character.[1] The skin is cold and moist, there are attacks of vertigo and syncope, and finally loss of consciousness, collapse, and death from heart failure.

In some instances violent delirium has preceded death,[2] while in other cases the patient has become drowsy or comatose.[3] Death has also been known to occur suddenly during a muscular effort on the part of the patient.[4]

Some one or more of the prominent symptoms may also remain absent. Thus in one instance (not fatal), although there were incessant nausea and great prostration, there was no purging. The dose, it is true, was small (fifteen minims of antimonial wine to an adult) and the patient recovered.[5] In two instances the patient did not vomit,[6] in two not during the first hour,[7] and in one only under the influence of an emetic.[8] In Gabb's case pain was also absent. In another case, although the other symptoms were pronounced, there was neither difficulty in swallowing nor sense of heat or constriction in the throat.[9] In one instance the urine was suppressed.[10] In two in-

[1] The occurrence of tetanus as an effect of tartar emetic was a point of great interest in the Palmer case (see Times Report of Trial, London, 1856, and Elliotson: M. Times and Gaz., 1856, n. s., xiii., 6–8). Antimony had been found in the cadaver of the deceased as well as in that of Palmer's mother, which had been exhumed, but the analysis had failed to show the presence of strychnin. Yet, although the symptoms in Cook's case were at first distinctly those of antimony, the later attacks, during one of which he died, were as markedly those of strychnin. Although, as in Elliotson's cases, the spasms due to antimony may be distinct and severe, they have not been known to be sufficiently severe and prolonged to cause death by asphyxia during the spasm, as strychnin frequently does, and as was the case with Cook. The theory of the crown that Cook was first dosed with antimony and afterwards killed by strychnin is most probably correct, notwithstanding the partial failure of the analysis.

[2] Beale: Lancet, 1854, i., 68. Beck: "Med. Jur.," 7th ed., 897.

[3] Mason: Brit. M. J., 1877, i., 674. Dobie: Lancet, 1887, i., 773.

[4] Skae: Northern J. of Med., Edinb., 1844, i., 289.

[5] Richardson: Lancet, 1856, i., 400.

[6] Beck: "Med. Jur.," 7th ed., 897 (Serres). Orfila: "Toxicologie," 5ème ed., i., 625.

[7] Gabb: Med. Times and Gaz., 1866, ii., 379. Gleaves: West. J. M. and S., Louisv., 1848, 3 s., i., 23.

[8] Orfila: *Op. cit.*, 621.

[9] Procter: Assoc. M. J., 1853, 513.

[10] Taylor: "Poisons," 3d Am. ed., 443 (Carling).

stances a peculiar metallic taste is noted, not as the first but as a rather later manifestation.[1] A pustular eruption similar to that produced by inunction was observed in one case of internal administration.[2]

The external application of tartar emetic causes local and systemic effects. A pustular eruption, resembling that of variola in appearance (ecthyma antimonale), attended with pain, fever and œdema, is produced upon the skin. The pustules first resemble little knots at the openings of the skin follicles, soon increase in size, become inflamed and purulent, and form deep crater-like ulcers, which readily become gangrenous, and sometimes involve exfoliation of bone. The action upon the skin is not limited to the surface of application, but may make its appearance at points far distant, as upon the genitals and internal surface of the thighs, after inunction to the breast or shoulders. The usual systemic symptoms—nausea, vomiting, purging, great prostration, and frequent fainting—also follow absorption from external application; and complete suppression of urine has been observed. Death has been caused by application of tartar emetic to the uninjured skin,[3] as well as by its application to wounded surfaces.[4]

The administration of repeated doses causes nausea, vomiting, purging, great prostration and weakness, pains in the stomach and bowels, heat and uneasiness about the throat and mouth, constant thirst, and a foul tongue. The person becomes emaciated; the eyes are sunken and watery, but clear and intelligent; the features are sharp and flushed; the skin is moist and cool. The pulse is weak, contracted, and very rapid. There are local spasms, as of the wrist, early in the poisoning. The person becomes weaker and weaker; the cheeks become more hollow, sharp, and pinched looking, though still flushed; the eyes red and sunken, with a peculiar wild expression; the pulse very weak and rapid, and the tongue more foul. Death finally results from exhaustion. During such a case more violent acute symp-

[1] Rynd: Penins. and Ind. Med. Jour., Detroit, 1859-60, ii., 717. Hulot: Arch. gén. d. méd., 1853, 5 s., i., 475.

[2] Gleaves: *Loc. cit.*

[3] Krebs: Beitr. z. prkt. Heilk., 1834, i., 670. Sigmund: Lancet, 1838, i., 250. Lewin: "Nebenwirk. d. Arzneimittel," 2d ed., 1893, 681. See also Gillet: Ann. Soc. méd.-chir., Liége, 1886, xxv., 83.

[4] Sigmond: *Loc. cit.* Tardieu and Roussin: "Empoisonnement," ed. 1866, p. 610.

toms may occur, following upon a renewed administration of the poison, and the patient may die during one of these.

The **method of action** of antimony is very similar to that of arsenic. It causes continuous diminution of the blood pressure in consequence of dilatation of the vessels, of direct action upon the heart, and of disorders in the central nervous system; and also gastro-intestinal symptoms due to congestion and changes in the mucous membranes of the stomach and intestines. In experiments upon animals Falck found a notable diminution in the body temperature in acute antimonial poisoning, from 4°.4 to 6°.2 C. (7°.9 to 11°.2 F.).[1]

Distribution—Elimination.—By the copious and persistent vomiting and purging which it provokes itself, tartar emetic is in large part expelled soon after having been swallowed. Not only is tartar emetic taken by the mouth expelled by vomiting and purging, but even when it enters the circulation by other channels it is, like arsenic, eliminated by the gastric and intestinal mucous membranes. Brinton[2] found antimony in the stomach contents of a dog fifteen minutes after its injection into the femoral artery. The gastro-intestinal symptoms observed after external applications of tartar emetic are no doubt accompaniments of its elimination, as they are with arsenic. Owing to this action of the gastric mucous membrane we would expect that the tissue, at least, of that organ would contain antimony in cases in which death had followed the ingestion of a single poisonous dose within the average period of duration, even though there had been copious emesis. Yet in Ranke's case, in which the patient died in six hours, and in which the accused confessed to having adminstered 1 gm. (15.4 grains) of tartar emetic, Professor Buchner failed to find antimony in either the stomach or intestines or their contents.[3] With such results we should be disposed, in the absence of chemical examination of other portions of the body, rather to question the accuracy of the confession than to conclude with Buchner that a large dose had been taken. In two early cases, in one of which death followed an ingestion of tartar emetic in about

[1] "Lehrb. d. pr. Toxikol.," 104. See also Nobiling: Zeitsch. f. Biol., 1868, iv., 40. Soloweitschyk: Arch. f. exp. Path. u. Ph., 1880, xii., 438. Kobert: *Ibid.*, 1882, xv., 22.

[2] Lancet, 1853, ii., 599.

[3] Friedreich's Bl. f. ger. Med., 1879, xxx., 241.

ten hours, while in the other life was prolonged for more than two days, antimony was found in the stomach.[1] Unfortunately, as bearing upon this and other questions of elimination and distribution of antimony, we have no investigation by modern methods made in cases of accident or suicide; while to draw inferences from homicidal cases, in which the conditions of administration are not known but sought to be proved, is to argue in a circle.

That antimony is eliminated by the urine (which is usually increased in quantity) was shown by observations upon the human subject by Orfila,[2] and by Millon and Laveran.[3] The elimination begins very shortly after the ingestion. Wormley[4] mentions an instance in which antimony was found in the urine five minutes after "0.01 gm. of antimony in solution" had been taken. Millon and Laveran also noticed that there were intermissions in the elimination, during which the urine did not contain antimony, and that antimony was present in the urine in several cases sixteen, seventeen, eighteen, nineteen, twenty, and twenty-four days after the last administration. One of the two twenty-four-day patients died of phthisis, and antimony was found in the liver. That antimony may be detectable in the liver at longer periods after administration is shown by experiments upon animals; but for what period it may remain detectable in the human subject, whether after single or repeated administrations, is, so far as we know, not determined by observation. The statements of Chapuis that "if the doses of the poison have been repeated, it may still be characterized (in the cadaver) three months after death;"[5] and of Kobert, that "the metal (antimony) may remain stored up in the liver for several months,"[6] while valuable as opinions, do not, so far as we know, rest upon actual observation in the human subject.

We cannot agree with Chapuis in the opinion that the liver will not contain antimony if death have resulted rapidly from a single dose.[7] Antimony acts only after absorption, and absorption from the alimentary canal involves passage

[1] Ellis: Boston M. and S. Jour., 1856–57, lv., 400. Beale: Lancet, 1854, i., 68.

[2] "Toxicologie," 5ème ed., i., 619.

[3] Ann. d'hyg., Paris, 1846, xxxvi., 221.

[4] "Micro-chemistry of Poisons," 2d ed., 238.

[5] "Précis de toxicologie," 2ème ed., 189.

[6] "Intoxikationen," 266.

[7] See p. 302.

through the liver. While localization in the liver tissue may continue, and the maximum of saturation be only reached later, the poison, in its passage from the alimentary canal, must reach the liver within a few minutes of its ingestion, and probably remains present in that organ so long as any of it is in the body, or at all events until shortly before the last traces are in course of elimination by the kidneys.

Antimony also passes into the milk; not, however, in sufficient quantity to render the milk of cows, to which one of many antimonial preparations used in veterinary medicine is administered, dangerous to adults or even to children.[1]

Experiments on Animals.—Orfila in 1840,[2] experimenting with dogs, found that, after death from single large doses of tartar emetic given by the stomach, antimony was found in large amount in the liver and particularly in the kidneys, and only in small quantity in the spleen, lungs, and heart; and that antimony is also found in the liver and urine after endermic application.

Millon and Laveran[3] in 1846 administered three grains of tartar emetic in repeated doses during ten days to each of five dogs. In one, which died in six days, antimony was found in the liver, muscle, intestines, lungs, and brain. In another which died in thirteen days the brain contained a larger proportion than the other organs. In another which died in six weeks from another cause, antimony was found in notable quantity in the liver and fat, but particularly in the bones. The fourth was killed in three and one-half months, and, while the liver, bones, and other organs contained antimony, 50 gm. of the fat contained as much as 500 gm. of the other tissues. In the fifth, four months after the administration, antimony had accumulated in the bones; the liver also contained a large quantity, the other organs but little.

In 1856 Richardson[4] found antimony in the blood, vomit, rectum, lungs, liver, stomach, bladder, kidneys, and small intestines, in proportion diminishing in the order named, of a dog which died in one and two-thirds hours after the injection of ℥i. of tartar emetic in solution into the cellular tissue; while in another dog, killed in seven days by the daily application of tartar-emetic ointment to a wound, no antimony was found in the brain, but it was found in larger amount in the liver and spleen than in other organs.

Nevins in 1856[5] administered dry tartar emetic in doses of gr. ss., gr. i., and gr. ij. respectively, four times a day to each of twelve rabbits,

[1] Baum: "Ber. u. d. Veterinärwesen in Sachsen, 1892," 156.
[2] Tox. gén., 5ème ed., i., 618.
[3] *Loc. cit.*
[4] Lancet, 1856, i., 400, 508.
[5] Liverp. Med.-Chir. J., 1857, i.

which were killed from day to day until they began to die from the effects. Antimony was always present in the liver after gr. v. had been given, and it appeared in that organ before its presence was clearly proved elsewhere; it next appeared in the kidneys, and after fifteen days was present in the bones. Its presence was also easily proved in the blood, lungs, urine, and faeces. In the brain it was never clearly present, and its evidence in the muscles was very slight. Antimony was constantly passing off in the urine and faeces, and it was abundant in both in some rabbits which had survived twenty-one days after the last dose had been given; and in the faeces it was slightly present thirty days after the last dose. It disappeared from the liver before the fifteenth day, and from the kidneys somewhat later than this, whilst it was found abundantly in the bones thirty-one days after the last dose had been given.

The results of the experiments of Chittenden and Blake,[1] in which more perfect analytical methods were used, may be thus tabulated:

Mgm. in 100 gm.	I.	II.	III.	IV*a*	IV*b*.	V*a*	V*b*	VI*a*	VI*b*.
Liver	12.21	2.38	4.03	2.10	2.96	5.85	5.24	9.6	2.28
Brain	2.18	2.22	.....	3.63	4.4	1.51	0.5	.....	.....
Heart	2.18	1.47	.....	1.93	trace	0.66	0.76	2.22	.....
Lungs						0.56	1.28		
Stomach and intestine	1.08	2.04	.....	0.92[2]	15.3	.....	.....	.....	.....
Muscle	0.90	0.66	.....	0.45	1.11	1.06	0.45	0.91	0.9
Kidney	1.25	5.21	1.72	6.34	1.92	2.15	0.12	7.4	.....

I. Cat: 0.12 gm. tartar emetic, under the skin, died in two hours. II. Rabbit: 0.083 gm. tartar emetic, hypodermic in three doses, died in twenty-two hours after first. III. Cat: 0.15 gm. tartar emetic, hypodermic, died in four and one-half hours. IV. Two rabbits: *a.*, 0.8 gm. tartar emetic, hypodermic, in four hours ten minutes 0.8 gm. more, died twenty minutes later. *b.*, 0.08 gm. tartar emetic, in solution into rectum, in four hours five minutes 0.16 more by rectum. First injection, 11:45 A.M., died during night. V. Two dogs: *a.*, 0.762 gm. tartar emetic fed during seventeen days, two to three doses daily, no vomiting, killed by chloroform six hours after last dose. *b.*, 2.073 gm. antimony trioxid fed during seventeen days. Killed by chloroform eight hours after last dose. VI. Two rabbits: *a.*, 2.34 gm. tartar emetic fed in gradually increasing doses; died in seventeen days. *b.*, antimony trioxid, containing the same amount of antimony (1.08 gm.) fed during seventeen days; killed by chloroform.

The principal conclusions drawn by the authors from these experiments are that the brain has a decided tendency to absorb and retain

[1] Studies Lab. Sheffield Sc. Sch., Yale, 1887, ii., 68.

[2] Stomach and small intestines: the rectum and adjoining intestine contained 3.05.

antimony, even when administered as antimony trioxid, which is less completely absorbed than tartar emetic; that under the conditions in experiment II., the distribution is more even than when death occurs more rapidly, as in experiment I.; that the elimination of absorbed antimony proceeds somewhat rapidly, the proportion in the liver diminishing and that in the kidneys increasing relatively; and that the epithelial cells of the stomach and small intestines have a special absorptive action for antimony.

Treatment.

The best antidote is tannin, either in aqueous solution or in aqueous extracts of oak bark, cinchona, coffee, or tincture of cinchona, or other tinctures containing tannin. The purpose is the formation of the insoluble tannate. As this is, however, soluble in solutions of tartaric acid, that acid or substances containing it are to be avoided. The stomach should be washed out. Oils, mucilage, milk, albumin, are of some value but only mechanically. Pain is to be controlled by opiates, and heart failure to be combated by caffein, alcohol, ether, atropin, camphor, etc.

In chronic cases elimination is promoted by administration of iodids.

Post-Mortem Appearances.

The lesions caused by antimony closely resemble those due to arsenic.

A scarlatinal or vesico-pustular eruption is sometimes observed upon the skin in places, both in cases of short and of prolonged duration. Pustules and aphthæ are also found in the mouth, œsophagus, and stomach. Or the mouth and tongue are not sensibly altered, except that the former contains much viscid mucus, while the pharynx and œsophagus are coated with a dense, yellowish-white pseudo-membranous deposit, forming a hollow tube and filled with a reddish frothy mucus.[1] The mucous membrane of the stomach is marked with the pustules already mentioned, which appear to be the most characteristic lesion, although they may be absent and the stomach appear normal, as in a suicidal death which followed an ingestion of the poison in ten hours,[2] and in which no lesions were found

[1] Durand-Fardel: Bull. gén. d thér., 1843, xxv., 370.

[2] Ellis: Boston M. and S. J., 1856-57, lv., 400.

above the lower part of the small intestines; and in an accidental death after 0.15 gm. (2.3 grains) were taken.[1] Perforations of the stomach are never observed as the result of the action of tartar emetic and ulcerations only exceptionally.[2] The stomach may be empty, but usually contains considerable thick, grumous, or gruel-like reddish liquid.

Fatty degeneration of the liver, kidneys, heart, muscular tissue of the diaphragm and gastric follicles, first noted by Saikowski[3] in the chronic form, is a constant effect of the prolonged action of tartar emetic, and probably also exists in some degree in all cases. If slight in degree it is only recognized on microscopic examination.

The lungs are usually dark in color and are the seat of inflammation and hepatization; although this is not constant, as in Laveran's[4] case they were healthy, and rather anæmic than congested. As secondary appearances congestion of the dura mater, effusions into the arachnoid and ventricles, and congestion of the cerebral vessels may be noted.

Analysis.

The detection of antimony is a comparatively simple matter. The method followed is the same as that used in the search for arsenic (to the description of which in this volume we refer), except in certain points, some of which will be discussed here, others under "Arsenic."

The separation of antimony from arsenic and from tin is an essential step in the process. The *distinction* between antimony and arsenic is easy; their *complete separation*, when both are present, by no means so easy.

The method most generally used for their separation is by the Meyer fusion, based upon the extremely sparing solubility of sodium pyroantimonate and the ready solubility of sodium

[1] Gaz. d. hôp., Paris, 1853, xxvi., 147.

[2] In Le Boutillier's case *(loc. cit.)* the intestines are described as ulcerated, the muscular coat being deeply involved in the small intestine; the gastric mucous membrane inflamed, disorganized at the larger curvature, and, at the pylorus, suppuration of the follicles. And in one of Hartley's cases (Lancet, 1846, i., 460) there were two or three white spots in the gastric mucous membrane of the size of split peas which under a magnifying glass appeared to be beginning of ulceration.

[3] Arch. f. path. Anat., etc., 1865, xxiv., 73.

[4] Tardieu: "Empoisonnement," ed. 1866, 627.

arsenate in water and in alkaline liquids, which effects not only a complete destruction of any remaining organic material, but also serves to afford one point of distinction between antimony and arsenic, and to separate them almost completely from one another, if not completely. The precipitated sulfids left after evaporation of the solution in ammonium monosulfid (see p. 158) are first oxidized partially by fuming, chlorin-free nitric acid, first at the ordinary temperature, and then on the water-bath, the nitric acid being then removed in great part by moistening the residue with water and drying it on the water-bath several times. The residue is then treated with water and a small quantity of caustic soda (free from carbonate) to alkaline reaction; a suitable quantity of a mixture of one part of disodic carbonate and two parts of sodium nitrate (both free from chlorid and pulverized) is added, and the mass dried in a capsule. The completely dried residue is transferred to a porcelain crucible and very gradually heated to fusion. Under this treatment it turns brown or black at first and subsequently colorless or faintly greenish. Should it remain dark after continued fusion a further quantity of sodium nitrate is to be added and the fusion continued until the color is completely discharged, unless, indeed, the color be in a heavy black powder which readily settles to the bottom and which consists of cupric oxid. Any antimony present will now exist as sodium pyroantimonate, any arsenic as sodium arsenate, and any tin as stannic oxid. The cooled residue is then treated with a small quantity of water, with which it is warmed, and, if necessary, with further quantities of water, until the cake is completely dissolved, except possibly a small quantity of white, powdery material and some heavy black powder. The solution, whether clear or cloudy, is, after having cooled completely, treated with carbon dioxid for a few minutes to convert any sodium hydroxid possibly present into the carbonate, and thus precipitate a small quantity of stannic oxid which may have dissolved in the caustic alkali. The solution is separated from that which remains undissolved by filtration through a very small filter, and the undissolved residue is washed first with water and then with dilute alcohol. Arsenic passes into the solution, antimony and tin remain in the residue. To separate antimony from tin, should the latter be present, the dried filter and its contents are heated in a

weighed porcelain crucible until the paper is reduced to ash; small quantities of dry powdered potassium cyanid are added, and the heating continued to fusion of the cyanid and incipient redness. The material turns dark, and a gray powder or metallic globules, or both, consisting of antimony or tin liberated by reduction of their compounds by the cyanid, separate. Should copper be present it will appear with its characteristic color. The cooled, fused mass is extracted with water and transferred to a weighed capsule, in which it is washed by decantation—first with water, then with alcohol, and finally with ether—dried, and weighed.[1] A portion sometimes adheres tenaciously to the crucible, the weight of which may be determined. The washings when decanted are passed through a small filter, which with proper manipulation should remain perfectly clean. To detect the presence of tin a portion of the metal-like powder is treated with hydrochloric acid and warmed; tin is dissolved as stannous chlorid, antimony remains. The liquid is tested for tin by addition of a few drops to a small quantity of a dilute solution of mercuric chlorid. A white cloud or precipitate indicates the presence of tin. The residue, free from tin, is next warmed with concentrated nitric acid, evaporated to dryness, and the residue treated with water. The water separated from the undissolved material, which is now white, is tested for copper.[2] The remaining antimonial compounds are dissolved in a mixture of hydrochloric and nitric acids; the excess of nitric acid is expelled by warming over the water-bath, and the remainder is diluted with water. If a cloudiness be produced on dilution hydrochloric acid is added, and the solution so obtained is used for the application of the other tests for antimony.

Although the language of Otto would seem to indicate that by this method a complete separation of antimony and arsenic is attained,[3] such is not the case absolutely. Sodium pyroantimonate is not insoluble, but only extremely sparingly soluble, even in alkaline solutions; and as W. Fresenius has said,[4] small

[1] The washing, drying, and weighing should be performed as rapidly as possible.

[2] In the liver copper is almost invariably present, and is here washed out and quantitatively determined in part.

[3] "Ausmittelung der Gifte," 6te Aufl., 1884, 164. The latter part of the note modifies the statement in the text somewhat.

[4] Ztsch. f. an. Chem., 1881, xx., 536.

quantities of antimony pass into the aqueous solution. The quantity dissolved is, however, extremely small if the amounts of solvent and wash liquid are kept within proper limits; and of the processes hitherto suggested for the separation of antimony and arsenic, this yields the best results. Moreover, this method affords one important safeguard against the possibility for mistaking antimony for arsenic in the Marsh test, for if antimony be not present in quantity sufficient to leave an insoluble residue at this stage, it will also not be present in sufficient amount to respond to the Marsh.

Methods for the separation of antimony, arsenic, and tin, based upon the same fundamental facts as to solubilities as the Meyer method, in which, however, oxidation is effected at the ordinary temperature by hydrogen peroxid or by sodium peroxid, have been suggested. Trials of these, however, with various modifications have convinced us that they are in any form yet devised less manageable and less reliable than the Meyer method.

In ROSE'S METHOD,[1] which is more applicable to analyses of alloys, antimony and arsenic are brought into the same forms of combination as in Meyer's method by fusion with caustic soda in a silver crucible. The resulting mass is treated with hot water until that which is insoluble appears finely granular; a little water is added and enough alcohol of 0.83 specific gravity to make the proportion of alcohol to water 1 : 3. After standing twenty-four hours, with frequent stirring, the liquid is filtered off and the residue washed with alcohol of increasing strength, first 1 : 3, then 1 : 2, then 1 : 1, then 3 : 1, the washing being continued until a sample is not colored when acidulated with hydrochloric acid and treated with hydrogen sulfid. Antimony remains in the residue, arsenic and tin pass into the solution.

BUNSEN'S METHOD,[2] based upon the precipitation of antimony and tin from a solution of the three sulfids in potassium sulfid solution by sulfurous acid, while the arsenic remains in solution; and CLARK'S METHOD,[3] based upon the different solu-

[1] "Anal. quantit.," Paris, 1862, 557; and Fresenius: "Quant. Anal.," 6te Aufl., i., 634.

[2] Ann. d. Chem. u. Ph., cvi., 3. Fresenius: *Op. cit.*, ii., 636. See also Nilson: Ztschr. f. an. Chem., 1877, xvi., 417; 1879, xvii., 165, 264.

[3] Chem. News, 1870, xxi., 124. Fresenius *Loc. cit.*

bilities of the sulfids in oxalic acid solution and the different actions of hydrogen sulfid upon the solutions of those dissolved, are not so satisfactory or reliable as those above mentioned.

Or the separation may be effected by the MAGNESIA METHOD. The sulfids are oxidized by hydrochloric acid or otherwise; tartaric acid and a considerable quantity of ammonium chlorid are added, and then an excess of ammonium hydroxid. If a cloudiness be formed at this point the quantity of tartaric acid or of ammonium chlorid is insufficient. Magnesia mixture[1] is then added in sufficient quantity and the mixture allowed to stand forty-eight hours, when the precipitate is collected on a filter (using the liquid itself until all the solid is on the filter) and washed with a mixture of three parts of water and one of ammonium hydroxid, until the washings do not precipitate with nitric acid and silver nitrate. The filtrate and washings contain the antimony, the precipitate the arsenic.

This method is sometimes available in toxicological investigations, although more cumbrous than the Meyer method, and generally not to be preferred to it.

DAVY'S[2] OR NAQUET'S[3] METHOD and THIELE'S[4] METHOD aim at the separation of arsenic and antimony when existing in their gaseous hydrogen compounds. In the former the action of silver nitrate is taken advantage of. The arsenical compound, when decomposed by the silver solution through which it is passed, deposits elementary silver as a dark powder, while all of the arsenic remains in the solution as silver arsenite; but when the antimonial gas is similarly decomposed all of the antimony combines with the liberated silver, and is precipitated as silver antimonid. The separation of antimony from arsenic may then be effected by simple filtration and washing.

Thiele's method is based upon the fact that hydrogen antimonid is decomposed at a lower temperature than hydrogen arsenid. The mixed gases are passed through a coil of tubing heated to 208°–210° (406°.5–410° F.), beyond which antimony is deposited, while the arsenic is only separated beyond in a second tube heated to redness.

[1] Eleven parts of $MgCl_2$ and twenty-eight parts NH_4Cl, dissolved in one hundred and thirty parts H_2O, and seventy parts dilute NH_4HO added, and filtered after two days.

[2] Chem. News, 1876, xxxiii., 58.

[3] "Legal Chemistry," Battershall's transl., 2d ed., 34.

[4] Ann d. Ch., 1891, cclxiii., 361. Baumert: "Lehrb. d. ger. Chem.," 212.

While these methods may effect a complete separation of the two elements in a mixture of gases (and the first mentioned certainly does), neither these nor any other method based upon the formation of the hydrogen compounds by nascent hydrogen are applicable, as pointed out by Fresenius,[1] to the separation of arsenic from antimony in solutions, for the reason that "portions of the arsenic and antimony are not given off as hydrogen arsenid or antimonid, but remain in the generating vessel." So far as arsenic is concerned this objection is groundless, as it has been shown by Gautier,[2] and by Chittenden and Donaldson,[3] and repeatedly verified by others, that with proper precautions (see "Arsenic," Marsh Test) the totality of the arsenic introduced into the generator is evolved as hydrogen arsenid. But with antimony, in repeated experiments under the most varied conditions, we have never succeeded in regaining all of the antimony introduced, and in some instances the loss was as great as that observed by Reichardt,[4] of one-third of the amount introduced.

For the methods of SCHNEIDER AND FYFE, FRESENIUS AND VON BABO, MARSH, and RENISCH, see under "Arsenic."

Reactions of Antimony.—1. *Water* in sufficient amount produces in solutions of antimony trichlorid (solutions produced by the action of hydrochloric acid, either alone or in connection with potassium chlorate, or of nitric acid upon antimony or any of its compounds capable of solution) a white precipitate of antimony oxychlorid (powder of Algaroth). The precipitate redissolves readily in tartaric acid, and if this acid be added to the hydrochloric acid solution before dilution, the precipitate is not produced.

2. *Hydrogen sulfid* forms in acid solutions, if the quantity of free mineral acid be not excessive, an orange-colored precipitate, which usually consists of antimony trisulfid, Sb_2S_3;[5] which is not formed in alkaline solutions, and only to a slight extent in those which are neutral.[6] This precipitate dissolves readily in

[1] *Op. cit.*, i., 641.

[2] Bull. Soc. chim., Paris, 1875, xxiv., 258.

[3] Amer. Chem. Jour., 1880–81, ii., 238.

[4] Otto: "Ausmitt. d. Gifte," 6te Aufl., 198.

[5] The precipitate from solution of antimonic acid in hydrochloric acid is a mixture of the trisulfid and pentasulfid (Sb_2S_5) along with free sulfur. This precipitate is readily soluble in ammonium hydroxid.

[6] The presence of nitric acid or of other oxidizing agents is to be avoided.

solutions of potassium or sodium hydroxid, in solutions of the alkaline sulfids and hydrosulfids, in cold concentrated hydrochloric acid (specific gravity 1.18), and in the same acid still more dilute if hot. It is very sparingly soluble in ammonium hydroxid, and still less soluble in ammonium carbonate in the absence of antimony pentasulfid. It does not dissolve in dilute mineral acids nor in monopotassic sulfate solution.

The dried precipitate, when heated with sodium nitrate, yields sodium sulfate and sodium pyroantimonate, the latter almost insoluble in water. The pyroantimonate is also formed when the precipitate is fused with a mixture of sodium carbonate and nitrate. If the precipitate be heated in a current of chlorin, or ignited with a mixture of five parts of ammonium chlorid and one part of ammonium nitrate, it is completely volatilized. If the dried precipitate be heated with potassium cyanid to fusion, elementary antimony is produced as a gray powder or in metal-like globules, and potassium thiocyanate is formed. If the fusion be conducted in air or in a current of carbon dioxid, none of the antimony is volatilized; but if it be done in a current of hydrogen a metal-like mirror of antimony is formed in the tube near where the heat was applied.

3. *Ammonium sulfids* in small amount form precipitates of antimony trisulfid, which dissolve in excess of the precipitants.

4. *Metallic zinc* (or *tin* or *magnesium*) in the absence of nitric acid deposits elementary antimony as a gray powder. The solution should be so far concentrated that the greater part of the excess of acid is expelled, transferred to a platinum crucible cover, and a fragment of pure zinc added. The platinum surface moistened by the liquid is colored brown or black with small quantities of antimony, the stain disappearing when moistened with cold nitric acid. With cold hydrochloric acid (specific gravity 1.12) the stain soon disappears if it be faint, but if thick it only disappears slowly when heated. In this test hydrogen is generated and a portion of the antimony escapes as hydrogen antimonid. Tin is deposited from its solutions when they are subjected to this test, but it does not attach itself to the platinum.

5. *Metallic iron*, when added to a solution containing antimony and free hydrochloric acid and the liquid warmed, causes

the separation of the antimony as a heavy, black, flocculent deposit. Tin is not similarly deposited.

6. If a solution containing antimony be added to one of sodium thiosulfate to which a solution of sulfurous acid has been added, and the mixture boiled, it becomes cloudy from the separation of sulfur; afterward the red antimony pentasulfid (Sb_2S_5) is deposited.

7. *Potassium hydroxid*, *sodium hydroxid*, *ammonium hydroxid*, *disodic* or *ammonium carbonate* form voluminous, white precipitates much more slowly and incompletely from solutions of tartar emetic or similar compounds than from solutions of the chlorid. The precipitate dissolves quite readily in excess of sodium or potassium hydroxid, but is almost insoluble in ammonium hydroxid, and in disodic carbonate only when warmed.

8. For the action of antimonials with the Marsh and Reinsch tests, see under "Arsenic."

Quantitative Determination.—The determination of the quantity of antimony present is best attained by weighing the metallic antimony separated by reduction of the pyroantimonate by fusion with potassium cyanid as above described (see p. 318), or by electrolytic deposition.

Should tin or copper be also present their quantity is to be determined. To this end the alloy (or an aliquot part) is finely powdered, placed in a bulb tube through which a gentle stream of perfectly dry chlorin may be passed, and which is in connection with two U tubes of sufficient capacity, containing a solution of tartaric and hydrochloric acids in water. The bulb tube is heated, at first moderately, afterward to slight redness. The copper remains as chlorid in the bulb, the antimony and tin[1] volatilize as chlorids, which pass into the tube beyond the bulb and into the liquid in the U tubes. The bulb tube is cut near the bulb and between it and the U tubes, and any sublimate contained in it added to the liquid from the latter.

The cupric chlorid is dissolved in a small quantity of dilute hydrochloric acid, the solution transferred to a weighed platinum dish, a fragment of metallic zinc (known to be soluble in hydrochloric acid without residue) is added, and the dish covered with a watch glass, which is afterward washed into the

[1] A minute trace of tin remains with the copper.

dish. The proportion of acid should be sufficient to cause generation of hydrogen, but not too rapidly. With the quantities of copper usually present, the metal attaches itself firmly to the platinum, and may be washed, dried, and weighed, after the deposition is complete. If the quantity of copper be large it may separate partly as a non-adherent spongy mass. When this is the case, having made sure that no zinc remains, the deposit is rapidly washed by decantation with boiling water until the washings are free from chlorin, dried, and weighed.

Tin is only exceptionally present. Should it be, the quantity of antimony is determined in the liquid from the U tubes. To an aliquot portion hydrochloric acid is added to strongly acid reaction and a bar of tin, and the whole warmed for some time; the precipitated antimony is separated from the tin bar, collected on a weighed filter, washed in succession with dilute hydrochloric acid, alcohol, and ether, dried at 100° (212° F.), and weighed.[1]

The electrolytic determination gives very good results when conducted according to Vortmann's modification.[2] The precipitated sulfid is placed in a weighed platinum dish, along with a weighed quantity of mercuric chlorid containing something more than twice as much mercury as there is antimony in the sulfid. Warm water sufficient to dissolve the mercuric chlorid is added, and then Classen's sodium sulfid solution,[3] until the precipitate formed and the sulfid are completely dissolved, and the liquid is then diluted as far as the capacity of the dish will allow. The platinum dish is then connected with the negative pole of a weak battery (four moderate-sized gravity cells), while a platinum plate welded to a stout platinum wire and connected with the positive pole is immersed in the liquid. The current is allowed to pass until a sample of the liquid only gives a clear white precipitate with dilute sulfuric acid (about fifteen hours). The liquid is then poured off and the adherent steel-gray amalgam is washed, first with water, then with alcohol, finally with ether, dried by holding the dish in the hand and then in the desiccator, and weighed. From this weight, that of the dish, plus that of the mercury added, is subtracted, the difference

[1] There is a slight loss of antimony, which is not absolutely insoluble in dilute hydrochloric acid.

[2] Ber., Berl., 1891, xxiv., 2762.

[3] Ber., Berl., 1885, xxviii., 1109.

being the weight of antimony present. Tin if present is not deposited at the negative electrode.

For reasons already mentioned (see p. 321) quantitative determinations of antimony by the Marsh apparatus are valueless.

Antimony may also be determined as the sulfid or as the intermediate oxid. For the methods we refer to Fresenius and other works on quantitative analysis. It may also be determined as sodium antimonate.[1]

ARSENICAL POISONS.

The toxicology of the arsenicals, and particularly of the trioxid and of Paris green, overshadows in forensic importance that of any other class of poisons. The facility with which white arsenic, Paris green, and arsenical vermin poisons can be obtained, and the acquaintance of even the most ignorant with their destructive powers, sufficiently account for the frequent murderous administration of the former and for the favor in which the latter appears to be held by suicides in the larger cities.

Elementary arsenic, as well as such of its compounds, mineral or organic, as are either soluble in water or are rendered soluble by the action of the liquids of the body, are capable of causing arsenical intoxication when introduced by any channel into the human body.

ELEMENTARY ARSENIC.

The element (symbol = As, atomic weight = 74.9) occurs in small quantity in nature, and is usually met with as a dull black or dark-brown amorphous material. When freshly isolated it constitutes a brittle, crystalline, steel-gray solid, having a metallic lustre, which it soon loses on exposure to air. When deposited upon and viewed through a glass tube, as in the reduction and Marsh tests, it forms a brilliant black "mirror." It is tasteless and odorless. Its specific gravity is 5.7 to 5.9. When heated in an atmosphere of an indifferent gas at the ordinary pressure, it volatilizes without fusion at 180° (356° F.). When heated under strong pressure it fuses at a dull-red heat.

[1] Beilstein and v. Blaese: J. Chem. Soc., London, 1890, lviii., 830.

It burns in air with a bluish-white flame, giving off a dense, heavy, white cloud of arsenic trioxid. It also ignites spontaneously and burns in chlorin with formation of the trichlorid. It is insoluble in water and in other neutral solvents if oxidation be prevented. It is dissolved by nitric acid with formation of arsenic acid, and in hydrochloric acid with formation of arsenic trichlorid.

Although elementary arsenic, so long as it remains such, is insoluble and therefore incapable of absorption from the alimentary canal, there is abundant evidence of its poisonous character after exposure to the air. The early experiments of Bayen and of Renault[1] showed that while dogs suffered no ill-effects from taking from 4 to 8 gm. of metallic arsenic, or of mispickel (sulfid of iron and arsenic), they were fatally poisoned by 0.2 to 0.3 gm. of arsenic which had been exposed to the air. The poisonous character of arsenic under these conditions is further established by numerous experiments upon animals (Sprögel, Hilefeld, Monro, Schroff), and by cases of poisoning in the human subject. Arsenic when exposed to moist air loses its metallic lustre and is converted into a dull-brown powder (*mort aux mouches*, *cobalt*) by a limited oxidation.

The experiments of Schroff[2] seemed to prove that unoxidized, elementary arsenic is poisonous to rabbits, probably by undergoing oxidation in the alimentary canal, causing death in twenty-eight to forty-two hours, in doses of 0.6 to 1 gm. Schmidt and Bretschneider,[3] however, from experiments made at about the same period, consider the element as innocuous, and question the purity of the arsenic used by Schroff.

The more recent experiments of Paschkis and Obermayer,[4] made with great care to insure the prevention of atmospheric oxidation, show conclusively that elementary arsenic may be absorbed by the skin; that it undergoes oxidation in the body; that it produces gastro-enteritis, nephritis, great thirst, and eczema; and that it is eliminated in solution in the urine.

The "fly poison," "fly stone," "cobalt," and "fly paper," (*mort aux mouches*, *poudre aux mouches*, *fliegenstein*, *cobal-*

[1] Orfila: "Toxicologie," 5ème ed., pp. 377, 611.

[2] Zeitsch. d.k.k. Gesell. d. Aerzte z. Wien, 1858, xiv., 3; 1859, xv., 689.

[3] Moleschott: "Untersuch. zur Naturlehre," 1858, vi., 146–147.

[4] Med. Jahrb., Wien, 1888, n. F iii., 117–124.

tum minerale), formerly used, owed its efficacy to elementary arsenic, partially oxidized by the action of air and moisture.

Of twenty-two cases of poisoning by fly poisons certainly containing elementary arsenic, seven were homicidal, two suicidal, six accidental, and seven uncertain. The accidental and uncertain cases occurred in four instances in children,[1] and in the others in consequence of the poison having been accidentally mixed with articles of food or taken in mistake for medicine. In three instances the cause of the poisoning of several individuals was found in the existence of elementary arsenic in bottles from which wine or spirits had been taken. It is possible that in some of these the arsenic may have been originally put into the bottles as a constituent of the shot used in cleaning them (see below). In one case all the persons in the house of a priest suffered from arsenical poisoning, and upon investigation "*mort aux mouches*" was found in the bottom of a bottle of brandy from which they had drunk.[2] In a second case[3] six persons were severely poisoned and one died. Elementary arsenic was found in the bottle from which they had drunk wine. In another case[4] five persons were similarly poisoned with wine, which had been eight months in a bottle in the bottom of which elementary arsenic was found. An experiment in this instance showed that wine was capable of dissolving elementary arsenic to the extent of about 1.5 parts in 1,000 of white arsenic during a contact of twenty-four hours.

A man drank beer in which there were the bodies of flies killed by "cobalt." In two hours he was attacked with colic, nausea, and vomiting, and later with diarrhœa, headache, insomnia, and prostration; but recovered slowly after the administration of an emetic.[5] No other instance of serious poisoning from this cause has been found. Payen[6] fed three hundred flies, poisoned by solution of white arsenic, mixed with bread crumbs to a spaniel, which was not at all incommoded. An

[1] Leger: Lancet, 1835, i., 516–518. Evans: Nelson's Amer. Lanc., 1853, vii., 180. Hurd: Boston M. and S. Jour., 1845, xxxi., 316. Schobbens: Ann. Soc. de méd. d'Anvers, 1846, 224.

[2] Galtier: "Tox.," i., 446.

[3] Rapp: Tr. Soc. d'émul., Rouen, An vii.

[4] Batillat: J. d. chim. méd., etc., 1840, 2 s., vi., 33.

[5] Grimm: "Misc. Ac. nat. cur.," Lips., 1699–1700, Dec. iii., Ann. 7–8, p. 292.

[6] J. de chim. méd., Paris, 1825, i., 196.

equal number of poisoned flies similarly fed to a hen provoked no symptoms of poisoning. He therefore considered that it would be impossible for a human being to involuntarily take a sufficient number of poisoned flies to cause poisoning.

The use of elementary arsenic to kill insects has also caused serious poisoning in the human subject in consequence of the false impression that it is only fatal to insect life. The members of two families suffered from severe arsenical poisoning, but ultimately recovered, in consequence of eating cheese in the manufacture of which "*mort aux mouches*" had been used to prevent the appearance of "skippers."[1]

Bucholtz relates an instance in which a family of six adults and a child were seized with nausea, pain, vomiting, and other symptoms of arsenical poisoning within an hour after partaking of a soup in which "cobalt" had been cooked.[2] He does not state whether the addition was intentional or accidental. Galtier[3] cites the case of a woman who was seized with vomiting and died in ten hours. The stomach contained about 180 gm. of a sanguinolent liquid and 15 gm. of "black oxid of arsenic" (elementary arsenic). He also mentions another instance in which three persons died in a few hours after violent pain and vomiting from taking "*cobalt mineral arsenical.*" The stomachs were highly inflamed and contained a sanguinolent liquid. Schobbens[4] relates the case of a man who died after exhibiting the usual symptoms of arsenical poisoning, in consequence of having taken "fly poison" by mistake for a purgative. The "fly poison" taken in the two cases reported by Schobbens (see above) was the powdered "Tunaberg ore," which is a compound of cobalt, arsenic, iron, and sulfur.

The earliest case of homicidal poisoning by elementary arsenic of which we find record occurred in 1740.[5] A girl poisoned her father and three sisters by serving to them dried pears boiled with water and 24 gm. of "cobalt." The deaths of the father and two sisters occurred in thirteen, nine, and eighteen hours; that of the other sister, who ate but little of the poisoned pears, on the sixth day. The stomachs were highly inflamed, and in two cases contained extravasated blood.

[1] Gollier: J. d. chim. méd., etc., Paris, 1844, 2 s., x., 571.

[2] J. d. pr. Arzneik u. Wissensch., Jena, 1797–98, v., 375.

[3] "Toxicologie," i., 446, 447.

[4] *Loc. cit.*

[5] "Misc. Ac. nat. cur.," Lips., v., obs. 102, p. 355.

Hahnemann[1] relates the case of a wood-cutter who was poisoned by his wife in 1784 by several grains of fly stone. He suffered from vomiting, burning pains in the stomach, and tearing pains in the bowels, but recovered. Orfila, Chevalier, and Barruel,[2] in examining the stomach in a case of supposed homicide, found about 5 gm. of a powder, consisting of elementary arsenic, oxid of iron, quartz sand, and flakes of mica; but state that the contents of the stomach contained no trace of arsenious acid. Briand, Chaudé, and Bouis[3] state that a case of supposed homicide by "*mort aux mouches*" was tried in France in 1844, but give no particulars. Aguilhon[4] reports a case of homicidal poisoning by fly poison which terminated fatally, after the symptoms usual in arsenical poisoning, on the sixth day. Wormley[5] cites a case of criminal poisoning by fly powder in which death took place in thirty-six hours, and in which a quantity of arsenic equivalent to forty-two grains (2.7 gm.) of arsenious acid remained in the stomach at the time of death, although there had been almost incessant vomiting for over thirty hours. The classic case of homicidal poisoning by elementary arsenic is that of Dombrowsky, reported by Schütte.[6] The accused was convicted of the murder of his wife by the repeated administration of fly stone. The stomach contained at death nearly twenty-four grains (1.5 gm.) of arsenic, partly in the form of elementary arsenic, partly in that of the trioxid. The mucous membrane of the stomach was softened, reddened in streaks, excoriated near the cardia, where there were also a few spots of extravasated blood. Attached to the walls of the stomach were small black bodies, some possessed of a metallic lustre, which were found to consist of finely powdered elementary arsenic. A similar powder was also found in the pockets of a night-dress worn by the prisoner. The deceased survived for six days after the date of the first alleged administration, during which she suffered from the usual symptoms of arsenical poisoning.

A suicidal case is reported by Patissier:[7] that of a girl of

[1] "Die Arsenvergiftung," Leipzig, 1786, p. 57, note.

[2] J. d. chim. méd., Paris, 1839, 2 s., v., 3–16.

[3] "Méd. lég.," 7ème ed., Paris, 1874, p. 473.

[4] Ann. d'hyg., Paris, 1851, xlv., 159–181.

[5] "Micro-Chemistry of Poisons," 1st ed., p. 239.

[6] Viertljschr. f. ger. u. öff. Med., 1854, vi., 230–293.

[7] "Bibl. méd.," 1827, ii., p. 59.

nineteen years who died in sixteen hours after taking an unknown dose of "fly poison." Jäger[1] relates the case of an adult female who poisoned herself with water which had stood in contact with "black oxid of arsenic" (elementary arsenic). She suffered no pain and remained conscious till near her death.

Pluskal[2] reported two cases in which wounds caused by small shot provoked symptoms not accountable for by the mechanical injury, and which he attributed to the poisonous action of the arsenic contained in shot. Shot metal consists of lead to which about two per cent. (forty pounds to the ton) of arsenic are added. Each ounce of shot therefore contains about nine grains of arsenic.

Tin is frequently contaminated with arsenic in small amount. In St. Petersburg several persons suffered from gastro-intestinal disturbances, accompanied by fever, nausea, vomiting, lassitude, prostration, and emaciation, from the use of food prepared in vessels tinned with an English tin containing 0.05 to 0.1 per cent. of arsenic and only one per cent. of lead.[3]

HYDROGEN ARSENID.

This substance, also known under the names *arseniuretted* or *arsenetted hydrogen*, *arsonia*, *arsenamin*, and *arsin*, has the formula AsH_3. It is produced: 1. By the action of water upon an alloy obtained by fusing together native sulfid of antimony 2 parts, cream of tartar 2 parts, and arsenic trioxid 1 part. 2. By the action of dilute hydrochloric or sulfuric acid upon the arsenids of zinc and tin. 3. Whenever a reducible compound of arsenic is in presence of nascent hydrogen (see Marsh test). 4. By the action of water upon the arsenids of the alkali metals. 5. By the combined action of air, moisture, and organic matter upon the arsenical pigments (?). 6. By the action of hot solution of potash and powdered zinc upon reducible compounds of arsenic.

It is a colorless gas, having a strong odor of garlic; soluble in five volumes of water free from air; neutral in reaction. It is partially and slowly oxidized by air and moisture, with depo-

[1] "Diss. de effectibus arsenici," etc., Tüb., 1808.

[2] Oest. med. Wchnschr., etc., 1843, iii., 505, 507.

[3] Ann. d'hyg., etc., 1890, 3 s., xxiv., 113.

sition of elementary arsenic. It is decomposed into hydrogen and arsenic when in contact with a red-hot surface, and by the passage through it of luminous electric discharges (see Marsh test). Its mixtures with air or oxygen are explosive. It burns in air with a greenish flame from which a white cloud of arsenic trioxid arises. A cold surface held above this flame becomes coated with a white, crystalline deposit of the trioxid. If the flame be cooled by the introduction of a cold surface into it, the hydrogen alone is oxidized and the arsenic is deposited upon the cold surface. Chlorin decomposes hydrogen arsenid explosively, with formation of hydrochloric acid and arsenic trioxid. Bromin and chlorin behave similarly but with less violence. Active oxidizing agents convert it into water and arsenic acid; less powerful oxidants into water and arsenic trioxid. Solid potassium hydrate decomposes it partially, and becomes coated with a dark deposit, which seems to be elementary arsenic. Solutions of the alkaline hydroxids absorb and decompose it, hydrogen being given off and an alkaline arsenite remaining in solution. It is reduced by solution of silver nitrate, metallic silver being deposited, and silver arsenite remaining in the solution. Hydrogen arsenid and hydrogen sulfid do not mutually decompose each other in the absence of air.

Hydrogen arsenid is the most violently poisonous of the mineral compounds of arsenic, partly by reason of the large proportion of arsenic which it contains (about ninety-six per cent.), and partly because its absorption by the pulmonary surfaces is more rapid than that of dissolved arsenicals by the alimentary canal. Van Hasselt[1] and other toxicologists have considered hydrogen arsenid apart from the other arsenicals as a poison *sui generis*, because of its much greater activity in minute doses and because of certain differences between its symptomatology and that of other compounds of arsenic. Its solubility in water is not sufficiently great to account for its absorption, and it probably enters into combination with some constituent of the blood. The amount of arsenic capable of causing death when absorbed as hydrogen arsenid is much less than that required in the other forms of combination. According to Brandes the quantity inhaled by Gehlen (see below) could not have exceeded one-one-hundredth grain. In the case re-

[1] "Allg. Giftlehre," 1862, 390.

ported by Schindler [1] the amount inhaled was greater, although it probably did not exceed one-eighth grain of arsenic.

We have collected reports of thirty-eight cases of poisoning by hydrogen arsenid, all of which were accidental. Twelve cases, of which eight were fatal, were those of chemists who inhaled the gas more or less diluted with hydrogen. The other twenty-six cases occurred among operatives engaged in chemical industries in which hydrogen is liberated from the action upon each other of chemicals contaminated with arsenic. Eighteen of the cases terminated in death.

The earliest recognized case of hydrogen arsenid poisoning was that of the chemist Gehlen in 1815. While experimenting upon the action of a hot concentrated solution of potash upon arsenic trioxid, and the action of water upon the solid residue of the reaction, he inhaled a small quantity of the gas in order to detect the presence of hydrogen arsenid by its odor, and died nine days subsequently. In 1819 Professor Emmert, of Tübingen, lost his life by inhaling hydrogen arsenid during a chemical experiment.[2] Taylor [3] cites a case which occurred in England in 1836, that of a young chemist who died twenty-four days after inhaling a small portion of the gas evolved from a mixture of arsenic, zinc, and sulfuric acid.

Schindler [4] gives a full account of the poisoning of his brother, aged thirty-two, who, in 1836, inhaled about half a cubic inch of gas containing at most 0.008 gm. (one-eighth grain) of metallic arsenic during forty minutes. No ill effects were observed for three hours, when he experienced vertigo in going upstairs (less on a level and none on a descent), later a sense of pressure in the region of the kidneys, which extended over the back and shoulders, which soon became severely painful. At the same time there were chilliness of the surface, gouty pains in the joints, and cold extremities. The pains in the back increased in severity, and were accompanied by cutting pain in the abdomen, vomiting, great thirst, an almost imperceptible pulse, and strangury. The urine passed was almost black, contained blood, and coagulated in the vessel. The skin was dark brown, and the hands, feet, and nose were insensible. The eyebrows were bleached white upon the second day. On the third day the symptoms abated, but hiccough was almost continuous. The patient gradually improved during six days, and the eyebrows resumed their natural color (brown), but he complained of a sensation as of a stone in the body. In the

[1] Rept. f. d. Pharm., 1840, lxix., 271–276.

[2] Müller: Ann. d. Staatsarznk., 1837, ii., 422.

[3] "Poisons," 3d Am. ed., 350.

[4] J. f. Chir. u. Augenhlk., 1838, xxvi., 626; also Mag. f. Phys. u. kl. Arznw. u. Tox., 1846, i., 364.

third week the prepuce and glans were covered with purulent vesications, which collapsed, leaving small round ulcers, which healed in ten to twelve days. He recovered in seven weeks. Schindler had much experience with hydrogen arsenid, was fully aware of its danger, and was possessed of an extremely acute sense of smell, upon which he relied.

Dr. O'Reilly[1] has given a full report of the case of Mr. Brittain, of Dublin, who, experimenting upon the physiological effects of hydrogen gas, inhaled a quantity, made from impure materials, estimated to have contained an amount of hydrogen arsenid equivalent to twelve grains of arsenic. He died upon the seventh day thereafter, having exhibited the usual symptoms.

An Italian chemist, Bietani, met his death in 1846 under circumtances almost identical with those of the Brittain case.[2]

Vogel[3] cites another similar case of a man who inhaled air containing hydrogen and some hydrogen arsenid, and was shortly attacked with symptoms of poisoning, but who recovered. The urine was almost black, coagulated on boiling, but was devoid of blood corpuscles.

The case of Professor Robertson of the Calcutta Medical College is reported by Mouat.[4] While lecturing, he inhaled a small quantity of the gas escaping from an imperfectly fitted Marsh apparatus. He only recovered after twenty-six days of severe illness.

Ollivier[5] describes the case of a man of twenty-two years who, experimenting with anilin colors, inhaled hydrogen contaminated with hydrogen arsenid, suffered from violent headache, vomiting, severe lumbar pain, feeling of constriction in the chest, thirst, cold extremities, jaundice, suppression of urine and hæmoglobinuria, and who died upon the fifth day.

Professor X. and two of his pupils were severely poisoned in consequence of inhaling hydrogen, generated from arsenical materials, for the purpose of repeating Tyndall's experiments with regard to the influence of the gravity of the gas upon the voice. They ultimately recovered.[6]

Dr. S——, a teacher of chemistry, was fatally poisoned by inhaling hydrogen made from commercial zinc and hydrochloric acid.[7]

We have found record of twenty-six cases of poisoning from the generation of hydrogen arsenid in industrial processes.

[1] Dublin Jour. Med. Sc., 1841–42, xx., 422–433.

[2] Ferré: "Arsenicisme professionel," Paris, 1882, 24.

[3] Arch. d. Ver. f. gemeinsch. Arb., 1854, i., 209.

[4] Ind. Ann. Med. Sc., Calcutta, 1856–57, iv., 657–660.

[5] C. r. soc. d. biol., Paris, 1863, 3 s., v., 77; also Gaz. des hôp., 1863, xxxvi., 509.

[6] Eitner: Berl. kl. Wchnschr., 1880, xvii., 256.

[7] Boström: Med.-chir. Rundschau, 1886, xvii., 325.

Commercial sulfuric and hydrochloric acids are always contaminated with arsenic, and consequently, whenever these acids are decomposed with liberation of hydrogen by the action of a metal, particularly if the metal itself contain arsenic, the conditions for the generation of hydrogen arsenid are fulfilled.

In the manufacture of green vitriol, blue vitriol, tin crystals, zinc chlorid, and zinc sulfate the acid is neutralized with scrap metal and hydrogen is liberated. Sury-Bienz[1] has reported the case of a workman, aged forty-eight years, who died upon the seventh day after inhaling the gas given off in the manufacture of zinc chlorid from commercial hydrochloric acid and waste zinc. Müller[2] describes the case of a man who manifested all of the symptoms of poisoning by hydrogen arsenid after exposure to the gas disengaged in the manufacture of tin salt, and who made a slow recovery. Martin[3] has reported the fatal poisoning of a man of thirty-one years engaged in the manufacture of zinc chlorid from arsenical materials.

In the anilin industry arsenical compounds are used and advantage is taken of the action of nascent hydrogen produced by the action of iron turnings upon sulfuric or hydrochloric acid. Coester[4] reports two cases, one terminating in death the other in recovery, in workmen in an anilin factory. Sury-Bienz[5] describes the simultaneous poisoning of five workmen employed in the manufacture of anilin blue, in which process arsenical zinc and hydrochloric acid were used. Valette[6] describes two cases, one terminating in recovery, the other in death in twenty-eight days, caused by the use of a concentrated solution of arsenic acid in mistake for sulfuric acid for the generation of hydrogen in an anilin factory.

Trost[7] gives the particulars of nine cases, three of which terminated fatally, which occurred at a trial of a method for desilvering lead, in a smelting works at Aix-la-Chapelle. In one stage of the process an alloy of zinc, silver, and a small quantity of arsenic was treated with commercial hydrochloric acid (afterward found to contain 0.027 per cent. of arsenic). The apparatus was consequently simply an enormous Marsh generator.

Not only are workmen in strictly chemical industries, such as those mentioned, liable to hydrogen arsenid poisoning, but also those engaged in mechanical arts in which impure acids are used for cleaning

[1] Viertljschr. f. ger. Med., 1888, n. F., xlix., 349.
[2] Ann. d. Staatsarznk., 1837, ii., 418.
[3] Med. Chron., Manchester, 1893–94, xix., 108.
[4] Berlin kl. Wchnschr., 1884, xxi., 119-121; 1886, xxxiii., 209.
[5] Vrtljschr. f. ger. Med., 1888, n. F., xlix., 353.
[6] Lyon méd., 1870, iv., 440–454.
[7] Vrtljschr. f. ger. Med., 1873, n. F., xviii., 269–279.

or other purposes. Dr. W. S. Barker[1] reports the case of a workman in a foundry who was engaged in cleaning castings with sulfuric acid, and who was severely poisoned, but recovered after ten days. Waechter[2] describes at length the cases of four Italians whose trade it was to manufacture toy balloons, filling them with hydrogen prepared from commercial materials. They worked at filling the balloons for five hours, at intervals during a day, in a small room. All suffered from violent symptoms of poisoning. Three were discharged, recovered in fourteen, fifteen, and seventeen days. The fourth died upon the tenth day.

Numerous instances of the occurrence of chronic, and even of acute, arsenical poisoning in persons inhabiting rooms whose walls are painted or covered with paper colored with arsenical colors (see "Arsenical Pigments," p. 487) have been recorded. The poison can find its way from the wall to the system of the inhabitant in one of two ways: either by mechanical detachment of minute fragments which float in the dust of the room, or by the generation of a gaseous or volatile compound of arsenic. That particles of the pigment become detached, particularly when the paper is new and when its surface is rough, cannot be questioned. But poisoning has also been observed and arsenic has been detected in the air of rooms which have been papered with arsenical paper for twenty-five and thirty years, and in which the first paper was covered with a second containing no arsenic, as well as in rooms in which the walls were painted with an arsenical pigment. The experimental results also of Fabian,[3] Müller,[4] Kletzinski,[5] Hamberg,[6] Fleck,[7] Sonnenschein,[8] and Bischoff[9] prove conclusively that an aeriform arsenical compound is given off from paints and papers containing arsenic, particularly when the walls are damp and the paste becomes mouldy. The nature of the gaseous arsenical produced under these conditions is not known as yet. It has been supposed to be hydrogen arsenid or cacodyl oxid (dimethyl arsin oxid). The supposition of Fleck that hydrogen arsenid is pro-

[1] St. Louis M. and S. Jour., 1864, n. s., i., 29–31.
[2] Vrtljschr. f. ger. Med., 1878, n. F., xxviii., 251–261.
[3] Arch. der Pharm., 1860, ix., 433.
[4] Wien. m. Wchnschr., 1860, x., 277; 292; 308; 324.
[5] Wien. med. Wchnschr., 1859, 43.
[6] Pharm. J. and Tr., London, 1874, 3 s., v., 81.
[7] Viertjschr. f. ger. Med., 1873, xviii., 391.
[8] "Handb. d. gerichtl. Chemie," 1869, 153.
[9] Repert. f. an. Chem., 1883, iii., 310.

duced by the reducing action of the nascent hydrogen produced by the growth of mould organisms upon the arsenic trioxid, which always exists in arsenical greens, is quite plausible. In wall-paper poisoning, even in severe cases, however, two of the prominent symptoms of hydrogen arsenid poisoning, jaundice and hæmoglobinuria, are conspicuous by their absence. The odor observed in rooms with arsenical walls is, moreover, not the garlic-like odor of hydrogen arsenid, but is of a musty character, sometimes described as "mouse-like." On the other hand, the reducing action of the gaseous arsenical from wall papers upon silver nitrate solution is inconsistent with the supposition that it is cacodyl oxid, a substance which does not reduce the silver salt, but combines with it to form a crystalline compound.

The symptomatology of poisoning by hydrogen arsenid differs in several essential respects from that of the solid arsenical poisons. The duration of the poisoning, whether it terminate in recovery or in death, is materially longer. The shortest period of complete recovery was in Valette's case, eight days; while in many cases several weeks or months elapsed before the patient was discharged. The shortest time in which death occurred was upon the third day (in two of Trost's cases), and the longest twenty-eight days (in Valette's second case). The average duration of eleven fatal cases was ten days and four and one-half hours. About one-half of the deaths from poisoning by white arsenic occur within the first twenty-four hours, and cases in which the duration of the poisoning is prolonged for two or three days are exceptional.

The onset of a poisoning by white arsenic is, in the great majority of cases, sudden, beginning with a violent attack of vomiting and abdominal pain, and occurs within one hour. In poisoning by hydrogen arsenid abdominal pain and vomiting are either absent or occur later in the history of the case. In most cases the attack begins with a feeling of faintness and giddiness, particularly on going upstairs, pain in the head, chills more or less severe, and pain in the kidneys. Occasionally, as in one of Valette's cases, nausea and vomiting are early symptoms. In this case the workman experienced nausea at the time of the exposure, and going out to vomit, urinated, and was struck by the dark-red color of his urine. In Boström's case

the attack is described as apparently one of renal colic, but the urine was dark-brown, almost black, in color, and the skin was copper-colored.

The three prominent symptoms of poisoning by hydrogen arsenid, which are not observed in poisoning by the other arsenicals, are lumbar pain, hæmoglobinuria, and icterus.

Lumbar pains occur early and are severe in most cases, although in some instances they were not observed, as in four of Trost's nine cases and in Eitner's case. Almost pure hæmoglobinuria occurs in all cases. The urine passed is bright-red, dark-brown, or black in color, coagulates almost solid by heat, and on standing deposits a very sparing sediment, which contains no blood corpuscles, but sometimes granular matter, the product of their disintegration. The quantity of urine passed is usually normal or somewhat increased at first, while afterward there is marked diminution or complete suppression. In Coester's first case, which terminated in death in nine days, the elimination was 1,500 c.c. (50¾ fluid-ounces) on the first day; while in the succeeding five days the total elimination was only 24 c.c. (6½ fluidrachms), or 5 c.c. (1⅓ fluidrachms) in twenty-four hours; and on the sixth day 4 c.c. (1 fluidrachm). Or the suppression may begin at the outset. Thus in O'Reilly's case, on the first day the patient passed 60 c.c. (2 fluidounces) of blood, on the second 15 c.c. (4 fluidrachms) of blood, and on the fifth, sixth, and last days no urine was obtained by the catheter, and the breath was strongly ammoniacal. In cases which terminate in recovery the blood pigment gradually disappears from the urine, which resumes its natural color and increases in quantity. In Coester's second case the elimination during the nine days was 2,000 c.c. (67½ fluidounces), 2,500 c.c. (84½ fluidounces), 2,500 c.c., 3,500 c.c. (118 fluidounces), 3,000 c.c. (101½ fluidounces), 2,800 c.c. (94½ fluidounces), 2,900 c.c. (98¼ fluidounces), 4,500 c.c. (152 fluidounces), and 4,500 c c. The urine contains methæmoglobin, oxyhæmoglobin, and traces of bile pigment. In some cases other manifestations of extensive disorganization of the blood are observed. In Valette's second case, which terminated in death in twenty-eight days, there was almost complete suppression of urine, a very small amount of black urine only being passed during the first twenty days, when there were sanguinolent diarrhœal

stools, hemorrhages from the gums, buccal mucous membrane, glans penis, and prepuce, the blood being pale and escaping rather by leaking than by bleeding. At the same time the skin was covered with an extensive papular eruption. On the twenty-first day the urine was abundant and clear. On the twenty-eighth day the patient vomited half a glassful of black blood, had two attacks of syncope, and died quietly in the afternoon. In one case in which the blood was examined microscopically (Sury-Bienz), it was found to be very poor in corpuscles, but contained much detritus of their disintegration.

Jaundice is an unfailing symptom. The only case whose symptoms have been described in which it is not mentioned is that recorded by Müller (1837), in which there was general subcutaneous emphysema. The face was pale, bluish, livid; the left arm and left leg were particularly swollen and covered with innumerable large and small petechial spots. In some cases the conjunctivæ and skin became yellow on the second day, the color of the skin deepening to a bronze or copper, which gradually diminished toward the fifth or sixth day, and the conjunctivæ become brownish-red from a combination of icterus and injection. In other cases jaundice is among the first symptoms to appear. Thus in Trost's cases the workmen noticed the yellow color of each other while at work, and in Valette's two cases the sclerotic was observed to be yellow one hour after the exposure. The alvine discharges are dark brown-black, liquid, and in some cases consist of almost pure bile.

The experiments of Stadelmann [1] have demonstrated that the icterus of hydrogen arsenid is hepatogenous, and is due to the production by the liver of a bile so modified in composition that it is peculiarly fitted for absorption. He considers, however, that the fundamental cause of the icterus is in the disorganization of the blood.

The post-mortem appearances after death from hydrogen arsenid differ also in some respects from those caused by white arsenic. The surface is dirty yellow or bronzed in color, and all the tissues are more or less yellow. The liver is normal in size or somewhat enlarged; in some cases of a deep indigo color and congested, in others yellow, gray, or slate-colored, and not congested, or even anæmic. The gall bladder

[1] Arch. f. exp. Path. u. Pharm., 1883, xvi., 221–255.

always contains much bile and is sometimes greatly distended. The kidneys are highly congested, dark brown-red or indigo in color, the surface sometimes almost black, and enlarged. The mucous membrane of the stomach is yellow or slate-colored, and evidences of inflammation were absent in all cases except in that reported by O'Reilly, in which death occurred in six days three and one-half hours, and in which there were two distinct inflammatory patches at the greater curvature.

ARSENIC TRIOXID, OR WHITE ARSENIC.

Physical and Chemical Properties.

This substance, the common arsenic of the shops, is also known under the names *arsenious acid* and *arsenious anhydrid*. Its formula is As_2O_3 (molecular weight=197.68);[1] one hundred parts therefore contain As 75.78 and O 24.22, and one part of elementary arsenic represents 1.32 As_2O_3. It is obtained industrially by roasting the native sulfid of arsenic and iron (arsenopyrites, mispickel) in a current of air.

It exists in three forms: *vitreous*, *porcelainous*, and *crystalline*. It is odorless, whether in the form of solid or vapor. (For taste and solubility, see below.)

Aqueous solutions of arsenic trioxid are acid in reaction, and probably contain the true *arsenious acid*—H_3AsO_3. They are neutralized by the alkalies and by alkaline carbonates, as well as by bases in general, with the formation of salts called *arsenites*. Consequently solutions of white arsenic in solutions of potassium or sodium carbonate do not contain "arsenious acid," but potassium or sodium arsenite.

Arsenic trioxid is readily reduced, with separation of elementary arsenic, when heated with hydrogen, carbon, potassium cyanid, or other reducing agents. Oxidizing agents, such as nitric acid, the hydrates of chlorin, and chromic acid, convert it into *arsenic pentoxid*, As_2O_5, or *arsenic acid*, H_3AsO_4. Its solution, acidulated with hydrochloric acid and boiled in presence of copper, deposits on the metal a gray film, composed

[1] The correct formula is more probably As_4O_6, as the specific gravity of its vapor, determined by Mitscherlich, at 571° (13.85 A.), and later by V. and C. Meyer (Ber., 1879, xii., 1116), at 1560° (13.78 A.=198.93 H.) nearly corresponds with the molecular weight (395.36) represented by that formula (198.93×2=397.86).

of an alloy of copper and arsenic consisting of As 32, Cu 68 in 100.

Crystalline Form.—When white arsenic is fused and allowed to solidify, it forms translucent or transparent, yellowish, *vitreous* masses, having no visible crystalline structure. When it is sublimed in moderate quantity it deposits in the warmer part of the condensor, first in small, brilliant octahedral crystals, which gradually increase in size, and as the temperature rises fuse together and finally form a colorless, liquid film,

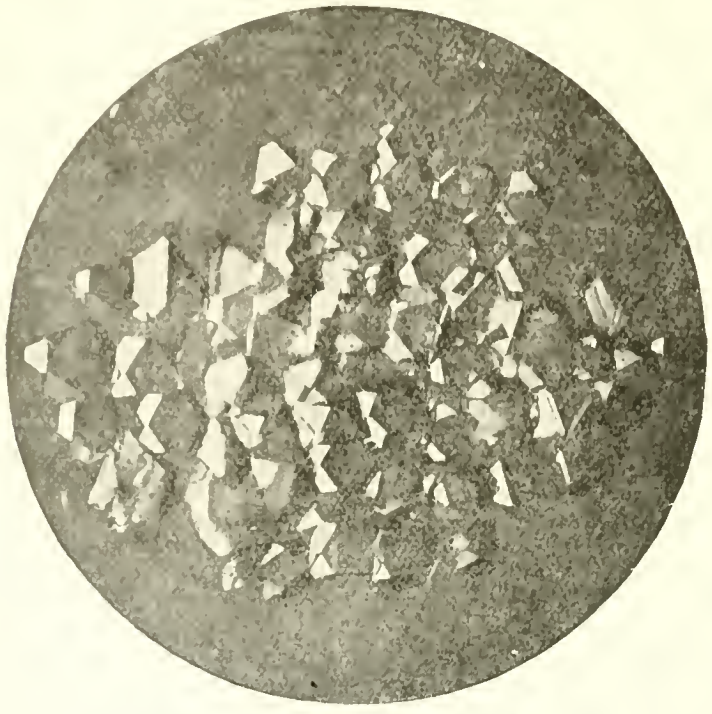

FIG. 7.—Crystals of Arsenic Trioxid. Sublimed. Transmitted light. × 75.

which on solidification has precisely the appearance of glass. This *vitreous arsenic* shrinks and cracks on cooling, and in a short time, in air containing the merest trace of moisture, becomes granular and milky, beginning at the edges, with formation of crystals. This change, which progresses gradually throughout the mass, results in the transformation of the vitreous material into a milk-white, rather friable, opaque substance, having the appearance of porcelain, and hence known as the *porcelainous* variety.[1]

The cakes or lumps of arsenic met with in commerce consist

[1] I. Sickels: "Arsenic Trioxid: a Study of Some of its Forms." Researches Loomis Lab., 1892, ii., 121.

externally of porcelainous arsenic, usually with more or less of the remaining vitreous modification in the centre of the mass, diminishing in amount with the age of the product.

When arsenic trioxid is sublimed or when it is produced by heating the element in a current of air or oxygen, it is deposited in brilliant isometric octahedra (Figs. 7; 8), which are larger and more perfectly formed the nearer the temperature of the condensing surface is to 180° (356° F.). If the condensing surface be cold the crystals are formed rapidly and are consequently

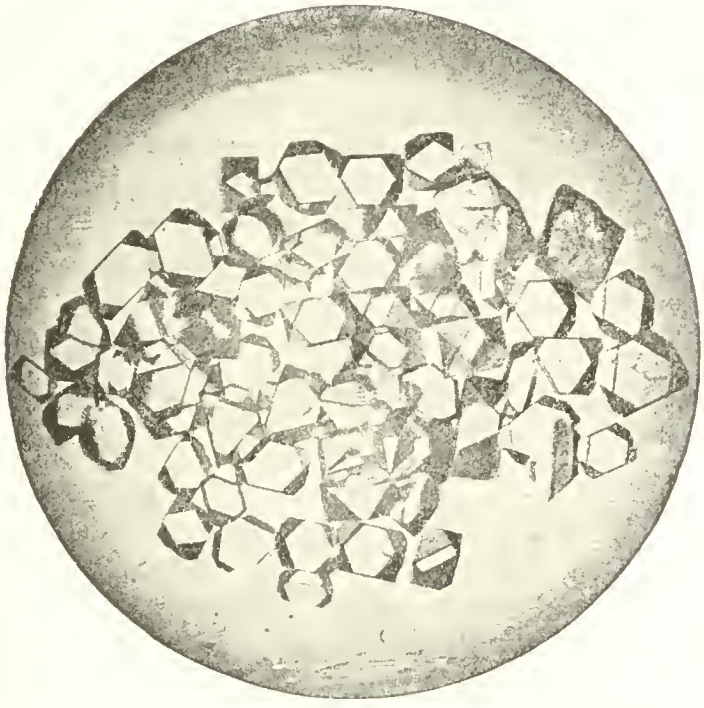

Fig. 8.—Same Specimen as in Fig. 7. Reflected light. × 75.

smaller; while if it be too hot the edges of the crystals are partly rounded by volatilization, or they fuse together to form the vitreous variety. If the temperature varies, aggregated or distorted forms are produced, or small crystals are deposited on the facets of larger ones.

The *flowers of arsenic*, or powdered *white arsenic* of the shops, is a more or less crystalline powder, obtained by grinding either the vitreous arsenic or a more or less crystalline raw product, or by rapid condensation of the vapor in suitable chambers. If prepared by the last-named method, without subsequent grinding, it consists entirely of octahedral crystals. If obtained by grinding the vitreous variety, which is rarely the

case, it consists of a powder whose particles exhibit no crystalline structure when examined microscopically. By far the greater proportion of commercial white arsenic is obtained by grinding a product consisting partly of crystallized and partly of fused arsenic, and consequently consists of a mixture of microscopic octahedral crystals and amorphous particles of varying size.

A microscopical examination of the crystals of white arsenic may be of value under certain conditions for the purpose of distinguishing between two samples. Such a differentiation was of importance, and was the subject of extended inquiry in the trial of State *v.* H. H. Hayden, for the murder of Mary Stannard.

In this case the body of the deceased was found in the woods with the throat cut and a hole in the head, into which a blood-stained stone found near by accurately fitted. An analysis revealed the presence of nearly ninety grains of arsenic in the stomach and liver. The death was abundantly accounted for by the mechanical injuries, and the poison found was considered of importance by the prosecution mainly for the purpose of fixing the crime upon the defendant. It was proved and admitted by the prisoner that, two days before the murder, he had purchased an ounce of arsenic, "to kill rats," which he claimed to have secreted in his barn to prevent the children getting it. A tin box containing one ounce 38.4 grains of arsenic was found in the barn. The prosecution sought to show that the arsenic found in the barn could not have come from the source claimed by the defence, and that the arsenic found in the stomach was similar to that purchased by the defendant. The theory being that the arsenic found in the barn had been placed there either before or afterward, that the accused had administered the poison proved to have been purchased by him to the deceased, that he was with her at her death, and that it was his hand that wielded the knife and the jagged stone. To effect this distinction and identification, Prof. E. S. Dana was sent to England to study the processes of manufacture of arsenic, and upon the trial nearly five days were consumed by the State in taking the testimony of Professors Dana, Brewer, and Wormley, who affirmed that the "barn" arsenic could be readily distinguished from samples obtained from the stock of which the defendant had purchased, and that the latter closely resembled the arsenic found in the stomach of the dead girl. The characters upon comparison of which this opinion was based were: 1. The number of crystals as compared with the number of irregular fragments. 2. The size of the crystals. 3d. The degree of lustre or dul-

ness of the crystals. 4th. The proportion of large fragments to dust-like particles.

The distinction between the "sample" and "barn" arsenics was certainly established by the following differences in characters: 1. About one-third of the "sample" consisted of well-defined crystals, in some mountings only one-fourth, never one-half; while fully nine-tenths of the "barn" was in regular crystals. 2. More than four-fifths of the "sample" crystals measured $\frac{1}{1000}$ to $\frac{1}{500}$ of an inch in their greatest diameter, while the crystals of the "barn" arsenic measured $\frac{1}{2000}$ to $\frac{1}{1000}$, except about one per cent. measuring $\frac{1}{800}$ to $\frac{1}{500}$. 3. The crystals of the "sample" were dull-surfaced, while those of the "barn" were smooth and lustrous. 4. In the "sample" some of the irregular fragments were larger than the crystals, and they constituted more than one-half of the mass, while in the "barn" they did not exceed from one-tenth to one-fourth. The distinction, however, became of little or no import when it was shown that the "sample" arsenic had been taken one month later from the almost empty jar, and consisted of the scrapings of a vessel which had been in use for many years and from which many pounds of arsenic, obtained from different wholesalers, had been dispensed by the druggist, who was consequently unable to state whether the scraping "sample" was from the same wholesale lot as that from which the defendant had purchased or was the remains of another lot put into the jar ten years before.

The investigations of Professor Dana have therefore shown that under favorable circumstances it is possible by microscopic examination to disprove the common origin of two samples of arsenic. But the result of the Hayden trial has also shown that caution is necessary in the use of such evidence. The attempt in this trial to show similarity between the "sample" arsenic and that obtained from the stomach of the dead girl failed; nor does it seem possible that under similar conditions such an attempt would prove successful, for the reason that the solvent action of the gastric secretions produces an undetermined modification of each of the characters used for differentiation.

In the case of Com. *v.* Goersen (Philad. Oy. and T., May, 1883), Professor Leffman found the arsenic adherent to the mucous membrane to be in crystals, which they could not have been had the substance been administered in a homœopathic trituration. He also observed some difference between the regularity of the crystals found in the stomachs of the two victims.[1]

[1] Paper book of plaintiff in error, pp. 19, 70.

In England, where arsenic when sold in small quantity must be mixed with one-sixteenth its weight of soot, or one-thirty-second its weight of powdered indigo, that it may not be mistaken for one of the many white powders used in the household (14 Vic., c. 13, s. 3), it may be possible to establish the certainty of dissimilarity or the probability of similarity between arsenic found in the stomach or vomit and that of the particular preparation alleged to have been administered, by the detection or non-detection of these insoluble colored materials. This

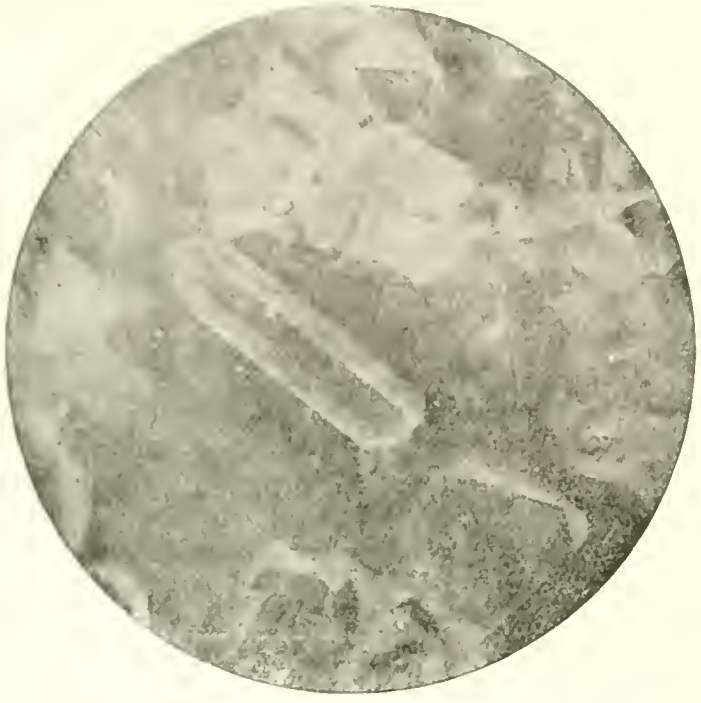

Fig. 9.—Prismatic Crystal of Arsenic Trioxid, among Octahedra. By Sublimation in Sulfur Dioxid. × 250.

point was the subject of inquiry on the trial of Madeleine Smith (1857), in which it was shown that of two samples of arsenic purchased by the accused one was colored with soot and the other with waste indigo. Neither pigment was found in the stomach or intestines.[1]

Arsenic trioxid is dimorphous and isodimorphous with antimony trioxid, and under certain conditions forms crystals which are modifications of the right rhombic prism. Thus when the trioxid is sublimed in a test tube that portion of the sublimate nearest to the heat contains long prisms among the octahedra,

[1] Morison's Report, pp. 33, 35, 38. Browne and Stewart: "Trials for Murder by Poison," pp. 311–312, 316–317.

their number being increased by slight pressure and by using a temperature of about 400° (752° F.)[1] (Fig. 9). The circumstances, however, under which prismatic crystals are produced are not such as obtain in the application of the Marsh or Reinsch test as directed below.[2]

Taste.—Although the taste of white arsenic has been described as hot, acrid, sweetish, metallic, and rough, it is under the most favorable circumstances faint. Taken in small quantity in the solid form it is, as might be expected from its sparing solubility, tasteless, and in very few of the many instances in which it has been taken in large quantity mixed with articles of food is any reference made to an impression of taste: in 15 cases out of 822. In these the taste was referred to as "bitter" in 6 cases. This, if correctly observed, was certainly not due to arsenic. In one instance[3] a "nauseous" taste was noticed, but as it escaped the notice of 14 other persons who ate of the same poisoned pudding, it was probably more imaginary than real. Of a family of 8 persons poisoned by arsenic in their food all are said to have experienced a harsh metallic taste.[4] This and another[5] are the only cases among 822 in which this quality of taste is referred to.

The aqueous solutions of arsenic, although not acrid in any degree, have a very faint though distinct taste, which is at first sweetish, somewhat nauseous, and later slightly metallic in character. It is probable that this taste is observable when the substance is taken in large quantity and unmixed with articles of food. Indeed, Liman[6] states that rescued suicides who have taken it under these circumstances describe the taste as harsh and somewhat salty. Taylor,[7] however, reports an instance in which three persons were severely poisoned by drinking port wine "saturated with arsenic," yet "not the least taste was perceived by any of the parties."

[1] Sickels: *Loc. cit.*

[2] Occasionally crystals are met with among the octahedra in a sublimate which have the appearance of being prisms with oblique ends. These, however, are thin, flat derivatives of octahedra so placed that they present their narrow faces. Their true character becomes evident on changing the focus and rendering the illumination somewhat oblique.

[3] Morley: Brit. M. Jour., 1873, i., 88.

[4] Thompson: Northwest M. and S. Jour., 1851–52, viii., 89–98.

[5] Vogler: Ztschr. f. Staatsarznk., 1846, lii., 276.

[6] Casper-Liman: "Handb.," 8te Aufl., ii., 389.

[7] "Poisons," 3d Am. ed., 302.

The sensation described as a "rough taste" is more a mechanical irritation, caused by the gritty character of the crystalline powder, than a true impression of taste. This sensation has been noted in several cases. The earliest is that of Mr. Blandy, who on two occasions "perceived an extraordinary grittiness in his mouth" on eating gruel prepared by his daughter.[1] In the case of Reg. *v.* John Guy, the victim is said to have complained of a "sandy taste on the tongue."[2] In his confession the Rev. Jacob S. Harden, executed at Belvidere, N. J., in 1860 for the murder of his wife by arsenic, says that he "gave it to her on an apple; she said there was something gritty on it."[3] In a case reported at length by Reuter, the victim shortly after taking two spoonfuls of soup on the day preceding his death "thinks he has sand in his mouth." At the post-mortem particles of a white powder, which proved to be arsenic, were found between the teeth, on the gums, and in the depressions of the buccal mucous membrane.[4] Maschka[5] cites the case of a woman who abstained from eating more than a few mouthfuls of food, upon which her husband had "salted" arsenic, because of the gritty sensation it produced between the teeth.

Solubility.—The degree of solubility of white arsenic in water and other liquids is frequently a subject of chemico-legal importance. The question usually arises in cases in which large quantities of arsenic are found in the stomach or vomit, and the defence advances the theory of suicide. In the case of Reg. *v.* Madeleine Smith 87.9 grains of arsenic were found in the stomach, which it was claimed by the crown had been administered in gruel or cocoa. The defence contended that it was impossible that such a quantity could have been taken unknowingly by the deceased in these, or indeed in any liquid media.[6] This view presupposes the fact that solution is a requisite to secret administration in a liquid medium. While this may be true with a transparent medium, and where the victim is in the possession of his senses, it must not be forgotten that a much larger quantity than could be dissolved may be stirred into a thick and opaque liquid, and taken without pro-

[1] "Tryal of Mary Blandy," 1752, p. 12.
[2] Pharm. J. and Tr., 1857–58, xvii., 385.
[3] "Confession," etc., 1860, p. 20.
[4] Med. Jahrb. f. d. Herzogth. Nassau, 1846, 101, 113, 123.
[5] Gutacht. Prag. med. Fak., 1867, iii., 269.
[6] Morison's Report, 34, 35, 167.

ducing any effect upon the senses, except possibly the "rough taste" referred to above.

Unfortunately it is not possible to assign any definite solubility to arsenic even in water, as it varies with many conditions: with the nature of the oxid, whether crystalline, vitreous, porcelainous, or in amorphous powder; if vitreous or crystalline, whether freshly prepared or old; with the time during which contact is prolonged; with the amount of agitation to which the mixture is subjected; with the relative proportions of solid and solvent; with the temperature; and with the nature of other substances present.

Under all circumstances pure water at the ordinary temperature dissolves the oxid slowly, and leaves a portion undissolved, even after very prolonged contact and when the amount of water present is greatly in excess of that required to dissolve the arsenic.

Although the specific gravity of crystalline arsenic is high (3.689) it does not all sink when thrown upon water. A portion floats upon the surface, forming a film, consisting of particles of the solid buoyed up by adherent air and larger air bubbles surrounded by the powder.[1] That which sinks does not form an even or a heaped layer at the bottom of the vessel as do other insoluble or difficultly soluble powders, but is disposed in nodular lumps, each spheroidal mass consisting of an air bubble surrounded by the powder, which itself is not moistened by the surrounding water. The slow and imperfect solution of crystallized arsenic in water under these circumstances is due to the tenacity with which air adheres to the solid, preventing contact between it and the solvent, and to the great cohesive force of the arsenic. Gmelin[2] found that if 1 part of the oxid be digested with 80 parts of water for several days at the ordinary temperature, the resulting solution contains $\frac{1}{90} = 11.11$ per thousand; with 160 parts of water $\frac{1}{180} = 5.55$ per thousand, with 240 parts, $\frac{1}{280} = 0.83$ per thousand, and, even when 16,000 or 100,000 parts of water are used, a portion of the oxid remains undissolved. Powdered white arsenic which had been left in contact with cold water in closed vessels for eighteen years, dissolved to the extent

[1] Corrosive sublimate in small crystals behaves in the same manner when thrown upon cold water, but does not float for so long a time as arsenic trioxid.

[2] "Handb. d. Chem.," Cavendish Soc. transl., iv., 257.

of 1 part in 54 of water, or 18.5 parts per thousand, which may be considered as the maximum solubility of white arsenic in cold water.

The property of arsenic of floating upon the surface of liquids has attracted attention in several cases of homicide and in some instances has prevented the consummation of the crime.[1]

By adopting different methods of experimentation and operating upon different varieties observers have reached widely divergent numerical expressions of the solubility of arsenic. There is not concordance of results even regarding the relative solubilities of the transparent, opaque, and crystalline varieties. Guibourt[2] and Tidy[3] find the opaque more soluble than the transparent in hot water, in which Taylor[4] finds the transparent the more soluble, and in which Bussy[5] finds the two forms to be equally soluble. Guibourt and Winckler[6] find the opaque more soluble than the transparent in cold water, while Bussy and Tidy find the contrary to be the case. According to Buchner[7] the amorphous is more soluble than the crystalline in both hot and cold water, while Tidy's figures indicate that the reverse is the case.

The solubility of powdered white arsenic by several days' digestion in cold water is given as follows by different authors, expressed in parts dissolved by 1,000 of water: Bucholtz, 20; Fischer, 15.16; Bergmann, 12.5; Guibourt, 12.5 if opaque, 9.71 if transparent; Spelmann and Hahnemann, 10.41; Nasse, 3.12; Klaproth, 2.5.

The results obtained by Taylor[8] are as follows:

	Per 1,000.
1. Boiling water poured on powdered opaque arsenic and let stand seventy-two hours dissolves	2.385
2. Another experiment under similar conditions	2.6825
3. Finely powdered white arsenic gradually added to gently boiling water, and boiling continued one hour	31.5

[1] "Tryal of Mary Blandy," p. 26; Ztschr. f. d. Staatsarznk., 1821, i., 29; 1833, xxvi., 7; 1856, lxxi., 313; J. d. chim. méd., etc. 1853, 3 s., ix., 690; Buff. M. Jour., 1852-53, viii., 68.

[2] Journ. d. chim. méd., 1826, ii., 61.

[3] "Handb. of Chem.," 397.

[4] "Poisons," 3d Am. ed., 287.

[5] Journ. d. chim. et de pharm., 1847, 3. s., xii., 321.

[6] Journ. f. pract. Chem., 1885, n. F., xxxi., 247.

[7] N. Rept. f. Pharm., 1873, xxii., 265.

[8] Guy's Hosp. Repts., 1883, ii., 103.

	Per 1.000.
4. Last set aside seventy-two hours	17.0
5. Same experiment as No. 3, but water boiling violently	46.3
6 Same as No. 5	44.5
7. Same as No. 5	40.76
8. No. 5 cooled to the ordinary temperature	24.7
9. Boiling, saturated solution set aside for six months	28.0
10. Same as No. 9	24.0
11. Transparent arsenic finely powdered and boiled with water an hour	46.0
12. No. 11 repeated	47.55
13. No. 11 cooled	18.7
14. No. 12 cooled	13.4
15. Temperate water added to powdered opaque arsenic let stand seventy-two hours after one agitation	1 0
16. Same as No. 15 with frequent agitation	2.125

Tidy[1] obtained results which would indicate, when compared with those of Taylor, that cold water dissolves more arsenic in twenty-four hours than in seventy-two, and that the solubility in hot water is greater than that given by Taylor. Tidy's results are as follows:

	Transparent form.	Opaque form.	Fresh crystals.
One thousand parts of cold distilled water after standing twenty-four hours dissolved	1.74	1.16	2.0
One thousand parts of boiling water poured on the acid and allowed to stand twenty-four hours dissolved	10.12	5.4	15.0
One thousand parts of water boiled for one hour, the quantity being kept uniform by the addition of boiling water from time to time and filtered immediately, dissolved	64.5	76.5	87.0

We have obtained results higher than those of Taylor and Tidy. In one series of experiments 10 gm. of each form of the solid were placed in corked flasks with 100 c.c. of distilled water and agitated three times daily for two minutes, the last agitation being half an hour before taking the sample, which measured 20 c.c., except the last, of 10 c.c. The sample was filtered through a moist filter which was then washed with 2 c.c.

[1] Woodman and Tidy: "Forensic Med.," 1st Am. ed., 139.

of water and the filtrate evaporated to fixed weight in a weighed porcelain capsule. Under these conditions the amount dissolved in 1,000 c.c. of solution was

	In twenty-four hours.	In forty-eight hours.	In seventy-two hours.	In seven days.	In ten days.
A. Glass arsenic and large crystals from body of retort, freshly sublimed; in lumps.	6.565	8.95	11.78	15.18	15.67
B. Same as A, but finely powdered	9.695	11.635	12.24	14.19	15.28
C. Small crystals from beak of retort, freshly sublimed	6.095	8.76	10.025	13.81	15.04
D. Commercial powdered arsenic, about equal proportions of crystals and amorphous fragments. Saxony	5.00	6.12	7.15	11.015	13.54
E. Porcelainous arsenic, fifteen years old, in fine powder	6.455	9.665	11.38	14.86	15.74
F. Same as E, in lumps	1.805	5.335	7.755	11.175	13.65

The same experiment was repeated with A and F under the same conditions, except that A consisted of glass arsenic alone, without any crystals, and the contents of the flask were only agitated once a day, with the following results:

A	8.785	13.69	15.45	15.845	15.99
F	0.665	2.02	3.935	7.84	12.17

A, B, E, and F were apparently readily wetted by the water, and sank to the bottom in a short time, leaving a more or less clear liquid above; C and D formed nodular lumps and did not completely deposit. The glass arsenic rapidly became opaque, and, when introduced in masses, rapidly disintegrated to a moderately fine powder. From these experiments it would appear that: 1. The glass arsenic is more rapidly soluble in cold water than either of the other forms, whether in mass or in powder. It is probable that solution of this form is favored by the change in molecular aggregation which it undergoes in contact with water. 2. That the porcelainous variety in mass is less readily soluble than the other varieties at all stages, while the same form in powder dissolves slightly more readily than the commercial arsenic. 3. That the solution of all forms continues to increase in strength for at least ten days. 4. That the maximum solubility in cold water of any form by a contact of ten days is about sixteen per

thousand. The solubility of the glass arsenic is of little practical importance, owing to the rapidity with which it is modified by contact with air.

When boiling water is poured upon arsenic the solution is more rapid with all varieties except the powdered porcelainous form, which seems to be about as soluble in cold as in hot water. With the other forms, except C and D, the amount dissolved in seventy-two hours is very nearly the same when cold water is used in the first instance as when the water is boiling. In the following experiments the conditions were the same as in the first series, but actively boiling water was poured upon the solid:

	In twenty-four hours.	In forty-eight hours.	In seventy-two hours.	In seven days.	In ten days.
A.....	9.99	11.28	12.24	13.75	14.47
B.....	10.16	12.087	13.61	15.10	16.20
C.....	12.33	13.73	14.305	15.09	15.85
D.....	8.57	9.69	11.10	12.88	Lost.
E.....	7.215	9.28	11.895	14.46	15.45
F.....	4.545	6.475	8.77	11.91	14.20

If water be maintained in ebullition in contact with arsenic the solution is still more rapid: Water boiled for half an hour in contact with excess of the solid in a flask fitted with a returning condenser dissolved in 100 c.c.: 8.0112 from porcelainous arsenic in lumps; 9.6092 from crystalline arsenic, and 15.3785 from freshy prepared glass arsenic.

The presence of organic matter is usually said to diminish the solvent power of water for white arsenic. It is doubtful, however, whether this applies to organic substances other than fats. Taylor says that hot tea and cold porter dissolve about 0.6 per thousand (one-half grain per fluidounce Br.), and hot coffee about twice that amount. Dr. A. A. Hayes,[1] however, found the fluid part of a decoction of coffee which had caused severe poisoning of a family to contain "apparently as much arsenic as a cold saturated solution," and Silliman[2] found a sample of tea which had severely poisoned a woman and destroyed her nursing infant to be "nearly if not quite a saturated solution."

[1] Boston M. and S. Jour., 1854, xlix., 42.

[2] Buffalo Med. Jour., 1852–53, viii., 730.

Blondlot[1] has shown that the presence of a very small amount of fat suffices to greatly impede the solution of arsenic, probably by coating the surface of the solid with a greasy film, which further increases its repulsive action toward the liquid. In one of his experiments, the inside of a test tube was touched with a rod moistened with oil, and then wiped with a cloth; 15 c.c. of water and a fragment of white arsenic weighing 0.05 gm. were placed in this tube and equal quantities in a clean tube. The two tubes were then heated in a water-bath, when, in half an hour, the arsenic in the clean tube had completely dissolved, while that in the other had suffered a diminution in size almost imperceptible to the eye. It would seem, therefore, that in liquid articles of food which contain fat or oil even in small amount, such as soup, milk, and tea or coffee to which milk has been added, the quantity of white arsenic which can be dissolved is very much less than that which would be dissolved by an equal amount of an aqueous liquid in the absence of fatty material. Yet in one series of experiments with the same (commercial crystalline) arsenic trioxid under identical conditions we found 100 c.c. of "tea" to dissolve 7.234 of arsenic trioxid, and 100 c.c. of coffee 4.9046, while 100 c.c. of soup dissolved 7.76 gm. In each case the liquid was hot, but not boiling, and the duration of the contact was half an hour. The quantity of arsenic was determined after destruction of organic matter by the usual method and as ammonio-magnesian arsenate. If the arsenic be introduced in lumps it will sink to the bottom, taking with it a protecting film of oil, and solution will be almost completely prevented. But if it be introduced in powder, particularly if the liquid be thick or dense, although solution will be similarly interfered with, a large proportion of the powder will remain in suspension for a long time, and may be swallowed with the liquid. If the oil form a layer upon the surface of an aqueous liquid the totality of the powder may remain in suspension in the oily layer, or at its junction with the aqueous liquid.

Niemann[2] has shown that milk left in contact with powdered white arsenic at the ordinary temperature for a quarter

[1] "Mém. Ac. Stanislas," *ex* J. d. chim. méd., etc., 1860, 4 s., vi., 139–154.

[2] Viertljschr. f. ger. Med., 1857, xi., 154.

of an hour does not dissolve sufficient to be recognized by the Marsh test. The interference of milk with the absorption of arsenic was illustrated in a case of attempted homicide in which a man and his wife took about the same quanity of poisoned coffee. The man drank two glasses of milk and suffered only a short time; the wife, who drank no milk, had severe symptoms for four days. Both vomited freely and early.[1] The interference with solution effected by fats and oils (all fats are oils at the temperature of the body) explains both the delay in the appearance of symptoms and the recovery after large doses which have been observed in many cases in which the poison in the form of powder has been taken in soup or other food articles containing fat. As the oils, however, merely prevent solution and exert no precipitant action, the above does not apply in cases in which the poison is added in the form of Fowler's or other solution. In such cases the taking of milk, butter, etc., does not delay a rapidly fatal action.[2]

The solubility of the oxid in alcohol varies with the strength of the spirit and the nature of the oxid; the vitreous variety being more soluble in strong than in weak alcohol, while the contrary is the case with the crystalline, as is shown in the following table:[3]

100 parts of alcohol dissolve.		Alcohol of 56 per cent.	Alcohol of 79 per cent.	Alcohol of 86 per cent.	Absolute alcohol.
Crystallized arsenic,	at 15° (59° F.)	1.680	1.430	0.715	0.025
	at the boiling point	4.895	4.551	3.197	3.402
Vitreous oxid, at 15° (59° F.)		0.504	0.540	....	1.060

The commercial white arsenic dissolves in alcoholic spirits and in wine in about the same proportion as in pure water.

The presence of mineral acids and alkalies, ammonia and the ammoniacal salts, alkaline carbonates, tartaric acid, and the tartrates increases the solubility of arsenic trioxid in water. In the case of the alkalies and alkaline carbonates the increase in solubility is great, particularly at elevated temperatures, by reason of the formation of the soluble alkaline arsenites.

Owing to the great variations in the solubility of white arsenic, experiments should be made in all cases in which the

[1] Kilpatrick: Texas M. and S. Reptr., 1881, i., 89.

[2] Fahrenhorst: Mag. f. d. ges. Heilk., 1852, xx., 483.

[3] From Wurtz: "Dict. d. chim.," i., 398.

degree of solubility is in question, the conditions of the experiments being as nearly as may be identical with those of the case in debate.

The question whether arsenic found in the stomach was taken in the solid form or in solution can only be answered when arsenic in the solid form is found in a body which has not been completely mummified. In that event it was not taken in solution. The theory advanced by the defence in the case of Reg. *v.* Sturt, cited by Taylor,[1] that arsenic may be taken dissolved in a liquid and reassume the solid form in the stomach is not tenable except under the very unusual condition of complete desiccation of the cadaver. When only dissolved arsenic is found it may have been taken in the solid form or in solution. If, however, the quantity found be large, and a careful examination has failed to reveal the presence of solid particles, it becomes highly probable that it was introduced either ante- or post-mortem in the form of a soluble arsenite—a probability which may, under exceptional conditions, become a certainty when the presence of the arsenite as such is demonstrated.

ARSENICAL POISONING.

Statistics and Origin.

White arsenic, which was first prepared in the third century[2] has in the past caused more destruction of life than any other poison, except opium and its derivatives, although tabulated statistics usually show a local preference for certain other poisons.

In England the Reports of the Coroners to the House of Commons for the years 1837–38[3] show that 35.1 per cent. of the deaths from poison were caused by arsenic, and 37.2 per cent. by the preparations of opium. In 1840 arsenic caused only 9.1 per cent. and the opiates 21.5 per cent.[4] Of the cases of poison-

[1] "Poisons," 3d Am. ed., 319.

[2] The discovery of arsenic trioxid is usually attributed to Geber in the eighth century, but among the Greek manuscripts published by Berthelot is a commentary of Olympiodorus which contains a description of its preparation, attributed to Africanus in the third century. See Berthelot: "Alchimistes grecs," Paris, 1888, Trad., p. 82.

[3] London Med. Gaz., 1839, xxv., 204.

[4] "Sixth Rept. Reg.-Gen. Great Brit.," 1844.

ing admitted to Guy's Hospital in 1860–74, only 7.6 per cent. were caused by arsenic—a percentage exceeded by those of hydrochloric acid, mercurials, oxalic acid, lead acetate, and the opiates.[1] In England and Wales during 1863–67, the percentages of deaths from known poisons were: from arsenic, 5.5 per cent.; from opiates, 31.4 per cent.; from the salts of lead, 16 per cent.; from cyanic poisons, 10 per cent.; and from other poisons percentages less than that of arsenic.[2] In the Berlin hospitals the percentage of cases of arsenical poisoning in 1876–78 was only 2.7, which was exceeded by those of carbon monoxid, sulfuric acid, phosphorus, potassium cyanid, oxalic acid, and alcohol.[3] Kobert[4] has given the number of cases of poisoning referred to in medical periodicals in all languages in the years 1880–89, of which 8.3 per cent. are by arsenic, 8.7 per cent. by poisonous foods, and 10.5 per cent. by lead, while the other poisons are represented by a percentage less than that of arsenic. This, however, is an indication rather of the degree of interest awakened in the medical profession than of the number of cases which actually occurred.

Beck has given[5] an abstract of the deaths from poisoning recorded by the coroners of New York in the years 1841–43, of which 14 per cent. were by arsenic and 61.4 per cent. by the preparations of opium. The same records for the years 1867–80 refer to 839 poisonings, of which 4.4 per cent. were by arsenic, 28.2 per cent. by Paris green, and 29.5 per cent. by the preparations of opium. Arsenical poisoning appears to be of more frequent occurrence in India than elsewhere. In the five years 1885–89, the percentages of poisoning by arsenic in the Presidencies of Bengal, Madras, and Bombay were respectively 34.1; 50, and 51, of those in which the nature of the poison was known.[6]

Homicides.—The first recorded attempt to murder with white arsenic was probably that of Woudreton to poison Charles VI. of France in 1384, although the poisonous quality of "sublimed" arsenic was evidently previously known. The poison of

[1] Taylor: "Poisons," 3 Am. ed., 178.

[2] "Rept. Reg.-Gen. Great Brit.," 1869.

[3] Lesser: "Atl. d. ger. Med.," i., 1.

[4] "Lehrb. d. Intoxikationen," 1893, p. 32.

[5] "Infant Therapeutics," pp. 136–143.

[6] Gribble and Hehir: "Outlines of Med. Jur. for India," 1892, pp. 400–401.

Toffana and Spara in the end of the seventeenth century was also probably arsenical.[1]

Although in the past arsenic has been the agent most frequently employed for homicidal poisoning, the frequency of its use in this manner appears to have diminished in those countries in which reliable data for comparison are available. The French records are the most complete and show clearly the diminishing frequency of use of arsenic and the corresponding increase of that of phosphorus in that country. Cormenin[2] gives the number of criminal arsenical poisonings in 1833–39 as 149 in 221 or 67.4 per cent., while phosphorus is not mentioned. Lacassagne[3] has tabulated the cases of criminal poisoning which occurred in France from 1825 to 1880, in quinquennial periods, from which the following percentages are derived:

	1835 to 1840.	1840 to 1845.	1845 to 1850.	1850 to 1855.	1855 to 1860	1860 to 1865.	1865 to 1870.	1870 to 1875.	1875 to 1880.	Entire period.
Arsenic.....	49.8	67.2	71.0	57.5	32.7	20.4	21.8	13.1	24.3	38.8
Phosphorus .	0.0	0.4	1.5	11.5	33.5	40.8	36.3	43.4	33.3	15.8

Tardieu[4] gives a similar table covering a portion of the same period, between 1851 and 1872, from which the following percentages (of all criminal poisonings) are obtained:

	1851.	1852.	1853.	1854.	1855.	1856.	1857.	1858.	1859.	1860.	1861.
Arsenic....	55.6	64.1	46.5	44.6	52.6	30.0	31.0	18.3	18.3	9.4	35.9
Phosphorus.	20.6	7.7	5.6	21.4	26.9	30.0	39.6	40.8	32.6	47.0	33.3

	1862.	1863.	1864.	1865.	1866.	1867.	1868.	1869.	1870.	1871.
Arsenic.....	13.9	42.1	15.6	23.1	12.5	10.5	32.0	40.0	15.3	11.1
Phosphorus...	44.4	26.3	55.6	46.2	25.0	47.3	44.0	31.4	30.7	16.7

In the United States we are under the impression that arsenic still holds the first place in frequency of criminal administration, although there are no available published or unpublished statistics from which a comparison can be made. In an endeavor to obtain information upon this point from the district attorneys of New York State it was learned that during the years 1879–89 there were twelve indictments for murder by poison in thirty-one counties; in six of which the poison alleged

[1] See p. 21.

[2] "Mém. à l'Acad. d. Sc. morales," 1842.

[3] Arch. de l'anthrop. crim., 1886, i., 260.

[4] "Empoisonnement," 2ème ed., 164.

to have been given was arsenic. Neither phosphorus nor morphin was mentioned, and strychnin but once.

It is probable that the diminishing frequency of arsenical poisoning in European countries is due partly to the rigid restriction of the sale there of arsenicals, and partly to dissemination by the reports of trials of the information that arsenic may be detected in the cadaver with certainty at an unlimited period after death. Neither of these causes is operative in the United States, where such restrictions to the sale of arsenicals as exist practically fail of enforcement, and where the deterrent influence of the knowledge of the analyst's power is more than overcome by the information which accompanies it of the general practice of arsenical embalming (see p. 476).

It would seem, from a comparison of the cases reported in medical literature and elsewhere, that the percentage of homicidal cases is higher than with any other poison. Thus of 820 cases collected at random and extending over the period from 1752 to the present time 45.2 per cent. are homicidal, 27.3 per cent. suicidal, and 27.5 per cent. accidental. This excess of homicidal cases over either suicidal or accidental is, however, more apparent than real at the present time, because accidental and suicidal cases are rarely reported unless they present some unusual feature. The records of any large hospital will show that homicidal arsenical cases are in a very small minority of all cases occurring. On the other hand, the absolute number of cases of criminal poisoning in which arsenic was the agent used is much greater than with any other poison.

Arsenic has also been, in almost every instance, the agent used by those who, having succeeded in a first attempt at secret poisoning, have seemed to develop a lust for murder and have continued to add to the list of their victims until their very number has aroused suspicion and led to detection.[1]

[1] In Germany the widow Ursinus, who moved in the higher circles of Berlin society, was convicted in 1803 of an attempt to poison a manservant. She had previously destroyed her husband, a lover, and an aunt by arsenic. In 1811 Anna Marg. Zwanziger was executed, having since 1807 poisoned three persons and made numerous attempts. In 1831 G. Marg. Gottfried was executed at Bremen. She had poisoned fifteen persons, among whom were her parents, her three children, her two husbands, and an intended third. An imitator and namesake of Gottfried, G. Marg. Brockmann, poisoned her sister, father, and brother within a few years. In 1875, fifteen years after the first crime, Franziska K—— confessed to the poisoning of an illegitimate child

Suicide.—Suicides at the present time resort to white arsenic much less frequently than formerly, probably owing to the popular knowledge of the existence of other poisons more rapid and painless in their action. According to Hofmann,[1] in Vienna there were 63 suicides by poison in 1874 of which 2 were by arsenic and (maximum) 32 by potassium cyanid; in 1875, 57 suicidal poisonings, none by arsenic, and 11 (maximum) by caustic potash; in 1876 there were 2 suicides by arsenic. Falck[2] mentions 103 suicides by poison in Prussia in 1869, in 56 of which the nature of the poison was undetermined, in 3 it was arsenic, and in 13 (maximum) sulfuric acid. In England,[3] in the years 1858–1877, there were 673 suicides by poison, of which 29 were by arsenic, and 121 (maximum) by cyanic poisons. In the years 1871–80[4] only 7.97 per cent. of the suicides by poison used arsenic, and 42.36 per cent. opiates. In this list, however, are included 24.21 per cent. by "vermin-killers or fly-killers," at least a portion of which must have been arsenical. In the city of New York during the eleven years ending December 31st, 1880, there were 1,521 suicides. Of these 513 used poison, in 26 cases only the poison was white arsenic, in 200 it was Paris green, and in 134 opium or its derivatives.[5]

of her daughter, and of two husbands of her own, and to the attempted killing of a third by the same means. In France, not to mention earlier cases, Pel was convicted after two trials in 1884 of having poisoned his mistress and burnt the body; his wife had died four years before with symptoms of arsenical poisoning and arsenic was found in the cadaver; he had attempted to poison his second wife and was strongly suspected of having destroyed his mother in 1872 and two girls, one of whom disappeared completely. Yet on the second trial, the jury finding extenuating circumstances (!) he was sentenced to imprisonment for life. Another adept in poisoning, a pharmacist, was acquitted at Havre, although there can be no doubt that fifteen persons were poisoned by him in 1886–1888, and of these three died. In 1885 Maria Cath. Swanenburgh was convicted at Leyden of poisoning her father, mother, and son, as "the final scenes of a series of poisonings by her during several years." In England Mary Ann Cotton was convicted in 1873 of the murder of her stepson. One of three husbands, and four children had died in rapid succession, and "this woman succeeded in destroying by poison twenty persons before her crimes were discovered." In the United States we may cite the case of Henrietta Robinson, convicted in 1888 of the murder of her son by arsenic. Eight others of the family and those in domestic relations with her had previously died with symptoms of arsenical poisoning, and arsenic was found in the cadavers of all but two.

[1] "Lehrb. d. ger. Med.," 5te Aufl., 662.

[2] "Lehrb. d. pract. Tox.," 1889, 18.

[3] "Rept. Reg.-Gen. Great Brit."

[4] Blyth: "Poisons," 1884, 31.

[5] Dr. J. T. Nagle in N. Y. World, Aug. 19th, 1894.

Probably the majority of suicides who die from the effects of white arsenic take it in the shape of some arsenical rat poison (see p. 364). Arsenic has also been used in several cases of combined suicide and homicide.[1]

In cases in which arsenic is shown to be the cause of death, and Paris green is excluded, the question whether it was taken knowingly by the deceased or administered by another is one which must be left to a jury to determine from the moral evidence. We can hardly imagine conditions under which it can be determined by medical or chemical evidence, and we cannot agree with Liman, who,[2] in the case of Henke in 1867, expressed the opinion that because arsenic was found in the stomach in the form of powder and in large quantity, and because the deceased had vomited profusely it was "at least highly probable" that the deceased had poisoned himself, and based his opinion upon another—*i.e.*, "that suicides preferably use arsenic in the form of powder and in doses greatly in excess of that required to produce the desired effect."

It is true that suicides as a rule take large doses and that almost all of the cases (87.5 per cent.) in which solid arsenic is taken in substance are suicidal. Suicides almost always take a single large dose; Paterson,[3] however, reports the case of a woman who attempted to kill herself with repeated very small doses with the object of escaping pain. It is very rare that the dose administered in homicidal cases is known, and in some of these it has been large, while the detection of such quantities in the stomach as were found in the Madeleine Smith and Hayden cases indicate the administration of excessive quantities.[4] Moreover, the detection of crystalline arsenic in the stomach does not indicate that it was taken as a dry powder, as it may be, and frequently has been in homicidal cases, adminis-

[1] Foster: Lancet, 1840–41, ii., 305–317. A woman poisoned herself and two children. Consbruch: Vrtljschr. f. ger. Med., 1854, v., 267–86. A father gave arsenic to three children and took it himself; a boy died. In the Reger case in Erie Co., N. Y., in 1882, a woman killed herself and child and severely poisoned her husband and another child with arsenic in a pancake.

[2] Casper-Liman: "Handb. d. ger. Med.," 8te Aufl., ii., 408.

[3] Edinb. M. Jour., 1857, iii., 395.

[4] In only six of three hundred and seventy-one homicidal cases was the dose determined, in one of which (Ann. d'hyg., 1875, 2 s., xlii., 350) it was said to be 22 gm. (=340 grains), and in another (Med. Times and Gaz., 1878, ii., 626) about 11.5 gm. (two teaspoonfuls).

tered in suspension in a liquid or incorporated as a powder in solid articles of food. Furthermore, the assumption that suicides "preferably use arsenic in the form of powder" is not warranted. Of 126 suicides by white arsenic only 28, or 22.2 per cent., took the poison in that form. (See "Solubility" *ante*, "Administration," and "Paris Green.")

Accidental.—Unintentional poisonings by white arsenic have occurred either from its accidental admixture with articles of food or medicine, or among operatives in industries in which arsenic is used, or by its improper medicinal use.

Although published statistics are very meagre, it would seem that this class of cases are of less frequent occurrence than homicidal or suicidal. In New York City, 1867–80, there occurred 235 accidental poisonings, of which 3 were by arsenic and 2 by "rat poison." In the years 1879–81 there were but 2 accidental poisonings investigated by the examiners in Massachusetts,[1] both of which occurred in 1880. Of 820 cases of arsenical poisoning noticed in medical journals 225 were accidental.

By Mistake, Negligence, or Misadventure.—Arsenic or arsenical rat poison has been mistaken for baking soda,[2] cream of tartar,[3] baking powder,[4] sugar,[5] salt,[6] flour,[7] and meal,[8] and in many cases has been "accidentally mixed" with various articles of food.

As white arsenic volatilizes and sublimes easily at 200° (392° F.), and as the usual temperature of the baker's oven is about 300° (572° F.), the claim may be advanced in cases in which it is supposed that arsenic has been incorporated in bread and other baked articles of food, that such arsenic would not remain in the bread but be expelled by volatilization. That this claim would be false is shown by the fact that bread still retains

[1] Tr. Mass. Med.-leg. Soc., i., 174, 201, 254.

[2] O'Reilly: Dublin J. M. Sc., xx., 1841, 429.

[3] Davis: Chicago Med. Exam., 1861, ii., 421. Otto: Viertljschr. f. ger. Med., 1865, n. F., ii., 177.

[4] Morley: Br. M. Jour., 1873, i., 88. Jeffreys: London M. Times, 1851, n. s., iii., 229.

[5] Hayes, A. A.: Boston M. and S. Jour., 1854, xlix., 42. Sanborn: Boston M. and S. Jour., 1847, xxxvii., 239. Hayes, J., Canada Lancet, 1878, x., 196. Netolitzky: Prag. med. Wchnschr., 1876, i., 225. Friedberg: "Gerichtsärztl. Gutacht.," 1875, 266.

[6] Shaw: Lancet, 1879, i., 699.

[7] Paterson: Edinb. M. Jour., 1857, iii., 397. Ogston: London Med. Gaz., 1851, xii., 183 (in this case three women stole a package labelled "Arsenic, 2 lbs.," and used it for flour to make a cake [!]).

[8] Otto: Vierteljschr. f. ger. Med., 1865, n. F., ii., 175. Puchelt: Med. Ann., Heidelb., 1839, v., 606.

water, which is still more volatile, and by the detection of white arsenic in baked food articles in many instances.[1]

An instance is related by Geissler,[2] which not only evidences the persistence of arsenic in baked articles but indicates conditions under which a suspicion of intentional poisoning might easily have arisen:

Two persons suffered from arsenical poisoning from eating cakes found to contain each about 3 mgm. of white arsenic, which were obtained from a bakery. Other cakes of the same baking and the materials used were found free from arsenic, and the entrance of arsenic into these particular cakes was ascribed, with great probability, to the spattering of some of a solution of arsenic used to kill meal worms into baking pans not customarily used but employed on this occasion.

In this class belong also occasional "mass poisonings" in which a great number of persons have suffered from "mistakes" which are the result of a combination of negligence and intent to adulterate.[3]

In the "Bradford lozenge case"[4] more than two hundred persons were poisoned and twenty-one died. The manufacturer sent to a druggist for "daff" (terra alba, or ground gypsum) with which to adulterate his product. Through the negligence of the druggist he obtained white arsenic instead, and made up his lozenges of arsenic twelve pounds, gum and water four pounds, essence of peppermint one and one-half ounces, sugar about forty pounds, in such manner that each lozenge, weighing forty-three and one-half grains, contained from eleven to sixteen grains of arsenic. The druggist and confectioner were indicted for manslaughter. The former was acquitted on the ground that the negligence was not sufficiently "gross" and the latter does not appear to have been tried.

In the Wurzburg poisonings[5] in 1867, some four hundred persons were more or less severely poisoned by eating cakes into the dough of which arsenic had been mixed by an "unfortunate accident." Fortunately none died.

[1] Morley (Bennett): Br. M. Jour., 1873, i., 88, in pudding. Lupton: Nashville J. M. and S., 1883, xxxi.-xxxii., 7, in biscuit. Riddell: Va. Cl. Rec., 1872, i., 79, in bread. In the Reger case a flat pancake weighing 61 gm. (about two ounces) contained 0.1632 gm. white arsenic (2.07 grains). See also Seisser below.

[2] Pharm. Centh., Berlin, 1884, n. F., v., 43.

[3] See also "Sulfids of Arsenic"

[4] Ph. J. and Tr., 1858-59, xviii., 340, 351, 390; Med. Times and Gaz., 1858, 476, 657. Taylor: "Pr. and Pr. Med. Jur.," 3 ed., i., 284.

[5] Seisser: Aerztl. Int.-Bl., München, 1869, xvi., 45.

Chronic arsenical poisoning of a mild type has also been traced to the use of artificial glucose, manufactured by the action of arsenical sulfuric acid upon starch. This substance almost always contains arsenic to the amount of from 0.0025 to 0.1094 gm. per kilo (.017 to .76 grain per pound avoirdupois),[1] and it is probable that in some cases the presence of arsenic in wine, attributed to the use of arsenical fuchsin, is due to the use of arsenical glucose or of "burnt sugar" made from it.[2]

Arsenic may also be taken up by articles of food from the tinning and soldering of vessels and packages with tin containing an excess of arsenic alloyed with it. This danger was appreciated by the French police as early as 1781, and the Comité d'Hygiène at the present time has fixed the permissible proportion of arsenic in tin at 0.01 in 100.[3] In our own country a poisoning by sardines was found to have been caused by arsenic.[4]

Taylor[5] cites the occurrence of the poisoning of three hundred and forty children in an English institution in 1857, caused by the use of an arsenical liquid to clean a copper boiler in which the water used to dilute milk was heated. He estimates that each child received about a grain of arsenic.

Water may also be contaminated with arsenic by carelessly using vessels which have previously contained an arsenical liquid for drawing water,[6] or from the proximity of a manufacturing establishment in which proper precautions are not taken in the disposal of arsenical waste liquors.[7]

An unusual source of multiple poisoning is related by Köfter:[8] one hundred and thirty-eight persons were poisoned, one fatally, by eating bread which had been made of flour which had been ground upon millstones which had been washed with an arsenical solution.

Arsenic is also a constituent, in small amount, of many nat-

[1] Clouet: Ann. d'hyg., 1878, 2 s., xlix., 145. Ritter, E.: "Des glucoses arsenicales," Paris, 1878.

[2] Schweissinger: Ph. Centralh. f. Deutschl., 1887, 62. Barthelemy: C. rend. Ac. Sc., Paris, 1883, xcvii., 752. The author has also known of a similar case due to the use of beer, which was found to contain a trace of arsenic, in whose manufacture glucose was probably used.

[3] Chevallier: J. d. chim. méd., Paris, 1840, 2 s., vi., 250–254. Pouchet: Ann. d'hyg., Paris, 1890, 3 s., xxiv., 113.

[4] Jenkins: Med. Herald, Louisville, 1882–83, iv., 17.

[5] "Poisons," 3d Am. ed., 290, 336.

[6] Prov. M. and S. Jour., 1841, iii., 257. Bell: Lancet, 1866, ii., 506.

[7] Hakes: Edinb. M. and S. Jour., 1846, lxvi., 43. Braconnot: Ann. d'hyg., 1838, xx., 90.

[8] Allg. Wien. med. Ztg., 1863, viii., 93.

ural waters, in which it exists as sodium, calcium, or ferrous arsenate. It is most abundant in the mineral water of Roncegno, in the Tyrol, which contains 259.2 mgm. of sodium arsenate in the litre (one and one-half grains in a quart), a quantity sufficient to render its use dangerous. Indeed Hirt[1] has reported the poisoning of a child of twelve years by a tablespoonful of this water. Other mineral waters containing arsenic in notable amount are those of La Bourboule (12.6, 14.6, 71.7, and 72.2 mgm. sodium arsenate in the litre); Arène (21.0 mgm. sodium arsenate); Mont d'Or (0.96 mgm. sodium arsenate) and Vichy (2 mgm. sodium arsenate) in France; Levico (8.69 and 0.95 mgm. arsenic trioxid) in the Tyrol; Huber Quelle (6.1 mgm. arsenic trioxid) in Bosnia; Crockett arsenic-lithia water in Virginia (0.267 mgm. sodium arsenate); and Thomsen's bromin-arsenic spring (0.104 mgm. arsenic trioxid) in Ashe County, N. C.[2]

In districts where arsenical mineral deposits exist the waters of streams have been found to contain arsenic in appreciable quantity. This has been shown to be the case in a stream called Whitbeck in Cumberland.[3] No fish live in the stream, but the inhabitants upon its banks use the water for household purposes and exhibit the effects which are observed among the Styrian arsenic eaters. Hofmann[4] has found that the ochre deposited by the water supplied to Leipzig contains 0.0188 per cent. of arsenic trioxid, and estimates the quantity in the water actually used by the inhabitants at 6.79 to 28.45 gm. in 1,000 cm.

That the milk of women who are taking arsenical preparations medicinally contains arsenic was known as early as 1838, when Thomson recognized its presence after the use of the iodid.[5] A woman was given 0.008 gm. (.012 grain) of arsenic daily for six days; 100 gm. of her milk were found to contain 0.001 gm. of arsenic.[6] One case is recorded of the fatal poisoning of a nursing infant to whose mother arsenic had been given with homicidal intent. Silliman obtained decisive evidence of the presence of arsenic in the stomach and liver.[7] In a case of alleged homicide of an infant of two months, in whose

[1] Breslauer ärztl. Zeitschr., 1886, viii., 25.

[2] See also Raspe: Deut. med. Ztg., 1887, No. 57.

[3] Pharm. J. and Tr., 1860–61, n. s., ii., 286.

[4] Diss., Leipzig, 1878.

[5] Lancet, 1838–39, i., 176.

[6] Lewin: "Nebenwirkung d. Arzneimitt.," 2te Aufl., 415.

[7] Smith, J. B.: Buffalo Med. Jour., 1852–53, viii., 730.

cadaver Brouardel and Pouchet[1] found a quantity of arsenic estimated at 0.005 gm. (.08 grain), they expressed the opinion that the poison found might have originated from arsenical preparations taken by the nursing mother.[2]

A large proportion of accidental cases are caused by negligence in exposing articles of food to which arsenical preparations have been added for the purpose of destroying rats, mice, flies, in the household.[3] The possibilities have also been suggested that poisoned rats may fall into wells and contaminate the water,[4] or that poisoned flies may fall into articles of food and drink.[5] So far as we know, however, no authenticated case of arsenical posioning has been traced to these sources, nor to the use of arsenic to protect grain from mice.

The following are some of the more prominent arsenical rat and fly poisons: Arsenic, white arsenic, cobalt, Paris green and London purple are arsenicals sold in bulk. The last mentioned is a waste product of the anilin industry, and is sold as a potato-bug poison. We have found the following proprietary articles to contain arsenic in the proportions mentioned: *Rough on Rats*, a grayish powder, containing 98.89 per cent. As_2O_3, mostly in perfect crystals, mixed with a black, granular powder. *Shoo fly poison fly paper*, each sheet, 18¾x12½ inches, contains 0.3828 gm. (5.9 grains) As_2O_3. *William's fly paper*, each sheet, circle four inches diameter, contains 0.1058 gm. (1.63 grains) As_2O_3. *Dutcher's fly paper*, each sheet, 13¾x17¾ inches, contains 0.6335 gm. (9.78 grain) As_2O_3. *Nickel's poisoned oats*, contains 9.5 per cent. As_2O_3. *Allan's fly brick* contains 2.34 per cent As_2O_3, mixed with powdered earth, probably dolomite. *Daisy fly killer* contains 68.9 per cent. As_2O_3 (both arsenite and arsenate) mixed with sugar. *Lyon's poisoned cheese* contains 93.5 per cent. As_2O_3. *Scatter rats* contains 36.2 per cent. As_2O_3, mixed with flour. *Rat dynamite*, contains 91.3 per cent. As_2O_3, mixed with bran. *Tannin rat and squirrel paste* contains 5.6 per cent. As_2O_3, with fat and meal. *Raticide* contains 40.7 per cent. As_2O_3 mixed

[1] Ann. d'hyg., 1885, 3 s., xiv., 73.

[2] See also Ewald: Berl. kl. Wchnschr., 1882, xix., 544. Dolan: Practitioner. London, 1881, xxvii., 123.

[3] The use of poison for this purpose is as old as Libavius (1560-1616), who recommends a mixture of white arsenic with flour and milk made into cakes ("Syntagma selectorum," Lib. vii., c. 26, ed. Francof., 1615).

[4] Schumburg: Vierteljschr. f. ger. Med., 1893, 3 F., v. 289.

[5] See p. 327.

with a reddish powder, probably rouge. *Rats pat* contains 32.9 per cent. As_2O_3 with meal. *Parry's infallible fly paper*, one sheet 6x7⅝ inches, contains 0.1426 gm. (2.2 grains) As_2O_3. *Saunders infallible fly paper*, one sheet 6¼x7½ inches, contains 0.3546 gm. (5.47 grains) As_2O_3. With the one exception mentioned the arsenic was present as arsenic trioxid or an arsenite.

The occurrence of the unfortunate *Affaire des vins d'Hyères* in France in 1888 well illustrates the insidious danger of the careless handling and storage of white arsenic. A wine grower experimented with arsenic to combat the ravages of the phylloxera, and finding it useless for that purpose stored away a package containing about 75 kgm. Next season four hundred and thirty-five cases of poisoning, including three deaths, were traced to the use of his wine, and it was found that some of this arsenic had been used in "plastering" the wine, some of which was found on analysis to contain as much as 0.16 gm. to the litre (about two and a half grains to the quart) of arsenic.[1]

Cases are also recorded of poisoning by the incautious or improper use of the arsenical liquor used by shepherds to destroy the sheep tick,[2] and of the arsenical soap used by taxidermists to prevent the ravages of insects,[3] as well as by the handling of stuffed animals impregnated with arsenic,[4] and even by the presence of specimens so prepared in a sleeping-room.[5] (See also: "Poisoning by Inhalation," and by "External Application, Arsenical Greens, Sulfids of Arsenic.")

Industrial poisonings by arsenic belong to two distinct classes. In one the toxic agent is hydrogen arsenid, unexpectedly produced by a chemical reaction from arsenic in some form existing as an impurity in the materials used. These cases are almost always acute, and present a clinical history (discussed above) which differs entirely from that of ordinary arsenical poisoning. The second class are mostly due to contact with

[1] Cougit: Ann. d'hyg., 1888, 3 s., xx., 348-360. Marquez: *Ibid.*, 1889, 3 s., xxi., 74-77. Ollivier: Bull. Ac. méd., Paris, 1888, 2 s., xx., 617.

[2] Nicholls: St. Geo. Hosp. Rept., 1869, iv., 220. Bell: Lancet, 1866, ii., 506.

[3] Nevermann: Jahrb. d. ges. Staatsarznk., 1837, ii., 305. Isidore: Rec. d. mém. méd. mil., 1868, 3 s., xx., 419. Both suicides.

[4] Headland: Lancet, 1848, ii., 697. Two children poisoned by a toy white rabbit.

[5] Boston M. and S. Jour., 1890, cxxii., 398. See also "Arsenical Wall Papers."

arsenic trioxid, intentionally used or prepared in the form of vapor or of powder, or to the manipulation of substances containing white arsenic in considerable proportions, and are almost invariably chronic in character.

The industries in which poisoning by white arsenic occurs are: the smelting and roasting of arsenical ores, particularly those of cobalt, nickel, and tin; the manufacture of arsenic, arsenic trioxid, and arsenic acid; the manufacture of anilin colors; taxidermists, candle makers, and enamellers.[1] (See also "Arsenical Greens, Sulfids of Arsenic.")

MEDICINAL.—Although occasional instances of severe and even fatal poisoning have resulted from the legitimate medicinal administration of arsenicals (usually Fowler's solution),[2] most of the cases of so-called accidental medicinal poisonings have resulted from the external application of arsenical pastes by quacks as alleged cancer cures, or from the use of arsenic in some form to destroy parasites. (See "External Application," under "Symptoms.")

In alleged homicidal cases the question sometimes arises, whether arsenic found in the body may not have been administered to or taken by the deceased as a medicine. It is clear that this theory is only tenable when the quantity found is small, and when the poison was not present in the stomach in crystalline form. Arsenic is administered medicinally only in solution and combination, as in Fowler's solution, or in a finely triturated form, mixed with sugar of milk, as in the homœopathic triturations, and in the various pills, granules, tablets, etc., in both of which forms the crystalline structure is destroyed.

In the case of Com. *v.* Goerson, tried in Philadelphia in 1883, the defendant, a homœopathic physician, of intemperate habits, was convicted of the murder of his wife by arsenic. The theory of possible medicinal administration, with the possibility also of an overdose having been given in consequence of the defendant's drunkenness, was advanced, and a physician of the same school was called by the defence

[1] See Ferré: "Arsenicisme professionel," Thèse, Bordeaux, 1882. Schmmburg: Vrtljschr. f. ger. Med., 1893, 3 F., v., 290. Little: M. Times and Gaz., 1870, i., 581; Lancet, 1845, i., 139. Bird: Lancet, 1843, i., 98. Horst: Wchnschr. f. d. ges. Hlk., 1840, ix., 57. Barton: Lancet, 1890, i., 119. Wille u. Jörger: Corr.-Bl. schweiz. Aerzte, 1886, xvi., 242.

[2] Spever: Wochenschr. f. d. ges. Hlk., 1840, ix., 58. Castle: Prov. M. and S. Jour., 1848, 347; N. C. Med. Jour., 1879, iii., 68.

to show the propriety of the use of arsenicum in Bright's disease, from which it was claimed the deceased suffered. As, however, the quantity of arsenic found in the stomach was four grains, and as it was present in crystals, Professor Leffman, the expert for the prosecution, very properly expressed the opinion that it had not been triturated and "had been taken in a solid form, undissolved and unpowdered," and the physician called for the defence admitted that arsenic in this form "had not been prepared in accordance with the rules laid down in our (his) school."[1]

It is only exceptionally, however, and when it is taken in the solid form, that arsenic is found in substance in the stomach. When it is not, the theory of medicinal administration may still be negatived if the quantity found be large. When arsenic is administered medicially it is in doses which do not exceed 0.006 gm. ($\frac{1}{10}$ grain), and usually in doses of 0.0006–0.003 gm., ($\frac{1}{200}-\frac{1}{2000}$ grain) three times daily, and continued for a long time, with periodic interruptions. As arsenic is not a cumulative poison (see Absorption, Elimination, etc.), the existence of more than 0.03 gm. ($\frac{1}{2}$ grain) of unabsorbed arsenic in the stomach is not explainable by the supposition of its introduction by legitimate medicinal administration.

When arsenic is found only in unweighable quantity, and not in crystals, the exclusion of possible medical administration as a "reasonable doubt" must be based upon grounds other than quantity and form. It has been shown by repeated experiences that a person may die of arsenical poisoning, and still none of the poison be detected in the body after death, and the quantity which is found in the cadaver is only indicative of the dose taken in so far that it proves the minimum of that dose, if it was taken during life. (See "Forensic Questions.") The detection of the mere presence of arsenic consequently constitutes one of the factors which, along with symptoms, post-mortem appearances, and history of administration enters into the sum of the evidence for or against medicinal administration.

The supposition of medical administration was an important factor in the defence in two recent *causes célèbres*. In the case of Reg. *v.* Maybrick, tried at Liverpool in 1889, death occurred on the eighth day after the supposed administration, and no arsenic was found in the

[1] A. G. F. Goersen *v.* Commonw. Penn., Paper book of "Plaintiff in Error," pp. 17, 19, 90, 91; also 99th Pa. St. Reps. In this connection see also Liman in Casper-Liman: "Handb.," 8te Aufl., 408.

stomach, while one analyst found 0.02 grain in six ounces of liver, and another 0.32 grain in four ounces by the distillation process and 0.019 in eight ounces by the chlorate process. Unweighable quantities were found also in the kidney. The defence made good use of the fact that this condition of affairs is equally consistent with a lingering death from the criminal administration of toxic doses, and with death from other causes and during or within an undetermined time after the administration of medicinal doses, and sought by every means to establish it as a fact that the deceased was or had been in the habit of dosing himself with arsenic. Prof. William Carter, one of the witnesses for the crown, in discussing the expert evidence upon this point subsequently, very properly remarks: "It seems, therefore, from the above evidence—and no doubt much more of the same general character could be collected—that it cannot be certainly concluded that a person has not been poisoned by arsenic, if no trace of that substance be discoverable in the tissues fifteen or sixteen days after the onset of symptoms; nor that he *must* have been poisoned within that or even a much longer period if traces *be* discoverable. The one thing, and the only thing that is certain, is that arsenic forms no part of the normal human tissues, and that when present in them it must have been introduced from without; but whether introduced feloniously or otherwise must be determined in every case by a consideration of all the facts in connection with it, and not by the opinions of experts on such dubious points as the length of time possible since the last administration, etc." [1]

In the "Affaire Pel," tried at Paris in 1884, the body of one of the alleged victims was exhumed four years after death, and 0.0012 gm. (0.0185 grain) of arsenic was found in 200 gm. of the "part corresponding to the liver and kidneys," and an unweighable quantity in the "part corresponding to the abdomen." None was found in the brain nor in the muscular tissue of the thigh. At the trial the defendant claimed that his wife (the deceased) had undergone an arsenical treatment during the last months of her life; that the quantity found in her body was within the medicinal dose, and that his wife had long been taking arsenic medicinally, principally in the form of Fowler's solution.

To this defence Professor Brouardel makes answer: "According to this hypothesis the exclusive localization of arsenic in the liver is not readily explained: its dissemination in all of the organs, the brain in particular, is the rule; but in the present condition of science I cannot say that the explanation of Pel should be absolutely discarded. What I can affirm is this: None of the observations which I have made is antagonistic to a poisoning by arsenic, and the hypothesis of Pel, while it may not be absolutely rejected, has serious objections against it." [2]

[1] Liverpool Med.-Chir. Jour., 1890, p. 138.

[2] Ann. d'hyg., Paris, 1886, 3 s., xv., 131, 18, 32.

It seems to us that the learned French toxicologist places too much reliance upon the indications of localization, and that he would have been upon surer ground had he left the determination of this point to the jury.

Another method by which arsenic may be medicinally introduced into the human economy is by its existence as an impurity in a medicine.

The preparation which is most frequently of forensic interest in this respect is bismuth subnitrate or subcarbonate, partly because of its liability to contamination with arsenic, and partly because it is a medicine to which the practitioner naturally resorts for the treatment of cases of gastro-intestinal disturbance not recognized as arsenical in origin.

In every case of alleged homicidal arsenical poisoning in which there is history of the administration of bismuth its arsenical contamination is called in question to account for arsenic found on analysis.[1] In one instance a trial for murder was cut short and the defendant released because it was shown by the evidence that the deceased had received bismuth subnitrate, and the chemist for the defence thereupon examined ten samples of the drug and found eight to contain arsenic. A specimen of the identical stock from which the prescription had been made up was not available.[2] In another case of supposed homicide by arsenic the small quantity found in the cadaver was accounted for by the administration of bismuth subnitrate.[3]

Two questions are of importance in this connection: 1. The quantity of arsenic present in bismuth subnitrate. If this can be determined by an analysis of a sample of the same stock from which the medicine administered was taken, such determination should be made. If this be impossible, it must be conceded that the preparation administered may have contained the maxi-

[1] In the Maybrick case (Liverpool Post Report) the bismuth administered was shown to be free from arsenic. In the Riddle case in Connecticut in 1879 "the defence advanced the theory that the arsenic found in the exhumed remains had been absorbed from bismuth, of which the deceased had taken large quantities for medicinal purposes" (Chittenden and Lambert: Am. Ch. Jour., 1881-82, iii., 396). In the case of State of Va. *v.* Emily E. Lloyd (1872) the question of arsenical bismuth seems to have been of importance. (Compare Taylor: "Poisons," 3d Am. ed., 470, with Wormley: "Micro-Chem. of Poisons," 2d ed., 317).

[2] Rogers: Tr. Coll. Phys., Phila., 1857, n. s., iii., 197.

[3] Brunner: Ph. Jahresb., 1889, 558.

mum of impurity. The preparation is rarely absolutely arsenic-free. Gunning found all of six samples examined to contain arsenic.[1] Hawkes [2] found but one arsenic-free in seven. Chittenden and Lambert [3] found but one arsenic-free in fourteen. Salisbury [4] found arsenic in thirteen out of eighteen samples of subnitrate examined, and in all of five samples of subcarbonate. Baylor [5] examined four samples of subnitrate and failed to find arsenic in any. Coad [6] found arsenic in only one of eight samples of subnitrate examined, and none in six samples of subcarbonate.

The highest proportion of arsenic found in recent analyses was in Coad's exceptional sample, which contained 0.33 per cent. of elementary arsenic. Salisbury made quantitative determinations of three specimens, which contained respectively 0.0714, 0.1, and 0.2 per cent. (one-fourteenth, one-tenth, and one-fifth per cent.), calculated as arsenic acid, which would correspond to 0.0377, 0.0527, and 0.1055 per cent. of elementary arsenic. This is somewhat below the maximum as given by Stillé,[7] apparently from an analysis of Lassaigne in 1855,[8] who says that 0.17 per cent. (one-sixth per cent.) has been known to occur. The maximum found by Hawkes was 0.0133 per cent., and by Chittenden and Lambert, 0.0585 per cent.

2. Is the arsenic in bismuth present in a form capable of absorption? So far as deductions can be drawn from experiments upon animals, Chittenden and Lambert [9] have shown that arsenic was not present in the sample experimented upon in a form capable of being readily absorbed into the system. It was, however, absorbed to a slight extent and found in the liver, spleen, brain, blood, kidney, heart, lungs, and muscles. It does not exist in the subnitrate in a form soluble in boiling water, but is dissolved by dilute sulfuric acid. It consequently does not exist there as arsenic trioxid, nor as arsenic acid, but most probably as bismuth arsenate.

We know of no fatal case of arsenical poisoning attributed

[1] Chem. News, May, 1868, 260.
[2] Deut. Amer. Apoth. Ztg., N. Y., 1888, ix., 237.
[3] Amer. Chem. Jour., 1881–82, iii., 398.
[4] Chic. M. Jour. and Exam., 1878, xxxvi., 601.
[5] Tr. M. Soc. Va., 1879, ii., 413.
[6] Amer. Chem., N. Y., 1875, vi., 44.
[7] Wharton and Stillé: "Med. Jur.," 4th ed., ii., 283.
[8] J. d. chim. méd., 1855, 4 s., i., 276.
[9] *Loc. cit.*

to this origin.[1] Cases of non-fatal poisoning have, however, occurred from this cause.[2]

Arsenic has also been found to exist as an impurity in sodium sulfate, and Cameron[3] has reported the fatal poisoning of ten cows by impure Glauber's salt, containing over 0.1 per cent. (8.4 grains to the pound) of arsenic trioxid.

Arsenical magnesium sulfate (Epsom salt) is now frequently met with, owing to the substitution of magnesian limestone for marble as a source of carbon dioxid in the manufacture of soda water. Commercial arsenical sulfuric acid is used, and the magnesium sulfate produced as a by-product is now an important source of that salt. Rattinger[4] has shown that the arsenic is present as an arsenic, and not as an arsenious compound, and in a proportion of 0.411 gm. to the kilogram. A dose of this salt of 30 gm. (about 1 ℥) would contain 0.012 (one-fifth grain) of arsenic acid.

Mylius (E.)[5] has found traces of arsenic in commercial sodium bicarbonate (salaratus, baking-soda).

Buchner[6] has shown that the officinal solution of sesquichlorid of iron (liquor ferri chloridi) frequently contains arsenic, as an arsenic compound, probably ferric arsenate. As this solution is sometimes used in the preparation of the antidote for arsenic, and as reduced iron and other iron compounds have also been found to be sometimes contaminated with arsenic,[7] it has been suggested that traces of arsenic may thus be introduced into the system in the treatment of a supposed case of poisoning. This theory was advanced in the Laffarge case, in

[1] The early case of Kerner (Heidelberger kl. Ann., 1829, v., 348), quoted by Wibmer (i., 416), Orfila ("Tox.," 5th ed., ii., 14), and other writers, of a man who died after symptoms of violent irritant poisoning from a dose containing 8 gm. of magistery of bismuth mixed with cream of tartar, and in whose cadaver appearances similar to those observed in arsenical poisoning were found, has been supposed to be due to arsenic. The substance, however, was delivered by a barber in mistake for magnesia, and the entire quantity was taken. There seems to be no evidence other than the barber's statement that it really was bismuth subnitrate.

[2] Fullerton: Am. J. M. Sc., Phila., 1874, n. s., lxvii., 280. Underhill: Cincin. Lanc. and Cl., 1878, n. s., i., 231. Martiris: J. Soc. d. sc. med. de Lisboa, 1878, xlii., 307. Dalché: Med. Contemp., Lisboa, 1886, iv., 335.

[3] Analyst, London, 1887, xii., 32.

[4] Pharm. Jahresb., 1883–84, 515.

[5] Pharm. Centralh., 1886, n. F., vii., 268.

[6] Chemiker Ztg., 1887, 417; also H. Will: Süddeutsche Apoth. Ztg., 1887, 410.

[7] Beckurtz: Pharm. Centralhalle, 1883, n. F., iv., 570. Bergmann: *Ibid.*, 1888, n. F., ix., 94.

which the quantity of arsenic found was estimated as only 0.0005 gm. ($\frac{1}{130}$ grain.)[1]

The question may also arise whether death was caused by arsenic or by a mistake in the antidote administered. Thus Ranke[2] reports the case of an apothecary who was sentenced to fine and imprisonment for having at least accelerated the death of a child, who had taken arsenic in the form of rat poison, by the administration of liquor ferri sulfurici oxydati (Liquor ferri tersulphatis, U. S. P.) in place of the antidote which should have been prepared from it.

Jaroschi[3] has reported a poisoning which followed the administration of glycerol which was apparently arsenical. The patient was a diabetic who suffered the characteristic arsenical symptoms in consequence of dosing himself with large quantities of glycerol. E. Jahns found arsenic in eight samples of commercial glycerol examined by him, and Barton found seven out of eight samples to be arsenical.[4]

Pharmacists' errors of substitution of arsenic for other substances are of rare occurrence, probably because it is not dispensed in the form of powder. Such errors have, however, occurred, as in a case reported by Preston[5] in which a No. 2 gelatin capsule was filled with arsenic trioxid in mistake for quinin, and caused severe poisoning. In another case a nurse caused the death of a boy by giving him white arsenic in mistake for magnesia.[6]

Method of Introduction.—In the great majority of cases arsenic is taken or administered by the mouth, more or less disguised, in homicidal cases, by mixing with food, drink, or medicine. In some instances it has not been mixed with the food but only placed upon the surface in such manner that the person taking that part would be affected while the remainder of the mass remained pure. Thus Maschka[7] reports the case of a man who attempted to poison his son by placing coarsely powdered white arsenic on the surface of the table salt.

Fodéré[8] has reported a case of administration of white arsenic by the rectum. That of a servant girl executed in 1807

[1] Taylor: "Poisons," 2d ed., 416.
[2] Friedreich's Bl. f. ger. Med., 1881, xxxii., 273.
[3] Prag. med. Wchnschr., 1889, xiv., 308.
[4] J. Am. Ch. Soc., 1895, xvii., 883.
[5] Tr. Indiana Med. Soc., 1880, xxx., 47.
[6] Ph. J. and Tr., 1870, 3 s., i., 66.
[7] Friedreich's Bl. f. ger. Med., 1855, vi., iv., 66.
[8] "Méd. lég.," iv., 266.

for having destroyed her mistress by boiling an ounce of arsenic in a liquid subsequently administered as an enema, after having failed to accomplish the same object with tartar emetic in a "tisane." In a homicidal case tried at Otsego, N. Y., in 1860, the theory of the prosecution was that arsenic had been injected into the rectum.[1] Administration by the vagina has occurred in five instances, in one of which it was self-administered, either with suicidal intent or to produce abortion.[2] Three were homicidal, one unsuccessful[3]; the others: the case of Lambert Couvelance, convicted in 1800 of the murder of his wife,[4] and the case of a Finnish peasant who murdered two wives in succession by this means. The second had been accessory to the first crime.[5] Another case of vaginal administration is reported to have occurred in Russia in 1890.[6]

A case was recently reported[7] in which arsenical poisoning, with the characteristic gastro-intestinal symptoms, was caused by the application of a mixture of creosote, arsenic, and glycerol in the ear.

Intentional poisoning of wells, a crime of which innocent persons have been so frequently accused, is not often attempted with arsenic. One case is reported[8] of an attempt to commit this crime which proved unsuccessful because of the property of arsenic of floating upon the surface of water.

Poisoning caused by the inhalation of vapor of white arsenic is most frequently accidental, occurring either industrially or by the application of the poison to walls in mistake for paint (baryta),[9] or as an insecticide,[10] or by burning some material containing arsenic.[11] In this connection it may be noted that small quantities of arsenic may be introduced into food cooked

[1] Peo. *v.* McCraney, 6 Harris C. R., 49.

[2] Brisken: Vrtljschr. f. ger. Med., 1864, xxv., 110.

[3] Schallgruber: Zts. f. Sttsarznk., 1825, Erg. Hft., iv., 302.

[4] Ansiaux: Zts. f. Sttsarznk., 1821, ii., 187.

[5] Mangor: Act. Soc. med. Havn., 1792, iii., 178.

[6] Jonstoff: "Westnik obsh. hig. sudeb. i prakt. med.," St. Pet., 1890, vii., pt. 3, 27.

[7] Prentiss: Therap. Gaz., Detroit, 1892, 3 s., viii., 105.

[8] J. d. chim. méd., etc., Paris, 1853, 3 s., ix., 690.

[9] Ogston: London M. Gaz., 1851, n. s., xii., 184.

[10] Ph. J. and Tr., 1870, 3 s., i., 66.

[11] Candles to which arsenic has been added to render them harder (see Husemann: "Handb. d. Tox.," 817. Errard: Gaz. méd. d. Paris, 1842, 2 s., x., 713. Pegna: L'Orosi, Firenza, 1879, i., 161–164) or which are colored with arsenical pigments (Lowe: Analyst, London, 1889, xiv., 83).

over a fire of wood which has been painted with an arsenical pigment.

Homicidal administration in this way has, however, occurred. Oxley[1] reports the conviction of a man of manslaughter for having caused the death of a child, and the severe poisoning of seven others, by burning pyrites at the door of a small room in which they were. Two cases of homicidal poisoning in which the administration was by cigars or smoking tobacco impregnated by arsenic are recorded,[2] the latter of which proved fatal, and arsenic was found in the viscera.

Poisonings by the external application of arsenic have been either accidental or caused by improper medical use. Christison[3] has collected references to five cases of poisoning by the application of powdered white arsenic in mistake for face or hair powder, and Tidy has reported[4] the deaths of two infants, upon whose bodies "violet powder," found on analysis to contain 38.5 per cent. of arsenious oxid, was dusted. Several cases are recorded of death or severe poisoning caused by the application of ointments or solutions containing arsenic trioxid to the uninjured skin to destroy lice or to cure the itch,[5] and a number in which the application was to a blistered or abraded surface for the alleged cure of disease.[6] (See Forensic Questions.)

It may be noted that the experiments of Binz and Schulz[7] have demonstrated that poisoning may be caused in animals by the hypodermic injection of a saturated aqueous solution of white arsenic or by its application to the conjunctiva, in the latter case with the production of little more local disturbance than is produced by a weak salt solution similarly applied.

[1] Taylor: "Poisons," 3d Am. ed., 300.

[2] Schlegel: J. d. prakt. Heilk., 1827, lxiv., 13; Friedreich's Bl. f. ger. Med., 1858, ix., iii., 40.

[3] "Poisons," Am. ed., 258.

[4] Lancet, 1878, ii., 250.

[5] Taylor: Guy's Hosp. Repts., 1864, 3 s., x., 220, ointment, death. Fristo: Ann. d'hyg., 1830, iv., 437, ointment, death. Portalez: J. méd.-ch. pharm., 1803, v., 60, with oil, death. Wagner: Wchnschr. f. d. ges. Hlk., 1839, viii., 213. Pyl: Aufs. u. Beob., 1783, i., 43, solution in water, death. Washbourn: London M. Rev. and Mag., 1799–1800, ii., 197, strong solution, recovery. Desgranges: Rec. per. d. l. Soc. d. méd., 1756, vi., 22, ointment, recovery. Mitchell: Med. Times and Gaz., 1853, n. s., vii., 612, with soft soap, recovery. Beck: Am. J. M. Sc., 1851, xxii., 259, with gin, death.

[6] Graham: Glasg. M. J., 1868–69, n. s., i., 56, death. Cameron: Brit. M. Jour., 1890, ii., 203, death. Speyer: Wchnschr. f. d. ges. Hlk., 1840, ix., 58, recovery. Errard: Gaz. méd. d. Paris, 1842, 2 s., x., 713, one death, one recovery. Barker: St. Louis M. and S. Jour., 1864, n. s., i., 29, recovery.

[7] Arch. f. exper. Path. u. Pharm., 1879, xi., 200–230.

ACUTE ARSENICAL POISONING.

Lethal Dose.

The question: "What is the smallest quantity of arsenic capable of destroying human life?" is one which is asked of experts in every trial for murder by arsenic. It is a question which, notwithstanding the great number of cases of arsenical poisoning observed, is still without a satisfactory solution. The answer usually given places the minimum lethal dose at between two and four grains (0.13–0.26 gm.). While this is in consonance with the statements of most writers upon the subject, it is rather an expression of opinion as to what might occur than a statement of observed fact.

The opinion that two grains may prove fatal was first expressed by Hahnemann,[1] whose belief in the efficacy of small doses is well known. In its support the observations of Lachèse fils are quoted by many writers.[2] He concludes that 0.01 gm. (one-eighth grain) taken in food may cause vomiting; that 0.02 gm. (one-fourth grain), taken once only, causes vomiting, colic, and prostration; that the same quantity repeated next day renews these symptoms in such force as to render the individual unfit for work till three or four days afterward, and that four such doses taken at intervals during two days—that is, 0.1 to 0.13 gm. (one and one-half to two grains) in all—excite gastro-enteritis and may prove fatal, since two individuals who had taken this much died, one in seven weeks the other three weeks later.[3] It will be observed that this statement, so far as it refers to a lethal action, applies, not to the administration of one dose, but of repeated doses, the absorption of which is more perfect. Moreover, it is questionable whether a death occurring seven or even three weeks after the taking of such doses can be justly considered as caused by them directly (see Chronic Poisoning).

[1] "Ueber die Arsenikvergiftung," Leipzig, 1786, p. 53. Eine geringere Menge als fünf Gran weisser Arsenik u. s. w. unter weniger günstigen Umständen verschluckt, vermag das nämliche zu thun; ja nur ein bis zwei Grane können, wenn viele der beim ersten Grade angezeigten Umstände zusammen treffen, eine nach verschiednen Tagen, ja noch geschwinder tödliche Vergiftung selbst bei einer Person von reiferm Alter hervorbringen.

[2] Ann. d'hyg., 1837, xvii., 334–350. Quoted by Christison, Beck, Orfila, Tardieu, Taylor, Wormley.

[3] Orfila ("Tox.," 5ème ed., i., 425) remarks that this last conclusion "ne saurait être adopté sans de grandes restrictions."

The statement that 0.13–0.2 gm. (two to three grains) is, under certain circumstances, a lethal dose is further based upon the reports of two alleged cases.[1] An examination into the circumstances of these cases, as given in the original reports, convinces us that the consideration of arsenic as the cause of death in them is purely on the principle of *post hoc ergo propter hoc*.

The two-grain case is reported by H. T. Castle.[2] It was that of an adult female in whom abortion had been recently produced, who took half an ounce of Fowler's solution in unknown doses between Saturday and Wednesday, and died on the following Saturday, having suffered from general illness and fever, with no sickness, purging, or other particular pain in the stomach, and having had repeated attacks of syncope, shortly after one of which she died. General and intense inflammation of the stomach and intestines, and an enlarged, red uterus, bedewed with mucus, were found at the autopsy, and traces of arsenic were detected in the stomach and liver.

It will be observed that there is no history of the administration of "two grains of arsenic," but of a quantity of Fowler's solution containing an amount of potassium arsenite corresponding to two grains of arsenic trioxid. The nature of the symptoms and the post-mortem appearances indicate, moreover, that the abortion had quite as much to do with the death as did the Fowler's solution.

Letheby's[3] case is that of a "healthy, robust girl of nineteen years" who "took about two ounces of fly-water containing two and one-half grains of white arsenic" at night. She suffered the symptoms of arsenical poisoning, from which she rallied during the following day and passed a comfortable night. On the second morning she became drowsy, with a pale, anxious countenance, cold extremities bathed in clammy perspiration, hardly perceptible pulse, and incipient coma. In this condition she was transferred to the hospital, where the coma became more and more intense until she died, thirty-six hours after hav-

[1] Taylor ("Med. Jur.," 11th Am. ed., 125, and "Poisons," 3d Am. ed., 302): "The smallest fatal dose of arsenic hitherto recorded is two grains. Mann ("For. Med. and Tox.," 431): "Two grains of arsenious acid have proved fatal." Wormley ("Micro-Chem. of Poisons," 2d ed., 246): "In a case quoted by Dr. Taylor, two grains of the poison, in the form of Fowler's solution, taken in divided doses during a period of five days, destroyed the life of a woman. The same writer cites another instance, reported by Dr. Letheby, in which two grains and a half killed a robust, healthy girl, aged nineteen, in thirty-six hours." Blyth ("Poisons," 505): "The smallest dose of arsenic known to have proved fatal to a human being is 0.16 gm. (2½ grains)," etc.

[2] Prov. M. and S. Jour., 1848, 347.

[3] Lancet, 1847, i., 44.

ing taken the fly-water. On admission to the hospital it is stated that "when roused she spoke of what she had done and seemed conscious of her danger." At the autopsy the brain was found congested, the lateral ventricles full of half coagulated blood, the heart flabby, distended with jelly-like blood, and with hemorrhagic spots on the endocardium; the stomach pale, nearly empty, and gamboge yellow toward the pylorus. Arsenic was found in the tissue of the stomach.

Here again we have not to deal with "two and one-half grains of arsenic" but with "about two ounces" of a liquid fly poison, notoriously variable in composition, which contains potassium or sodium arsenite, and, in the absence of any statement in the report as to how the amount of arsenic was determined, we must consider that the dose taken is not given with sufficient accuracy. It would seem also from the report, although it is not specifically stated, that the deceased was a suicide and that much, if not all, of the history previous to entrance in the hospital is hearsay evidence. Conceding, however, that the deceased did take the dose mentioned and was thereby severely poisoned, the later history of the case and the post-mortem appearances are much less consistent with death from arsenic than with death from cerebral hemorrhage.

We have now to consider other cases in which it is claimed that death has been caused by small doses.

Christison[1] refers to the following cases: 1. A case of death in six hours from four and one-half grains. This is reported by Fahrenhorst[2] and is the case of a boy of four years who received a solution of arsenic by mistake, suffered from constriction of the œsophagus, retching, vomiting, cutting pains in the stomach and abdomen, burning sensation in the pharynx, cramps, trembling, weakness, and collapse, and died in six hours. The autopsy, made fifteen days after death, revealed the existence of gastritis and duodenitis, the lungs were congested, the pharynx and larynx inflamed, the great vessels full of liquid blood and the heart flabby. An analysis of a portion of the liquid administered to the child showed that it contained arsenic in such proportion that the dose given was four and one-half grains of white arsenic. There does not seem to be reason to doubt that death in this case was caused by arsenic, and, if we may trust the accuracy of quantitative analysis made at this early period, we must admit that this quantity of arsenic may cause the death of a child of the age mentioned when given in solution.

[1] "Poisons," Am. ed., 232–233. [2] Mag. f. d. ges. Heilk., 1823, xx., 483.

2. The case of a man who died from taking six grains, mentioned by Alberti, the particulars of which Christison was unacquainted with. This[1] is a report, in a case of alleged homicide, upon the question (among others) whether a quantity of arsenic "of the size of a pea" and estimated as weighing six grains, which the accused had confessed to having given, was capable of causing death. In the "responsum," dated 1738, the question is evaded, it is considered probable (p. 640) that the deceased received a large amount, and from all the circumstances the conclusion is reached that he died of arsenical poisoning.

3. The cases of two children poisoned by rather less than sixteen grains. This is an instance related by Aguilhon and Barse[2] of two children, aged four and a half and seven years, who, between them, ate a quantity of cheese containing 0.8 gm. (12.35 grains) of arsenic, prepared to poison a cat. Both manifested the symptoms of arsenical poisoning, and one died on the third day. The symptoms and post-mortem appearances were consistent with arsenical poisoning, and arsenic was found in the blood, heart, and contents of the stomach. It is not stated what proportion was taken by the child that died, but even if it took the entire amount this would still remain the smallest dose of solid arsenic known to have destroyed a child.

4. A case alluded to by Valentini in which thirty grains of the oxid killed an adult in six days. This[3] is a "responsum" to the question whether a quantity of white arsenic of the size of a bean (fabæ magnitudine quod pondere drachmam dimidiam æquat) was capable of causing death, made in the affirmative by Valentine in 1652.

Woodman and Tidy[4] refer to Sigmond's case and to two cases of death from external application in which the quantity presumed to have been absorbed was small, although indefinite.

The following modern authors are silent upon the subject of lethal dose: Casper, Filippi, Severi and Montalti, and v. Jaksch (Nothnagel). The following state the lethal dose as between the limits of 0.1 and 0.2 gm. (one and a half to three grains), without citing any cases in support of the view: Tardieu, van Hasselt, Hofmann, Husemann, Kobert, Seidel (Maschka), and Lewin. The dose is similarly stated by Briand, Chaudé and Bouis as 0.05–0.1 gm. (three-fourths to one and a half grains), and by Wharton and Stillé as 0.012–1.15 gm. (one-fifth to twenty-two grains). The following refer to the cases above cited but to no others in this connection: Wormley, Taylor, Blyth, Falck, Naunyn (Ziemssen), Hills

[1] "Jurispr. Med.," Lips., 1740, v., 619.

[2] Bull. Ac. de méd., 1840, v., 144.

[3] "Corp. Jur. Med.-leg.," Francof., 1722, 132.

[4] "For. Med.," 140.

(Wood's "Handbook"), Rabuteau, Lutaud, Guy and Ferrier, and Le Grand du Saule, Berryer and Pouchet.

A careful search through the medical and pharmaceutical periodical literature has failed to bring to light any record of the clearly established death of an adult from a dose of less than 2 gm. (thirty grains) of arsenic in the solid form. This quantity of commercial white arsenic taken with suicidal intent in water, by a female of twenty-seven years, caused her death in twenty-six hours after the manifestation of the characteristic symptoms.[1] Another case is reported[2] in which the dose taken was about the same, although the symptoms did not so closely correspond to those of a typical case of arsenical poisoning, and the poison was, in part at least, in solution. It is the case of a man of fifty-one years who died in one hour[3] after taking by mistake a preparation for softening the hands supposed to be chiefly glycerol. The quantity of arsenic taken was estimated from knowledge of what was in the bottle and of what remained, and from an analysis of the latter as "upward of twenty-six grains (1.7 gm.) of arsenious oxid."

It is true that other cases of alleged arsenical poisoning are on record in which the dose taken is said to have been much smaller, but in them either the dose is not fixed with sufficient accuracy, or the death is improperly ascribed to arsenic. The cases of this character which we have found are the following:

Page[4] relates the case of a child of three and a half years who took eight grains (0.52 gm.) of arsenic on bread, which had been prepared for rats. Copious vomiting was induced by an emetic and in twenty minutes "the stomach appeared to be free from the drug." For three days the child was as well as usual, but on the fourth day manifested interference of respiration and quick pulse, but had no sickness, purging, or pain, and died on the following morning. At the autopsy, ten hours after death, there were few traces of inflammation found in the stomach, and the lungs and pleurae were healthy. The pericardium was filled with pale, straw-colored, transparent fluid. A lump of semi-transparent, organized, coagulable lymph was found in the vena cava close to the auricle, and much more in its cavity. In

[1] S. M. Ward: Therap. Gaz., Detroit, 1885, 3 s., 519.

[2] D. W. Finlay: Lancet, London, 1883, ii., 943.

[3] The rapid action might be explained by the greater solubility of arsenic in glycerol, one hundred parts of which (Wurtz: "Dict. de Chim.," i., 1592) dissolve twenty parts As_2O_3.

[4] Lancet, London, 1837, ii., 626.

the ventricles strings of the same substance were interwoven with the columnæ carneæ, extending into the aorta and nearly filling the auriculo ventricular opening. The walls of the heart were pale, flabby, and soft. And yet this report is headed: "Poisoning by Arsenic"!

In the case of Hiram Cole,[1] charged with murdering his wife by the administration of from three to eight grains (0.2 to 0.5 gm.) of arsenic and one and a half ounces (45 c.c.) of laudanum, the symptoms were distinctly those of strychnin. Professor Cassel, who made the analysis, found arsenic in small quantity in the liver, "but not more than I (he) should expect from previously taking arsenical preparations in medicinal doses," and further testified that he: "Thought that I (he) detected slight traces of strychnin, but nothing upon which I (he) could rely. Wish to be understood as saying that I (he) found none. Found no poison in the stomach." It is lightly probable that this was a case of strychnin poisoning in which the detection in the stomach failed because of insufficient purification of the residue tested.

In this connection it should be mentioned that the dose taken is mentioned in a small proportion only of the cases reported,[2] and that it is most exceptional that the dose is accurately known in homicidal cases.[2]

There are also numerous instances upon record, in which very severe poisoning has been caused in adults by doses as small as 0.065 gm. (one grain), and in children by even less quantities. And it may be conceded that in some of these cases death might have resulted had not the patients received suitable treatment.[3]

[1] Mixer: Ohio M. and S. Jour., 1858–59, xi., 273.

[2] Only in nine cases of which we have knowledge.

[3] Christison (Edinb. M. and S. Jour., 1830, xxxiii., 67): Six persons poisoned by 0.065 (one grain) each in champagne. Murray (Lancet, London, 1838, i., 54): Male, twenty-two, severe poisoning by 1 gm. (fifteen grains) "white arsenic." Recovery in five days. Burne (Lancet, London, 1839, i., 416): Female, adult, "in imminent danger from debility and exhaustion." Had taken 0.013 gm. ($\frac{1}{5}$ grain) As_2O_3 in four pills in three days. Taylor (Guy's Hosp. Rept., 1841, iv., 29): Male, forty, female, fifty-two, severely poisoned by about 0.13 (two grains), and child, sixteen months, by 0.022 gm. (one-third grain) taken in port wine. Bissel (Am. J. M. Sc., 1848, n. s., xvi., 121): Male, twenty-seven. Suicide, severely poisoned by 1.3 gm. (twenty grains). Caner (M. and S. Reptr., Phila., 1878, xxxviii, 359): Female, twenty-four. Suicide, severe poisoning from 0.65–1.0 gm. (ten to fifteen grains) of "arsenic." "A North Carolina Physician" (N. C. M. Jour., 1879, iii., 68): Male, adult. Symptoms of poisoning, including violent abdominal pain, from three doses of ten drops each of Fowler's solution (=0.02 gm.=three-tenths grain As_2O_3). Powell (Westm. Hosp. Rept., 1889, v., 215): Male, forty. Took three tablespoonfuls of Fowler's solution (nearly 0.4 gm.= 6 gm. As_4O_3) with suicidal intent.

On the other hand, numerous instances of recovery after taking large quantities of white arsenic (15 gm. = half ounce and over) have been reported, among which are several in which the amount has been from 30 gm. (one ounce) to 60 gm. (two ounces).[1] In these cases, however, the poison has either been taken in lumps,[2] or early and copious vomiting has removed it before time for solution and absorption has elapsed. The influence of milk and other articles of food containing fat in impeding solution has already been noted.

In a few cases, however, notable quantities of white arsenic have remained in the stomach under circumstances apparently favoring absorption, without causing death:

Kelso[3] reports the case of a girl of eight years who took almost half an ounce (15.5 gm.) of powdered white arsenic, mixed with oatmeal. In one and a half hours she was listless; the pulse was weak, fluttering, and unequal; the respiration was slow and imperfect; there were heat and pain in the throat and stomach, and nausea. Chalk, magnesia, and ferric hydrate were administered, and the patient recovered. The history details the other symptoms of arsenical poisoning, but does not refer to vomiting.

In Jackson's case, cited above, a man of twenty-eight years took not less than two ounces (62 gm.) of white arsenic on an empty stomach. The greater part of it remained in the stomach for six hours. Zinc sulfate and ferric hydrate were administered, and the patient recovered.

Owing to the variations in the rapidity and perfection of the absorption of arsenic under varying conditions, and the differences in the time during which in different cases the process is allowed by circumstances to continue, it is probable that it will remain impossible to foretell the effects of any given quantity of arsenic when taken into the stomach. But it seems

Caused immediate vomiting and very severe poisoning, from which he recovered in fourteen days. Arsenic was detected in the urine of the fifth day, but not in that of the sixth.

[1] Cornish: Lancet, 1849, i., 35 (two cases). Rademaker (Richm. and Louisv. M. Jour., 1873, xv., 383): About one ounce (31 gm.). Walsh (Annalist, N. Y., 1849, iii., 136): 600 grains (39 gm.). Barnum (N. Y. Med. Reposit., 1802, v., 43): Two ounces less a tablespoonful (about 40 gm.). Underhill (Lancet, 1844, ii., 282): Two tablespoonfuls (about 47 gm.). Jahresb. f. Staatsarznk., 1809, ii., 181. Bryant (Lancet, 1852, ii., 299); Jackson (Am. J. M. Sc., 1858, 77): two ounces (62 gm.).

[2] As in a case cited by H. C. Wood: "Mat. Med. and Tox.," 1874, 320.

[3] Lancet, 1844, ii., 154.

possible that at some future time the question concerning lethal dose may, in a somewhat modified form, become more definitely answerable. What is the smallest quantity of *absorbed* arsenic capable of destroying life?

An indication toward an answer to this question was attempted by experiments upon dogs by Rouyer and Feltz,[1] who found that the absorption of 0.0006 gm. per kilogram of body weight (.0042 grain per pound) was capable of causing symptoms of poisoning; that 0.0025 gm. per kilogram (.018 grain per pound) caused severe poisoning and sometimes death in twenty-four hours; and that 0.003 gm. per kilogram (.021 grain per pound) always caused death in eight hours. These results would place the lethal dose for a man of one hundred and fifty pounds at 2.7–3.15 grains (0.17–0.20 gm. per kilogram) of absorbed arsenic. Such calculations, however, based upon experiments upon animals, are to be considered merely as indications, and are by no means to be relied upon.

The determination of the minimum lethal dose of absorbed arsenic may ultimately be reached by a comparison of two series of quantitative determinations: (1) The total quantity of arsenic eliminated in non-fatal cases; and (2) the sum of the quantity eliminated during life and that remaining in the body in fatal cases.

It is possible, by careful but most elaborate analyses, to determine with approximate accuracy the quantity of absorbed arsenic remaining in the body after death. Such determinations have, so far as we know, been made in two instances only:

Johnson and Chittenden[2] estimated the total arsenic trioxid in the body of a woman (in a case of homicide) as 5.2261 grains in the entire body, of which the stomach, spleen, intestines, and uterus contained 1.2255 grains, leaving a trifle over four grains (0.26 gm.) of absorbed arsenic. In another homicidal case Chittenden[3] estimated the total amount of arsenic trioxid in the cadaver of a woman as 3.1192 grains, of which 0.472 grain was in the stomach and intestines, leaving 2.6472 grains (0.1721 gm.) in the remainder of the body. In the case last referred to, however, the body was found floating in shallow water, and there were some grounds for the belief that the deceased had

[1] Paris méd., 1875–76, i., 962, from Gaz. d. hôp.

[2] Am. Ch. Jour., 1880–81, ii., 332.

[3] Am. Ch. Jour., 1883–84, v., 8.

been addicted to the use of arsenic during life, hence it cannot be considered as certain that arsenic was the sole cause of death.

The determination of the total amount eliminated, while apparently a much simpler problem, is only possible under exceptional circumstances. Arsenic is eliminated, not by the kidneys alone, but by the liver as well. Bingley[1] reports the analysis of the cadaver of a woman who died in about six hours, in which 1.13 grains (0.0735 gm.) of arsenic trioxid were found in the gall bladder and bile. Consequently, when the poison is taken by the mouth, the arsenic eliminated by the liver becomes mixed with that which has remained unabsorbed in the intestines and cannot be estimated. The only cases therefore available for the determination of the quantity eliminated are either those in which the administration is by a channel other than the alimentary canal, or those in which purging does not occur, in which death occurs early, and in which the gall bladder is found filled with bile. Both of these classes of cases are of rare occurrence and, so far as we know, have not been examined with a view to determining this question.

The question of the amount of the lethal dose loses much of its apparent importance when we consider that the conditions under which analyses are made in alleged homicidal cases are only most exceptionally such that the amount taken can be determined, even by the most elaborate analysis of sampled portions of all the organs and tissues of the body, of all the excreta and of all of the vomited matter. If any portion of these materials be lost the quantity of arsenic found in the remainder is only a portion of that taken and, *a fortiori*, the amount found in the stomach alone is but an undetermined fraction of the quantity taken. Indeed, it is quite possible that a person may be killed by arsenic and no determinable quantity remain in the body after death. (See Absorption, etc., and Forensic Questions.)

Duration.

Interval Before Appearance of Symptoms.—The first manifestations of the symptoms of arsenical poisoning usually appear in from half an hour to an hour after the poison has been swallowed.

[1] Lancet, 1864, i., 697, 732.

This interval has exceptionally been of less duration. Indeed, several cases have been reported in which the action has been described as being "immediate."

In one case, reported by Dr. Iliff,[1] a woman took a glassful of a saturated solution, fell directly and died instantly, without a struggle. As this is the only case of which we find mention in which death has been said to follow in so short a time, as it was a suicide, and as the report contains no account of attendant circumstances, post-mortem or analysis, we believe rather that death was due to some other cause than that arsenic should produce results so much at variance with other observations.

Taylor[2] reports the case of a woman of twenty-two years, who was immediately attacked with violent pain, etc., on taking two drachms (7.78 gm.) of powdered arsenic in warm water on an empty stomach. This was a suicide. In considering such cases in this connection it must be remembered that previous doses may have been taken, and that, in the absence of proof to the contrary in the report, the time may have been fixed by the statements of the patient, which for our present purpose are liable to be misleading. In homicidal cases, also, the possibility of previous and unknown administrations must be held in view. It is only in cases of accidental poisoning in which the time of taking the poison is definitely known and in which previous doses are clearly out of the question that we find reliable data as to time.

Shaw[3] relates the poisoning of a family of seven persons by an ounce or more of arsenic baked in a meat pudding. A girl of five years was attacked immediately, and died in about six hours. The symptoms in the others appeared "in a short time."

Shepard[4] reports the case of a man, a suicide, who took about an ounce (31 gm.) of arsenic in beer, and vomited immediately.

Taylor[5] states that "in the case of Lofthouse, tried at the York Lent Assizes in 1835, the symptoms were proved to have attacked the deceased while he was in the act of eating a cake in which the poison was administered.

In the case of State *v.* Sager, cited by Elwell,[6] tried in Maine in 1834, great distress was experienced immediately after taking the

[1] Lancet, 1845, ii., 432.
[2] Guy's Hosp. Rep., 1851–52, 2 s., vii., 203.
[3] Lancet, 1879, i., 699.
[4] Tr. S. Car. M. Assoc., 1880, xxx., 21.
[5] "Poisons," 3d Am. ed., 288.
[6] "Malpractice and Evidence," 1860, 504.

poison, which had been added to wine in which an egg had been stirred. (See also Horsford's case below.)

When the interval is short the poisoning is usually severe in character, and its duration short if it terminate fatally. Thus of ten fatal cases in which the interval preceding the manifestation of symptoms was less than twenty minutes the patients expired in the following times: immediately, 3, 6, 6½, 8, 8, 8½–9, 9, 18, and 24 hours. In the case of longest duration (Shepard's case, see above), there were remissions, and the final attack was rapidly fatal. The poison was taken in the afternoon and produced vomiting, which lasted during the night, with little or no pain. The next morning he went into the street and in about an hour was seized with terrible spasmodic pain in the epigastrium, after which the pain ceased and he slept lightly occasionally. In three hours he was again seized with violent epigastric pain, terrible vomiting and purging, finally became delirious and died in convulsions.

Recovery is in some instances slow, the acute attack leaving the patient greatly exhausted and being succeeded by chills, cramps in the abdomen and legs, pain in the back and trembling, which persist for several days.[1] In other cases, however, recovery progresses rapidly.[2]

The rapid appearance of symptoms does not seem to depend upon the form in which the poison is taken. Thus in one of the cases cited above, in which they were immediately manifested (Iliff's case) the poison was in saturated solution, while in another (Lofthouse) it was baked in a cake. Moreover, in several instances in which the same poisoned food has been eaten by several persons the symptoms appeared earlier in some than in others.

Paterson[3] relates an instance in which arsenic was used in mistake for flour and was thus served in an oyster soup to the guests at a dinner party. They were all seized with sickness and vomiting. One of the ladies was taken before she had finished her soup.

An accidental poisoning of a family consisting of father, mother, two girls, and a boy was caused by using water contaminated with arsenic to prepare a dish of bacon and greens.[4] One of the girls, aged

[1] Hemenway: M. News, Phila., 1883, xliii., 456.

[2] Taylor: Guy's Hosp. Rep., 1851–52, 2 s., vii., 203 (two cases).

[3] Edinb. Med. Jour., 1857, iii., 397.

[4] Prov. M. and S. Jour., 1841, iii., 257.

two years, was attacked in a few minutes, afterward the mother, then the other girl, aged three and a half years, and the boy, and finally the father. The father and the two girls died.

A number of guests at a hotel in Winona, Ill., were poisoned by biscuit containing arsenic.[1] One had immediate nausea, retching, and vomiting, while in another the symptoms appeared only after several hours, and were then obscured by hysteria. Some took large doses of emetics, and these apparently suffered most. (See also Shaw's case above.)

Nor is the acceleration of symptoms due solely to the magnitude of the dose taken. Thus in a case reported[1] by Goschler,[2] burning pain in the throat and stomach were experienced by a man of twenty-four years within five minutes after taking 2 gm. (thirty-one grains) of arsenic, dissolved and suspended in water. This amount is much less than has been taken in cases in which the appearance of symptoms has been delayed beyond the average period. In ten instances of short interval in which the amounts taken are given, these have been: 2, 3.9, 5.8, 5.8,–8.7, 7.8, 7.8, 11.6, 15.6, 15.6, and 31 gm.

In cases such as Shaw's and that of Lofthouse, cited above, in which the poison is taken in such form as to preclude the possibility of absorption in so short a period, the first effects must be due to a local action upon a stomach peculiarly sensitive.

Instances are also recorded in which the interval has been prolonged to two hours or more.[3] The longest duration of the interval of which we can find record has been nine hours:

Ryan[4] reported this case, which was that of a man of thirty-five years, who, after a week's debauch, took half an ounce (15.6 gm.) of "arsenious acid" in porter, adding water to get the dregs. The poi-

[1] Horsford: Phila. M. and S. Reptr., 1867, xvi., 45.

[2] Allg. Wien. med. Ztg., 1877, xxii., 156.

[3] Cases in which the interval has been from two to three hours will be found reported by Williams: Lancet, 1840, i., 706. McGee: Lancet, 1849, i., 311. Booth: London M Gaz., 1834, xiv., 62. Todd: Taylor, "Poisons," 3d Am. ed., 288. Giraud: Tr. M and Phys. Soc., Bombay, 1849, ix., 236. Cotting: Boston M. and S. Jour., 1838, xviii., 78. Heebner: M. and S. Reptr., 1887, lvi., 187. Wimmer: Mag. f. d. Staatsarznk., 1846, v., 227. Ward: Edinb. M. and S. Jour., 1830, xxxiii., 61. Cameron: N. Y. M. Press, 1858–59, i., 44. Foster: Lancet, 1840–41, ii., 305. Bissel: Am. Jour. M. S., 1848, n. s., xvi., 121. Bryant: Lancet, 1852, ii., 299. Ogston: London M. Gaz., 1851, n. s., xii., 181. Devergie: J. univ. et hebd., etc., 1832, vi., 333. Orfila: "Tox.," 5th ed., i., 390, 392.

[4] Lancet, 1851, i., 410.

son was taken at 2 P.M. At 11 P.M. he vomited, but did not get out of bed until 7:30 the next morning, when he vomited and suffered from thirst, but did not complain of pain. At 1 P.M. he was found greatly prostrated, almost pulseless, and suffering great pain and intense thirst. The symptoms of intense gastro-intestinal irritation continued for two days and twenty-two hours, when he died. Dr. Ryan observes "the fact of a week's tippling so completely enervating, narcotizing the stomach as to incapacitate it from its usual healthy action, and in its deranged state allowing the common effects of the poison to be delayed."

In another case, reported by Tonnelier,[1] the interval seems to have been eight hours, although the facts, depending to some extent upon the statements of a suicide, are not so positively established as they might be.

A girl of nineteen years confessed to having taken arsenic during the morning, and it was thought that she took it at eleven o'clock in soup. No very serious consequences were observed before evening, although she several times changed color and gave other signs of suffering and anxiety. She dined fairly well at 2 P.M. At 7 P.M. she vomited with extreme violence. She was seen at 11 P.M. by Tonnelier, who observed the symptoms of severe arsenical poisoning. She died at 5 A.M. She had made two previous attempts at suicide by arsenic, the first nine months previously. In the contents of the stomach was found a cyst, supposed to have been formed by the gastric mucous membrane, in which traces of the vessels could be seen and in the interior of which were thin partitions, forming a cellular structure containing fragments of crystalline arsenic of varying size. In the opinion of Dupuytren, this cyst dated from one of the previous attempts at suicide.

Two cases have been reported in which the interval was six hours, in both of which death occurred within two hours after the first symptoms:

Fox[2] reported the case of a man of twenty-one who took by accident a teaspoonful (= ʒ iss. = 5.8 gm.) of arsenic trioxid, mixed with flour. In three hours vomiting was provoked by an emetic, and in a few minutes he seemed quite well. In six hours there were sudden diarrhœa, and vomiting without any of the irritative symptoms of arsenic. He died in about seven hours.

Virchow[3] describes the case of a man of forty years who took a

[1] J. d. med., chir., et pharm., etc., an x. (1802), iv., 15. Flandin: "Tr. d. Poisons," i., 530. Orfila: "Tox.," 5th ed., i., 381.

[2] Lancet, 1848, ii., 503.

[3] Arch. f. path. Anat., 1869, xlvii., 524.

tablespoonful (= ℥ vi. = 23.3 gm.) of arsenic trioxid in fine powder. Symptoms of arsenical poisoning appeared in six hours, and he died in eight hours.

In two instances the interval was about five hours. In one of these the same difference in the time of action in different persons which has been remarked when the interval is short was observed:

Taylor[1] describes the poisoning of a boy of six years and two girls of fifteen and nine years who took at the same time a quantity of arsenic trioxid mixed with flour and sugar. The symptoms appeared in the boy in two hours, and in the girls only in five hours.

Thomson[2] relates the poisoning of a woman of fifty years who took a quantity of fly powder consisting of chrome yellow and white arsenic, containing at least forty grains (2.6 gm.) of the latter, at 3 P.M. No ill effects were observed until evening.

The symptoms of arsenical poisoning are modified and delayed when it is taken in conjunction with opium, or by a person already under the influence of opiates. This affords some explanation of the peculiarities of the following case, reported by Clegg:[3]

A girl of seventeen years, a confirmed opium eater, took a teaspoonful of white arsenic (= ℥ iss. = 5.8 gm.) before 12:30 A.M. At 4 P.M. she appeared as if intoxicated, dull, and reeling. There was neither distress, vomiting, diarrhœa, nor burning in the throat or epigastrium. She had been once sick after dinner. Vomiting was induced by an emetic. At 9 P.M. there were still no arsenical symptoms. At 11:30 the next morning, twenty-three hours after the arsenic had been taken, there was sudden excruciating pain in the body and excessive prostration. She went upstairs to lie down, and at 12 M. was found dead at her bedside. Arsenic was found in abundance in the vomited matters, and arsenic in substance in the stomach.

The appearance of symptoms is also delayed when arsenic is taken by a person already under the influence of alcohol. This is shown by Ryan's case, cited above, as well as by others:

Ward[4] relates the case of a boy of seventeen years who, after a debauch and seventeen hours after having taken food, swallowed three

[1] Guy's Hosp. Rept., 1865, 3 s., xi., 277.

[2] Lancet, 1853, ii., 480; Med. Chir. Rev., 1854, 294.

[3] Med. Times, London, 1849, xix., 26.

[4] Edinb. M. and S. Jour., 1830, xxxiii., 61.

drachms (11.7 gm.) of arsenic in water. He was disposed to sleep and severe symptoms only appeared in two and one-half hours.

Another man of thirty years,[1] while drunk, took at least two ounces (62 gm.), which acted between two and three hours later.

(See Gérard's case below, and Storer's case p. 401.)

If, as has occasionally occurred, the person goes to sleep after taking arsenic the appearance of symptoms is usually delayed:

Christison[2] quotes a case of Macauley's in which a man took seven drachms (27.2 gm.) of the poison at 8 P.M., went to bed at 9:30, and slept until 11, when he awoke with slight pain.

In the case of Rex *v.* Mary Smith,[3] tried at Edinburgh in 1827, one dose was administered at night, the girl immediately went to bed, and no symptoms were observed until six o'clock the next morning.

A man[4] who took a quantity of the trisulfid by mistake went to sleep, and was awakened three hours later by violent abdominal pain.

Delayed symptoms are caused rather by the fact that the poison was taken in a form difficult of solution, or by interference with absorption, than by smallness of dose. In nine cases of delayed symptoms in which the doses are stated they were: 1.3, 2.6, 5.8, 11.7, 15.6, 15.6, 27.2, 62 and 62 gm. (two grains to two ounces).

When the interval is long the poisoning has been in most instances severe and rapidly fatal. Of twenty-nine cases in which symptoms were late in appearing twenty-one terminated in death, and of these thirteen in less than twenty-four hours.

The following are instances of delayed action with rapidly fatal termination:

A man[5] who drank a pint of brandy daily was observed to take arsenic. He continued perfectly tranquil for nearly five hours, when he vomited a little; coldness of the extremities and spasmodic flexion of the legs soon followed, and in a few minutes more he died.

A man[6] took a teaspoonful of arsenious acid mixed with flour. An emetic was administered, and acted in three hours. He seemed quite well until, six hours after taking the poison, he was suddenly attacked with diarrhœa and vomiting, and died in about seven hours.

[1] Bryant: Lancet, 1862, ii., 299.

[2] "Poisons," Am. ed., 235.

[3] Edinb. M. and S. Jour., 1827, xxvii., 450. Christison: "Poisons," Am. ed., 236. Elwell: "Malpr. and Evid.," 1860, 500.

[4] Devergie: "Méd. lég.," 3d ed., iii., 519.

[5] Gérard: Rev. méd., Paris, 1822, vii., 105.

[6] Fox: Lancet, 1848, ii., 503.

A man of forty-five years [1] took about 12 gm. (three drachms) of powdered white arsenic in water at 8 A.M. He went about bidding adieu to his friends, returned at about 10 A.M. and consented to take an emetic. At 1 P.M., not having previously experienced any suffering, he complained of a sense of constriction in the stomach, burning heat, thirst, etc., and died at 5 P.M.

A woman [2] took half an ounce (15.6 gm.) of arsenic. In one hour vomiting was provoked by an emetic. One hour later she was taken with symptoms of collapse, and died in six and one-half hours after having taken the poison.

On the other hand, in eight cases of long interval life has been prolonged for from forty-one hours to twenty-four days.[3]

Period Until Death.—The usual duration of a fatal case of acute arsenical poisoning is about twenty-nine hours.

Leaving out of consideration three doubtful cases,[4] the shortest duration which has been observed is one hour, in a case in which the poison was taken in a most exceptional form, greatly favoring solution and absorption.

Finlay [5] reports this case, which is that of a man of fifty-one years who took twenty-six grains (1.7 gm.) of white arsenic in glycerol. He was almost immediately seized with a feeling of faintness and with collapse; there was slight pain in the epigastrium. In half an hour the skin was cold; the face covered with clammy sweat; the pulse slow and very feeble; the respiration shallow; the pupils somewhat dilated, acting to light. There were appearances of collapse, headache, epigastric pain, and a sense of constriction across the chest. He was quite conscious, speedily became pulseless, sank rapidly, and died in one hour. The post-mortem showed the lungs to be emphysematous and engorged. The right heart contained fluid blood and clots, the left was contracted and nearly empty. There were many ecchymoses in the endocardium of the left ventricle. The mucous membrane of

[1] Missa: Arch. gén. de méd., 1825, vii., 14. Orfila: "Tox.," 5th ed., i., 390.

[2] Booth: London Med. Gaz., 1834, xiv., 62.

[3] Ward (Edinb. M. and S. J., 1830. xxxiii., 61), forty-one hours. Giraud (Tr. M. and Phys. Soc., Bombay, 1849, ix., 236), forty-one hours. Ryan (London M. Gaz., 1851, n. s., xii., 722), seventy hours. Williams (Lancet, 1840, i., 706), fourth day. Ogston (London M. Gaz., 1851, n. s., xii., 181), one hundred and seventeen and a half hours. Devergie (J. nn. et hebd. d. méd., etc., 1832, vi., 333), three cases: four, thirteen, and twenty-nine days; London M. Gaz., 1841–42, 288, six days.

[4] Iliff's case, quoted above: "immediate." Thompson's case, quoted by Taylor ("Poisons," 3d Am. ed., 303), twenty minutes. Case of Clara Ann Smith (London M. and S. Jour., 1834–35, 760, and London M. Gaz., 1835, xv., 519), one hour.

[5] Lancet, 1883, ii., 943. See also p. 379 *ante*.

the stomach was intensely congested, and showed a few small ecchymoses but no erosions. Arsenic in large quantity was found in the tissue and contents of the stomach.

The shortest duration which has been observed under usual conditions is two hours, and of this several instances are recorded:

Foster[1] relates the circumstance of a woman aged thirty-seven years, who poisoned herself and her two-and-a-half-year-old child with arsenic mixed with the yolk of egg. Both died; the woman in three and one-half hours, the child in two hours.

An army officer[2] took a large quantity of coarsely powdered arsenic, refused all treatment, and died in two hours.

A woman[3] took a large dose of arsenic and died within two hours. Upward of an ounce (31 gm.) of arsenic trioxid was found in the stomach.

An individual[4] died with narcotic symptoms only within two hours after having taken nearly a quarter of a pound (227 gm.) of arsenic.

A woman[5] of twenty-seven years died in collapse two and a half hours after having taken one hundred and fifty grains (9.7 gm.) of arsenic in fine powder suspended in water.

A woman[6] in her twentieth year died two and one-half hours after having taken two ounces (62 gm.) of arsenic. She had had violent vomitings by which most of the poison was ejected.

Besides the above we have collected from the medical literature 49 cases in which death occurred in 10 hours or less: 3 in 3 hours; 8 in 4 hours; 6 in 5 hours, 1 in 6 hours; 11 in 7 hours; 5 in 8 hours; 7 in 9 hours; and 3 in 10 hours.

So far as we are aware no recent homicidal case has occurred in which a rapid fatality has been the ground for question as to the cause of death, although Cauvet[7] reports a French case in 1875 in which the deceased died only four hours after the supposed administration, having suffered from colic, diarrhœa, and vomiting on the day preceding, and from vomiting, diarrhœa, thirst, weakness and cramps in the legs after having taken the soup alleged to have been poisoned. From a portion of the intestine 1.73 gm. of arsenic trioxid were separated.

[1] Lancet, 1840–41, ii., 305.
[2] Dieberg: Friedr. Bl. f. ger. Med., 1866, xvii., 53.
[3] Clegg, cited by Taylor: "Poisons," 3d Am. ed., 297, 303.
[4] Macauley: Christison, "Poisons," Am. ed., 243.
[5] Taylor: Guy's Hosp. Repts., 1850–51, 2 s., vii., 183.
[6] Dymock: Edinb. M. and S. Jour., 1843, lix., 350.
[7] Ann. d'hyg., 1875, 2 s., xliii., 350.

Taylor[1] states, however, that in the case of Rhymes, tried in 1841, it was sought to refer the ulceration found in the stomach to some cause other than arsenic, the deceased having died in ten hours after the alleged administration. Christison also[2] states that during the controversy which succeeded the trial of Rex *v.* Russell and Leny, at the Lewes Summer Assizes in 1826, in which the former was convicted of the murder of her husband, who had died three hours after the alleged administration, it was claimed by one of the parties, among other reasons for believing arsenic not to have been the cause of death, that this poison never proves fatal in so short a time as three hours. The same ground was taken by several medical witnesses for the defence upon the trial. It does not seem probable that a defence based upon this ground, if attempted, could be maintained at the present time.

In many instances also the victim of arsenic has survived for a much longer period than twenty-four hours and has finally succumbed, either to a prolonged acute poisoning terminating in death from exhaustion, or to a chronic poisoning succeeding the acute attack:

Paterson[3] relates the instance of a woman of thirty-five years who took, with suicidal intent, an unknown quantity of white arsenic. She was found early in the evening suffering from sickness, vomiting, and purging, which increased in severity until the morning, when the symptoms were very much like those of cholera, except that the vomited matter was like thin coffee grounds; pulse feeble; tongue white; epigastrium tender. There was then no suspicion of poisoning, and the treatment consisted in the administration of opiates and the application of mustard cataplasms. Next day the symptoms were all much aggravated; abdomen very tender to the touch; pulse much weaker, more rapid; everything taken into the stomach rejected; purging frequent, with straining and pain. The condition remained much the same until next morning, when she died convulsed, sixty hours after having been found. Arsenic in substance, tinged with sulfid, was found in the stomach, which was inflamed.

A somewhat similar instance, but of longer duration, is that of the Duc de Praslin[4]: At 3 P.M. August 18th, 1847, he was seen in health. (He was under suspicion of having assassinated his wife the previous night.) At 10 P.M. he was taken with vomiting, had a very feeble

[1] "Poisons," 3d Am. ed., 297, and Guy's Hosp. Rep., 1841, vi., 265.
[2] "Poisons," Am. ed., 240.
[3] Edinb. M. J., 1857, iii., 394.
[4] J. des conn. méd.-chir., Oct., 1847 (from reprint).

pulse, and vomited and purged during the night. His condition went from bad to worse, with occasional remissions, during one of which on the 21st at 5 A.M., he was transferred to prison, where he was placed under strict surveillance, although suspicion of poisoning does not appear to have been seriously entertained until the 22d, four days after the poison had been taken. The treatment was limited to efforts at allaying the gastro-intestinal irritation. He died at 4:35 P.M. on the 24th, six full days after the poisoning, during the greater part of which time it was impossible that he could have taken a further quantity of poison. Arsenic was found in the liver.

In both of these cases the cause of the symptoms was unrecognized, and the poison remained in the stomach, except in so far as it was removed by the efforts of nature. It was consequently gradually absorbed, as is indicated by its presence in the stomach in the first case, and in the liver in the second.

In most cases of protracted poisoning the symptoms of gastro-enteritis are succeeded by those of more or less pronounced chronic poisoning:

A man[1] of twenty-seven years took an unknown quantity of arsenic by mistake in coffee. After the usual symptoms of acute arsenical poisoning, eczema appeared around the mouth on the third day, and he had pain in the forehead. On the fourth day the parotid and submaxillary glands and one tonsil were enlarged, the breath was fetid, and he had fever. On the sixth day urticaria and pyrosis appeared. On the seventh day there was pain in the chest and back, aggravated by breathing, cough, and hæmoptysis. He had strabismus and double vision, delirium and spasmodic contractions of the muscles, and died eight days after the accident.

A case of still longer duration is that of Dr. Alexander[2] who took a quantity of arsenic, mixed with arrowroot, by mistake. He exhibited the usual symptoms and died on the seventeenth day. The stomach was found to be ulcerated. No arsenic was found in the stomach, liver, and spleen, which were analyzed.

Taylor[3] notes an instance of death on the twentieth day after an external application of arsenic to the head. The same author quotes as the longest duration of an arsenical poisoning a case reported by Belloc[4] in which it is inferred that a woman of fifty-six years died two years after an application of a solution of arsenic to the skin. Belloc, however, does not specifically state that the arsenic was the cause of death.[5]

[1] Kersten: Deutsch. Klin., 1851, iii., 393.

[2] Geoghegan: Med. Times and Gaz., 1857, 389.

[3] "Poisons," 3d Am. ed., 303.

[4] "Cours de méd. lég.," Paris, 1807, p. 153.

[5] "Elle traîna une vie languissante

If rapidly fatal cases are rarely the subject of legal inquiry, there have been many instances of alleged homicide in which several days have elapsed between the time when it has been claimed that the poison was administered and the death.[1] Although in some of these cases death may have been caused by the administration of a single dose, it is certain that in many, if not in most of them, repeated administration of small doses was resorted to.

Symptoms.

The clinical history of acute arsenical poisoning is by no means the same in all cases. Apart from peculiarities to be noted later, two distinct types are recognized: The first, of most frequent occurrence, is a very intense toxic gastro-enteritis; the second, more rarely met with, is marked by symptoms referable to a severe cerebro-spinal action, the so-called narcotic or paralytic form.

Gastro-Enteric Form.—This begins with a sense of faintness, depression, and nausea, accompanied usually by a feeling of irritation and heat in the throat and stomach. Vomiting and retching occur early, increase in frequency and in severity, and continue even after the stomach has been emptied completely of its contents except such portions as adhere to its walls. The vomited matters at first consist of whatever food, drink, etc., the stomach may contain; later they are mucous or watery in

pendant deux ans, au bout desquels elle mourut, ayant toujours conservé un tremblement dans tous les membres."

[1] Rex *v.* Blandy, 1752; fourth day. Case of Töllner (Jahrb. d. Staatsarznk., 1809, ii., 221), eight days. Peo. *v.* Kessler, 1818 (Am. Med. Recorder, 1818, i., 386; 3 Wheeler's Crim. Cases, 19), five days. Case of Ober-Tödtmann, 1819 (Mag. f. d. ges. Heilk., 1819, v., 57), six days. Case reported by Christison (Edinb. M. and S. Jour., 1828, xxix., 23), four days. Reg. *v.* Hunter (Taylor: "Poisons," 3d Am. ed., 42, 124, 312), three days. Affaire Gloeckler, 1846 (Ann. d'hyg., 1847, xxxvii., 121), ten days. Case reported by Chevallier and Lassaigne (Ann. d'hyg., 1850, xliii., 420), four days. Case reported, *ibid.* (1850, xliv., 432), five days. Reg. *v.* Wooler, 1855 (Edinb. M. Jour., 1856, ii., 625), fifty days. Case reported by Maschka ("Samml. gerichtsärztl. Gutacht.," Prag, 1858 218), five days. Reg. *v.* Holmes, 1860 (Taylor: *Loc. cit.*, 329), seven days. Case of Franziska Schmidmaier, 1862 (Freidreich's Bl. f. ger. Med., 1862, xiii., 353), sixth day. Reg. *v.* Williams, 1863 (Lancet, 1863, ii., 47), fifteen days. Case of U. V., 1869 (Maschka: *Loc. cit.*, 1873, 232), eleven days. Case of T. C. (Maschka: *Loc. cit.*, 1867, 265), fourteen days. Case reported in Friedreich's Bl. f. ger. Med., 1870, xxi., 447, fourth day. Case of A. E., 1871 (*ibid.*, 1872, xxiii., 395), four weeks and three days. Reg. *v.* Maybrick, 1889, eight days.

character, and still later may be tinged with blood either in streaks or in spots. In most cases the vomited matters are tinted or highly colored, yellow, green or bluish, from admixture with bile regurgitated from the intestine by the violent efforts at vomiting. The yellow color is sometimes ascribed to the yellow sulfid of arsenic. It is in part or in whole due to this cause when King's yellow has been taken, but never otherwise, as white arsenic is not converted into the sulfid in the stomach during life to any considerable extent. (See Patterson's case, p. 392.)

A bright green color points to the strong probability that Paris green has been taken, yet Day [1] has reported the case of a boy of sixteen years who on the second day after having accidentally taken "rough on rats" vomited about a pint of a grass-green fluid. He had vomited about twenty times previously. In some cases the vomited matters are brown in color. This may be due to the administration of ferric hydrate as an antidote, but in some instances it certainly is not, as in a case reported by Paterson,[2] in which the appearance of the ejecta was "like thin coffee grounds," although the arsenical origin of the trouble was not recognized and opiates only were administered; also in a case of Taylor's [3] in which the brown color changed to green on exposure to air, which it might if due to bile, but not if due to ferric compounds. A black or blue-black color is sometimes observed when, as in England, the arsenic has been mixed with lampblack or indigo. A greenish-black color has also been observed,[4] and a pinkish color in another instance.[5] The quantity of blood in the ejecta is usually small and in streaks, although an exceptional instance is recorded by Dennott [6] in which the matter discharged was dark and glutinous, with clots of blood, and about a tablespoonful of bright florid blood followed after each effort at vomiting. It was the case of a suicide who had taken an unknown quantity of arsenic in water, but who recovered. Possibly the hemorrhage was due to some cause other than arsenic. The quantity of vomited liquid is sometimes very great, particularly if large quantities of liquid

[1] N. Y. Med. Jour., 1885, xli., 189.
[2] Edinb. M. Jour., 1857, iii., 394.
[3] Guy's Hosp. Rep., 1851-52, n. s., vii., 187.
[4] Beresford: South. Pract., 1881, iii., 109.
[5] Wilks: Guy's Hosp. Rep., 1855, 3 s., i., 364.
[6] Lancet, 1851, ii., 552.

are taken; thus a suicide who took half an ounce (15.6 gm.) of arsenic[1] was known to have ejected fourteen pints (6,620 c.c.), although the first vomit was not measured and the stomach had been pumped out. The ejecta, particularly those first discharged, should be carefully examined for fragments of the poison, which are very frequently present either in the form of lumps or masses or of a gritty powder which tends to sink to the bottom of the vessel, or is imprisoned in shreds or masses of mucus.

The vomiting is soon accompanied by pain, first in the stomach, then extending from the epigastric to the umbilical region as the intestines become involved, and radiating toward the back and loins. The pain is burning in character, frequently described as "like a red-hot coal in the stomach," and increases rapidly in severity until the patient writhes in agony. The abdomen is tender to the touch and later distended, and the pain is increased by pressure, even the most gentle. Early in the poisoning the tongue becomes coated with a white fur, the mouth is dry and parched, and there is frequently a sense of constriction in the throat, which is sometimes painful. Great thirst is an early and persistent, and probably the most constant, symptom. To relieve it large quantities of liquid are greedily taken at first, although the victim soon finds that this but increases the pain and the vomiting. Sometimes a persistent hiccough torments the patient and causes severe abdominal pains.

Later the vomiting is attended with purging, the discharges being suddenly expelled and frequently passed involuntarily. Soon the purging is accompanied by tenesmus and pain and irritation about the anus. The stools are at first dark-colored and extremely fetid, later yellowish and finally watery, colorless and odorless, resembling the "rice-water" stools of cholera. The urine is diminished in quantity, sometimes completely suppressed, and darker in color than normal. There are severe cramps in the calves of the legs, which usually appear with the beginning of purging. The respiration is interfered with only in so far as abdominal respiration is rendered painful by the tenderness of the abdomen. The patient is very restless, rapidly becomes greatly prostrated, and in a short time enters into the stage of collapse. The pulse is small, frequent, irregular, and

[1] Ryan: London M. Gaz., 1851, n. s., xii., 722.

subsequently becomes imperceptible. The surface is cold and covered with clammy perspiration. The face is anxious, at first pale, later cyanosed. There are attacks of syncope and convulsive movements of the arms and legs. Death is usually quiet, without impairment of intellect until the last, and occurs in about twenty-four hours. (See Duration.)

In its most acute form (asphyxia arsenicalis) arsenical gastro-enteritis closely resembles Asiatic cholera—vomiting, purging of "rice-water" stools, tenesmus, tonic convulsions of the lower and later of the upper extremities, cyanosis and collapse, and death within a few hours. (See Diagnosis.)

Narcotic Form.—The gastro-enteric symptoms, if present at all, are not severe. There is some pain, nausea, and one or a few acts of vomiting, but the patient rapidly becomes somnolent, with a small pulse and cool surface, experiences a feeling of formication, has vertigo, is sometimes delirious, soon becomes comatose, and dies without regaining consciousness. The duration of cases of this class is usually short, less than twenty-four hours, more frequently from four to eight hours.

The two following cases may be quoted as instances of this form:

A woman of twenty-three years, who, as was subsequently ascertained, had taken a teaspoonful of "rough on rats" with suicidal intent, was found unconscious, with matter about her that had evidently been vomited. She could not be aroused, but threw her hands about and pressed them upon her abdomen; there were no spasms of either extremities and no resistance to pressure over the stomach; the face was extremely pale, the pupils widely dilated and not responsive to light, the eyeballs slightly congested. The pulse was feeble, about 80, and the respiration 30. There were involuntary discharges from the bowels, and an emetic caused the vomiting of about a pint of liquid. About five hours later there were signs of returning consciousness and in two hours later she became conscious and answered questions intelligently, but felt weak and sleepy. The stupor increased and she died comatose, fifteen hours after the poisoning, without any convulsions or struggling. At the autopsy the stomach was found to be highly inflamed.[1]

A family of four persons were poisoned by arsenic in coffee. Soon after all had abdominal pain and vomiting, two were carried to bed unconscious. One of them, a girl of six years, awoke in three hours, complained of pain in the abdomen and asked to drink. She drank

[1] Willard: Maryl. M. J., Balt., 1884-85, xii., 333.

sweetened water and again went to sleep. A short time after she was found to be dead. Arsenic was found in the coffee and in the girl's stomach.[1]

The condition of the pupils has not been often noted, but it affords one of the points of distinction between opium narcosis and that sometimes observed in arsenical poisoning. In fourteen out of twenty-two cases in which the condition of the pupils was described they were dilated, even in one homicidal case in which, according to the theory of the state, morphin was administered as well as arsenic.[2] In one of these,[3] in which the symptoms were of the cerebro-spinal type, the pupils were not sensitive to light. In one case,[4] in which the patient had violent tetanic convulsions, the pupils from being minutely contracted became much dilated. In another,[5] in which there were marked gastro-enteric symptoms, the pupils in one and a half-hours alternately dilated and contracted, and in four hours there was little movement of the iris. In another, rapidly fatal case[6] the pupils in two hours were somewhat dilated and susceptible to light. Shortly before death, in collapse, they became contracted and fixed. In one case[7] in which there was some contraction of the pupils, which remained sensitive to light, laudanum had been given along with the arsenic and the symptoms of opium poisoning predominated. Of the four cases in which the pupils were contracted, one[8] was that of a man in whom the pupils were contracted on the afternoon of the second day, he having manifested the characteristic gastro-enteric symptoms during the first day. Another[9] was a homicidal case, in which the pupils, although contracted, were sensitive to light, and in which Hofmann, by reason of the lack of intense inflammation of the stomach and intestines and the slight development of symptoms during life, did not feel justified in attributing the death to arsenic. Only a small quantity was found

[1] Friedberg: "Gerichtsärztl. Gutacht.," 1te R., 1875, 271.

[2] Com. *v.* Francis E. Hayden. Jewett: Boston M. and S. Jour., 1880, cii., 148.

[3] Willard: Maryland M. Jour., 1884–85, xii., 333.

[4] Foster: Lancet, 1840–41, ii., 305.

[5] Kelso Lancet, 1844, ii., 154.

[6] Booth: London M. Gaz., 1834, xiv., 62.

[7] Friedreich's Bl. f. ger. Med., 1862, xiii., 353.

[8] Taylor: Guy's Hosp. Rep., 1851–52, 190.

[9] Friedreich's Bl. f. ger. Med., 1862, xiii., 353; and Hofmann: Ztschr. f. Staatsarznk., 1864, lxxxvii., 260–284.

in the stomach. In a third case,[1] in which the pupils were much contracted, the patient remained perfectly rational to the end, and, although he vomited copiously and the urine was suppressed, there is no mention made of his having suffered from pain or thirst, and the bowels were constipated. He died without a struggle and apparently without pain in forty hours. As the case was a suicide it seems probable that morphin may also have been taken in this case. There is no mention of an analysis. In the fourth case[2] the gastro-enteritic symptoms were prominent.

It is clear that none of these cases could have been mistaken for one of opium narcosis.

Another mixed form of arsenical poisoning is sometimes designated as **subacute.** In cases of this character the beginning of the attack is violent and the symptoms those of either of the forms above described, almost always those of the gastro-enteric form. In from twenty-four to forty-eight hours the manifestations of gastric and intestinal irritation diminish in intensity, and the condition of the patient seems to be ameliorated. The tenderness of the epigastrium usually disappears, although in a non-fatal poisoning which Dr. Sanborn observed in his own person[3] considerable burning pain at the epigastrium and right hypochondrium, increased on exercise, persisted until the tenth day. The lower abdomen remains sensitive longer. The mouth and tongue become sore, dry, and painful, the latter white in the centre, red at the tip and margins. The salivary glands and tonsils swell in some cases.[4] There is complete loss of appetite and continued severe thirst. The pulse is small, weak and rapid (100–130). The temperature is said to he high, although Roth[5] reports a case in which upon the fourth day (the day of death) the temperature was "very low." The urine is diminished in quantity, and has been known to be completely suppressed for three days preceding death,[6] and contains albumin and arsenic. The skin is marked by the most varied exanthemata; general redness, like scarla-

[1] Beresford: Southern Pract., 1881, iii., 109.

[2] Isidore *et al.:* Rec. d. mém. méd. mil., 1868, 3 s., xx. 419.

[3] Boston M. and S. Jour., 1847, xxxvii., 239.

[4] Kersten: Deut. Klin., 1851, iii., 393.

[5] Arch. f. path. Anat., 1868, xliv., 131.

[6] Roth's case, above. Merbach: Vrtljschr. f. ger. Med., 1875, n. F., xxiii., 48–61.

tina, roseola, petechiæ, urticaria, herpes, pemphigus, eczema. Sometimes there is œdema[1] and occasionally icterus.[2] The patient is extremely restless, tosses about, is sleepless and has pain in the head. The eyes are suffused and the conjunctivæ are injected,[3] although this is not so universally the case as was claimed by Dr. Tidy in the Maybrick case. Seidel[4] in describing this form of poisoning does not mention these eye symptoms. In Kersten's case (*l.c.*) they are not mentioned, although strabismus and double vision are referred to as occurring; nor in the equally typical cases reported by Laure (*l.c.*), Merbach (*l.c.*), Roth (*l.c.*), and Williams.[5]

The strength fails, the limbs become weak or paralyzed, the surface cold, the heart's action weak and irregular, respiration difficult, and the pulse imperceptible. There are wandering delirium, loss of consciousness, and sometimes spasmodic contractions of the muscles, and death in convulsions (Merbach's case). More usually death occurs quietly in from three to ten days.

It is from this form of poisoning that James Maybrick died, according to the theory of the prosecution. The principal points from the symptomatology, raised by the defence in opposition to this theory were: 1. The late onset of diarrhœa, and the succession of tenesmus and diarrhœa, concerning both of which there seems to have been a variation in the statement of fact. 2. The slightness of the abdominal pain. 3. The fact that the urine was increased in quantity. 4. The absence of suffusion of the eyes and injection of the conjunctivæ.[6] (See above.)

The **sequence of symptoms** is usually continuous. Occasionally, however, there are remissions or even complete intermissions. Such cases are of rare occurrence, and in homicidal or other cases in which the possibility of a second administration is not distinctly excluded a cessation of symptoms followed by another attack points strongly to a repetition of the ingestion of poison. This view was held by Maschka[7] in a homicidal case

[1] Laure: Lyon méd., 1869, iii., 48.

[2] Taylor (quoting Marshall): "Poisons," 3d Am. ed., 293. Jaundice as a symptom of arsenical poisoning was the subject of inquiry on the trial of Madeleine Smith (Morison's Report, pp. 28, 29, 176).

[3] Taylor: Guy's Hosp. Repts., 1851–52, 2 s., vii., 190. Ward: Edinb. M. and S. Jour., 1830, xxxiii., 61.

[4] Maschka: "Handb. d. ger. Med.," ii., 242.

[5] Lancet, 1840, i., 706.

[6] Carter: Liverp. Med.-Chir. Jour., 1890, x., 117.

[7] "Samml. Gutacht. Prag. med. Fak.," 1858, 218.

in which there was almost complete cessation of symptoms on the fourth day and death on the fifth.

In the case of the Duc de Praslin, however, there were distinct remissions and intermissions of individual symptoms. At some times there was no pain, at others the vomiting ceased. Yet in this instance it was practically impossible that there could have been more than the first large dose taken. This was, however, a case of long duration (six days) whose arsenical character was not recognized until very late and in which, consequently, absorption from the stomach and intestine continued for some days, producing, so far as absorption is concerned, the same effect as a continuous administration. Maclagan[1] cites a case in which, on the second day after having been taken with vomiting and purging, the patient was much better and had ceased to vomit. On the fourth day vomiting recommenced, and he died on the seventh day. Although Taylor, in quoting this case, states that "the symptoms recurred without, so far as could be ascertained, any fresh dose of poison having been given," the case was an alleged homicide, and there does not appear to be sufficient evidence that a fresh dose was not given.

Lothrop[2] has reported the case of a girl of ten years in whom the intermissions were quite marked:

> Soon after taking the poison she vomited at short intervals, with considerable retching, but between the attacks of vomiting she was apparently well. This continued until midnight, when the vomiting occurred at longer intervals and toward morning ceased and she went to sleep. The next morning the trouble seemed over. Vomiting began again and continued until the next day at noon, when it ceased. Then there was considerable retching, pain, distress, and soreness. She was excited, restless and thirsty. After midnight on the second night she slept some, and during the following morning vomited occasionally but had no pain. She ultimately recovered.

Shepard[3] reports a suicidal case in which pain was absent during three hours, although very violent before and after the interval.

Storer[4] relates an accidental poisoning in a workman who, after taking the poison at 9 P.M., and exhibiting the usual symptoms, was much better and made no complaint of pain during the following day, but in the evening again suffered great pain.

[1] London and Edinb. Mthly. J. M. Sc., 1853, xvi., 15.

[2] Buffalo M. and S. Jour., 1865-66, v., 66.

[3] Tr. S. Car. M. Soc., 1880, xxx., 21.

[4] Boston M. and S. Jour., 1841, xxiii., 348.

Ogston[1] reports the poisoning of a girl of sixteen years in which there were not only remissions and intermissions of symptoms, but other peculiarities. She was seen October 28th, at 1:30 P.M., two hours after having taken the poison. The eyes were much injected, the countenance was livid, the pulse slow and feeble. There were salivation and involuntary passages of urine and faeces. She was drowsy and complained of pain in the head. Zinc sulfate did not produce emesis, which was, however, provoked by tickling the fauces, and was kept up for two hours. In the matter first vomited a white powder was found and identified as arsenic. In the evening there were severe thirst and headache, the lower extremities cold. She passed a restless night, with frequent evacuation of fetid material from the bowels, and frequent vomiting. On the morning of the 29th vomiting continued. The abdomen was not tender and there was neither pain nor uneasiness. In the evening the vomiting ceased, there was no thirst and she was hungry. On the morning of the 30th she walked four hundred to five hundred yards to the infirmary. The face was flushed; the pulse weak, quick, and compressible; thirst urgent; tongue red and dry; lips parched, cuticle exfoliated. There were epigastric pain and inclination to vomit everything swallowed. Micturition painful and difficult. On the 31st vomiting ceased and she was nearly free from pain. She suffered from thirst and the lips and tongue were dry and parched. She had no motion for twenty-four hours. November 1st, castor oil was vomited, still no movement of the bowels; thirst urgent, otherwise she was better. November 2d she was incoherent at times during the night, and passed faeces in bed. At 5 A.M., when raised to the night stool, she was seized with rigidity of the joints, then clonic convulsions, which gradually ceased when she was returned to bed. At 8:50 A.M. the convulsions returned, and she died at 9 A.M. Arsenic was found in the first vomit to the extent of 23.312 grains in two fluidounces, and also in the blood, liver, and stomach after death.

All of the symptoms described above may not be met with in every case. Indeed some of the most prominent, such as pain, vomiting, purging, thirst, may be absent or of little intensity.

In some instances these prominent symptoms have been either entirely absent, very slight, or have occurred only late and shortly preceding death. Such exceptional cases have been usually, though not always, observed in old persons or in individuals under the influence of opium or of alcohol:

A family of five persons were all accidentally poisoned by arsenic baked in a cake in mistake for soda. They all suffered from the usual symptoms except an elderly lady in whom there was great prostration

[1] London M. Gaz., 1851, n. s., xii., 181.

of strength and only slight vomiting. She died in six hours with little pain or suffering.[1] (See Clegg's case, p. 388.)

A man, who had been very drunk the day before, and who had eaten nothing for two days, took two drachms (7.8 gm.) of arsenic in solution at 2 P.M. At 3 P.M. he came down-stairs so weak he could hardly stand; appeared very feeble, with a pale countenance, and very thirsty. The water which he drank did not provoke vomiting. At 4 P.M. an emetic which had been administered had failed to act. There were slight cramps in the legs, which were very weak. There was no pain and no swelling of the abdomen. The eyes were red and blood-shot. The skin was not cold or damp even after death. He died at 7 P.M. perfectly rational and without a struggle. Twitchings of the muscles were seen about twenty minutes after death.[2]

In a young woman, free vomiting of matters in which arsenic was shown to be present was produced by an emetic, but she manifested none of the prominent painful symptoms which follow arsenical poisoning.[3]

In some cases only one of the prominent symptoms is present. Thus in Clegg's case, cited above, pain was the only one observed, and that occurred quite late. Ludicke[4] and May[5] have reported cases in which vomiting occurred, but neither pain, purging, nor thirst. In Storer's case, quoted above, thirst was the only one of the prominent symptoms present, and this may have been due to alcohol.

In other cases either pain, vomiting, purging, or thirst, or two of them have been absent. Instances in which there was no pain have been reported by Choulant,[6] Fox,[7] Headland,[8] Taylor,[9] Kerr,[10] Hakes,[11] and Roché;[12] others in which it was very slight by Taylor,[13] Paterson,[14] and Beck;[15] and still others in which it only appeared late in the history of the case by Hodgson,[16] Ogston,[17] and Hofmann.[18] In Paterson's case the woman

[1] O'Reilly: Dublin J. M. Sc., 1841, xx., 429.
[2] Storer: Boston M. and S. Jour., 1841, xxiii., 349.
[3] Seymour: Lancet, 1842–43, ii., 587.
[4] Wochenschr. f. d. ges. Heilk., 1839, viii., 201.
[5] Prov. M. and S. Jour., 1845, 453.
[6] Ztschr. f. d. Staatsarznk., 1841, xlii., 402.
[7] Lancet, 1848, ii., 503.
[8] Lancet, 1848, ii., 697.
[9] Guy's Hosp. Rep., 1865, 3 s., xi., 277.
[10] Edinb. M. and S. Jour., 1831, xxxvi., 94.
[11] Edinb. M. and S. Jour., 1846, lxvi., 43.
[12] Bull. soc. méd. de l'Yonne, 1871, xii., 59.
[13] Guy's Hosp. Rep., 1837, ii., 68; 1851–52, 2 s., 187.
[14] Edinb. M. and S. Jour., 1857, iii., 395 (Case 7).
[15] Am. J. M. Sc., 1841, n. s., i., 57.
[16] Prov. M. and S. Jour., 1851, 490.
[17] London Med. Gaz., 1851, n. s., xii., 181.
[18] Friedreich's Bl. f. ger. Med.,

had determined to poison herself by small doses, as she supposed they would cause death without pain. The doses were very small, measured on the point of a pen knife, but taken daily, and caused griping pains of the abdomen, and diarrhœa. Roché's patient was a woman of sixty-two years who took a teaspoonful of white arsenic by mistake. Vomiting was delayed for half an hour. There was no pain in abdomen or epigastrium even on pressure, and no tenesmus; but cramps in the legs were frequent and very painful. Vomiting and purging were abundant in the earlier stages. In ten hours narcotism appeared and continued to increase in depth until her death in thirteen and one-half hours.

In a report of the medical history of the case of Com. *v.* Francis E. Hayden, Dr. Jewett[1] remarks that "there was and had been entire absence of thirst, painful or difficult deglutition, heat in the throat, and pain or tenderness at the epigastrium." An analysis of the viscera of the deceased by Professors Hills and Wood showed the presence of 5.78 grains (0.3746 gm.) of arsenic trioxid in the stomach of the deceased, 2.72 grains (0.1767 gm.) in the intestine, and 2.77 grains (0.1799 gm.) in the liver. The theory of the prosecution was that the deceased was poisoned by her brother, a medical student, by the administration of about 20 grains (1.3 gm.) of arsenic, followed about two hours later by a large dose of morphin.

In the Maybrick case,[2] the slightness of the abdominal pain was one of the points dwelt upon by the defence.

A case is reported by Spengler[3] in which no vomiting occurred during the entire poisoning. Another by Pyl and Kastner[4] in which even the exhibition of emetics failed to provoke vomiting; and still another by Davies[5] in which four children were poisoned and all vomited after the administration of an emetic except a girl of five years who did not. She and one of the others died. In one of twelve persons whose poisoning by arsenic in fowl soup is related by Ramsay[6] there was no purging. Indeed cases have been reported in which the bowels were

1862, xiii., 353; Ztschr. f. Staatsarznk., 1864, lxxxvii., 260.

[1] Boston M. and S. Jour., 1880, cii., 148. See also Wharton and Stillé: "Med. Jur.," 3d ed., ii., 556.

[2] Carter: Liverp. Med.-Chir. Jour., 1890, x., 135.

[3] Zeitschr. f. Staatsarznk., 1848, lv., 448.

[4] "Aufsätze u. Beob.," 1787, v., 105.

[5] Med. and Phys. J., London, 1812, xxviii., 345.

[6] Am. J. M. Sc., 1834, xv., 261.

constipated. In those recorded by Kelso,[1] Paterson,[2] Bridges,[3] Beresford,[4] and Wormley,[5] the poison was taken by the mouth. In the last-mentioned case the victim suffered from flying pains in the head, dizziness, thirst, constipation, and swollen feet. Five days later he got up and fell dead. At the time death was attributed to apoplexy (he was seventy years old). Seventeen months later the accused while intoxicated confessed to having given three doses of arsenic. Arsenic was found in the stomach, liver, and other viscera. Constipation has also been observed in two cases of external application reported by Barker[6] and Graham.[7] Cases are reported in which there was neither pain nor thirst by Yellowly,[8] Warren,[9] Shipman,[10] Haughton,[11] and A—— P. E.,[12] one by Taylor[13] in which, while pain was present at first, later there was neither thirst nor pain, and the patient complained of a sense of *coldness* at the pit of the stomach; and another by Cotting[14] in which there was neither thirst nor pain until immediately before death.

Hitchcock[15] relates the instance of a woman in whose cadaver, fourteen years after death, Professor Webster found over four grains (0.26 gm.) of arsenic trioxid, during whose last illness there were neither vomiting nor purging, nor were these provoked by the administration of ipecac, zinc sulfate, and purgative enemata. Christison[16] also reports the case of a girl of fourteen who only vomited a little at first, but had no diarrhœa.

Heebner[17] reports the case of a girl, fatally poisoned by "rough on rats," who suffered no pain except shortly before death, and did not purge. A somewhat similar case is reported by Ward.[18]

[1] Lancet, 1844, ii., 154.
[2] Edinb. M. J., 1857, iii., 393.
[3] Am. Med. Bi-Monthly, 1879, x., 298.
[4] South. Pract., 1881, iii., 109.
[5] Ohio M. and S. Jour., 1863, xv., 467.
[6] St. Louis M. and S. Jour., 1864, n. s., i., 29.
[7] Glasg. M. J., 1868–69, n. s., i., 56.
[8] Edinb. M. and S. Jour., 1809, v., 389.
[9] Boston M. and S. Jour., 1843, xxviii., 214.
[10] Am. J. M. Sc., 1843, n. s., vi., 520.
[11] Lancet, 1846, ii., 216.
[12] N. Orl. M. and S. Jour., 1884–85, xii., 324.
[13] Guy's Hosp. Rep., 1851–52, vii., 199.
[14] Boston M. and S. Jour, 1838, xviii., 78.
[15] Boston M. and S. Jour., 1849, xxxix., 489.
[16] Tr. M.-Chir. Soc., Edinb., 1826, ii., 298.
[17] Med. and Surg. Reptr., Phila., 1887, lvi., 187.
[18] Edinb. M. and S. Jour., 1830, xxxiii., 61.

Although usually the urine is scanty and micturition is difficult and painful, cases are not wanting in which the urine is passed freely [1] or even involuntarily.[2] The urine may be suppressed in the first stages of the poisoning, and subsequently excreted, even in cases terminating in death;[3] or, on the contrary, suppression may only occur at a later stage.[4] In a case reported by Stanley,[5] which presented other abnormalities, there was incontinence of urine on the fourth day of the poisoning, and retention on the fifth, when over a pint was drawn with the catheter. Another instance of painful distention of the bladder, relieved by the catheter, in an advanced stage of a non-fatal case, is reported by Jones.[6] There have also been instances in which the quantity of the urine was increased. Thus Spengler [7] reported a case in which the urine was increased in quantity and dark brown in color; and Angouard [8] one in which urine was not passed for the first seven hours, after which ten litres (ten and a half quarts) were passed in ten hours. Potassium nitrate was administered in large quantity.

The urine responds to Fehling's reaction for sugar,[9] probably by the reducing action of arsenious acid contained in it.

The *temperature* in acute arsenical poisoning does not vary much from the normal, and in the few cases in which it has been noted it has been slightly subnormal in some and in others somewhat above the normal. In a somewhat protracted non-fatal poisoning by Fowler's solution, in which the patient was discharged on the fourteenth day, the temperature on the first day was 98°.6 on the second day 97°.8 F., and on the third day 98°.[10] In a homicidal case in which the victim died on the eighth day, the temperature was "natural" on the first day, and "subnormal" on the fifth.[11] In a non-fatal case presenting the characteristic symptoms, in which the patient was discharged on the ninth day, the temperature was 99° F.

[1] Hemenway: M. News, Phila., 1883, xliii., 456.
[2] Ogston: London M. Gaz., 1851, n. s., xii., 181.
[3] Bouillet: Bull. Ac. d. M., Paris, 1840–41, vi., 544. Powell: Westm. Hosp. Rep., 1889, v., 215.
[4] Merbach: Vrtljschr. f. ger. Med., 1875, xxiii., 48.
[5] Amer. Lancet, 1890, n. s., xiv., 446.
[6] Virg. M. Monthly, 1875, ii., 194.
[7] Ztschr. f. d. Staatsarznk., 1848, lv., 452.
[8] Gaz. méd. de Paris, 1843, 2 s., xi., 222.
[9] Vrijens: Diss., Amsterdam, 1881.
[10] Powell: Westm. Hosp. Rep., 1889, v., 215.
[11] Whitford: Lancet, 1884, i., 419.

on the morning of the second day, and 97°.5 on the morning of the third.[1] In a homicidal case—in which, however, there were probably more than one administration, and in which the death occurred on the ninth day—the temperature was 100° on the second and sixth days and "natural" on the morning of the seventh.[2] In one case only do we find record of a marked elevation of temperature, and in that the symptoms departed widely from the typical clinical picture of arsenical poisoning, although there is no reason to doubt that arsenic was taken. In this poisoning, from which the patient recovered very slowly, the temperature on the first day was 102° and on the evening of the eighth day 104° F., having been at about 100° during the interval; on the evening of the eleventh day it was 105°; on the twenty-second day it dropped to 100° and remained at 99°–100° for five months.[3]

In experiments upon animals Tamassia[4] observed, after injection of 0.01 to 0.02 gm. of arsenic in solution, a fall of one-tenth degree (Centigrade) per minute in the first two minutes; in the following fifty minutes of one-tenth degree every two minutes, and in the last eight minutes a fall of one-tenth degree per minute. The range was from an initial temperature of 39°.6 (103°.3 F.) to that at death of 36°.4 (97°.5 F.), a fall of 3°.2 (5°.76 F.).

Unusual Symptoms.—In some instances the convulsions exhibit a marked tetanic character. Indeed in some of the cases recorded the symptoms have so much more closely resembled those of strychnin than those of arsenical poisoning that it is difficult to avoid the suspicion that strychnin may not have been taken along with arsenic.

Foster[5] relates the case of a woman of thirty-seven years who was seized with violent vomiting, burning pain at the pit of the stomach, constant tenesmus, with dejection of mucous stools. She had violent delirium and tetanic convulsions of such severity as to require the efforts of four persons to hold her in bed. The conjunctivæ were intensely injected, the eyeballs fixed upward and the pupils from being

[1] Day: N. Y. Med. Jour., 1885, xli., 189.
[2] Durell: Boston M. and S. Jour., 1889, cxxi., 200.
[3] Stanly: Amer. Lancet, 1890, n. s., xiv., 446.
[4] Friedr. Bl. f. ger. Med., 1878, xxix., 140; *ex* Riv. sper. med.-leg., 1876, 77.
[5] Lancet, 1840–41, ii., 305.

minutely contracted became much dilated. The mouth was drawn in all directions, exhibiting a most horrid spectacle. This condition continued for some minutes, when she became quite calm and died three and one-half hours after taking the poison. At the autopsy the mucous membrane was found intensely inflamed and a white powder, which proved to be arsenic, adherent to it in places.

Thompson [1] describes the non-fatal poisoning of eight persons at the same meal. All had vomited, and had epigastric pain and some diarrhœa. Two women of about twenty-five years were in violent convulsions in about eight hours with the hands clenched, the feet spasmodically extended, the jaws locked, and occasional opisthotonos. One of these women again had tetanic spasms early the following morning. One of the men, aged nineteen years, had tetanic spasms on the evening of the first day (the poisoned meal was eaten at midday) and early the following morning; during the following day he had frequent violent tetanic seizures, struggling furiously at times and requiring four men to prevent him from harming himself.

Donnelly [2] relates the case of a girl of eleven years who attempted to destroy herself with arsenic, in whom, six hours after taking the poison, there were rigid spasms of the muscles of the arms and legs, the heels and tendones Achillis being very painful. Arsenic was detected in the urine.

Jones [3] describes a case more closely simulating strychnin poisoning. A man of twenty-two years took nearly a tablespoonful of arsenic in cold coffee after a hearty meal, presuming it to be quinin. In five minutes he suffered from dizziness, slight distress in the stomach, great swimming in the head, vertigo, retching, slight vomiting, slight general pain and spasms. During the first hour he only vomited once, but had three or four fits or spasms which grew more severe, it requiring three men to hold him in bed. Vomiting was provoked by an emetic. In the third hour there were furious convulsions, succeeding each other rapidly, so that it was impossible to flex any joint by force; the jaws were locked, the patient entirely unconscious, the tongue swollen, and the eyes bloodshot. After hypodermics of morphin there were no convulsions; the spasms gradually subsided; there was profuse perspiration, and six hours later not a muscle moved. After a peaceful night's sleep he was perfectly conscious and complained only of general soreness and a painfully distended bladder.

Kortum [4] gives an extended account of the poisoning by a girl of her father and two brothers, in which, from the moral evidence and from the detection of arsenic in the alimentary canal of one of the victims and in articles of food and vessels, it is certain that arsenic was administered,

[1] Northwest. M. and S. Jour., 1851–52. viii., 89.

[2] Pac. M. and S. Jour., 1880–81, xxiii., 70.

[3] Virg. M. Monthly, 1875, ii., 194.

[4] Ztschr. f. Staatsarznk., 1833, xxvi., 1–72.

probably upon three occasions, while the probability of the accessibility of strychnin at that early date is extremely remote. The father suffered from violent vomiting, purging, and burning abdominal pain after the first administration; after the second, of which he took little, from vomiting; and after the third from violent pain, after which he was seized with tetanic spasms and pronounced opisthotonos. He shrieked so loudly that the assistants fled in panic terror, and when they returned they found him dead. One of the boys died about an hour afterward under similar circumstances.

McLeod,[1] in the year succeeding the discovery of strychnin, relates the poisoning of three maidservants by arsenic, in which all three suffered the characteristic gastro-enteric symptoms, while two on the fourth and fifth days exhibited marked tetanic symptoms, convulsive seizures, loss of speech and of the power of swallowing, locked jaws, etc. All ultimately recovered.

In a few cases the delirium which is frequently observed, particularly in the later stages of the poisoning, and in the cerebro-spinal form, becomes very violent. Thus one of McLeod's patients, in the instance just cited, on the evening of the fifth day was highly delirious, insensible of her situation, not knowing the attendants, and giving foolish answers to questions, and remained in this condition until early the following morning. Rademacher[2] relates the case of a man of forty-five years who during the entire third and fourth nights was sleepless and delirious. He had taken an ounce (31 gm.) of arsenious acid, but made a slow recovery.

In the following case reported by Stanly[3] the mental condition of the patient was probably due to causes other than the arsenic:

A girl of fourteen years took a large quantity of "rough on rats" with suicidal intent. When seen she was actively delirious; temperature 102°, pulse 110, respiration 25; abdomen tender; stomach not retentive. In three or four hours she became maniacal, struggled furiously with and bit at the attendants, using vulgar and abusive language, the face flushed, the eyes wild, and the expression painful. She was quieted by opium. The next day the delirium had almost passed away, but she was sullen and uncommunicative. The bowels were very loose. The subsequent recovery was very slow, she having paralyses, atrophy of muscles, and other symptoms of chronic arsenical poisoning.

[1] Edinb. M. and S. Jour., 1819, xv., 553–57.

[2] Richm. and Louisville M. Jour., 1873, xv., 383.

[3] Amer. Lancet, 1890, n. s., xiv., 446.

In this connection a curious case reported by Shipman[1] may be cited: A man who had been insane for between two and three years took a teaspoonful of arsenic trioxid with suicidal intent immediately after a hearty dinner. He vomited freely, had diarrhœa for a day or two, but no other symptoms referable to arsenic. After a week or so he suffered for several months with pains and swelling in the limbs, and paralyses, which might be attributed to the secondary action of arsenic. These, however, gradually disappeared with complete restoration of reason and ultimately of health.

Among the symptoms of unusual occurrence the following may be noted:

Pain in the mouth and pharynx, which is frequently observed in the later stages of the poisoning, has been noted as occurring early by Shafer,[2] Meriwether,[3] Kersten,[4] Powell,[5] and Delarue.[6] In the report last referred to, the burning sensation in the alimentary canal is described as being particularly in the pharynx. Clemens reports the case of a man who took a large quantity of arsenic, in whom the abdominal pain was limited to colic on the second day, and in whom the pain due to excoriations in the mouth, pharynx, and tongue was the only painful symptom.[7]

Salivation has been observed by Dunnell,[8] Greening,[9] Ogston,[10] Delarue,[11] Hamill,[12] and Netolitzky.[13]

The voice is frequently hoarse, and in some cases there has been complete *loss of the power of speech*,[14] or the difficulty of articulation has been such that only unintelligible sounds or unconnected words were uttered.[15]

Disturbances of vision and audition have also been observed. Intolerance of light, usually with dilated pupils, has been noted by Ramsay,[16] Cornish,[17] Morton,[18] and Donnelly;[19]

[1] Amer. J. M. Sc., 1843, vi., 520.
[2] Nelson's North. Lancet, 1853–54, viii., 145.
[3] Transylv. J. M., 1829, ii., 233.
[4] Deutsche Klin., 1851, iii., 393.
[5] Westm. Hosp. Rep., 1889, v., 215.
[6] Journ. d. chim. méd., etc., 1858, 4 s., iv., 107.
[7] Würzb. med. Ztschr., 1865, vi., 52.
[8] N. Y. Med. and Phys. Jour., 1829–30, n. s., ii., 114.
[9] Lancet, 1835, i., 812.
[10] London M. Gaz., 1851, n. s., xii., 181.
[11] *Loc. cit.*
[12] Chicago M. Exam., 1865, vi., 643.
[13] Prag. med. Wchnschr., 1876, i., 225.
[14] McLeod: Edinb. M. and S. Jour., 1819, xv., 553. Morley: Br. M. Jour., 1873, i., 88.
[15] Beck: Am. J. M. Sc., 1841, n. s., i., 57. Hartmann: Cleveland M. Gaz., 1859–60, i., 201. Cohn: Arch. f. Kinderhlk., 1868, vii., 418.
[16] Am. J. M. Sc., 1834, xv., 259.
[17] Lancet, 1849, i., 35.
[18] M. Reptr. W. Chester, Pa., 1855, iii., 55.
[19] Pac. M. and S. Jour., 1880–81, xxiii., 70.

total blindness by Vogler[1] and Morley;[2] strabismus and double vision by Kersten[3] and "disordered vision" by Bridges.[4] Tinnitus aurium has been observed by Schumacher[5] and Roché,[6] and total deafness was one of the symptoms observed in the case of William Chapman.[7]

Respiration is in many instances seriously interfered with, particularly toward the approach of a fatal termination, and death may be due to asphyxia. (See method of death below.)

Although arsenic has been frequently taken or administered with the view of producing abortion, and although it has been known to bring on menstruation in non-pregnant women,[8] in one case even in a woman of fifty years who had not menstruated for five years,[9] it has been repeatedly taken by pregnant women, many of whom have died from its effects, without causing the expulsion of the fœtus.[10] In one case reported by Edwards,[11] the woman recovered, and three months later the fœtus in utero was vigorous. Three cases are, however, recorded in which the woman died of the poisoning after having been delivered of a living child.[12] In a case in which arsenic was introduced into the vagina of a woman eight months pregnant, for the purpose of producing abortion, both mother and child were saved.[13]

Acute arsenical poisoning is frequently followed and sometimes preceded by chronic symptoms. Even when a single

[1] Ztschr. f. Staatsarznk., 1846, lii., 276.

[2] Brit. M. Jour., 1873, i., 88.

[3] Deut. Klin., 1851, iii., 393.

[4] Am. Med. Bi-monthly, 1879, x., 298.

[5] Wien. med. Presse, 1868, ix., 11.

[6] Bull. Soc. méd. de l'Yonne, 1871, xii., 59.

[7] Trial of Lucretia Chapman, Du Bois' report, p. 33.

[8] "Med.-ger. Beobacht.", 1789, ii., 361. Taylor: Guy's Hosp. Rep., 1837, ii., 77. Demuth: Ver.-Bl. d. pfälz. Aerzte, 1887, iii.

[9] Ramsay: Am. J. M. Sc., xv., 1834, 260.

[10] Elwell: "Malpr. and Evid." (1860), 505 (Peo. *v.* Eldridge); six to eight weeks, death. Paterson: Edinb. M. J., 1857, iii. 393 (Case III.), three months, death. Lesser: "Atl. d. ger. Med.," 89, about four months, death. Paterson: *Loc. cit.*, (Case I.), five months, death. De Bartolome: Prov. M. and S. Jour., 1849, 72, five to six months, death. Beck: Am. Jour. M. Sc., 1841, n. s., i., 57; six months, death. Taylor: Guy's Hosp. Rept., 1850–51, n. s., vii., 186; eighth month, death. Greaves: London M. Gaz., 1850, n. s., x., 677; ninth month, death.

[11] London M. and Phys. Jour., 1823, lxix., 117.

[12] Christison: Edinb. M. and S. Jour., 1828, xxix., 23. Brown: Assoc. M. Jour., London, 1853, 878. Elwell: "Malpr. and Evid." (1860), 501 (Rex *v.* Wishart).

[13] Schallgruber: Ztschr. f. Staatsarznk., 1825. Erg.-hft. iv., 302.

large dose has been taken, the acute poisoning has gradually merged into a chronic form in which death has finally resulted or from which a slow recovery has been made. A similar condition might be produced by the administration of a large, though non-fatal dose followed by smaller quantities repeated at intervals. The occurrence of acute symptoms following those of chronic poisoning indicates the administration of a larger dose to a person already under the influence of arsenic either by previous repeated small doses, by exposure to the inhalation of arsenical fumes, or otherwise. A case of this character was the subject of inquiry in Germany.[1] (See Chronic Arsenical Poisoning.)

When arsenic is applied to the skin or when it is introduced by a channel other than the mouth, the symptoms are the same, with the addition of other manifestations due to its local action. When it is applied to the head or neck the face swells greatly, and there is severe pain at the point of application and in neighboring parts, with the formation of a well-defined slough. Local paralysis of neighboring parts has been observed.[2] When applied to or about the scrotum, the cuticle separates and the testes enlarge and become extremely painful.[3] When introduced into the vagina, local inflammation, ulceration, and vaginal discharge are added to the usual symptoms.

Mode of Death.—Death from acute arsenical poisoning usually occurs either from exhaustion and heart failure, in collapse, or in coma from central paralysis, according as the gastro-enteric or the cerebro-spinal action predominates. In such cases the death is gradual, easy, and quiet, however much the patient may have previously suffered. Instances, however, are by no means infrequent of deaths from arsenic attended by violent manifestations. Cases in which death has occurred during violent convulsions have been reported by Crowfoot,[4] Coates,[5] Ryan,[6] Paterson,[7] Friedreich,[8] and others. Deaths during violent paroxysms of pain, consciousness remaining perfect to the

[1] Vrtljschr. f. ger. Med., 1871, n. F., xiv., 1.

[2] Prentiss: Therap. Gaz., Detroit, 1892, 3 s., viii., 105.

[3] Michell: Med. Times and Gaz., 1853, n. s., vii., 62.

[4] Med. and Phys. Jour., London, 1815, xxxiv., 441.

[5] London M. Gaz., 1837, xx., 308.

[6] Lancet, 1851, i., 410.

[7] Edinb. M. J., 1857, iii., 394, 395 (two cases).

[8] Friedr. Bl. f. ger. Med., 1862, xiii., 353.

last, have been recorded by Kaiser,[1] Grabadier,[2] and Colting.[3] On the other hand, Wright[4] relates the case of a woman who died in sleep four hours after having taken an ounce (31 gm.) of white arsenic. Death in several cases has occurred suddenly and unexpectedly. Thus Christison[5] reports the case of a girl of fourteen years who suddenly became blue in the face, seemed to be fainting, and expired without a struggle; Taylor[6] that of a woman of twenty-five years who sat up in bed, and, while in the act of drinking, had a slight convulsive fit, and died almost instantly; Dymock[7] that of a woman of twenty-five years who walked across the room unassisted, sat down, slipped to the floor, was put to bed again, and died without a struggle; Wormley[8] that of a man of seventy years who got up to stool alone and fell dead, so that at the time the death was attributed to apoplexy; and Graham[9] that of a woman who had been treated for cancer of the breast by a quack, who called out suddenly "there it is again" (meaning a paroxysm of pain), went into a fit, and died immediately.

In some cases death is due to asphyxia. Thus in the case of Soufflard[10] during the last hours the interference with respiration was the predominant phenomenon; two hours before death the symptoms of asphyxia declared themselves in the highest degree; and in the last moments there were no convulsions, no cerebral symptoms, nor any disorder of the nervous system.

Death may also result from secondary effects, the arsenical poisoning being the remote but not the immediate cause. Thus the case is reported[11] of a man who was found dead, hanging partly out of a window; he had vomited, the mouth was open and the face blue. Arsenic was proved to have been administered and the stomach was found inflamed, but a ruptured aneurism of the aorta was also discovered at the autopsy.

[1] Ztschr. f. Staatsarznk., 1827, xiii., 264.
[2] Oest. Ztschr. f. prk. Hlk., 1866, xii., 172.
[3] Boston M. and S. Jour., 1838, xviii., 78.
[4] Lancet, 1829, ii., 612.
[5] Tr. M.-Chir. Soc., Edinb., 1826, ii., 298.
[6] Guy's Hosp. Rep., 1837, ii., 68.
[7] Edinb. M. and S. Jour., 1843, lix., 350.
[8] Ohio M. and S. Jour., 1863, xv., 467.
[9] Glasgow M. Jour., 1868–69, n. s., i., 56.
[10] James: Bull. Ac. Med., 1838–39, iii., 664.
[11] Mag. f. ger. Arzneiwis., 1832, i., 222.

Diagnosis.

As acute arsenical poisoning does not present any symptom peculiar to itself, unless it be the existence of arsenic in the urine, diseased conditions arising from natural causes, particularly those affecting the alimentary canal, which present resemblances to the effects of arsenic which frequently render a positive diagnosis difficult, if not impossible, from the symptoms alone.

Acute Gastritis, Enteritis, and Gastro-Enteritis.—Catarrhal inflammation of the intestines is of frequent occurrence. In its diffuse form affecting the whole or nearly the whole alimentary canal, it is usually not an essential disease, but is symptomatic, as an accompaniment of measles, scarlet fever, cholera, etc. When the inflammation is limited to the lower small intestine and upper large intestine (ileo-colitis), to the duodenum (duodenitis), or to portions of the lower intestine (typhlitis, appendicitis, proctitis), the diagnosis may be easily made from the history of the onset, the existence of high temperature and other febrile symptoms, the localization of the pain, the absence of pain in the throat, and the duration and course of the disease.

It is now generally conceded that idiopathic gastritis and gastro-enteritis do not occur, that when they exist they are the results of some obvious cause, to the action of a thermal, mechanical, or chemical irritant or corrosive. The theory of possible idiopathic gastro-enteritis is, however, frequently advanced by the defence in cases of alleged homicide by arsenic. Taylor[1] states that "the case of Reg. *v.* Hunter (Liverpool Spring Assizes, 1843) was successfully defended on the theory of gastro-enteritis from natural causes, in spite of the strongest suspicions that arsenic was the cause of death." In the Maybrick case one of the witnesses for the defence expressed the opinion that gastro-enteritis might be produced by "getting wet."[2]

[1] "Poisons," 3d Am. ed., 97.

[2] Dr. McNamara, of Dublin, in the Maybrick case testified for the defence that gastro-enteritis may be produced without the introduction of any substance from the outside. In answer to a question he said: "I can perfectly believe that a wetting, coupled with neglect of precautions and a weak stomach and circulation, may produce these consequences. Q. Then do you mean to say that by

It being conceded that gastro-intestinal inflammation does not arise spontaneously, the differentation of *causes* becomes the only important point of diagnosis. Thermal causes, such as overheated fluids, or even large quantities of ice water in some cases, are most unusual. The former would produce scalds in the mouth and lips if sufficiently hot, and the history of the taking of either would usually be clear.

Gastro-enteritis produced by mechanical irritants presents symptoms practically identical with those of arsenical poisoning. Maschka[1] relates the following case:

J. B. on April 13th mixed about a tablespoonful of pounded glass in coffee which was taken by R. F., a woman of seventy-six years. After she had drunk the coffee and noticed a considerable glassy sediment, she was seized with abdominal pain and diarrhœa, which ceased toward the morning of the 14th. On the 15th he again mixed two tablespoonfuls of pounded glass in a soup of which R. F. took a part and again noticed the peculiar sediment, which she collected and submitted to the authorities. On the night of the 15th and 16th she suffered severe abdominal pain and had a frequent diarrhœa. On the 17th she complained of headache, vertigo, loss of appetite, and increased thirst. The tongue was dry and coated white; the epigastrium very tender to the touch; the abdomen distended and painful; the urine voided only in drops; the skin dry; and the pulse small and irregular. On the 18th the diarrhœa was diminished, the pains less, and the symptoms gradually improved until she was completely restored to health April 27th. The sediment was found on examination to be coarsely powdered glass.

The effects of other chemical irritants and corrosives may be distinguished from those of arsenic by the existence of other attendant symptoms, salivation and pain in the mouth in the case of mercurials, corrosion of the mouth and lips by the corrosives, lumbar pain in poisoning by oxalic acid, the appearance of the urine and of stains with carbolic acid, the luminous appearance of the vomit and the garlic taste in the mouth with phosphorus, etc. It is clear that the diagnosis is made positive

getting wet this illness of gastro-enteritis, this acute inflammation, may be produced in the stomach and bowels? A. That I think is the evidence I have given." In this opinion Dr. McNamara differs from Professor Tidy, also called for the defence, from all the medical witnesses called for the crown, and from all writers upon the subject of whom we have knowledge.

[1] "Samml. ger. Gutacht. Prag. med. Fak.," ii., 1858, 213.

by the detection of one or another of these poisons in the urine or vomit, by an analysis conducted with proper precautions, in the absence of its medicinal administration.

Gastro-enteric symptoms are produced by poisonous foods, sausage, mussels, decayed fish, meat, etc., and cheese. In these, however, a number of persons are usually attacked simultaneously, and the source of the poisoning is directly suspected; the interval preceding the manifestation of symptoms is usually longer than with arsenic, and with all, except in some cases of cheese poisoning, there are nervous symptoms and effects upon the eye which are not, or but most exceptionally, met with in arsenical poisoning. Cheese poisoning, which more nearly resembles arsenical poisoning, although frequently attended with very severe symptoms, has, so far as we know, been fatal in one case only, that of a child of three years who was poisoned along with four other persons.[1]

In a homicidal case[2] in which the arsenic was administered spread upon cheese, which was described as spoiled and stinking, and in which neither symptoms nor post-mortem appearances were well marked, Hofmann very properly considered cheese poisoning as a possible factor in the cause of death.

Asiatic Cholera.—Although the statement of Taylor[3] to the effect that the symptoms with arsenic and other irritant poisons are wholly different from those of Asiatic cholera, excepting the intense thirst which is present in both, is true so far as it relates to the ordinary type of arsenical poisoning either in its gastro-enteric or its cerebro-spinal form, it does not hold good with regard to the rapidly fatal variety, designated as *asphyxia arsenicalis*, which in many respects bears a striking resemblance to an attack of Asiatic cholera. In cholera, however, purging usually precedes vomiting in time, which is never the case with arsenical poisoning. The stools in cholera rapidly assume the characteristic "rice-water" appearance—liquid, whitish, grumous, resembling whey or a decoction of rice or meal, nearly clear, exceptionally tinged with blood or bile, and emitting an insipid spermatic odor, and are discharged in an almost continuous, involuntary jet. These differ entirely from

[1] Gläsel: Ugeskr. f. Läger, Kjøbenh., 1879, 3 R., xxvii., 309.

[2] Case of Françiska Schmidmaier, Friedreich's Bl. f. ger. Med., 1862, xiii., 353; Ztschr. f. Staatsarznk., 1864, lxxxvii., 260.

[3] "Poisons," 3d Am. ed., 94

the usual feculent, highly colored, fetid stools of arsenical poisoning, discharged with straining and tenesmus. Nevertheless cases of arsenical poisoning in which the alvine discharges have assumed the "rice-water" character have been reported by Wyss,[1] Taylor,[2] Wilks,[3] Fox,[4] Headland,[5] Storer,[6] and Gairdner.[7] Of these cases Headland's recovered, Storer's died in thirty-six hours, and the others died in fourteen and one-half hours or less. In cholera the voice has a peculiar, rough, whistling tone (vox cholerica) which has been observed in arsenical poisoning but once.[8] According to Tardieu[9] the eruption in cholera is limited to the forms of roseola, erythema, or urticaria, while in arsenical poisoning it takes on the most manifold forms, and to it the petechial form belongs almost exclusively.

In cases of death the diagnosis may be established by the results of the autopsy. After death from arsenic the venous congestion which is observed in cholera cadavers is not present. The serous surfaces after death from cholera are found coated with a sticky, viscid exudation, which is absent after death from arsenic. The blood in the vessels of those who have died from cholera is thicker than normal, usually contains small clots, coagulates slowly, and the separation into clot and serum is very imperfect. Virchow[10] has described coagula found in the heart in cholera, which extend from the right heart into the pulmonary artery, and from the pulmonary veins into the left heart.[11]

If the cholera bacillus of Koch were peculiar to cholera it is clear that its identification, either ante or post mortem, would prove the existence of that disease.

It is only during epidemics of cholera, or in India where it is endemic, that a diagnosis between it and arsenical poisoning is of interest, but instances are not wanting in which poisoners have availed themselves of a cholera epidemic to more safely murder by arsenic.

Jackson[12] makes mention of the exhumation in 1858 of the body of

[1] Arch. d. Heilk., 1870, xi., 15.
[2] Guy's Hosp. Rep., 1851-52, 2 s., vii., 194.
[3] *Ibid.*, 1855, 3 s., i., 364.
[4] Lancet, 1848, ii., 503.
[5] *Ibid.*, p. 697.
[6] Boston M. and S. Jour., 1841, xxiii., 345.
[7] Edinb. M. and S. Jour., 1829, xxxii., 305.
[8] Maclagan: London and Edinb. Monthly J. M. Sc., 1852, xiv., 132.
[9] "Empoissonnement," 2ème ed., 369.
[10] Med. Reform, 1849, No. 12, 82.
[11] See Kraus: Friedreich's Bl. f. ger. Med., 1888, xxxix., 278.
[12] Extr. Rec. Soc. M. Impr., Boston, 1859, iii., 291.

a man who had died in 1849 of a disorder supposed to have been Asiatic cholera, in which an abundance of arsenic was found. The finding of arsenic in the cadaver of another supposed victim, who died in 1850, led to this investigation.

Niemann[1] gives an account of the case of Bernh. Hartung, who was executed for having poisoned his aunt during a cholera epidemic. His first wife and mother-in law had died during a previous outbreak, and his second wife twenty months after the first, having suffered from the symptoms of arsenical poisoning. The bodies of the aunt and second wife were exhumed; four and one-half grains (0.28 gm.) of arsenic trioxid were obtained from the stomach of the former, and 6.08 grains (0.38 gm.) from the cadaver of the latter. Keber[2] describes a case of homicidal poisoning by arsenic of a person suffering from Asiatic cholera, in whose cadaver both arsenic (although in small quantity) and the lesions of cholera were found. In this case it was difficult to decide which was the cause of death. The defendant was convicted of an attempt to murder.

Tardieu[3] relates the case of a young girl who was supposed to have committed suicide by taking an arsenical green during the cholera epidemic of 1849. The autopsy and analysis showed that she had died of the disease and not of poison.

In the case of the Duc de Praslin, who committed suicide by taking a large dose of arsenic, the symptoms were at first attributed by the attending physicians to an attack of cholera.[4]

Taylor[5] also cites the cases of Reg. *v.* Chesham (1847) and Reg. *v.* Foster (1847) in which arsenical poisoning was mistaken for cholera, and the fact of poisoning remained concealed until an analysis was made.

Cholera Morbus—Cholera Nostras.—The diagnosis between this disorder and arsenical poisoning is more difficult than in the case of Asiatic cholera, and it is questionable whether a positive distinction can be made from the symptoms alone. Cholera morbus occurs in the summer and autumn, usually follows some indiscretion of diet, and does not attack all or several persons eating of the dish or article of food which provokes the outbreak. It is very rarely fatal except in the very young and the very old, and, when it is fatal, death only occurs in three or four days from exhaustion, while in that form of arsenical poisoning whose symptoms simulate those of cholera death follows rap-

[1] Ztschr. f. Staatsarznk., 1862, lxxxiv., 153.

[2] Vrtljschr. f. ger. Med., 1863, xxiv., 131.

[3] "Empoisonnement," 2ème ed., 36.

[4] J. des conn. méd.-chir., Oct., 1847. Reprint.

[5] "Poisons," 3d Am. ed., 96.

idly, usually in less than twenty-four hours. In cholera morbus, moreover, the throat symptoms usually observed in arsenical poisoning are absent. The detection of arsenic in the urine, vomit, or dejecta is the only ground for a positive diagnosis.

Peritonitis.—The onset of peritonitis resembles that of arsenical poisoning. There is usually no initial rigor. Pain, severe, burning or boring in character, at first located in some particular part of the abdomen, rapidly extends over a large area; vomiting, great thirst, diminished or suppressed urine, cramps in the legs are common to both. The chief points of distinction during life are the localization of the early pain in the abdomen elsewhere than in the region of the stomach; the almost constant occurrence of constipation, the absence of throat symptoms, the rapidly increasing tympanites producing a deep resonant note on percussion, and great diminution of the normal dulness of the liver and spleen, and sometimes an elevation of temperature in peritonitis.

The autopsy will remove all doubt; the injection of the peritoneum, sometimes very intense, extravasations, the thin grayish-yellow coating of plastic lymph covering and in places causing adhesions of the peritoneal surfaces, and the serum collecting in the peritoneal cavity to a greater or less extent in peritonitis are not observed after death from arsenic. Moreover, peritonitis is almost invariably the result of a cause detectable at the autopsy: a perforation of the stomach or intestine, rupture of a hydatid cyst of the liver, strangulated hernia, cancer, cystic and other tumors, tubal or abdominal pregnancy, etc., or of an altered condition of the blood, as in septicæmia, pyæmia, Bright's disease, small-pox, and other eruptive fevers.[1]

Intussusception—Ileus.—Although these disorders, due either to the invagination of one portion of the intestine into another or to its strangulation within the abdomen, are attended by the most obstinate constipation, Taylor states[2] that in one case at least intussusception in an infant was mistaken for arsenical poisoning, and the mistake nearly led to the conviction of the mother and grandmother of the child on an unfounded charge of murder (Reg. *v.* Dore and Spry, Central Criminal Court, August 28th, 1848).

[1] See also Schuchardt, in Maschka's "Handb.," ii., 34 *ff.* Tardieu and Roussin: "Empoisonnement," 2ème ed., 28.

[2] "Poisons," 3d Am. ed., 99.

Method of Action.

The views of authors concerning the corrosive action of arsenic have differed widely at different times. The older writers attributed to it a corrosive power but little inferior to that of the mineral acids. In the Affaire Mercier in 1839, Rognetta[1] advanced as one of the reasons in opposition to the theory of arsenical poisoning that there was no cauterization of the mouth and tongue of the deceased, which he claimed to occur as a characteristic of arsenical poisoning. At a later date Taylor held the opposite view that arsenic is not a corrosive. Concerning it he says: "Arsenic is an irritant poison; it has no decided chemical or corrosive action on the animal tissues, and the changes met with in the stomach and bowels of a person poisoned by it are referable to the effects of inflammation. I have not found that arsenic produces any effect on dead mucous membrane."[2]

The truth lies between these extreme views. While arsenic is not a corrosive in the sense in which we have used the word,[3] since it does not produce death by its chemical action upon a tissue with which it comes in direct contact, but always as a true poison—that is, after entrance into the blood, and by its subsequent action upon tissues, including the alimentary canal, to which it is carried by the circulation—it certainly does exert a local chemical action, and does produce alterations in dead mucous membranes as well as in living. Its corrosive action is, however, secondary to and independent of those powers which render it so powerful a poison.

The local chemical action of arsenic has been utilized from early times in the various depilatory and escharotic preparations of which it is an ingredient: Pulvis arsenicalis Cosmi, Dupuytren's caustic powder, Plunket's caustic, Hunter's caustic, Rousselot's powder, Esmarch's paste, Sir Astley Cooper's ointment, Marsden's mucilage, and Hebra's caustic—all contain arsenic in the form of the trioxid. When applied to the skin it produces a pustular eruption and severe pain. Thus Wagner[4] relates a fatal arsenical poisoning caused by washing the entire

[1] Gaz. d. hôp., 1839, 2 s., i., 581.
[2] "Poisons," 2d ed., 1859, 355; 3d Am. ed., 1875, 288.
[3] See Definition, p. 43.
[4] Wchnschr. f. d. ges. Hlk., 1839, viii., 213.

body with a solution of arsenic trioxid. It immediately produced severe pain all over the body, as if the person were laid on a fire or upon needles, which constantly increased in spite of the external application of remedies, and over most of the body the cuticle was raised in blisters. Where arsenic is applied to mucous membranes it produces a local inflammation, followed by the formation of a well-defined eschar. This has been repeatedly noted in the cases of extra-gastric administration already cited (see p. 412), and in some instances in the mouth when arsenic has been swallowed; not only as a late symptom and in chronic poisoning, but also, as in the case of Soufflard, who poisoned himself with 12 gm. of arsenic, as one of the earliest manifestations. Dr. James, who saw him very shortly after he took the poison, says that the lower lip was strongly cauterized and extremely painful to the touch.[1] All doubt as to the nature of the poison taken is removed by the fact that Orfila separated from the stomach, liver, etc., of Soufflard a quantity of arsenic such that he estimated the amount absorbed at four to five grains. At the post-mortem also, the gums, inner surface of the cheeks, palate, fauces, uvula, were found to be bright red; the lower lip strongly cauterized, of double its natural size; the tongue suburral, its epithelium destroyed in places, leaving the enlarged and reddened papillæ exposed and greatly swollen. Death occurred in thirteen hours.

Nor can it be doubted that arsenic has a distinct action upon dead mucous membrane. This subject will be discussed under the head of "Post-mortem Appearances" (*q. v.*).

On the other hand, the inflammation of the alimentary canal which is the cause of the intense gastro-intestinal disturbances observed in arsenical poisoning, is produced only in part, and probably only in a comparatively slight degree by the local or corrosive action. This is shown to be the case by numerous observations of the symptoms of gastro-enteritis during life, produced by arsenic which has not been taken by the alimentary canal, but by application to the skin or introduction into the vagina, etc. In these cases the post-mortem appearances of the stomach and intestine are absolutely identical with those ob-

[1] Bull. Ac. méd., 1838–39, iii., 664. "La lèvre inférieure avait été fortement cauterizée, car sa membrane muqueuse était blanche, fendillée, et le moindre attouchement y provoquait une excessive douleur."

served when arsenic has been taken by the mouth.[1] In several instances of extra-gastric administration arsenic has also been found in the stomach, although the quantity has always been quite small.[2] These observations, taken in conjunction with the fact that an interval of about half an hour or more elapses in all but the most exceptional cases between the taking of the poison by the mouth and the manifestation of symptoms, prove conclusively that the violent gastro-enteric symptoms of arsenical poisoning are produced in some way by arsenic which has entered the circulation, rather than by that which is placed in contact with the gastro-intestinal mucous membrane.

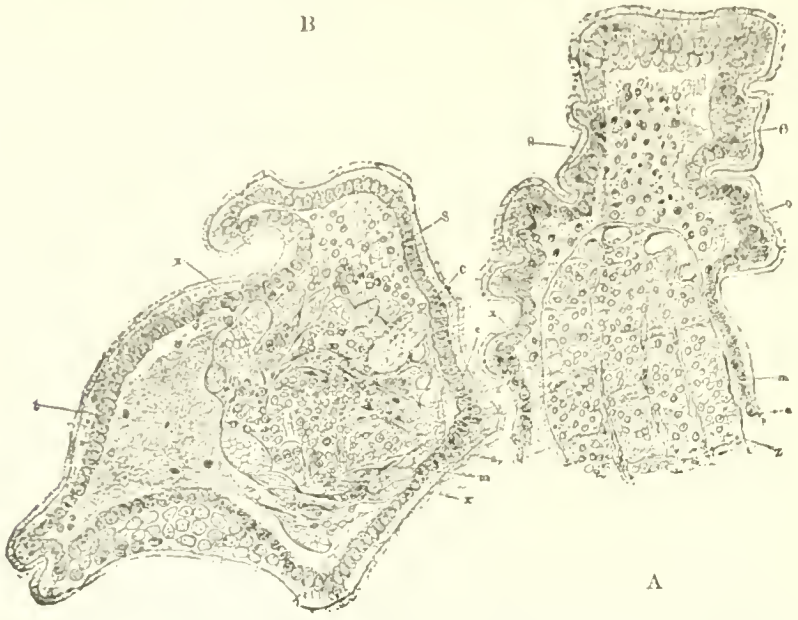

FIG. 10.—Villi of Duodenum. Dog. × 320. A, vertical section; B, transverse section; (*z*) body of villus, with (*a*) adenoid elements, (*m*) muscular fibres, (*c*) capillaries, (*g* A) adenoid-like tissue, with round cells, separating end of villus and epithelial layer (*e*), which still adheres at *x*; (*t*, B) large transudation coagulum, with a few adenoid cells between epithelium and body of villus; (*s*, B) somewhat oblique section of adenoid-like tissue. (Pistorius.)

The method by which the inflammation is produced has been variously interpreted. Boehm and Unterberger[3] conclude from their experiments that arsenic trioxid injected into the circulation causes great diminution of the blood pressure, which may be attributed partly to paralysis of the abdominal vessels and

[1] See Boehm and Unterberger: Arch. f. exper. Path. u. Pharm., 1874, ii., 96.

[2] Taylor: Guy's Hosp. Rep., 1864, 3 s., x., 220. Brisken: Vrtljschr. f. ger. Med., 1864, xxv., 110. Tidy: Lancet, 1878, ii., 250 (two cases). Boehm and Unterberger: *Loc. cit.*, p. 98 (experiments upon animals).

[3] *Loc. cit.*, 89–98.

partly to a diminished force of the heart muscle; and they attribute arsenical gastro-enteritis to a vasomotor paralysis of the terminations of the sympathetic, resulting in diminished blood pressure and great congestion of the abdominal viscera, which, extending to the gastro-intestinal mucous membrane, there produces the appearances of intense gastro-enteritis. Under the direction of Boehm, Pistorius[1] has shown by experiments upon animals that arsenic trioxid, administered by the stomach or hypodermically, causes congestion of the vessels of the intestinal villi and the formation of a false membrane, consisting of amorphous coagulated masses, which enclose large numbers of

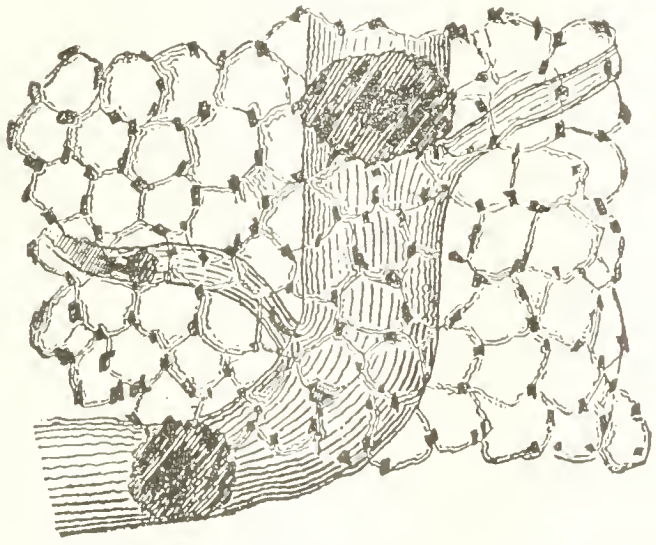

Fig. 11.—Numerous Thrombi in the Capillary Plexus and two large Plugs in a Larger Vessel. The capillary thrombi very numerous and of varying size. × 80. (Heilbronn.)

epithelial and adenoid cells, and which, being thrown out between the body of the villus and its epithelial covering, causes the detachment of the latter, shreds of which are found in the contents of the gut (Fig. 10). Lesser[2] attributes the diarrhœa to an irritation of the ganglia located in the intestinal walls. Heinz at Breslau,[3] and Heilbronn at Würzburg[4] have simultaneously given another explanation of the method of production of arsenical gastro-enteritis by showing that arsenic produces thrombi in the minute blood-vessels. They differ, however, in

[1] Arch. f. Path. u. Pharm., 1883, xvi., 188.

[2] Arch. f. path. Anat., etc., 1881, lxxxiii.

[3] "Natur und Entstehungsart der bei Arsenick-Vergiftung auftretenden Gefäss-Verlegungen," Diss., Breslau, 1891.

[4] "Veränderungen im Darme," etc., Diss., Würzburg, 1891.

that the former found that the coagulation was always in the small arteries and veins, never in the capillaries, while the latter found the coagulation to take place in the capillaries also (Fig. 11).[1] It is probable also, from the presence of arsenic, albeit in small quantity, in the stomach after its external application, that the mucous membrane of that organ and of the intestines performs to some extent an excretory function for absorbed arsenic, an action which would necessarily be attended with irritation and inflammation. Finally Binz and Schultz[2] have offered an original and ingenious explanation of this and other actions of arsenic on the animal economy. They found that the protoplasm of many tissues, intestine, brain, spleen, etc., have the power of setting the oxygen atoms combined with arsenic in alternating motion, of converting arsenious into arsenic acid, and reversing the process. According to them this continuous series of oxidations and reductions within the albuminoid molecule finally causes the disintegration of the cell and fatty degeneration. (See Chronic Poisoning.)

As no one of these methods of action is inconsistent with the others it is quite probable that all are operative, aided in some slight degree by the local action.

Aside from its action upon the alimentary canal arsenic also produces serious modifications in the normal chemism of the body. It has been shown by Saikowski[3] to cause fatty degeneration in the kidneys, liver, heart, diaphragm, and stomach. In consequence of fatty degeneration in the tissues of the blood-vessels multiple hemorrhages occur in different organs. Death by heart failure is produced by degeneration of the heart muscle, which, with large doses, may also involve the heart ganglia. The occurrence of coma, delirium, and eclamptic seizures may be traced to the degeneration of cerebral ganglia and to imperfect nutrition, due to diminished blood pressure. When the dose is too small to cause a degeneration sufficiently extensive or complete to impede the function of heart or brain the case terminates in recovery, or merges into a chronic form which is frequently observed to follow an acute poisoning. The skin symptoms produced by arsenic are due to the excretion of small

[1] A similar effect is produced by other poisons: cyclamin, sapotoxin, ricin, and abrin.

[2] Arch. f. exp. Path. u. Pharm., 1879, xi., 200.

[3] Arch. f. path. Anat., 1865, xxxiv., 73.

quantities of the poison by the skin and consequent irritation of that organ.

As arsenic readily replaces phosphorus in organic compounds it is quite within the possibilities that the phosphorized constituents of nerve tissue, protagon, nucleins, etc., may be converted into corresponding compounds containing arsenic in place of phosphorus. This has, however, not been proved to be the case.

Treatment.

When an arsenical compound has been taken by the mouth the objects of the treatment are: first, the removal as completely as practicable of the poison from the stomach, and second, the conversion of what remains into an insoluble or, at least, a very sparingly soluble combination.

The first indication is usually fulfilled more or less completely by the vomiting provoked by the poison itself. The physician may, however, be called in during the interval preceding the manifestation of symptoms, or vomiting may not have been free and copious. In that event the stomach should be thoroughly washed out by the pump or siphon, using several quarts of water. Even if the case be seen in a later stage, and there be reason to suppose that arsenic still remains in the stomach, this procedure should be resorted to. The same danger does not attend the introduction of the pipe into the stomach in arsenical poisoning as exists in corrosion by acids or alkalies; still, if some time have elapsed and the symptoms of irritation are violent, some caution is necessary in view of the possible existence of ulcerations in the stomach. The same end may be attained, though much less completely, by the administration of emetics, such as zinc sulfate, ipecacuanha, or apomorphin, never of tartar emetic. But even by a washing with a large volume of water the poison is frequently not completely removed by reason of the tenacity with which it adheres to the walls of the stomach, particularly if it have been taken when that organ is empty, and in the form of powder.

In no case should the fulfilment of the second indication by the administration of chemical antidotes be neglected. Of these the most efficacious is the normal ferric hydroxid, the use of which was first suggested by Bunsen in 1834, with the object of

the conversion of the arsenic trioxid into ferric arsenite,[1] a compound which, although not absolutely insoluble, is only very sparingly soluble.[2] To be of service this antidote must be freshly prepared, as it rapidly undergoes modifications which greatly diminish its capacity of combination. It should be administered in quantities of two to four tablespoonfuls, suspended in warm water, every ten minutes until the local symptoms abate, or until a quantity about twenty times as great as the dose of arsenic presumed to have been taken is reached. The greater portion of the arsenic taken should be removed from the stomach by the pump or siphon or by emesis before the administration of the antidote, which should be given even if the case have progressed for some time, with a view to neutralizing the action of particles of arsenic adherent to the walls of the stomach. For the preparation of the antidote a solution of normal ferric sulfate is retained in the United States Pharmacopœia, under the name of *Liquor ferri tersulphatis*, and in the British Pharmacopœia under the name of *Liquor ferri persulphatis*. From this the hydroxid is prepared when wanted by precipitation with ammonia in excess, after dilution with water, and subsequent washing on a muslin filter until the odor of ammonia is barely perceptible. Dialyzed iron has been used as a substitute, but although it has proved beneficial in some cases, it is not so trustworthy as the hydroxid prepared as directed in the Pharmacopœia.[3]

Another antidote, suggested by Bussy, in 1846,[4] is magnesium hydroxid prepared by mixing magnesia usta with twenty times its weight of water. This antidote, whose action depends upon the formation of the sparingly soluble mangesium arsenite, is preferred by some[5] because an excess is better borne by the stomach, because it acts as a cathartic to remove any of the poison which may have reached the intestine, and because it is more easily prepared and may be kept ready. It should be given in doses of four to six tablespoonfuls every fifteen minutes. The magnesia from which it is made must not have been

[1] "Das Eisenoxydhydrat, ein Gegengift der Arsenigen Säure," Göttingen, 1834.

[2] As to solubility of ferric arsenite see Schlagdenhauffen and Reeb: J. d. Pharm. v. Elsass-Lothr., Strassb., 1888, xv., 191.

[3] See Ferri oxidum hydratum, U. S. Ph.

[4] J. de pharm. et de chim., 1846, 3 s., x., 81.

[5] Kobert: "Intoxicationen," 255.

too strongly heated in its preparation. A combination of the two antidotes is officinal in the German Pharmacopœia under the title *Antidotum arsenici (Fuchsii)*, and in the United States Pharmacopœia under that of *Ferri oxidum hydratum cum magnesia*, and is prepared when wanted by mixing together 10 gm. of magnesia rubbed up with water, 6.5 gm. of liquor ferri tersulphatis previously diluted with twice its weight of water, and water to the total volume of one litre. This antidote possesses the powers of both of its constituents and has the advantage over the ferric hydroxid of being more rapidly prepared.

Although milk, melted butter, or lard have no chemical action upon arsenic, their administration, if the poison be taken in the solid form, may be of service in interfering with its solution, and consequently with its absorption.

Post-Mortem Appearances.

The post-mortem appearances after death from arsenical poisoning are the same, irrespective of whether it has been taken by the mouth or introduced through some other channel of absorption, except that in the latter event its local effects will be found at the point of application. The appearances differ somewhat, however, with variations in the nature of the symptoms, and according as the duration of the case has been short or has extended over several days.

The **external appearance**, if diarrhœa and vomiting have been profuse, as is usually the case, will be that of emaciation, although the quantity of subcutaneous fat remains undiminished. The skin is cyanosed, particularly that of the hands and feet, although by no means so deeply as after death from Asiatic cholera. The process of putrefaction is delayed, and the body remains odorless longer than usual. These effects are due to a diminution in the quantity of water in the cadaver.

In rapidly fatal cases it is rare that any change is met with in the **mouth, pharynx**, or **œsophagus**. Occasionally, however, they are the seat of inflammation, or even of ulceration.

Thus in Fahrenhorst's case[1] death occurred in six hours, and the pharynx and larynx were found to be inflamed. Booth[2] reported a

[1] Mag. f.d. ges. Hlk., 1825, xx., 483. [2] London M. Gaz., 1834, xiv., 62.

case of death in six and a half hours in which the œsophagus was inflamed to within half an inch of the cardia. Taylor[1] reported two cases, in one of which death occurred in seventeen hours, and the œsophagus was inflamed and the mucous membrane of the larynx and trachea highly injected. In the other the patient died in two and a half hours after taking one hundred and fifty grains (9.72 gm.) of arsenic in fine powder, and the œsophagus was found to be inflamed in places. In one of Rayner's cases[2] the gullet was highly inflamed. In a case reported by Knott,[3] in which the poison was taken in powder, suspended in brandy and water, the œsophagus was found vascular in the upper half to a distinct line, below which there were "gangrene of the mucous coat" and thinning of the tube to within about one and a half inches of the cardia. Greenhow[4] reported a case of death in six hours and ten minutes, in which the upper half of the œsophagus was found to be pale but the lower half purple, with silvery-white patches which could not be detached without injury to the mucous coat, and several slightly raised opaque granules at the cardiac end. In an alleged homicidal case Demuth[5] found a parchment-like drying of the mucous membrane at the angle of the lips. The exceptional post-mortem appearances of the lips, mouth, and tongue in the case of Soutllard have already been referred to.[6] In this case the pharynx and œsophagus were also found injected.

It is in the **stomach** that the most prominent lesions are observed. Its mucous membrane over a greater or less extent, or in spots, is swollen, thickened, somewhat spongy and covered with a thick, viscid mucus tinged with blood or bile. The color of the mucous surface is sometimes a uniform dull or brownish red, becoming brighter on exposure to air, and presenting a velvety appearance. In some cases the color has been described as "bright scarlet"[7] either throughout or in portions;[8] in others it has been said to be vermilion[9] uniformly or in places. Usually, however, the color is not uniform, the redness appearing in spots or patches, more frequently toward the greater end and chiefly on the prominences of the rugæ. Fre-

[1] Guy's Hosp. Rep., 1837, ii., 77; 1850–51, 2 s., vii., 183.
[2] Prov. M. and S. J., 1840, i., 155.
[3] Lancet, 1849, ii., 1.
[4] Med. Times and Gaz., 1878, ii., 626.
[5] Vereinsbl. f. pfälz. Aerzte, 1887, iii.
[6] See p. 421.
[7] Booth: London M. Gaz., 1834, xiv., 62. Foster: Lancet, 1840–41, ii., 305. Buchner: Friedreich's Bl. f. ger. Med., 1884, xxxv., 170.
[8] Dymock: Edinb. M. and S. Jour., 1843, lix., 350. Milford: Australian M. Gaz., 1889-90, ix., 90, 95.
[9] Foster: Lancet, 1840–41, ii., 305. Silliman: West. Journ. M. and S. 1850, 3 s., v., 49. Carter: Liverpool, Med.-chir. Jour., 1890, x., 130.

quently the redness is in the form of minute dots (petechiæ) arranged in striations stretching in curved lines between the cardia and pylorus, or scattered uniformly over the entire mucous membrane. This appearance, which is undoubtedly due to minute extravasations caused by the plugging of small vessels,[1] is claimed by Tidy to be characteristic of arsenical poisoning.[2] It is certain, however, that it is by no means always present after death from arsenic, and that it is frequently observed after death from other causes. Indeed, Professor Tidy, who laid great stress upon the absence of this appearance in the Maybrick case, himself goes no further in his work than to say that it is "generally" present.[3] Submucous extravasations of blood also occur frequently and, by their dark or even black appearance, may be mistaken for gangrene. When the body has been long buried the mucous membrane is frequently of a bright-yellow color, due to the formation of the yellow arsenic trisulfid, a change which has in one case been observed in as short a time as twelve days.[4] A pale ochry or bright-yellow color has also been observed when the post-mortem has been made twenty-one and twenty-seven hours after death;[5] but in these cases the color is due to a thick coating of mucus more or less tinged with bile, on scraping which off the highly reddened mucous membrane is exposed. Ulcerations of the stomach are not of frequent occurrence, and are more frequently observed in cases of longer duration. Ulcerations and erosions have, however, been observed in cases in which death followed in thirteen,[6] twelve,[7] ten,[8] and even six and one-sixth[9] hours after the poison was taken. Perforation has not, so far as we are aware, been observed in any rapidly fatal case.

It requires but a short time to produce these inflammatory changes in the gastric mucous membrane. Indeed, as the gastric symptoms are the result of the gastric inflammation, the

[1] See p. 423.

[2] See Woodman and Tidy: "Forensic Med.," Pl. i.

[3] Woodman and Tidy: "Forensic Med." (1877), 142. See also Prof. Tidy's testimony in Liverp. Post Rep., and Carter: *Loc. cit.*

[4] Scheulen: Wchnschr. f. d. ges. Hlk., 1844, xiii., 372.

[5] Letheby: Lancet, 1847, i., 44. Taylor: Guy's Hosp. Rep., 1851-52, 2 s., vii., 187. Greenow: Med. Times and Gaz., 1878, ii., 626.

[6] Wyss: Arch. d. Heilk., 1870, xi., 15.

[7] Cotting: Boston M. and S. Jour., 1838, xviii., 78.

[8] Taylor: Guy's Hosp. Rep., 1841, vi., 265. "Poisons," 3d Am. ed., 297.

[9] Greenhow: Med. Times and Gaz., 1878, ii., 626.

supposition that the latter precedes the former in all cases is not unwarrantable. Observation has also shown that in those cases in which death has occurred with the greatest rapidity the evidences of gastric inflammation have been present.

In Finlay's case[1] of death in one hour the mucous membrane of the stomach was intensely congested, and was the seat of a few ecchymoses, but there were no erosions.

In Foster's case[2] of death in two hours the villous coat of the stomach was of a vermilion hue. In Dieberg's case[3] of death in the same time the mucous membrane of the stomach was marbled red, with black spots the size of peas here and there. In Clegg's case,[4] which also proved fatal in two hours, the stomach was found intensely inflamed. In a case reported by Taylor,[5] in which death occurred in two and a half hours, the stomach presented internally several red patches, especially toward the pylorus. In another case, fatal in the same time, reported by Dymock[6] the rugæ were numerous, prominent, and firm; in the fundus and at the pylorus were bright scarlet patches which disappeared near the centre of the stomach, except a few isolated ones. Some patches had a bruised and purplish appearance, but there were no extravasations, and on microscopic examination they showed the color to be due to enlarged veins. Taylor[7] mentions another case, fatal in barely three hours, in which there was intense inflammation, especially at the pyloric end. Jeffreys[8] also reports a case in which death occurred in less than three hours, in which the mucous coat of the stomach was covered with a tenacious mucus, red, highly inflamed, and ecchymosed. A dark, tenacious, bloody patch three inches in circumference existed near the cardiac end. The rugæ were corrugated, and there was an appearance of disorganization. The pyloric end was highly inflamed.

It cannot be denied, on the other hand, that death may be due to arsenic and still no signs of inflammatory action remain detectable in the mucous membrane of the stomach or intestines. Several such cases are upon record, in none of which, unfortunately, is the dose taken stated, and in only three the duration.

[1] Lancet, 1883, ii., 943.
[2] Lancet, 1840–41, ii., 305.
[3] Friedreich's Bl. f. ger. Med., 1866, xvii., 53.
[4] Taylor: "Poisons," 3d Am. ed., 297.
[5] Guy's Hosp. Rep., 1850–51, 2 s., vii., 183.
[6] Edinb. M. and S. Jour., 1843, lix., 350.
[7] "Poisons," 3d Am. ed., 327.
[8] London M. Times, 1851, n. s., iii., 229.

O'Reilly [1] reports the case of an elderly lady who died in six hours, with little pain or suffering. There was scarcely a trace of change of structure in the whole alimentary canal. The stomach exhibited vascularity in two or three points only. A boy of twenty months [2] died in eight hours after having taken an arsenical mouse poison. He vomited, but had no pain. In the intestine and stomach there was little deviation from the normal appearance. Taylor refers to two homicidal cases.[3] In one (Reg. *v.* Newton) the coats of the stomach were thickened and pulpy, but entirely free from inflammation, although the "death was clearly due to arsenic." In the other (Reg. *v.* McCracken) a large quantity of arsenic was found in the stomach of the deceased, but there was no appearance of inflammation, either in that organ or in the intestines. In the Kloss case [4] the mucous membrane of the stomach of one of the victims was free from any semblance of inflammation. Arsenic was found in the stomach in small quantity, and a little more than half a grain (0.032 gm.) of arsenic trioxid was separated from the liver and kidneys.

In a clear case of suicide by Paris green, in which death occurred in less than sixty hours, there was entire absence of inflammation of the alimentary tract, although vomiting and purging had been excessive.[5]

Virchow has described [6] a modification of the minute structure of the gastric mucous membrane, under the name of *gastro-adenitis arsenicalis parenchymatosa*, which is said to occur in arsenical poisoning even when the coarser gross appearances are wanting. The surface of the mucous membrane is covered with a thick, granular mass, which penetrates its whole substance, dipping into the peptic glands, the epithelium of which is cloudy and partly fatty. The interstitial tissue is infiltrated with round cells. Kobert [7] considers this appearance as typical of arsenical poisoning, and states that it may be produced in animals by hypodermic injection of Fowler's solution, and that it has been observed in the human subject after external application of arsenic.

An examination of the surface of the mucous membrane with the aid of a magnifying glass will, if the poison have been taken in the shape of a crystalline powder, usually reveal the presence of minute particles firmly adherent to, and embedded

[1] Dublin J. M. Sc., 1841, xx., 429.
[2] May: Prov. M. and S. Jour., 1845, 453.
[3] "Poisons," 3d Am. ed., 298.
[4] Davidson: Buff. M. and S. Jour., 1882, xxii., 117.
[5] Chittenden and Smith: Med.-leg. J., N. Y., 1885, iii., 148.
[6] Arch. f. path. Anat., etc., 1869, xlvii., 524.
[7] "Intoxicationen," 256.

in, the layer of mucus or even in the substance of the membrane. These particles are in the form of octahedral crystals, which sometimes glisten as little diamond-like specks when the surface is exposed to the sunlight, and which communicate a "sandy" sensation when the finger is drawn over the surface. They are usually white, but sometimes assume a yellowish or yellow color from conversion into the yellow sulfid (see below). They are surrounded by a reddened area, due to injection of the vessels, and beneath them the mucous membrane is frequently distinctly eroded. Sometimes the particles are of nearly uniform size, or they may vary from coarsely granular fragments to the finest powder,[1] according to the method of manufacture of the arsenic used. From these and other characters it is sometimes possible to show that arsenic found in two cadavers was not of the same origin, or not of a certain given origin.[2] The quantity of arsenic found adherent to the stomach is usually not great, although it may be. Thus Taylor[3] refers to a case in which upward of an ounce was found spread over the mucous membrane in a pasty state. Bartley separated three hundred grains (19.5 gm.) of arsenic from the stomach of a girl who had died of arsenical poisoning. In the case of Reg. *v.* Madeleine Smith, Professor Penny separated 5.2 grains (0.337 gm.) of crystallized arsenic from the stomach.[4] These particles are most frequently found in the stomach, but they have also been found elsewhere: in the duodenum,[5] in the ileum and jejunum,[6] in the large intestine,[7] and in the mouth and between the teeth.[8] In an early case[9] a cyst containing crystallized white arsenic was found in the stomach. These particles must be shown to be arsenical, which is easiest accomplished by heating a small portion in a subliming cell and collecting the sublimed product partly on a dry glass cover and partly on one upon which

[1] Med. Times and Gaz., 1872, ii., 490.

[2] See p. 342.

[3] "Poisons," 3d Am. ed., 297.

[4] Morison's Report, p. 34.

[5] James: Bull. Ac. méd., Paris, 1838–39, iii., 664 (case of Soufflard). Ewen: Prov. M. and S. Jour., 1842, iii., 505. Wimmer: Mag. f. d. Staatsarznk., 1846, v., 227. Case of Mad. Smith, *loc. cit.*, p. 32. Greaves: London M. Gaz., 150, n. s., x., 677. Wyss: Arch. d. Heilk., 1870, xi., 15.

[6] Wyss: *Loc. cit.* Hodgson: Prov. M. and S. Jour., 1851, 490.

[7] Hodgson: *Loc. cit.* Greaves: *Loc. cit.*

[8] Kissel: Deut. Ztschr. f. Staatsarznk., 1852, xi., 137. Reuter: Med. Jahrb. f. d. Herz. Nassau, 1846, 97, 236. Grabacher: Oest. Ztschr. f. prk. Hlk., 1866, xii., 172.

[9] Tonnelier: J. d. méd., chir., pharm., etc., 1802, iv., 15.

is a drop of water. The octahedral crystals upon the first, and the formation of a yellow color or precipitate in the second upon the addition of ammonio-silver nitrate solution are sufficient evidence that the material in question was arsenic, when taken in conjunction with its original crystalline form.

One case is reported[1] in which the powdered porcelainous form had been taken, and in which more than 0.36 gm. (five and a half grains) of the amorphous white powder was scraped from the mucous membrane of the stomach.

If the body has been long buried non-arsenical crystalline deposits may exist. Maschka[2] found the walls of the alimentary canal (which were well preserved) of a cadaver exhumed eighteen months after death to contain numerous, hard, firmly adherent incrustations, varying in size from that of a hempseed to that of a pea, which consisted of ammonio-magnesian phosphate. Three-quarters of a grain (0.048 gm.) of arsenic trioxid was also obtained from a portion of the viscera.

The contents of the stomach are usually fluid, sometimes quite thin, generally viscid, red brown in color from the presence of blood, or yellowish from the presence of bile, varying in quantity from one or two ounces to two or three pints, and containing, if vomiting have not been abundant, particles of food substances. These, however, by reason of the copious vomiting, are usually absent. In some cases the stomach, and indeed the entire alimentary canal, is found empty or nearly so, and in some cases the stomach is distended with gas—usually a product of putrefaction.

The appearances of inflammation observed in the stomach are frequently extended into the **intestine**, usually in patches; the **duodenum** and **rectum** being the portions of the gut most frequently affected. It is possible, however, that these portions may not be inflamed, while patches of inflammation are found in other portions of the intestine. Thus Taylor[3] describes a case in which the duodenum and rectum were not inflamed, but red patches existed in the jejunum and ileum. The intestinal hyperæmia varies greatly in degree, and does not

[1] Casper-Liman: "Handb. d. ger. Med.," 8te Aufl., 400.

[2] "Samml. Gutacht. Prag. med. Fak.," 1867, iii., 253.

[3] Guy's Hosp. Rep., 1851–52, vii., 194.

necessarily increase with the duration of the poisoning. Thus Lesser[1] gives examples of portions of the small intestine from two fatal cases: one that of a woman of twenty-two years who died in eighteen and a half hours after taking white arsenic, in whom the intestinal mucous membrane was anæmic, the injection being limited to the larger vessels; the other that of a man of twenty-two years who died in seven hours after having taken Schweinfurth green, in whom the intestinal mucous membrane was reddened by capillary and venous congestion, and in places corroded and marked with minute hemorrhages. The solitary follicles and Peyer's patches are swollen, prominent, and yellowish-white from extensive cell infiltration. The mucous membrane is extensively denuded of epithelium, shreds of which float in the intestinal contents, communicating to it, in the lower part of the small intestine, a "rice-water" or "thin gruel-like" appearance.[2] The contents of the duodenum usually resemble those of the stomach. Multiple ecchymoses throughout the intestinal tract are not infrequently met with. In the **large intestine** the mucous membrane is also denuded of epithelium in great part, and abundantly infiltrated with white corpuscles. The **colon** is frequently found contracted. Wilks[3] reports a case, fatal in twelve hours, in which the contraction was such as barely to admit the point of the enterotome. The end of the ileum was also contracted, as well as other parts of the small intestine to a less degree. The **omentum** and **mesentery** have been found congested and the **mesenteric glands** enlarged and reddened in some cases, and Wyss[4] reports a case of death in thirteen hours in which there were evidences of peritonitis of recent origin, without perforation or extensive ulceration of the stomach or intestine. The viscid exudation on the peritoneum, observed in Asiatic cholera, was absent. The **gall bladder** is rarely empty, more usually distended with bile. The **urinary bladder** is almost always empty, or nearly so.

The **heart** usually contains loosely coagulated blood in moderate quantity, and ecchymoses have frequently been observed beneath the endocardium and in the muscle of the left ventricle in cases which have been rapidly fatal,[5] as well as in others in

[1] "Atlas d. ger. Med.," Pl. xiv., Figs. 2, 5, pp. 90, 93.
[2] See pp. 417, 423.
[3] Lancet, 1862, i., 325.
[4] Arch. d. Hlk., 1870, xi., 15.
[5] Finlay: Lancet, 1883, ii., 943; death in one hour.

which life has been prolonged for several days.[1] The **blood** is dark, and, if the autopsy be made soon after death, becomes brighter on exposure to air, and is thicker than normal, owing to the loss of water by purgation during life. The coagula are not firm, and the corpuscles are normal. The **lungs** are usually congested, with subpleural ecchymoses, and occasionally œdema.

The appearances in the **brain** and its membranes are not constant.

The condition of the **liver** and **kidneys** may be dealt with in passing from the consideration of cases with rapidly fatal termination to that of those of longer duration. Although in many early accounts of autopsies in cases of arsenical poisoning the liver has been described as "greatly enlarged," "mottled," "pale," or even "yellow-fatty,"[2] the occurrence of fatty infiltration and degeneration of that and other organs as a consequence of poisoning by arsenic (and antimony) was not recognized until the experiments of Saikowsky[3] upon animals demonstrated its occurrence. Nor was an observation of the same changes in the liver, kidneys, heart, and diaphragm in the human subject long wanting, for in the same volume[4] may be found the account of the autopsy, by Grohe and Mosler, of a child of two years which died in thirteen hours after an accidental taking of an arsenical green, in which these lesions were found to exist. This infiltration and degeneration proceeds progressively, and is consequently best marked in cases in which death is postponed for some days. When the patient succumbs during the first twenty-four hours, the gross appearances of the liver and kidneys usually present nothing abnormal; yet even in rapidly fatal cases microscopic examination will show the existence of fatty infiltration and beginning degeneration in the liver, kidneys, and later in the heart, diaphragm, and central organs generally. The most rapid development of this lesion was in the case reported by Lesser,[5] in which a man of twenty-two years died in seven hours after taking Schweinfurth green.

[1] Roth: Arch. f. path. Anat., etc., 1868, xliv., 131; death on the fourth day. See also Wilks: Tr. Path. Soc., London, 1862, xiii., 54.

[2] Williams: Lancet, 1840, i., 706.

[3] Arch. f. path. Anat., etc., 1865, xxxiv., 73–80. See also Munk and Leyden: Berl. klin. Wchnschr., 1865, ii., 503.

[4] *Ibid.*, pp. 208–225.

[5] "Atlas f. ger. Med.," Pl. xvi., Fig. 9, p. 133.

The liver was enlarged, presented the gross appearances of fatty degeneration, and on microscopic examination showed the liver cells to be the seat of acute parenchymatous degeneration. In other instances the same changes have been observed in death after eight and a half, nine, thirteen, and eighteen hours.[1] Pointer[2] in 1856 reported a death from arsenic in twelve hours, in which some fatty degeneration was observed in the liver. When death is postponed for some days these changes extend in this and other parenchymatous organs, and in the heart and muscular system, attended by extensive derangements of nutrition and by extravasations beneath the serous membranes.

In fatal cases of long duration other lesions are sometimes observed, some of which have been already noted as occurring in a less marked form and exceptionally in rapidly fatal cases. Among these are inflammation and ulceration of the mouth, tongue, pharynx, and œsophagus. Glossitis, with swelling of the salivary glands, has been observed even after external application.[3] Cystitis has been noted in some cases.[4] Gangrenous discolorations have occurred in the scrotum, glans penis, and beneath the ears, when the poison has been taken by the mouth,[5] and similar changes are said to have been observed in the female genitals and at the anus under like conditions.[6]

When the application has been to the skin, the cuticle is raised in blisters, or the points of application present excoriations, ulcerations, or eschars. When the poison has been introduced into the vagina, the vulva, vagina, and uterus rapidly become intensely inflamed, and soon become gangrenous.

Post-Mortem Changes—Mummification.—During the eighteenth century the general opinion among writers on forensic medicine was that the cadavers of those who have died from the effects of poisons, including arsenic, putrefy more rapidly than usual. At the present time the opposite view, so far as arsenic is concerned, is very generally entertained by those who have not made a special study of the subject.

[1] Lesser: *Loc. cit.*, and Wyss Arch. d. Hlk., 1870, xi., 15.

[2] Am. med. Monthly, 1856, v., 310.

[3] Fristo: Ann. d'hyg., 1830, iv., 437. Ingerslev: Hosp.-Tid., Kjobenh., 1867, x., 161.

[4] Taylor: Guy's Hosp. Rep., 1851–52, 2 s., vii., 187. Hemann: St. Petersb. med. Ztschr., 1869, xvi., 104.

[5] Ward: Edinb. M. and S. Jour., 1830, xxxiii., 61.

[6] Seidel-Maschka: "Handb. d. ger. Med.," ii., 246.

That arsenic, when present in sufficient quantity, acts as an antiseptic cannot be doubted. The embalming liquids now so generally used are almost invariably arsenical solutions, and when used *secundem artem* and in sufficient quantity, they certainly perform their intended purpose of preventing decay. We have seen a cadaver embalmed with arsenic exhumed after fifty-four days' burial, whose external appearance, save for patches of mould on the eyes, lips, and forearm, did not differ from that of a living person, while the internal organs, even to the brain, were as firm in texture and, so far as gross appearances went, as well preserved as are those of a body within a day after death in winter. But to produce this result a notable quantity of arsenic is required, and if the quantity be insufficient, or if the injection be limited to the abdominal cavity, putrefaction proceeds as usual, notwithstanding the rapid diffusion of arsenic throughout the cadaver.[1]

As arsenic does not act as a dehydrating or coagulating agent, and as, of course, it cannot prevent putrefaction by removal of air or restraint of temperature, its antiseptic power must depend entirely upon its germicidal action. That its toxic action toward the lowest forms of life is very greatly inferior to that which it exerts upon the higher forms is shown by many observations. Miquel[2] found that to prevent putrefaction of beef tea by arsenic trioxid a solution of the strength of 1:166 is required, and with sodium arsenite the strength must be increased to 1:111, while the same end is attained by a solution of mercuric chlorid of 1:14,300, and by mercuric iodid in the dilution of 1:40,000. The experiments of Johannsohn[3] have shown that although arsenic trioxid by long contact with yeast gradually destroys its power of causing alcoholic fermentation, it accelerates putrefaction of the ferment, if present in small amount, by favoring the development of *bacterium termo*. The growth of moulds is not interfered with by arsenic, and we have repeatedly seen solutions of arsenious acid become mouldy on exposure to air.

That a cadaver may putrefy rapidly after having been more than usually impregnated with arsenic during life is evidenced

[1] Witthaus: "Researches Loomis Lab.," 1890, i., 48.

[2] "Les organismes vivants de l'atmosphère," Paris, 1883, c. ix.

[3] Arch. f. exp. Path. u. Pharm., 1874, ii., 99–122.

by the case of an arsenic eater who died from an overdose, which is reported by Parker.[1] He had taken arsenic daily for four years, the dose per diem during the last few months having been between two and three grains (0.13–0.2 gm.). In twenty-four hours after death the whole body was swollen and disfigured, the abdomen was distended to its utmost limits, all voluntary muscles were very rigid, the countenance was livid, the skin of the face glistened with emphysematous distention and venous congestion; there were general emphysema and capillary congestion of the surface of the body; and the penis and scrotum were black and swollen from decomposition and gaseous distention. This occurred in November.

On the other hand numerous observations show that the cadavers of those who die from arsenical poisoning generally putrefy less rapidly than other bodies under like conditions and are frequently exempted from the usual odorous putrefactive changes. This we believe is in many instances due to extensive loss of water by purgation during life, rather than to any truly antiseptic influence of the arsenic. Exceptional instances have, however, been observed in which this cause could not have been operative. Hedley[2] reports the case of a man of twenty-four years who died in twenty hours without having purged, and whose body one hundred and forty-one days after death was sufficiently preserved to admit of identification. Hitchcock[3] describes the case of a woman who died in eighteen hours from the effects of arsenic (more than four grains of which were separated from the cadaver) whose body was identified ten years later by the features alone, although other bodies buried later in the same cemetery had wholly decayed.

When suspicion of homicidal poisoning has been awakened months or years after the alleged commission of the crime, and the body of the deceased has been exhumed, its degree of preservation has frequently been an important subject of consideration. The preservation of the bodies exhumed and examined by Welper in the case of the widow Ursinus in 1803, and by Bachman in that of the widow Zwanziger in 1812, led to the impression that mummification[4] is not only a result of arsenical

[1] Edinb. M. Jour., 1864. x., 123.
[2] Lancet, 1842-43, ii., 801.
[3] Boston M. and S. Jour., 1849. xxxix., 489.

[4] In this connection natural mummification is understood; a drying of the body, in contradistinction to the ordinary soft putrefaction, re-

poisoning, but is an indication that it has occurred. So firmly was this view entertained by Burdach and Butzke[1] that in a case of suspected homicide in 1837, they, having made an autopsy, buried the body, exhumed it after five months, and declared in favor of arsenical poisoning from the symptoms, post-mortem appearances, and mummification, although arsenic was not detected.

In a more recent case, that of Speichert[2] in 1876, undue weight was given to the fact that the body of the deceased was found mummified after eleven months' burial, and the conviction seems to have depended largely upon the statements of Koch, Zagrodski, and Wolff that they could not explain the condition of the cadaver on any other theory than arsenical poisoning. A small quantity of arsenic had been found in the cadaver by Sonnenschein, the presence of which was, however, subsequently accounted for by the fact that the deceased had taken tartar emetic which was shown to have contained arsenic.

Bischoff,[3] in a report in May, 1885, says that in twenty cases of arsenical poisoning investigated by him he has but seldom met with mummification, and then only in the abdomen, without noticeable preservation of the remainder of the body, and that he has frequently seen total mummification under the most varied conditions, without the presence of a trace of arsenic.

In the Buffenbarger case in Ohio in 1871, the body of the victim was found in a remarkable condition of preservation after four years' burial, the tissues being quite firm and the stomach parchment like. In their report of the case Bartholow and Whittaker[4] discuss the subject of the preservative action of arsenic, and do not consider it to have been the cause of the condition of the organs in this instance, particularly as the stomach was not so well preserved as other parts.

In connection with the investigation of the crimes of that most industrious of poisoners, Frau van der Linden (Swanen-

sulting in its conversion into a brown, dry, almost odorless parchment-like or powdery material.

[1] Burdach: "Gerichtsärztl. Arbeiten," 1839, pp. 35-65.

[2] Löwig: "Arsenikvergift. u. Mummifikation," Breslau, 1887. Kobert "Intoxikationen," 257.

[3] Vierteljschr. f. ger. Med., 1893, 3 F., vi., 85.

[4] Cincinnati Clinic, 1871, i., 9, 25. See also Wormley: "Micro-Chem. of Poisons," 2d ed., 252.

burg) at Leyden in 1883–85, Zaaijer[1] examined the bodies of sixteen supposed victims at periods varying from fourteen and a half hours to two years and eleven months after death, in all but two of which arsenic was found; and made an extended study of the occurrence and conditions of mummification. We give his conclusions as expressing the most enlightened view upon the subject:

1. Mummification is of very frequent occurrence.

2. Bodies containing no arsenic under the same conditions as arsenical cadavers are equally well preserved and also become mummified.

3. The relative frequency of mummification of the abdominal and thoracic walls, of the skin upon the wrists, knees, ankles, hands, fingers, feet, and toes are easily explainable irrespective of the influence of arsenic.

4. There is no so-called arsenic-mummification (that is for toxic doses).

5. Cadaveric mummification is without significance in forensic toxicology.

Partial conversion of the tissues into adipocere has also been observed in arsenical cadavers,[2] under circumstances similar to those existing when the same change is observed in the bodies of those who have died from other causes.[3]

During the progress of putrefaction the sulfur existing in the albuminoid constituents of the tissues is converted into hydrogen sulfid, which acting upon any arsenical compound present converts it to a greater or less extent into arsenic trisulfid, which, being insoluble in acid liquids, is deposited and communicates its bright-yellow color to the tissues which it impregnates, or tinges the crystals of white arsenic which remain adherent to the stomach and intestine more or less deeply yellow. It may also be dissolved out when the reaction becomes alkaline and may thus migrate so as to communicate its yellow color to the skin and even to the grave clothes and linings of the casket. Deposits of arsenic trisulfid have also been found on the endocardium.[4]

[1] Viertljschr. f. ger. Med., 1886, n. F., xliv., 249–277.

[2] London M. and S. Jour., 1834–35, xi., 760; Ohio M. and S. Jour., 1863, xv., 467; Wien. med. Wchnschr., 1867, xvii., 533; Ann. d'hyg., 1886, 3 s., xv., 24.

[3] See vol. i., p. 451.

[4] Bose: Indian Med. Jour., Calcutta, 1892, xxvii., 142.

It is probable, however, that the yellow spots, streaks, and stains observed in the stomach, intestine, and elsewhere in earlier cases were not always due to arsenic trisulfid. As early as 1840 Widemann[1] found that a yellow material from the stomach of a man who had died in six hours from the action of arsenic did not consist of arsenic trisulfid, while white particles from the same were identified as arsenic trioxid. In two of the four bodies examined in the case of Reg. *v.* Flannagen and Higgins (Liverpool Winter Assizes, 1884), Brown and Davies found the bright-yellow stains which existed in the large and small intestines—in one case after over thirteen months' burial and in the other after over three years' burial—were not due to arsenic trisulfid but to bile pigment more or less modified.[2] A chemical examination of these yellow stains should therefore never be neglected when they are found. Arsenic trisulfid may be dissolved out by ammonium hydroxid solution, which, after filtration, deposits it unchanged on evaporation. The biliary stains, on the other hand, may be dissolved out, after drying, by chloroform, which on evaporation leaves a residue which responds to the Gmelin reaction and other tests for bile pigments. The residue of their aqueous or ammonical extract also responds to the Pettenkofer reaction.

CHRONIC ARSENICAL POISONING.

The chronic form of arsenical poisoning may be produced by a single large dose, the acute symptoms gradually diminishing in intensity and merging into those characteristic of the chronic form. Most cases of acute poisoning in which death is delayed manifest this mixed type.[3] Usually the duration, whether the case terminate in death or in recovery, is less than a fortnight, but in non-fatal cases the paralyses may continue for a much longer period. Thus Morton[4] relates the case of a woman of thirty-five years who required three months, after having taken a drachm (31 gm.) of white arsenic and a phial of laudanum, to

[1] Ztschr. f. d. Staatsarznk., 1840, xxxix., 182.

[2] Whitford: Lancet, 1884, i., 419–42. Brown and Davies: *Ibid.*, 506; and Med. Times and Gaz., 1884, i., 319.

[3] See p. 393.

[4] Med. Reptr. West Chester, Pa., 1855, iii., 55.

regain the use of her hands and feet. Barnett[1] and Stanley[2] report cases of attempted suicide in each of which a year elapsed before recovery was complete.

It is possible also that a patient may apparently recover entirely from an acute poisoning, and be taken after an interval with chronic symptoms. Thus Kovács[3] reports the case of a man who was discharged after a week's illness, caused by his having taken 0.6 gm. (nine and a quarter grains) of white arsenic, and who was readmitted one month later, suffering from pronounced arsenical paralysis, etc., for which he remained under treatment for two months.

In most forms of industrial arsenical poisoning, as well as in those due to contact with arsenical compounds or to inhabiting rooms papered with arsenical papers, or containing specimens of the taxidermist's art in too great abundance, the symptoms follow the chronic type. This is also the case in many medicinal poisonings when the drug is exhibited for too long a period or in too large quantity. (See pp. 365–372, and Arsenical Greens.)

To this class also belong those homicidal cases in which the poison is administered in repeated small doses, with or without a larger dose at the beginning or end of the series of crimes. Frequently the clinical history of these cases differs from that of those which may be classed as accidental in that there occur during the course of the chronic poisoning a series of more or less pronounced acute attacks corresponding to each administration, the dose given by the poisoner being small, yet larger than is usually accidentally absorbed, and sufficient to produce acute symptoms. Excellent illustrations of such cases is found in the several poisonings at Havre in 1886–88 (Affaire Pastré Beaussier) which formed the subject of the thorough investigation of Brouardel and Pouchet.[4]

Symptoms.—From a study of the Havre poisonings and of the accounts of Vidal and others of the phenomena observed in the poisonings caused by the wine of Hyères,[5] Brouardel and Pouchet[6] have traced the clinical picture of chronic arseni-

[1] Tr. Med. Soc. N. Y., 1858, 225.
[2] Amer. Lancet, 1890, n. s., xiv., 446.
[3] Wien. klin. Wchnschr., 1889, ii., 629.
[4] Ann. d'hyg., 1889, 3 s., xxii., 137, 356, 460.
[5] See p. 365.
[6] *Loc. cit.*, p. 479.

cal poisoning. They divide its course into four distinct and well-defined phases or periods:

I. *The Period of Digestive Disturbances.*—The case usually begins with gastric disorders, which are marked by mild forms of the symptoms already described as those of acute poisoning with certain differences: the vomitings are not generally accompanied or followed by painful sensations in the stomach (as in acute poisoning), they occur suddenly, seven or eight times a day, are abundant, and consist of a glairy liquid tinged with bile. Constipation is more usual than diarrhœa, but sometimes there are a few sanguinolent stools. The intestinal disturbance lasts but a short time. Febrile symptoms, resembling those of typhoid, with a pulse of 110–120 also occur.

II. *Period of Eruption—Laryngo-Bronchial Catarrh.*—The laryngeal and bronchial symptoms are those of spasmodic grippe. The laryngoscope reveals the existence of laryngeal catarrh. In some instances there is aphonia, almost without cough, which may persist for a fortnight. There are sibilant or snoring bronchial râles, and mucous, sometimes bloody expectoration. At the same time there is intense coryza, accompanied by suffusion of the eyes and injection of the conjunctiva.

During this period, before the catarrh sometimes, and occasionally at a later period, cutaneous eruptions appear, and a redness and puffiness of the eyelids and scrotum: various erythemata upon different parts of the body, furfuraceous or squamous exfoliations of the epidermis, loss of hair and even loss of one or more nails, vesicles, vesico-pustules, urticaria, rubeola, and pigmented patches. The epidermic and laryngeal symptoms are attributed to the elimination of arsenic by the skin and laryngo-bronchial epithelium. Arsenic was found in the Havre cases in the epidermis, nails, and hair.

III. *Period of Disorders of Sensibility.*—A very frequent and early symptom is severe general headache, which persists for quite a time. Then there is a feeling of numbness in the legs and feet, sometimes accompanied by rather severe cramps. Later there are severe pains, sometimes shooting, but more frequently crushing in character, principally in the tibio-tarsal and tarso-metatarsal articulations. The sensations are sometimes peculiar; one described them as if "he had animals, dogs, in his ankles and soles which were gnawing the skin," another as

if "he had butterflies crawling in his back." In almost all the pressure of the bed coverings on the feet and legs becomes insupportable.

The general sensibility is not notably affected. There is, however, diminution of sensation, frequently quite marked. The patients do not have a clear impression of the resistance of the ground upon which they walk; touch or slight pressure may not be perceived, and pricks are less distinctly felt. In some cases the loss of sensation in the upper extremity has been such that the patients do not hold objects in the hand when they look away. There is no interference with the special senses. The secretions, notably the perspiration, are increased, particularly in young patients. Loss of sexual desire is almost constantly observed.

IV. *Period of Paralysis.*—The motor disturbances follow, but if the intoxication be light they are absent or only slightly marked. They begin with a certain degree of muscular feebleness, afterwards the patient becomes fatigued much more quickly, he has difficulty in going up-stairs, and throws his legs directly forward in walking. The paralysis increases, the patient can neither walk nor stand upright without support. When seated the foot hangs limp. The muscular paralysis begins with the extensor communis digitorum and attacks in preference the extensors and supinators, which become atrophied. Faradic excitability is absent in the muscles most affected, and diminished in others. Galvanic excitability is at least partially retained even in the severest cases. The tendinous reflexes of the lower extremity are absent. The cutaneous reflexes are less affected, the plantar reflex only being much weakened. The cremasteric and abdominal reflexes are normal in intensity.

Termination.—Recovery is frequent in cases in which the phases are distinct. It is slow when paralysis has been clearly defined, and may require over a year.

Death is most frequently by the heart. In two cases, after a period of gastro-intestinal symptoms, there was an attack of dyspnœa and then fatal syncope; in another death occurred suddenly during paralysis. Or life may be extinguished in another way. The quantity of poison taken may not be sufficient to cause death in the few days following its absorption. It may

have been entirely eliminated, but the anatomical changes which it has caused in the liver, kidneys, and muscular tissue remain and lead to a fatal termination.

The above is a statement of the phenomena observed in two series of cases. But chronic arsenical poisoning assumes the most protean forms, and in exceptional instances has manifested this or that symptom not observed in these cases: arsenical melanosis, anæsthesia dolorosa arsenicalis, anaphrodisia arsenicalis, arsenical amaurosis, multiple neuroses, etc., for descriptions of which we can only refer to special treatises.[1]

ARSENOPHAGIA—ARSENIC EATERS—TOLERANCE OF ARSENIC.

That the habit of taking arsenic exists in certain localities and among certain classes of the inhabitants of many large cities does not admit of question.

Attention was first drawn to this habit, which was then probably limited within very narrow boundaries, by von Tschudi, who published an account of his observations in the mountainous districts of Styria and Hungary in 1851,[2] the publication being prompted by the outcome of a trial for murder by arsenic in which the defendant was acquitted, the defence having set up the theory that the deceased was an arsenic eater.

In his two papers von Tschudi refers to three cases of arsenophagia: 1. That of a thin and pale girl, who after four months' use of arsenic became plump and red-cheeked, but who died of poisoning in consequence of having incautiously raised the dose. 2. That of a man of

[1] Lewin: "Nebenwirkungen der Arzneimittel," 1893, 419–30. Imbert-Gombeyre: "Suites de l'empoisonnement arsenical," Paris, 1881. Müller: "Arsenmelanose," Diss., Berlin, 1892. Da Costa: Phila. Med. Times, 1880–81, xi., 385–90, 614–616. Erlicki u. Rybalkin: Arch. f. Psychiatr., Berlin, 1891–92, xxiii., 861–895. Mařik: Wien. klin. Wochnschr., 1891, iv., 565, 595, 614, 648, 666, 686, 710, 728, 747. Leszynsky: Med. News, 1889, 251. Seeligmüller: Deut. med. Wchnschr., 1881, vii., 185, 200, 217. Alexander: "Beiträge zur Kenntn. der Lähmungen nach Arsenikvergift.," Breslau, 1889. Wyss: Corrbl. f. schweiz. Aerzte, 1890, 473. Charcot: Bull. gén. de thérap., 1864, lxvi., 529–534. Hutchinson: Arch. Surg., London, 1889, i., 75. Falkenheim: "Mitth. a. d. m. Klin. zu Königsberg," Leipzig, 1888, 114–131. Engel, Reimers: Jahrb. d. Hamb. Staatskrankenanst., 1890, Leipzig, 1892, ii., 428. McPhedran: Canada Pract., 1890, xv., 1. Startin: Lancet, 1879, ii., 940. Putnam: Boston M. and S. Jour., 1889, cxx., 235, 253.

[2] Wien. med. Wchnschr., 1851, i., 454; 1853, iii., 8.

sixty years, in excellent health, who had been an arsenophage for forty years, who had acquired the habit from his father, whose dose at that time was four grains, and in whom there was not the faintest trace of an arsenical cachexia. 3. That of a man who had begun the habit in his twenty-seventh year and continued it until his sixty-third, beginning with a piece the size of a hempseed and increasing the dose until he took between three and four grains for eight to ten days each month. During thirty-five years he had taken twenty to twenty-two ounces, yet there was no marked change in his organism other than a peculiar hoarseness, which is common among arsenic eaters. On one occasion, being intoxicated, he had taken too large a dose and had suffered the usual symptoms of arsenical poisoning. For two years past he had discontinued the habit, for fear of having dropsy, as did one of his acquaintance, and since stopping the arsenic he suffered from gastrodynia. Von Tschudi further stated that the object of these peasants in taking arsenic is twofold: (1st) The improvement of the personal appearance, and (2d) the improvement of the "wind" in ascending mountains and carrying loads, etc. He further stated that the symptoms of chronic arsenical poisoning never appear in those who maintain their doses within the limits to which they have become habituated, but that if they be deprived of arsenic they suffer from symptoms which bear the closest resemblance to those of mild arsenical poisoning, from which they are immediately relieved by a return to arsenic. He also refers to the common practice of giving arsenic to horses in Vienna for the purpose of improving their condition and appearance, but if a horse so treated be sold to an owner who does not give arsenic the animal loses flesh and becomes dull, even with the best of fodder. He also makes mention of the owner of an arsenic works who daily took three and three-quarter grains of arsenic with his breakfast, and instructed his operatives in the same habit for the purpose of counteracting the ill effects of the arsenical fumes.[1]

The statements of Tschudi were discredited in England, and Taylor,[2] Christison,[3] Kesteven,[4] and others united in condemnation of both doctrine and teacher.

[1] Thilenus ("Med.-chir. Bemerk.," 2d ed., Frankf., 1809) refers to the existence of arsenophagia in southern Germany and Bavaria. Schallgruber (Med. Jahrb. d. œsterr. Staates, 1822, n. F., i., 99,) noticed the case of a healthy peasant who daily took two grains of arsenic trioxid. Flechner (Verhandl. d. k. k. Ges. d. Aerzte zu Wien, 1843, ii., 237) refers to the use of arsenic among the mountaineers of Austria and Styria.

[2] "Poisons," 2d ed., 1859, 89–94. In the 3d ed., and in later editions of the "Med. Jur.," this violent polemic is omitted. See "Poisons," 3d Am. ed., 68–69; "Med. Jur.," 11th Am. ed., 75.

[3] Edinb. M. J., 1855–56, ii., 709–710. In connection with the case of Reg. *v.* Wooler, which was the first English case in which arsenophagia was set up as a defence.

[4] Assoc. M. Jour., London, 1856, ii., 721, 757, 808.

Subsequent observations have shown, however, that von Tschudi's accounts were correct upon all material points. In 1860 Professor Roscoe[1] and Heisch[2] each published a paper, consisting of communications upon the subject received by them, the latter from Professor Lorenz, Professor Arbele of Salzburg, and Dr. Kettowitz of Newhaus, the former through Professor Pebal, of Lemberg, in which statements substantiating those of von Tschudi were made, and Professor Roscoe expressed the opinion that decisive evidence had been brought forward that arsenic was regularly eaten in Styria, in quantities usually considered sufficient to produce death rapidly. Heisch also refers to two cases of alleged homicidal poisoning in Austria in which the defendants were acquitted on the ground that the deceased were arsenophagists.[3] Previously Schäfer had detected arsenic in the urine of an arsenic eater,[4] and later reported[5] the case of a man of thirty years who in his presence and that of Dr. Knappe took four and a half grains of white arsenic, and on the succeeding day five and a half grains, whose urine, passed on the two days, contained arsenic, and who left quite well on the succeeding day, without having manifested any arsenical symptom while under observation during the three days. In 1863 Knapp[6] published his observations of two other arsenic eaters: (1) (Mathias Scholer) A man of twenty-six years who had taken orpiment for a year and a half. (2) (Joseph Flecker) A man of forty-six years, who had taken arsenic since 1849, usually in the form of the trisulfid (orpiment). These cases were also investigated by Maclagan and Rutter, in whose presence the former took nearly five grains (0.32 gm.) of arsenic trisulfid, and the latter about six grains (0.39 gm.) of the trioxid, without manifesting any symptoms of poisoning. The urine in both cases was collected and found to contain arsenic.[7]

In 1864 Parker[8] reported the case of an English arsenic

[1] J. Brit. and For. Med.-chir. Rev., 1862, xxix., 142–153. A review of various papers.

[2] Mem. Lit. and Phil. Soc., Manchester, London, 1860, 3 s., i., 208.

[3] Ph. J. and Tr., 1859–60, n. s., i., 556.

[4] *Loc. cit.*, p. 558.

[5] Sitzb. d. k. k. Akad. d. Wissench. zu Wien; Math.-Phys. Cl., 1857, xxv., 489.

[6] *Ibid.*, 1860, xli., 573.

[7] Jahresb. d. Ver. d. Aerzte in Steirmark, Graz, 1863–64, 19–21. The name is sometimes spelled Knappe and sometimes Knapp.

[8] Maclagan: Edinb. M. Jour., 1864, x., 200–207.

eater whose daily dose was between two and three grains (0.13–0.19 gm.) of white arsenic, who had taken it for four years, and finally died of an overdose taken with suicidal intent.

In 1866 Professor La Rue[1] published an observation of an arsenic eater in Quebec, who in his presence took four grains (0.26 gm.) of white arsenic and smoked a further quantity, mixed with tobacco in a pipe, without suffering any inconvenience during two days of observation.

In 1875 Knapp presented the same two arsenic eaters, accompanied by a third, a man of twenty-five years, at a meeting of a scientific society at which one took 0.3 gm. (about five grains) of arsenic trisulfid, and another 0.4 gm. (about six grains) of arsenic trioxid.[2] In 1885 Knapp and Buchner[3] published the results of their observations of the cases of eight arsenic eaters, including Flecker, then sixty-six years of age and still in excellent health. All of these were in good health and manifested none of the symptoms of acute or chronic arsenical poisoning. The amounts of arsenic given to each and the results of examination of their urine were as follows:

Age.	Dose taken.	Quantity of urine.	As_2O_3 in urine.
66	0.14 gm. As_2S_3	1,515 c.c.	0.0326 gm.
43	0.05 " As_2O_3	1,215 c.c.	0.0292 "
54	0.05 " As_2S_3	645 c.c.	0.0185 "
57	0.02 " As_2O_3		
23	0.12 " As_2S_3		
48	0.12 " As_2S_3	1,525 c.c.	0.0271 "
47	0.10 " As_2O_3		
43	0.10 " As_2O_3		

All of the cases of arsenophagia which have been the subject of investigation in Styria, with one exception, have been men, for the most part strong, healthy, and hard workers. The writers upon the subject from Tschudi to Mařik[4] indicate the probability of the existence of the habit among women as well. The tolerance established is limited, and cases have been referred to by Schäfer, Knapp, Schauenstein, and Parker in which

[1] Edinb. M. Jour., 1864, x., 116–123.

[2] Boston M. and S. Jour., 1866, lxxiv., 439.

[3] Allg. Wien. med. Ztg., 1875, xx., 355, 362.

[4] Knapp: Erghft. z. Centbl. f. allg. Gesundhtspfl., Bonn, 1885, ii., 1–15. Buchner: Sitzber. d. Gesellsch. f. Morph. u. Physiol. in München, 1885, i., 109–112.

an overdose has caused death. Nor is the dose necessarily increased as with opium. Sexual power is not diminished, but rather increased, and cases are referred to by Mařik[1] in which the habit has been resorted to for the purpose of sexual stimulation. The arsenophagists, when maintaining their doses within the limits for which tolerance has been established, never suffer the symptoms of chronic arsenical poisoning, but if the use of arsenic be suddenly discontinued, they suffer from discomfort, weakness, and trembling, which disappear with resumption of the habit. By gradual diminution of the dose, however, the use is readily broken off.

Upon trials for murder by arsenic in this and other countries, the use of arsenic internally without medical prescription is frequently invoked either to account for the presence of the poison in the body, when the quantity found is small, or to explain the purchase of arsenic by the accused. Thus in the Maybrick case it was claimed by the defence that the deceased had been in the habit of dosing himself with arsenic for years, and in the case of Peo. *v.* Ford (New York, 1888) the defendant alleged that he purchased pills containing arsenic (which he administered to his wife) for the purpose of improving his complexion. In another trial for murder by arsenic in New York (Peo. *v.* Sarah Harrington, Del. Co., 1861) it was proved that the deceased had for years used arsenic as a medicine for horses and had taken it himself.[2]

There is every reason to believe that popular arsenical medication has of late years become much more general than it formerly was, although Mařik[3] refers to a countess who related that in the thirties arsenophagia was generally disseminated among the younger ladies in the upper circles of society at Florence (Italy), and that they took arsenic to render them more handsome and lively. Buchner[4] states that at a girls' school in Switzerland arsenic is regularly fed to the pupils. Lewin[5] refers to the very general use of arsenic in the form of Fowler's solution or of Roncegno mineral water by women in better circumstances, as well as by maid-servants, actresses, and

[1] Wien. kl. Wochnschr., 1892, v., 145, 157.

[2] Porter: Tr. M. Soc., N. Y., 1862, 155.

[3] *Loc. cit.*

[4] "Die Aetiol., Ther. u. Prophyl. d. Lungentuberk.," München, 1883, p. 120.

[5] "Nebenwirk. d. Arzneimit.," Berlin, 1892, p. 417.

prostitutes in Germany. In the United States the publication of entire columns of advertisements of "arsenical wafers for the complexion" in the most prominent daily papers is at once an indication of the prevalence of arsenophagia and a means for its wider dissemination.[1] (See also Arsenical Mineral Waters, p. 363.)

In several instances it has been claimed that arsenic or arsenical fly papers were purchased by the defendant to prepare a solution for use as an external application, as a cosmetic. Probably the earliest case in which such alleged use was the subject of inquiry was upon the trial of Madeleine Smith in 1857. The defendant had upon three occasions purchased arsenic colored with indigo and soot, a watery extract of which she claimed to have applied to the face, neck, and arms. Upon the trial Dr. Lawrie testified to having washed his face and hands with water in which he had "mixed" half an ounce of arsenic; but it is not stated whether sufficient time was allowed for solution of any portion of the arsenic "mixed" with the water.[2] Two druggists also testified to applications having been made to them by persons desiring to purchase arsenic "for the complexion," and reference was made to popular articles upon the subject in *Blackwood's* and *Chambers'* magazines.[3] In the case of Minnie Walkup (Kansas, 1886), it was shown that the defendant had purchased four and a half ounces of arsenic from two druggists, and had on two occasions attempted to purchase strychnin. The purchases of arsenic occurred in the week preceding her husband's death by arsenical poisoning, and were made upon representation that it was for use as a cosmetic.[4] In the Maybrick case it was shown that in April the defendant had purchased three dozen arsenical fly papers, some of which were found soaking in a basin. To explain these purchases the defendant made the following statement:

"The fly papers were bought with the intention of using as a cosmetic. Before my marriage, and since for many years, I have been in the habit of using a face wash prescribed for me by Dr. Greggs, of Brooklyn. It consisted principally of arsenic, tincture of benzoin,

[1] Campbell's arsenical wafers contain 0.0000126 grm. (.0002 grain) As_2O_3 in each. The directions are to take two a day for twenty days.

[2] Morison's Report, p. 125.

[3] *Ibid.*, pp. 120, 123.

[4] Med.-leg. Jour., N. Y., 1885, iii., 343.

elder-flower water, and some other ingredients. This prescription I lost or mislaid last April, and as at that time I was suffering from slight eruption of the face, I thought I should like to try to make a substitute myself. I was anxious to get rid of this eruption before I went to a ball on the 30th of that month. When I had been in Germany many of my young friends there I had seen use a solution derived from fly papers, elder water, lavender water, and other things mixed, and then applied to the face with a handkerchief well soaked in the solution. I used the fly papers in the same manner. But to avoid the evaporation of the scent it was necessary to exclude the air as much as possible, and for that purpose I put a plate over the fly papers, and put a folded towel over that, and another towel over that. My mother has been aware for a great many years that I have used an arsenical cosmetic in solution."[1]

No evidence was introduced upon the trial in support of this statement, except that of a hairdresser who testified that upon a few occasions he had been asked for arsenic as a cosmetic by ladies. Macdougall[2] alleges that "the prescription has been found" since the trial, and gives a fac-simile reproduction of it. It is, however, not signed by Grace or Greggs, of Brooklyn, but by a New York chiropodist, and is as follows: "Fowler's sol. arsenic, ℥ ss.; chlorate potass., ℥ ij.; aqua rosæ, ℥ vij.; spts. rect., ℥ i. S. Apply with a sponge twice a day," containing two grains of arsenic in eight and a half fluidounces (0.0517 per cent.).

Arsenic is not used in solution as an external application in the treatment of skin diseases by physicians, and when so applied in strong solution has been known to cause serious symptoms and even death.

Wagner[3] reported the case of a man of twenty-five years whose entire body was washed with a strong solution of arsenic trioxid in water, applied with a wash rag. There immediately followed severe pain all over the body, as if it were laid on fire or needles, which constantly increased. The cuticle was raised in blisters over most of the body. Death followed in three days.

It is certain, however, that there exists a popular impression that preparations containing arsenic may be used externally

[1] Liverpool Post Rept., Aug. 6th, 1889. In Macdougall's account (p. 402) the name of the doctor is given as "Dr. Grace of Brooklyn."

[2] "The Maybrick case," London, 1891, p. 413*.

[3] Wchnschr. f. d. ges. Heilk., 1839, viii., 213.

with benefit, and consequently there is no inherent improbability in the claim that a person may have purchased arsenic for such use. Indeed, toilet preparations containing arsenic, exclusive of depilatories, are openly sold.

ABSORPTION—DISTRIBUTION—ELIMINATION.

Absorption.—Although arsenic may be absorbed into the circulation through every possible avenue of entrance (see page 372) it is chiefly with absorption from the alimentary canal that forensic toxicology has to deal.

When dissolved arsenic is taken into the stomach its absorption probably begins immediately. It makes its appearance in the blood in a few minutes, and is soon partially excreted by the kidneys,[1] and the time which usually elapses before the manifestation of severe symptoms is consumed rather in the production of those tissue changes which are the cause of the symptoms than by delayed absorption. Exceptional cases have already (page 384) been referred to in which the action of the poison was manifested within a few moments after its ingestion.

Arsenic was detected in the blood drawn from the veins of the human subject in life as early as 1839.[2] It is said by Seidel[3] to be found in the corpuscles but not in the plasma.

The RAPIDITY with which absorption progresses is the greater the more the conditions under which arsenic is taken favor its solution, and is greater from an otherwise empty stomach than from one containing food. Taylor[4] cites an instance in which a man died in barely three hours after having taken a large quantity of white arsenic in water, in whose liver, spleen, and kidneys the poison was found in abundance. Smith and Peck[5] in the cadaver of a girl who died in six and a half hours after having taken 45 gm. of "rough on rats," found 0.0548 gm. (0.8 grain) in the liver, and smaller quantities in the kidney and brain.

The DURATION of absorption depends, of course, upon its

[1] Dragendorff: "Ermittel. v. Giften," 4te Aufl., 372.

[2] Bull. Ac. de méd., Paris, 1838–39, iii., 676.

[3] Maschka: "Handb. d. ger. Med.," ii., 249.

[4] "Poisons," 3d Am. ed., 327.

[5] Med.-leg. Jour., N. Y., 1891, ix., 129.

rapidity and upon the quantity absorbed. What its minimum and maximum may be is not definitely determined, if it be determinable.

The shortest duration of fatal arsenical poisoning in which none of the poison remained in the contents of the stomach at death of which we can find satisfactory record is in a very exceptional case reported by Smith:[1] A nursing infant died on the second day, poisoned by the milk of its mother, to whom arsenic had been criminally administered. An analysis by Professor Silliman gave decisive evidence of the presence of arsenic in the liver by the Marsh and Reinsch tests, but the same tests gave no evidence of arsenic in the fluid from the stomach. The child had refused to nurse on the first day. In this case the poison was certainly entirely in solution and in a form readily absorbable.

In one of the victims of the Leyden poisonings,[2] who died eleven days after the poisoning, arsenic was found in the liver to the amount of 0.05211 gm. (0.78 grain), in the kidneys, contents of the small and large intestines, and in the urine voided during life, but could not be detected in the contents of the stomach and duodenum, nor in the pericardial fluid.[3]

In neither of these cases does the tissue of the stomach appear to have been analyzed. While it is quite possible that at some stage of arsenical poisoning the processes of absorption and elimination have proceeded in such manner that the poison may still remain in the liver and kidneys, and be absent from the tissue as well as the contents of the alimentary canal, we know of no analysis whose results prove the existence of such a condition; the nearest approach to it being in the case of Paris-green poisoning, referred to below, in which the tissue of the stomach still contained a trace of arsenic, while the contents were entirely free from it. The fact that arsenic, by whatever channel absorbed, causes tissue changes in the gastro-intestinal walls, and the symptoms resulting therefrom, fairly warrants

[1] Buffalo Med. Jour., 1852–53, viii., 730.

[2] Zaaijer: Vierteljsch. f. ger. Med., 1886, n. F., xliv., 257; analysis by Professor van der Burg.

[3] Taylor ("Poisons," 2d ed. [1859], p. 411) refers to a case in which "nearly two ounces of arsenic were swallowed and the person died in eight hours. No arsenic was discovered in the stomach." No particulars are given, and the statement is omitted in the third edition.

the inference that it enters into chemical reaction with the constituents of those tissues, and if it disappear therefrom sooner than it does from the liver and other parenchymatous organs, it is because the form of combination is less stable in the former than in the latter organs, and because elimination of *absorbed* arsenic from the tissues of the alimentary canal is consequently more rapid than from the liver, kidneys, spleen, etc. In five cases of death from arsenic absorbed otherwise than by the alimentary canal, in which analyses were made, the poison was found in the stomach in four, and in two of these it was not detected in the liver.

A girl of nine years died in six days after the application to the scalp of an ointment containing arsenic. Arsenic was found not only in the scalp but also in the liver and in the mucous contents of the stomach and intestines in small quantity and in solution.[1]

A woman of twenty-five years aborted at three and one-half months, and died nineteen days after, after having manifested the symptoms of arsenical poisoning. Arsenic was found in the vaginal discharges and in the vagina itself, and in less quantity in the stomach and intestines,[2] but none in the liver, kidneys, heart, or muscle.

A woman of thirty-nine years died in six days after application of an arsenical ointment to a cancer of the breast. Arsenic was found in the liver, kidneys, and tissue and contents of the small intestine, but not in the stomach, spleen, or blood.[3]

An infant succumbed in twenty-four hours after inhaling the fumes from heated arsenical pyrites. Arsenic was found in the tissue and the contents of the stomach, and the lungs, but none in the liver.[4]

Two infants died from the effects of arsenical violet powder applied to the skin. In one the kidneys, stomach, and intestines contained distinct traces of arsenic, the spleen two grains (0.13 gm.) and the liver about one grain (0.065 gm.). In the other the liver yielded 1.5 grains (0.0975 gm.) of arsenic, the stomach and intestine one grain (0.065 gm.), and the kidneys distinct traces; four grains (0.26 gm.) more was obtained from the rest of the body.[5]

Maschka [6] reports a case of supposed homicidal poisoning by repeated doses in which death occurred after an illness of eight days, and an autopsy was made on the second day thereafter, and in which somewhat larger quantities of arsenic were found

[1] Taylor: Guy's Hosp. Rep., 1864, 3 s., x., 220.

[2] Brisken: Viertljschr. f. ger. Med., 1864, xxv., 110.

[3] Graham: Glasgow M. Jour., 1868–69, n. s., i., 56.

[4] Allan: In Taylor's "Poisons," 3d Am. ed., 300.

[5] Tidy: Lancet, 1878, ii., 250.

[6] Vierteljschr. f. ger. Med., 1880, n. F., xxxiii., 223.

in the liver and spleen than in the alimentary canal. The chemical experts expressed the opinion that "the arsenic found in the stomach and intestines is only a small portion of the poison originally swallowed, as the greater portion must have been removed by elimination and absorption during the long illness." It is also possible that the poison found in the alimentary canal was not the remains of unabsorbed arsenic, but was that which had been absorbed by the tissue of the alimentary canal, and, as the quantity was less than that in the liver and spleen, it is quite possible that had life been further prolonged a time would have been reached when elimination would have been complete as to the alimentary canal, but not as to kidneys and spleen. In two other instances,[1] in each of which the autopsy was made eight days after death, the quantity of arsenic found in the liver was greater than that in the stomach, but in these the distribution existing at death had been notably modified by post-mortem imbibition, as it must also have been in two other instances in which the bodies were exhumed after burial for still longer periods.[2]

With regard to the maximum limit of time during which some portion of a poisonous dose of arsenic may remain *unabsorbed* in the stomach, the data are equally unsatisfactory. In very few fatal accidental or suicidal cases of long duration have analyses been made. In the case of the Duc de Praslin, whose death occurred on the sixth day, arsenic was found in the intestines in small amount, but no mention is made of the result of the examination of the stomach, although that organ was submitted to the experts.[3] In a case of accidental poisoning in which death occurred in three days, Aguilhan and Barse[4] found arsenic in the stomach by Marsh's test. In a suicidal case in which death occurred in two days de Bartholome[5] found crystals of arsenic trioxid in the stomach. And in a suicidal case in which more than sixty hours elapsed before death, Paterson[6]

[1] Lawrence and Wormley: Ohio M. and S. Jour., 1864, xvi., i.; death in about three days. Davidson: Buffalo M. and S. Jour., 1882, xxii., 117; death in twelve hours.

[2] Devergie: "Méd. lég.," 3d ed., iii., 539 (Affaire Lacoste, 1843). Watt (Wormley): Ohio M. and S. Jour., 1860-61, xiii., 181 (Peo. *v.* Priest).

[3] Orfila: "Toxicologie," 5ème ed., i., 582. Tardieu: "Empoisonnement," 2ème ed., 414–430.

[4] Bull. Ac. de méd., 1840, v., 144.

[5] Prov. M. and S. Jour., 1849, 72.

[6] Edinb. M. Jour., 1857, iii., 394 (Case IV.).

found white arsenic in substance, tinged with the trisulfid. These are the only suicidal or accidental cases of which we have knowledge in which life was prolonged for two days or longer in which a satisfactory analysis revealed the presence of arsenic in the contents of the alimentary canal. In several cases of homicidal poisoning arsenic has been found in the stomach at a longer interval after the *supposed* time of administration; in one case[1] the interval was fourteen days: but in this, as in other cases, the symptoms and other circumstances pointed very strongly to the administration of repeated doses, the interval having been calculated from the first manifestation of symptoms. In other cases the date of administration was insufficiently defined. In one homicidal poisoning, however, reported by Niemann,[2] there appears to have been but one administration by which three individuals were fatally poisoned, two of whom died in two days and the third in five days. The analysis showed the presence of three grains (0.2 gm.) of arsenic in stomach and intestines of one of those who died in two days and of traces in the contents of the other two. Tardieu[3] reports a case in which arsenic was found in the stomach, liver, and spleen in a case of arsenical poisoning of eleven days' duration, but the particulars are insufficient.

We have already suggested that arsenic may exist in the *tissues* of the alimentary canal at a later period after death. Chittenden [4] reports the analysis in a case in which the date of administration is clearly fixed, and in which life was prolonged for two weeks. The stomach was entirely empty, but its tissue yielded 0.00013 gm. ($\frac{1}{5\,0\,0}$ grain) of arsenic trioxid, and one-half of the intestine 0.00026 gm. ($\frac{1}{2\,5\,0}$ grain). The author very properly remarks that: "That contained in the stomach as well as that in the intestines represents the mere trace still retained by the muscle and other tissues of those organs." The quantity of arsenic separated from one-third of the liver equalled that obtained from one-half of the intestines. In another case, reported by Chittenden and Smith,[5] of suicide by Paris green

[1] Maschka: "Samml. Gutacht. Prag. med. Fak.," 1867, iii., 265.
[2] Ztschr. f. Staatsarznk., 1862, lxxxiv., 148.
[3] "Empoisonnement," 2ème ed., 433.
[4] Med.-leg Jour., N. Y., 1884, ii., 243.
[5] Med.-leg. Jour., N. Y., 1885, iii., 148.

(in which, unfortunately, the time when the poison was taken was unknown, although it could not have been as early as sixty hours before death), there were evidences of excessive vomiting and purging about the body when it was found, and the contents of the stomach were entirely free from arsenic, while the tissues of the organ still contained it in small amount.

If arsenic be found in the stomach, and if it have been the cause of death, it must also exist elsewhere in the body, and particularly in the liver. Its absence in that organ while it is present in the alimentary canal is only possible if it have been introduced into the stomach after death and shortly before the autopsy (see Post-mortem Imbibition).[1]

While poisons in solution as a rule act more rapidly and intensely when injected into the circulation than when absorbed from the alimentary canal, the experiments of Boehm and Unterberger[2] show that the contrary is true with regard to arsenic administered to dogs. These observers found that the smallest lethal dose given by the mouth was insufficient to cause death when injected into a vein of an animal of equal size, and that with the latter method of administration death always ocurred somewhat later than when the poison was given by the mouth.

Distribution.—It was from the liver, spleen, kidneys, lungs, heart, and muscles of the suicide Soufflard that Orfila first separated absorbed arsenic from the human cadaver in 1839; and very shortly thereafter the fact was generally recognized that the first-named organ contains notable quantities of arsenic after death caused by that poison. Herapath, in 1842, in a suicidal case, found a much larger quantity in the liver than in the heart;[3] and in the Affaire Lacoste in 1844, Pelouze, Devergie and Flandin, having found a larger quantity in the liver than in

[1] Two recorded cases are in apparent conflict with this view. In one of Casper-Liman's cases ("Handb.," 8te Aufl., ii., 412), an alleged poisoning by an arsenical green, Hoppe found 0.0429 gm. As_2O_3 in 122.5 gm. of stomach and contents, but none in the heart, and only a trace in 291 gm. of liver and blood. The other was the case of a child of four years, reported by Pinkham (Tr. Mass. Med.-leg. Soc., 1878, i., 43), who is said to have died in forty-four hours from the effects of arsenic, to whose action the fatty degeneration of the liver and other organs found at the autopsy is (properly) ascribed, and in whose stomach arsenic was found in crystals, yet in whose liver Professor Hills found no arsenic in 50 gm. of tissue.

[2] Arch. f. exp. Path. u. Pharm., 1874, ii., 96.

[3] Lancet. 1842-43, ii., 285.

the intestine or muscular tissue, remark in their report that "this is in accordance with what is known of poisoning by arsenic."[1]

It is quite possible that an accumulation of results obtained from analyses made under varying conditions may show that there exist constant differences in the relative distribution of arsenic in different organs, due to variations in the time of introduction, the form of combination, the number and amount of the doses, or the habituation of the individual, which are sufficiently marked to enable the toxicologist of the future to positively answer questions concerning those points, but the results hitherto obtained are neither sufficiently concordant nor sufficiently numerous to afford reliable standards of comparison. Our present knowledge of the quantitative distribution of arsenic in the different tissues and organs of the body after death caused by that poison rests upon a small number of more or less complete analyses, in cases of poisoning in the human subject, and upon the results of experiments upon animals.

In Table A we have tabulated all of the analyses known to us which are sufficiently complete for this purpose, and which have been made within a sufficiently short time after death to exclude the unknown influences of post-mortem imbibition.

Of these I., II., III., IV., V., and VII., may be considered as fairly comparable in the elements of duration, the form of the poison taken and the age of the individuals, yet if the quantities found in the different organs other than the alimentary canal be compared with each other it will be found that the distribution varies greatly. Thus, if the quantities in brain, muscle, and liver be successively taken as unity we have:

	Brain = 1.				Muscle = 1.		
	Liver.	Kidneys.	Muscle.		Spleen.	Liver.	Kidneys.
I.....	300	253	..	IV.....	....	28.2	38.1
II.....	125	...	..	V.....	10.2	3.4	10.2
IV.....	84	128	3	VII.....	....	3.3	5.1

	Liver = 1.				
	I.	III.	IV.	V.	VII.
Kidneys.....	0.84	0.03	1.52	3.05	1.53
Spleen.....	....	...	....	3.04	....

Under very similar conditions, therefore, in one case (V.) 100 gm. of kidney contained three times as much arsenic as

[1] Devergie: "Méd. lég.," 3d ed., iii., 544–545.

A. Human Bodies—Autopsies Made Soon After Death.

No.	Conditions.	Stomach.		Intestines.		Spleen.		Liver.		Kidneys.		Brain.		Muscle.	
		Found.	In 100 gm.	Found.	In 100 gm.	Found	In 100 gm.	Found.	In 100 gm.	Found	In 100 gm.	Found	In 100 gm.	Found	In 100 gm
I.	F., 16, S., D. 6½ h., "Rough on rats."	342.1	46.1	.....	.. ....	.....	.....	54.8	4.5	In one 4.4	3.8	0.1 As. in ½	0.015 As.		
II.	M., 47, S., D. 7½ h., "Arsenik pulver" dissolved in water.	.....	.....	.....	..... .	.....	.....	112.58	4.36	.....	.....	0.43	0.034		
III.	M., ad., D. 9 h., A. n. d., acute symptoms.	.....	.....	.....	.....	.....	.....	76.00	6.03	And b ladder. In one 0.6	0.18	Faint	trace.		
IV.	M., 27, S., D. < 12 h., As_2O_3 in water.	16.802	gm. soli	d As_2O_3	in c.	.....	.....	66.03	4.46	9.79	6.80	0.78	0.053	Thi gh. 0.95	0.158
V.	F., 22, S., D. 18½ h., A. 30 h., As_2O_3.	t. 21.57 c. 70.08	t. 6.51 c. 18.20	t. 79.7 c. 100.	t. 7.95 c.37.73	17.2	16.21	In half 33.04	5.32	29.2	16.22	Minute	trace.	Glu teal. 4.9	1.581
VI.	F., 38, H., D. 26 h., A. 32½ h., repeated doses.	c. 375.7	.....	c. 176.8	.....	.....	.....	180.05	.....	.....	.....	Tr	ace.		
VII.	F., ad., S., D. < 60 h., Paris green.	t. tr c. no	ace. ne.	.....	. ....	. .....	.....	12.78	0.13	And b ladder. 3.40	0.66	Tr	ace.	Thi gh. 0.97	0.13
VIII.	F., 17., S., D. 5 d., A. 2d d., 50 gm. mitis green.	.....	And pancreas 1.26	.....	0.66	.....	.....	.....	1.85	.....	0.53	.....	0.26	.....	0.32
IX.	M., 35, H., D. 11 d., A. 14½ h.	None	in c.	Present in duo	but not denum.	.....	.....	52.11	.....	Pre	sent.				
X.	M., ad., D. 14 d., As_2O_3 in bread.	t. 0.13 Empty.	0.035	In half 0.26	0.053	. ..	.....	In ⅓ 0.26	0.06	In one 0.2	0.07	Faint	trace.	Thi gh. 0.23	0.06
XI.	F., 43 H., D. 5½ d., A. 52 h.	Pres	ent.	Pres	ent.	.....	... .	123.6							
XII.	M., 8. m., H., A. 35 h.....	Small	quant.	Less stom	than ach.	.....	.. ....	16.5	5.32						
XIII.	M., ad., S., phosphorus, kaiser green, and caustic lye.	.....	.....	.....	.....	.....	.....	.....	0.92	.....	0.66	Tr	ace.		
XIV.	F., ad.....	And œso 10.3	phagus. 1.71	20.4	.....	.... .	.....	In half 7.1	.....	1.2	Dry 1.16	In ⅓ 1.6	.....	See p	age 464.

Abbreviations.—F. = female; M. = male (the number following indicates the age); ad. = adult; D. = death; h. = hours; m. = months; y. = years; H. = homicide; S. = suicide; A. = autopsy; t. = tissue; c. = contents; n.d. = next day. Quantities in milligrams of As_2O_3. I. Smith and Peck: Med.-leg. J., N. Y., 1891, ix., 129. Matter vomited in last hour of life and a little urine = 414.1. II. Ludwig: Med. Jahrb., 1880 (Repr. p. 21). III. Chittenden and Smith: Med.-leg. Jour., N. Y., 1885, iii., 46. IV. Ludwig: *Loc. cit.*, p. 19. Tibia and fibula (255 gm.) gave a distinct mirror. V. Bischoff, Lesser: "Atl. d. ger. Med.," 1884, p. 89. The duodenum was included with the stomach. In blood from heart, 19.3 = 14.84 in 100; heart muscle, 18.1 = 9.28 in 100; uterus, 20.8 = 5.47 in 100; embryo and coverings, 23.0 = 8.68 in 100. No trace in 275 gm. liquor amnii. VI. Wood and Hills. Wharton and Stillé: "Med. Jur.," 4th ed., 556. VII. Chittenden and Smith: *Loc. cit.*, p. 148. VIII. Bergeron, Delens and L'Hôte: Ann. d'hyg., 1880, 3 s., iii., 27. In heart, traces. In 100 gm. lung, 0.92. In 9 gm. hair, 0.13 = 1.44 in 100. In 100 gm. mammary gland, 0.43. IX., XI., XII., Zaaijer (van der Burg): Vierteljschr. f. ger. Med., 1886, n. F., lxiv., 256. In XI. present in left breast; woman was nursing. X. Chittenden: Med.-leg. J., N. Y., 1884, ii., 243. For distribution in different muscles see below, p. 464. XIV. Chittenden: Am. Ch. J., 1883-84, v., 8-14. Body found floating in shallow water; had not been long immersed. Lungs and spleen, ⅔ = 7.4; heart, ¼ = 1.82 (these had been preserved in alcohol together); trachea, larynx, and tongue = 5.3; diaphragm = 0.65.

the same quantity of liver, and in another (III.) the liver tissue contained thirty-three times as much as the same quantity of the kidney tissue; in one case the relations of the amounts in equal quantities of brain and liver was as 1 : 300 and in another as 1 : 84; and in one case the amounts in muscle and liver were as 1 : 28.2 and in another as 1 : 3.3.

Cases VIII. and X., in which life was prolonged for several days and in which comparable analytical results were obtained, indicate, so far as they go, that the distribution is more nearly equal in cases of long duration than in those which are more rapidly fatal. Thus, taking the amount in 100 gm. of brain tissue in Case VIII. as unity, the proportion in like quantities of other organs were: liver 7.1, kidney 2, muscular tissue 1.2, which is much nearer to equality than in I., II., and IV. The preponderance in the liver and kidneys is the greater the more rapidly fatal the case. In X. also the amounts in 100 gm. of liver (0.06), kidneys (0.07), and muscle (0.06) are very nearly equal.

It would seem that the only inference which can fairly be drawn from these results is that the liver, spleen, and kidneys uniformly contain a greater proportion of arsenic than an equal weight of muscle or brain, and that this preponderance, in some cases at least, tends to diminish as life is prolonged.

The total quantity of arsenic trioxid in the liver at death probably never exceeds 0.2 gm. (three grains), if indeed it ever reaches that amount. The largest quantity actually found has been 2.77 grains (0.18 gm.) in a homicidal case in which death was supposed to have occurred in twenty-six hours after the administration of the first dose.[1] In other cases in which the determination was made either with the entire organ or with a properly sampled fraction the results were:

	I.	II.	III.	IV.	V.	VII.	X.
Death in.....	6½ h.	7½ h.	9 h.	< 12 h.	18½ h.	< 60 h.	14 d.
As_2O_3 in milligrams	54.8	112.38	76.0	66.03	66.08	12.78	0.78
As_2O_3 in grains....	0.82	1.68	1.14	0.99	0.99	0.19	0.011

From these results it might be inferred that the proportion in the liver reaches its maximum at seven and a half-hours after

[1] See Case VI., Table A. In three cases larger quantities have been said to have been found in the liver. In two (Barker: Wharton and Stillé, "Med. Jur.," 4th ed., ii., 563, five grains; and Walther:

the ingestion of the poison, and that thereafter it is gradually and progressively removed by elimination. We do not, however, believe that such an inference is justified. In Case IV., although the amount of arsenic in the liver was less than in II. and III., there still remained in the contents of the stomach the enormous quantity of 16.803 gm. (259¼ grains) of arsenic trioxid in substance. Clearly in this case absorption would not have ceased had life been prolonged, and the relatively small amount of arsenic found in the liver may be due to one of two different conditions: Either absorption was not so rapid as in Cases II. and III., owing to a less soluble condition of the poison or to other causes; or the liver had at some previous stage contained a larger proportion of arsenic, which had been removed by an increased activity of elimination, coupled or not with a diminished rapidity of absorption. Although the former supposition is much more probably the true one, the impossibility of the latter is not demonstrated. Certain it is that the more soluble the form in which arsenic is taken the more rapid, *cæteris paribus*, will be the absorption, and consequently the sooner will the liver reach its maximum of saturation; and all facts support the view that there is such a maximum at or before which death necessarily arrests the process. (See Elimination.)

The table given by Taylor,[1] based upon the analyses of Geoghegan, which is so frequently referred to in support of the view that the liver reaches its "maximum of saturation" at about fifteen hours after ingestion, is affected with two sources of error: It is inferred that the liver in each case weighed three and a half pounds—which is merely an average weight—and it is assumed that the amount in the entire organ may be calculated from that found in an *unsampled* fraction, an assumption which has frequently led to inaccurate deductions. (See Quantitative Determination.)

Complete analyses have also been made in several cases of homicidal poisoning after a burial of from three months to two years and eight months. These are arranged in the following table, the amounts representing milligrams of As_2O_3:

Vierteljschr. f. ger. Med., 1862, xxii., 188, 2.8 grains) the amount was calculated from the results of an analysis of a small, unsampled portion, and are therefore unreliable (see Quantitative Analysis). In the third case (Bingley: Lancet, 1864, i., 697) one-half of the liver is said to have yielded 4.15 grains!

[1] "Poisons," 3d Am. ed., p. 40.

B. Human Bodies—Late Autopsies.

No.	Conditions.	Stomach.		Intestines.		Spleen.		Liver.		Kidneys.		Brain.		Muscle	
		Found	In 100 gm.	Found.	In 100 gm.	Found	In 100 gm.	Found	In 100 gm.	Found.	In 100 gm	Found	In 100 gm	Found	In 100 gm
XX.	M., ad., D. 7 d., A. 3 m....	.....	.....	t. 20.3 c. 6.9	2.49 3.48	.....	.....	17.9	3.08	11.6	3.75	Part of 10.8	1.72		
XXI.	F., ad., D. 2 d., A. 5 m....	t. 41.2 c. 9.7	21.7 16.2	t. 63.8 c. 20.5	13.0 33.06	.....	.....	49.7	4.05	6.9	2.51	8.8	1.69		
XXII.	M., ad., A. 11 m.....	t. 9.0 c. 2.2	9.2 44.0	t. 10.3 c. 4.3	2.99 10.75	3.2	6.96	87.8	8.54	One 4.3	6.11	5.2	0.68		
XXIII.	F., ad., H., A. 1½ y.....	And spleen. 53.59	10.40	And uterus. 25.82	2.60	.....	.....	47.88	8.11	6.60	8.25	Unweighable trace.		See page 464.	
XXIV.	F., ad., H., D. 53 d., A. 5 m., chronic.	.....	.....	.....	.....	No trace.		No trace.		No trace.		No trace.			
XXV.	M., 36, H., D. 12 d., A. 14 m., acute upon chronic.	200 gm.		= 3 to 4.		.....	.....	.....	3.8	With spleen and heart. 100 gm. = 2 to 3.		200 gm = distinct mirror.			
XXVI.	F., 26, H., D. 11 d., A. 2 y. 8 m., chronic.	200 gm.		= 0.5 to 1.		.....	.....	.....	1.5	With spleen and heart, 100 gm. = 1 to 2.		200 gm = distinct mirror.			
XXVII.	No conditions mentioned.	In 205 15.7	21.7	In 300 5.28	1.76	.....	.....	In 266 3.69	1.39	.....	.....	In 700 gm. trace.		In 352 0.4	0.15

XX. Wood and Hills, Wharton and Stillé: "Med. Jur.," 4th ed., ii., 159 (Case 2). XXI. *Ibid.*, Case 1, p. 158. XXII. *Ibid.*, Case 3. XXIII. Johnson and Chittenden: Am. Ch. J., 1880-81, ii., 332. One lung and heart = 14.54 = 3.29 per 100; one lung and liquid from thorax: 5.83 = 1.40 in 100; urinary bladder, etc., distinct trace. XXIV.-XXVI. Brouardel and Pouchet: Ann. d'hyg., 1889, 3 s., xxxii., 362. The determinations in these cases (Aff. Pastré-Beaussier), except those of the livers, were by comparison. (See Quantitative Determination.) XXVII. Guareschi: Riv. d. ch. med.-farm., etc., Torino, 1883, i., 17-20. Lungs and heart, 300 gm., 1.8 mgm., = 0.6 mgm. per 100. No particulars are given as to the nature of the case or the interval between death and analysis.

Inferences as to the distribution which existed at death, drawn from such analyses, are, we believe, entirely unreliable, as it is impossible in the present condition of our knowledge to form any adequate idea of the degree to which it has been subsequently modified by post-mortem migration. That such post-mortem distribution occurs with arsenic introduced into the cadaver after death cannot be questioned (see page 481); and if arsenic introduced during life does not travel from one tissue to another after death it is because it has formed an insoluble compound with the constituents of the tissues, a supposition in favor of which there is no evidence and against which the rapid elimination of arsenic during life militates strongly. The amounts of arsenic found in the brains in Cases XX., XXI., XXII. are greatly in excess of any of the analyses made shortly after death (see Table A, page 459), and those in Cases XXV. and XXVI. are also relatively large. To explain the presence of these large amounts in the brain we are forced to one of two suppositions: Either in these cases the brains contained large quantities of arsenic, or the arsenic travelled to the brain post mortem. The former supposition is in opposition to the results of all analyses made shortly after death, the latter is in accordance with what is known concerning post-mortem imbibition. In Case XXI. a difference between the amounts in the two kidneys—the right, weighing 125 gm., containing 2.6 mgm. or 2.08 per cent., and the left, weighing 149 gm., 4.3 mgm., or 2.88 per cent.—may be due either to a greater activity of the heavier organ during life, or to more direct post-mortem imbibition from the stomach to the left than to right kidney. Whatever the relative activity of the two kidneys may be during life, it is certain that post-mortem imbibition from the stomach carries a larger quantity into the left than into the right kidney.[1]

In a few cases the relative distribution of arsenic in different parts of the muscular, osseous, and epidermic tissues has been determined. The results were as follows, expressed in milligrams of As_2O_3 in 100 gm. of tissue:

[1] A greater difference between the amounts in the two lungs in Case XXIII. is partly explained by the fact that the heart was included with one and some liquid from the thoracic cavity with the other; but whether or no the difference is entirely due to this cause is uncertain. (See p. 483.)

C. Distribution in Muscles and Bones.

No.	Arm.	Forearm.	Hand (entire).	Thigh muscle.	Leg.	Foot.	Muscle from breast and ribs.	Abdominal muscle.	Transverse section at pelvis.	Muscle from back.	Trachea, larynx, and tongue.	Diaphragm.	Femur.	Vertebræ.	Skull bones.	Skin.	Hair	Nails
XXX.	0.81	0.55	0.12	0.51	0.65	0.22	0.91	0.58	1.56	.....	.....	.....	0.06					
XXXI.	Left arm 0.5			Right leg	except 0.16	femur.	Muscle alone, 1.25	.....	0.3	3.73	3.01	0.12	Absent.					
XXXII.	....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	Present.	Present.	Scalp, present.	Present	
XXXIII.	....	.....	.....	.....	.....	.....	.....	....	.	.....	.....	.....	0.2	0.28 to 0.56	0.15	.....	2.5 to 5	About 10.0
XXXIV.	...	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	....	200 gm. Very faint.	0.11 to 0.22	0.16 to 0.31	Abdomen, faint	1.0	2.30 gm distinct.
XXXV.	..	.....	.....	0.16	.....	.....	.....	.....	.....	.....	.....	.....	Tibia and fibula = distinct.					
XXXVI.																		

XXX. Johnson and Chittenden: Am. Ch. J., 1880-81, ii., 332 (see also Case XXIII., Table B, p. 462). The medical testimony in this case leaves no room to doubt that it was one of repeated administrations. The body was exhumed one and one-half years after death. XXXI. Chittenden: Am. Ch. J., 1883-84, v., 8 (see also Case XIV., Table A, p. 459). These parts were taken at a second autopsy six months after death. XXXII., XXXIII., XXXIV. Brouardel and Pouchet: Ann. d'hyg., 1889, 3 s., xxii., 362, *et seq.* (For analysis of internal viscera see XXIV., XXV., XXVI., Table B, p. 462.) Determinations by comparison. Poisoning by repeated doses. Exhumation five months, fourteen months, and two years eight months, after death. XXXV. Ludwig: Same case as IV., Table A, p. 459; acute poisoning. In another acute case (XIII., Table A) Ludwig found more arsenic in the skull bones and in a tibia than in the brain.

Unfortunately in most of these the body had been buried for several months or years, and in them, consequently, the distribution was subject to post-mortem modification. But, notwithstanding this, the absence of arsenic in the femur in a case presumably (although by no means certainly) acute in character, and its presence in a case of poisoning by repeated doses would retain all of the importance attached to it by Chittenden[1] for the purpose of distinguishing between acute and chronic poisoning in doubtful cases, were such a difference found to exist constantly in recent cadavers. But in other acute cases the bones have been found to contain arsenic, as in Ludwig's cases above mentioned. That author, in summing up the results of his experiments and observations, says: "Arsenic is taken up by the bones, although in small yet distinctly detectable quantity. So far as human bones are concerned I could only observe this fact in acute poisoning; without doubt it is also true in chronic poisoning, in which I have noted it in the dog."

The three analyses (XXXII.–XXXIV.) by Brouardel and Pouchet were made in investigation of the Affaire Pastré Beaussier in 1889, and the clinical histories leave no doubt of their chronic character. In XXII. the illness was of five weeks' duration after the last administration. Arsenic in minuter quantity was still found in the skull and vertebral bones, although no trace of it remained in the liver, spleen, kidneys, or brain. In XXXIII. and XXXIV. the amount was also greater in these bones than in the femur. This is in accordance with the results of experiments upon animals made by Brouardel,[2] which indicated that whatever the method of introduction "arsenic accumulates very sensibly in the *spongy tissue* of bones and becomes fixed in such manner that its presence can be detected, in the bones of the skull or vertebræ particularly, some time after every trace has disappeared from those organs, such as the liver, in which it is localized in greater amount." "This localization in the bones is less accentuated and the elimination is more rapid when arsenic is absorbed in quantities sufficient to cause immediate symptoms of poisoning. When animals are poisoned by very large doses, there is a general diffusion of the

[1] Med.-leg. Jour., N. Y., 1884, ii., 244, 246.

[2] Ann. d'hyg., 1889, 3 s., xxxi., 486. Bull. Ac. de méd., Paris, 1889, 2 s., xxii., 4, 53.

arsenic, and the osseous tissue presents nothing particular in regard to localization: arsenic is found in all the organs."

The localization of arsenic in the bones is explainable on the supposition that the phosphates which they contain are partially replaced by arsenates. The chemical relationship of these two classes of salts is well known, and the formation of arsenate in this manner is quite as probable *a priori* as the conversion of phosphate into carbonate, which has been shown by Milne-Edwards[1] to take place normally in bone. In support of this view we have also the results of Binz and Schultz, who have shown that arsenious acid is converted into arsenic acid in the economy;[2] and those of Caillol de Poncy and Livon, who have shown that under the influence of arsenic the elimination of phosphates by the urine is increased.[3]

Arsenic has been found in the bones after long burial, during which the soft parts have been almost completely disintegrated. In one instance Liman,[4] in a skeleton examined after eight years, found traces of arsenic in the upper part of the femur and in the pelvic bones, and a distinctly larger quantity in the tissue adherent to the sacrum. In 1847 arsenic was found in the bones of a skeleton exhumed after ten years' burial, but none in those of another from an adjoining grave.[5]

When a pregnant woman dies from the effects of arsenic the poison is found in the fœtus and its coverings, but not in the amniotic fluid.[6] In a case of supposed arsenical poisoning after abortion Liman found arsenic in the remains of the woman two years and three months after death, but none in the fœtus, which had been preserved in alcohol.[7]

Garnier[8] gives the results of analyses in the cases of three patients who died of tuberculosis while under treatment with Fowler's solution. Traces of arsenic were obtained from the liver and brain in each, the brain containing somewhat more than the liver in two cases, while in the third the reverse was the case.

Experiments upon animals with regard to distribution have

[1] "Études sur les Os," 1860, 151, Repr. fr. Ann. d. Sc. nat., 4 s., xiii.

[2] Arch. f exp. Path. u. Ph., 1879, xi., 212.

[3] Jour. de pharm. et de chim., 1879, 4 s., xxx., 344.

[4] Casper-Liman: "Handb. d. ger. Med.," 8te Aufl., ii., 446.

[5] Jour. de chim. méd., etc., 1847, 3 s., iii., 82.

[6] Bischoff: In Lesser's "Atl. d. ger. Med.," i., 89. Husemann: "Handb. d. Toxikologie," p. 823.

[7] Casper-Liman: "Handb. d. ger. Med.," 8te Aufl., 425.

[8] Thèse, Nancy, 1880, No. 107, p. 47.

D. Distribution—Experiments on Animals.

No.	Conditions.	Stomach.	Intestines.	Liver.	Kidneys.	Urine.	Spleen.	Heart and lungs	Brain.	Spinal cord.	Muscles.	Bones.	Bile.
1	Dog, received increasing doses sodium arsenite 34 days; 5–15 mgm. daily. Healthy; killed.	.....	.....	2.71	.....	.....	.....	.....	8.85	9.33	0.25		
2	Rabbit, received 5–50 mgm. sodium arsenite, 15 days; death, 15th day.	.....	.....	Faint.	.....	.....	.....	.....	5.94	Very large.	Very faint.		
3	Dog, received 5–60 mgm. sodium arsenite; 32 days; killed.....	.....	.....	Not weighable.	.....	.....	.....	.....	4.22	Large mirror.	2.10		
4	Dog, received 2 gm. As_2O_3 in solution; death after symptoms of acute arsenical poisoning.	.....	.....	8.4	.....	6.0	.....	.....	0.5				
5	Dog, same conditions	.....	.....	5.3	.....	.....	.....	.....	0.4				
6	Dog, received 6.5 gm. solid As_2O_3 during 8 days; doses, 0.1–2.5 gm.; killed 24 hours after last.	.....	2.0	1.0	0.44	0.3	.....	.....	Faint mirror.	.....	0.2		
7	Dog, killed by stomachal injection of 2 gm. sodium arsenite in solution.	.....	.....	1.05	0.69	.....	.....	0.39	0	.....	0.52		
8	Dog, same conditions.....	.....	.....	0.50	3.75	.....	3.33	0.08	0	.....	0.07		
9	Rabbit, same conditions; dose, 0.2 gm.	.....	.....	5.00	0.3	.....	.....	0.02	0.5	.....	1.16	0.33	
10	Rabbit, Fowler's solution = 1 gm. As_2O_3 in 92 days.	.....	.....	4.00	0	.....	.....	Trace.	Trace.	.....	Trace.	Trace.	Trace.
11	Cow, 0.4–0.5 gm. As_2O_3 daily, 44 days.	1.9	.....	1.14	0.45	.....	0.76	Lungs 0.3	.....	.....	0.38		

1–3, Scolosuhoff; 4, 5, Ludwig; 6, Johnson and Chittenden; 7–10, Garnier; 11, Stroppa and Monari (Selmi): Riv. d. chim. med. e. farm., etc., Torino, 1883, 1., 325; Blood, 0.38 mgm. in 100 gm.

also been made by Scolosuboff,[1] Ludwig,[2] Garnier,[3] and Johnson and Chittenden,[4] the results of which are given in the preceding table, in milligrams of elementary arsenic in 100 gm.

From the results of his experiments Scolosuboff inferred that arsenic is localized particularly in the nervous tissues, and that in acute poisoning the expert should search for the metalloid particularly in the brain, as the liver, if the poisoning be very rapid, may sometimes not contain it. This view is in opposition to the universal experience of toxicologists in analyses of the human subject, in which the quantity of arsenic found in the brain is usually very small absolutely and relatively, and so far as we are aware has never been found to exceed 1.66 mgm. in one-third of the organ,[5] in an analysis made shortly after death. The experiments of others given in the above table have also led to different results.

As an explanation of this supposed localization in the brain Scolosuboff advanced the theory that arsenic is substituted for phosphorus in the lecithins or other phosphorized organic constituents of nerve tissue. This theory has not, so far as we know, been supported by experimental or other proof.

The view of Chittenden and Smith[6] that the localization in brain tissue, while not occurring with arsenic trioxid, does take place as found by Scolosuboff in poisoning with sodium arsenite, is not substantiated by the results of the experiments of Garnier given above. It is to be regretted that, so far as we can find, no quantitative analysis of the brain has been made in a case of poisoning in the human subject by Fowler's solution or by arsenite in other form.

Elimination.—Several questions of great forensic interest depend upon the elimination of arsenic from the system.

It is stated by most writers that arsenic is not a cumulative poison. This expression is used in three distinct senses. If by "cumulative" is meant that repeated medicinal doses may gradually accumulate in the body in such manner that finally, acting unitedly and without the administration of a dose poisonous in itself, they may cause the symptoms of acute poisoning, then

[1] Bull. Soc. chim., Paris, 1875, xxiv., 125.
[2] Med. Jahrb., 1880.
[3] Ann. d'hyg., 1883, 3 s., ix., 314; and Thèse, Nancy, 1880.
[4] Am. Ch. J., 1880, ii., 337.
[5] Case XIV., Table A, p. 459.
[6] Med.-leg. Jour., N. Y., 1885, iii., 145.

arsenic is certainly not a cumulative poison, as it is constantly used in medical practice, over long periods of time, without the production of such accidents.[1] Or it may be considered cumulative in its action in the sense that when given in repeated small doses it produces effects which do not result directly from a single dose, however large. In this sense it is cumulative, as the clinical history of a poisoning caused by repeated small doses is distinct from that of one resulting from a single large dose, although the symptoms of "chronic" poisoning sometimes follow the acute symptoms due to a large dose without any further administration. Again, the question may mean: Does arsenic enter into combination with the constituents of the tissues to form compounds which are difficult of elimination and are retained in the body for a long period after administration has ceased? To some extent arsenic is cumulative in this sense, and that to a greater degree when given in repeated small doses than when a single large dose has been taken. It is probable, however, that this action is limited to the bony and epidermic tissues, in the former of which there is every reason to believe that it to a certain extent replaces the natural phosphate with an arsenate. On the other hand, it is not retained by the soft parts for so long a period, certainly not to the degree that lead and copper are.

Elimination of arsenic probably begins very early and during the progress of its absorption, although we can find no record of analyses of urine passed by the human subject during the first hours of poisoning. According to Husemann[2] it makes its appearance in the urine often in five to six hours, and with potassium arsenite in an hour. On the other hand, Taylor[3] quotes a case of Geoghegan's in which no arsenic was found in the small quantity of urine passed up to the fourteenth hour, and that passed from fourteen to thirty-six hours yielded only faint indications.

The elimination from the bones, particularly those rich in spongy tissue, takes place more slowly than from the soft parts in cases of poisoning by repeated doses, and probably in acute cases also. Thus in the case of the woman Morisse, one of the

[1] See Taylor: "Poisons," 3d Am. ed., 294. Seidel: Maschka, "Handb. d. ger. Med.," ii., 249.

[2] "Handb. d. Tox.," 823.

[3] "Poisons," 3d Am. ed., p. 38.

victims of the Havre poisonings, who died in six weeks after having been removed from the possibility of further administration, the liver, spleen, kidneys, and brain contained no traces of arsenic, which was readily detected in the bones of the skull, the vertebræ, and the hairs.[1]

Elimination from the soft parts, when a single large dose has been taken, may be complete in about fifteen days, to such extent at least that no detectable quantity remains. In a few instances analysis has failed to reveal the presence of any trace of arsenic in the soft parts when death has occurred at about this interval.

In the case of Dr. Alexander a large quantity of arsenic trioxid mixed with arrow root was taken by mistake. He suffered the usual symptoms and died on the sixteenth day. The stomach was found to be ulcerated, but no arsenic was found on analysis of stomach, liver, and spleen.[2]

In the case of Reg. *v.* Williams (South Wales Circ., July, 1863) a clear case of arsenical poisoning was made out. Death occurred in fifteen days after the administration, and no arsenic was found in the cadaver on analysis.[3]

Elimination, aided by copious vomiting, caused the removal of all detectable quantities of arsenic in the case of a man who died in eleven days after having been poisoned by arsenic in coffee. No arsenic was found in either stomach, intestines, liver, or spleen on analysis.[4]

On the other hand, arsenic has been found in the liver, kidneys, muscles, and tissue of the stomach and intestines, although in small quantity, in one case in which death followed in fourteen days after a single administration.[5]

Although we know of no instance in which the poison was detected in the cadaver in an acute case of longer duration than this, there are some observations which indicate that in non-fatal cases elimination by the urine may continue for a much longer period, even when but one dose has been taken:

A woman of thirty-five years took by mistake half a dessertspoonful (about 8 gm.) of white arsenic in water. She suffered the usual symptoms of the mixed type and was under treatment in the hospital

[1] Brouardel and Pouchet: Ann. d'hyg., 1889, 3 s., xxii., 362, 484.

[2] Geoghegan: Med. Times and Gaz., 1857, 389.

[3] Herapath: Lancet, 1863, ii., 47.

[4] Maschka: Vrtljschrf. f. ger. Med., 1863, xxiii., 261–270.

[5] See Case X., Table A, p. 459.

for twenty-five days. The urine of the second day gave an abundant arsenical deposit with the Marsh test. A similar result was obtained with the urine of the fourth day. Samples passed on the fifth, eighth, sixteenth, and twenty-first days gave evidence of the presence of arsenic in diminishing quantity. Negative results were obtained with the urine passed on the twenty-fifth day, as well as with that passed on the twelfth day.[1]

Carter[2] quotes a case from Gubler, concerning which, however, there appears to be some doubt as to the date of administration and the dose administered: The patient had probably taken 8 gm. (about two drachms) of arsenic trioxid on September 25th, 1864. On October 27th an analysis showed the presence of "a large quantity of arsenic in the urine." Another analysis made thirteen days later, forty-five days after the poisoning, still showed the presence of arsenic in the urine. On November 10th another analysis was made without revealing any traces of arsenic. On November 15th or 16th potassium iodid was given and "on the 19th another examination of the urine was made, and a considerable quanity of arsenic found to be present."

Wood,[3] using the more direct method of extraction suggested by Sanger,[4] found arsenic in the urine ninety-three days after the ingestion of a single large toxic dose of arsenic, which had caused the usual acute symptoms, followed by arsenical paralysis.

When arsenic is administered in repeated doses, whether in the proper medicinal quantities or in somewhat larger amounts, the elimination, after cessation of the dosing, continues during periods about as long as in acute cases:

In the observations of Garnier and of Ritter[5] of six cases of administration of arsenic in medicinal doses in the shape of granules of arsenic trioxid or of Fowler's solution, for periods varying from seven days to six months, arsenic was no longer detectable in the urine in from seven to twenty-four days after discontinuance of the treatment.

A girl of twenty-two years was directed to take Fowler's solution for the treatment of eczema, in doses of fifteen drops morning and evening for fifteen days, then three times a day for fifteen days, and finally twenty drops three times a day for fifteen days. She followed this treatment for the first two periods, taking, consequently, during the first period of fifteen days daily doses equivalent to 0.015 gm. (equal to one-fourth grain) of arsenic trioxid, and during the second to 0.022 gm. (equal to one-third grain); but on attempting to increase

[1] Maclagan: London and Edinb. Monthly Jour. M. Sc., 1852, xiv., 132.

[2] Liverpool Med.-Chir. Jour., 1890, x., 137.

[3] Bost. M. and S. J., 1893, cxxviii., 414.

[4] See pp. 492, 508.

[5] Garnier: Thèse, Nancy, 1880, 76.

the dose to 0.032 gm. (equal to one-half grain) she was obliged to abandon the treatment in consequence of the vomiting, pain, cramps, and paralyses which resulted from it. She was seen by Dr. Gaillard five weeks after having stopped the Fowler's solution, during which she had suffered the usual arsenical symptoms. The urine then contained arsenic, which continued to be eliminated by the urine until forty days after the date of cessation of the arsenical treatment.[1]

Wood[2] examined the urine of two patients by Sanger's method after the cessation of medicinal administration of Fowler's solution. In one arsenic was detected up to the fifty-ninth day, and in the other up to the eighty-third day after the last administration.

The elimination by the urine of quantities which can be extracted by the usual methods may also cease at an earlier time, even in non-fatal cases:

In a case of poisoning by about an ounce (31 gm.) of white arsenic in water, from which the patient finally recovered, arsenic was detected in the urine in diminishing quantity during the first four days, but not on the fifth.[3] In a case of non-fatal poisoning by Fowler's solution, arsenic was detected by the Reinsch test in the urine of the fifth day, but not in that of the sixth.[4]

When, as occasionally happens, the secretion of urine is suppressed in the early history of the poisoning and is subsequently resumed, that which is then voided contains arsenic:

In a woman poisoned by 15 gm. (about half an ounce) of white arsenic, no urine appeared during seven hours, after which she passed ten litres (two and one-half gallons) in ten hours, in which arsenic was detected.[5] A woman died in five days from the effects of 6 gm. (ninety-three grains) of white arsenic suspended in water. For the first three days the urine was entirely suppressed, and on the last two she passed 100 and 150 gm. (three and one-half and five fluidounces) of urine containing arsenic.[6]

Although the urine is the most obvious and, during the earlier stages of the poisoning, probably the most active channel of elimination of arsenic, in cases of long duration the excretory action of the kidneys may be diminished to such an extent that the urine no longer contains any detectable quantity of the

[1] Gaillard: Bull. Soc. méd.-lég., Paris, 1873–74, iii., 249. Also Ann. d'hyg., 1874, 2 s., xlii., 406.

[2] *Loc. cit.*

[3] Rademacher: Richm. and Louisville M. Jour., 1873, xv., 383.

[4] Powell: Westm. Hosp. Rep, 1889, v., 215–219.

[5] Angonard: Gaz. méd. de Par., 1843, 2 s., xi., 222.

[6] Bouillet: Bull. Ac. de méd., Paris, 1840–41, vi., 544, 553.

poison, which after death may still be detected even in the soft parts. The failure to detect arsenic in the urine by the usual methods is consequently not sufficient proof that elimination is complete.

A young woman had been treated with Pearson's solution for two months. On admission to the hospital this was discontinued. She died in nineteen days. The urine examined on the fifteenth day showed no trace of arsenic. The liver, which was very large, yielded a mirror of arsenic estimated to weigh 1 to 2 mgm.

A young woman had taken Fowler's solution for eight months, with an interval of two months. On entrance into the hospital a medication with bismuth subnitrate (non-arsenical) in large doses was substituted. She died in thirty-five days. The urine contained no arsenic after the twenty-seventh day. The liver yielded 1.9 mgm. of elementary arsenic on analysis.[1]

The Duc de Praslin died in six days after having taken a large dose of arsenic. The poison was found in the liver and intestines after death, but no trace was detected in the urine passed in the last moments of life.[2]

Although the gall bladder is frequently found full of bile after death from arsenic, we know of no instance in the human subject in which the bile has been analyzed separately from the liver. But while direct analytical evidence of elimination by the liver is thus wanting, there is every reason to believe that it occurs throughout the course of the poisoning. Sonnenschein[3] reports a case of attempted poisoning by repeated doses of extract of arsenical fly papers in which the analysis showed the presence of arsenic in the fæces but none in the urine. In this case, apparently, elimination through the alimentary canal continued after it had ceased through the kidneys, and the question is only whether the excretory organ was the liver or the gastro-intestinal mucous membrane.

Arsenic, when taken by the stomach, is found in the skin and in notable quantity in the nails[4] and is in all probability eliminated to some extent in the perspiration. Sonnenschein[5] in the Knothe case found small but distinct quantities in the bed clothing upon which the head of the deceased had lain, and in a

[1] Ritter: Rev. méd. de l'Est, Nancy, 1878, ix., 257–267.

[2] Tardieu: "Empoisonnement," 2ème ed., 422.

[3] "Gericht. Chemie," 1869, p. 120.

[4] See Table, p. 464.

[5] "Gericht. Chemie," 1869, p. 119.

position where neither the vomit nor the poison itself could have accounted for its presence. Chatin[1] found arsenic in the serum of a vesication produced upon the skin of an arsenical patient, and even suggested the application of blisters and the chemical examination of the serum to establish the diagnosis in doubtful cases.

When arsenic is applied to the skin or to exposed mucous membrane, the discharges which its irritation produces contain arsenic, which is, however, probably a portion of that which was applied.[2]

The hair has been found to contain arsenic in notable quantity in several cases in which the body was examined chemically after long burial. Thus Sonnenschein found it in the amount of 2.4 mgm. in 95 gm. of the hair of a cadaver exhumed after two and a quarter years, in which arsenic was also found in other parts of the cadaver, but none in the sawdust with which the body was surrounded,[3] and Brouardel and Pouchet found it in the amounts of 1 mgm. and 2.5 to 5 mgm. per 100 gm. in the hairs of two of the Havre victims, buried two and two-thirds years and fourteen months respectively.[4]

Liman, in discussing "an unsolved riddle" in which arsenic was found in the hair, but in no other part of the cadaver or its surroundings after a burial of eleven years, considers it probable that the arsenic in such cases reaches the hair rather by post-mortem processes than during life.[5]

On the other hand, in the description of Gubler's case, cited above,[6] it is stated that the head was shaved and that "her hair grew again, and I thought, after a certain number of months, that it would be curious to know if the hair contained arsenic. The analysis of the hair, made eight months after the poisoning, gave a quantity of arsenic considerable enough." Brouardel and Pouchet[7] also recommend that the hair be cut and analyzed for the purpose of establishing a diagnosis during life in doubtful cases.

[1] Jour. d. chim. méd., etc., 1847, 3 s., iii., 328.

[2] Brisken: Vierteljschr. f. ger. Med., 1864, xxv., 110–126. Taylor: Guy's Hosp. Rep., 1864, 3 s., x., 220–236. Cameron: Brit. M. Jour., 1890, ii., 203.

[3] Casper-Liman: "Handbuch d. ger. Med.," 8te Aufl., 424.

[4] Ann. d'hyg., 1889, 3 s., xxii., 486.

[5] Casper-Liman: *Op. cit.*, p. 411.

[6] Carter: Liverpool Med.-Chir Jour., 1890, x., 137.

[7] *Loc. cit.*

FORENSIC QUESTIONS.

We have already (pages 163–184) considered a number of questions which may be formulated in cases of supposed poisoning by any toxic agent. Besides these there are others which relate more particularly, almost exclusively, to arsenic. Of these we have considered such as relate to the non-homicidal and more or less legitimate introduction of arsenic into the body during life in the preceding pages. There still remains the cardinal question: **Was the arsenic found in the cadaver of ante-mortem or of post-mortem origin?**

We believe that in the present condition of the science this, in many cases, is a question which must be left to the jury to determine in the light of the entire testimony in the case, rather than one upon which an expert may safely express a positive opinion in the light of the scientific evidence alone. And this we say without prejudice, be it understood, to the opinion that a scientific jury would be much more competent to properly weigh the evidence than a lay jury.

As toxicologists are by no means unanimous upon this point, we will state the grounds for the opinion we have expressed somewhat at length, considering, to begin with, the possible sources of arsenic in a cadaver other than from ante-mortem *introduction*, whether criminal or legitimate:

1. Normal Arsenic.—The question of the existence of arsenic as a normal constituent of the human body may be summarily dismissed with a negative. The only toxicologist who considered the affirmative of the supposition favorably was Orfila, whose views were based upon defective analyses. To-day it is the unanimous opinion of toxicologists that arsenic is not normally present in the body, either strictly as a normal constituent, or, in the same manner as copper, by constant absorption with food articles.[1]

2. Undertaker's Arsenic.—European toxicologists only consider the intentional introduction of arsenic into a cadaver as a remotely possible manifestation of the acme of human depravity. In the United States, however, we are unfortunately under different conditions, and among us in recent years a non-

[1] Except among drinkers of arsenical waters (see p. 362).

arsenified body twenty-four hours after death is only met with when some accident or superior power has stayed the hand of the "funeral director." Bodies are all "embalmed" except in the coldest weather; and, even when no internal injection is practised, cloths saturated with embalming liquid are kept upon the face and other parts previous to the funeral. Of the many embalming liquids which we have examined all but two were arsenical. One of these, which was valueless for the purpose for which it was intended, was a tolerably strong solution of hydrogen peroxid; the other, which came from California, was a two-per-cent. solution of boric acid in dilute spirit, colored with carmine. All others were either simple aqueous solutions of arsenites or arsenates or combinations of these with corrosive sublimate, zinc chlorid, alum, methyl alcohol, or glycerol.

The method of introduction varies with the skill of the undertaker, from that of the village practitioner, who contents himself with the injection of a pint or two of liquid through a trocar pushed through the abdominal wall, to that of the graduate of a "College of Embalming" who ties a canula into what he believes to be an artery[1] and more or less rapidly and completely permeates the body with a gallon or so of liquid containing arsenic and other poisons. The practice is entirely unrestricted by regulations of any kind in New York State and, so far as we know, in other States, and tons of arsenical embalming liquids are manufactured and sold annually.[2]

3. "Cemetery Arsenic."—The presence of arsenic in many soils, and in that of some cemeteries was considered by some of the earlier toxicologists as a possible source of contamination of bodies buried therein, and many pages of the works of Orfila, Devergie, Galtier, and others are devoted to discussions of the subject. It was demonstrated, however, that arsenic existing naturally in the soil is always in a form of combination insoluble in water, and hence only transferable to a body buried in it by fracture of the coffin and physical admixture of the arsenical soil with the remains.

[1] We have seen such an "adept" make the injection into the basilic vein, believing it to be the brachial artery.

[2] In France the use of arsenic for embalming was prohibited as early as 1846 (Parcelly: "Embaumements," Paris, 1891, p. 128). See also Durrell: Boston M. and S. Jour., 1890, cxxi., 544. Suiter: Tr. M. Soc., N. Y., 1891, 394.

But, although the question of the possibility of contamination from natural "cemetery arsenic" has been definitely set at rest, that relating to artificial cemetery arsenic is one of recent origin, local in the United States, and still demanding solution. The arsenic in embalming liquids is, of course, not insoluble, but quite soluble. How long does it remain soluble, and how far may it permeate the soil of the cemetery and unembalmed bodies buried therein? It is true that during putrefaction of a body containing arsenic a part or all of the poison may be converted into arsenic trisulfid, which is insoluble in acid or neutral liquids. But the trisulfid is soluble in alkaline liquids, and an alkaline reaction is not an improbable condition in view of the generation of ammonia during putrefaction. Another condition necessary to the migration of arsenic is the presence of water. In this respect cemeteries differ markedly. In one cemetery during the month of March we have found a casket and the embalmed body to be perfectly dry when exhumed two months after burial, while in an exhumation in another cemetery two weeks later the grave filled with water nearly to the brim, and the casket and contents were thoroughly water-soaked.

It is true that arsenic may be rendered insoluble in neutral liquids not only by conversion into the trisulfid, but also by reaction with the calcareous and ferruginous constituents of the soil. The coverings of a body embalmed with sodium arsenate, a compound much more slowly converted into trisulfid than the arsenite, were found after one hundred and ninety-seven days' burial to contain notable quantities of arsenic trisulfid, but no arsenic soluble in water. But as the casket was water-soaked it is presumable that soluble substances had been leached out during the six months' burial with more or less contact of water. In some experiments of Garnier and Schlagdenhaufen[1] made with arsenates of iron, calcium, and potassium, it was found that permeation of the soil did not extend in fourteen months to distances 0.6 and 0.9 m. (twenty to twenty-nine and a half inches) below the point at which the samples were placed. In these experiments the quantities used (5 gm.=seventy-seven grains) were out of all proportion to the quantities used in embalming.[2]

[1] Ann. d'hyg., etc., 1887, 3 s., xvii., 33.

[2] The authors of these experiments make no mention of embalming in enumerating the possible sources of arsenic in the body.

Ludwig and Mauthner[1] refer to the possibility of the earth of cemeteries becoming contaminated with soluble arsenic from arsenical ornaments and colors, and from manufacturing establishments in the neighborhood.[2]

4. Arsenic in Articles of Ornament.—Arsenic is a constituent of many pigments, and, if such arsenical colors have been used in the manufacture of any colored article of dress or ornament which is put into the coffin, there is danger that arsenic may be thus introduced into the cadaver. The possibility of contamination from this source has been discussed in at least four cases:

In the remains of a child of six years which had been buried a little more than eight years, and which had been reduced almost to the skeleton, four grains of arsenic were found; and in a fragment of paper-like material of a green color, half a grain of arsenic. The fragment was the remains of four religious pictures which had been placed upon the breast of the cadaver before burial, and the question is discussed whether the arsenic found in the remains might not have been entirely derived from the arsenical colors of the pictures. The "*gutachten*" declares the probability that the deceased died of arsenic, principally in view of the amounts of arsenic found, and of the symptoms observed during life.[3]

The body of a child of two years was exhumed after three and a half years' burial. The soft parts were reduced to a brown, soapy mass. Upon the body remains of red bands, shreds of a red fabric, numerous remains of a green and blue-green fabric, some glass balls, and some more consistent green fragments, were found, and were probably the remains of artificial flowers. The colors of the fragments were in places well preserved, in others faded, and in others completely wanting. In the remains of the body a quarter of a grain of arsenic trioxid was found, and a small quantity of copper. In all of the colored objects described copper was present; the blue-green fabric contained traces of arsenic; and the remains of artificial flowers considerable quantities of arsenic. The "*gutachten*" concludes "that a distinct foundation for the supposition that a poisoning occurred is wanting, but on the other hand the possibility that such may have occurred is not excluded, as it may be that all of the arsenic found in the cadaver did not come from the pigments, but a part of it may have been administered during life; a positive judgment upon this point can,

[1] Wien. kl. Wochenschr., 1890, iii., 691.

[2] See also Sonnenschein: Vrtljschr. f. ger. Med., etc., 1870, n. F., xiii., 169.

[3] Reuter: Med. Jahrb. f. d. Herzogth. Nassau, 1846, 206–212.

however, not be given, either from the medical or the chemical standpoint."[1]

The body of a pregnant girl, which had been found in a pond, in which she was presumed to have drowned herself, was, upon the supposition that an abortive which she had taken was arsenical, exhumed three weeks after burial. Small pieces of small and large intestine, liver, spleen, and scalp with adhering hair were taken for analysis by the physicians who made the autopsy. All of this material was analyzed together, the whole weighing only 70 gm. (two and one-fourth ounces) of which only 23 gm. (three-fourth ounce) were of internal organs, and found to contain arsenic, not in weighable amount, but sufficient to yield three distinct mirrors. From this it was concluded that "the deceased had certainly taken arsenic, but whether in dangerous amount or not was uncertain." A second exhumation was had, at which abdominal and thoracic viscera, muscular tissue and hair were taken from the cadaver, as well as a wreath of artificial flowers which had been upon the head, and some colored silk fabric in which the body had been enclosed. The analysis of the viscera failed to show the presence of more than a trace of arsenic; the hair contained traces of arsenic and copper, but the muscles and skin of both upper and lower extremities were entirely free from arsenic. In the flowers there were notable quantities of arsenic and a little copper, and in the silk much copper and a little arsenic. The opinion of the experts was that it was not a case of poisoning, and that the arsenic found came from the artificial flowers.[2]

The possibility of arsenic in the cadaver being derived from the dye in silk was also considered in the Speichert case.[3]

The question under discussion is one which must arise in every trial for poisoning by arsenic in the United States; and will assume one of two forms, according to the conditions of the case: Either embalming liquids have been applied to the cadaver in some manner, in which case the question is whether the expert is capable of differentiating poison introduced during life from that injected after death; or the evidence as to embalming is negative or questionable, when the expert is called upon to state whether or not the arsenic found was certainly in the body at death and could not have been introduced subsequently. The possibility of obtaining an answer in either case must depend upon the existence of some known difference between the

[1] Maschka: "Gutacht. Prag. med. Fak.," 1867, iii., 262.

[2] Ludwig u. Mauthner: Med.-chir. Centbl., Wien, 1884, xix., 245; Ph. Centralh., 1884, n. F., v., 33.

[3] See Ph. Centralhalle, 1889, n. F., x., 165.

form in which arsenic introduced into the system during life is left there at death, or its *distribution* or *effects*, and the form, distribution, or effects of that introduced after death. The question must be determined by comparison, and that such comparison may not be utterly misleading the things or conditions compared must be known and understood; and we have stated the inability of the expert to answer the question unaided, because, in our belief, the knowledge of the conditions to be compared is not as yet in the possession of the toxicologist.

The FORM OF COMBINATION in which arsenic exists in the body at death if taken during life is not known, nor can we say in what form it will exist in a day or week or a month, whether it be present at death or injected thereafter. Any comparison of *form*, therefore, is an attempt to solve an equation which has no known factors. The supposition that arsenic enters into combination with the living tissue in a different manner and with the formation of different products than when acting upon dead tissue may be, and probably is, an excellent *a priori* hypothesis to serve as the starting-point for a line of investigation. But at the present time it is, so far as we know, a pure hypothesis, without a scintilla of experimental evidence of either its truth or its falsity.

It has been shown by Binz and Schulz[1] that in the living organism, if either an arsenite or an arsenate be injected the other form of oxidation is produced, and that after death a mixture of arsenite and arsenate is found. But the finding of such a mixture does not indicate ante-mortem introduction with certainty. Not only is the commercial sodium arsenate frequently used in embalming, generally contaminated with arsenite, but in a case in which pure arsenate, free from arsenite, was injected after death we have found a mixture of arsenate and arsenite. That post-mortem arsenic may be converted into the risulfid is well known, but the same is true of ante-mortem arsenic. Indeed, in a case of Hofmann's[2] it appears probable that a like conversion occurred during life. As a result of putrefactive changes a portion of arsenic present in the body is converted into volatile products, probably either arsin itself or organic arsins, but this post-mortem change affects ante-mortem

[1] Arch. f. exp. Path. u. Pharm., 1879, xi., 200.

[2] Wien. med. Wochenschr., 1886, xxxvi., 333, 377, 413.

as well as post-mortem arsenic. Concerning organo-metallic compounds of arsenic which may be formed during life we have as yet nothing beyond supposition.

In the methods now used for separating arsenic from organic mixtures, it is identified as elementary arsenic, and in a state which does not permit of the formation of an opinion as to the form in which it existed before separation.[1]

The DISTRIBUTION of arsenic in the body has been depended upon to solve the question, and it is believed by some to have been insufficiently studied not only to serve this end, but to permit of a distinction between acute and chronic poisonings, as well as between different forms in which it may have been taken. The fundamental idea is that when arsenic is introduced during life it is rapidly carried to all parts of the body, and there distributed among the various organs and tissues according to the differing retaining powers of each, and in a different manner after single large doses than after repeated small doses, and with other variations due to differences in the solubility of the poison in different forms; while, on the other hand, arsenic introduced post-mortem will remain at the point of introduction or be only slowly carried therefrom and will not exist in the different organs in the same quantitative proportions as ante-mortem arsenic.

We may distinguish between what may be called *qualitative distribution* and *quantitative distribution*.

The former is based upon the supposition that arsenic introduced after death by any means other than by injection into a vein or artery will remain entirely absent from parts anatomically far removed from the point of introduction. Orfila was the first to study the question experimentally with a human cadaver, and reached the conclusion that poisons would never migrate so far as the parts most distant from the points at which they were applied, at least in quantities sufficient to be detected, if the amount injected into the digestive canal were small.[2] This view was apparently upheld by the results of numerous experiments upon animals, and as late as 1877 Reese wrote that

[1] Some years ago we were informed by a Western toxicologist that he was engaged in experiments looking toward the determination of the behavior of ante- and post-mortem arsenic toward solvents, but we have seen no report of his results.

[2] "Traité des poisons," 5th ed., i., 59.

"it is scarcely conceivable that a poison, introduced into a body after death, could penetrate by imbibition within the cavity of the cranium or spinal column."[1] It was not until 1883 that, after the trial of the case of Peo. *v.* Matthew Millard in Michigan, in which post-mortem imbibition was invoked by the defence, Vaughan and Dawson again resorted to the only proper subject for experiment, the human cadaver, and found that the brain contained arsenic (1,028 gm. yielded 0.00363 gm. As_2O_3) twenty-four days after post-mortem injection into the mouth and rectum. In this experiment a portion of the liquid returned through the nose, and consequently the injection was practically into the head, and direct imbibition through the openings in the base of the skull.[2] In 1888, in connection with the case of Peo. *v.* Ford, the author made three experiments with human cadavers. The injections in two were by the mouth, in one of which the head was severed from the body immediately after the injection. In the third the injection was limited strictly to the stomach and abdominal cavity. Each body was kept fourteen days, after which each brain was found to contain arsenic in sufficient amount to yield quite heavy mirrors. In the experiment with the detached head the brain was found in a good state of preservation, and a portion of brain from the vertex was found to contain quite as much arsenic as another portion from the base. In the third experiment arsenic was found in the muscular tissue of the calf of the leg as well as in the brain.[3] In another experiment with a human cadaver the brain contained 8.28 mgm. of arsenic (As_2O_3) five days after the post-mortem injection of 16.56 gm. in solution into the stomach. In a case reported by Mason,[4] an embalming liquid containing arsenic and zinc had been applied by cloths to the face and chest of the cadaver for many hours, but "no injection of this embalming fluid was practised." After a burial of thirty-five days the body was exhumed and distinct traces of zinc, as well as notable amounts of arsenic, were found in the heart and stomach. From this it would appear that an external application of a solution of zinc (and probably of arsenic) for a

[1] Trans. Col. Phys., Phila., 1877, 3d ser., iii., 33.

[2] Jour. Am. M. Assoc., 1883, i., 115; Med.-leg. J., N. Y., 1889, vii., 345.

[3] Research. Loomis Lab., 1890, i., 38.

[4] Jour. Anal. and Appl. Chem., 1893, xv., No. 6.

period limited to "many hours" is followed by passage of the metallic salt into the interior of the body.

Strassman and Kirsten[1] having experimented with the cadavers of frogs, dogs, and of one human infant, assert that "the presence of poison in the left kidney and its absence in the right indicates the introduction of poison into the stomach of the cadaver. Poison in the two kidneys during the first weeks indicates absorption during life. . . . Arsenic does not penetrate the brain from the stomach within the first four weeks." Of the three propositions here enunciated the first only is admissible, although probably of most exceptional occurrence. The third has been positively disproved. The second is a proposition startling in its novelty. To test its accuracy we injected an arsenical solution into the stomach of an adult human cadaver, and, examining the kidneys after five days found the right to contain 9.37 mgm. of arsenic (As_2O_3) and the left 0.3584 gm.

The attempt to answer the question from the *quantitive distribution* of the poison is much more scientific. Indeed, we believe that comparisons of quantitative distribution will eventually aid materially in solving the questions at issue in many cases, if they do not do so unaided. But we must still maintain that at the present time the knowledge which we possess is entirely insufficient to be safely relied upon. We have already discussed at length (see pages 457–468) such information as is available concerning the distribution of poison taken during life, and have found it to be both meagre and inconsistent with itself. Of the other factor in the problem, the quantitative distribution of arsenic introduced post mortem, we have absolutely no information beyond a few isolated determinations of the quantity in one or another organ. It is clear also that quantitative post-mortem distribution will most probably differ according to the method of introduction, and that it may not be the same when the injection is into an artery as it would be if the poison were injected into a vein, or into the stomach, or the rectum, or the mouth, or imbibed by external application or contact with arsenical objects, or immersed in arsenical ground water. In a recent trial an expert for the people testified that "the arsenic and antimony are especially massed, so to speak, in the alimentary tract, in the centre of the body; the stomach,

[1] Arch. f. path. Anat., 1894, cxxxvi., 127.

the intestines, and the liver containing large amounts of both arsenic and antimony, a fact which to my mind shows conclusively that the arsenic and the antimony could not have entered the body by post-mortem imbibition. In other words, the very fact that the arsenic and antimony are to be found massed—centred, so to speak—in the very centre of the organism shows to my mind that that arsenic and that antimony could not have been absorbed after death, because it seems to me very naturally you would expect to find a larger amount in the external portions of the body."[1] This opinion, which confessedly has no experimental foundation, may be well founded if we consider only imbibition from the exterior, but (equally as an opinion) we fail to imagine how a condition differing from that described could possibly exist if the arsenic had been introduced into the stomach post-mortem, as it very frequently is by undertakers.

The EFFECTS which arsenic is said to produce during life, resulting in a petechial reddening of the gastric mucous membrane and the formation of thrombi in the arterioles and capillaries (see pages 421, 431), are appearances deserving of careful study. So far as we are aware the microscopic lesions due to arsenic have not been studied in the human subject. Should it be shown that these appearances are constantly produced by arsenic and not by disease, the evidence of the pathologist will have much greater weight in the determination of the ante- or post-mortem origin of the poison than that of the chemist.

POISONING BY ARSENICAL GREENS.

Among the arsenical compounds, the arsenical greens, Scheele's green and Schweinfurth or Paris green, are second in forensic interest to the trioxid; and of the two the latter is by far the more important.

Scheele's Green—*Cupric arsenite*—$CuHAsO_3$—is a pale-green powder, made by precipitating a solution of potassium arsenite with one of cupric sulfate. It dissolves readily in potassium hydroxid solution, but is decomposed into a mixture of arsenate, arsenite, cuprous oxid, and cupric oxid when the liquid is heated. It is also soluble in ammonium hydroxid.

[1] Peo. *v.* Meyer, stenographer's minutes, fol. 993.

The commercial Scheele's green is usually a mixture of cupric arsenite and hydroxid. It is only used to a limited extent as a pigment.

Paris Green—*Schweinfurth green, emerald green, mitis green, mineral green*, etc.—is an aceto-arsenite of copper, having, according to Ehrman, the formula $(CuAs_2O_4)_3\ Cu(C_2H_3O_2)_2$—mol. weight.=1011.36, and containing therefore 24.99 per cent. of copper and 44.43 per cent. of arsenic (=58.65 per cent. As_2O_3). It is a bright-green, crystalline powder, insoluble in water, but decomposed by prolonged boiling. It is readily attacked by alkaline solutions, with separation of cupric hydrate, which, on boiling, is converted into cupric oxid, and then reduced to cuprous oxid, the solution then containing an alkaline arsenite. Mineral acids and strong acetic acid decompose it completely, removing all of the copper and liberating arsenious acid.

Paris green is extensively used as a pigment, in the manufacture of artificial flowers, of wall papers, etc., and in the United States, where the name "Paris green" is local,[1] it is probably the most widely known poison for the destruction of potato bugs and other insect pests.

Cupric Arsenate—[$Cu_2(HAsO_4)_2$ or $CuHAsO_4$=31.13 per cent. Cu and 36.91 per cent. As. ?]—is said by Bergeron, Delens, and l'Hôte[2] to exist in the *vert de Mittis* used in France, accompanied by barium sulfate and sodium arsenate.

Poisonings by Paris green are of much more frequent occurrence in the United States than elsewhere, and are chiefly suicidal or accidental.

Homicides by Paris green are also practically limited to this country. We can find reference to but one case in England, which was a conviction for manslaughter by criminal negligence in 1848.[3]

It is true that a perusal of American medical literature does not indicate a greater frequency of criminal poisonings by this agent in this country, as we find reference to only one such case, in which a father was accused of an attempt to poison his

[1] *Vert de Paris*, or *Pariser grün*, is an anilin color.

[2] Ann. d'hyg., etc., 880, 3 s., iii., 23.

[3] Reg. *v.* Franklin and Randall. See Bryant: Prov. M. and S. Jour., 1848, 374; and Taylor: "Med. Jur.," 11th Am. ed., 135.

three-and-a-half-year-old child by Paris green given in water.[1] From other sources, however, we have knowledge of no less than twenty-five cases of homicidal poisonings, or attempts at such, during the years 1879–93, of which thirteen occurred in the State of New York:

In 1879 a young man in Westchester county attempted to poison two brothers by putting Paris green into their tea cups. At Malone in 1880 a young woman was charged with attempting to poison another with repeated small doses of Paris green in coffee. In 1881 Harriet Stone was tried in Essex county for the murder of an infant child with Paris green, and was acquitted. In 1881 a woman in New York city accused her husband with attempting to poison her with Paris green in porter. In 1882 a woman in Dutchess county was accused of having poisoned her husband with Paris green. In 1884 Annie Stewart was indicted in Orange county for assault in the second degree for an attempt to poison by Paris green. In 1885 at Batavia a man attempted to poison himself and family with Paris green in coffee. In New York city in 1885 a coroner's jury found that "Herman Lessman died of Paris green administered by some person other than himself, and that suspicion rests strongly on his widow, Johanna L." (May 18th, 1885). In the same year a woman in Brooklyn was forced to take Paris green and was nearly smothered by her husband. A large quantity of the poison was pumped from her stomach and she recovered. In New York city, in 1888, a young Russian Jewess attempted to destroy a family by sprinkling Paris green upon fish. In New York city in 1890 a man attempted to destroy himself and his wife and children with Paris green in coffee. In 1893 two women in Queens county were accused of attempting to poison their servant with Paris green in coffee. In the same year a man in Richmond county confessed to an attempt to poison another by putting Paris green into his dinner pail.

Of the cases in other parts of the country the following deserve mention with regard to methods of administration: In one a woman attempted to kill her husband by Paris green in a pie; in another the poison was scattered upon the food intended for a party of workmen; in another it was mixed with spices and food articles by a discharged negress previous to her departure; in another it was thrown into a well; in two cases mixed in coffee; in one case in whiskey; in one the administration was forcible, and in one figs were cut open, lined with Paris green and sent by mail to the intended victim.

A homicidal case was the subject of investigation in France in 1878,[2] in which the death of a girl of seventeen years was

[1] The Higbee case. Mayer: Am. Pract. and News, 1891, n. s., xii., 354.

[2] Affaire Gaudot; Bergeron, Delens, and l'Hôte: *Loc. cit.*

caused by *vert de Mittis*, containing cupric and sodium arsenate. Another homicidal case is reported from Germany in 1880, in which a man was tried for poisoning his wife with Schweinfurth green.[1]

Suicides by Paris green are of very exceptional occurrence in Europe, but are very common in the United States. In New York city in the years 1867–1880, of 604 suicides by poison 230 (38 per cent.) were by Paris green.

Accidental poisonings by the arsenical greens have been due to a variety of causes: 1. By the use of the pigment as an insect poison.[2] 2. By its use to color articles of confectionery.[3] 3. From children sucking green paint or articles colored therewith.[4] 4. From wearing articles of dress colored with arsenical greens.[5] 5. By drinking wine, the bottles containing which were sealed with arsenical green sealing-wax.[6] 6. As the results of absorption or inhalation in industries in which the arsenical greens are manufactured or used.[7] 7. As a consequence of inhabiting rooms the walls of which have been papered or painted with materials containing arsenic.

Concerning the last-named source of accidental arsenical

[1] Casper-Liman: "Handb. d. ger. Med.," ii., 433.

[2] Badger: Med. Rec., N. Y., 1878, xiv., 194. Wilson: M. and S. Reptr., Phila., 1878, xxxix., 107. Morrill: Boston M. and S. Jour., 1890, cxxii., 398; all non-fatal.

[3] Fergus: Lancet, 1849, i., 191; Lancet, 1849, i., 219. Bryan: *Loc. cit.* Taylor: Guy's Hosp. Rep., 1850–51, 2 s., vii., 218. Bulley: Med. Times, 1848–49, xix., 507; two deaths.

[4] Metcalfe: Lancet, 1860, ii., 535. Rose: Lancet, 1859, i., 237. Schumacher: Friedreich's Bl. f. ger. Med., 1868, xix., 295. Arnold: M. and S. Reporter, 1878, xxxviii., 339. Emsmann: J. d. prakt. Heilk., 1841, xciii., 134. Grohe u. Mosler: Arch. f. path. Anat., etc., 1865, xxxiv., 208. Niemann: Ztschr. f. Staatsarznk., 1859, lxxviii., 231. Jackson: Boston M. and S. Jour., 1861, lxiv., 121; v. Linprun: Aerztl. Int. Bl., München, 1869, xvi., 81. Traill: Monthly J. M. Sc., 1851, xiii., 1. Siebenhaar: Ztschr. f. Staatsarznk., 1844, xlvii., 188. Schneider: *Ibid.*, 1846, lii., 475; eight deaths.

[5] Image: Practitioner, London, 1880, xxiv., 110 (Case II.). Innhauser: Oest. Ztschr. f. prakt. Heilk., 1871, xvii., 17. Casper-Liman: *Op. cit.*, ii., 415, 417 (two cases). Riedel: Berl. kl. Wochenschr., 1870, vii., 471. Med. Pr. and Circ., London, 1871, xi., 55; no deaths.

[6] Artus: Vrtljschr. f. techn. Chem., 1867.

[7] Farrar: Brit. M. Jour., 1877, i., 8. Roussin: Ann. d'hyg., etc., 1867, 179. Gailletau: Lyon méd., 1869, ii., 543. Ferrand: Union méd., 1872, 3 s., xiv., 797. Gabourg: Union méd. du Canada, 1879, viii., 394. Isambert: Lancet, 1872, ii., 854. Griswold: N. Y. J. M., 1858, 3 s., v., 64. Guy: Arch. of M., London, 1857–59, i., 86. Hassall: Lancet, 1860, ii., 535 (two cases). Coote: Lancet, 1863, ii., 190; four deaths.

poisoning there has been some little discussion. The questions involved are, however, rather of hygienic than of forensic interest. Two propositions may be considered as proven: (1) That arsenical poisoning of a chronic type has been developed by this cause in persons susceptible to the effects of arsenic; and (2) that manufacturers of wall paper have succeeded by strenuous effort in preventing the enactment of such salutary legislation as exists in other civilized countries for the regulation of the materials which they may use. Public health in this as in other matters is secondary to private interest.[1]

The **symptoms** caused by Paris green do not differ from those produced by arsenic trioxid under like conditions, except for the green color of the vomit; and the **post-mortem appearances** are also the same except for the possible presence of Paris green in substance in the stomach in place of crystals of white arsenic.

POISONING BY SULFIDS OF ARSENIC.

Arsenic Trisulfid—*Orpiment, King's yellow*—As_2S_3.—The native trisulfid, ORPIMENT or AURIPIGMENTUM, was known to the ancients and is described by Dioscorides, who refers to its depilatory power. It is to this substance that the name ἀρσενικόν was given, and the red sulfid, REALGAR, was also known, under the name σανδαράχη. It is a bright golden-yellow mineral. The artificial trisulfid is a yellow powder, manufactured for use as a pigment, KING'S YELLOW. Owing to the presence of arsenic trioxid in the artificial powder it is much more actively poisonous than the natural. This substance constitutes the yellow precipitate produced by hydrogen sulfid in acid solutions of the arsenious compounds. It is almost insoluble in cold water, but sufficiently soluble in hot water to communicate to it a distinct yellow color. By continued boiling with water it is to some extent decomposed into hydrogen sulfid and arsenious acid. It is insoluble in dilute hydrochloric acid, but readily soluble in the alkaline hydroxids, carbonates, and sulfids.

[1] The question will be found completely stated in Senate Doc. No. 215, 1891, Massachusetts, and in a paper by Prof. E. S. Wood in 5th Rept. Mass. State Board of Health, etc., suppl., 1884, 213–267. Cases subsequent to 1884 are reported by Swift: Boston M. and S. Jour., 1891, cxxiv., 185, and Freer: British M. Jour., 1885, i., 1246.

Hence, although not absorbed from the stomach, it may be from the intestine. It volatilizes when heated. Oxidizing agents convert it into arsenic and sulfuric acids.

Arsenic trisulfid enters into the composition of a number of depilatory powders: *Pento-epilatoire* is an eight-per-cent. solution of As_2S_3 in a solution of sodium sulfite; *Poudre pilivore de Laforest* contains 60 parts mercury, 30 As_2S_3, 30 litharge, and 30 starch; *rhusma* is a mixture of 2 to 3 parts As_2S_3 with 15 parts quicklime; *Ergen's depilatory powder* gave 19.67 per cent. As_2S_3 and 74.2 per cent. calcium hydroxid. It appears to contain realgar (As_2S_2), arsenic trisulfid, and calcium arsenite or thioarsenite. A mixture similar to rhusma was formerly used by tanners for a like purpose.

Poisonings by King's yellow are of rare occurrence. They are sometimes the result of a combination of fraud and accident, the pigment being mistaken for some other yellow powder, such as turmeric, used to adulterate food articles. In the Clifton case, noted by Taylor,[1] six persons were severely poisoned by eating "Bath buns" into which King's yellow had been put in mistake for chrome yellow. Jochner[2] reports the poisoning of an old man of seventy years and his nephew by orpiment mixed with porridge in mistake for turmeric. The former died in nineteen and a half hours. Other cases in which it has been put into food by mistake are reported by Devergie,[3] Paterson,[4] and Crawford.[5] Three instances are upon record of women who have been killed by the application of arsenic trisulfid by quacks to tumors of the breast.[6] Four early cases of homicidal poisoning by arsenic trisulfid have occurred: Christison[7] refers to a woman having been tried in Glasgow in 1822 for the murder of a child by King's yellow, and to another instance of attempted homicide by the same poison mixed with tea. Chevallier[8] reports the poisoning of a woman of twenty-six years by her husband. The case of Reg. *v.* Burdock, in which a woman was convicted of the murder of another by King's yellow, which

[1] "Poisons," 3d Am. ed., 346.
[2] Ztschr. f. d. Staatsarznk., 1851, 43 Erghft., 162.
[3] "Méd. lég.," 3ème ed., iii., 519. In mistake for brown sugar.
[4] Monthly J. M. Sc., 1846, vi., 184.
[5] Glasgow M. Jour., 1856–57, iv., 49.
[6] Rousset and Lepelletier: Ann. d'hyg., 134, xi., 459. Manouvriez: Gaz. d. hôp., 1881, liv., 331. Chabenat and Leprince: Ann. d'hyg., 1890, 3 s., xxiv., 360.
[7] "Poisons," Am. ed., 226.
[8] Ann. d'hyg., 1849, xlii., 448.

was found on analysis to contain seventy-nine per cent. of arsenic trioxid, is reported by Symonds.[1] A more recent case is that reported by Krafft-Ebing[2] of an insane man who attempted to poison a woman with "yellow arsenic" in coffee.[3]

Arsenic exists in nature as sulfids combined with those of iron and cobalt. These minerals are sometimes used in the powdered form as fly poisons, and have caused accidental poisoning in the human subject. Thus Schobbens[4] has reported two cases, one fatal, by a fly poison consisting of cobalt, iron, arsenic and sulfur, from the mines of Tunaberg; and Berti[5] the fatal poisoning of a woman by a native double sulfid of arsenic and cobalt.

The symptoms, treatment, and post-mortem appearances are the same as with white arsenic, except for the yellow color of the trisulfid in the vomit and stomach contents. In exhumed bodies arsenic trisulfid may have been produced from the trioxid.

POISONING BY ARSENIC ACID AND ARSENATES.

Poisonings caused by arsenic acid (H_3AsO_4) or the arsenates taken internally are of very exceptional occurrence. Indeed we find but one case reported, in which a child of three to four years died in nine hours, after swallowing an unknown quantity of a "pest poison," found upon analysis to be a solution of sodium arsenate. At first there were thirst and nausea, but no vomiting in spite of the administration of emetics, nor was there pain or any sign of gastric trouble except during the first few minutes. The child became deeply comatose, with widely dilated pupils, and died from paralysis of the respiratory centres.[6] A German case is reported[7] of an unsuccessful attempt by a man to poison his wife with a native arsenate of iron ("Kuttenberger Erde"=Eisensinter=Pittizite?), used as a fly poison, administered in coffee.

[1] Tr. Prov. M. and S. Assoc., 1835, iii., 465. See also Beck: "Med. Jur.," 12th ed., ii., 602.

[2] Friedreich's Bl. f. ger. Med., 1882, xxxiii., 319.

[3] See also Fortier: Gaz. m. d'Algérie, 1858, iii., 66. Harvey: Ind. M. Gaz., Calcutta, 1879, xiv., 142. Chowdry: *Ibid.*, 1881, xvi., 282. Landerer: Arch. d. Pharm., 1882, 3 R., xx., 56. Majer: Med. Corr.-Bl. Würt. A.-Ver., 1849, xix., 55.

[4] Ann. Soc. d. m. d'Anvers, 1846, 224, 229.

[5] Gaz. med. it. prov. Venet., 1860, iii., 90.

[6] Silliman: Med.-leg. Jour., N. Y., 1883–84, i., 473.

[7] Friedreich's Bl. f. ger. M., 1862, xiii., 1 Hft., 78.

Barnett[1] has reported a number of poisonings caused by the use of a mixture of arsenic and nitric acids applied with a swab several times daily, by a "cancer doctor." Of these victims a woman of forty-seven years, another of sixty years, and a man of thirty-six years died.

A few cases of non-fatal arsenate poisoning have been caused by contact with arsenical anilin dyes or with fabrics dyed with them (red-stocking poisoning).[2]

ANALYTICAL.

The systematic process for the separation of arsenic from organic mixtures and from other poisons has already been described in the section of general toxicology and under antimony. It remains only to describe certain methods of separation adapted to arsenic alone, the tests by which it is identified, and the methods for quantitative determination.

The method of Schneider and Fyfe,[3] modified by **Beckurts,**[4] depends upon the volatility of arsenic trichlorid and its separation by distillation in the presence of ferrous chlorid from the less volatile chlorids, including those of antimony and tin. The materials, hashed if solid, or concentrated after neutralization with disodic carbonate if liquid, are placed in a capacious retort with sufficient pure hydrochloric acid to make a thin paste and 20 c.c. of a cold saturated solution of ferrous chlorid. The beak of the retort is directed upward and connected by an obtuse-angled tube with a Liebig's condenser. The contents of the retort are cautiously and gradually heated until about two-thirds of the acid have distilled over at the rate of not more than 3 c.c. a minute. If the material contain a large quantity of arsenic the distillation must be repeated two or three times with fresh quantities of hydrochloric acid; and if antimony or tin be present the first half of each distillate

[1] Tr. M. Soc., N. Y., 1858, 225.

[2] Meeres: Lancet, 1869, ii., 190. Whalley: M. Times and Gaz., 1866, ii., 222. Cheyne: Brit. M. J., 1874, ii., 643. Hofmann u. Ludwig: Med. Jahrb., Wien, 1877, 501.

[3] Arch. d. Pharm., 1851, 2 R., lxx., 40; J. f. pract. Chem., 1852, lv., 103.

[4] Arch. d. Pharm., 1884, ccxxii., 653. See also Fischer: Berichte, Berlin, 1880, xiii., 1778. Ambühl: Schweiz. Wochenschr. f. Pharm., 1892, 49. Duflos and Hirsch ("Das Arsenik," Breslau, 1842, p. 37) had previously suggested a similar process, using hydrochloric acid alone.

should be redistilled from about 5 c.c. of ferrous chlorid solution.

Method of Fresenius and v. Babo.[1]—The earlier stages of this method have been described, and are the best adapted to the systematic search for all mineral poisons. The continuation of the method after the Mayer fusion[2] consists in recovering the arsenic as arsenic trisulfid from the solution by reduction with sulfur dioxid and precipitation with hydrogen sulfid. The thoroughly dried arsenic trisulfid is then mixed with dry potassium cyanid and disodic carbonate, and the mixture placed in a porcelain boat, which is introduced into a Bohemian tube drawn out as shown in Fig. 12. This tube is attached to a carbon dioxid generator and a slow current of the gas passed

Fig. 12.—Reduction Tube, Fresenius and v. Babo Apparatus.

until the air is completely displaced. The mixture in the porcelain boat is then gradually heated, when the trisulfid is reduced, and elementary arsenic is deposited in the cool part of the tube beyond. The mixture of sulfid and cyanid and carbonate should not be placed in contact with the glass, as, if the glass contain arsenic, as it frequently does,[3] this may be reduced.

We prefer to depart from the method laid down by Fresenius and von Babo at the termination of the Mayer fusion. The cooled residue after the action of sulfuric acid (see page 159) is dissolved in sulfuric acid No. 3 (see page (499, cooled if necessary, and the solution (or an aliquot part of it if the bulk of the hydrogen sulfid precipitate has indicated a large quantity of arsenic) introduced into the Marsh apparatus (see page 498).

When urine or other substances containing but a small amount of organic matter are to be tested for arsenic only, the process may be much abbreviated and the extraction more completely effected by adding concentrated sulfuric acid containing about one-thirtieth of its volume of nitric acid, evaporating to dryness, heating the residue until dense white fumes are given

[1] Fresenius: "Qual. Anal.," 16te Aufl., 503.

[2] See pp. 153, 157, 316.

[3] See Fresenius: Ztch. f. anal. Chem., 1883, xxii., 397.

off, extracting the char with boiling water, concentrating the solution, and introducing directly into the Marsh apparatus.

Tests for Arsenic—1. Hydrogen Sulfid.—Colors neutral solutions of arsenious acid or of arsenites yellow. If the solution be acid a yellow precipitate of arsenic trisulfid is formed immediately; which is readily soluble in solutions of alkaline hydroxids, carbonates, or sulfids, and in nitric acid, but almost insoluble in hydrochloric acid. If the arsenic be present as arsenate hydrogen sulfid produces no precipitate in the cold except after long standing, when there is separation of arsenic pentasulfid and sulfur, and partial reduction of arsenic to arsenious compound, followed by precipitation of arsenic trisulfid, the action continuing until finally all of the arsenic is separated as trisulfid. If hydrogen sulfid be caused to act upon a solution of an arsenate at about 70° (158° F.) a precipitate of arsenic trisulfid is formed if there be a large excess of hydrogen sulfid; otherwise a mixture of arsenic trisulfid and pentasulfid and sulfur separates.

2. Reinsch Test.—If a solution containing an arsenite and strongly acidulated with hydrochloric acid, be boiled in the presence of metallic copper, a gray stain is formed upon the copper, which increases in thickness and finally peels off if the quantity of arsenic be large. The gray deposit is an alloy of copper and arsenic. The deposit is not formed in the presence of powerful oxidizing agents such as the chlorates. With an arsenate it is only slowly formed. A stain having an appearance similar to that caused by arsenic is also formed if the liquid contain compounds of sulfur, gold, platinum, palladium, silver, bismuth, antimony, or mercury. To distinguish the arsenical stain from the others the strip of thin copper foil, which should be about one-eighth of an inch wide and three-quarters of an inch long, is removed from the solution, gently washed, and dried by contact with filter paper. It is then placed near one end of a clean piece of thin Bohemian tubing, open at both ends and about eight inches long. This is held at an angle of about ten degrees to the horizontal and gently warmed along its entire length until the interior of the tube and the foil are perfectly dry. The tube is then held in the position shown in Fig. 13, and the copper heated while the inclination of the tube may be varied, and the forefinger more or less applied

to the upper opening in such a manner as to govern the current of air through the tube, which should be quite slow. Or the copper may be heated in a closed reduction tube, but in that case the copper and tube must be completely dried before being heated. We prefer the open tube because it is more readily cleaned and dried, and because the slight current of air carries the sublimate well away from the copper; there is, however, danger of loss if the air current be too rapid. Of the compounds mentioned arsenic, antimony, and mercury are the only ones

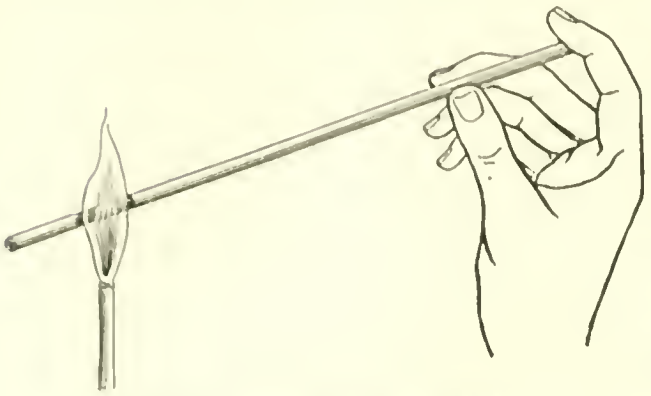

FIG. 13.—Reinsch Test, second stage.

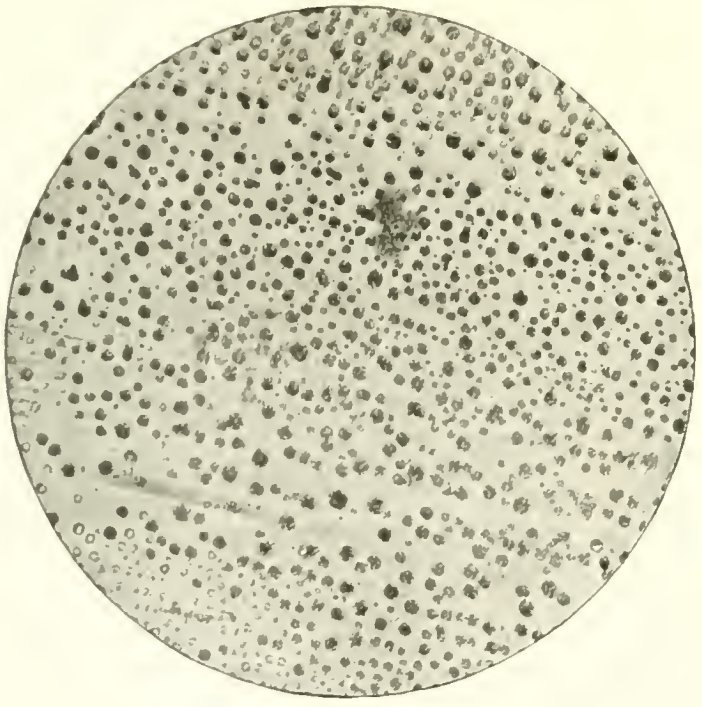

FIG. 14.—Reinsch Test. Mercury Globules. × 75.

which produce a sublimate in the tube. Sulfur is volatilized as sulfur dioxid, and the metals remain upon the copper either in the metallic form or as non-volatile oxids. The sublimate produced by mercury is grayish rather than pure white, and when

examined with the microscope is found to consist of an aggregation of shining globules of metallic mercury (Fig. 14). The arsenical and antimonial deposits are both white and more closely resemble each other, but differ in certain particulars: The antimonial deposit is nearer to the point at which heat was applied than the arsenical, and a portion of it may be in that part of the tube which was in the flame. After the formation of the sublimate it may be readily driven along the tube by a moderate heating if it be arsenic, while a much higher temper-

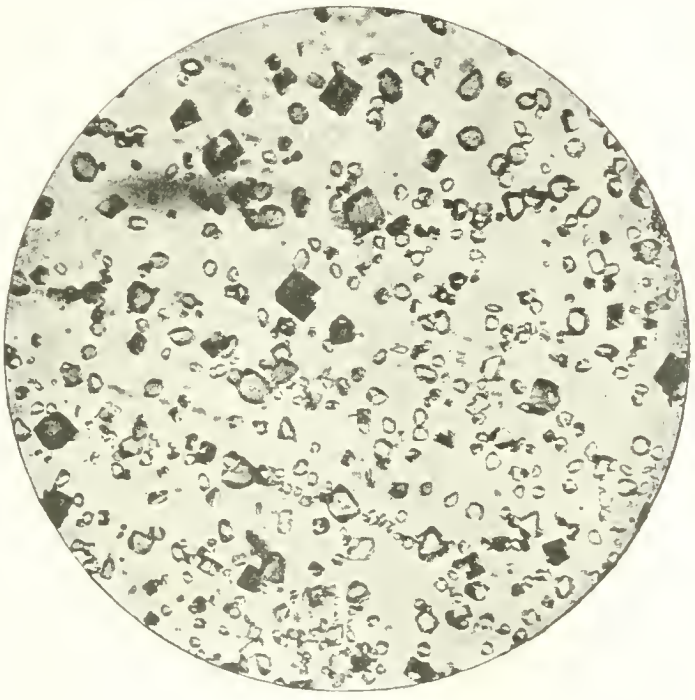

FIG. 15.—Reinsch Test. Arsenic. Octahedral Crystals. × 75.

ature is required to volatilize the antimonial deposit. The arsenical deposit consists entirely of brilliant octahedral crystals (Fig. 15), varying in size, the larger being in the portion of the sublimate nearest to where the heat was applied. The crystals are bright, with finely defined edges, and scintillate when the tube is rotated on its axis in the sunlight. The antimonial deposit is generally entirely amorphous (Fig. 16). It may, however, contain crystals, some of which may be octahedral, of the same shape as the arsenical crystals, but rather duller in lustre and less transparent, and whose edges appear as broader black lines (Fig. 17). These crystals, if present at all, are always few in number and surrounded by much granu-

lar, amorphous material, and require a high temperature for their volatilization. Occasionally prismatic crystals are also formed, either beyond the copper or in that part of the tube which was in the flame (Fig. 18). When arsenic trioxid is sublimed in air in the manner above directed it does not form prisms, as it does occasionally when heated under pressure or in sulfur dioxid (see Fig. 9, page 344), but sometimes flattened derivatives of the octahedron, when attached to the glass by their edges, have, with a certain focussing, a prismatic appearance;

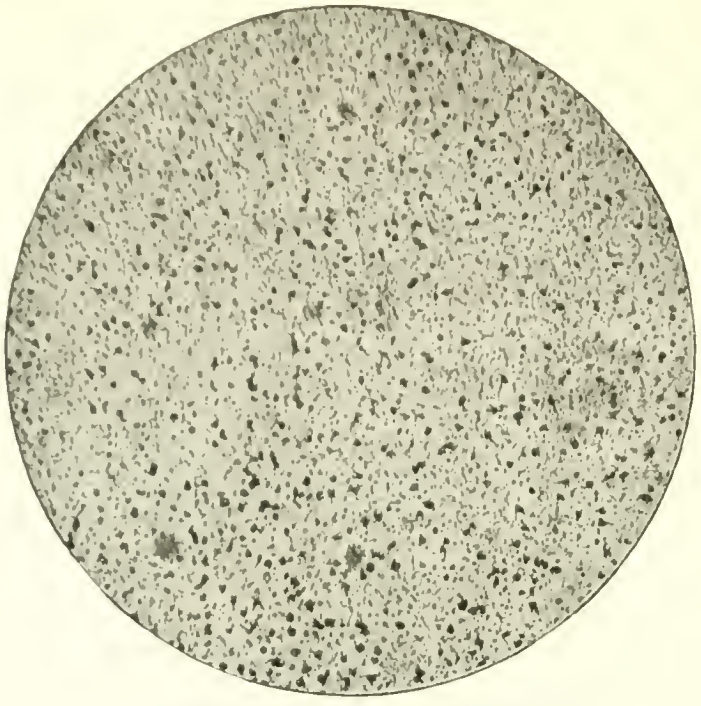

FIG. 16.—Reinsch Test. Antimony. Amorphous Deposit. × 75.

the apparent ends are, however, truncated, and on changing the focus the larger surfaces of the crystal come into view.

The deposit may be heated with a very small quantity of water and tested for arsenic and antimony with hydrogen sulfid, or ammonio-silver nitrate. The larger crystals, whether of arsenic or antimony trioxid, are visible with an ordinary hand magnifier of four diameters; the smaller require a magnifying power of about seventy-five diameters to be distinctly seen.

The Reinsch test possesses two advantages as a preliminary test when abundance of material is available, and for clinical purposes during the life of the patient. It is a test not only for arsenic, but for mercury and antimony as well. It may be

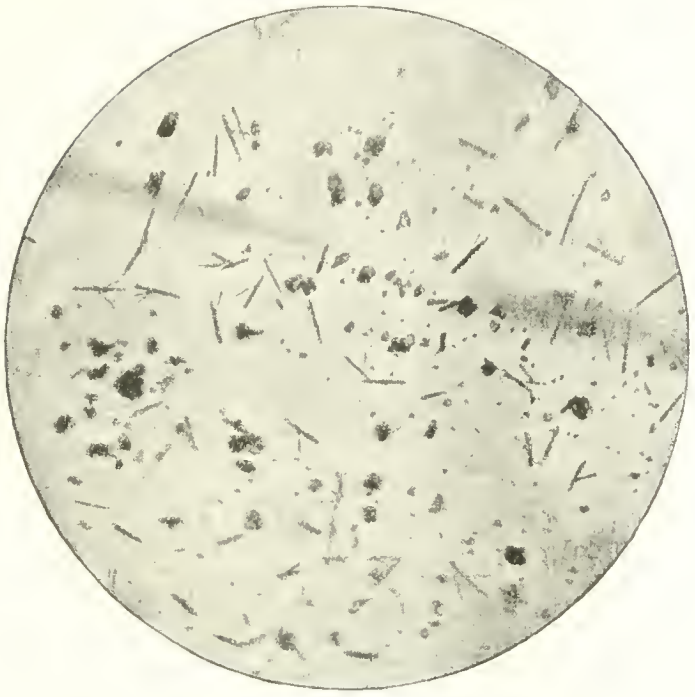

FIG. 17.—Reinsch Test. Antimony Deposit; Amorphous, Octahedra and Prisms. × 75.

FIG. 18.—Reinsch Test. Antimony, Amorphous and Prismatic Deposit with a few Octahedra. × 75.

applied directly to a liquid containing organic matter, such as the urine, and may be completed in a few minutes, while other methods require a tedious preliminary treatment for the removal of organic matter. Two points are, however, to be borne in mind in its use: Hydrochloric acid, even the chemically pure of the pharmacies, is rarely free from arsenic, and copper foil may also contain it. The method should, therefore, never be used without a blank testing, using as much acid and copper, and continuing the boiling for as long a period as in the actual testing. If the chemicals be pure the copper is, if anything, brightened. Should it becomed dimmed in the slightest degree the acid, which is usually at fault, must be rejected. Copper foil of a sufficient degree of purity is purchasable, but it is best prepared by electrolysis of a solution of pure cupric sulfate, using a plate of pure copper as an anode. Another objection to this process is that in its application copper is introduced into the articles under examination. It should, therefore, never be used except with a small sample of the available material.

The practical limit of delicacy of this test is about 0.0001 grain, 0.0065 mgm.; although Wormley[1] claims for it a delicacy 0.00001 grain, 0.00065 mgm. It is certainly inferior in delicacy to the Marsh test as now used.

3. **Marsh Test.**[2]—Of this, the most delicate and reliable of the tests for arsenic, nothing of the original process as described by Marsh remains in the present method of application except the fundamental principles, the generation of hydrogen arsenid and its decomposition by heat, upon which it is based.

The form of apparatus which we have found most convenient is a modification of that recommended by Chittenden and Donaldson,[3] whose method of manipulation we also recommend in the main. The apparatus (Fig. 19) consists of a generator, *a*, made from a Drexel wash bottle of about 250 c.c. capacity by cutting off the entering tube and fusing a blown separatory funnel upon it, and drawing out and bending up the lower end of the tube. The delivery tube of the generator communicates with a chlorid of calcium tube, *b*, which in turn communicates

[1] "Micro-Chemistry of Poisons," 2d ed., 274.

[2] London M. Gaz., 1836, xviii., 650.

[3] Amer. Chem. Jour., 1880–81, ii., 238.

with a tube of Bohemian glass, *cc*, about 5 mm. internal diameter and 50 cm. long, drawn out somewhat at one point and supported in a gas furnace capable of heating about 20 cm. of its length, that portion of the tube in the furnace being enclosed in a tube of wire gauze. About 20 cm. beyond the furnace the Bohemian tube is connected with a tube bent at right angles, whose longer limb is bent downward and dips about 2 cm. into a solution of silver nitrate (1:2) in the foot test-tube *d*. The chemicals required are: Granulated platinized zinc, made by adding a small quantity of platinum to arsenic-free zinc in fusion, and after stirring in and complete fusion pouring the

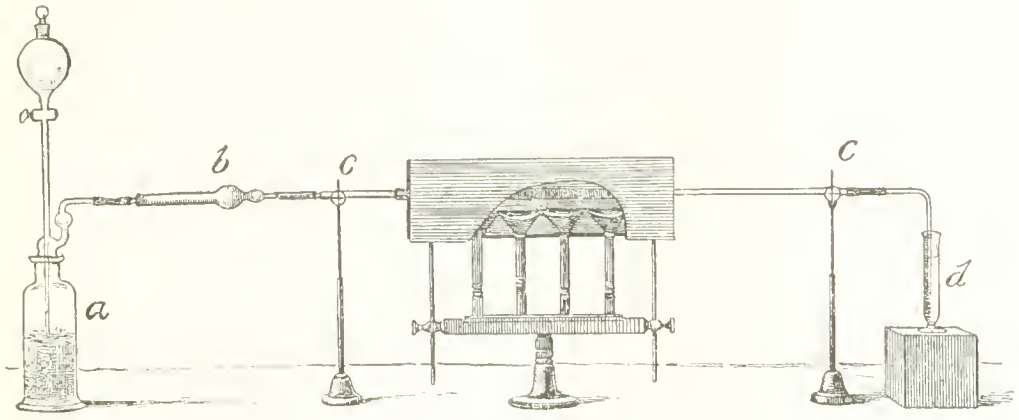

FIG. 19.—Marsh Apparatus.

alloy into water. Sulfuric acid of four degrees of concentration: No. 1, 545 c.c. pure concentrated sulfuric acid and 5,000 c.c. water; No. 2, 109 c.c. concentrated sulfuric acid and 1,640 c.c. acid No. 1; No. 3, 218 c.c. concentrated sulfuric acid and 1,640 c.c. acid No. 1; and No. 4, 530 c.c. concentrated sulfuric acid and 1,248 c.c. water.[1]

To conduct a testing, the tube *b* is charged with granulated calcium chlorid, enclosed between plugs of glass wool;[2] 25–35 gm. of the platinized zinc are placed in the generator, and the apparatus is connected, a suitable quantity of silver chlorid solution being placed in the test tube *d*, and the generator being set into a large beaker or other vessel filled with cold water,

[1] The acids must have become cold when they are used.

[2] It has been supposed that acid from the generator might liberate chlorin by contact with the calcium chlorid. We have never seen this to occur in the form of apparatus and with the manipulation described.

which is renewed from time to time during the operation. The integrity of the joints is tested by gently aspirating at the mouth of the separatory funnel until the silver nitrate solution has risen to near the bend of the tube and closing the stopcock; the level attained should remain constant for ten minutes. A small quantity of acid No. 2 is now put into the separatory funnel and allowed to flow upon the zinc, at first quickly, so as to fill the tube completely with the liquid, and afterward more slowly, until the apparatus is filled with hydrogen, for which fifteen to twenty minutes should be allowed. The gas furnace is then lit and the evolution of hydrogen continued for half an hour by the use of acid No. 2, at the end of which time the tube beyond its emergence from the furnace must be perfectly free from any stain or deposit.[1] The liquid supposed to contain arsenic, prepared by the methods previously described (see pages 153 *et seq.*, 316, 492), and having been mixed with about 40 c.c. of acid No. 3 and cooled, is poured into the separating funnel, whose stopcock is so adjusted that the bubbles of escaping gas pass through the silver solution at the rate of about one a second. When the liquid in the separatory funnel begins to run low, but before it has entirely run out, 40 c.c. of acid No. 3 are added, and after that 45 c.c. of acid No. 4. The entire duration of the testing is usually about four hours.

If arsenic be present in the liquid introduced into the generator a stain is produced in the tube beyond the furnace, which increases in density more or less rapidly, according to the quantity present, and forms a "mirror," which is usually double, the portion nearest to the heat being brown and rather thin, that beyond dark slate gray, almost black, and lustrous. With larger quantities the film increases in thickness so as to peel off. If the quantity of arsenic introduced be not very large it will be entirely contained in the deposit in the tube, and the silver nitrate solution, although somewhat darkened, will have formed no black deposit.

If in the treatment by hydrogen sulfid (see page 157) a considerable quantity of yellow precipitate was formed the final

[1] It is presumed that both acid and zinc have been previously tested by a blank testing, using the same quantities of acid and zinc as are to be used in the entire testing, and continued for the same period, and which have been found to be free from arsenic. This repeated testing of the chemicals is merely an additional precaution.

solution should not all be used in a single Marsh testing. The liquid should be divided into at least two parts bearing a known relation to the whole. One of these is to be used for a quantitative determination by the Marsh (see page 508). Another portion is worked in another Marsh until a mirror of sufficient magnitude is obtained, when the stopcock is closed and the apparatus allowed to stand until the escape of gas has nearly ceased. The furnace is then extinguished, the tube *c* removed, and another substituted; the stopcock is then again opened as before and the furnace again lighted after a few minutes, during which the silver solution is observed to turn noticeably darker and to deposit a black sediment. With a sufficient supply of material several mirrors may be thus obtained, which may be either retained as exhibits or used for further verification tests (see below). The silver solution may also be tested for arsenite by floating upon it dilute ammonium hydroxid solution, which in the presence of arsenic causes a yellow cloud of silver arsenite.

The limit of delicacy of this test is placed by Chittenden and Donaldson at 0.001 mgm. (=about $\frac{1}{70000}$ grain), and by Wormley at $\frac{1}{500000}$ grain (=0.0013 mgm.).

Modifications of the Test.—One of these consists of a reversion to the original Marsh process: In place of turning the tube through which the gas finally escapes downward, it is turned upward and drawn out to a fine point, at which the gas is ignited while the furnace is extinguished. If now a cold surface, such as a piece of porcelain, be held *in* the flame, a brown stain of elementary arsenic is formed; while, if a cold surface, such as a plate of glass or a section of glass tubing, be held *above* the flame a deposit of white, glistening octahedral crystals of arsenic trioxid is formed upon it. Necessarily this modification is attended with considerable loss of arsenic.

"*Morton's Method*,"[1] commonly called *Bloxam's*,[2] is the same as that of Marsh except that nascent hydrogen is produced by the electrolysis of dilute sulfuric acid by the battery, instead of by the action of zinc upon sulfuric acid; the object being to dispense with the use of zinc and to diminish the amount of sulfuric acid used, both of which were formerly obtained arsenic-free only with great difficulty. The most recent form of appa-

[1] Lancet, 1840–41, ii., 394.

[2] Quart. Jour. Chem. Soc., 1861, xiii., 12, 338.

ratus used in this modification is that suggested by Wolff[1] and shown in Fig. 20, in which the liquid to be tested is introduced through *d*, while the gases generated may be directed at will through the silver nitrate solution in *s* or though the heated reduction tube *k*. A battery of six Bunsen cells is used.

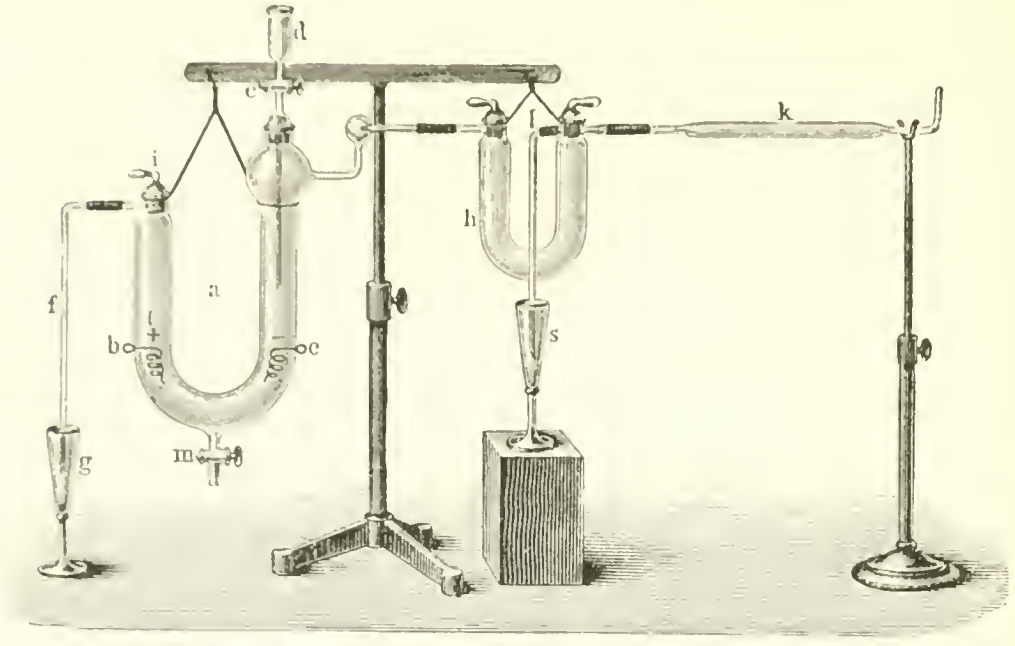

Fig. 20.—Marsh Apparatus. Morton-Wolff Modification.

In *Klobukow's* modification[2] the reduction tube *k* of Wolff's apparatus, or the heated tube in the ordinary Marsh is replaced by an arrangement such as is shown in Fig. 21, in which the hydrogen arsenid is decomposed by heat, not externally applied,

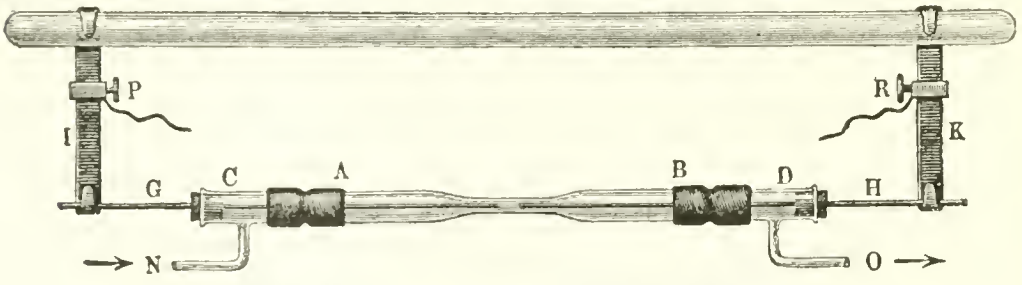

Fig. 21.—Marsh Apparatus. Klobukow's Modification.

but produced by the passage of the discharge of an induction coil between the platinum wires G and H while the gas passes in at N and out at O. The advantage claimed is that the arsenic

[1] Ztschr. f. an. Chem., 1888, xxvii., 125.

[2] Ztschr. f. an. Chem., 1890, xxix., 129.

is all deposited in the short space between the terminals in the constricted part of the tube.

Combination of Marsh and Hager's[1] *Methods.*—In this the reduction tube (*c*, Fig. 19) in place of being all of one piece is divided into two parts. One of them (*b*, Fig. 22) is charged with solid silver nitrate between plugs of glass wool. The silver nitrate turns yellow when *a*, which serves the ordinary purpose of the reduction tube in the Marsh, is not heated.

FIG. 22.—Marsh Apparatus. Hager's Modification

DISTINCTION BETWEEN ARSENICAL AND ANTIMONIAL DEPOSITS IN THE MARSH.—The earliest and only important objection raised against the Marsh test is that if arsenic be absent and antimony present appearances at first identical are produced: mirrors in the reduction tube, stains upon porcelain, and a black deposit in the silver solution. The two substances may be distinguished with absolute certainty: First, by operating in such manner that arsenic and antimony are separated during the preparatory process, as by the Meyer fusion (see page 316) or by Beckurt's modification of the Schneider and Fyfe method (see page 491).

Second, by noting certain differences in the appearances and products of the Marsh.

The arsenical stain is always beyond that part of the reduction tube which was heated.[2] Antimony forms two stains, one in front of the heated part and one beyond it. The external antimonial stain is nearer to the heated part of the tube than the arsenical.

The arsenical mirror, if slight, is brown in color; if greater in quantity it is usually double, the denser part being almost black with a rather dull metallic lustre. The antimonial mirror

[1] See below, and Klein: Arch. d. Pharm., 1889, ccxxvii., 913.

[2] It is assumed that the form of apparatus and method of procedure described are adopted. If curves are made in the reduction tube, back currents are produced and, with a slow evolution of gas, arsenic may deposit in a manner similar to antimony, or if the tube be constricted at the point at which the mirror is deposited arsenic may produce an anterior mirror if the evolution of gas be too rapid.

is usually single (each one) and of a brighter, more silvery appearance.

If heated in a slow current of hydrogen the arsenical mirror is driven along the tube with the application of a slight degree of heat, and without fusion; and the escaping gas has the odor of garlic.[1] The antimonial mirror requires a much higher degree of heat for its volatilization, and is redeposited nearer to the heated point, and fuses before volatilizing. The escaping gas is odorless.

If the arsenical mirror be gently heated in a slow current of air, it disappears and is replaced further along the tube by a white deposit consisting entirely of octahedral crystals of arsenic trioxid; under similar treatment antimony, with a higher degree of heat, is converted into the amorphous or crystalline antimony trioxid or tetroxid. (See Reinsch, p. 495.)

If the arsenical mirror be cautiously heated in a slow current of dry hydrogen sulfid it is converted into the yellow arsenic trisulfid. Under like conditions the antimonial mirror is converted into antimony trisulfid, which may be orange or black.

Arsenical mirrors or stains disappear by solution when moistened with sodium hypochlorite solution.[2] Antimonial stains or mirrors remain unaltered.

Both arsenical and antimonial stains or mirrors dissolve in nitric acid (sp. gr. = 1.3), and on evaporation of either solution a white residue remains. If this be now moistened with ammonio-silver nitrate it turns red-brown with arsenic, but with antimony remains colorless in the cold, and turns black when heated.

Both arsenical and antimonial stains and mirrors dissolve in ammonium sulfid solution, the latter somewhat more rapidly than the former. On evaporation of this solution arsenic leaves yellow arsenic trisulfid, soluble in ammonium hydroxid, insoluble in hydrochloric acid; while antimony leaves the orange antimony trisulfid, soluble in hydrochloric acid, but insoluble in ammonium hydroxid.

If an arsenical stain be exposed to vapor of iodin it is gradually converted into the yellow-brown arsenic triiodid, whose

[1] The odor should be very cautiously tested.

[2] Made when required by rubbing up chlorid of lime with soda solution and filtering the faintly alkaline liquid.

color is discharged when it is breathed upon, and if then the residue be moistened with strong hydrogen sulfid solution it turns yellow. Under like conditions the red-brown antimony triiodid is produced, whose color is not discharged when it is breathed upon, and is converted into the orange antimony trisulfid by hydrogen sulfid.

Although the alteration in appearance of the silver solution is the same whether hydrogen arsenid or antimonid has been passed through it, the reaction has been different: all of the arsenic remains in the solution as silver arsenite, while all of the antimony passes into the black deposit as silver antimonid (see page 320).

4. Hager's Method[1] in the various forms suggested by Hager, Gutzeit, and Flückiger depends upon the action of hydrogen arsenid upon silver nitrate. The object, suitably prepared if necessary, is placed in a test tube or flask with hydrochloric acid and zinc, a loose plug of cotton is placed above, and finally a cork having two slits into which are introduced slips of parchment paper, one impregnated with silver nitrate, the other with lead acetate. In the presence of arsenic the latter should remain colorless while the former turns yellow with dark-brown borders, and black when moistened. This method, while serviceable as a rapid preliminary test for chemicals and papers or fabrics, is not preferable to the Reinsch method for like uses.

5. Bettendorff's Reaction.[2]—A few drops of a solution of stannous chlorid are added to 5 c.c. of fuming hydrochloric acid (sp. gr. at least 1.123), and a few drops of the suspected liquid are added. If an arsenious or arsenic compound be present a brown-black precipitate is formed, slowly in the cold, rapidly when heat is applied. Bettendorff claims for this test a delicacy of 1 mgm. in a dilution of 500,000. Antimony is not similarly reduced.

6. Schlikum's Reaction.[3]—From 0.3 to 0.4 gm. of stannous chlorid is dissolved in 3 to 4 gm. of pure hydrochloric acid and a very minute fragment (0.01 to 0.02 gm.) of sodium sulfite added. The liquid to be examined is floated upon this test liquor and the test tube placed in warm water. In the presence

[1] Pharm. Centralh., 1872, xiii., 145.

[2] Ztschr. f. Chem., 1869, 2 R., v., 492.

[3] Pharm. Ztg., 1885, xxx., 465.

of arsenic a yellow band (of arsenic trisulfid) is formed at the junction of the layers.

7. Ammonio-Silver Nitrate[1] produces in neutral solutions of arsenites or in aqueous solutions of arsenious acid a pale-yellow precipitate of silver arsenite, soluble in acids and alkalies, and not formed in their presence. The same reagent under like conditions produces a brown-red precipitate of silver arsenate in solutions of arsenates.

8. Ammonio-Cupric Sulfate[2] produces in solutions of arsenious acid or of arsenites a green, amorphous precipitate of cupric arsenite; which is readily soluble in ammonium hydroxid or in free acids.

9. The reduction test is only applicable to dry powders in the absence of organic matter. The solid is placed at the

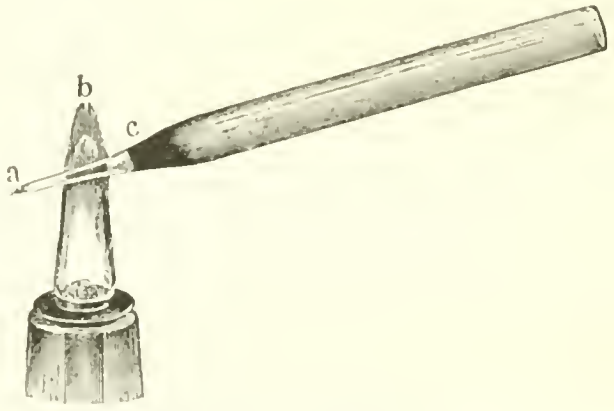

Fig. 23.—Reduction Test for Arsenic.

point *a* of the closed reduction tube (Fig. 23), and above it at *b* a splinter of recently ignited charcoal; *b* is first heated to redness, then *a*; the vapor of arsenic trioxid, in passing over the hot charcoal, is reduced, and elementary arsenic is deposited at *c* as a "mirror." The tube may then be cut between *a* and *c*, the larger piece, held with its wider end slightly elevated, and the mirror gently heated in the flame, when the deposit is volatilized, the odor of garlic is observed, and bright octahedral crystals are deposited in the tube above.

[1] Prepared by cautiously adding dilute ammonium hydroxid to a solution of silver nitrate until a very small quantity of the precipitate which forms at first remains undissolved.

[2] Prepared with cupric sulfate solution and ammonium hydroxid in the same manner as No. 7.

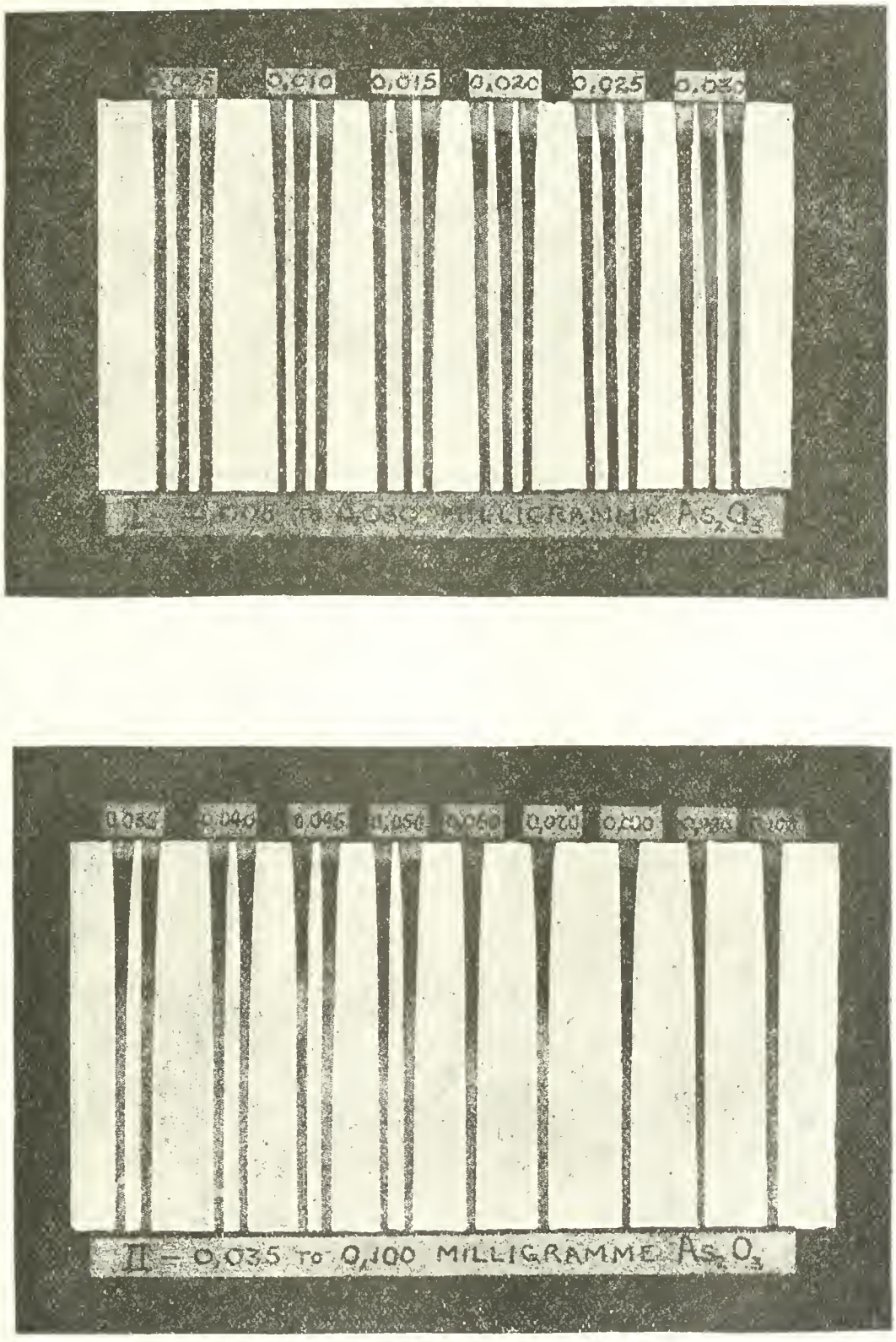

Fig. 24.—Sanger's Scale of Type Mirrors.

Quantitative Determination of Arsenic.—By far the most delicate and accurate method of determining small quantities of arsenic in forensic cases is by the Marsh apparatus. Prof. J. C. Draper[1] was the first to suggest an apparatus and method by which the Marsh process was made to deposit all of the arsenic introduced into the generator. The matter has been further studied by Gautier,[2] Johnson and Donaldson,[3] and Sanger.[4] If the proper precautions be taken the results are extremely accurate, and very small quantities may be determined. The main conditions necessary are that the quantity of arsenic be not too great, that the generator be not allowed to become heated, that the gas be made to traverse a sufficient length of red-hot tube, and that the evolution of gas shall be slow and uniform. When the quantity of arsenic separated is sufficient to form a large mirror the amount may be determined by direct weighing. The tube is cut at a safe distance from the mirror, and the section containing the arsenic is weighed on an assay balance, or other balance sensitive to 0.0000[illegible] gm.; the mirror is then removed by heat or, preferably, dissolved out with nitric acid, and the cold and dry tube again weighed. The difference is the weight of elementary arsenic, which, multiplied by 1.32, gives the amount of arsenic trioxid, As_2O_3. Even smaller than weighable quantities may be determined by comparison of minute mirrors with a standard scale of mirrors containing known quantities in tubes of equal size. A representation of such a scale of type mirrors is here reproduced (Fig. 24) from Sanger's paper. It will be seen that quantities of arsenic may be thus estimated with considerable accuracy down to 0.001 mgm. As_2O_3 or 0.0007 mgm. As.[5]

When the quantity of arsenic introduced approaches a decigram (one and one-half grains) the accuracy of the method is diminished, possibly by reduction of the hydrogen arsenid and retention of arsenic in the generator.[6]

When present in sufficient amount arsenic may also be de-

[1] Amer. Chemist, 1872, ii., 456.
[2] Bull. Soc. Chim., Paris, 1875, xxiv., 250.
[3] Amer. Chem. Jour., 1880-81, ii., 235.
[4] Proc. Am. Ac. Arts and Sc., 1891, xxvi., 24.
[5] See also Otto: "Ausmittelung der Gifte," 6te Aufl., 178.
[6] See Kühn und Saeger ("Berichte," Berlin, 1890, xxiii., 1798), whose results are no doubt due to the use of too concentrated a solution and to too rapid a generation of gas.

termined gravimetrically, preferably as magnesium arsenate, or ammonio-magesium arsenate, or as arsenic trisulfid;[1] or volumetrically by Mohr's,[2] Bernstein's,[3] or Mayrhofer's[4] methods.

[1] See Fresenius: "Quant. Anal.," 6te Aufl., i., 367 *et seq.* The directions must be strictly followed.

[2] "Titrirmethode," 3te Aufl., 275.

[3] "Ueber Arsenwasserstoffgas," etc., Diss., Rostock, 1870.

[4] Ph. Centralh., 1889, n. F., x., 387.

BARIUM COMPOUNDS.

Although the salts of barium, the chlorid, carbonate, nitrate and acetate, are actively poisonous in comparatively small doses, and cause death quite rapidly, their forensic interest is only possible, not present; we will therefore content ourselves with a summary of the few accidental poisonings which have occurred and the bibliography of the subject.

Cases.

1. *Walsh: Lancet*, 1859, *i.*, 211.—F., 22, A., D. in seventeen hours; about 4 gm. (one drachm) chlorid in water. By mistake for Epsom salt.

2. *Tidy: M. Press and Circ., Lond.*, 1868, *vi.*, 447.—M., 46, A., D. in six and one-half hours. Mixture of barium nitrate, etc. (green fire) in mistake for sulfur. Analysis.

3. *Wach: Ztschr. f. Staatsarznk.*, 1835, *xxx.*, *i.*—F., 42, A., D. in two hours; about 15 gm. Mistake for Glauber's salt.

4. *Seidel, Vrtljschr. f. ger. Med., etc.*, 1877, *n. F.*, *xxvii.*, 243.—F., 28, D. in twelve hours. Barium carbonate taken as abortive.

5. *Ogier and Socquet: Ann. d'hyg., etc.*, 1891, 3 *s.*, *xxv.*, 447.—M., ad., A., D. in four and one-half hours. About 20 gm. barium chlorid in mistake for magnesium sulfate.

6. *Reincke: Vrtljrschr. f. ger. Med., etc.*, 1878, *n. F.*, *xxviii*, 248.—Family of four persons poisoned by barium carbonate in cake, probably accidentally in mistake for chalk as an adulterant. One man died in four hours.

7. *J. Sc. and Arts, Lond.*, 1818, 382.—F., ad., A., D. in one hour from one ounce (31 gm.) barium chlorid, taken in mistake for Glauber's salt.

8. *Mather: Med. Comment. Edinb.*, 1795, *xix.*, 267.—M., ad., A. R., overdose of barium chlorid solution given as a medicine.

9. *Wilson: Lond. M. Gaz.*, 1833-34, *ii.*, 487.—F., yg. ad., S. R., half teacupful (?) of carbonate.

10. *Wolf: Wchnschr. f. d. ges. Heilk.*, 1850, *No.* 37.—Barium chlorid in mistake for Epsom salt.

11. *Pharm. Centralh.*, 1867, *No.* 5.—M., ad., A., D. soon. Barium chlorid in mistake for Carlsbad salt.

12. *Chevallier: Ann. d'hyg., etc.*, 1873, 2 *s.*, *xxxix.*, 395.—M., 40, D. from considerable quantity of barium acetate.

13. *Lagarde: Union méd., Par.*, 1872, *xiv.*, 537.—A., D. from barium acetate in mistake for sodium sulfovinate.

14. *Husemann; Ztschr. f. pr. Hlk.*, 1866, *iii.*, 232.—A., D. after 10 gm. (154 grains) barium chlorid taken in five doses during two days. H. questions Ba as the cause of death.

15. *Felletar: Pest. med.-chir. Presse*, 1892, *xxxix.*, 1078.—Not seen.

16. *Courtin: Rev. d'hyg., Par.*, 1882, *iv.*, 653.—Not seen. Chlorid.

17. *Eschricht: Ugeskr. f. Laeger., Kjøbenh.*, 1881, 4 *R.*, *iv.*, 241.—Not seen. Nitrate.

Method of Action, etc.

Bary, A.: "Beiträge zur Baryum-Vergiftung," Diss., Dorpat, 1888. —*Boehm u. Mickwitz:* Arch. f. exp. Path. u. Pharm.—*Cyon:* Arch. f. Anat., Physiol., u. wiss. M., 1866, 196.—*Linossier:* Ct. rend. Soc. biol., Par., 1887, 8 s., iv., 122.—*Onsum:* Arch. f. path. Anat., etc., 1863, xxviii., 233.—*Sommer, F.:* "Beiträge," etc., Diss., Würzb., 1890.—*Blake:* J. An. and Phys., 1874, 2 s., viii., 242.—*Ringer and Sainsbury:* Practitioner, 1883, 81.—*Brunton and Cash:* Tr. Roy. Soc., 1884, 222.

BISMUTH.

Bismuth is used medicinally in three forms of combination: the **citrate**, $BiC_6H_5O_7$, a white, amorphous, odorless, and tasteless powder, insoluble in water, but soluble in ammonium hydroxid; the **subcarbonate**, or *bismuthyl carbonate*, $(BiO)_2CO_3,H_2O$; and the **subnitrate**, or *bismuthyl nitrate*, $(BiO)NO_3,H_2O$, both white, amorphous, odorless, and tasteless powders, insoluble in water or alcohol, soluble in warm, concentrated nitric acid, the solution again depositing the subnitrate on dilution with water. The subnitrate is also known as *Magistery of Bismuth*, and is used as a cosmetic under the name *pearl white.*

It is very questionable whether either of these preparations *when pure* is possessed of any toxic powers, as they are only absorbed to a very slight extent,[1] and pass out with the fæces

[1] In two cadavers which contained notable quantities of arsenic we found 10.6019 and 3.6282 gm. (163.6 and 55.99 grains) of bismuth subnitrate in the intestines, while the livers yielded quantities of bismuth

unchanged or converted into the black or dark-brown bismuth sulfid, which is frequently found as a heavy powder in the dejecta. Those cases of alleged poisoning which have been reported by Pott,[1] Odier,[2] Guersent,[3] Kerner,[4] Wernek,[5] Traill,[6] Kocher,[7] Riedel,[8] Dalché,[9] Lewin,[10] and Wilson,[11] were caused, not by the bismuth, but by an impurity which it contained. We have already seen (see page 369) that the medicinal bismuth compounds frequently contain arsenic. But this is not the only contamination to which they are subject.

Reisert[12] and Braithwaite[13] have shown that they also contain tellurium. The latter found 0.076 per cent. in the subnitrate and 0.05 per cent. in the subcarbonate. It was also shown that the peculiar garlic-like odor observed in the breath, the so-called *bismuth breath*, is due to this impurity. According to Reisert an extremely small quantity of tellurium is capable of causing this odor, the occurrence of which is suggested as a physiological test for that element. Bismuth subnitrate has also been found to be contaminated with antimony and with lead.

Bismuth compounds are of forensic interest only in connection with arsenical poisoning. Being remedies customarily used to control gastric disturbance, they are frequently administered to patients suffering from arsenical poisoning when the cause of the illness is not recognized. When thus given in cases not rapidly fatal notable quantities may remain in the intestinal canal, and, by superficial conversion into sulfid, appear as a crystalline or amorphous, dark-green or black deposit attached to the walls of the stomach and intestine. We have found as much as 10.6019 gm. (163.6 gr.) of bismuth subnitrate in the intestines and their contents. The questions dependent upon

corresponding to .0052 and .0569 gm. (.08 and 0.9 grain) of bismuth subnitrate respectively.

[1] Wibmer: "Wirkung der Gifte," i., 416.

[2] J. d. méd., Paris, 1768, lxviii., 49.

[3] Orfila: "Tox. gén.," 3ème ed., i., 599.

[4] Heidelb. kl. Ann., 1829, v., 348.

[5] Med.-chir. Ztg., 1831, lxx., 312.

[6] "Outlines Med. Jur.," Phila., 1841, p. 130.

[7] Volkmann's Samml. kl. Vortr., 1882, No. 224.

[8] Berl. kl. Wochenschr., 1882, No. 19.

[9] Ann. d'hyg., etc., 1886, 3 s., xvi., 358.

[10] "Nebenwirkungen," 2te Aufl., 1893, p. 747.

[11] N. Y. Med. Jour., 1894, lix., 87.

[12] Am. J. Pharm., 1884, lvi., 177.

[13] Amer. Druggist, 1884, xiii., No. 5.

the presence of arsenic in bismuth compounds have already been discussed (see page 369).

Analytically the separation and quantitative determination of bismuth present no difficulties. In the systematic process bismuth sulfid remains in the residue insoluble in ammonium sulfid (see page 159), possibly along with mercury, lead, cadmium, and copper if these be present. The sulfids are extracted with warm nitric acid, which dissolves all except mercuric sulfid and some separated sulfur; the solution is filtered, the greater part of the acid is neutralized with ammonium hydroxid; ammonium chlorid is added in sufficient amount, and then considerable water. After standing several hours a portion of the clear liquid is further diluted, and, if necessary, water is added until a further addition causes no cloudiness. The precipitated bismuthyl chlorid is collected on a filter, washed with cold water, dried, and fused in a weighed porcelain crucible with five times its weight of postassium cyanid. The cooled fusion is rapidly extracted and washed with water by decantation in the crucible, then with aqueous alcohol and finally with strong alcohol, and dried. The crucible with the metallic globules of bismuth is then weighed. Or the bismuth may be precipitated by ammonium carbonate, washed, dried, ignited, and reduced by potassium cyanid in fusion.

CHLORATES.

Potassium chlorate—$KClO_3$—122.28—*Potassii chloras, U. S. P.*, a white, crystalline salt, soluble in 16.5 parts of cold water and in 2 parts of boiling water, only sparingly soluble in alcohol—and **sodium chlorate—$NaClO_3$** — 106.25 — *Sodii chloras, U. S. P.*, soluble in 1.1 parts of cold water, in 0.5 part of boiling water, and in 40 parts of alcohol, are the only chlorates of toxicological interest.

Of these the latter has been only exceptionally used in medicine, and has, so far as we can find, caused but one poisoning: the death of a man of 30 years on the fourth day after having taken 39 gm. (3 x.) in six hours, in place of in six days as directed.[1]

[1] Jacobi: Med. Rec., N. Y., 1879, xv., 243.

Poisonings by the potassium salt are of much more frequent occurrence, because of its more general use as a medicine. We find record in medical literature of 89 cases,[1] of which 76 terminated in death. Almost all of these were accidental, caused either (in 7 cases) by the chlorate having been taken in mistake for some other medicine, or, in the great majority of cases, by its having been administered in overdose, or swallowed by the patient when prescribed as a gargle. Of the fatal "overdose" cases four were the subject of judicial investigation, having been caused by administration to children of a remedy for sore throat, dispensed from a convent;[2] and one was in the person of a physician who died from the effects of 31 gm. (℥ i.), which he took to testify to his belief in the harmlessness of the drug.[3]

We find mention of but six suicides or supposed suicides by this agent;[4] and of these two were quite as probably accidental as suicidal.

Of chlorate poisoning having a homicidal element we find but three instances: One was that of a child of 1½ years which seems to have been intentionally destroyed by its mother;[5] another was the supposed poisoning of an illegitimate child of 2½ years by its mother;[6] and the third was the case of a girl who died from the effects of potassium chlorate. A priest was convicted of attempted abortion in having administered the chlorate to her for that purpose.[7] Maschka has reported the case of an illegitimate child supposed to have been poisoned by its mother in which, however, neither the cause of death, nor the probability of administration by the mother is established.[8]

The minimum **lethal dose** of the potassium salt may be

[1] Not including six in journals inaccessible to us.

[2] Aff. de la Supérieure d. Saint-Saturnin-du-Port-d'Envaux. Brouardel and l'Hôte: Ann. d'hyg., 1881, 3 s., vi., 232.

[3] Case of Dr. Fountain. Stillé: "Therap. and Mat. Med.," 2d ed., ii., 776.

[4] Marchand: Arch. f. path. Anat., etc., 1879, lxxvii., 455 (?). Meyer: Diss., Berlin, 1893, p. 16. Schuchardt: Deut. m. Wochensch., 1888, xiv., 835. Otto: St. Pet. m. Wochenschr., 1882, vii., 235 (?). Kostilew: Med. Obozr., Mosk., 1886, xxv., 759 (two cases).

[5] Pharm. Ztg., 1881, xxvi., 229.

[6] Seydel: Vrtljschr. f. ger. Med., etc., 1885, n. F., xliii., 273.

[7] Affaire T——, Chapelle de la Tour. Lacassagne: Arch. d. l'Anthrop. crim., 1887, ii., 359. Hougounenq: "Poisons," Paris, 1891, p. 467.

[8] Prag. med. Wochenschr., 1893, xviii., 223.

approximately stated as 12 gm. (3 ʒ) for an adult, 5 gm. (1¼ ʒ) for a child, and 1 gm. (15 gr.) for an infant.

With adults smaller doses appear to be capable of causing death when divided than when taken at one time. The smallest single dose which has caused the death of an adult was 25 gm. (386 gr.) taken by a man of 26 years in mistake for Epsom salts, who died in 36 hours.[1] In divided doses 11.75 gm. (180 grs.), taken during three days, caused the death of a man of 31 years.[2] Two hundred grains (13 gm.), taken during two days, caused the death of a girl of 11 years on the sixth day.[3] Twenty grams (308 gr.), given to a man of 64 years in small doses during three days after a lithotripsy, produced the symptoms of chlorate poisoning, and he died in 4 days.[4] In two instances young men of 15½ and 19 years respectively died from the effects of 25 gm. (386 gr.) taken in divided doses, the former in 5 days, the latter in 8 hours.[5] We find record of no less than 12 cases in which adults have been killed by doses of about 30 gm. (1 ℥).

An infant of 3 weeks died in 3 days from the effects of about 1 gm. (15 grs.) of powdered chlorate dusted into its mouth.[6] A child of less than a year died in 24 hours from the effects of 3.9 gm. (1 ʒ).[7] Another child "several years old" died from swallowing a gargle containing 4 to 5 gm. (62–77 grs.).[8] Still another child (3 years) died in 9 days from swallowing 30 three-grain chlorate lozenges, of which 12 were expelled by vomiting within a quarter of an hour, the amount remaining being about 20 grs. (1.3 gm.).[9] Jacobi, who was the first to call attention to the dangerous character of the chlorate, refers to a number of fatal poisonings by small doses, several previously unrecognized as such.[10]

[1] Ferris: Pac. M. and S. Jour., 1873–74, xvi., 18.

[2] Zillner: Wien. m. Wochenschr., 1882, xxxii., 1331.

[3] McShane: J. Am. Med. Assoc., 1894, xxiii., 860.

[4] Billroth: Wien. med. Wochenschr., 1880.

[5] Satlow: Jahrb. f. Kinderhlk., 1881–82, xvii., 311. Laaser, in Krückel: "Ueb. d. Tox. u. Therap. Wirk. d. Kal. Chlor.," Diss., Kiel, 1891, p. 12.

[6] Goldschmidt: Breslau. aerztl. Ztschr., 1883, v., 6.

[7] Hall: Med. Rec., N. Y., 1878, xiv., 396.

[8] Laaser: *Loc. cit.*

[9] Abrams: Occid. M. Times, 1892, vi., 23.

[10] Tr. M. Soc., N. Y., 1879, xv., 241, and "Treat. on Diphtheria," N. Y., 1880, p. 162. Jacobi's first publication was in 1860 (Amer. M. Times). Of the eighty-nine cases above referred to only one (Cheval-

The largest quantity which has been known to have been taken without causing death was 46.7 gm. (719 gr.), taken by a man of 25 years during 13 hours. He was discharged after an illness of 13 days.[1] A child of 7 years received about 25 gm. (386 grs.) in solution, and suffered the usual symptoms, but recovered slowly.[2] Rabow[3] reports the case of a woman who took 20 gm. (308 grs.) in mistake for Carlsbad salts and in whom not the slightest reaction was observed! As only ten cases of non-fatal poisoning in which the amount of the dose taken is given have been reported, it is quite possible that notably larger quantities than those stated may be recovered from.

The average **duration** of 59 cases in which it is given was 4 days and 22 hours. The shortest duration was in the case of a man of 30 years, who died in 6 hours from the effects of 75 gm. (19¼ ʒ).[4] A child of 2½ years died in 7 hours;[5] a man of 67 years in 7½ hours;[6] a young man of 19 years in 8 hours;[7] a boy of 3½ years in 8½ hours;[8] a child of 2¾ years in 9 hours;[9] a man in 11 hours;[10] and a boy of 2½ years in 12 hours.[11] In 3 cases the duration is stated as "a few hours."[12]

With the exception of 3 cases to be referred to below, the longest duration was in Schallmayer's case:[13] A young man swallowed the crystals three or four times daily for 5 days, on the seventh day after the first dose he was admitted to the hospital, where he died 12 days later. Two cases of 12 days' duration are also recorded, one by Marchand, in a boy of 3 years;[14] the other by Wegschneider, in a man of 25 years.[15] Three quasi-chronic cases of longer duration have occurred: A child

lier: J. de chim. méd., etc., 1855, 4 s., i., and Lacombe: *Ibid.*, 1856, 4 s., ii., 197) was published before that date. Koppel ("Vergift. d. Blutgifte," Diss., Dorpat, 1891) gives a bibliography of sixty-six cases, 1880–1889.

[1] Gesenius: Deut. m. Wochenschr., 1882, viii., 512.

[2] Baginsky: *Loc. cit.* (Case II.).

[3] Deut. med. Wochenschr., 1886, xii., 199.

[4] Neuss: Deut. m. Wochenschr., 1884.

[5] Kennedy: Am. Jour. Pharm., 1878, l., 113.

[6] Manouvriez: Ann. d'hyg., 1880, 3 s., iii., 543.

[7] Laaser: *Loc. cit.*

[8] Brouardel and l'Hôte: *Loc. cit.*

[9] Rodenstein: Am. Jour. Obstet., 1882, xv., suppl., 15.

[10] Maschka: Wien. med. Wochenschr., 1886, xxxvi., 534.

[11] Brouardel and l'Hôte: *Loc. cit.*

[12] Pharm. Ztg., 1881, xxvi., 229. Meyer: Diss., Berlin, 1893, p. 16. Riess: Berl. kl. Wochenschr., 1882, xix., 785.

[13] "Ein Fall von Vergiftung mit Kali chloricum," Diss., Würzburg, 1889.

[14] Arch. f. path. Anat., etc., 1879, lxxvii., 455 (Case III.).

[15] Deut. med. Wochenschr., 1880, vi., 533.

of 1½ years was treated for diphtheria by chlorate gargles for 22 days; the disease disappeared, but the child died from chlorate poisoning.[1] A girl of 9 years was allowed to chew crystals of the chlorate and died in 1 month in consequence.[2] A man had been in the habit of taking chlorate troches for chronic throat trouble for 2 years; he was taken with symptoms of chlorate poisoning and died in 4 days.[3]

The **symptoms** of chlorate poisoning are both local and general. The local action of the poison is that of a gastro-intestinal irritant, producing severe vomiting, copious diarrhœa, and epigastric and abdominal pain. These are most pronounced in those cases in which large doses are taken, and in which the course of the poisoning is most rapid.

In Maschka's case[4] of death in 11 hours, besides pain and vomiting, the patient complained only of weakness and great thirst. He went to sleep, when the heavy breathing and tracheal râles caused his wife to awaken him. After a few inarticulate words he had severe tonic convulsions of the upper extremities; and a physician who was summoned found the patient unconscious, perspiring profusely, and having severe spasms. He died shortly after having been transferred to the hospital.

But pain may be absent even in comparatively rapidly fatal cases, in which the cardiac action is well marked:

A woman of 70 years took about 30 gm. (℥ i.) of chlorate dissolved in water in mistake for Epsom salt. There were no symptoms for an hour, when the mistake was discovered and an emetic was given. Two hours later there was still neither pain nor distress. Four hours later, while defecating, she fainted, became collapsed, with blue lips, ashen face, purple tongue; temperature, 100° F.; pulse, 100, weak; there was profuse emesis of mucus but no blood. She died in collapse 15 hours after having taking the poison, without at any time having had suppression of urine, dyspnœa, or pain.[5]

The most marked action of the poison, however, is upon the blood corpuscles. Indeed, it is the type of the methæmoglobin-producing toxics, and the most prominent symptoms which it produces are due to its action upon the blood pigment, or rather

[1] Baginsky: Arch. f. Kinderhlk., 1880, i., 100.
[2] Jones: Med. Rec., N. Y., 1885, xxviii., 513.
[3] Peabody: Med. Rec., N. Y., 1888, xxxiv., 57 (Case II.).
[4] *Loc. cit.*
[5] Anderson: Med. Rec., N. Y., 1889, xxvi., 707.

to the consequences of such action manifested in the respiration and cardiac action, and in changes in the liver, spleen, nerve centres, and notably in the kidneys. The cardiac action is also due in part to the toxic power of the potassium.

The alteration of the blood corpuscles has been directly observed in the human subject: In Riess' case,[1] in blood drawn from the finger on the day of admission one-fourth to one-fifth of the corpuscles had lost all of their hæmoglobin, or contained but a very small quantity. Their outlines were well preserved. The normal corpuscles were in rouleaux. In Landerer's case[2] the corpuscles in the blood from the finger showed defective rouleaux formation, and were agglutinated in little piles. As an external manifestation of blood alteration the skin is frequently marked with gray-violet patches, or copper-colored spots, which disappear on pressure; or, in consequence of the effect upon the liver, it becomes markedly icteric.

The effects upon the kidneys are manifested in the urine, which is small in quantity, cloudy, and of a red-brown or almost black color, contains abundance of albumin, and when examined spectroscopically shows the bands of methæmoglobin or those of hæmatin. The sediment at first contains hyaline casts, but later broad brown or yellow casts, with abundant granular matter and detritus of red corpuscles, sometimes with corpuscles more or less modified, crenated, and agglutinated in masses. Frequently the urine contains potassium chlorate as such, or an excess of potassium chlorid. But the urine, although diminished in quantity, has been in a mild case described as clear and perfectly free from albumin.[3] Or the secretion of urine may be entirely suppressed.

The liver and spleen are notably enlarged, and there is tenderness, particularly on pressure, in that region, as well as over the kidneys.

The action upon the nervous system is manifested by delirium, or coma, or violent tonic or clonic spasms, with fixation of the muscles of the extremities, the neck or jaw; or sometimes a condition approaching opisthotonus. Usually the

[1] Berl. kl. Wochenschr., 1882, xix., 785.

[2] Deut. Arch. f. kl. Med., 1890–91, xlvii., 103.

[3] Leichtenstern: Deut. med. Wochenschr., 1884, x., 305.

patient becomes unconscious, but sometimes consciousness remains perfect until near the end. In one instance the patient went to sleep and died without a struggle.[1] Death may occur suddenly during a spasm or convulsion, but is usually in collapse. In one instance on the eleventh day the patient, a man of 51 years, had much improved, but was all at once seized with an attack of dyspnœa and fell back dead.[2]

Treatment.—Emetics should be given, or the stomach washed out. Excretion by the saliva may be stimulated by hypodermics of pilocarpin. Stimulants and digitalis are indicated. Inhalation of oxygen may be serviceable. Transfusion of blood was tried in one case with results beneficial at first, but afterward not so favorable, and without saving the patient.[3]

Post-Mortem Appearances.—These are more character-

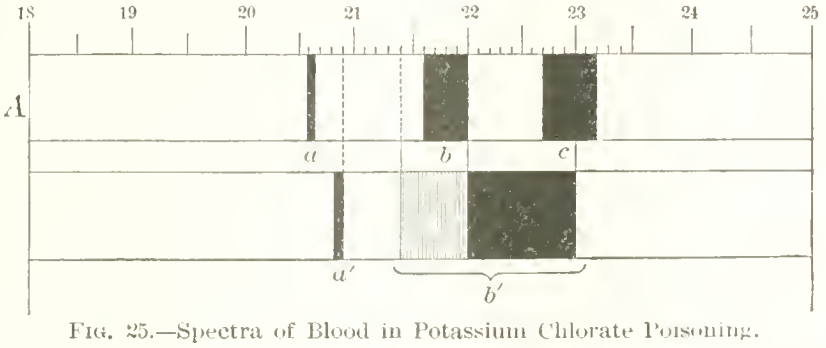

Fig. 25.—Spectra of Blood in Potassium Chlorate Poisoning.

istic than is usually the case with the true poisons, provided the autopsy is made soon after death. The *blood* is of a peculiar dark chocolate-brown color. Under the microscope the corpuscles are very pale, and some contain punctiform vacuoles. On dilution with water it forms a turbid solution which, when filtered, is Burgundy-red, and on sufficient dilution shows the spectrum A, Fig. 25. On addition of a trace of ammonium hydroxid *a* disappears. On addition of ammonium sulfhydrate, on the other hand, *a* disappears first, while *b* and *c* slowly fuse together to produce *b′;* before this is quite accomplished *a′* makes its appearance and is visible in dilute solution; *b′* plainly shows two parts, a lighter and a darker, the more sharply defined one being nearer to the blue.[4]

[1] Billroth: *Loc. cit.*

[2] Lenhartz: Deut. med. Wochenschr., 1887, xiii., 9.

[3] Satlow: Jahrb. f. Kinderhlk., 1881–82, xvii., 311.

[4] See Hoffmann: Med. Jahrb., Wien, 1881, 473.

The liver, spleen, and kidneys are enlarged. The *liver* may be normal in appearance, or icteric. Microscopically, hæmoglobin-colored masses are found in the capillaries, between the cells, but not in the cells. The *spleen* is easily torn, intensely hyperæmic, and filled with red corpuscles, more or less disintegrated, and some white corpuscles and pigment granules. The *kidneys* are intensely hyperæmic, with œdematous swelling of the interstitial tissue, the medullary portion gray, the cortex yellow, with chocolate-colored stripes in the papillæ. There is round-cell infiltration around the vessels; proliferation of nuclei in the glomeruli cells; within the glomeruli are fine fibrin coagula and pigment scales. The tubuli are filled with casts, especially the straight ones, the casts containing blood detritus and epithelial cells, and a few being hyaline. In the convoluted tubes the casts are not so plentiful, but the tubes are remarkably widened and flattened. The epithelial cells are granular, their nuclei do not stain in some cases, and some show vacuoles. The lumina of the tubes are traversed by dry reticulated coagula. The mucous membrane of the *stomach* is swollen and marked in places with small, shallow erosions, and with punctiform hemorrhages, or ecchymoses. In the *small intestine* the mucous membrane is œdematous and injected, with loss of substance in places. Peyer's patches and solitary follicles are swollen and sometimes ecchymotic. The capillaries of the mucous membrane of the *large intestine* are markedly congested. The *lungs* are marked with subpleural ecchymoses, brownish in color, and are hyperæmic and œdematous. The pleural cavity contains fluid, varying in color from yellow to deep red. The *heart* is dilated, containing brown-red cruor; and the pericardium contains yellow fluid. The *brain* is smoke-colored, the meningeal and cerebral vessels are injected with rust-brown-colored blood, and the sinuses and arteries at the base filled with fluid blood of the same color. The *marrow* of the bones is softer than normal, brown in color, marked with spots, and its cells contain numerous aggregations of granular detritus of blood corpuscles which is readily soluble in acetic acid. The *skin* is icteric, or of a peculiar marble whiteness.[1]

[1] For colored plates of the appearances of various organs in a case of chlorate poisoning, see Lesser: "Atl. d. ger. Med.," pl. xvii., Fig. 8; pl. xviii., Figs. 1–4, 9.

Analysis.—Potassium chlorate is eliminated in part unchanged in the urine, perspiration and saliva, as well as in the milk and tears. It may be separated from the urine or vomit, or from the contents of the stomach or blood in recent cases, as follows: The material is diluted with boiling water; if not acid, it is rendered faintly so with acetic acid, and then boiled for a moment or two to coagulate albumin. The liquid is then filtered, concentrated to a small volume over the water-bath, and allowed to stand in a cool place until quite cold. The chlorate being relatively sparingly soluble in water, crystallizes out, and the crystals may be separated from the mother liquor, dried between folds of filter paper, and identified by the following reactions: 1. Heated in a dry test tube the solid melts and gives off oxygen, which causes a glowing match-stick to burst into flame when introduced into the mouth of the tube. 2. When thrown on a glowing coal the crystal deflagrates with a violet flame. 3. Powdered and mixed with charcoal or sulfur the mixture burns with a bright light when ignited. 4. When moistened with concentrated sulfuric acid a crystal gives off a yellow, pungent, explosive gas having an odor somewhat resembling that of chlorin, and the acid is colored yellow. 5. A crystal moistened with hydrochloric acid gives off chlorin. 6. A solution of the chlorate when colored slightly blue by indigo and treated with sulfuric acid remains blue, but the blue color is discharged on the addition of a solution of sulfurous acid or of sodium sulfite.

A portion of the chlorate is converted in the system into the chlorid, and eliminated as such, but the presence of an excess of chlorids in the urine may be due to many other causes.

CHROMATES.

Of the compounds of chromium of toxicological interest, one, **chromic anhydrid**, has already been considered (see page 262); another, **lead chromate**, used as a pigment under the name **chrome yellow**, is rather a plumbic than a chromic poison so far as it is poisonous. Another chromic pigment, **chrome green**, may be either a mixture of chrome yellow with some blue pigment, such as Prussian blue, or *Guignet's green*, which is an hydroxid of the formula $Cr_2O(OH)_4$. This

may contain boric acid or borax as an impurity, but when properly prepared it is insoluble and non-poisonous. Of the chromates two are used in the arts: **Potassium chromate,** K_2CrO_4, a yellow salt readily soluble in water, used principally in the manufacture of chrome yellow, which is actively poisonous, but which has, so far as we know, been the cause of poisoning in the human subject but four times: Professor Parochow, of Charkow, took a tablespoonful of the chromate "in place of Glauber's salt" (!), and died in twelve hours.[1] Hjelt has reported another instance of the fatal poisoning of an adult by the same salt in Finland in 1872.[2] Gläser[3] reported an unsuccessful attempt at suicide with about 10 gm. of the chromate. Fagerlund[4] reported a fatal poisoning by a saturated solution of the chromate taken by a man while drunk.

Potassium dichromate—*bichromate of potash—potassium pyrochromate*—$K_2Cr_2O_7$—is an orange yellow, crystalline body, odorless, bitter and metallic in taste, soluble in 10 parts of water at 15° (59° F.) and in 1.5 parts of boiling water; insoluble in alcohol. It is quite extensively used in dyeing, in the preparation of "battery fluid" (see Chromic Anhydrid), in the manufacture of the chromate and of certain anilin dyes, and is also used in medicine (*Potassii bichromas, U. S. P.*).

Its toxic powers were first observed in Scotland in 1827, when Duncan and Cuming[5] noted its local action upon the operatives engaged in its recently introduced industrial applications.[6]

Besides numerous accounts of industrial poisonings, more or less severe, and which are of hygienic rather than forensic interest, we find record of 14 accidental poisonings by dichromate, 18 suicides, and 1 case in which a woman of 24 years was supposed to have died in 24 hours after having taken the salt as an abortive.[7] Of truly homicidal cases we can find no mention.

The smallest dose of the dichromate which has been known

[1] Neese: Pharm. Ztschr. f. Russl., 1862, No. 7.

[2] Schmidt's Jahrb., 1876, clxx., 232.

[3] Deut. med. Wochenschr., 1886, xii., No. 17.

[4] Vierteljschr. f. ger. Med., etc., 1894, viii., supplhft., 76.

[5] Edinb. M. and S. Jour., 1827, xxvi., 133; xxviii., 301.

[6] For bibliography of industrial and other poisonings by dichromate, see Pander: "Arbeit. d. pharm. Inst.," Dorpat, ii., 39.

[7] Schrader: Vrtljschr. f. ger. Med., etc., 1866, n. F., v., 113.

to cause the death of an adult was from 2 to 3 gm. (31 to 46 grs.), taken in the form of powder, with suicidal intent, by a man of 43 years, who died in 10 days.[1] Another suicide, a young man of 19 years, died in 10¾ hours after having taken about 7.5 gm. (114 grs.).[2] A boy of 20 months died in 9¼ hours after having accidentally swallowed a crystal weighing about 10 grs. (0.65 gm.).[3] On the other hand, a man of 48 years took 273 grs. (17.7 gm.) in solution in mistake for tea, and recovered;[4] and in two instances men have unsuccessfully attempted suicide with 15 gm. (℥ ss.).[5]

The **duration** of fatal cases is usually short. Excluding the 3 extreme cases mentioned below, the average duration in 12 cases in which it is accurately reported was 13¾ hours. In one instance a woman of 46 years, a suicide, died in 40 minutes;[6] and in another a man, also a suicide, died in 50 minutes.[7] On the other hand, in Klimesh's case, above referred to, life was prolonged for 10 days. This is, however, the only fatal case of which we find record in which the duration exceeded 24 hours, except one case of 54 hours' duration.[8]

The **symptoms** are prompt in appearance. Within five minutes or less there is most intense pain in the stomach and back, followed or accompanied by violent and continuous vomiting, the vomited matter consisting at first of the food, tinged with the poison, and subsequently becoming bilious and bloody. In a short time there is severe purging, and the patient rapidly becomes almost unconscious, pulseless, and collapsed. Usually the onset is rapid and sudden, and within a very short time the person is doubled up, unable to speak, or to perform any voluntary act. But cases are recorded in which, even when the poison has been taken in solution, the victim, although suffering violent pain, has been able to help himself for as long as half an hour after taking the poison.[9] In Schin-

[1] Klimesh: Wien. kl. Wochenschr., 1889, ii., 732.

[2] Gesenius: Deut. med. Wochenschr., 1882, viii., 511.

[3] McCrorie: Glasgow M. Jour., 1881, xv., 378.

[4] Philipson: Lancet, 1892, i., 138.

[5] Warwick: Lancet, 1880, i., 167. Waugh: *Ibid.*, 1885, ii., 1135.

[6] Stewart: Brit. M. Jour., 1888, ii., 420.

[7] Waugh: *Loc. cit.* (Case II.).

[8] Schindler: Repert. f. d. Pharm., lxix., 387.

[9] Johnson: M. Times and Gaz., 1877, ii., 447. See also Walker: Lancet, 1879, ii., 464. Bullock: *Ibid.*, 1890, ii., 560. Macniven: *Ibid.*, 1883, ii., 496. Knight:

dler's case, above referred to, the course of the poisoning seems to have been exceptional, probably because of prompt treatment. He was given warm water, soap and oil, and emetics, until the discharges were no longer yellow, after which he appeared well and passed a good night. The next morning he was excessively weary, suffered from pains in the back and kidneys, and had suppression of urine and frequent purging. The second night was very restless, and in the morning he was motionless and completely exhausted, and died 54 hours after having taken the solution. The emetics had obviously not caused the complete discharge of the poison, as chromium was found in the stomach after death.

The **post-mortem appearances** are not characteristic. The most diagnostic, when it is present, is a grayish-blue, greenish, or purplish-blue coloration of the skin in places, of the gums, meninges, stomach, liver, lungs, and other internal organs, caused by reduction of the chromate. The blood does not coagulate readily, is brownish in color, and shows the spectrum of methæmoglobin.[1] The mucous membrane of the lower œsophagus is injected. The gastric mucous membrane is swollen, ecchymotic, particularly at the fundus, and in some cases is shredded off, as is that of the upper small intestine. The intestinal mucous membrane, particularly in the large intestine, is hyperæmic and ecchymosed, with enlargement and elevation of Peyer's patches and solitary glands. The liver and heart have been found to be the seat of fatty degeneration. A brown-red exudation is found in the peritoneal and pleural cavities. The kidneys are dark and hyperæmic, and the convoluted tubes contain occasional hyaline casts, their cells being unaltered.

Analysis.—Occasionally, when the dichromate has been taken in the solid form, it may be found in substance in the stomach and intestine,[2] but usually the most that is to be expected, under favorable conditions, is the detection of chromium, either in the urine, vomit, alimentary contents, blood, liver, or kidneys.[3] In the systematic analysis chromium, if present, is found in the residue X (page 160); this is fused with disodic

Med. Press and Circ., 1885, n. s., xl., 532. Kiemann: Ber. . . . Rudolph Stift., Wien, 1886, 296.

[1] Ruttan and Lafleur: Montreal M. Jour., 1888, xvii., 81.

[2] Maschka: Vrtljschr. f. prakt. Hlk., 1877, cxxxvi., 37.

[3] Cf. Ruttan and Lafleur: *Loc. cit.*

carbonate and potassium chlorate, the cooled fusion extracted with water, and the solution tested for chromium as directed at page 263.

COPPER.

Are the compounds of copper, when not taken in excessive quantity, poisonous? This is a question the affirmative of which is firmly fixed in the popular mind, yet it is one upon which toxicologists and hygienists have been by no means unanimous. Copper and brass have been used from time immemorial in the manufacture of cooking utensils. Biblical and classical literature contains frequent references to them; and not only have numerous copper, brass, and bronze utensils been found in excavations in Europe and Asia, but upon this continent there is evidence that the copper deposits of Lake Superior were worked at an early period, and by a race probably differing from and possibly antedating the red Indian. In the early part of the eighteenth century deaths caused by food articles, which were undoubtedly due to the then unknown products of putrefaction, were first attributed to their having been prepared in copper utensils, and were considered as cupric poisonings. Probably the earliest writer to take this view was Mauchart in 1712.[1] A dissertation with a like title was defended at the University of Altdorf in 1722 by Anhalt;[2] and a treatise upon the poisonous quality of copper was published by Falconer in 1774.[3]

In France during the past forty years the *Comité consultatif d'hygiène publique* upon three occasions[4] declared that copper in articles of food is dangerous, and the use of salts of copper or of copper vessels was forbidden by law. But Galippe in 1875[5] contended for the innocuousness of the copper compounds and later, by numerous experiments upon himself, his family and friends, showed conclusively that, even when taken for a long time and in quantity sufficient to be perceptible to the taste, they are absolutely without deleterious effects upon the human economy.[6] In 1880 the *Council of Hygiene*, consist-

[1] "Mors in olla, sive de cupreorum vasorum noxa," Ephem. N. Cur. Cent., i., obs. 13, p. 54, 1712.

[2] Schulze: Præses.

[3] "Observations and Experiments on the Poison of Copper," 12mo, London, 1774.

[4] Febr. 28th, 1853; Nov. 12th, 1860; July 15th, 1877.

[5] Thèse, Paris, 1875.

[6] C. rend. Ac. Sc., Paris, 1877, lxxxiv., 718; Arch. d. phys. norm. et path., 1877, 2 s., iv., 206; Ann. d'hyg., etc., 1878, 2 s., l., 426; c.

ing of Pasteur, Poggiale, and Brouardel, reported to the Prefect of Police that "copper in the amounts found by them in canned goods is not capable of injuring health."[1] In 1889 Grimaux reported to the Minister of the Interior the innocuousness of the cupric salts,[2] in consequence of which the interdiction of the use of copper in canned goods was removed;[3] yet copper compounds were still included among the noxious colors in a list attached to an ordinance in 1891.[4] The Belgian Academy of Medicine in 1885, in answer to a question from the Government, declared that "the compounds of copper are not only useless in articles of food, but are deleterious;" but the discussion which preceded the expression of this opinion, which has been published by Du Moulin,[5] showed that a minority, respectable alike in numbers and ability, entertained the contrary view, which prevailed later, as the same academy at a session June 26th, 1886, declared that: "1. Copper contained in food in the proportion in which it has been found is not dangerous. 2. The greening of canned goods with the salts of copper is absolutely harmless." Yet the compounds of copper were included among the forbidden colors in a ministerial regulation of June 17th, 1891.[6] In Germany and Austria the use of copper is forbidden in regulations of 1889 and 1890, and the International Congress of Food Analysts in 1891 declared that "the use of copper in canned goods should be forbidden."[7] But as early as 1855 Hoenerkopf[8] held the opinion that copper sulfate is not poisonous in the sense that it can cause death when taken in small quantity, a view which has been supported by Wald[9] and many other toxicologists down to the most recent German writer upon the subject, Tschirch.[10] That the constant absorption of copper by workers in that metal does not produce deleterious effects is abundantly shown by numerous observa-

rend. Soc. de biol., 1877 (1879), 6 s., iv., 70, 77, 229; Ann. d'hyg., etc., 1879, 3 s., ii., 159; 1880, 3 s., iii., 531; 1883, 3 s., ix., 122.

[1] Ann. d'hyg., etc., 1880, 3 s., iii., 193.

[2] Rec. d. trav. Com. consult. d'hyg. pub. de Fr., 1889 (1890), xix., 146.

[3] May 7th, 1889.

[4] Dec. 17th, 31st, 1890; June 17th, 1891.

[5] "La Toxicologie du Cuivre," Bruxelles, 1886. See also France méd., 1889, ii., 1481.

[6] Monit. Belge, 1891, 1774.

[7] Ztsch. f. Nahrungsmittelchemie, etc., 1891, 557.

[8] Vrtljschr. f. ger. Med., etc., 1855, viii., 212.

[9] "Gerichtl. Med.," Leipzig, 1858, i., 393.

[10] "Das Kupfer," Stuttgart, 1893.

tions,[1] although they become saturated with it to such an extent that their hair and gums became green, and their urine colors the ground green.

On the other hand, the copper salts are used in medicine as emetics and as mild escharotics, and cases of severe poisoning have unquestionably been caused by them. To sum up the matter, therefore, it may be said that while copper is not a poison in the sense that it is dangerous when taken in small quantity, it is one under the definition which we have adopted, in that it is capable of causing harm when absorbed in sufficient quantity.

Indeed, for practical purposes, copper must be considered as a normal constituent of the animal body. Its presence in the human subject has been frequently noted by toxicologists, and we have yet to examine a human liver which does not contain it. Its presence has been usually attributed to the presence of copper in *prepared* food articles: but Cloez has found it in the blood of a wild animal (a stag) in the proportion of 5.5 mgm. to the kilogram.[2] Church[3] has described a pigment containing seven per cent. of copper, *turacin*, obtained from certain species of wild birds; Fredericq[4] has described an albuminoid containing copper, *hæmocyanin*, from the blood of the octopus, which has also been found by Krukenberg[5] in cephalopods, crustaceans, and oysters.[6] Copper is much more widely distributed in nature, in small quantities, than has been supposed. It exists in traces in almost all mineral waters, in sea water, in cereals,[7] in unmanufactured cocoa, in coffee, in potatoes, in beans, in spinach, in salads, in different fruits and berries, and in many other vegetable substances.[8]

The copper compounds of toxicological interest are the sulfate, subacetate, and subcarbonate.

Cupric sulfate, *blue vitriol, blue stone, Cupri sulphas,*

[1] Houlés: Jour. d'hyg., Paris, 1879, iv., 160, 170. Buck: "Hygiene," N. Y., 1879, ii., 50, 51; Index Cat. Libr. Surg.-Gen., Wash., iii., 411.

[2] Bull. Soc. chim., Paris, 1877, xxvii., 196.

[3] Proc. roy. Soc., 1892, li., 399.

[4] C. rend. Ac. Sc., Paris, 1878, lxxxvii., 996.

[5] "Vergl. phys. Studien," iii., 1880, 76.

[6] See also Wackenroder: Arch. d. Ph., 1853, lxxi., 9, and Gamgee: "Physiol. Chem.," i., 132.

[7] Galippe (Ann. d'hyg., etc., 1883, ix., 128) found in wheat from 0.005 to 0.0108 gm. of copper to the kilogram; in bran, 0.014; in wheat flour, 0.0084; in rye, 0.005; in oats, 0.0084; in barley, 0.0108; and in rice, 0.0016.

[8] See Tschirch: *Op. cit.*, 3–11.

U. S. P.—$CuSO_4 + 5Aq.$—159+89.8—forms deep blue, odorless crystals, having a nauseous, metallic taste and an acid reaction in solution. Soluble in 2.6 parts of water at 15° (59° F.) and in 0.5 part of boiling water; insoluble in alcohol.

Verdigris is a mixture in varying proportions of three basic acetates: $2[Cu(C_2H_3O_2)_2]$, $CuO+6Aq.$; $Cu(C_2H_3O_2)_2$, $CuO+6Aq.$; and $Cu(C_2H_3O_2)_2$, $2CuO+2Aq.$ It is a blue or green crystalline powder. The neutral acetate, $Cu(C_2H_3O_2)_2+Aq.$, is in large green prisms, known as *Crystals of Venus*. The blue-green material formed upon copper when exposed to air is a basic carbonate, $CuCO_3,Cu(OH)_2$. A **basic chlorid**, $CuCl_2$, $3CuO$, used as a pigment under the name of *Brunswick green*, was the cause of death in one instance.[1]

Although Tardieu in his table of criminal poisonings in France, 1851–1871, gives the number by copper salts as 159 (exceeded only by arsenic and phosphorus), we can find but few cases of acute copper poisoning detailed in medical literature. In most of the older cases and in many of the more recent ones in which the cause of death was ascribed to copper in articles of food, the real cause was most probably the presence of ptomains or of trichinæ or bacteria. This is undoubtedly the case where the food was some variety of sausage,[2] where, as in several instances, the poisonings occurred at sea.[3]

In 89 alleged copper poisonings referred to in medical literature there are but 12 suicides, 6 by verdigris, all unsuccessful save 1, and 6 by cupric sulfate, all fatal. Concerning Tardieu's 159 criminal cases we can learn nothing further than the mere mention of their alleged occurrence, except in four instances. In 17 of the 89 cases above referred to, homicidal intent was alleged. Of these 10 are French cases, 1829–1874,[4]

[1] A child of two and a quarter years. Siebenhaar: Ztschr. f. Staatsarznk., 1844, xlvii., 188.

[2] Langenbeck: Dent. Klinik, Berlin, 1851, iii., 418. Paasch: Vrtljschr. f. ger. Med., etc., 1852, i., 79.

[3] Ramsay and Duval, in Wibmer: "Wirkung d. Arzneimitt.," etc., i., 244. Moore: Lancet, 1846, i., 404.

[4] Chevallier and Barruel: Ann. d'hyg., 1829, ii., 465. Fodéré: J. compl. d. dict. d. sc. méd., 1830, xxxviii, 408. Orfila: Bull. ac. de méd., 1838-39, iii., 93 (Aff. Rittinghausen). Chevallier and Lassaigne: Ann. d'hyg., 1848, xxxix., 408. Orfila: "Tox. gén.," 5ème ed., i., 820 (Aff. Dupuy, 1851). Poirier: J. d. chim. méd., etc., 1858, 4 s., iv., 19. Tardieu and Roussin: "Empois.," 2ème ed., p. 648 (Aff. Lapeyre, 1866). *Ibid.*, p. 652 (Aff. Lefévre and Desnos). *Ibid.*, p. 643. Bergeron and l'Hôte: J. d. chim. méd., etc., 1874, xlviii., 503 (Aff. Moreau, 1874).

and 5 are English, 1856–1886.[1] Of the two remaining cases one is an ancient German case reported at great length by Hitzig and Häring,[2] the other an unpublished case of a man in New York City who in 1888 attempted to poison himself and his wife and children with cupric sulfate.

The **lethal dose** of cupric sulfate is very differently stated by different writers from v. Hasselt,[3] who places it at 0.4 to 0.5 gm. (6–8 grs.), to Böcker[4] who states it as 35 to 70 gm. (1–2 "unzen"). The medicinal dose as an emetic is 0.7 to 1.0 gm. (10–15 grs.). Of cases of undoubted cupric poisoning that in which the smallest dose caused death was the one reported by Starr[5] in which a woman of 46 years died in 4 days and 5 hours from the effects of about an ounce (31 gm.) of copper sulfate, taken in tea with suicidal intent. On the other hand, in Kétli's case[6] 120 gm. (nearly 4 ℥) did not cause death.

The **duration** of fatal cases is usually rather prolonged, the average of 8 cases having been 52 hours. The shortest duration was in a child of 16 months that died in 4 hours after having swallowed a few pieces of cupric sulfate.[7] The shortest duration in an adult was in a female suicide of 42 years, who died in 10 hours.[8] Excluding 2 homicidal cases in which the duration, although stated as 19 and 20 days, is uncertain,[9] and another in which a child died in 12 days after having swallowed a half-penny,[10] the longest duration of a case of unquestionable cupric poisoning was in a female suicide of 16 years who died in 7 days.[11]

The **symptoms** of copper poisoning, like those due to zinc, mercury, etc., are partly local and referable to its action upon

[1] Reg. *v.* Smith, 1856, Taylor: "Poisons," 3d Am. ed., 433. Tully *v.* Corrie, 1866, Taylor: Guy's Hosp. Rep., 1866, 3 s., xii., 329; Ph. J. and Tr., 1870–71, 3 s., i., 874. Reg. *v.* Mary Baker, 1874. Taylor (Stevenson): "Med. Jur.," 11th Am. ed., 150. Reg. *v.* Reynolds, 1886, *ibid.*

[2] "Die Gräfin von Görlitz und ihr Diener," Neue Pitaval, xvii., 143–358.

[3] "Handb. d. Giftlehre," 1862, ii., 273.

[4] "Lehrb. d. ger. Med.," 1857, p. 243.

[5] Med. Rec., N. Y., 1882, xxi., 564.

[6] Pest. med.-ch. Presse, 1883, xix., 310.

[7] Sonnenschein: "Handb. d. ger. Chem.," p. 82.

[8] Maschka: Vrtljschr. f. ger. Med., etc., 1883, n. F., xxxix., 55.

[9] Tully *v.* Corrie and Aff. Dupuy.

[10] Corrigan: Dublin Hosp. Gaz., 1854, n. s., i., 229.

[11] Maschka: Wien. med. Wochenschr., 1871, 628.

the alimentary canal, and partly those of true poisoning. The taste would be observed by an adult in the possession of his faculties. Epigastric pain, extending to the abdomen, is an early symptom, and is accompanied by repeated vomiting of greenish matters, which are turned blue on addition of ammonium hydroxid. There is purging, the stools being liquid, brown in color and not bloody. Cramps in the legs occur early, and are frequently very severe. The pulse in the earlier stages is full and strong, soon becoming more feeble, smaller, and more rapid. The respiration is normal. The patient becomes very weak and suffers from frontal headache. If the case be prolonged, there is a decided improvement in the patient's condition, which has also been observed in experiments upon animals. Then there is hypogastric pain, frequent, scanty micturition, accompanied by urethral pain, and with diminution in the quantity of urine. The urine passed is dark in color, almost black, cloudy, acid, contains a large quantity of albumin, deposits a sediment consisting of granular material and epithelial casts, but no blood corpuscles, and shows the oxyhæmoglobin bands before the spectroscope. Later there is jaundice, and the pain is more marked in the hepatic region. The patient becomes weaker and weaker and more somnolent, the abdomen tender to pressure throughout, the jaundice increases, and toward the end the intellectual capacity, which has hitherto remained unimpaired, becomes affected; the power of co-ordination is diminished, as well as the sensibility, and a condition of semi-coma is established; the pupils become contracted and insensible to light, and death follows in coma and complete paralysis.[1]

The **treatment** should consist in washing out the stomach with water containing potassium ferrocyanid (to produce the brown, insoluble cupric ferrocyanid) and in the administration of stimulants, hypodermically if necessary, and of nutritive enemata.

The **post-mortem appearances** are not characteristic except in one point, if it be present: the mucous surface of the alimentary canal and its contents may be greenish or distinctly green, and the color changes to blue on the addition of aqua ammoniæ. A greenish tinge may be due to bile, but it does not turn blue on addition of ammonia. Besides this the entire ali-

[1] For a detailed account of a typical case see Starr: *Loc. cit.*

mentary mucous membrane, from mouth to anus, may be reddened, swollen, ecchymosed, and even ulcerated—indeed, the rectum has been said to have been perforated. If the case have been one of long duration the liver is found to be fatty and the kidneys are the seat of parenchymatous nephritis. The skin may be icteric, and putrefaction is apparently delayed.

Analysis.—The detection of copper presents no difficulties; but the interpretation of the results requires caution. As we have said, copper is so frequently present in the human body that it must be considered as innocent, if not normal, unless the quantity be excessive. On the other hand, an inevitable effect of large doses of copper salts is to provoke frequent vomiting, by which the major part of a large dose taken into the stomach is expelled. An analysis of the vomited matters is therefore of prime importance. Of absorbed copper the largest amount is found in the liver, whether it be "normal" or "toxic." Raoult and Breton[1] found 0.005 gm. copper per kilogram of "normal" copper in one liver, and 0.015 gm. in another. Bergeron and l'Hôte[2] found 0.001 and 0.003 gm. copper in two "normal" livers. After medicinal treatment with cupric salts the quantity in the liver increases greatly. Thus Bourneville and Yvon[3] found as much as 0.295 gm. of copper in the liver of an epileptic woman who had taken 43 gm. of cupric sulfate in 4 months. In the Affaire Moreau, in which the livers of the two women supposed to have been poisoned were found to contain 0.12 and 0.08 gm. of copper respectively,[4] the inference drawn from the quantity should have been that this quantity might be accounted for by medicinal or criminal administration, but not by ordinary "physiological" absorption.

In the systematic method of analysis (see page 157) copper, if present, is found for the most part in the extract IV., partly in the insoluble residue V.[5] That portion in the extract is separated as cupric oxid in the subsequent fusion, and separated from antimony and tin in the manner described on pages 159, 317,

[1] J. de Ph. et de chim., 1878, 4 s., xxv., 118.

[2] *Ibid.*, 1875, 4 s., xxi., 252.

[3] *Ibid.*, p. 236.

[4] Bergeron and l'Hôte: *Loc. cit.*

[5] In ordinary analysis this division of the copper may be obviated, in the absence of mercury, by substituting sodium sulfid for ammonium sulfid, but in toxicological analysis this is not permissible, as the object is not fully attained, and the separation of mercury is interfered with.

318, 323. That portion remaining in the residue V. (see page 158) is separated from mercury and bismuth by solution in nitric acid, and dilution with water as directed on page 513. The solution, which may contain lead and cadmium, besides copper, is treated with excess of sulfuric acid, heated until white fumes begin to be given off, cooled, treated with water, and filtered immediately. If lead be present it remains upon the filter as the white lead sulfate. The filtrate may contain cadmium and copper. These are to be reprecipitated by hydrogen sulfid, and the sulfids boiled with dilute sulfuric acid (1 : 5), allowed to stand a short time, and filtered. The cupric sulfid remains undissolved, while the cadmium passes into solution. The black copper sulfid is dissolved in hot dilute nitric acid, sulfuric acid is added to the solution, which is evaporated to near dryness, the residue is heated to expulsion of nitric acid, and dissolved in water. The solution is transferred to a weighed platinum capsule and a bar of zinc is added. When the liquid has become colorless and a drop on a white surface gives no color with potassium ferrocyanid the liquid and remaining zinc are removed, the copper, adherent to the platinum, is washed with water, alcohol, and ether in turn, dried in the desiccator, and weighed.

The detection of copper in articles of food, etc., may be readily effected by acidulation with dilute sulfuric acid and introduction of a plate of bright iron, when the copper is deposited upon the iron. Or with pickles, a knitting needle stuck into the pickle becomes similarly coated.

Tests for Copper.—The identification is usually sufficiently complete from the formation and behavior of the black sulfid, and the appearance of the deposited metal. Other tests are: 1. *Potassium* or *sodium hydroxid* produces a pale blue precipitate, insoluble in excess of the precipitant (except in the presence of certain organic substances, such as tartaric acid, glucose, etc.), which turns black either on prolonged standing or on heating the liquid. 2. *Ammonium hydroxid* produces a similar blue precipitate which readily dissolves, however, in excess of the precipitant, forming a deep blue solution. 3. *Potassium ferrocyanid* produces in moderately dilute acid solutions a red-brown precipitate, in very dilute solutions a brown color. 4. Cupric salts produce a yellow color with potassium

iodid, or a blue color with potassium iodid and starch paste. This reaction, which is very delicate, is not available in the presence of other substances which liberate iodin from potassium iodid.

GOLD.

Gold compounds cannot be considered as toxic agents of forensic interest. We can find record of but one poisoning by the terchlorid: an accidental poisoning of a child which took about 12 grs. (0.78 gm.) of the crystallized chlorid and recovered.[1]

IRON.

Although the compounds of iron are not usually considered as poisonous, certain of them are distinctly toxic. Indeed, in a case of alleged arsenical poisoning the question was very properly raised whether death was due to the arsenic or to Monsel's solution given in mistake as an antidote.[2]

The iron compounds which have been of forensic interest are the chlorid and the sulfate:

Ferric chlorid — *sesquichlorid* — *perchlorid* — Fe_2Cl_6 — 323.98—is, as usually met with, a yellow, amorphous solid, acid in reaction and very soluble in water. It is used medicinally in aqueous (Liq. ferri chloridi, U. S. P.) and in alcoholic solution (Tinct. ferri chloridi, U. S. P.). Both preparations contain an excess of free acid.

Ferrous sulfate—*green vitriol*—*copperas*—*Ferri sulphas, U. S. P.*—$FeSO_4 + 7Aq.$—151.66+125.72—is in large, pale green, oblique rhombic prisms, odorless, having a salty styptic taste; soluble in 1.8 parts of water at 15° (59° F.) and in 0.3 part of boiling water, insoluble in alcohol, which precipitates it from its watery solutions (Ferri sulphas præcipitatus, U. S. P.). When exposed to air it effloresces and becomes oxidized. Its solutions also become brown and deposit by oxidation. When heated to 149° (300° F.) it loses 6Aq. (Ferri sulphas exsiccatus, U. S. P.) forming a grayish-white powder.

We find reference in medical literature to 10 non-medicinal

[1] Stevenson: Guy's Hosp. Rep. (1893), 1894, i., 127

[2] Ranke: Friedreich's Bl. f. ger. Med., 1881, xxxii., 273.

cases of poisoning by ferric chlorid, of which 2 were homicidal;[1] and to 14 poisonings by the sulfates, of which 8 were homicidal.[2]

The **symptoms** produced by ferrous sulfate are more clearly attributable to the action of the iron than those produced by the chlorid, which is usually taken in the form of tincture (see below). The local action is soon manifested in vomiting of greenish material; pain, at first epigastric, subsequently abdominal, and most marked over the sigmoid flexure; and purging of stools which in some instances have been liquid, greenish, and odorless (Hall), in others hard and black (Nistrand). The phenomena of gastro-enteric irritation are due in part to the direct local action of the acid salt, but in great part also to its absorption and elimination, partly by the bile, and partly by the enteric mucous membrane.[3] The systemic action is manifested in disturbances of the special senses, partial blindness and deafness, vertigo, partial paralysis of the lower extremities, weak and irregular pulse, and low temperature. The salt is rapidly absorbed from the intestine, but is not so rapidly eliminated, appearing only in small amount in the urine and

[1] Murray: Dublin M. Pr., 1849, xxi., 113. Combe, Christison: "Poisons," Am. ed., 393. Hiens. M. and S. Reptr., Phila., 1869, xx., 366. Herrick: Penins. J. M., Detroit, 1874, x., 345. Bérenger, Férand, and Porte: Ann. d'hyg., etc., 1879, 3 s., i., 312, 508 (H.). Ravaglia: Bull. soc. m. d. Bologna, 1884, 6. s., xiii., 361 (H.). Sternberg: Közeg-és Törvény, Orvos, Budapest, 1886, 53. Aymot: Prov. M. and S. J., 1847, April 7. Taylor: "Pr. and Pr. of Med. Jur.," i., 320 (abortive). Warburton: Lancet, 1869, i., 9 (S.).

[2] Mag. f. d. ges. Heilk., 1826, xxi., 247. Christison: *Loc. cit.* (two cases, one H., one H.?). Chevallier: J. d. chim. méd., etc., 1847, 3 s., iii., 78 (H.). Chevallier and Lesueur: Ann. d'hyg., 1850, xliii., 1850 (H.). *Ibid.*, p. 419 (H.). Limouzin-Lamothe: J. d. chim. méd., 1850, 3 s., vi., 380 (H.) [same case. Chevallier: Ann. d'hyg., 1851, xlv., 154]. J. d. chim. méd., etc., 1858, 4. s., iv., 24 (H). Wistrand: Ztschr. f. d. Staatsarznk, 1864, lxxxvii., 285 (2 H). Hall: N. Y. M. Jour., 1883, xxxviii., 401. Busey: Tr. Wash. Obstet. and Gyn. Soc., 1885–87, i., 123. Fitts: Atlanta M. and S. Jour., 1888–89, n. s., v., 198.

[3] Novi: Arch. ital. d. biol., Turin, 1890, xiii., 242. Anselm: "Arb. d. pharm. Inst.," Dorpat., 1892, viii., 51. Kobert: *Ibid.*, 1891, vii., 123 (a summary of four valuable papers by Damaskin, Kumberg, Busch, and Stender, *ibid.*). For other papers on the absorption, action, and elimination of iron see Bunge: Zts. f. physiol. Chem., 1892, xvi., 78, 173; 1893, xvi., 63. Gottlieb: *Ibid.*, 1891, xv., 371. Mörner: *Ibid.*, 1893, xviii., 13. Socin: *Ibid.*, 1891, xv., 63. Samojloff: "Arbeit.," Dorpat, 1893, ix., 1. Lipski: *Ibid.*, 62. Meyer and Williams: Arch. f. exp. Path. u. Ph., 1881, xiii., 70. Jacobi: *Ibid.*, 1891, xxviii., 256. Schmul: Diss., Dorpat, 1891.

remaining in the system for weeks.[1] Death is considered by Franzolini and Baldisera[2] to be due to paralysis of respiration.

The symptoms caused by ferric chlorid in the form in which it has been taken in the recorded cases, that of the tincture, are partly referable to the action of the metal, and partly to that of the ethyl chlorid and excess of free hydrochloric acid which the tincture always contains. Indeed, in a case observed by Combe[3] a man who took half an ounce of the tincture in mistake for whiskey recovered from the primary effects, but died in six weeks from inanition due to pyloric stenosis, in the same way as death is frequently caused by hydrochloric acid itself. The tincture has also caused death indirectly, when used as an hæmostatic in the puerperal uterus, the fatal result occurring suddenly from penetration of thrombi and air into the veins, or from peritonitis caused by passage of the solution itself through the tubes into the peritoneal cavity.[4] The death of a child of 9 months in 5 minutes from injection of tinct. ferr. chlor. into a nævus is reported by Kesteven.[5] Of the four Martinique victims, murdered by tincture of the chlorid, administered in an alcoholic liquid on an empty stomach[6] one complained in half an hour of weight in the stomach and severe pain in the left arm; in 7 hours he was found groaning, unable to move, unconscious, having vomited and purged abundantly, and died 13 hours after having taken the poison. Nothing is said of the micturition in the history of this case, but the bladder was found empty at the autopsy. In two of the others, who died in 65 hours, there were suppression of urine and violent abdominal pains. The fourth, or rather first, victim had died two years before the inquiry, and the history of the symptoms is not given.

The **post-mortem appearances** observed in the Martinique case were a hyperæmic condition of the peritoneum, liver, kidneys, and nerve centres; an anæmic condition of the alimentary canal, which was pale and decolorized throughout; and the presence of a black material on the tongue, œsophagus, stomach,

[1] See Gottlied, Stender, Schmul: *Op. cit.*

[2] Ann. univ. d. Med. e Ch., Milano, 1882, cclxi., 79.

[3] Christison: "Poisons," Am. ed., Phila., p. 393.

[4] Lewin: "Nebenwirkung d. Arzneimitt.," 2te Aufl., 433; Am. J. M. Sc., 1870, 506.

[5] Lancet, 1874, i., 195.

[6] Bérenger, Férand and Porte: *Loc. cit.*

and intestine, which not only colored the part but penetrated either by absorption or imbibition into neighboring organs.

Analysis.—In the systematic process iron, if present in notable amount, will be found in the residue X. (see page 160). It has been demonstrated by Damaskin,[1] however, that the method of disorganization by hydrochloric and chloric acid does not convert all of the iron present in urine into a modification precipitable by ammonium sulfid, from 23 to 79 per cent. remaining unprecipitated. When the presence of iron is suspected the method by desiccation and incineration recommended by Damaskin should be followed.

Iron is not only extensively used as a medicine, but is also an essential constituent of the blood-coloring matter, which contains it in the proportion of 0.33 to 0.59 per cent., and is also normally eliminated by the urine in some form of organic combination, probably in the coloring matter. According to Gottlieb[2] the average daily elimination of iron by the healthy human urine is 2.59 mgm.

Tests for Iron—*Ferrous.*—The ferrous compounds are usually pale green in color and readily oxidize to ferric. 1. With ammonium sulfid: in solutions containing no free acid a black precipitate. 2. Potassium, sodium, or ammonium hydroxid: an almost white precipitate which rapidly turns green and brown. 3. Potassium ferricyanid: a deep blue precipitate. 4. Potassium ferrocyanid: a bluish-white precipitate, subsequently turning blue.

Ferric compounds are yellow or brown in color. 1. Hydrogen sulfid: a milky white cloudiness or precipitate of elementary sulfur in acid solution. 2. Ammonium sulfid: a black precipitate in solutions containing no free acid. 3. Potassium, sodium, or ammonium hydroxid: a brown, gelatinous precipitate. 4. Potassium ferrocyanid: a deep blue precipitate. 5. Ammonium thiocyanate: a deep red color.

[1] "Arbeiten d. pharm. Inst.," Dorpat., 1891, vii., 54.

[2] *Loc. cit.*

LEAD.

ACUTE POISONING.

Although it appears from the statistical table on page 56 that lead poisoning is more frequently referred to in medical literature than that by any other toxic agent, these poisonings are for the most part chronic and industrial, and although the subject of lead poisoning is one of great medical and hygienic interest, it is one of very slight forensic importance, as acute lead poisonings are of very rare occurrence and almost always terminate in recovery. Homicidal poisonings by lead compounds are of most exceptional occurrence. In Tardieu's table of criminal poisonings in France, 1851–63, there is but 1 case of poisoning by lead acetate (1854), while Hugounenq[1] states that but 9 cases of criminal lead poisoning occurred in France in 60 years. We find mention in medical literature of but 12 cases of alleged homicidal lead poisoning, of which 8 were in England,[2] 1 in Germany,[3] 1 in Austria,[4] 1 in France,[5] and 1 in Algeria;[6] in all but 1 of which lead was alleged to have been administered as acetate or subacetate.

Lead Acetate—*sugar of lead—salt of Saturn—Plumbi acetas, U. S. P.*—$Pb(C_2H_3O_2)_2 + 3Aq.$—324.11 + 53.88—crystallizes in large, oblique rhombic prisms, sweetish with a metallic after-taste; soluble in 1.8 parts of water at 15° (59° F.), in 0.5 part of boiling water, or in 8 parts of alcohol at 15° (59° F.). Its solutions are acid. It effloresces in air and is superficially converted into carbonate.

Sexbasic lead acetate—$Pb(C_2H_3O_2)OH,2PbO$—726.91—

[1] "Poisons," Paris, 1891, p. 89.

[2] Christison: "Poisons," Am. ed., 396 (1827). Taylor: "Poisons," 3d Am. ed., 404, 405 (Reg. *v.* Edwards, 1844; Reg. *v.* Hume, 1847). London M. Gaz., 1850, n. s., x., 476 (Reg. *v.* Bridget Mann, 1850). Ph. J. and Tr., 1859-60, n. s., i., 342 (Reg. *v.* Keefe and Turner, 1859). Lancet, 1882, ii., 1087 (Reg. *v.* Louisa Jane Taylor, 1882). In the J. de chim. méd., etc., 1849, 2 s., x., 82, is a mention of an English case of Jos. Taylor, and in the Ph. J. and Tr., 1858–59, xviii., 293, the commitment of Ann and Chas. Taylor on a charge of murder by lead carbonate is noticed.

[3] Schniewind: Vrtljschr. f. ger. Med., 1862, xxi., 277.

[4] Maschka: Wien. med. Wochenschr., 1871, xxi., 312.

[5] Flandin: "Poisons," ii., 311. Orfila: "Tox. gén.," 5ème ed., i., 886, 896 (Aff. Pouchon, 1843).

[6] J. de chim. méd., etc., 1849, 2 s., x., 385 (Aff. Merle, 1832).

is the main constituent of *Goulard's solution=Liq. plumbi subacetatis, U. S. P.*, which is a colorless liquid, sp. gr. 1.228, of a sweetish, astringent taste, containing about 25 per cent. of the subacetate. It becomes milky on exposure to air from formation of the carbonate. If made with ordinary water, containing carbonates and sulfates it is milky (*eau blanche*).

White lead—*Plumbi carbonas, U. S. P.*—is a basic carbonate, usually of the composition $(PbCO_3)_2$, PbH_2O_2, which is very extensively used in oil painting and to a limited extent, mixed with lard, as an ointment. Although it is one of the most prolific sources of chronic lead poisoning it is not sufficiently soluble to provoke serious acute effects. Snow[1] reports the death of a child of 5 years 90 hours after swallowing a piece of white lead of the size of a small marble, ground in oil, in which lead poisoning was at least a contributory cause of death. This and the homicidal case referred to in a previous note are the only fatal cases of which we find record. In two other instances adults suffered the symptoms of poisoning from doses of 20–23[2] and 23–31[3] gm. (5–6 and 6–8 ʒ) but recovered; and Christison[4] cites two cases in which 30 and 45 gm. (1 and 1½ ℥) were taken by adults without producing any effects whatever.

The **lethal dose** of either of the above compounds of lead is not known, certainly it is not small, as cases are reported in which 30 gm. (1 ℥) of the acetate were swallowed without causing death,[5] and one case[6] of a woman who took three-quarters of a pint of the solution of the subacetate, containing about 110 gm. (3½ ℥) of the subacetate, and recovered. In a careful search of medical literature we have found record of but 23 alleged deaths from acute lead poisoning. Of these 9 were supposed homicides, in which nothing is known with certainty as to the doses, except that in 2 cases the administration was by repeated doses to an aged woman,[7] and to a man of 69 years.[8]

[1] Lancet, ii., 1844, 144.
[2] Schubert: Wochenschr. f. d. ges. Heilk., 1844, 588.
[3] Cross: London Med. Times, 1839, i., 786.
[4] *Op. cit.*, 428.
[5] Gorringe: Prov. M. and S. Jour., 1846, 181 (two cases). Leared: Med. Times and Gaz., 1858, i., 296.
[6] Aldis, *Ibid.*, 1860, i., 34.
[7] Lancet, 1882, ii., 1087.
[8] Maschka: "Samml. Gutacht. Prag. med. Fak.," 1873, 4 F., 253. In an English trial for attempted murder the extraordinary claim appears to have been set up by the prosecution that 26½ grains (1.7 gm.) of the acetate were sufficient to destroy the life of an adult.

Of the 14 non-homicidal cases 7 were in adults. Of these 2 are early cases, 1 of a man who died on the third day after taking an unstated quantity of subacetate solution;[1] the other that of a woman of 40 years who is said to have died in 8 months (!) from the effects of ℥ iij. (11.7 gm.) of the acetate.[2] A somewhat more modern case was that of a woman who is said to have died in 8 days after having accidentally swallowed an unstated quantity of the same salt.[3] Two instances of death in females who took lead carbonate to cause abortion are reported: one took 45 gm. (nearly ℥ iiss.).[4] Another early instance was the poisoning of a family of 9 persons by lead carbonate accidentally mixed with sugar: 2 adults and 2 children died, the former in 3 and 4 months, the latter in 24 hours and 2 weeks.[5] Freyer's case is therefore the only recent one in which the dose is known. No dose is mentioned in 2 of the 5 other cases in children; one is that by the carbonate already referred to; another that of an infant of 8 days said to have died from the effects of "Eau de M. Delacour," which had been applied to the nurse's breast.[6] The remaining case was also in an infant who died in 2 days from the effects of *eau blanche* given internally by misadventure by an ignorant person. The dose stated (apparently upon the evidence of the person who gave it) was two coffee-spoonfuls.[7] This quantity, about 8 c.c., would contain about 2.5 gms., or 38 grs. of the subacetate.

Symptoms.—Apart from the sweetish and somewhat metallic taste of soluble lead compounds, which would be observed at the time of ingestion, a large dose of acetate or subacetate would cause vomiting within a few minutes, certainly within half an hour, unless the poison were taken into a full stomach or by a person under the influence of alcohol, when it might be delayed for an hour or two. The vomited matters are white or tinged with blood, and vomiting is persistent and furthered by the liquids which thirst prompts the patient to take. There is also colic-like paroxysmal abdominal pain, diminished by pres-

[1] Kerchhoff: Ἱπποκρατης, Rotterdam, 1817, iii., 143. Orfila: "Tox. gén.," 5ème ed., i., 838.

[2] Lancet, 1828–29, ii., 234.

[3] Boston M. and S. Jour., 1852, xlvi., 74.

[4] Freyer: Ztschr. f. med. Beamte, 1888, i., 231. The dose is not stated in the other. Casper: Wochenschr. f. d. ges. Hlk., 1838, 457.

[5] Deering: Tr. M. Soc., London, 1810, i., Pt. i., 64.

[6] Bouchut: Gaz. d. hôp., 1873, xlvi., 5.

[7] Aigre and Planchon: Ann. d'hyg., etc., 1890, 3 s., xxiv., 444.

sure. Purging has occurred, although sometimes there is constipation. The stools may be dark or black from formation of lead sulfid. The tongue is coated, and the breath very fœtid, and a sweetish metallic taste is perceived, even late in the history. Later there are insomnia, great depression, muscular cramps, vertigo, headache, and in some cases a violent return of the vomiting and colicky attacks of pain, which had ceased. The symptoms produced by repeated administration of soluble lead compounds appear to be the same as those caused by larger doses, with exacerbations after each administration, and without the development of the effects of truly chronic lead poisoning. Strength diminishes gradually, and death follows the last administration rapidly.

The **treatment** in acute lead poisoning should consist in the administration of a sulfate, either sodium sulfate (Glauber's salt) or magnesium sulfate (Epsom salt), with the object of forming the insoluble lead sulfate, and in washing out the stomach. Milk or white of egg may be given while awaiting the chemical antidote, or an emetic in the absence of the pump or siphon. Pain should be controlled by opiates. In the subsequent treatment the elimination of absorbed lead may be accelerated by the administration of iodids, and the diet should be fluid and non-irritating for some time.

The **post-mortem appearances** have not been carefully observed in the human subject. In Maschka's case the autopsy was made twenty days after death and showed no characteristic lesion. Notwithstanding the time which had elapsed the stomach was uninjured, the mucous membrane somewhat loosened, but otherwise entirely normal, without signs of inflammation and without ecchymoses or erosions. The mucus membrane of the intestine was also normal. This case, although homicidal, was unquestionably one of acute or subacute lead poisoning, probably by repeated administrations of lead acetate, the last one not long before death. In Aigre and Planchon's case, in which an infant was destroyed by a solution of the subacetate and the autopsy was made the second day after death, the only notable lesion was in the stomach, which was found to be so greatly softened that it ruptured in the attempt to remove it. The analysis in this case showed the presence of a greater quantity of acetic acid than would correspond to the

amount of lead found and to the composition of extract of Saturn of the codex. The authors advance the supposition that the acetate is immediately decomposed on contact with the food in the stomach, forming solid insoluble compounds, which contain the whole of the metal and which are rapidly discharged by the dejecta, and that the liberated acetic acid accumulates in the stomach by imbibition of its tissues. In two children which died in 36 hours from the effects of the subacetate Taylor[1] found the mucous membranes of the stomachs of a gray color, but otherwise perfectly healthy. The intestines were much contracted. Taylor states that "the appearances observed in cases of acute poisoning by lead are very characteristic,"[2] but does not give facts sufficient to warrant the statement.

Analytical.—In the systematic analysis, if lead be not present in excessive amount it will pass entirely into the hot filtrate obtained after the attack by hydrochloric acid and chlorate (see page 154). It exists at this stage as lead chlorid, a salt which, although not readily soluble in cold water and still less so in the presence of about the proportion of hydrochloric acid here present,[3] is much more soluble in hot water. Consequently, if lead be present in notable quantity the filtrate will, on cooling, deposit crystals of lead chlorid, which may be separated by filtration. In the subsequent treatment the lead remaining in solution is found as sulfid in the residue insoluble in ammonium sulfid (IV., page 159), from which it is separated as sulfate in the manner already decribed (see pages 513, 532).

If a *quantitative determination* be desired the dried precipitate from the two filters (chlorid and sulfate) are detached from the filters as far as possible and collected on a watch glass. The filters are burnt in a weighed porcelain crucible, the contents of the watch glass are added to the cooled crucible, then sulfuric acid; and the contents of the crucible are gradually heated until white fumes cease to be given off. The cooled crucible and its contents are then weighed. The last weight, minus that of the

[1] "Poisons," 3d Am. ed., pp. 394, 404. On the latter page Taylor refers to a case of Orfila's, which we do not find, however, either in the 1st, 3d, or 5th edition. Kerchoff's case, to which Taylor also refers, occurred in 1817.

[2] *Op. cit.*, p. 404.

[3] Water at 165° (62° F.) dissolves 0.952 per cent. Water containing 1 to 10 per cent. HCl, dissolves 0.347 to 0.093 per cent. (Bell: Chem. News, xvi., 69).

crucible and filter ash, is that of the lead sulfate present. This weight, multiplied by 0.68317, is the weight of metallic lead. A portion or all of the sulfate may be redissolved by heating to about 70° (158° F.) with acid ammonium acetate solution,[1] and the solution so obtained used for further qualitative reactions.

Tests for Lead—1. *Hydrogen sulfid* or *ammonium sulfid* produces a black precipitate of lead sulfid, insoluble in cold dilute acids, soluble in hot nitric acid. 2. Dilute *sulfuric acid* produces a white precipitate of lead sulfate. 3. *Potassium chromate* produces a yellow precipitate of lead chromate (chrome yellow) soluble in potash or soda. 4. *Potassium iodid* produces a bright-yellow precipitate of lead iodid, readily soluble in potassium hydroxid, insoluble in potassium iodid, soluble in concentrated hydrochloric or nitric acid. 5. *Potassium*, *sodium*, and *ammonium hydroxids* produce white precipitates, soluble in excess of the first two, but insoluble in excess of the last. 6. *Hydrochloric acid* produces a white precipitate in concentrated solutions. The precipitate, if not too abundant, dissolves on heating the liquid, and is deposited in crystals on cooling.

MERCURY.

Modern experience from use of mercury in the form of corrosive sublimate in surgical and particularly in obstetrical practice shows that it is not only an extremely efficient germicide, but that it may be fatal to the human host when absorbed in very minute quantity. If the magnitude of the dose capable of causing death be the measure of "deadliness" of a poison, mercury is more deadly than arsenic. In a case reported by Saumer[2] a vigorous, healthy woman of 21 years died in collapse after having manifested all the symptoms of mercurial poisoning, and after death the intestines presented the lesions usually produced by corrosive sublimate. She had been delivered normally and had progressed well until the sixth day, when she had a slight chill and a temperature of 40°.5 (104°.9 F.). Four litres of a solution of corrosive sublimate 1:4,000 were used, as

[1] Made by adding acetic acid to concentrated ammonium hydroxid solution until the liquid has a faintly acid reaction.

[2] Charité Ann., 1888, xiii., 737.

a precautionary measure, in vaginal and uterine irrigation, the liquid flowing away freely. In this case the total quantity of mercuric salt injected was only 1 gm. (15.4 grs.), and as the solution was only in momentary contact with the parts the amount absorbed could only have been a minute fraction of the amount injected.

All the compounds of mercury, as well as the metal itself, are actively poisonous if absorbed.

Metallic mercury—*hydrargyrum*, **Hg**—199.8.—Metallic mercury in the liquid form can hardly be considered as poisonous. The older medical literature contains numerous references to individuals who have swallowed a pound or two of the liquid metal for the cure of obstinate constipation or intestinal obstruction, without suffering any evil consequences other than, in some cases, salivation and soreness of the mouth. In one instance an individual took two pounds daily for a long time with the view of removing a silver coin which had lodged in the œsophagus.[1] In one of two cases cited by Taylor,[2] the metal remained in the alimentary canal eight days, when it was discharged, partly in the metallic form and partly as black oxid; in the other[3] the mercury was retained in the body for nine days and the last of it only passed on the fourteenth day. In this instance there was slight salivation.

The notion that liquid mercury is poisonous is, however, popularly entertained, and a few cases of attempted murder by its administration have occurred in European countries.[4] In India it is said by Brown[5] to be frequently given to cause injury. In England a girl took four and a half ounces of liquid mercury as an abortive, and suffered from mercurial tremors, but did not abort.[6]

Metallic mercury enters into the composition of five pharmacopœial preparations, in the manufacture of which, how-

[1] Sue: Mém. Soc. méd. d'emulat., iv., 252, quoted by Wibmer: "Wirk. d. Arznmitt. u. Gifte," iii., 77, where other early cases are cited. (We do not find the case quoted from Abano in the ed., Padua, 1473.)

[2] "Poisons," 3d Am. ed., 351, Kerstein.

[3] *Loc. cit.*, from Wochenschr. f. d. ges. Heilk., 1845, 249.

[4] Friedreich's Bl. f. ger. Anthr., etc., 1858, ix., 5 Heft., 61, 64; "Samml. Gutacht. Prag. med. Fak.," 1858, ii., 215. Möhsen: Repert. d. ger. u. öff. Arzneyw., 1789, i., 248. (In the last a number of earlier observations are referred to.)

[5] Med. Rept. Bengal Pres., 1869, 152.

[6] Gibb: Lancet, 1873, i., 339.

ever, the metal is more or less oxidized. Of these, three are intended for external application: *Emplastrum hydrargyri*, and *Emplastrum ammoniaci cum hydrargyri*, plasters of no toxic interest; and *Unguentum hydrargyri=blue ointment*, which contains 50 per cent. of mercury, triturated with lard and suet until the metallic globules are not visible with a magnifying power of ten diameters. A somewhat similar combination in which vaselin is used in place of animal fat is used as a bedbug poison. A combination of mercury (30 per cent.) with lanolin and olive oil, similarly prepared by trituration, is the most recent form of an oily remedy applied by hypodermic injection, under the name *oleum cinereum*.[1] Two pharmacopœial preparations are intended for internal administration: *Hydrargyrum cum creta=mercury with chalk*, containing 38 per cent. of mercury "extinguished" with chalk and sugar of milk; and *Massa hydrargyri=blue mass*, or *blue pill*, containing, when first prepared, 33 per cent. of mercury, with a quantity of mercuric and mercurous oxids, increasing with the age of the preparation.[2]

In a few instances death has been caused by a too liberal external application of blue ointment,[3] and, although it has only been in use since 1887, *oleum cinereum* has been the cause of several accidental poisonings, and of at least two deaths.[4]

Mercury with chalk is said to have caused the death of a girl of 5 years in 8 days after administration of 3 grs. (0.2 gm.) daily for 3 days.[5] And in another instance a boy of 11 years is said to have died in 4 days, after having taken 3 doses, 2 of 6 grs. (0.4 gm.) each, and the third of 4 grs. (0.26 gm.) during 6 days.[6] In both cases there were salivation and extensive sloughing of the cheeks. In the absence of any mention of

[1] Pharm. Jahrb., 1887, 484; 1888, 412; 1889, 457–58.

[2] Senier (Ph. J. and Tr., 1876, 3 s., vi., 621) found in pills two years old 24.4 per cent. metallic Hg, 1.8 per cent. HgO, and 4.22 per cent. Hg_2O.

[3] Lancet, 1845, ii., 108. Letaux: Thèse, Paris, No. 194, 1893 (complicated with lead poisoning); Ph. J. and Tr., 1868, 2 s., ix., 395; Tr. Assoc. King's and Queen's Coll. Phys. Irl., Dubl., 1824, iv., 91. Sackow: Berl. kl. Wochenschr., 1892, xxix., 618. Lowe: Brit. M. J., 1882, i., 1881. Braus: Deut. med. Wochenschr., 1887, xiii., 593. See also Lamberg Upsala läk. Förh., 1867–68, iii., 681, and mercuric sulfid below.

[4] Kaposi: Verh. d. deut. derm. Gesellsch., 1889, i., 319 (five cases, 1 D.). Lang: "Aerztl. Ber. allg. k. k. Krankenh.," Wien, 1891, 123. Klein: Deut. med. Wochenschr., 1893, xix., 745.

[5] Taylor: "Poisons," 3d Am. ed., 357.

[6] Harper: Lancet, 1851, ii., 579.

symptoms of mercurial poisoning beyond those of salivation, mercury in these cases can only be considered as an indirect cause of death. An old sample of mercury with chalk has been found to contain a large proportion of mercuric oxid.[1] *Blue pill* is said to have been instrumental in causing death in 4 instances. In one a rural practitioner in England in 1838 ascribed a death to 18 grs. (1.2 gm.) taken in divided doses during 3 days.[2] In another a woman is said to have died from "excessive doses of blue pill."[3] In the third a man brought an action against an apothecary for injuries alleged to have been sustained by taking blue pills prescribed by the defendant.[4] A child of 5 years is said to have died from maxillary gangrene in 8 days after the administration of 4 blue pills and a dose of calomel during 24 hours.[5]

Black wash, containing calomel and mercurous hydroxid, has caused ptyalism in 2 cases.[6]

Mercuric oxid—HgO—215.76—is used in medicine in two different physical modifications: the *yellow oxid; Hydrargyri oxidum flavum, U. S. P.*, a yellow impalpable powder, obtained by precipitation of a solution of mercuric chlorid with potassium hydroxid; and the *red oxid=red precipitate=Hydrargyri oxidum rubrum, U. S. P.*, an orange-red crystalline powder, obtained, not by precipitation, but by heating the nitrate. Both are used in the preparation of ointments. The former is the more active in its chemical and medicinal actions, and exists both in solution and in suspension in *yellow wash*. When improperly prepared the yellow oxid may contain corrosive sublimate as an impurity, and the red oxid may contain the nitrate. Mercuric oxid decomposes the chlorids of many metals in solution, with formation of a metallic oxid and mercuric oxy-chlorids. It combines with alkaline chlorids to form soluble double chlorids, called *chloromercurates* or *chlorhydrargyrates*, and forms similar compounds with alkaline iodids and bromids. Mercuric oxid is used in veterinary practice in the shape of ointment, and enters into the

[1] Hendricks: Am. J. Pharm., 1878, 4 s., l., 325.

[2] Potter: Lancet, London, 1838, i., 215.

[3] Med. Times and Gaz., 1863, i., 446. *Ex* Taylor: *Op. cit.*, p. 353.

[4] Jones *v.* Fay, 1865, Taylor: *Op. cit.*, p. 358.

[5] Stewart: N. Y. J. of M., 1846, vii., 19.

[6] Lucas: Ind. Med. Gaz., 1886, xxi., 79. Walker: Brit. Med. Jour., 1891, ii., 1147.

composition of a number of proprietary preparations, chiefly eye salves.[1]

We find mention of but one fatal case of poisoning by the red oxid taken internally, that of a hat maker of 26 years, who swallowed an ounce (30 gm.) and died on the second day.[2] Accidental, non-fatal poisonings by swallowing the oxid are reported by Kennedy (2 ʒ = 7.8 gm.),[3] Allison (35 grs. = 2.3 gm.),[4] Taylor ("a quantity"),[5] Smyth ("a quantity in beer"),[6] Stevenson (120 grs. = 7.8 gm.),[7] Ord (about a teaspoonful),[8] and Lee (half an ounce = 15.5 gm.),[9] all in adults. Prince[10] has reported two attempts at suicide, each with 40 grs. (2.6 gm.). White gives an incomplete account of the non-fatal alleged poisoning of a girl of 15 years by red precipitate, given to her by a boy under the representation that it was sugar (!).[11] A fatal poisoning of a girl of 1¾ years by daily inunctions for 3 weeks of an ointment containing red precipitate by an empiric has been carefully reported by Gwalter.[12]

Mercuric sulfid—*cinnabar; vermilion; Æthiops mineralis; Hydrargyri sulphidum rubrum, U. S. P.*—**HgS**—231.78—exists in nature as the principal ore of mercury and is manufactured artificially, either as a brilliant red powder (vermilion) or as a black powder, formerly used as a medicine (æthiops). It has been known to cause chronic mercurial poisoning from having been used to color artificial gums.[13] In two instances it has caused acute poisoning by having been administered in fumigation by empirics.[14] Three persons were found dead in bed the morning after having been treated for the itch by a quack by inunction of nine ounces of a salve which contained mercury, cinnabar, and other substances.[15]

[1] Hahn u. Holfert: "Specialitäten und Geheimmittel," 5te Aufl., Berlin, 1893, Nos. 124, 129, 132, 134, 135, 136, 137, 138, 1092, 1312, 2246.

[2] Sobernheim: "Handb. d. prakt. Tox.," Berlin, 1838, 250, *ex* Repert. f. d. Pharm., 1837, 2 R., iii., 361.

[3] Med.-chir. J. and Rev., London, 1816, i., 189.

[4] Lancet, 1836, i., 401.

[5] "Poisons," 3d Am. ed., 383.

[6] Brit. M. Jour., 1878, ii., 101.

[7] Taylor: "Med. Jur.," 11th Am. ed., 145, *ex* Brit. M. Jour., 1884, i., 56.

[8] Lancet, 1888, ii., 570.

[9] Brit. M. Jour., 1889, ii., 718.

[10] Lancet, 1859, ii., 506.

[11] Irish Hosp. Gaz., 1873, i., 308.

[12] "Ein Fall von Quecksilbervergiftung," Diss., Zurich, 1877.

[13] Wells: Brit. M. Jour., 1863, ii., 366. Woodman: Med. Pr. and Circ., London, 1874, ii., 502. Buckley: Tr. N. Y. M. Assoc., 1884 (1885), i., 126.

[14] Sutro: Med. Times, London, 1845, 27. Szabo: Pest. med.-chir. Presse, 1883, xix., 671.

[15] Leiblinger: Wien. med. Wochenschr., 1869, xix., 1595.

Mercurous chlorid—*calomel ; mild chlorid of mercury ; protochlorid of mercury ; Hydrargyri chloridum mite, U. S. P.; Hydrargyri subchloridum, Br. P.*—Hg_2Cl_2—470.34—is prepared by one of three methods: 1. By sublimation of a mixture of mercurous sulfate and sodium chlorid, and washing out the corrosive sublimate, which is also formed, with water. 2. By precipitation of a solution of mercurous nitrate with hydrochloric acid or a chlorid. 3. By heating together an intimate mixture of 4 parts of mercuric chlorid and 3 parts of mercury. The product is purified by a sublimation with steam (*calomel à la vapeur*). Ordinary sublimed calomel is in imperfectly formed quadratic prisms, steam calomel in smaller but more perfectly formed prisms, and precipitated calomel in very much smaller crystals. It is a heavy, faintly yellowish-white powder, insoluble in alcohol and in water, soluble in boiling water only to the extent of 1 part in 12,000, slightly soluble at the temperature of the body in artificial or natural gastric juice, soluble in notable amount in solutions of alkaline sulfates. When heated it sublimes without fusing.

Calomel is decomposed slowly by sunlight into mercuric chlorid and mercury. A similar decomposition is produced more rapidly by common salt and by alkaline chlorids. Solutions of the hydroxids and carbonates of the alkaline metals decompose it with formation of mercuric oxid and liberation of mercury; if alkaline chlorids be also present mercuric chlorid is produced. Potassium iodid acts upon it with formation of potassium chlorid, mercuric iodid, and mercury. Chlorin or aqua regia convert it into mercuric chlorid.

Calomel is one of the ingredients of the compound cathartic pill (28.2 per cent. Hg_2Cl_2), and of Plummer's pills (25 per cent. Hg_2Cl_2). It enters into the composition of a few proprietary medicines,[1] and of many cosmetics.[2]

Excepting the single supposed suicide by calomel mentioned by Bernt,[3] the poisonings by this agent reported in medical literature are medicinal, and for the most part of early date. In some of these death was caused, probably indirectly, from septic

[1] Hahn u. Holfert: *Op. cit.*, Nos. 519, 761, 1319, 1324, 1325, 1363, 1409, 1787

[2] *Ibid.*, Nos. 1876, 1952, 1963, 1977, 1981, 2013, 2152, 2183, 2298, 2316, also in Goulard's oriental cream, eau de Ninon, and eau de lys.

[3] Visa reperta, 1827, i., 374.

poisoning from extensive ulceration and gangrene of the salivary glands and surrounding parts.[1] Of the more modern cases one, reported by Leuteri[2] is an instance of susceptibility (idiosyncrasy) to calomel: A man of 43 years suffering from aortic insufficiency, cardiac hypertrophy, and atheromatous arteries, with ascites and œdema, was given 0.2 gm. (3 grs.) of calomel thrice daily for 7 days. On the fourth and fifth days there was marked increase in the elimination of urine from 1,200 c.c. on the third to 11,200 c.c. on the fourth and 6,800 c.c. on the fifth. On the sixth day the quantity sank to 1,200 c.c., on the seventh to 650 c.c., and on the eighth, the day of his death, there was suppression. In the mean time diarrhœa had begun on the third day and had increased, accompanied by abdominal pain and vomiting. At the autopsy, besides the organic lesions, those in the large intestine and kidneys due to mercurial poisoning were found to exist. In another case a woman of 68 years died in 5 days after having received 1 gm. (15½ grs.) of calomel in 2 days, having suffered from severe purging and suppression of urine.[3]

Fatal poisonings have also resulted from the hypodermic injection of calomel in the treatment of syphilis (Scarenzio, calomel rubbed up with glycerol). Thus Runeberg reported[4] the case of a woman of 34 years who received three injections of 0.1 gm. (1½ grs.) March 12th, 20th, April 13th, and died in coma May 6th, having manifested symptoms of mercurial poisoning. Although in this instance calomel was not the sole cause of death, it was a largely contributory factor. Claeys[5] has reported a mercurial poisoning caused by a single insufflation of calomel into the conjunctival sac.

The smallest quantity of calomel which has been alleged to have caused death was in the case of a boy of 14 years who died in 3 weeks from the secondary effects of 6 grs. (0.4 gm.).[6] In Graves and Stokes' case a girl of 11 years died from fatal slough-

[1] London M. Gaz., 1836, xviii., 484. Sobernheim: "Toxicologie," p. 253. Graves and Stokes: Dublin Hosp. Repts., 1827, iv., 299. Grattan: Tr. Assoc. King's and Queen's Coll. Phys., Irl., 1820, iii., 236. Jackson: Am. J. M. Sc., 1828, iii., 89.

[2] "U. d. anatom. Veränd. d. Sublimatintox.," Berlin, 1895, p. 15.

[3] Adam: "Darm u. Niere nach Calomelvergiftung," Diss., Würzb., 1892.

[4] Deut. med. Wochenschr., 1889, xv., 4.

[5] Livre jubil. . . . Soc. de méd. de Gand, etc., 1884, 287.

[6] London M. Gaz., 1836.

ing of the mouth and palate in 8 days after having taken 8 grs. (0.5 gm.). In Sobernheim's case, an adult died in 8 days from extensive sloughing of the face following a dose of about 15 grs. (1 gm.). In Grafton's case a hydrocephalic girl of 10 years died in 13 days from gangrene of the face 13 days after having received 20 grs. (1.3 gm.) in 10 pills during 6 days. In Leutert's case the total quantity taken during 7 days was 4.3 gm. (64 grs.) in doses of 0.3 gm. (3 grs.) each. These doses, although large, do not greatly exceed the maximum medicinal doses, and are less than have been given in many instances without dangerous effects. Two early accidental cases are reported in which calomel was taken by mistake in doses of 1 ℥ (31 gm.)[1] and 14 ℥ (54.4 gm.)[2] at one time without causing death.

Owing to imperfect purification or to exposure to sunlight calomel may contain corrosive sublimate. Although this impurity is now rarely met with, an early case is recorded in which death was caused by the administration of 12 grs. (0.8 gm.) of calomel, a remaining sample of which was found to contain a large proportion of corrosive sublimate.[3]

Mercuric chlorid—*bichlorid of mercury; corrosive sublimate; perchlorid of mercury; Hydrargyri chloridum corrosivum, U. S. P.; Hydrargyri perchloridum, Br. P.*—$HgCl_2$—270.54—crystallizes by sublimation in octahedra and by evaporation of its solutions in flattened, right-rhombic prisms. It is soluble in water to the extent of 6.57 parts in 100 at 10° (48° F.); 7.39 at 20° (68° F.); 11.84 at 50° (122° F.); 24.3 at 80° (196° F.); and 53.96 at 100° (212° F.). It is more soluble in alcohol, which dissolves 40 per cent. in the cold and 66.6 per cent. when hot. It dissolves in absolute ether to the extent of 11.36 parts in 100 at 15°.5 (60° F.), and of about 30 parts in 100 of commercial ether. Chloroform dissolves only about 1 part in 1,700. The taste of corrosive sublimate is very marked, styptic, nauseous, and metallic in character, and is persistent.

Corrosive sublimate is easily reduced to calomel or to elementary mercury by heating with mercury, or with zinc, iron, lead, copper, or bismuth, or even, slowly, by exposure of its solution to light. The hydroxids of potassium, sodium, calcium, or

[1] Robarts: London M. Gaz., 1838, xxii., 610.

[2] Bochardt: Allg. med. Annal., 1827, 441.

[3] Lancet, ii., 1848, 187.

magnesium produce in its solutions precipitates of a brown oxychlorid, or of the orange-colored mercuric hydroxid or oxid. It readily combines with the alkaline chlorids to form double chlorids, known as *chloromercurates* and *chlorhydrargyrates*. Many organic substances decompose it into calomel and mercury, particularly under the influence of sunlight. With albumin it forms a compound insoluble in water but soluble in an excess of fluid albumin or of alkaline chlorids, and in tartaric acid solution. A solution of *mercury albuminate* in a solution of sodium chlorid, containing 1.5 per cent. of mercuric chlorid, is used medicinally for hypodermic injection.

Corrosive sublimate is sometimes administered internally, but its chief use in medicine at present is as an antiseptic, as it is one of the most powerful of germicides, for which reason it is also largely used as a disinfectant and preservative of anatomical preparations. *Liquor Plenckii* consists of 2 gm. each of bichlorid, lead acetate, lead carbonate, and camphor, with 16 gm. each of alcohol and vinegar, and consequently contains 6.25 per cent. of mercuric chlorid. *Van Swieten's* solution, according to the French Codex, is made by dissolving 0.9 gm. mercuric chlorid in 96 gm. of alcohol and diluting with 928 gm. of water, and therefore contains about 1 : 1,000.

It enters into the composition of several proprietary remedies intended for external application,[1] and of some cosmetics in which it is either intentionally introduced or produced from calomel.[2] It is also the active agent in many liquid "bedbug poisons."

Poisonings by corrosive sublimate have increased greatly in frequency during the past few years. Of 368 cases of which we find mention in medical literature of the present century 231 have been reported since the year 1879; while during the decade 1870–1879 only 22 cases were reported. The cause of this sudden and large increase is to be found in the very extensive use of solutions of the bichlorid as antiseptics. Of the 231 cases reported since 1879, 143 were caused by irrigation of the vagina or uterus or both with solutions of 1 : 1,000 or of still higher dilution in obstetrical or gynæcological cases, and of these 48 were

[1] Hahn u. Holfert: *Op. cit.*, Nos. 586, 622, 788, 934, 1056, 1398, 1537, 1596, 1607, 1814.

[2] *Ibid.*, Nos. 1933, 1952, 1977, 2006, 2015, 2134, 2188, 2264.

fatal.[1] In many of the recent cases death has followed surgical operations in which solutions of corrosive sublimate of 1:1,000 or 1:2,000 have been used, and in which there have been distinct ante-mortem and post-mortem evidences of mercurial poisoning.[2] Of 172 cases other than surgical or obstetrical 70 were accidental, 47 suicidal, 15 by administration by quacks, and 27 homicidal, the remainder having no cause assigned. Of the ACCIDENTAL CASES, several followed medicinal administration either of solution or ointment externally applied, or hypodermically[3] or by the mouth[4] or rectum[5] in the treatment of syphilis. Or it has been taken in mistake for other white powders (Epsom salt, tartar emetic, potassium bromid, sugar, bismuth subnitrate, santonin, calomel), or for whiskey; or by accidental interchange of medicine, or by druggists' error in three cases in place of calomel, and in two instances in supplying prescriptions calling for "Hyd. chlor.," intended by the careless writer to mean chloral hydrate. Two poisonings, one fatal, were caused by prolonged contact of the solution with the skin.[6] One case, which was the subject of medico-legal investigation, was caused by the use of a mercurial cosmetic.[7] The antiseptic tablets of bichlorid, although usually colored blue, have been swallowed accidentally.

Of the 47 SUICIDES there was but 1 which was in any way peculiar: that of a young girl who took an alcoholic solution of corrosive sublimate (bedbug poison) by rectal injection.[8]

The HOMICIDAL cases include 18 poisonings by quacks and 27 deliberate homicides, real or alleged. Among the latter are included some early *causes célèbres*. The investigation of the series of poisonings in France in the middle of the seventeenth

[1] Sehillotte ("Intoxications par le sublimé corrosif chez les femmes en couche," Thèse, Paris, 1891) gives full reports of many cases. See also Delaunay: Thèse, Paris, 1893 (No. 359). Leutert: *Op. cit.*

[2] Peabody: Med. Rec., N. Y., 1885, xxvii., 290 (eleven cases, seven deaths). Fraenkel: Arch. f. path. Anat., etc., 1885, xcix., 276 (fourteen cases, $HgCl_2$ cause of death in two). Grawitz: Deut. med. Wochenschr., 1888, xiv., 41. Czernohoss: Wien. med. Presse, 1889, xxx., 958.

[3] Marocco: Verhandl. d. X. Int. Congr., 1891, iii., 170.

[4] Barthélemy: Ann. d'hyg., 1883, 3 s., iv., 337. Koboyner: Bull. gén. de thérap., 1878, xcv., 75.

[5] Huber: Ztschr. f. kl. Med., 1888, xiv., 459.

[6] Orfila: "Tox. gén.," 3ème ed., i., 671 (Cloquet). Dittrich: Vrtljschr. f. ger. Med., etc., 1891, 3 F., i., 71 (D.).

[7] Stavely and Pedley: Brit. M. Jour., 1893, i., 889. For another case of local poisoning by a mercurial cosmetic see Marro: Giorn. d. r. Ac. di med. di Torino, 1891, 3 s., xxxix., 224.

[8] Hofmann: "Lehrb. d. ger. Med.," 5te Aufl., 615.

century, in which the Marchioness of Brinvilliers was the central figure, showed that corrosive sublimate was probably the poison most frequently used.[1] At an earlier period (1613) Sir Thomas Overbury had perished in the Tower of London, a victim of poisons administered to him at the instigation of the Countess of Somerset, and, although many real and supposed poisons had been previously mixed with his food, he finally succumbed to a clyster containing corrosive sublimate.[2] Two alleged homicides were the subjects of trials in the eighteenth century, one in Germany,[3] and one in England.[4] We can find mention of but two French cases, one in 1810,[5] the other in 1857.[6] Concerning nine cases in England during the present century we find only brief notices.[7] Information as to American cases known to have been the subjects of trial is equally inaccessible. A man was tried in New York State for the murder of his wife by corrosive sublimate in 1824;[8] a supposed attempt to poison a woman was investigated at Buffalo, N. Y., in 1838;[9] a man was accused of having poisoned a female servant, pregnant by him, near Boston, Mass., in 1861;[10] a man named Williams was supposed to have been poisoned in Westchester County, N. Y., in 1880; a man was tried in New Jersey

[1] See Introduction, p. 22.

[2] Hargrave's State Trials, i., 323, 345. Amos: "The great Oyer of Poisoning," London, 1846.

[3] Eller: "Rept. f. d. ger. u. öff. Arzneyw.," 1789, i., 248.

[4] Rex *v.* Butterfield, 1775. Ingram: "Enquiry into the cause of death of William Scawen." London, 1777. Paris and Fonblanque: "Med. Jur.," i., 303, ii., 262. Beck: "Med. Jur.," 12th ed., ii., 632.

[5] Aff. Bridon, Chaussier: "Consultations médico-légales sur une accusation d'empoisonnement par le sublimé corrosif," Paris, 1811. Tardieu: "Empois.," 2ème ed., 685. Ztschr. f. d. Staatsarznk., 1824, 3 Erghft., 1.

[6] Lassaigne: Ann. d'hyg., 1858, 2 s., x., 200.

[7] Rex *v.* Mary Bateman, 1809: "Celebrated Trials," vi., 44. Case of Mich. Whiting, 1812. Beck: "Med. Jur.," 12th ed., 634. Rex *v.* Wm. Patterson, 1817. Christison: "Poisons," Am. ed., 319, 335, Cases of Mrs. Byrne and son, 1820. Lendrick: Trans. Assn. King's and Queen's Coll. Phys. Irel., 1820, iii., 310. Reg. *v.* Walsh: 1850, London M. Gaz., 1850, n. s., xi., 253. Reg. *v.* Abrah. Kaley, 1862; Ph. J. and Tr., 1861-62, n. s., iii., 627. Alleged murder of an infant, Lancet, 1878, ii., 876. Man tried in 1884, Taylor: "Med. Jur.," 11th Am. ed., 139. Reg. *v.* Eliz. Berry (the Oldham case), Harris: Med. Chron., Manchester, 1887, vi., 122. To these may be added the case of a woman of twenty years who was induced to take corrosive sublimate by ill treatment and apprehension of pregnancy, Edinb. M. and S. Jour., 1811, vii., 150.

[8] Peo. *v.* Hodgson, Dean: "Med. Jur.," 1854, 317. See also Edinb. M. and S. Jour., 1824, xxii., 438.

[9] Burwell: Am. Jour. M. Sc., 1841, n. s., ii., 515.

[10] White: Boston M. and S. Jour., 1861, lxv., 169.

in 1881 for the murder of an infant; a negro boy in Pennsylvania was tried in 1884 for an attempt to poison a family; an attempt at poisoning in New York City in 1886 proved unsuccessful; a woman was tried in New Jersey in 1893 for the murder of her son, and acquitted. Of none of these cases do we find any mention except in the secular press.[1]

The DOSE of corrosive sublimate which may cause death, once it is absorbed, is probably much smaller than is indicated by the smallest quantities which have been known to prove fatal when swallowed. Taylor[2] has reported the case of a man of 25 years, who died in 7 days from the effects of 5 grs. (0.32 gm.) taken in solution in vinegar. Ketli[3] relates the case of a physician who swallowed 50 c.c. of a one-per-cent. solution of corrosive sublimate (=0.5 gm.=$7\frac{3}{4}$ grs.) and died on the eighth day. But much smaller quantities have been known to cause the death of parturient females when used in intra-uterine or vaginal irrigations. Thus Vöhtz[4] used a quantity of a 1:750 solution (for irrigation after an abortion at three months) which contained about 0.25 gm. ($3\frac{3}{4}$ grs.). The woman (æt. 33) manifested the usual symptoms of mercuric poisoning and died on the tenth day. In another instance, reported by Braun,[5] a woman died of mercurial poisoning in 7 days after intra-uterine irrigation with 1 litre of a 1:3,000 solution, the total quantity injected being therefore 0.34 gm. ($5\frac{1}{4}$ grs.). In these and other similar cases only a fraction of the amount injected can possibly be absorbed. On the other hand, very large doses have been recovered from: A man of 24 years accidentally swallowed a quantity of a solution of corrosive sublimate, used for photographing purposes, containing 100 grs. (6.5 gm.) of the poison; he swallowed a pint of milk on discovering the mistake and was under medical treatment within ten minutes, and recovered after an illness of a week.[6] A woman of 40 years attempted suicide by swallowing an ounce of a supersaturated alcoholic

[1] The case last referred to is that of State *v.* Mattie C. Shann, the salient points of which are given in the note on p. 115.

[2] Guy's Hosp. Rep., 1864, 3 s., x., 183. Taylor ("Poisons," 2d ed., 460; "Pr. and Pr. Med. Jur.," 2d ed., i., 282) refers to the fatal poisoning of a child by three grains, as reported in Lancet, 1845, 297. We fail to find the report in either volume for that year.

[3] Pest. med.-chir. Presse, 1878, xiv., 129, 150.

[4] Hosp. Tid., Kjøb., 1884, 3 R., ii., 557.

[5] Wien. med. Wochenschr., 1886, xxxvi., 740.

[6] Lodge: Brit. M. Jour., 1888, ii., 720.

solution containing 4 gm. (4.5) of mercuric chlorid, and ultimately recovered from the effects.[1] An early case is reported by Budd[2] in which a woman is said to have recovered from the effects of an ounce (31 gm.) of corrosive sublimate taken with suicidal intent.

It has been stated by Pouqueville, Rigler, and Tschudi that opium eaters in the Orient are in the habit of taking enormous quantities of corrosive sublimate (40 to 60 grs. = 2.6 to 3.9 gm.) along with their opium without the manifestation of any untoward symptoms. These statements have been insufficiently verified. It may be possible, however, that the mercuric salt may be rendered insoluble by combination with constituents of the opium.[3]

White precipitate—*mercury chloramidid; ammoniated mercury; Hydrargyrum ammoniatum, U. S. P.*—**NH_2-HgCl**—251.27—is a white, odorless, and tasteless powder produced by adding a slight excess of ammonium hydroxid to a solution of mercuric chlorid. It is known as *infusible white precipitate* to distinguish it from a related compound, NH_2-HgCl, NH_4Cl, called *fusible white precipitate*. When heated, it volatilizes completely without fusion. It is insoluble in cold water, alcohol, or ether. It dissolves completely in hydrochloric acid and in acetic acid. It is used medicinally in its own form and in an ointment (Ung. hydrarg. ammon., U. S. P.), and is contained in a few proprietary articles.[4]

Poisonings by white precipitate have been of not infrequent occurrence, and the proportion of attempts at homicide, 6 out of 22 cases, is relatively large. But 1 of these, however, was successful (Reg. *v.* Moore, 1860) in which a child of 3 months was poisoned by its mother.[5] Two cases were unsuccessful attempts at suicide, 1 with a mixture of 90 grs. (5.8 gm.) each

[1] M. and S. Reptr., Phila., 1872, xxvi., 248.

[2] Phila. Med. Museum, 1806, ii., 180.

[3] See Pouqueville: "Voyage dans la Grèce," etc., Paris, 1820–21. Thornton: "The Present State of Turkey," London, 1809. Rigler: "Die Turkei und ihre Bewohner," Wien, 1852. Tschudi: Wien. med. Wochenschr., 1851, i., 454. Byron: "Notes to Childe Harold," ii., 33.

[4] Hahn u. Holfert: *Op. cit.*, Nos. 125, 1723, 2249, 2357.

[5] Taylor: Guy's Hosp. Repts., 1860, 3 s., vi., 483. The other cases, also referred to in Taylor, "Poisons," are Reg. *v.* Daniel, 1855; Reg. *v.* Hargreaves, 1866; Reg. *v.* Seaham, 1869; a boy of twelve years, convicted in 1873; Reg. *v.* Clapp, 1874.

of white precipitate and red oxid.[1] In 6 instances the poison was taken in consequence of errors on the part of the druggist or patient.[2] In one case a woman suffered from the local effects of ointment of white precipitate applied as "dandruff cream."[3] Among the 22 cases reported only 3 were fatal, 2 in children of 3 months,[4] and 1 a woman who died in 7 days after taking an unstated quantity by mistake.[5] Only in Short's case is the dose stated, 20 grs. (1.3 gm.). Michael[6] reports the case of a woman of 37 years who recovered after having taken 100 grs. (6.5 gm.); and Graham[7] that of a man of 35 years, who was severely poisoned by about 2 ʒ (7.8 gm.) but recovered.

Mercuric iodid—*biniodid* or *red iodid*; *Hydrargyri iodidum rubrum*, *U. S. P.*—HgI_2—452.88—is a brilliant red powder produced by precipitating a solution of mercuric chlorid with one of potassium iodid. If an excess of the alkaline iodid be used the precipitate redissolves as potassium iodhydrargyrate. It is almost insoluble in water (1 : 20,000) but forms a colorless solution with about 130 parts of alcohol. It dissolves readily in potassium iodid solution, in many dilute acids, and in solutions of ammoniacal salts, alkaline chlorids, and mercuric salts. It is one of the constituents of Donovan's solution (Liq. Arsen. et Hydrarg. Iod., U. S. P.), and is contained in a few proprietary remedies.[8]

We find mention of but two instances of poisoning in the human subject by this agent. One that of a man of 25 years, who, with suicidal intent, swallowed the whole of a prescription which he had been taking for syphilis. The symptoms differed from those usually observed in mercurial poisoning, although ptyalism appeared in about 30 hours, and there were vomiting and abdominal distress, but there was no disposition to diarrhœa, and nothing is said in the account of suppression of urine. The most prominent symptoms were muscular spasms,

[1] Benham and Hendley: Brit. M. Jour., 1885, i., 484.

[2] Sandberg: Brit. M. Jour., 1889, i., 709 (for sal ammoniac). Stevenson: Guy's Hosp. Rep., 1874, 3 s., xix., 415 (for magnesia); Lancet, 1871, ii., 540 (for bicarbonate of soda). Hardy: Brit. Med. Jour., 1876, ii., 76 (for seidlitz powder). Stephens: Brit. M. Jour., 1876, i., 781 (for lac sulfuris). Cockle: Med. Times and Gaz., 1882, i., 302.

[3] Green: Brit. M. Jour., 1884, i., 853.

[4] Taylor: *Loc. cit.* Short: Med. Times and Gaz., 1860, i., 444.

[5] Stevenson: *Loc. cit.*

[6] Brit. M. Jour., 1857, ii., 909.

[7] *Ibid.*, 1869, i., 329.

[8] Hahn u. Holfert: *Op. cit.*, Nos. 876, 880, 1483, 1608.

stupor, and a peculiar regurgitation at each attempt to swallow.[1] The second case was that of a woman who died in 4 days from the effects of an irrigation with a solution of the biniodid 1 : 2,000 after normal delivery. There was suppression of urine and the autopsy showed the lesions of acute nephritis.[2]

Mercuric sulfate—$HgSO_4$—295.62—is a white, crystalline powder, manufactured as the first stage in the production of corrosive sublimate. On contact with water it is decomposed into an acid salt, which dissolves, and turpeth mineral (see below).

Two cases of poisoning by this salt have been reported. In one a man swallowed a quantity of the salt used in the mounting of certain electrical apparatus, by mistake. He recovered in 8 days.[3] In the second case death occurred in 22 days, after complete anuria.[4]

Turpeth mineral—*turbith mineral*—$HgSO_4$, $2HgO$—a basic mercuric sulfate, formed as a lemon-yellow powder by the action of water upon mercuric sulfate, soluble in 2.000 parts of cold water, was formerly official and is still used to some extent in medical practice.

It has proved poisonous to persons beyond the age of childhood in three reported instances: In one eight men received each 5 grs. (0.3 gm.) of turbith and 3 grs. (0.2 gm.) more in half an hour; they all suffered from a sense of burning in the throat and fauces, vomiting, diarrhœa, much griping and depression. In one there were soreness of the gums and salivation. None died.[5] In the other two instances the dose taken was larger, and death followed after the usual manifestations of mercurial poisoning: In one a youth of 16 died in a week from the effects of 1 3 (3.9 gm.);[6] in the second, which was the subject of a trial for manslaughter in England in 1862, a man of 27 years died in 11 days from the effects of 40 grs. (2.6 gm.) dispensed by mistake of the druggist.[7] Some instances have been reported in which doses of from 3 to 6 grs. (0.2 to 0.4 gm.) of turpeth, medicin-

[1] Taylor: Boston M. and S. Jour., 1870, n. s., v. (lxxxii.), 499.

[2] Grossier, in Sebillotte: *Op. cit.*, p. 155. See also Morell Lavallée: Schmidt's Jahrb., 1893, ccxxxii., 127.

[3] Van der Burg: Nederl. Tijdsch. v. geneesk., 1880, 333.

[4] Ludwig: Wien. kl. Wochenschr., 1889-90, *ex* Leutert: *Op. cit.*, p. 89.

[5] Randolph and Roussel: Med. News, Phila., 1884, xliv., 275.

[6] Letheby: Lancet, i., 1847, 285.

[7] Taylor (Snoad): Guy's Hosp. Rep., 1864, 3 s., x., 180.

ally given, have proved fatal to young children, death having occurred in collapse in from 3 to 15 hours, apparently from acute gastritis rather than the usual nephritic action of the mercurials.[1] Another who received 0.36 gm. in two doses given 15 minutes apart as an emetic without producing vomiting, suffered from diarrhœa and ulceration of the mouth and gums, but recovered.[2]

Mercuric nitrate—$Hg(NO_3)_2$—323.76—exists, along with excess of nitric acid, in the *Liq. hydrargyri nitratis, U. S. P.*, and, incorporated with lard oil, in the *Ung. hydrargyri nitratis, U. S. P.*, or *citrine ointment*. The *Liqueur de Belloste* or *remède de capucin* or *du duc d'Antin* is a solution of mercurous nitrate ($Hg_2[NO_3]_2$), with excess of nitric acid. Mercuric nitrate is also used in painting on porcelain; and, either alone or mixed with white arsenic and corrosive sublimate, by hatters and furriers; and in veterinary medicine.

Poisonings by this salt may be divided into three classes: 1. Those in which the acid solution is swallowed. 2. Those in which the solution or ointment is externally applied. 3. Chronic cases occurring in hatters and furriers, etc. The first class of cases manifest the symptoms of nitric-acid corrosion (see page 250) and death occurs within a few hours, usually without any of the appearances of mercurial poisoning. In 3 cases of suicide death occurred in 25 minutes and $2\frac{1}{4}$ and 3 hours.[3] In another suicide life was prolonged for $12\frac{1}{2}$ hours, and although the symptoms were those of corrosion, and death was the result of the local effects, at least one of the effects of mercury, suppression of urine, was observed.[4] In one instance death from suffocation immediately followed an application of the acid nitrate to the throat as an escharotic.[5] The pronounced taste of the poison and its immediate local action frustrated an attempt at homicide by the administration of mercuric nitrate in chamomile tea.[6] The death of a man of 73 years following in 23

[1] McPhedron: Med. News, Phila., 1883, xliii., 682. "Medicus," Med. and Surg. Reptr., Phila., 1884, l., 93. Northrup: Proc. N. Y. Path. Soc., 1890, 34 (two cases).

[2] Woodbridge: Occid. Med. Times, 1891, v., 123.

[3] Hickenbotham: Brit. M. Jour., 1872, i., 113. Fauvel: Bull. Soc. d'anat., Paris, 1837, xii., 175. Bigsley: London M. Gaz., 1830, vii., 329.

[4] Chambé: "Empoisonnement par le nitrate acide de mercure," Thèse, Strasb., 1857.

[5] Guy's Hosp. Rep., 1850, 206.

[6] Reg. *v.* Smith, 1857. Taylor: "Poisons," 3d Am. ed., 388. A

days after he had accidentally swallowed half a glassful of the solution is reported by Prévost.[1]

When the intoxication is produced by external application the poison is absorbed, and the symptoms are not those of corrosion but those of mercurial poisoning, death is usually delayed for several days, and the lesions of acute mercurial poisoning are observed at the autopsy.[2]

When mercurial poisoning occurs in hatters, furriers, and others from constant contact with mercuric nitrate it assumes the chronic type.[3]

Mercuric cyanid—*Hydrargyri cyanidum, U. S. P.*, $Hg(CN)_2$—251.94—white, prismatic, odorless crystals having a bitter, metallic taste, which become darkened on exposure to light; soluble in 12.8 parts of water and in 15 parts of alcohol at 15° (59° F.).

The few instances of acute poisoning by this substance observed in the human subject have presented the characters of mercurial rather than those of cyanic poisoning: A man took 1.3 gm. (20 grs.) with suicidal intent, manifested all the symptoms of mercurial poisoning vomiting, diarrhœa, salivation, fetor, suppression of urine, and died of syncope on the ninth day.[4] The same symptoms were observed in a physician who made an unsuccessful attempt to destroy himself by swallowing about 2 grs. (0.12 gm.) of the cyanid.[5] On the other hand, animals experimented upon by Tolmatscheff[6] and Wladikin[7] manifested marked cyanic symptoms and died within 1½ hours. An instance of chronic poisoning by mercuric cyanid (used in the manufacture of percussion caps) complicated with and terminating in death from an acute poisoning, is reported

case of poisoning by $Hg(NO_3)_2$ is reported by Scalzi (Giorn. med. di Roma, 1867, iii., 14), to which we have not access.

[1] Rev. méd. d. l. Suisse rom., 1882, ii., 554, 605.

[2] Jour. de chim. méd., etc., 1847, 3 s., iii., 197. Hutin (Cloquet): Child, quoted by Chambé: *Op. cit.* Vidal: C. rend. Soc. de biol., 1863 (1864), 3 s., v., 193. Dubar: Gaz. des hôp., 1867, xi., 493. Stevenson-Taylor: "Med. Jur.," 11th Am. ed., 145. Cabaret: Gaz. des hôp., 1854, xxvii., 119.

[3] Taylor: Guy's Hosp. Rep., 1864, 173. Axenfeld: Gaz. des hôp., 1870, xliii., 97. Stadthagen: Deut. med. Wochenschr., 1884, x., 202. Charpentier: Bull. soc. de méd. publ., etc., 1885, viii., 12. Adler: Med. News, Phila., 1891, lix., 186.

[4] Ollivier: J. de chim. méd., etc., 1825, i., 209 (269); also in Orfila: "Tox. gén.," 5ème ed., i., 735.

[5] Moos: Arch. f. path. Anat., etc., 1866, xxxi., 117.

[6] Hoppe-Seyler: "Med.-chem. Unt.," Berlin, 1871, 285.

[7] "Wirkung des Cyankaliums und des Cyanquecksilb.," Diss., Bern, 1878.

by Klob.[1] Other cases of chronic poisoning from cyanid or fulminate are reported by Bamberger,[2] Lancereaux,[3] and Faucher.[4]

Other Organic Compounds of Mercury.—MERCURIC THIOCYANATE (sulfocyanid)—$Hg(CNS)_2$—enters into the composition of the toys called *Pharaoh's serpents*. An instance in which death resulted in 15 days from the effects of a powder containing the thiocyanate accidentally administered is reported by Brouardel and Ogier.[5]

An account of the fatal poisoning of two chemists by inhalation of the vapor of MERCURY DIMETHYL, $Hg(CH_3)_2$ is given by Edwards.[6] The poisonous action of MERCURY DIETHYL, $Hg(C_2H_5)_2$ upon animals has been studied by Hepp.[7]

ACUTE MERCURIAL POISONING.

DURATION.

The usual duration of fatal cases of mercurial poisoning is from 5 to 12 days. In a few instances death has occurred within a few hours. The most rapidly fatal case of true mercurial poisioning of which we can find record is that reported by Skegg[8] in which a man of 54 years died in 3¼ hours from the effects of 112 grs. (7.26 gm.) of corrosive sublimate. A large dose of the same mercurial taken in vinegar caused the death of a man of 32 years in 5 hours;[9] and 40 grs. (2.6 gm.) of white precipitate proved fatal to a man in the same time.[10] A man of 76 years died in 5½ hours, in collapse and without

[1] Wien. med. Presse, 1868, xxvii., 64.

[2] Deut. Klin., 1850, 110.

[3] Gaz. d. hôp., 1891, lxiv., 417.

[4] Rev. San. d. Bordeaux, 1886, iii., 165.

[5] Ann. d'hyg., etc., 1893, 3 s., xxix., 352. See also Ouchinsky: *Ibid.*, 347, and Love: St. Louis M. and S. Jour., 1881, xlii., 291.

[6] St. Barth. Hosp. Rep., 1865, i., 141.

[7] Arch. f. exp. Path. u. Pharm., 1887, xxiii., 91.

[8] Lancet, 1863, i., 119. There are four reported cases of shorter duration; but of these three [Bigsley: London M. Gaz., 1830, vii., 329 (three hours). Fauvel: Bull. Soc. d'anat., Paris, 1837, xii., 175 (two and a quarter hours); and Hickenbotham: Brit. M. Jour., 1872, i., 113 (twenty-five minutes)] were by mercuric nitrate, which acted as a corrosive rather than as a mercurial poison; and in the fourth the evidence that death was due to mercurial poisoning is insufficient (Bigham: Amer. M. Times, 1862, iv., 347).

[9] Lucas: Med. Times and Gaz., 1871, ii., 382.

[10] Moore: Brit. M. Jour., 1885, ii., 15.

having been salivated.[1] A child of 2½ years died in 6 hours from the effects of 0.7 gm. (gr. i.) of corrosive sublimate given in mistake for santonin.[2]

The longest duration of a fatal poisoning caused by a single dose of a mercurial taken by the mouth was in a woman of 67 years who took an alcoholic solution of corrosive sublimate (bed-bug poison) with suicidal intent and died in 23 days.[3] A youth of 14 years died in 2 weeks from the secondary effects of 6 grs. (0.39 gm.) of calomel.[4] In another instance of calomel poisoning in which the administration was hypodermic, life was prolonged for 24 days.[5] In a poisoning by mercuric sulfate death occurred in 22 days.[6] Kaufmann[7] has reported the death of a woman in 19 days from the effects of 200–300 c.c. of four-per-cent. solution swallowed with suicidal intent. In an obstetrical case a woman died of sepsis and mercurial poisoning in 24 days.[8]

Symptoms.

Certain effects of the soluble mercurials when taken by the mouth are immediate. During the act of swallowing the acrid, metallic taste of corrosive sublimate is perceived, and at the same time, or within a very few minutes, a burning sensation is experienced in the throat and gullet. These are not only important points of distinction between arsenical and mercuric poisoning in its initial stage, as arsenic is practically tasteless, and the burning sensation in the throat which it sometimes produces only appears much later; they also may serve, and have served to attract the attention of an intended victim (unless the poison be enclosed in a capsule or wafer) and thus frustrate an attempt at murder. The taste and action upon the œsophagus are more pronounced if the mercurial be in solution, but they are

[1] Walker: Brit. M. Jour., 1885, ii., 599.

[2] Plant: Obstetr. Gaz., Cincin., 1879, i., 489.

[3] Smith: Pac. M. and S. Jour., 1875, xvii., 448. Belfield (Med. Rec., N. Y., 1886, xxx., 197) has reported the death from mercurial poisoning of a man of seventy years which occurred in twenty-three days after cessation of the surgical use of the bichlorid.

[4] London M. Gaz., 1836, xviii., 484.

[5] Runeberg: Finska läk.-sälsk. handl., Helsingfors, 1888, xxx., 626.

[6] Ludwig: Wien. kl. Wochenschr., 1890, No. 28, 32.

[7] Jahresb. d. schles. Ges. f. vaterl. Kult., 1889, lxvii., 23.

[8] Braun: Wien. med. Wochenschr., 1886, xxxvi., 740.

also observed when it is taken in the solid form if it be soluble. Indeed, while the total duration of a fatal mercurial poisoning notably exceeds that of an arsenical intoxication in most cases, the initial stage of the former is much more rapid and intense than that of the latter. When corrosive sublimate is swallowed violent symptoms usually follow within less than 5 minutes. In exceptional cases this interval has been extended to 10 or 15 minutes. In Hort's case[1] a man took half a teaspoonful of mercuric chlorid in warm water in mistake for tartar emetic (!) and did not vomit during the following quarter of an hour. In Heidgen's case[2] a woman took 100 c.c. of a half-percent. solution (7¾ grs.) and only vomited when emesis was otherwise provoked, 15 minutes afterward. She had, however, taken a quantity of opium shortly before.[3] In two instances 10 minutes elapsed after the ingestion of the poison, before the manifestation of violent symptoms.[4] In a woman who received 0.75 gm. (11½ grs.) by rectal injection purging was produced in 5 minutes and vomiting in 10. She died in 5 days.[5] When white precipitate is taken the interval is longer than with corrosive sublimate, and may be extended to half an hour;[6] and with less soluble mercurials two hours may elapse before their action becomes manifest.[7] Within a few minutes the mucous membrane of the tongue, mouth, and pharynx swells and becomes coated with a gray film. There is violent, almost continuous, vomiting of gray mucoid material, frequently containing blood and shreds of mucous membrane, attended with pain in the stomach of a burning character. Soon the vomiting is accompanied by purging, the stools being liquid, dysenteric, bloody, extremely fetid, very copious, sometimes passed involuntarily, and attended with severe tenesmus, rectal pain, and colic. The abdomen is sometimes painful and meteorized. When the mercurial is absorbed by other channels than the mouth, as in obstetrical or surgical poisonings, the first symp-

[1] Tardieu: *Op. cit.*, 691.

[2] "Sublimatintoxication," Diss., Bonn, 1890, p. 24.

[3] In a case reported by Sheehy (Med. News, Phila., 1891, lix., 512) the interval is said to have been half an hour, but the statement is upon insufficient hearsay evidence.

[4] Thornton: Therap. Gaz., Detroit, 1893, 3 s., ix., 368. Delaunay: "Intox. par le sublimé," Thèse, Paris, 1893, p. 73 (taken in powder in a wafer).

[5] Huber: Ztschr. f. kl. Med., 1888, xiv., 459.

[6] Taylor: "Poisons," 3d Am. ed., 380.

[7] Ord: Lancet, 1888, ii., 570.

toms are those referable to the intestine just described, which may be manifest in a few minutes. The mouth and throat symptoms and epigastric pain are absent or only appear later in the history. Vomiting may occur, but is neither so constantly present nor so severe as when the poison has been swallowed.

A prominent symptom, and one present even in cases having a rapidly fatal termination[1] is diminution or total suppression of urine. This may begin almost at the outset of the poisoning and continue during one of long duration. Thus in Ludwig's case,[2] in which mercuric sulfate caused death in 22 days, there was complete anuria from the beginning, although, under the influence of pilocarpin, upon two days there were copious perspiration and the elimination of 900 c.c. (about 30 ℥) of urine. In other cases the suppression is gradual, as in Kaufmann's case[3] of suicide by mercuric chlorid, fatal in 19 days. On the third day the quantity of urine was scanty, and on the fourth there was complete anuria, after which only small quantities of urine were eliminated under the influence of digitalis and potassium acetate. In one case of suicide by mercuric nitrate in a man of 73 years, who died in 23 days, anuria did not occur,[4] but it must be remembered that the action of this salt is that of a corrosive rather than that of a mercurial poison. The small quantities of urine which are passed are albuminous, respond to the tests for mercury, and deposit casts and blood corpuscles.

The pulse is accelerated, small, irregular, and easily compressed. The temperature is subnormal. In one case, fatal in 9 days, it fell as low as 33°.4 (92°.12 F.) on the second day before death.[5] Even when the poisoning occurs in an individual in a febrile condition the temperature usually falls below the normal. Thus in a woman who died in eight days from mercurial poisoning caused by uterine irrigation while she had a temperature of 41° (105°.8 F.), the temperature on the second day had fallen to 36°.4 (97°.5 F.).[6] But if the febrile condition be sufficiently intense, although the temperature is reduced, it may not become subnormal. Thus in a woman who died in seven

[1] Maschka: Vrtljschr. f. d. prakt. Heilk., 1877, cxxxvi., 38.
[2] *Loc. cit.*
[3] *Loc. cit.*
[4] Prévost: Rev. méd. de la Suisse rom., 1882.
[5] Löwy: Wien. med. Presse, 1874, xv., 793.
[6] Steffeck: Centbl. f. Gynäk., 1888, xii., 65.

days after mercurial irrigation while having a temperature of 40° (104° F.) the temperature only fell to 37°.3 (99°.14 F.) on the day preceding death.[1]

Upon the second or third day, or even later, there are salivation, stomatitis, gingivitis, with loosening of the teeth, and enlargement and inflammation of the salivary glands. These mouth symptoms vary greatly in intensity. Sometimes, even in prolonged cases terminating in death, there is no salivation.[2] In other cases they progress to extensive ulceration and necrosis, and may become the cause of death secondarily from septic poisoning. Indeed, in a man of 25 years an external application of black-wash to a chancre caused soreness of the gums and profuse salivation.[3] In rapidly fatal cases mouth symptoms other than those due to local action are absent.

Toward the end in prolonged cases there are somnolence, cramps in the muscles, abolition of the reflexes, paralyses, convulsions, and death in a manner similar to that from uræmia. Or death may occur in collapse at a much earlier period, as in Maschka's case,[4] in which a woman fell unconscious shortly after having taken a large quantity of bedbug poison, was received at the hospital in a condition of collapse, and died of œdema of the glottis five hours after having taken the poison. In this rapidly fatal case there were dysenteric evacuations and suppression of urine, but no salivation.

Localization and Elimination.

Ludwig found from comparative analyses, as well with human subjects as with experiments upon dogs, that the quantity of mercury in the large intestine is greater than in the small intestine, whether it be taken by the mouth or administered hypodermically, except when death follows the ingestion rapidly, when the quantities in the small intestine and in the stomach are greater than that in the large intestine. The quantity in the liver was always large, and that in the kidneys and

[1] Frantzen: St. Pet. med. Wochenschr., 1890, n. s., vii., 217.

[2] Roberts: M. Times and Gaz., 1859, xviii., 210. Ullrich: Wien. med. Presse, 1883, xxiv., 1381. Walker: Brit. M. Jour., 1885, ii., 599.

[3] Lucas: Ind. Med. Gaz., Calc., 1886, xxi., 79.

[4] *Loc. cit.* and "Handb. d. ger. Med.," ii., 301.

muscles still greater; the spleen contained less and the bile little and sometimes none; the thyroid, the lungs, and the brain contained small quantities, and the bones mere traces. In later analyses he found the kidneys to contain the maximum amount, and the liver the next largest quantity. In the large intestine certain portions were found to contain ten to twenty times as much as neighboring portions of the same viscus.[1]

In a case of homicidal poisoning by arsenic in which the deceased had been given blue pills by the medical attendant twelve days before his death, Porter[2] found 0.872 gr. (0.0565 gm.) of mercury in the tissue of the stomach, but does not refer to its presence in the liver.

Once absorbed, the elimination of mercury from the system is extremely slow. Kobert places the average time required for complete elimination at six months;[3] and in a poisoning by external application of mercurial ointment Lamberg[4] found mercury in the urine four months after the absorption.

Prognosis and Treatment.

The **prognosis** in poisoning by corrosive sublimate by the mouth is not favorable. Of 145 cases of reported accidental or suicidal poisonings 90, or 62.1 per cent., terminated in death, and 55, or 37.9 per cent., in recovery, and among the latter are several cases of severe poisoning by minute doses. With white precipitate the percentage of mortality has been much less: of 16 non-homicidal cases in which it was swallowed only 3 were fatal. On the other hand, mercuric nitrate caused death in all of the 7 cases in which it was taken internally.

In the **treatment** of acute poisoning by the stronger mercurials albumin should be administered in the form of white of egg or of large quantities of milk, preferably the former. As, however, the mercury albuminate formed is soluble either in solutions of the alkaline chlorids or in excess of albumin, the administration of albumin should be followed by emesis, which usually occurs spontaneously from the effects of the poison, or may be provoked by mechanical means or by the administration

[1] Ztschr. d. œst. Apoth. Ver., 1889, 54; Wien. kl. Wochenschr., 1890, iii., 534, 552, 572, 615; Wien. med. Presse, 1892, No. 47.

[2] Tr. M. Soc., N. Y., 1862, 152.

[3] "Intoxikationen," 1894, p. 270.

[4] Upsala läk. Förh., 1867–68, iii., 681.

of an emetic (apomorphin hypodermically) if the case come under treatment early. The stomach pump (or siphon) is, however, to be preferred to emetics, provided the pipe be soft and carefully introduced, as the corrosive action of the mercurials does not produce that disintegration which is caused by the mineral alkalies and acids (including mercuric nitrate), yet it causes lesions which with an emetic may produce rupture and sudden death in collapse, particularly if the case be seen late. The best plan is to administer white of egg, and then to wash out the stomach with albuminous water, holding magnesia usta in suspension. An excellent antidote, theoretically, is freshly precipitated ferric sulfid, but it is not available outside of a chemical laboratory. In the subsequent treatment digitalis, pilocarpin, and stimulants are indicated. Salt or any article containing it is to be avoided, as it favors absorption of the mercury. The maintenance of nutrition is difficult, as the duration of the poisoning is long, and not only is food given by the mouth rejected, but rectal nourishment is difficult from the action of the mercurial upon the large intestine.

Post-Mortem Appearances.

When death has been caused by a mercurial taken by the mouth the most noteworthy appearances found at the autopsy are in the alimentary canal and the kidneys. The appearances in the former vary according as the case has been one of rapid or of prolonged course. In rapidly fatal cases there is evidence of intense acute inflammation of the mouth, œsophagus, and stomach. The tongue is swollen and deprived of epithelium in part, and the mucous membrane of the buccal cavity, pharynx, and œsophagus is swollen, injected, partly loosened or in shreds, gray-white and corrugated. The œdema may extend to the glottis. The gastric mucous membrane is highly reddened, ecchymosed, or even necrotic in parts, easily detachable or detached in shreds and easily torn. The muscular coat is also softened. Although it is said by Seidel[1] and others that "perforation is among the rare occurrences," we can find no instance in which it has been observed, yet it might be caused by mercuric nitrate solution containing a large excess of acid. A

[1] Maschka: "Handb. d. ger. Med.," ii., 298.

black color of the gastric mucous membrane is considered by Seidel as of questionable possibility, yet it has been observed in several cases,[1] in one of which (Downs) death followed the ingestion in forty hours. In very rapidly fatal cases the intestines may be normal; but in most instances the lesions there are more intense than those found in the stomach, particularly in the large intestine. The appearances are those of a diphtheritic enteritis; the mucous membrane is highly injected, reddened, detached in shreds, marked with numerous eschars, and the submucosa also reddened and injected. Frequently also there are evidences of peritonitis.

In cases of more prolonged duration the appearances in the alimentary canal are those of a more advanced inflammatory state. The mouth and cheeks may be the seat of extensive ulceration and necrosis. The gastric mucous membrane is slate-gray, ulcerated, or necrotic. Whatever the channel of entrance of the mercurial may have been, the changes in the large intestine are met with, if it cause death. They are similar to those met with after death from dysentery.[2]

The kidneys are large and soft, the cortex is pale yellowish-white and sharply defined from the red medullary portion. The organ grits on section, and the cut surface presents yellowish-white spots and streaks, which are produced by deposits in the straight and convoluted tubes of amorphous or nodular masses of calcium carbonate and phosphate, from the former of which escape of bubbles of gas may be observed when a section is treated under the microscope with sulfuric acid, when crystals of calcium sulfate may also be formed. There are other evidences of acute parenchymatous nephritis, necrosis and fatty degeneration of the epithelium, and the presence of casts, hyaline, epithelial, or blood, in the tubules.[3]

Among the other lesions observed are ecchymoses of the endocardium and degeneration of the heart muscle; injection of

[1] Seidel: *Loc. cit.* Valentine: Edinb. M. and S. Jour., 1818, xiv., 468. Wade: Lancet, 1848, i., 500. Fagerlund: Vrtljschr. f. ger. Med., 1894, 3 F., viii., supplhft., 70. Westrumb: Mag. f. d. ges. Heilk., 1834, xliii., 448. Downs: Tr. M. Soc., N. Y., 1861, 69.

[2] See Virchow: Berl. kl. Wochenschr., 1888, xxv., 72. Runeberg: Deut. med. Wochenschr., 1889, xv., 4.

[3] See Kaufmann: "Die Sublimatintoxication," Breslau, 1888. Leutert: "U. d. anatom. Veränderungen d. Sublimatintoxication," Berlin, 1895, and the references therein contained.

the tracheal mucous membrane; an inflammatory reddening of the marrow of the bones; and, in prolonged cases, fatty degeneration of the liver.

After the prolonged use of mercurials in medicinal doses Williams[1] has observed a blackening of the large intestine, caused by deposition of mercuric sulfid in its submucous tissue.

CHRONIC MERCURIAL POISONING.

This form of poisoning occurs in operatives who have to deal with metallic mercury, and is produced by inhalation of the vapor which it gives off at all temperatures. It has been observed in the operatives engaged in the mining and separation of the metal at Almaden in Spain, at Idria in Austria, in Peru, California, China (Kwei-chan), and in the Ural mountains.[2] Other industries in which the operatives are exposed to mercurial vapors are in the manufacture of mirrors,[3] of percussion caps,[4] of barometers and thermometers,[5] of incandescent electric lamps,[6] also goldsmiths, fire gilders, gold "retorters,"[7] and in hatters and furriers who handle the nitrate.[8] A "mass poisoning" of some 200 sailors on the British ship *Triumph* in 1809 was caused by inhalation of the vapor from a quantity of

[1] Tr. Path. Soc., London, 1867, xviii., 111.

[2] Baaz: Wien. med. Presse, 1886, xxvii., 711, 741, 774, 879, 944, 977, 1010, 1104, 1133. Raymond: Progrès méd., Paris, 1884, xii., 1017.

[3] Percival: Edinb. M. and S. Jour., 1813, ix., 32. Kalb: "Ein Fall v. lethalem Mercurialismus," Diss., Erlangen, 1876. Koch: Protoc. d. Ver. d. Kreis. v. Mittelfrank. Aerzte, Nürnb., 1854. Bayer: Arch. f. med. Erfahr., 1820, i., 115. Mayer: Friedreich's Bl. f. ger. Med., 1884, xxxv., 176, 285. Schoull: Ann. d'hyg., 1882, 3 s., viii., 261. Draper: N. Y. M. Jour., 880, xxxi., 626. Wollner: Münch. med. Wochenschr., 1892, xxxix., 533, and in Penzoldt and Stintzing: "Handb. d. spec. Therap.," 1895, ii., 114. According to the last-named the disease has been exterminated at the factory at Fürth, where during 1885 there were 5,463 days of mercurial illness among the operatives, in 1890 only 148, and in 1891 none.

[4] Chevallier: Ann. d'hyg., 1844, xxxii., 322. Fancher: Rev. san. de Bordeaux, 1886, iii., 165. Marie and Lande: Rev. d'hyg., Paris, 1885, vii., 16. Bamberger: Deut. Klinik, 1850, 110. Lancereaux: Gaz. d. hôp., 1891, lxiv., 417.

[5] Pleischl: Œst. Ztschr. f. prakt. Heilk., 1856. Schmitz: "Ueber Quecksilbervergiftung," Diss., Berlin, 1869, p. 22.

[6] Donath: Wien. med. Wochenschr., 1894, xliv., 888.

[7] Schmitz: *Op. cit.*, p. 26. Chapin: Am. J. Insanity, 1863, xx., 335. Merget: Rev. san. de Bordeaux, 1884–85, ii., 33. Whiteley: Rep. Med. Off. Privy Council, 1863, p. 358.

[8] See Mercuric Nitrate, p. 557; also Reiz: Ztsch. f. Staatsarznk., 1829, xvii., 381. Letulle: Ann. d'hyg., 1889, 3 s., xxi., 169.

mercury which had broken loose in the hold.[1] Chronic poisonings have also been caused by the excessive administration of mercurials as medicines, although such cases are of infrequent occurrence at present (see also page 546), and from inhabiting rooms adorned with mirrors whose silvering was originally or has become defective.[2]

In chronic mercurial poisoning all of the symptoms observed in the acute form may be met with except those due to the primary corrosive action. To these the following are added: *Mercurial tremors*, severe trembling, first of the arms and hands, afterward of the muscles of the legs and trunk; *mercurial erethism*, a condition of hypersensitiveness, as well of the special senses as of general sensation and of mental impressions, particularly such as are unpleasant, and which may extend to insomnia, irregularity of the heart's action, and great weakness; *mercurial cachexia*, manifested in anæmia, loss of teeth, disturbance of digestion, defective capillary circulation, emaciation, and muscular atrophy; *mercurial necrosis of the jaws*, which follows the ulcerative processes in the mouth and leads to anatomical changes similar to those produced by phosphorus; *mercurial cirrhosis of the kidneys*, following upon the parenchymatous nephritis observed in acute poisoning.

Chronic mercurial poisoning may terminate in death from the kidney lesions, from exhaustion, or from the secondary effects of the intense stomatitis;[3] and has also resulted in insanity.[4]

ANALYTICAL.

If the systematic process have been followed in the manner previously directed (pages 152, 157) any mercury which may have been present in combination will be found in the residue VI. (page 159) as the black mercuric sulfid, which remains alone after extraction with hot dilute nitric acid. It must be remembered that, as has been shown by Lecco,[5] even corrosive subli-

[1] Burnett: Phil. Trans., London, 1823, 402. Edinb. M. and S. Jour., 1810, vi., 513; Med. chir. Rev. and Jour., 1823–24, iv., 1010. Paris and Fonblanque: "Med. Jur.," ii., 461.

[2] Neukirch: Berl. kl. Wochenschr., 1883, xx., 820.

[3] See several cases cited by Kalb: *Op. cit.*

[4] Chapin: *Loc. cit.*

[5] "Berichte," Berlin, 1886, xix., 1175; *ibid.*, 1891, xxiv., 928.

mate is readily reduced by organic substances to metallic mercury, and that this distils partly with vapor of water and resists the action of hydrochloric acid and potassium chlorate obstinately. Consequently the metal may be encountered in the distillate obtained in the search for volatile poisons, and that the residue I. (page 157) must be submitted to a further prolonged treatment with hydrochloric acid and chlorate to insure complete extraction of mercury, or preferably, treated according to Ludwig's method:[1] the materials are heated in a flask, fitted with a return condensor, with twenty per cent. hydrochloric acid until the solid matter is completely dissolved (two to four hours), the heat being first raised to boiling, and then maintained just below that point. It is then cooled to about 60° and potassium chlorate added in portions of 0.5 gm. until the dark fluid remains light. The cooled liquid is filtered, the filter washed, the acid reaction nearly neutralized with soda, and filtrate and washings repeatedly shaken with about 5 gm. of zinc dust for some minutes. The clear liquid, after deposition of the zinc, is decanted off and the zinc washed by decantation, first with water and then with water containing a few drops of soda solution, then again with pure water, and is finally collected in a funnel over a plug of glass wool, washed with alcohol and with ether, and dried by a current of air. The mercury is then separated from the zinc by distillation; a combustion tube open at both ends is bent at one end into a U tube and charged by pushing in an asbestos plug to near the U, behind this is a layer of freshly burnt lime in pieces as large as hempseeds, then a second asbestos plug, then a layer of granular cupric oxid, then another asbestos plug, then the dry amalgamated zinc dust (which is transferred with the glass wool and without loss, removing the last portions with unused zinc dust) and finally another asbestos plug. The tube is then mounted in a combustion furnace, with the U portion projecting and kept cold by immersion in water, and the other end connected with a gasometer or other contrivance from which a gentle current of pure, dry air is caused to traverse the tube in the direction toward the U. The lime and cupric oxid are then heated to redness, after which the zinc is more moderately heated (short of fusion), and the heat maintained for an hour. The U portion is then separated from the

[1] Ztschr. f. an. Chem., 1878, xvii., 395; 1881, xx., 475; 1891, xxx., 258.

remainder of the tube, dried by a gentle current of dry air and weighed. The mercury is then removed, the last portions by heat and a current of air, and the empty tube weighed. The difference is the weight of mercury in the material used.

If the presence of mercury be suspected and it is to be specially sought for, this process should be used in the first instance with the viscera, without previous treatment with hydrochloric acid and chlorate. The process can be also applied to the urine by simply acidulating a measured volume with hydrochloric acid and warming before shaking with zinc dust. Under unfavorable conditions 90 per cent. of the mercury present is recovered, under favorable conditions 97.5 to 98 per cent.

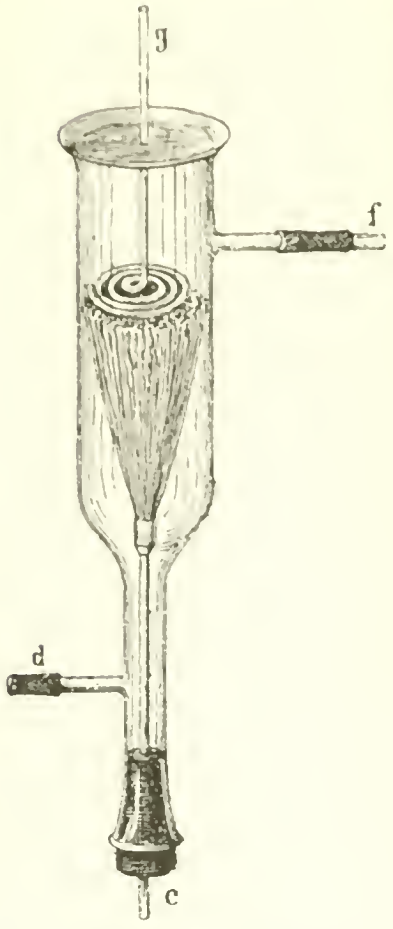

FIG. 26.—Wolff-Schneider Apparatus for Separation of Mercury.

Copper dust or gauze or brass wool have been used in place of zinc dust, in modifications of this method.[1]

In examinations of urine Mayer's method[2] may be used. It consists of treating a mixture of the residue of evaporation of the urine with quicklime and slacked lime in a combusion tube and condensing the mercury. Or the mercury may be separated from urine or other liquid, after acidulation, by electrolysis. Either by the simple method of immersing in it for twenty-four hours a bar of zinc, or stout copper wire upon which has been wound a spiral of dentist's gold foil in such manner that alternate spirals of gold and zinc or copper remain exposed; or, preferably, by Wolff's modification of Schneider's method.[3] The apparatus used

[1] See Fürbringer: Ztschr. f. an. Chem., 1878, xvii., 526. Güntz: *Ibid.*, p. 57. Teubner: *Ibid.*, 1880, xix., 198. Pashkis: *Ibid.*, 1883, xxii., 295. Lehmann: *Ibid.*, 1882, xxi., 472; 1884, xxiii., 109. Winternitz: *Ibid.*, 1889, xxviii., 753. Wolff u. Nega: Ph. Centrh., 1886, n. F., vii., 392. Böhm: Ztschr. f. physiol. Chem., 1891, xv., 1.

[2] Ztschr. f. an. Chem., 1878, xvii., 402.

[3] Repert. d. an. Chem., 1883, iii., 114.

consists essentially of a glass tube (Fig. 26) through which the liquid is allowed to flow slowly in at *d* and out at *f*, and which contains the electrodes. The cathode, *c*, consists of a bundle of gilded fine platinum wire, attached to a stout platinum wire; and the anode, *g*, of a spiral of stout platinum wire to the centre of which a handle of fine platinum wire is so attached that it hangs without contact in the conical cavity of the cathode. A current is used of sufficient amperage to yield 500 c.c. of H_2+O per hour. The mercury may be removed from the gold by heating the cathode, previously washed with water, alcohol, and ether, and dried in a tube drawn out to small calibre; or it may be converted into the red mercuric iodid by suspending the cathode, or the tube containing the sublimate, for a few hours in a bottle containing a few crystals of iodin. The quantity may also be determined by this method, by weighing the cathode before and after the operation. The Reinsch test may also be used for the separation and identification of mercury, and depends upon the same principles as Ludwig's method (see page 493).

Other Reactions of Mercury—MERCUROUS.—1. *Hydrochloric acid*, or a *chlorid*, produces a white precipitate of mercurous chlorid, Hg_2Cl_2, insoluble in water and in acids, which turns black on addition of ammonium hydroxid. 2. *Hydrogen sulfid*, a black precipitate, consisting of a mixture of mercury and mercuric sulfid, insoluble in alkaline sulfids, in dilute acids, and in potassium cyanid; partly soluble in hot nitric acid. 3. *Potassium hydroxid:* a black precipitate, insoluble in excess. 4. *Potassium iodid:* a greenish precipitate, converted by excess into mercury, which is deposited, and mercuric iodid, which dissolves.

MERCURIC.—1. *Hydrogen sulfid:* a black precipitate. If the reagent be slowly added the precipitate is at first white, then orange, and finally black. The precipitate, which is mercuric sulfid, is insoluble in nitric acid, even boiling, but dissolves readily in aqua regia. 2. *Ammonium sulfhydrate*, or *sulfid:* a black precipitate, insoluble in excess except in the presence of organic matter (see page 158). 3. *Potassium* or *sodium hydroxid:* a yellow precipitate, insoluble in excess. 4. *Ammonium hydroxid:* a white precipitate, soluble in great excess or in solutions of ammonium salts. 5. *Potassium iodid:* a

yellow precipitate, rapidly turning to salmon color, then to red, easily soluble to a colorless solution in excess of the alkaline iodid, or in great excess of the mercuric salt. 6. *Stannous chlorid*—in small quantity a white precipitate; in large quantity a gray precipitate; and, when boiled, deposits globules of metallic mercury.

PHOSPHORUS.

Elementary phosphorus is known in four different allotropic modifications. Of these two, the black and white varieties, are only formed in small quantity or under exceptional conditions and are not met with in commerce. The forms commonly used are the yellow and the red.

Yellow phosphorus, *ordinary* or *crystallizable phosphorus*, is sold in sticks about 1.5 cm. in diameter and 20 cm. long, which must be preserved under water. It is yellow in color and, unless acted upon by light, translucent; but after exposure to sunlight it becomes opaque from the formation of a film of the white variety upon the surface. It has the consistence of wax, being brittle when cold, softens when warmed, and fuses at 44° .3 (111 .7 F.). If heated in an atmosphere incapable of acting upon it chemically it boils at 290° (554° F.), being converted into a colorless vapor. It volatilizes at much lower temperatures, and water boiled upon it gives off steam charged with its vapor. When exposed to the air it gives off white fumes and produces ozone. It is luminous in the dark. It gives off a pungent odor, particularly when heated, which reminds one of the combined odors of garlic and ozone. It has a disagreeable, garlic-like taste.

Phosphorus is generally considered as insoluble in water. Its insolubility is, however, not absolute, as it has been shown by the experiments of Vohl[1] and of v. Bamberger[2] to diffuse in an unoxidized form with air-free water, through an animal membrane. The amount dissolved is, however, extremely small, being estimated by Hartmann[3] at 0.000227 gm. in 100 gm. of water. A much larger proportion may be present

[1] Berl. kl. Wochenschr., 1865, ii., 336.

[2] Würzb. med. Ztschr., 1866, vii., 41.

[3] "Zur acuten Phosphorvergiftung," Diss., Dorpat, 1866, p. 35.

in water, suspended in a finely divided condition; at all events, water which has stood over phosphorus is luminous in the dark and has the odor of phosphorus. According to Hartmann phosphorus is much more soluble in bile than in water, 100 gm. dissolving as much as 0.02996 gm. It is soluble in absolute alcohol to the extent of 1 part in 350. It dissolves more abundantly in carbon disulfid, sulfur chlorid, phosphorus trichlorid, benzene, ether, petroleum, acetic acid, and the fixed and volatile oils. On evaporation of its solutions it crystallizes in octahedra or dodecahedra.

Of the physical properties of yellow phosphorus, which is the only modification which is poisonous, the odor, taste, luminosity, and solubility are particularly of toxicological interest. Numerous cases are on record in which the attention of the intended victim has been attracted by one of the three first-named qualities. Indeed, the earliest recorded instance of attempted homicide by phosphorus[1] was unsuccessful because the man went in the dark to get a soup which his wife had prepared with rat poison for his use, and, observing its luminous appearance and its very pungent odor, submitted it to the authorities for examination in place of eating it.

Yet neither the odor, taste, nor luminosity is to be depended upon to reveal the presence of phosphorus. All of these characters may be absent or very much diminished in intensity. Phosphorus is not luminous in the presence of alcohol, and its taste and odor are readily masked, and the former is common to many substances. Consequently if the poison be mixed with an alcoholic drink this will not be rendered phosphorescent, and if the beverage be one of marked taste and odor, such as rum, the phosphorus taste and odor become too faint to be particularly noticeable.[2] On the other hand, phosphorescence may be observed in the absence of phosphorus. We have frequently observed this appearance most brilliantly in pieces of meat in which putrefaction had not advanced sufficiently to develop any odor or other appearance of taint; and such meat has been the cause of false accusation of poisoning honestly made, when an analysis has demonstrated the absence of any trace of phosphorus.

[1] In Germany in 1838. Schneider: Ann. d. Statsarznk., 1839, iv., 2 Hft., 205.

[2] See also Dannenberg's experiments quoted by Schuchardt in Maschka: "Handb. d. ger. Med.," ii., 184.

It may be claimed that phosphorus cannot be given in aqueous or alcoholic liquids by reason of its extremely slight solubility therein. It is proved, however, by a great number of suicidal deaths from phosphorus so taken that water by mere contact (and more rapidly and completely if warm than if cold) may take up sufficient of the poison, whether it be in solution or suspension, to cause the death of an adult. It will be taken up still more readily by liquids such as soup, coffee, and tea, which are swallowed warm, and its true solution will be favored by the presence of liquid fats or oils.

The most notable chemical character of phosphorus is the facility with which it combines with oxygen, the yellow modification much more readily than the red. The former ignites in air at 60° (140° F.) and burns with a bright flame, giving off dense white fumes so long as any oxygen remains. When finely divided it bursts into flame by the heat of its own oxidation.

The use of yellow phosphorus as a medicine was suggested by Kunkel (about 1721), who was the first to publish the method of its preparation in 1678.[1] During the latter part of the eighteenth century it seems to have been extensively used as a medicine, and is said to have been administered in doses of one or two grains (!). At the present time it is used medicinally in doses of 0.003–0.0006 gm. ($\frac{1}{20}$—$\frac{1}{100}$ gr.) in the form of a solution in almond oil, *Oleum phosphoratum*, *U. S. P.*, or preferably in cod-liver oil, or, less advantageously, in pill form (*Pil. phosphori*, *U. S. P.*). A solution of phosphorus in absolute alcohol is also sometimes used (*Thompson's solution of phosphorus*), but is not officinal. It is intended to contain $\frac{1}{20}$ gr. in each fluidrachm (0.003 in 4 c.c.). *Glover's chloroformum phosphoratum* is said to be a solution of 1 part of phosphorus in 40 of chloroform.

The principal use of phosphorus is in the manufacture of sulfur matches, which are tipped with a paste containing phosphorus, glue, an oxidizing agent, and some pigment. The quantity of phosphorus in match heads varies greatly. In Germany the proportion of phosphorus in the paste is limited by law to 10 per cent., yet this proportion is frequently exceeded and in-

[1] Brandt had previously obtained phosphorus in 1669, but kept the process secret.

creased to 14, 23, and even 32 per cent.[1] Sonnenschein[2] states that the quantity of phosphorus contained in 100 match heads is from 1.152 to 1.666 "gran" (0.0717 to 0.1036 gm. = $1\frac{2}{10}$–$1\frac{3}{4}$ troy grains), but, according to Schuchardt,[3] Böcker found the quantity as small as 0.0249 gr. (0.4 gm.) in 100 matches, and Ure as high as 0.1625–0.195 ($2\frac{1}{2}$ to 3 grs.); Cheveau[4] found 0.052 to 0.058 and Fischer[5] 0.05 to 0.075; while Gunning found the variations to extend between 0.012 and 0.062 gm.

Phosphorus is also extensively used in the preparation of rat pastes and vermin-killers. We have found *Coster's Rat and Roach Exterminator* to contain 2.13 per cent. of free phosphorus; *Parsons & Co.'s Vermin and Insect Exterminator*, 0.4 per cent., and *phosphor paste* 1.01 per cent. The label of the first-named preparation bears the words "not poisonous, no danger" (!). Two children were killed by it in Yonkers, N. Y., in November, 1894, and the proprietors were arrested on a charge of manslaughter. Blyth[6] gives analyses of five phosphorus vermin pastes containing from 1.2 to 4 per cent. of phosphorus.[7]

In only 10 out of 522 cases of phosphorus poisoning was the substance taken in a form other than match heads or vermin poison, and of these 10 only 2 have occurred in the past 40 years: one the case of a student who took phosphorus "to improve his memory" and died in 3 days;[8] the other that of a child of 2 years who died from the effects of phosphorized cod-liver oil given in medicinal doses.[9] It is patent, therefore, that phosphorus poisoning might be eradicated by the enactment and enforcement of legislation forbidding the use of yellow phosphorus in these articles. In Finland, where the phosphoric rat poisons are permitted although yellow phosphorus matches have been prohibited since 1872, there were 12 poisonings by phosphorus (rat poison) in the 14 years 1880–93, of which 2 were murders, 7 suicides, and 3 accidents.[10]

[1] Custer: "Fort mit dem Gift d. Phosphor-Zündhölzchen," Zurich, 1887, p. 20.

[2] "Handb. d. ger. Chemie," 29.

[3] Maschka: "Handb. d. ger. Med.," ii., 185.

[4] Ann. d'hyg., etc., 1880, 3 s., iv., 256.

[5] Pharm. Jahrb., 1892, 799.

[6] "Poisons," London, 1884, 197.

[7] The incorporation of the phosphorus in the paste is by no means uniform.

[8] Achergee: Ind. M. Gaz., Calcutta, 1887, xxii., 171.

[9] Aff. Métivier: Ann. d'hyg., etc., 1891, 3 s., xxvi., 517.

[10] Fagerlund: Vrtljschr. f. ger. Med., 1894, 3 F., viii., supplh., 80.

Red phosphorus, *amorphous phosphorus*, is produced from the yellow variety by maintaining it at a temperature of 240° (464° F.) to 280° (536° F.) for two or three days in an atmosphere of carbon dioxid, and washing the cooled product with carbon disulfid. In the commercial product this washing is rarely complete and small quantities of yellow phosphorus, from 0.6 per cent. downward, remain. This and other impurities may amount to 4.6 per cent.

It is a brownish-red, amorphous, tasteless, odorless powder, which does not fume in air or dissolve in the solvents of the yellow variety. It dissolves in molten lead, from which it separates on cooling in violet-black crystals. It is not luminous at ordinary temperatures, and only ignites when heated to a temperature approaching 250° (482° F.)

This form of phosphorus is practically non-poisonous, probably by reason of its insolubility. It has been administered to dogs to the extent of 200 gm. (nearly ½ lb.) in 12 days without causing poisoning.[1] A woman of 20 years attempted suicide by swallowing the heads of three boxes of Swedish matches, along with water in which they had been boiled. She suffered some abdominal pain, but recovered completely in a few days. The match paste contained a trace of yellow phosphorus and a small quantity of arsenic.[2]

In the better class of "snapping matches," which ignite when rubbed upon any rough surface, the paste contains red phosphorus and potassium chlorate incorporated with glue. In the "safety matches," which ignite only when rubbed upon a prepared surface, the match heads contain no phosphorus, but consist mainly of potassium chlorate and sulfid of antimony, the red phosphorus with some very fine sand and glue being attached to the box. Some German matches have no phosphorus either upon the match or box.

ACUTE PHOSPHORUS POISONING.

Statistics.—The history of the toxicology of phosphorus may be said to have begun with the introduction of phosphorus

[1] Sonnenschein : "Ger. Chem.," 25.

[2] Edeling : Svensk. läk. förh., 1879, 113.

matches in 1839.[1] Lewin in his monograph, published in 1866,[2] collected 44 cases, of which only 5 had occurred previous to 1840 and only 13 previous to 1850. Of these 44 cases 18 (40.9 per cent.) were suicidal, and 14 (31.8 per cent.) homicidal; in 19 the poison was taken in match heads and in 15 as vermin poison. At an earlier date, in 1855, Henry and Chevallier[3] had presented a monograph to the Society of Medicine of Toulouse in which they refer briefly to 68 cases, of which 42 were by matches and 19 by rat poison; 26 (38.2 per cent.) were homicidal and 21 (30.9 per cent.) suicidal. In Tardieu and Roussin's table of criminal poisonings in France in the years 1851–63 there are 170 cases of phosphorus poisoning in a total of 617 by all poisons (27.5 per cent.). We have consulted the reports of 521 poisonings.[4] Of these 110 were homicidal, 294 suicidal, 66 accidental, 2 by empirics, 4 taken to cause abortion, 1 taken as an experiment. In the remaining 44 the origin of the poisoning is not given.

Of the **homicidal** cases 47 occurred in Germany, 51 in France, 4 in Great Britain, 2 in Finland, 2 in Denmark, 5 in Belgium, and 3 in the United States. Death was caused in 66 cases; in 18 the poisoned food was not taken on account of its appearance or taste; in 11 instances the victim recovered; and the remaining 15 cases are reported as "attempts." The agent used was "rat poison" in 35 cases, match heads in 35, and unspecified in the remainder. In 65 instances the victims were adults, in 35 children; and in 15 two or more persons were poisoned at the same time. The poison was given in soup in 27 cases. In 1 case match heads were given to an illegitimate child by its mother in a pear; in another a child was given matches to play with, with criminal intent; in another a woman mixed match heads with her husband's chewing tobacco; in another a 15-year-old girl was made the ignorant agent of administration; and in another a woman destroyed her 15-year-old son by introduction of match heads into the rectum. Of the **suicidal** cases 179 were in Germany, 55 in France, 30

[1] Matches were made in 1830 by Walker, of Stockton, in England (Ph. J. and Tr., 1871, 3 s., ii., 41), but they were not in general use until the period mentioned in the text.

[2] Arch. d. path. Anat., etc., 1866, xxi., 506.

[3] Ann. d'hyg., etc., 1857, 2 s., vii., 414; viii., 208.

[4] To which may be added eighty-five cases reported in journals to which we have not access.

in Great Britain, 6 in Sweden, 7 in Finland, 4 in Belgium, 3 in Switzerland, 1 in Russia, 1 in Canada, 1 in Italy, 3 in New Zealand, and 3 in the United States. Only 80 of the suicides were men, as against 172 women.[1] Of the latter probably many took the poison with the object of provoking abortion rather than intending to destroy themselves.

Among the 66 **accidental** poisonings were 31 in which the victims were adults. In 5 of these the origin of the accident is not stated; eight adults ate food poisoned for rats, etc.; in 3 matches fell into food by accident; 6 were medicinal poisonings, of which 3 were anterior to 1826, 1 in a child which was destroyed by phosphorized cod-liver oil in 1891,[2] and 2 in adults.[3] In the remaining cases the origin of the poisoning was exceptional: a student died in 3 days from phosphorus taken "to improve his memory;"[4] a cook was severely poisoned by rat poison which she prepared upon a table from which she afterward ate her meal;[5] an English colonel and family suffered violent enteric pains after eating meat which had lain upon a surface upon which matches were scratched;[6] operatives in a match factory were poisoned by match paste put into a coffee pot for safe-keeping;[7] a man was killed by matches accidentally present in a cooking utensil;[8] a man died from drinking white wine in which match heads had been macerated "as a joke;"[9] two daughters of a match manufacturer died from eating bread which had been left in a basket along with a large quantity of matches;[10] a woman was killed by eating soup in which she had cooked "rat pills"[11] in mistake for barley;[12] a "man with an ostrich stomach" died from flavoring his diet of glass, leather,

[1] In forty-two cases the sex is not given.

[2] Aff. Métivier: Ann. d'hyg., etc., 1891, 3 s., xxvi., 517.

[3] A slight poisoning of a man of thirty-three years (Anstie: Practit., London, 1873, xi., 103); and the fatal poisoning of a woman (Ph. J. and Tr., 1879, 80, 3 s., x., 747).

[4] Achergee: Ind. M. Gaz., Calcutta, 1887, xxii., 171.

[5] Starcke: "Zur Casuistik d. acute Phosphorverg.," Diss., Jena, 1868, p. 11.

[6] Chevallier and Poirier: J. d. chim. méd., etc., 1858, 4 s., iv., 140.

[7] *Ibid.*

[8] *Ibid.*

[9] *Ibid.* See also Hebra: Phila. Med. Times, 1881–82, xii., 866.

[10] *Ibid.*

[11] The extent to which these rat pills are used in Germany is evidenced by the fact that the number of the Pharm. Ztg. for Nov. 12th, 1881, contains advertisements of five makers of them and of a machine guaranteed to turn out 40 kgm. per hour.

[12] Memorabil., Heilbronn, 1890–91, n. F., x., 513.

soaps, etc., with matches.[1] A girl of 15 years died in 7 days after having anointed her hands and face with "rat paste" for the purposes of a "dark séance."[2]

Lethal Dose.—The minimum lethal dose of phosphorus is not accurately known, as the quantity actually swallowed has been determined in a few cases only. It is certainly not large. Anderson[3] reported the case of a child of $1\frac{2}{3}$ years who died in 7 days after having sucked the heads of about 20 matches. Assuming that the paste was all swallowed, and contained the maximum amount of phosphorus (0.195 gm. in 100), the quantity taken was 0.04 gm. (0.6 gr.); Shephard[4] states that a child died from the effects of 8 match heads (0.0156 gm. = $\frac{1}{4}$ gr.?). In both of these cases the action of the poison was favored by the administration of oil as an antidote. In one of Lamprecht's cases[5] a healthy woman of 22 years died in 84 hours after having taken match heads estimated to contain 1 to 2 grs. (0.065–0.13 gm.) of phosphorus. Probably 0.05 gm. ($\frac{3}{4}$ gr.) of phosphorus, taken in a finely divided form, as in match heads or rat poison, or in solution, might cause the death of a healthy adult.

Blaschko[6] has reported the case of a girl of 18 years who took, with suicidal intent, the heads of 2,000 matches along with the hot water in which they had been macerated, yet recovered. Singer[7] relates the case of a woman of 21 years who made an unsuccessful attempt at suicide by swallowing the heads of ten packages of matches.

Duration.—This will vary in fatal cases from a few hours to several days, according as death is caused by collapse or by the more remote effects upon the liver and other organs. Usually life is prolonged during several days of intense suffering. Among 208 fatal cases in which the duration was accurately reported, 33 died during the first day; 170 in a period varying from 2 days to 2 weeks (16 in 2 days; 29 in 3; 34 in 4; 25 in 5; 22 in 6; 20 in 7; 6 in 8; 8 in 9; 2 in 10; 3 in 11; 3 in 12; and 2 in 14 days), the average duration being $5\frac{1}{4}$ days, and 5 in longer periods.

[1] Freyhan: Deut. med. Wochenschr., 1894, xx., 58.
[2] Lancet, 1890, i., 398.
[3] Lancet, 1871, ii., 189.
[4] *Ibid.*, i., 435.
[5] Friedreich's Bl. f. ger. M., etc., 1868, xix., 243.
[6] Vrtljschr. f. ger. Med., 1877, n. F., xxvii., 563.
[7] Prag. med. Wochenschr., 1883, viii., 509.

The shortest duration was 7 hours in a case reported by Jäderholm.[1] In 3 instances reported by Maschka[2] death occurred in collapse in 7 to 8 hours. The victims, suicides, were aged 50, 27, and 61 years. Hammer[3] reports the case of a woman of 46 years who destroyed herself in 9 hours with about 3,000 match heads (38 packages); and Langer[4] that of a man of 71 years who died in 9¾ hours after having taken with suicidal intent the heads of one package infused in coffee.[5] A most protracted case, whether death can be entirely attributed to the phosphorus poisoning which had occurred 8 months previously or not, was that quoted by Tardieu and Roussin from Brullé.[6] A woman destroyed herself by swallowing a piece of phosphorus paste of the size of a walnut and afterward half a pint of paraffin oil. After suffering from the usual primary symptoms she was discharged as cured in 12 days. After 5 weeks of immunity she was readmitted, suffering from jaundice, and died suddenly in 6 days, or 53 days after the poisoning.[7] In an early case[8] a man took 0.05 gm. of elementary phosphorus, afterward 0.1 gm. on the same day, and the following day 0.15 gm. in syrup (in all 0.3 gm. = about 4½ grs.), and died in 17 days. A man of 32 years died in 15 days from the effects of about 2 ʒ of phosphorus paste;[9] and a woman of 21 years in the same period after having taken the heads of two packages of matches.[10] Both were suicides. In severe non-fatal cases

[1] Hygiea, 1873, xxxv., 12. Svensk. läk. förh., 303. In four homicidal cases (v. Bünau: Vrtljschr. f. ger. Med., 1859, xvi., 305, one-half hour. Schaeffer: "Samml. ger. Gutachten," Berlin, 1848, 287, one and a half hours. Otto: Memorabilien, Heilbr., 1870, xv., 134, three to four hours. Kessler: Vrtljschr. f. ger. Med., 1866, n. F., iv., 271, four hours) the period *alleged* was shorter; and in one (v. Maschka: Vrtljschr. f. ger. Med., 1885, n. F., xliii., 17) as short. In a double suicide reported by Liman (Casper-Liman: "Handb. d. ger. Med.," 8te Aufl., ii., 479) both are said to have died in two hours. But so unusual a coincident seems rather to point to some other unrecognized cause of death.

[2] Wien. med. Wochenschr., 1884, xxxiv., 608.

[3] Prag. med. Wochenschr., 1889, xiv., 79.

[4] *Ibid.*, 1892, xvii., 451.

[5] Other accidental or suicidal cases fatal in less than a day are reported by Alexander (Berl. kl. Wochenschr., 1866, iii., 59), Chevallier and Poirier (*Loc. cit.*, two cases), Casper ("Handb.," 5te Aufl., ii., 513, 514, two cases), and Fagerlund (Vrtljschr. f. ger. Med., 1894, viii., supplh., 80).

[6] "Empois.," 2ème ed., 553.

[7] West: Lancet, 1893, i., 245.

[8] Julia Fontenelle: Rev. méd., 1829, iii., 429; *cf.* Orfila: "Tox.," 5ème ed., i., 84.

[9] Harris: Lancet, 1886, ii., 582.

[10] Rummel: Ztschr. f. rat. Med., 1868, xxxiii., 227.

the illness is frequently prolonged. Thus Knoevenagel[1] has reported a poisoning of almost 6 months' duration.

The **prognosis** in acute phosphorus poisoning is very unfavorable. Of 393 cases 284, or 72.1 per cent., terminated fatally. Among the recoveries are included 33 alleged homicides, in which the fact of administration is more or less problematical, and in 23 instances the cause of the illness is questionable, or the amount taken insignificant. Among 268 cases of severe, non-homicidal cases there were, therefore, 53 recoveries, or a mortality of 80.2 per cent. Thirty-four of the recoveries were in very severe cases, in which the hepatic symptoms were marked, and in most of which oil of turpentine was administered. If the statistics be derived from hospital cases in which suitable treatment has been followed, the prognosis becomes more favorable, although the percentage of deaths is still high. Thus in von Jaksch's 39 cases there were 16 deaths and 23 recoveries—a mortality of 41 per cent.[2] In a few instances recovery has been complete after the manifestation of violent primary symptoms within two or three days after the poisoning;[3] but in most instances some of the symptoms persisted for several weeks. In one instance recovery may be attributed to the fact that the intended suicide, a man of 63 years, took oil of turpentine with the match heads, believing thus to intensify their action.[4] In 2 cases in which suicides have taken powerful corrosives along with the phosphorus and have recovered from the effects of both, the action of the phosphorus appears to have been completely masked or absent. One of these was a girl who took a quantity of match heads containing at least 3 grs. (0.2 gm.) and attempted to swallow an ounce of sulfuric acid, but spat it out;[5] the other that of a woman who drank a mixture of match heads and caustic potash solution, and in whom the cardiac stricture produced by the latter was subsequently successfully dilated.[6]

Symptoms.—The clinical history of most cases of acute phosphorus poisoning may be divided into two distinct periods

[1] Berl. kl. Wochenschr., 1869, vi., 157.

[2] See p. 583.

[3] Smoller: Wien. med. Halle, 1863, iv., 346. Vetter: Arch. path. Anat., etc., 1871, liii., 168.

[4] Vetter: *Loc. cit.*

[5] Graff: Ztschr. d. Staatsarznk., 1842, xliv., 283.

[6] Schrötter: Aerztl. Ber. allg. k. Khaus., Wien (1886), 1888, 38.

which are frequently separated by an interval of apparent recovery. This interval may be of two or three days' duration, and during it patients have been frequently discharged from hospitals as cured. Or the intensity of the primary action may be so slight that the person affected only comes under medical observation after the second stage has been fully established. In some cases, indeed, the symptoms of the first stage have been insignificant in cases which have subsequently terminated in death.[1] On the other hand, instances are not wanting in which death has been caused by the violence of the primary effects within twenty-four hours after the taking of the poison, and before any of the symptoms of the second stage have been exhibited.[2]

There is usually an interval of from two to six hours after the ingestion, during which no effects are observed save the taste of the poison, which may or may not be experienced at the time it is swallowed, and a sense of warmth in the throat and stomach. Then follow pain of a burning character in the epigastrium, which varies greatly in intensity in different cases, frontal headache, which is severe and persistent, thirst, a subnormal temperature, and a small, soft, and easily compressible pulse. The expired air has an alliaceous odor, and there are eructations of gas having a similar character, and in dark places there may be a luminous appearance about the mouth, either from a luminosity of the expired air or from the adherence of particles of phosphorus to or about the lips, which, in exceptional cases, have been reddened or ecchymosed from the latter cause.[3] There is nausea, followed by vomiting of matters which have the odor and luminosity of phosphorus, and which may be tinged with bile.[4] Somewhat later there is purging, the fæces being dark and offensive, and sometimes luminous. The duration of this stage does not usually

[1] v. Sturck: Deut. Arch. f. kl. Med., 1884, xxxv., 488, Case III.

[2] See cases cited on p. 580, and Alexander: Berl. kl. Wochenschr., 1866, iii., 59. Casper: "Handb. d. ger. Med.," 3te Aufl., ii., 464.

[3] Cutler: Boston M. and S. Jour., 1862, lxvi., 393. Stevenson: Lancet, 1880, i., 644. Friedreich's Bl. f. ger. M., 1852, iii., 4 Hft., 73. Coupland: Lancet, 1879, ii., 309. Curtis: Boston M. and S. Jour., 1876, xciv., 433. Alexander: *Loc. cit.* Woodman: M. Times and Gaz., 1864, ii., 386.

[4] The luminous appearance is not observed if the stomach contain oil of turpentine or alcohol (see p. 591), even if phosphorus be present.

exceed two or three days. The symptoms usually abate in severity and there is the semblance of recovery, except in rapidly fatal cases.

In from two to ten days after the poisoning the second stage begins with jaundice, which increases in depth with the intensity of the poisoning, and a recurrence or exacerbation of the symptoms of the first stage. The vomiting and hepatic pain are very distressing. At the same time the area of hepatic dulness is notably increased, the increase in some cases being followed by contraction. The vomit contains blood, and there are hemorrhages from the nasal and other mucous membranes, and notably, in the female, from the vagina and uterus. There is intense headache, fever, an accelerated pulse, and death in coma or from exhaustion. Even in this later stage the breath is sometimes alliaceous,[1] and the vomit and dejecta may be luminous.[2] Priapism has been observed in the male[3] and sexual excitement in the female.[4] In some instances death is preceded by convulsions or delirium; in others it has occurred suddenly, during a condition of apparent improvement.[5]

Taussig[6] and von Jaksch[7] have shown that with the appearance of hepatic symptoms there is a notable transitory increase in the number of the red corpuscles in the BLOOD, in one case reaching 8,600,000 per cubic millimetre in a woman,[8] without an increase in the quantity of hæmoglobin, while the number of leucocytes is diminished. At the same time the alkalinity of the blood is notably diminished.[9]

The TEMPERATURE is slightly subnormal during the first

[1] Anderson: Lancet, 1871, ii., 189. Blaschko: Vrtljschr. f. ger. Med., 1877, n. F., xxvii., 563.

[2] Cayley: Lancet, 1883, i., 1041. Rothhammer: Diss., Würzb., 1890, p. 22. Lion: Wien. med. Presse, 1866, vii., 941.

[3] Cooper: Brit. M. Jour., 1858, 846 (Case II.). Solon: "Dict. de méd. et de chir.," xii., 707.

[4] Neuman: Wien. med. Halle, 1864, v., 134. Zeidler: Charité Ann., 1860, xi., 1 Hft., 1.

[5] Cocker: Med. Times and Gaz., 1882, i., 303. Krug: Arch. d. Heilk., 1865, vi., 566. Fritz, Ranvier, and Verliac: Arch. gén. de méd., 1863, ii., 25. v. Jaksch: In Nothnagel, i., 132, Case XXXVI. Reith: Brit. M. Jour., 1867, ii., 560.

[6] Arch. f. exper. Path. u. Pharm., 1892, xxx., 161.

[7] Deut. med. Wochenschr., 1893, xix., 10, and in Nothnagel's: "Spec. Path. u. Ther.," 1894, i., 146. In the latter (pp. 107–159) will be found an excellent discussion of the symptomatology and diagnosis, based upon forty cases observed during 1884–93 in the services of Nothnagel and von Jaksch at Vienna and Prague.

[8] Tausig: *Op. cit.*, p. 165. (Normal=5 mill. in man, 4 to 4.5 mill. in woman).

[9] Kraus: Ztschr. f. Heilk., 1889, x., 106. v. Jaksch: *Loc. cit.*

stage, frequently with a sharp and marked elevation, which may reach 39.4 (103° F.), which, however, continues only for one to three days, and is followed by subnormal temperature.

The URINE contains serum albumin in the earlier stages. With the establishment of the second stage it contains bile pigment; and the elimination of nitrogen, which had diminished somewhat at first, is enormously increased.[1] The quantity of phosphates eliminated is markedly increased from the first. The sediment, in the later stages, contains large numbers of epithelial cells and casts, which may be hyaline or epithelial, or blood casts, in any event tinged with bile pigment and the attached epithelium infiltrated with oil globules. Leucin and tyrosin crystals have been observed in the later stage in many instances, and these substances have also been identified by their chemical reactions.[2] The urine is also said to contain peptone in some instances,[3] and also lactic acid,[4] and an aromatic oxyacid.[5] The urine may also contain sugar,[6] but its presence has no connection with the action of phosphorus. In a case reported by Erman[7] the urine passed on the morning of the eighth day was milky from the presence of a notable amount of suspended fat (chyluria).

Diagnosis.—A positive diagnosis, in the absence of a clear history, can only be made during life from the detection of unoxydized phosphorus in the excreta or vomit.

The points of divergence between the symptoms of acute phosphorus poisoning and those of ACUTE YELLOW ATROPHY OF THE LIVER are that: 1. In the disease the liver rapidly diminishes in volume, while in most cases of phosphorus poisoning it increases in size. Yet this enlargement may not occur, or may be followed by diminution. 2. In yellow atrophy there

[1] von Sturck: Deut. Arch. f. kl. Med., 1884, xxxv., 481. Münzer: *Ibid.*, 1893–94, lii., 199.

[2] Wyss: Arch. f. path. An., 1865, xxxiii., 422. Rothhammer: Diss., Würzb., 1890, p. 22. Ossikowsky: Wien. med. Presse, 1872, xiii., 121–294. Fraenkel: Berl. kl. Wochenschr., 1878, xv., 265. Starke: Diss., Jena, 1868. Badt: Diss., Berlin, 1891. Liman: Casper-Liman, "Handb. d. ger. M.," 8te Aufl., ii., 487. See also Münzer and von Jaksch: *Loc. cit.*

[3] Maixner: Prag. Vrtljschr., 1879, cxliv., 75. v. Jaksch: Ztschr. f. kl. Med., 1883, iv., 413. Robitschek: Deut. med. Wochenschr., 1893, xix., 569.

[4] Schultzen u. Riess: Charité Ann., 1869, xv., i.

[5] *Ibid.*, and Baumann: Ztschr. f. physiol. Ch., 1882, vi., 192.

[6] Grose: Lancet, 1889, ii., 902. Huber: Ztsch. f. kl. M., 1888, xiv., 483.

[7] Vrtljschr. f. ger. Med., 1880, n. F., xxxiii., 61.

are usually severe cerebral symptoms, delirium, convulsions, etc., which are only exceptionally caused by phosphorus.

Nor can the distinction be absolutely made from HYPERTROPHIC CIRRHOSIS OF THE LIVER. In this disease, toward the end, there is frequently vomiting of masses containing blood, which symptom, although uncommon at this stage of phosphorus poisoning, has been observed in several cases.

The differentiation from INFECTIOUS ICTERUS—WEIL'S DISEASE,[1] is more obvious by the acute nephritis and the high fever of that disease.

In certain forms of SEPSIS and of PUERPERAL ECLAMPSIA the symptoms resemble those of phosphorus poisoning. In the former, however, the elevation of temperature is considerable and continuous, while in phosphorus poisoning the temperature is subnormal or only temporarily high. The chill of sepsis is also absent in phosphorus poisoning, and in the former condition enlargement of the liver is slight or absent, while that of the spleen is considerable; in the latter the reverse is the case. In puerperal eclampsia cerebral symptoms and nephritis are present, and the history of the case will aid in the differentiation. From VARIOLA HÆMORRHAGICA in the absence of the prodromal eruption and of high temperature of the disease, or the presence of phosphorus in the vomit, etc., a diagnosis from the symptoms is difficult.

Treatment.—Unabsorbed phosphorus, which may remain in the alimentary canal for several days after having been swallowed, is to be removed by emesis and later by purgatives. The best emetic is cupric sulfate in doses of 0.1 to 1 gm. (1½ to 15 grs.) in solution. It is to be preferred to zinc sulfate as it may serve in another manner by coating the phosphorus with copper. It is preferable, however, to wash out the stomach thoroughly as early as possible with a 0.1 to 0.3-per-cent. solution of potassium permanganate, and to leave about 500 c.c. (a pint) of the solution in the stomach. Diluted hydrogen peroxid (one to three per cent.) has also been recommended for washing the stomach.[2] The use of hot oil for this purpose as practised by Macewen[3] is not to be recommended, although his patient re-

[1] Deut. Arch. f. kl. Med., 1886, xxxix., 209.

[2] Thornton : Therap. Gaz., Detroit, 1893, 3 s., ix., 8.

[3] Glasgow M. Jour., 1872-73, 407.

covered, as the absorption of any phosphorus remaining in the stomach or passing into the intestine is greatly favored. The stomach having been washed out, unrectified French oil of turpentine is to be given in doses of 1 to 2 gm. (15 to 30 grs.) several times daily, either in capsules or in mucilaginous emulsion. The older and more acid the oil the greater is its tendency to unite with the phosphorus to produce the crystalline, spermaceti-like, and inert phosphorous acid compound.[1]

As the alkalinity of the blood is diminished by phosphorus, the administration of alkalies is indicated. Transfusion of defibrinated blood has been practised with benefit in some cases,[2] and Kobert[3] recommends transfusion of an alkaline solution of salt mixed with ten to twenty per cent. of water which has been agitated with oil of turpentine.

As phosphorus is soluble in the fixed oils and fats, all substances containing them should be rigidly excluded from the dietary and medication.

Post-Mortem Appearances.—The SKIN is yellow, and all internal organs are more or less jaundiced. Absence of icterus has only been observed in very few cases in which death occurred within less than two days.[4] The blood is dark and fluid, and in subacute cases does not coagulate.[5] MULTIPLE EXTRAVASATIONS of blood, either in the form of minute petechiæ or of larger ecchymoses, are found in various situations, most frequently in the pleuræ, pericardium, mediastina, subcutaneous and perimuscular cellular tissues, lungs, and mesentery.[6] In the female large hæmatomata are frequently found in the ovaries,

[1] See Thornton: *Loc. cit.* Rommelaere: Bull. de l'Ac. r. de méd. de Belg., 1871, 3 s., v., 1043–1121. Köhler: "Sauerstoffhaltiges Terpentinöl," etc., Halle, 1872. Busch: "Wirksamkeit des Terpentinöls," etc., Riga, 1892.

[2] Eulenberg and Landois: Deut. Arch. f. kl. Med., 1867, 440; Centbl. f. d. med. Wissensch., 1867, 280. Jürgensen: Berl. kl. Wochenschr., 1871, 241, 253, 292, 304, 364, 392.

[3] "Intoxikationen," p. 422.

[4] Jäderholm (*Loc. cit.*), seven hours. Baumann (Arch. d. Heilk., 1871, xii., 257), about twenty-four hours. Coupland (Lancet, 1879, ii., 309), forty-four hours. Paltauf (Wien. kl. Wochenschr., 1888, 513), two cases; one nine hours, the other about twenty-four hours after having been found poisoned. Although icterus is not specifically mentioned in the cases reported by Blix, and quoted by Rossbach (whose other alleged cases of absence of jaundice are misquotations), it is implied in the appearances mentioned (Hygiea, Stockh., 1865, xxvii., 115).

[5] See Corin and Ansiaux: Vrtljschr. f. ger. Med., 1894, 3 R., vii., 79, 212.

[6] See Hessler: *Ibid.*, 1882, n. F., xxxvi., 32. Lesser: "Atl. d. ger. Med.," pl. xvi., Figs. 1, 2.

and blood may be found in the uterus or escaping from the vulva.[1] In one of Casper's cases luminous vapors were observed to issue from the vagina, although the poison had been taken by the mouth. Death had occurred in twelve hours.[2] The MOUTH, PHARYNX, and ŒSOPHAGUS are rarely affected beyond being colored by the dark or black stomach contents which frequently exude. Exceptionally, however, the lips, internal surface of the cheeks, pharynx, and œsophagus have been found injected, reddened, deprived of epithelium, covered with fibrinous exudation, or luminous.[3]

On opening the abdominal cavity, or, more frequently, on opening the stomach or intestine, the garlic-like odor of phosphorus, and its luminosity in the dark, may be observed, although these appearances are by no means constant. If the odor of putrefaction be marked that of phosphorus is sometimes only observed after the gases of putrefaction have escaped. The odor is most frequently present in cases of comparatively short duration; it has, however, been detected in persons who have survived for 7,[4] 5,[5] $4\frac{1}{2}$,[6] and 4[7] days. In one case of death in 3 days the odor was present in the large intestine but absent from the stomach.[8] In two of three cases fatal in 8 hours, in all of which phosphorus was detected on analysis, the odor was present in the stomach; but in the third it was present in the intestine and absent in the stomach.[9] In a case fatal in $9\frac{3}{4}$ hours the odor was absent, although the contents of the intestine were luminous.[10] The luminous appearance is not observed in the presence of alcohol or of oil of turpentine. The STOMACH is congested, with infiltration of blood into the cellular tissue and ecchymoses into the submucous tissue, the mucous membrane swollen and softened and frequently marked

[1] Baader: Corr.-Bl. f. Schweiz. Aerzte, 1872, ii., 419.

[2] "Handb. d. ger. Med.," 5te Aufl., ii., 513.

[3] Liman: Casper-Liman, "Handb. d. ger. Med.," 8te Aufl., ii., 479. Starke: Diss., Jena, 1868, p. 25. Niemann: Ztschr. f. d. Staatsarznk., 1862, lxxxiii., 185. Casper: Vrtljschr. f. ger. Med., 1858, xiv., 185. Bünau: *Ibid.*, 1859, xvi., 305. Martin: Brit. M. Jour., 1878, i., 478. Tüngel: Deut. Klin., 1863. xv., 395, 402 (Cases XV. and XVI.).

[4] Anderson: Lancet, 1871, ii., 189.

[5] von Maschka: Vrtljschr. f. prakt. Heilk., 1867, xcvi., 26.

[6] Mowat: Lancet, 1891, ii., 1387.

[7] Werner: "Memorab.," Heilbronn, 1890–91, n. F., x., 513.

[8] Kieman: Ber. . . . Rudolph Stift., Wien, 1883, 407.

[9] von Maschka: Wien. med. Wochenschr., 1884, xxxiv., 608.

[10] Langer: Prag. med. Wochenschr., 1892, xvii., 451.

with patches of purple or black-gray. The peptic glands are at first swollen and filled with oil globules, in the later stages they are empty.[1] In some cases slight erosions of the gastric mucous membrane have been observed.[2] The contents of the stomach are liquid, "coffee-ground," or pitchy in consistence, dark or black in color, strongly acid, and sometimes contain large clots of blood.[3] The quantity of liquid in the stomach, and in the intestine as well, is sometimes sufficient to cause their distention and the escape of the liquid. In other cases the stomach is empty or nearly so. The INTESTINAL MUCOUS MEMBRANE is frequently unaffected. In other cases it is reddened and is the seat of alterations similar to those found in the stomach.

The LIVER is usually enlarged, sometimes to an enormous size.[4] In some cases, however, it has been found to be smaller than normal.[5] Its color is a bright or dirty yellow, with red spots or blotches.[6] It is friable, and pulpy in consistence. On section it is icteric, oily, and greases the knife. In one instance, however, it was described as perfectly bloodless, white, and of the appearance of having been boiled;[7] while in another it is said to have been very dark.[8] On microscopic examination it is found to be the seat of more or less advanced fatty degeneration, even in some of the cases of short duration.[9] The SPLEEN is enlarged in most instances, sometimes normal in size and occasionally diminished.[10] The KIDNEYS are somewhat larger than normal, smooth, and yellow or yellowish in color. On section the cortex is fatty, anæmic, yellowish, firm, and dry, the

[1] For colored plate see Lesser: "Atl. d. ger. Med.," pl. x., Figs. 1 and 2.

[2] Rothhammer: Diss., Würzb., 1890, p. 22. Bünau: *Loc. cit.* Leudet: Arch. gén. d. méd., 1857, 5 s., ix., 308.

[3] von Maschka: Vrtljschr. f. ger. Med., 1886, n. F., xlv., 232.

[4] Starke (*Loc. cit.*) death in four days; weight of liver, 4,300 gm. Robitsheck (Deut. med. Wochenschr., 1893, xix., 569): four days; 2,450 gms. Starling (Guy's Hosp. Rept., 1890, xxxii., 275): seven days; 2,115 gm. Henry (Boston M. and S. Jour., 1883, cix., 14): seven days, 1,770 gm. (normal, 1,450 to 1,700 gm.).

[5] Harris: Lancet, 1886, ii., 582. Kieman: *Loc. cit.*, Case III. Diesterweg: Diss., Greifswald, 1868, p. 18 (five cases). Schleiss-Löwenfeld: Diss., München, 1869, p. 12. Rummel: Ztsch. f. rat. Med., 1868, 3 R., xxxiii., 227. Wallis: Hygiea, Stockh., 1884, xlvi., 340. Langer: Med. Jahrb., 1881, 475. Ebstein: Arch. d. Heilk., 1869, x., 379.

[6] For colored plates see Lesser: "Atl. d. ger. Med.," pl. xvii., Figs. 1, 2. Kast u. Rumpel: "Anat. Path. Taf.," Pt. XI., D. III.

[7] Cutler: Boston M. and S. Jour., 1862, lxvi., 393.

[8] Bünau: *Loc. cit.*

[9] Maschka (Wien. med. Wochenschr., 1884, xxxiv., 608): three cases of death within eight hours.

[10] Hessler: *Loc. cit.*, p. 17.

glomeruli appearing as red spots; the medullary portion pale and yellow in striations corresponding to the straight tubes. Occasionally there are ecchymoses. The microscope shows the existence of fatty degeneration.[1] The BLADDER is empty or contains bloody urine. The HEART is fatty, pale; its valves yellow, and ecchymoses are found in the endocardium, pericardium, and intermuscular connective tissue. The LUNGS are normal, save for the presence of ecchymoses. The BRAIN and its coverings are in some cases normal, in others congested, with capillary hemorrhages and effusions in the arachnoid and ventricles. Microscopically fatty infiltration of the ganglion cells of the cortical portion, and of the walls of the capillaries, have been observed.[2] The same degenerative changes occur in the spinal cord.[3] Hemorrhages into the sheath and substance of nerve trunks have also been noticed.[4]

Analysis.—Although phosphorus is very readily oxidized in air it appears not only to remain in the elementary form in the human body for a long time, but also to interfere with the usual process of oxidation. It has been found in the unoxidized form in the cadavers of persons who have survived for six[5] and nine[6] days after having taken it. On the other hand a properly conducted analysis has frequently failed to reveal the presence of unoxidized phosphorus in much more rapidly fatal cases of unquestionable phosphorus poisoning. Under favorable conditions phosphorus sometimes remains unaltered for long periods in decaying cadavers. As early as 1848 Schaeffer detected phosphorus in the cadaver of a child which had been buried 14 days.[7] Wald[8] isolated fragments of the element by levigation from the stomach contents of a body buried 5 weeks. Elvers[9] separated 0.094 gm. of phosphorus from the intestines of a cadaver buried 42 days. Fischer and Müller[10] detected free phosphorus after 8 weeks, in cadavers of animals killed by it and

[1] For colored plate see Lesser: *Op. cit.*, pl. xvi., Figs. 3, 10, 11.

[2] Hammer: Prag. med. Wochenschr., 1889, xiv., 79. Elkins: Brit. M. J., 1891, ii., 1302.

[3] Gurrieri: Rif. med., Napoli, 1893, ix., pt. 4, 410.

[4] Reichel: Wien. kl. Wochenschr., 1894, vii., 155.

[5] Dutoit: Corr.-Bl. f. Schweiz. Aerzte, 1886, xvi., 359.

[6] Casper: "Clin. Novellen," p. 419.

[7] "Samml. gerichtsärztl. Gutacht.," Berlin, 1848, 287.

[8] "Gerichtl. Med.," 1858, i., 413.

[9] Vrtjschr. f. ger. Med., 1876, n. F., xxv., 25.

[10] *Ibid.*, 1876, n. F., xxiv., 1.

phosphorous acid after 12 weeks. Hoffmann,[1] in a case fatal in 8 hours, detected the element in the putrid intestinal contents after 2 months, and in another case, fatal in 3 days, after 5 months. Such instances are, however, exceptional.

Sometimes a careful examination of the stomach contents will lead to the discovery of fragments of phosphorus, usually in globules, in substance; or even of fragments of wood with adherent sulfur, pigment, and phosphorus, when match heads have been taken.

As phosphorus in the form of phosphates and in organic combination exists in extremely varying amount in different food articles and in the tissues and fluids of the body, its discovery or quantitative determination in this form is without forensic interest. That the analysis shall afford any affirmative indication of phosphorus poisoning phosphorus must be detected in the elementary form, or in its lower condition of oxidation as phosphorous acid, in which states it does not occur in the animal or vegetable world naturally. The fact that an organic mixture is luminous, without distillation, is not sufficient evidence of the presence of phosphorus.[2]

FIG. 27.—Absorbing Apparatus.

For the detection of free phosphorus a combination of the Mitscherlich process[3] with Scheerer's modification,[4] and Dussard's method[5] is best employed. This is conducted in its first and original stage as a part of the systematic analysis for volatile poisons in the manner described on page 128 and in the apparatus there figured. In the presence of free phosphorus, under favorable conditions, a bright phosphorescent light is seen at the point of greatest condensation when the liquid approaches the boiling point, and continues throughout the distillation.

[1] "Lehrb. d. ger. Med.," 5te Aufl., 679.

[2] See p. 573.

[3] Vrtljschr. f. ger. Med., 1855, viii., 1; J. f. prakt. Ch., 1855, lxvi., 238.

[4] Ann. d. Chem. u. Pharm., 1859, cxii., 216.

[5] C. rend. Ac. Sc., Paris, 1856, xliii., 1126. See also Blondlot: "Sur la recherche tox. du phosphore," etc., Nancy, 1861.

Care must be had that *all* reflexions of light are excluded; and a dim visibility of condensed steam which is sometimes seen in a slightly illuminated room is not to be mistaken for phosphorescence. When the phosphorescence is distinctly seen it is proof positive of the presence of elementary phosphorus. The phenomenon is, however, interfered with by many substances, among which are alcohol, oil of turpentine, carbolic acid, ammoniacal and cupric salts, which are likely to be present. If alcohol be the only interfering agent the luminosity will appear after about two-thirds of the liquid have distilled over. Should the phosphorescence not appear, the distillation should be interrupted after about one-third of the liquid has been distilled and the condenser (*e*, Fig. 2, page 128) disconnected. In its place an absorbing apparatus charged with solution of silver nitrate (Fig. 27) is substituted and the distillation continued gently, after the apparatus has been filled with carbon dioxid by opening the cock (*b*, Fig. 2), and a gentle current of CO_2 is maintained through the apparatus. Should the silver solution remain colorless the absence of phosphorus may be inferred. If a black deposit be produced it remains to prove that it consists of silver phosphid. For this purpose the apparatus shown in Fig. 28 is used. The Wolff bottle is charged with pure zinc[1] and dilute sulfuric acid; the tube *b* is filled with fragments of calcium chlorid. When the apparatus has been filled with hydrogen the pinchcock *d* is closed until the funnel tube *f* becomes three-quarters filled with liquid; then the pinchcock *e* is closed and *d* opened, and the black silver deposit, which has been collected on a filter and washed, is thrown into *f*; *e* is then

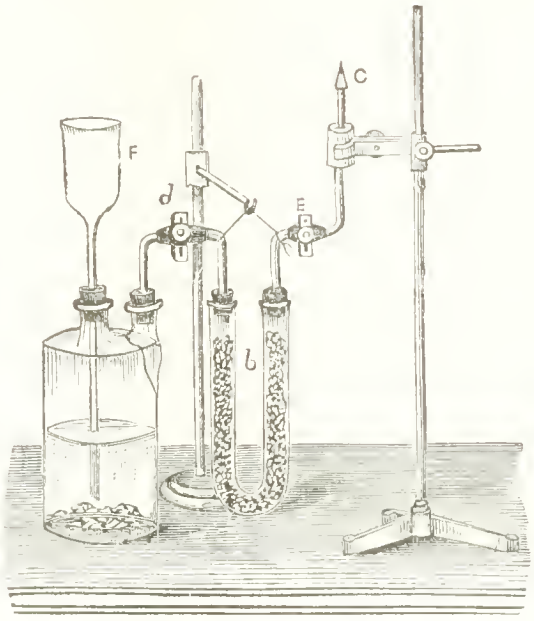

FIG. 28.—Apparatus for Examination of Silver Phosphid.

[1] A fair sample of the zinc used must be tested for phosphorus in blank in the manner described in the text.

slightly opened and the escaping gas ignited at the platinum tip *c*, the size of the flame being regulated by *e*. If the black deposit be silver phosphid a green core is seen in the flame, and when this is observed with the spectroscope it will show a spectrum of three bright lines in the green (Fig. 29). As phosphorous acid is decomposed by heat into phosphoric acid, which is non-volatile, and hydrogen phosphid, which is gaseous, the silver deposit and reactions obtained from it indicate, not the presence of free phosphorus, but that of either free phosphorus or phosphorous acid. Consequently it is preferable, if possible, to obtain the indication of free phosphorus from the luminosity and then, having started the CO_2 generator, collect the distilled

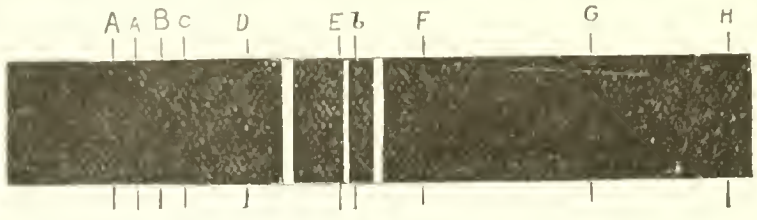

FIG. 29.—Spectrum of Phosphorus.

phosphorus in a closed flask, connected with a U tube charged with silver nitrate, through which the excess of gas bubbles. The phosphorus so distilled may be collected and weighed.[1]

Scheerer's process may also be used as an independent test. The materials to be examined are warmed in a bottle fitted with a cork to which are suspended two strips of filter paper, one moistened with silver nitrate (or sulfate) solution, the other with lead acetate. A darkening of the silver paper while the lead paper remains white indicates the presence of phosphorus or of phosphorous acid.[2]

Lafargue has proposed a method for the detection of very minute quantities of free phosphorus by extraction with ether and examination of the residue under the microscope upon a warmed plate in the dark, the instrument having been previously focussed.[3]

[1] See Dragendorff: "Ermittl. von Giften," 4te Aufl., 93.

[2] See Dragendorff: *Op. cit.*, 90.

[3] Union méd. de la Gironde, *ex* Friedreich's Bl. f. ger. Med., 1868, xix., 245.

CHRONIC PHOSPHORUS POISONING.

Although poisonings by repeated administration of small quantities of phosphorus may occur and have occurred,[1] the effects are practically the same as when the poison is taken in a single dose, owing to the slowness of its absorption in any case. The only difference noted is that the patient is worse after each administration.

By *chronic phosphorus poisoning*, or *lucifer disease*, a condition is usually understood which is peculiar to the operatives employed in the manufacture of yellow phosphorus matches. Its principal manifestation is necrosis of one or both maxillary bones. Owing to improvements in processes of manufacture, and particularly to the more extended use of red phosphorus, the disease is of much less frequent occurrence now than formerly.[2]

ZINC.

Poisonings by the zinc compounds are of rare occurrence, although apparently more common in England than elsewhere. Of 65 cases reported in medical literature 46 were in Great Britain, 6 in Germany, 5 in France, 3 in the United States, and 1 each in Belgium, India, and Australia.

The form of combination was either the chlorid or sulfate.

Zinc chlorid, $ZnCl_2$, is a white, hygroscopic solid, very soluble in water, used in medicine as a caustic. It has caused death in two instances when so applied to wounded surfaces.[3] Most of the poisonings by this agent have been accidental or suicidal (of 40 cases 27 were accidental and 7 suicidal). In 4 instances "soldering liquid," made by adding zinc to hydrochloric acid, was swallowed. In 26 cases some form of disinfecting

[1] In Ph. Jahrber., 1869, 463, an instance is referred to in which a woman repeatedly administered phosphorus in cakes to her child while it was in a hospital in France, as well as a similar case which occurred in Germany.

[2] For bibliography of lucifer disease see Index Cat. Surg. Gen. Libr. Wash., vii., 222, viii., 680, and Schuchardt in Penzoldt and Stintzing: "Handb. d. spec. Ther.," 1895, ii., 67, to which add Kocher: "Zur Kenntn. d. Phosphornecrose," Biel, 1893. Brouardel: Ann. d'hyg., etc., 1889, 3 s., xxi., 193. Caseneuve: *Ibid.*, 289. Pouchet: *Ibid.*, 168. Bührer: Hyg. Rundschau, 1893, iii., 372. Schüler: *Ibid.*, 1892, ii., 860.

[3] Stimson: Med. News, Phila., 1883, xlii., 572. Nichols: Boston M. and S. Jour., 1886, cxv., 343.

fluid containing the chlorid was taken: Burnett's solution (205 to 230 grs. per ounce) in 21 cases, Crew's in 2, and Morell's in 1. In an English case a woman was tried for the murder of her child by administration of a solution of zinc chlorid,[1] and in another it is probable that a similar administration by a woman to her child was intentional rather than accidental.[2] A non-fatal poisoning by Burnett's solution, sold in mistake for fluid magnesia, was the subject of a suit for damages in England in 1858.[3]

Zinc sulfate, $ZnSO_4$, 7 Aq.—*white vitriol*—is a white, crystalline solid, very soluble in water, sparingly soluble in dilute alcohol. Its solutions have an acid reaction and a strong styptic taste. It is very extensively used in medicine both externally as an astringent, and internally as an emetic.

In 8 of the 25 poisonings by zinc sulfate of which we find record, it was swallowed accidentally in mistake for Epsom salts (magnesium sulfate) which it closely resembles in appearance. In 5 instances it was taken with suicidal intent, and once to produce abortion. In one mass poisoning 60 convicts were more or less severely, though not fatally, poisoned by zinc sulfate added to milk, probably as a joke.[4] The salt was used in the prison in quantities in gilding picture frames. In one case a horse lotion was swallowed by a man by mistake. Four supposed homicidal cases are reported: 2 German,[5] 1 French,[6] and 1 English,[7] the victims having all been adults.

Lethal Dose.—The smallest quantity of the CHLORID known to have caused the death of an adult was about 3.5 gm. (55 grs.) taken in the form of two drachms of Burnett's solution.[8] A woman of 40 years died from the secondary effects of a quantity of Burnett's fluid containing about 100 grs. (6.5 gm.);[9] and a man of 55 similarly from two tablespoonfuls (about 14 gm. $ZnCl_2$) of the same liquid.[10] A child of 2 years died in 26½ hours

[1] Case of Mary Robinson (1849). Stratton: Edinb. M. and S. Jour., 1854, lxxxi., 497.

[2] Letheby: Lancet, 1850, ii., 23, and Stratton: *Loc. cit.*

[3] Richards *v.* Cocking: Ph. J. and Tr., 1858–59, xviii., 139.

[4] Lutier: Gaz. d. hôp., 1877, l., 676.

[5] Niemann: Ztschr. f. Staatsarznk., 1859, lxxvii., 219. Werres: *Ibid.*, 1832, xxii., 169, 313.

[6] J. d. chim. méd., etc., 1845, 3 s., i., 528.

[7] Herapath: Ph. J. and Tr., 1865, n. s., vii., 13.

[8] Cousins: Med. Times and Gaz., 1862, ii., 404.

[9] Markham: Tr. Path. Soc., London, 1858–59, i., 164.

[10] Ph. J. and Tr., 1867, n. s., viii., 420.

from the effects of a teaspoonful of a 50-per-cent. solution.[1] On the other hand a man of 54 years took a wineglassful of a solution said to have contained 88 grs. of zinc chlorid to the fluid-drachm (= about 90 gm.);[2] and in another case[3] three ounces of Burnett's solution (about 43 gm. $ZnCl_2$) were swallowed by a man without causing his death.

The smallest reported lethal dose of the SULFATE was 15.5 gm. (½ ounce), which quantity, taken in solution, caused the death of a man of 52 years.[4] But a woman of 35 years took 46 gm. (1½ ounces) in mistake for Epsom salt, and, although she did not vomit for a quarter of an hour or purge for half an hour, ultimately recovered.[5]

Prognosis.—So far as can be judged from the statistics the chlorid is fatal in a larger percentage of cases than the sulfate: 65.8 per cent. of the former to 50 per cent. of the latter.

Duration.—When death is due to the primary shock and collapse caused by the chlorid, particularly when it contains excess of acid, it may occur within a few hours. Thus a woman of 52 years died in about two hours from the effects of Burnett's solution,[6] and a man of 62 years in four and a half hours after swallowing two ounces of "soldering fluid."[7]

On the other hand, the primary effects may be recovered from and the patient may die weeks or months afterward from inanition or perforation. Deaths in eleven,[8] twelve,[9] and fourteen[10] weeks, and in three to four[11] and over six[12] months are reported.

In the few fatal cases of poisoning by the sulfate in which the duration is reported death has occurred in five days or less, if we except Herapath's case[13] in which it is supposed that the homicide was committed by repeated administrations of sulfates of zinc and iron. The shortest duration was in a man of 50

[1] Seydel: Vrtljschr. f. ger. Med., 1896, 3 F., xi., 286.
[2] Stratton: Edinb. M. and S. J., 1848, lxx., 335.
[3] Hassall: Lancet, 1853, ii., 159.
[4] Marsh: Med. Times and Gaz., 1862, ii., 252.
[5] Woodman and Tidy: "Forens. Med.," p. 226, Case XXIII.
[6] Brunton: Glasgow M. Jour., 1869–70, 5 s., ii., 514.
[7] Crosse: Brit. M. Jour., 1883, ii., 820.
[8] Jalland: Brit. M. Jour., 1887, i., 1387.
[9] Markham: *Loc. cit.*
[10] Wilks: Guy's Hosp. Rept., 1859, 128.
[11] Habershon: M. T. and Gaz., 1859, 525.
[12] Ph. J. and Tr., 1867, n. s., viii., 420.
[13] *Ibid.*, 1865, n. s., vii., 13.

years who died in two hours after having taken about 125 gm. (four ounces) of a mixture of sulfates of zinc and magnesium containing about seventy-five per cent. of the former.[1]

Symptoms.—Chlorid.—The action of this salt is essentially that of a corrosive. Consequently its effects are rarely delayed. Its styptic taste is experienced during the act of swallowing and is accompanied by a burning sensation in the mouth, throat, and stomach which increases in severity and extends in area, and is aggravated by pressure. Vomiting usually follows within a few minutes, particularly if the stomach contain food; in other cases it must be provoked by other means. Sometimes the lips and tongue are cauterized, most frequently not. There is severe thirst, and the voice is husky or extinguished. The countenance is pale, the expression anxious, and the forehead bathed in perspiration. Later there is purging, and in some cases the vomit and stools are tinged with blood. In rapidly fatal cases the prostration is great, and the patient soon becomes collapsed and dies. In more prolonged cases there is a more or less complete remission of all symptoms, and the patient may not come under medical observation until after the primary effects have disappeared and the secondary stage is established. This usually begins rather suddenly with a recurrence of the pain and vomiting, which in some cases continue obstinately for several days, while the victim becomes more and more emaciated and finally dies of exhaustion;[2] or large quantities of altered blood may be passed per anum and vomited, and the patient die in a few hours from syncope.[3]

In a fatal case from external application death was due to profound shock, and occurred in eight and a half hours, and the condition resembled that of narcotic poisoning.[4] In another instance, there was also narcosis, accompanied by vomiting. The patient died in six days, and the autopsy showed the existence of nephritis.[5]

In non-fatal cases recovery is slow and sharp relapses frequently occur.

[1] Buchner: Friedreich's Bl. f. ger. Med., 1882, xxxiii., 255.

[2] Finlay: Brit. M. J., 1889, ii., 15. Jalland: *Ibid.*, 1887, i., 1387.

[3] Willis: Assoc. M. J., 1855, ii., 743. Wilks: Guy's Hosp. Rept., 1859, 128.

[4] Nichols: Boston M. and S. Jour., 1886, cxv., 343.

[5] Stimson: M. News, Phila., 1883, xlii., 572.

SULFATE.—This salt does not act as a corrosive, but as an irritant. The styptic taste is observed on swallowing the poison, and is soon followed by persistent vomiting, pain in the mouth, throat and stomach, salivation, and diarrhœa. At first the pulse is accelerated, afterward it and the respiration become slower, there are attacks of vertigo and syncope, with cramps in the legs, convulsions, and death in collapse.

Treatment.—Until the stomach pump or siphon is obtainable vomiting should be favored by draughts of lukewarm water and tickling the fauces. Emetics should not be given. The stomach should be thoroughly washed out with water containing an alkaline carbonate in solution. In an emergency milk, white of egg, and materials containing tannin are beneficial by formation of zinc albuminate or tannate, but their use should be followed by that of the siphon. In the later treatment ice fragments may be given to control persistent vomiting, opium to allay pain and arrest diarrhœa, and stimulants to combat collapse. When the chlorid has been taken rectal alimentation may prolong life somewhat.

Post-mortem Appearances.—We find in medical literature reports of 16 autopsies after death from zinc compounds taken internally. In 11 the chlorid was the cause of death and in 5 the sulfate. In 4 of the deaths from the chlorid death occurred within twenty-four hours. The mucous membrane of the mouth, fauces, and œsophagus is deprived of epithelium, white, opaque, and swollen. The mucous membrane of the stomach is softened and disintegrated, at the cardia and greater curvature particularly, and toughened and greatly corrugated toward the pylorus. Its color varies from ashen gray to purple and blackish. The vessels are gorged with blood, the smaller capillaries as well as the larger vessels. There may or may not be ulcers or eschars. The contents consist largely of blood in some cases, in others the stomach is found empty or nearly so. The small intestine, particularly in its upper part, is congested and inflamed, and has been found thinned and eroded.

In one case of death from the effect of one and a half ounces of Burnett's solution the body, notwithstanding the high temperature of the weather at the time, showed no signs of putrefaction after forty-eight hours.[1] We do not believe, however,

[1] Crossing: Lancet, 1864, ii., 267.

that this condition can be properly ascribed to the preservative action of the small quantity of zinc salt present.

In one instance in which death occurred in eleven days, from sudden and copious hemorrhage, the stomach was so disorganized that the pyloric end was torn asunder in the attempt to remove the organ.[1]

When longer periods have elapsed before death, still more marked gastric lesions have been observed. The stomach has been found ulcerated and perforated, and in two instances the organ has been found replaced by a cavity made up of adhesions between surrounding organs.[2] In one instance the immediate cause of death was found in a constriction of the pylorus;[3] and in another fatty degeneration of various organs was observed.[4]

The observations of appearances after death from zinc sulfate are neither sufficiently numerous nor sufficiently detailed to show more than that an inflamed condition of the gastro-enteric tract, and fatty degeneration of the liver may be looked for.[5]

Analysis.—In the systematic process of analysis for mineral poisons any zinc present will be found in the liquid VIII. *a* (see page 160).

The TESTS of zinc are: 1. Potassium, sodium, or ammonium hydroxid—a white precipitate, soluble in excess; 2. Hydrogen sulfid—a white precipitate in neutral solutions. In the presence of mineral acids the formation of this precipitate is prevented except sodium acetate be also present; 3. Ammonium sulfid—a white precipitate, insoluble in excess or in sodium or ammonium hydroxid solutions or in acetic acid, but soluble in dilute mineral acids; 4. Ammonium carbonate, a white precipitate, soluble in excess; 5. Disodic phosphate, a white precipitate, soluble in acids or alkalies. Not formed in presence of ammoniacal salts; 6. Potassium ferrocyanid, a white precipitate, insoluble in hydrochloric acid.

Zinc may be present in human remains in small amount as a

[1] Willis: Assoc. M. Jour., London, 1855, ii., 743.

[2] Wilks: Guy's Hosp. Rept., 1859, 128. Jalland: Brit. M. Jour., 1887, i., 1387.

[3] Markham: Tr. Path. Soc., London, 1858-59, i., 164.

[4] Tuckwell: Brit. M. Jour., 1874, ii., 297.

[5] See Jones: Lancet, 1850, i., 342. Niemann: Ztschr. f. Staatsarznk., 1859, lxxviii., 219, 220. Marsh: M. Times and Gaz., 1862, ii., 252. Buchner: Friedreich's Bl. f. ger. Med., etc., 1882, xxxiii., 255.

consequence of the use of food which has been in contact with zinc or "galvanized iron" vessels; or it may be introduced post-mortem as a constituent of a disinfectant or embalming solution. In an instance reported by Mason it was shown that even after an application of a zinc-arsenic embalming liquid limited to the exterior of the cadaver, both poisons are found in the internal organs.[1]

The observations of Mazkewitz concerning the distribution of zinc after hypodermic injection of the acetate, as quoted by Kobert,[2] have led to results which, in view of their departure from that which is known concerning other metallic poisons, require confirmation.

[1] Jour. of Anal. and Appl. Chem., 1893, xv., No. 6 (reprint).

[2] "Intoxikationen," p. 283.

VEGETABLE POISONS.

ACIDS.

ACIDS OF THE ACETIC SERIES.

Formic acid, HCOOH—occurs in vegetable nature in the sting of the nettle and in the leaves of trees of the pine family; more abundantly in the acid secretions of red ants and in the stinging hairs of certain insects, and, probably accompanied by a more actively poisonous toxalbumin, in the secretion of the stinging glands of bees, wasps, etc. Formic acid, accompanied by higher acids of the same series, is also a product of the human economy,[1] and, according to the experiments of Pellacani,[2] the formates produce in the animal economy symptoms of paralysis and sopor similar to those observed in certain autointoxications.

We know of but one instance in which formic acid has been of forensic interest: that of a child of two days, whose death was due to natural causes, but upon whose cadaver were marks similar to those which might have been produced by a mineral acid, but which it was claimed were caused post mortem by ants.[3]

Acetic acid, CH_3.COOOH—is the acid of vinegar and is prepared in a more concentrated form by the destructive distillation of wood, and purification of the crude *pyroligneous acid.* The pure acid, known as *glacial acetic acid*, is a colorless, highly acid liquid which crystallizes at 17° (62°.6 F.) and boils at 119° (246°.2 F.).

Acetic acid intoxication has been rarely observed. Schuchardt[4] refers to six cases mentioned in the literature, including

[1] v. Jaksch : Ztsch. f. physiol. Chem., 1886, x., 536.

[2] "Therap. mod.," 1890, i., 65.

[3] v. Maschka : Vrtljschr. f. ger. Med., 1881, n. F., xxxiv., 193. This case was quoted in the discussion of the Harbaum case, of alleged murder by sulfuric acid, *ibid.*, 1882, xxvi., 211.

[4] v. Maschka : "Handb. d. ger. Med.," ii., 112.

two caused by the injection of *Liquor Villati* into purulent wounds.[1] Of the four cases of internal administration but three are particularly referred to.[2] The first of these is the only fatal case of which we find record, that of a woman of nineteen years who died in convulsions in a short time after having been found suffering in the street. At the autopsy no alterations were observed about the mouth and lips, although they were covered with a frothy, partly dried, brownish liquid. The stomach was distended, violet externally, almost black toward the pylorus; its mucous membrane not destroyed, grayish-white toward the cardia, brown to black toward the pylorus; and contained about eight ounces of a brown, black, slimy, fœtid liquid which yielded acetic acid on distillation. In Melion's case a teaspoonful of *radical vinegar* (glacial acetic acid) caused severe poisoning. In Birkett's case two to three ounces (60–90 c.c.) of a 33 per cent. acid, immediately caused acute laryngitis in a drunken man of forty years, who was rescued by tracheotomy and artificial respiration. David[3] reported the case of a woman who suffered severely from the effects of a quart of common vinegar swallowed with suicidal intent. A man of thirty-six years suffered instant and excruciating pain and great sickness from swallowing a teaspoonful of *aromatic vinegar*.[4] A case reported by Bojasinski is probably the one referred to by Kobert, although we have not access to the publication.[5]

The third to tenth acids of this series, *propionic*, *butyric*, *valerianic*, *caproic*, *œnanthylic*, *caprylic*, *pelargonic*, and *capric*, are volatile products of the decomposition of butter and other fats and oils, and probably have actions upon the economy similar to those of acetic and formic acids. The number of isomers increases from two of butyric acid, four of valerianic, eight of capric, etc.

A derivative of butyric acid, *β-oxybutyric acid*, CH_3-$CH(HO).CH_2.COOH$, has been shown to be produced in the

[1] Heine: Arch. f. path. Anat., 1867, xli., 24. The liquor of Villati contains 30 parts lead subacetate, 15 parts each of zinc sulfate and cupric sulfate in 200 parts of vinegar

[2] Orfila: Ann. d'hyg., 1831, vi., 159. Melion: Vrtljschr. f. d. prakt. Heilk., 1845, i., 86, and Birkett: Lancet, ii., 98.

[3] Brit. Am. J. M. Sc., Sept., 1847.

[4] London M. Times, 1865, ii., 471. A solution of essential oils in highly concentrated acetic acid.

[5] Medycyna, Warszawa, 1892, xx., 302. Kobert: "Intoxikationen," 221.

human economy in diabetes, and is considered as a causative agent of diabetic coma.[1]

HYDROCYANIC ACID, AND OTHER CYANIC POISONS.

As the various cyanic poisons—hydrocyanic acid, the cyanids, impure oil of bitter almonds, cherry-laurel water, etc., all owe their poisonous qualities to the presence in them or liberation from them of hydrocyanic acid, their action is practically the same in kind, and differs in intensity and rapidity according to the quantity of the acid present; being to some extent modified with some, such as potassium and mercuric cyanids, by the action of the material with which the cyanic radical is combined.

Hydrocyanic acid, *prussic acid*, *azomethan*, **CNH**, appears to exist preformed in the juice of the cassava (*Manihot utilissima*), the root from which "Brazilian arrowroot" and tapioca are obtained, which according to Francis,[2] contains an average of 0.0168 per cent. in the "sweet" variety, and 0.0275 per cent. in the "bitter." Most vegetable substances which yield hydrocyanic acid do not contain the acid as such, but contain a glucosid, **amygdalin**, having the composition $C_{20}H_{27}NO_{11}$ which, under the influence of **emulsin** (an enzym which they also contain) in the presence of moisture and a slightly elevated temperature, is decomposed into glucose ($C_6H_{12}O_6$), benzoic aldehyd (C_6H_5,COH), and hydrocyanic acid; 100 parts of amygdalin yielding 5.909 of prussic acid. Amygdalin exists in the bitter almond, the kernels of peach, apricot, cherry, plum, apple and pear pits, and in the bark, leaves, flowers, and fruit of the cherry-laurel, and of certain other plants, notably in those of the genera *Prunus*, *Cerasus*, *Sorbus*, and *Spiræa*.[3] Forty to sixty bitter almonds yield 0.05 to 0.07 gm. ($\frac{3}{4}$ to 1 grain) of hydrocyanic acid.[4]

[1] Stadelmann: Arch. f. exper. Path. u. Ph., 1883, xvii., 441. Minkowski: *Ibid.*, 1884, xviii., 35, 147. Külz: *Ibid.*, 1884, xviii., 291.

[2] Analyst, London, 1877, ii., 4.

[3] See Husemann and Hilger: "Pflanzenstoffe," 2te Aufl., 1017. Also Gresshoff (abst.): Ph. Jahrb., 1890, 14; 1891, 13. Thümmell: Apoth. Ztg., 1889, 518. Davidson and Stevenson: Practit., London, 1884, xxxii., 435. Loesecke: Leipz. Apoth. Ztg., 1871, 157. Jorissen and Hairs: Rep. d. Pharm., 1891, 416. Senior: Ph. Ztg., 1885, xxx., 779. Bettinck: Ph. Ztg., 1892, 135.

[4] See also Apoth. Ztg., 1890, 607.

Prussic acid is also an animal product and exists in the poisonous secretions of certain myriapods.[1]

Hydrocyanic acid and the cyanids used in medicine and in the arts are obtained synthetically, directly or indirectly, by the decomposition of a cyanid by a stronger acid, usually by the action of sulfuric acid upon potassium ferrocyanid.

The pure acid is a colorless liquid, has a penetrating odor resembling that of peach kernels; sp. gr. 0.7058 at 7° (44°.6 F.); crystallizes at − 15° (5° F.); boils at 26°.5 (79°.7 F.), and is more or less rapidly decomposed by exposure to light.

Since the first introduction of hydrocyanic acid into the materia medica by Borda in 1804, it has been used in degrees of concentration varying from 1 to 50 per cent.: Robiquet's acid was 50 per cent.; Koller's, 25 per cent.; Ittner's, Pfaff's and that of the French codex, 10 per cent.; Duflos', 9 per cent.; Scheele's, 4–5 per cent.; that of the Pharm. Bavar., 4 per cent.; Vauquelin's and that of the Edinburgh Pharm., 3.2–3.3 per cent.; that of the Dublin Pharm., 1.6–2.82 per cent.; those of the United States and British Pharm., 2 per cent.; Schrader's, 1.5 per cent.; and that of the Prussian Pharm., 1 per cent. At the present time the medicinal acid dispensed in the United States is invariably intended to be the U. S. P. acid, while in England both the Br. Ph. and Scheele acids are used. But in appreciating doses actually taken it must be remembered that hydrocyanic acid is decomposed by light, and that in samples not freshly prepared the percentage has diminished.

The crude *essential oil of bitter almonds*, whose principal constituent is benzoic aldehyd, C_6H_5COH, contains from 2 to 14 per cent. of hydrocyanic acid[2] which is *presumed* to have been removed from the *oleum amygdalæ amaræ*, U. S. P. The numerous poisonings which have been caused by the oil itself and by the *almond flavor* (which is an alcoholic solution) are sufficient evidence that the purification is usually incomplete in commercial samples.

The *aqua laurocerasi* or *cherry-laurel water* prepared by distillation from the leaves of the cherry-laurel, still official in

[1] Weber: Arch. f. mik. Anat., 1883, xxi., 468. Budge: Deut. Rev., 1883, viii., 401. Gulden steeden: Rec. de trav. Ch. d. Pays Bas, 1882, i., 282.

[2] Hager: Commentar z. Ph. German., ii., 456—two to five per cent. Blyth: "Poisons," 177—five to fourteen per cent.

the British and German Pharmacopœias, but not in that of the United States, contains quantities of hydrocyanic acid varying from 0.7 to 1 per cent. Madden in 1737[1] called attention to its toxic powers, and it was alleged to have been the agent used in the earliest English trial for murder by cyanic poisons.[2]

The *cyanids* of potassium, gold, and silver are extensively used in photography and in electroplating. The potassium salt, the one in most general use, has been the agent of a large proportion of the recorded cyanic poisonings.

The metallo-cyanids, such as potassium ferrocyanid, Prussian blue, Turnbull's blue, etc., are usually considered as non-poisonous.[3]

Hydrocyanic acid also exists in several liqueurs, such as *Kirschwasser*, *noyeau*, etc., the inordinate use of which might cause cyanic poisoning.

Statistics and Origin.—Dioscorides casually mentions the poisonous nature of bitter almonds,[4] and the Egyptian priests appear to have known of a poison derived from the peach.[5] Fodéré[6] quotes from Murray cases of poisoning by cherry-laurel water which occurred in 1728. But the toxicological history of hydrocyanic acid itself begins with the year 1803, when its poisonous nature seems to have been first recognized by Schrader.[7] The acid had been discovered twenty years earlier (1780) by Scheele, whose death has been frequently attributed to accidental inhalation of its vapor. It would seem, however, from the account given by Crell[8] at the time of Scheele's last illness, that his death was due to disease and not to poison.

According to Stillé,[9] Borda (1804) first suggested the use of the artificially prepared hydrocyanic acid as a remedy in cases in which cherry-laurel water had been previously used. A few years later (1813) two cases of fatal poisoning by it were reported:[10] one that of a suicide who died in five minutes, the other

[1] Phil. Trans., 1737, xxxvii., 85. See also Mortimer: *Ibid.*, p. 175. Kaempfer; Wibmer, "Wirkung d. Arzneim.," etc., ii., 90. Fodéré: "Méd. lég.," iv., 126.

[2] Rex *v.* John Donellan, 1781. Paris and Fonblanque: "Med. Jur.," iii., 243.

[3] See, however, Huber: Ztsch. f. kl. Med., 1888, xiv., 515.

[4] Lib. i., c. 176.

[5] See p. 5.

[6] "Méd. lég.," iv., 26.

[7] Gilbert's Ann. d. Phys., xiii., 503.

[8] Crell's Annalen, 1787, i., 192.

[9] "Therap. and Mat. Med.," ii., 207.

[10] Arch. f. m. Erfahr., 1813, 510 Hufeland's Journal, 1813, 113.

that of a child who swallowed a quantity of the dilute acid intended for the mother. Within the few succeeding years deaths occurring in a similar manner, from misadventure, or by reason of the varying degree of concentration of the medicinal acid used, became very numerous and in consequence the drug fell into disrepute.

In more recent years the extensive use of the cyanids of potassium, silver, etc., in the processes of photography, electroplating, and gilding has placed these active poisons at the easy disposal of many, has led to numerous cases of poisoning through carelessness or misadventure, and has brought the cyanid of potassium into prominent notice as one of the most frequently employed of poisons.

Of 402 cases of cyanic poisoning which we have collated, 181 were by hydrocyanic acid, 118 by potassium cyanid, 61 by oil of bitter almonds, 5 by eating bitter almonds, 7 by cherry-laurel water; in 16 the nature of the compound is not definitely stated, while in 17 the preparation was an unusual one: ratafia,[1] almond cake,"[2] peach kernels,[3] Prussian blue,[4] bitter almond milk;[5] potassium ferrocyanid in solution followed by a mixture of hydrochloric and nitric acids,[6] inhalations of hydrocyanic acid liberated by the action of sulfuric acid on potassium cyanid,[7] seeds of *Jatropha multifolia*,[8] a solution of silver cyanid in potassium cyanid used by counterfeiters,[9] "argentine,"[10] oil of bitter almonds and opium,[11] almond flavor,[12] gilding solution,[13]

[1] Smith: Lancet, 1844, i., 335—F., 8½, A., R.

[2] Turchetti: Gaz. m. it. prov. Venet., 1860, iii., 226—M., ad., A., R.

[3] Dalla Torre: *Ibid.*, p. 162—F., ad., A., R.

[4] Huber: Ztsch. f. kl. Med., 1888, xiv., 515—M., 50, H. (?), R.

[5] Ponzaire: J. d. m. ch. and ph., Montpel., 1803, Ann. 2, T. i., 297—5 y., A., D.

[6] Volz: Vrtljschr. f. ger. Med., 1877, n. F., xxvi., 57—M., ad., S., D.

[7] Graff: Ztschr. f. d. Staatsarznk., 1838, xxxvi., 1—M., 28, S., D.

[8] Porter: Ind. Med. Gaz., 1885, xx., 143—three children, A., R.

[9] Letheby: Med. Times, London, 1851, n. s., iii., 41—two F., 24, S., D. In one case the woman's paramour was accused of causing her death by inducing her to swallow the poison.

[10] Martius: Bayer. Int. Bl., 1872, xi., *ex.* Ph. Jahrb., 1872, 585—F., 58. A., R. Argentine is a mixture of the cyanids of silver and potassium with chalk.

[11] Ph. J. and Tr., 1861, n. s., ii., 535—a double suicide. The woman recovered and was held for trial as aiding and abetting suicide.

[12] *Ibid.*, 1863, n. s., v., 89—M., 9, A., D.

[13] *Ibid.*, 1871, 3 s., i., 856—M., ad., A., D. Potassium cyanid, hydrochloric acid, ammonia, and water (*sic!*). Another by Witlacil in Wien. m. Wochenschr., 1862, xii., 231—M., 27, S., D.

Kirschwasser,[1] cherry kernels,[2] and plum kernels.[3] The ordinary French bean (*Phaseolus vulgaris*), when eaten raw has been known to cause severe cyanic poisoning, and a related kidney bean (*Phaseolus lunatus*) has proved fatal.[4]

The greater proportion of cases were suicidal: 256 out of 391 cases in which the motive is stated (=65.4 per cent.). Many of the suicides by hydrocyanic acid were pharmacists, physicians, or chemists, while many of those by potassium cyanid were among photographers, electroplaters, and chemists, or soldiers who purchased the salt under the pretense of using it to clean gold lace. In two early instances physicians narrowly escaped fatal poisoning consequent upon their taking the acid for experimental purposes.[5]

Hydrocyanic acid was taken by 120 suicides; potassium cyanid by 83, and oil of bitter almonds by 38. In 7 cases the nature of the preparation is not given, and in 8 it was peculiar; in 2 instances by eating a large quantity of bitter almonds.[6] Only 43 of the 256 suicides were women. Of these 15 took oil of bitter almonds used in confectionery or in the household for flavoring; 5 were female connections of photographers, and 4 of platers, who took potassium cyanid; 5 were female connections or servants of physicians, who took hydrocyanic acid; 3 were double suicides in which the man obtained the poison. In one instance a woman attempted suicide by injecting a solution of potassium cyanid into her rectum.[7]

Of the 102 cases of poisoning by accident, misadventure, and negligence, 17 resulted from the use of oil of bitter almonds as a flavor; 7 from eating bitter almonds, peach kernels, etc.; 27 from mistake on the part of the victim himself, by hydrocyanic acid, and 14 similarly by potassium cyanid; 10 were medicinal

[1] Schneider: Ztschr. f. d. Staatsarznk., 1825, ix., 399—M., ad., A., D.

[2] Wahlen: Upsala läk. förh., 1884, xix., 129—F., 5, A., D.

[3] Seferowitz: Wien. m. Bl., 1882, v., 391—M., 12, A., R.

[4] Forrest: Glasgow M. Jour., 1882, xviii., 68. See also Soltsien: Arch. f. Pharm., 1883, xxii., 29. Davidson and Stevenson: Practitioner, 1884, xxxii., 435. Broadbent: Brit. M. Jour., 1884, i., 267.

[5] Coullon: "Rech. et consid. méd. s. l'acide hydrocyan.," 1819; Revue méd., 1825, 265.

[6] See Volz's, Graff's, Letheby's, Wahlen's, and Witlacil's cases above cited; and Kennedy: London M. and Ph. Jour., 1827, n. s., ii., 150—M., ad., S., D. Maschka: "Samml. Gutacht. Prag. med. Fak.," 1874, 4 F., 259—F., 31, S., D.

[7] Carrière: Bull. gén. de thérap., 1869, lxxvii., 458.

poisonings, all but two anterior to 1850.[1] Three of these were non-fatal cases, one was the fatal poisoning of seven epileptic patients at the hospital of Bicêtre in 1830,[2] and one was the subject of a trial for manslaughter.[3] In 17 instances the poisoning was the result of an error in compounding a prescription.[4] As "chlorodyne" contains hydrocyanic acid, poisonings by that mixture sometimes assume a distinctly cyanic type, particularly when large quantities are taken.[5] Accidental poisonings have also been caused by inhalation of the vapor of the acid,[6] by absorption from a wound caused by breaking a bottle of the medicinal acid in the hand,[7] and a peculiar chronic poisoning in a photographer from contact with potassium cyanid.[8]

Although cyanic poisons have pronounced taste and odor, they have been alleged to have been used, or used in 31[9] cases of intentional homicide of which we find mention. The earliest of these, Rex v. Donellan, 1781, was in all probability a homicide by cherry-laurel water, although the scientific evidence adduced would hardly be considered conclusive at the present time.[10] In 1829 a man was acquitted upon trial for the murder of his mistress by hydrocyanic acid. There was no question of the cause of death, but reasonable doubt concerning the administration.[11] The defendant was also acquitted in Reg. *v.*

[1] Ph. J. and Tr., 1875–76, 3 s., vi., 559. An overdose given to a patient in an infirmary, by error of the nurse. Death, *ibid.*, 1861–62, n. s., iii., 341. Two medicinal doses of hydrocyanic acid given to allay vomiting in a case of poisoning by aconite.

[2] Orfila: "Tox. gén.," 5ème ed., ii., 326.

[3] Reg. *v.* Cronin: Edinb. M. and S. J., 1847, lxvii., 598; London Med. Gaz., 1847, n. s., iv., 388, 431, 695.

[4] Among these are the cases of Reg. *v.* Boroughs, 1857, Ph. J. and Tr., 1857, xvi., 484. Reg. *v.* Ball, 1860, *ibid.*, 1860–61, n. s., ii., 198. Burman's case, Lancet, 1854, i., 39. Garstang's case, *ibid.*, 1888, ii., 15. Guinness' case, Ph. J. and Tr., 1869–70, n. s., xi., 42. Arnold's case, Am. J. M. Sc., 1869, n. s., lvii., 103.

[5] See opium. Also case by Browne; Austral. M. J., 1879, n. s., i., 587.

[6] Taylor: "Poisons," 3d Am. ed., 563—two cases: F., ad., A., D.; F., ad., A., R. Post: New York M. J., 1876, xxiii., 403—M., 55, A., D.; Ph. J. and Tr., 1877–78, 3 s., viii., 356—M., ad., A., D. Martius: Ph. Jahrb., 1872, 585. Professor Bescherer, of Rudolstadt, died in 1849, poisoned during preparation of hydrocyanic acid. Poggendorff: "Handwb.," i., 175.

[7] Sobernheim: "Handb. d. Tox.," 1838, 449.

[8] Souwers: Phila. M. Times, 1877–78, viii., 345.

[9] We find reference to two other cases in a journal inaccessible to us: Blumenstok: Przegl. lek., Krakow, 1882, xxi., 95; *ibid.*, 1886, xxv., 7, 24, 36, 53, 67, 81, 98.

[10] Gurney's Report, Paris and Fonblanque: "Med. Jur.," iii., 243.

[11] Rex *v.* Freeman, London M.

Belaney, 1844, in which there was no doubt of the cause of death or of the administration, but a question as to the intent.[1] In 1845 John Tawel was convicted of the murder of his mistress by hydrocyanic acid.[2] In Reg. *v.* Fisher oil of bitter almonds was supposed to have been poured down the throat of a man while he was asleep.[3] William Palmer, subsequently executed for the murder of Cook, by strychnin, is supposed to have destroyed his brother Walter by prussic acid in 1856.[4] In Reg. *v.* Walker the defendant was convicted, largely upon moral evidence,[5] of the murder of Agnes Montgomery, at Egglesham near Glasgow, in 1857. Taylor refers to another trial for murder, also by hydrocyanic acid, in Glasgow in the same year.[6] In 1863 one Hunt administered hydrocyanic acid to his wife and two children in a cab, which he afterwards left, directing the cabman to drive to a certain address, upon arriving at which, the dead bodies were found in the cab. Two days after Hunt killed himself with aconite.[7] In the same year one Dalby poisoned his wife with hydrocyanic acid and committed suicide with the same poison seven days later.[8] In 1856 one Southey *alias* Forward poisoned three children in a hired room in London, with prussic acid;[9] and in 1872 one Williams poisoned two children with oil of bitter almonds.[10] In 1883 a girl of sixteen years died from the effects of a teaspoonful of oil of bitter almonds administered to her by a preacher, it was claimed without intent to kill.[11] In an early *French* case portions of a body were found in different places, and hydrocyanic acid was detected in the stomach contents.[12] In the *Affaire Pralet*, or *Aff. l'Héritier* in 1841, death was caused by apoplexy, and not

Gaz., 1831, viii., 759, 795. Christison: "Poisons," 2d ed., 666.

[1] Taylor: "Poisons," 3d Am. ed., 572.

[2] Skae: North. J. Med., Edinb., 1844–45, ii., 396; Lancet, 1845, i., 379. Brown and Stewart: "Trials for Murder by Poison," 16–49.

[3] Taylor: *Op. cit.*, 584.

[4] Taylor: *Op. cit.*, 564, 578.

[5] M. Times and Gaz., 1858, n. s., xvi., 36. Brown and Stewart: *Op. cit.*, 52.

[6] Reg. *v.* Thompson. Taylor: *Op. cit.*, 579.

[7] Ph. J. and Tr., 1863–64, n. s., v., 281. Taylor: *Op. cit.*, 721. In the case of Dr. Warder (Ph. J. and Tr., 1866–67, n. s., viii., 94) the selection of poisons was reversed, the wife was destroyed by aconite and W. subsequently committed suicide with hydrocyanic acid.

[8] Ph. J. and Tr., 1863–64, n. s., v., 187.

[9] *Ibid.*, 1865–66, n. s., vii., 129.

[10] *Ibid.*, 1873–74, 3 s., iv., 486.

[11] Reg. *v.* Timmins: *Ibid.*, 1882–83, 3 s., xiii., 579, 619; 1883–84, 3 s., xiv., 58.

[12] Aff. Ramus, Chevallier and de Loury: Ann. d'hyg., etc., 1833, ix., 337.

by hydrocyanic acid as alleged.[1] We find no reference to a French case between this and the affaire Troppmann in 1869. In this case, singular in many respects, a mechanic, in a crude apparatus, prepared impure hydrocyanic acid, with which he poisoned Jean Kinck. Three months later he slaughtered the widow and six children.[2] Tardieu and Roussin, however, refer to another poisoning by a young man without professional knowledge, who prepared the poison himself; and they include in their table of criminal poisonings from 1857 to 1871, two by hydrocyanic acid in 1864 and 1865, and two by potassium cyanid in 1868.[3] The *Affaire Danton* followed in 1882.[4] In *Germany* Liman reports the Melchior case, in which a man poisoned two children and his wife, strangled an elder daughter, and then destroyed himself by a pistol-shot wound in the head.[5] In November, 1867, the Countess Chorinsky died at Munich from the effects of potassium cyanid, administered by a woman sent by the Count from Vienna for that purpose.[6] In 1879 a woman was destroyed by potassium cyanid, added by her lover to a liquor which she was accustomed to use.[7] Huber reported a case of alleged attempt at homicide by Prussian blue mixed with milk in 1888.[8] In the *United States* Paul Schoeppe was convicted of murder by prussic acid in Pennsylvania in 1869, but was acquitted upon a second trial.[9] A young man was accused of the murder of his father by hydrocyanic acid in Nebraska in 1880; and a man was tried in Delaware Co., New York, in 1886 for attempted wife murder by potassium cyanid mixed with salt.[10] In seven instances murder and suicide were committed either simultaneously or in immediate succession. In one of these a woman and two sons were poisoned by one of the latter;[11] in another a man and woman murdered their six children

[1] Orfila "Tox. gén.," 5ème ed., ii., 364–404; Ann. d'hyg., 1841, xxvi., 399, 1843, xxix., 104; Arch. gén. de méd., 1843, 4 s., i., 383; Gaz. méd. de Par., 1843, xi., 82.

[2] Ann. d'hyg., 1870, 2 s., xxxii., 181.

[3] "Empoisonnement," 2ème ed., 1187, 165.

[4] Vibert and l'Hôte: Ann. d'hyg., 1883, 3 s., ix., 393; 1888, 3 s., xx., 235.

[5] Casper-Liman; "Handb. d. ger. Med.," 8te Aufl., ii., 65.

[6] Martin: "Process Ebergenyi-Chorinsky," Allg. Wien. med. Ztg., 1868, xiii., 297, 305, 353, 377. Buchner: N. Rept. f. d. Pharm., 1867, xvii., 534.

[7] Hofmann: "Process Leop. Winckler," Wien. med. Wochenschr., 1880, xxx., 9, 40, 64.

[8] Ztschr. f. kl. Med., 1888, xiv., 515.

[9] Papers, Med.-leg. Soc., N. Y., 1874, i., 124.

[10] Peo. *v.* Arthur F. Hoops.

[11] Smithers' case, Ph. J. and Tr., 1858, xvii., 383.

and then destroyed themselves;[1] in 1871 a man poisoned four of his children and himself;[2] in Manchester a woman poisoned her daughter and herself in 1872;[3] in 1877 a woman poisoned herself and her infant child,[4] and another woman in 1883 destroyed herself and her six-year-old child.[5] An English case in 1890 was more probably one of suicide and homicide than one of double suicide.[6]

Lethal Dose.—The minimum lethal dose of hydrocyanic acid is difficult to fix with certainty, owing to the varying strength of the medicinal acid. It is stated by Christison[7] that the Parisian epileptics received a quantity containing two-thirds of a grain of anhydrous acid each. It has been shown by Letheby, however, that the quantity actually taken in these cases was 5½ Troy grains (0.3575 gm.).[8] In a case reported by Hicks, a woman of twenty-two years died in twenty minutes from the effects of a lotion containing 0.9 grain (0.0585 gm.) of anhydrous acid;[9] and in the Bristol Infirmary case the dose did not exceed 1.2 grains (0.078 gm).[10] A man of forty years died from swallowing the whole of a medicine containing 30 drops of the Br. Ph. acid.[11] It is probable that even so small a quantity as 0.05 gm, (¾ grain) of the anhydrous acid might prove fatal. On the other hand, in a few instances when prompt treatment has been possible, much larger doses have been recovered from. Thus in Burman's case the quantity taken certainly contained 2.4 grains (0.156 gm.) of hydrocyanic acid,[12] and in a case reported by Shively[13] the dose taken is said to have equalled 4.8 grains (0.312 gm.) of anhydrous acid, taken by a student of pharmacy in the form of a freshly prepared U. S. Ph. acid. Potassium cyanid has caused death in doses of 3 and 4 grains (0.2–0.26 gm.),[14] but persons have recovered after taking 19½, 30, and 50 grains (1.26, 1.94 and 3.25 gm.).[15] The

[1] "The Smithfield tragedy." Wilson: Lancet, 1869, ii., 30; Ph. J. and Tr., 1869, n. s., xi., 87.
[2] Nimmo case, Ph. J. and Tr., 1871–72, 3 s., ii., 137.
[3] *Ibid.*, 1872–73, 3 s., iii., 394.
[4] *Ibid.*, 1876–77, 3 s., vii., 403.
[5] *Ibid.*, 1883–84, 3 s., xiv., 617.
[6] "The Newcross tragedy."
[7] "Poisons," 4th (Am.) ed., 592.
[8] Ph. J. and Tr., 1845, iv., 515.
[9] Lancet, 1845, i., 98; Med. T. and Gaz., 1845, xxxv., 896. Letheby: Lancet, 1845, i., 638.
[10] Med. Times, London, 1839, i., 109.
[11] Ph. J. and Tr., 1870, n. s., xi., 727.
[12] Lancet, 1854, i., 39.
[13] Am. M. J. Sc., 1890, n. s., c. 47.
[14] J. d. ch. méd., etc., 1843, 3 s., ix., 95, 98. Perry: Am. J. M. Sc., 1852, n. s., xxiv., 94.
[15] Higgins: Med. Rec., N. Y..

effect of a given dose of oil of bitter almonds is more variable, owing to the varying quantity of prussic acid which the oil contains. Twenty drops have proved fatal to a woman of forty-nine years,[1] and doses of from four to six drachms have been recovered from in several instances.[2]

Prognosis and Duration.—The prognosis in cyanic poisoning is extremely unfavorable. Of 455 subjects 382, or 84 per cent., died. In this number are included many slight poisonings by small doses, and very few of those who have taken truly lethal doses have recovered. This high mortality is in great measure due to the extremely rapid action of the poison. A large proportion of the victims have been "found dead," many within a short time after they have been seen perfectly well. The interval intervening between the ingestion of the poison and unconsciousness and loss of voluntary motion is measured in seconds rather than in minutes. Indeed it was formerly questioned whether a person having taken a lethal dose of a cyanic poison could perform any voluntary act thereafter. A number of observations, however, of instances in which death has been unquestionably due to a cyanic poison, and in which the victim has spoken, walked, and performed other voluntary acts before yielding to the action of the poison, render the opinion that evidences of such acts, performed after the taking of the poison, are inconsistent with the theory of suicide untenable.[3]

The average **duration** of fatal cyanic poisoning is shown in the cases of the seven epileptics, above referred to, who died in from fifteen to forty-five minutes. In 78 of 151 reports of fatal poisonings by hydrocyanic acid, in which reference is made to the duration, the person was "found dead;" in 35 death is said to have occurred in "a few minutes" or "soon." In 4 early cases death is said to have occurred in five minutes or less. But in these unconsciousness and an imperceptible pulse were probably mistaken for death. Thus in Hufeland's case, a thief upon

1891, xl., 687. Stevenson: Lancet, 1871, i., 806. Gillibrand: Lancet, 1876, ii., 223.

[1] Bull: Prov. M. and S. Jour., 1844, 364.

[2] Pursell: Assoc. M. J., 1854, ii., 885. Chavasse: Lancet, 1839, ii., 930. Allen: Int. M. Mag., Phila., 1893, ii., 126.

[3] See in this regard Gierl: Med.-Chir. Ztg., Innsbruck, 1829, i., 396. Godfrey: Prov. M. and S. J., 1844, 398. Burman: Lancet, 1854, i., 39. Quain: Tr. Path. Soc., London, 1855–56, vii., 189; also Med. Times and Gaz., 1855, ii., 609; Lancet, 1858, i., 128. Hickman: Lancet, 1866, i., 310.

being taken into custody swallowed a large dose of the acid. When seen by the physician four or five minutes later he was stretched out completely lifeless, with no trace of pulse, heart's action, or respiration, and after a few minutes there was one terribly deep respiratory act, followed afterward by two others before death.[1] Taylor[2] refers to the case of a physician who destroyed himself in from four to five minutes by swallowing several drachms of the medicinal acid; and Tripe[3] speaks of another suicide in whom life was extinguished in eight or nine minutes. In but few fatal cases has the duration exceeded three-quarters of an hour, and in most cases it was less than half an hour. We find record of but three instances in which death has occurred after the patient has survived for an hour. In one of these a woman died in an hour and twenty minutes after taking half a drachm of the Br. Ph. acid;[4] in another a man died in an hour and a half,[5] and, in a most exceptional case, a woman of twenty-three years rallied for a time, but subsequently died in three and one-half hours.[6] The action of oil of bitter almonds and of potassium cyanid is equally prompt, and deaths caused by each in less than ten minutes have been reported.[7] But survivals for an hour or more and subsequent death by these cyanics are of more frequent occurrence than with the acid itself. Instances of death caused by oil of bitter almonds in two and a half,[8] three,[9] and fifteen[10] hours; and by potassium cyanid in two,[11] "several,"[12] and twenty-four[13] hours have occurred. The duration of fatal poisonings by bitter almonds, etc., is usually

[1] Hufeland: J. d. prakt. Heilk., 1815, xl., 1 st., 85.

[2] "Princ. and Pr. Med. Jur.," 2d Am. ed., i., 363.

[3] Brit. M. J., 1877, i., 11.

[4] Garstang: Lancet, 1888, ii., 15.

[5] Fagge (Stevenson): Guy's Hosp. Rep., 1869, 3 s., xiv., 259.

[6] Brit. M. J., 1883, i., 131.

[7] By oil of bitter almonds: Frank: Vrtljschr. f. ger. Med., 1868, n. F., ix., 179—M., 20, S., about five minutes. Quain: *Loc. cit.*—M., 39, S., ten minutes. By potassium cyanid: Casper-Liman: "Handb. d. ger. Med.," 8te Aufl., ii., 496—F., 20, S., "immediately." Valcourt: Union méd., 1875, 3 s., xx., 626—boy, A., two minutes. Haskins: Boston M. and S. J., 1870, lxxxii., 21—M., ad. (intoxicated), S., four to five minutes. Frank: *Loc. cit.*—M., 21, S., four to five minutes; Ph. J. and Tr., 1858, xvii., 534, boy, A., seven minutes.

[8] Armstrong: Austral. M. J., 1880, n. s., ii., 202—3 yr., A.

[9] [Barclay] Lancet, 1866, i., 255—M., 57, S.; *ibid.*, 1845, ii.—M., 19, S.

[10] Ph. J. and Tr., 1863, n. s., v., 282—F., 60, A.

[11] *Ibid.*, 1872, 3 s., 14—M., ad., A.

[12] *Ibid.*, 1861, n. s., ii., 535—M., 19, S.

[13] *Ibid.*, 1875, 3 s., vi., 235, 239—M., ad., S.

longer. In one instance a girl of five years died in seventy hours from eating a quantity of cherry kernels.[1]

Symptoms. — HYDROCYANIC ACID. — *By Inhalation.* — Coullon[2] observed that "the odor of the acid caused one to fall unconscious and motionless." Ittner[3] suffered from "constriction of the chest, interference with respiration, vertigo, lassitude, and shuddering" in consequence of having inhaled the vapor. Taylor relates the case of a lady who "was immediately seized with dizziness, stupor, inability to stand, and faintness" from inhalation of the vapors produced by spilling a quantity of the five-per-cent. solution upon her dress while standing before a fire.[4] A student, engaged in preparing the acid in an apparatus whose joints permitted the escape of the vapor, was found, after several hours, insensible, with closed eyelids, widely dilated pupils, cold limbs, and barely perceptible pulse. The muscles of the arms and legs were firmly contracted, the patient was in a condition of profound coma, but recovered after several hours.[5]

Persons exposed for a number of hours to an atmosphere charged with vapor of the acid in relatively small proportion suffer for some days from disturbances of vision, shooting pains in the forehead, pain in the region of the heart, palpitation, difficult respiration, a sense of constriction and irritation of the throat, fits of weakness and somnolency, cough and attacks of suffocation. Operatives also, who are exposed to the constant inhalation of minute quantities of the vapor, develop a form of chronic poioning.[6]

By the Stomach.—The action of hydrocyanic acid when taken by the stomach varies with the magnitude of the dose, the degree of concentration having little, if any, influence. When taken in quantities above the medicinal dose but still below that capable of causing death (ɱx. to xx. = 0.6 to 1.2 c.c. of the U. S. Ph. acid), it causes a sense of constriction of the chest, a feeling of impending suffocation, irritation of the

[1] Wahlen: Upsala läk. förh., 1884, xix., 129.

[2] "Dissert. s. l'ac. pruss.," Paris, 1808.

[3] Med. Jahrb. d. oest. Staates, 1809, ii., 4.

[4] "Poisons," 3d Am. ed., 571.

[5] Regnauld: Ann. d'hyg., etc., 1852, xlvii., 455.

[6] See Martius: Bayer. Int. Bl., 1872, xi. Martin: Friedreich's Bl. f. ger. Med., 1888, xxxix., 3. Mittenzweig: Ztschr. f. Med.-Beamte, 1888, i., 97. Tatham: Brit. M. J., 1884, i., 409.

throat, a sense of heat in the epigastrium, dizziness, impeded locomotion, a sense of constriction and heaviness of the head, and in some cases nausea, or a craving for food. Salivation and ulceration of the mouth may occur. A sample of blood exhibits the peculiarities due to the formation of cyanmethæmoglobin.[1]

When small poisonous doses are taken, the patient notices the hot, bitter taste of the acid during the act of swallowing, and almost immediately experiences a sense of constriction of the throat. In the course of one or two minutes there are severe pains and pressure in the head, increasing vertigo, progressive confusion of intellect, obscuration of vision, and insensibility, with loss of muscular power. The face is pale and bloated, the finger nails are blue or purple, the eyes, prominent and glassy, are directed upward or have a rolling motion, and the mouth is marked with froth. The patient is then seized with convulsions of a tetanic character, with the jaws and hands firmly clenched. The pulse is quick, the respiration is slow and stertorous, and mucous râles are heard over the chest. Vomiting is uncommon. If it occur it is usually the beginning of recovery. The vomited matters, as well as the breath, have the odor of bitter almonds. If the case terminate in death there are usually strong tetanic convulsions, involuntary evacuations of urine and fæces, and opisthotonus; then succeeds a condition of general paralysis, and death from asphyxia follows. When recovery occurs it does so gradually, the respiration slowly returning to the normal condition within a few hours, and without any further complications.[2]

With large doses subjective symptoms may be entirely absent. In less than a minute consciousness is suddenly lost, and the patient falls to the ground if standing or sitting. Then there is usually a short convulsive seizure, during which involuntary evacuations of urine and fæces frequently occur. After this the patient lies perfectly still, with prominent, glassy eyes, jaws firmly clenched, mouth covered with foam, face at first bloated, afterward pale and sunken, pupils dilated and insensible, surface cold and clammy, and muscles completely relaxed. The pulse, at first somewhat more frequent than normal,

[1] See test 5, pp. 622.

[2] For a graphic account of the sensations during cyanic poisoning see Arnold: Am. J. M. Sc., 1869, lvii., 103. Also Nunneley: Prov. M. and S. J., 1845, 517.

rapidly becomes weaker, until in the later stages it is imperceptible. The respiration is spasmodic, the inspirations are short and immediately followed by a protracted and very deep expiration, after which succeeds a pause of considerable duration, increasing as the case progresses to a fatal termination. Death occurs during one of the pauses, the only apparent difference in the condition of the patient before and after death being that no succeeding inspiration occurs. Recovery even from large doses of hydrocyanic acid, is, when it occurs, rapid and complete within a few hours.[1]

The syptoms produced by OIL OF BITTER ALMONDS are the same as those caused by prussic acid itself.

POTASSIUM CYANID.—As this salt is strongly alkaline and frequently contains a very notable proportion of potassium carbonate, it produces local symptoms besides those of cyanic poisoning if the duration be sufficiently prolonged. The lips and mucous membrane of the mouth are corroded, reddened, and covered with a whitish film, and there is burning pain in the throat and stomach. Vomiting occurs in cases which are not rapidly fatal, particularly if the poison be taken with food or into a full stomach.

In non-fatal cases recovery is much slower than from hydrocyanic acid. In an exceptional case the patient gradually regained consciousness on the third day, and was not discharged until the eleventh day after the poisoning.[2] (See Forensic questions, p. 624.)

Treatment.—Although in the majority of cyanic poisonings the physician arrives too late to be of service, several instances of recovery under suitable treatment in apparently hopeless cases have been reported in recent medical literature.[3]

The first indication is to wash out the stomach as soon as possible with water containing either an oxidizing agent such as potassium permanganate (0.3 per cent.)[4] or hydrogen per-

[1] See Burman's and Shively's cases above cited.

[2] Med.-chir. Rundschau, 1867, 195, *ex* Friedreich's Bl. f. ger. Med., 1868, xix., 452.

[3] See Shively's and Burman's cases, and Allen: Int. M. Mag., Phila., 1893, ii., 126. Ord: Lancet, 1886, ii., 1174. Carmichael: M. Times and Gaz., 1865, i., 647 (prussic acid and chloroform). Quintin: Berl. kl. Wochenschr., 1885, xxii., 120. Gillibrand: Lancet, 1876, ii., 223. Scholz: Wien. med. Wochenschr., 1866, xvi., 1529, 1548.

[4] Kossa: Therap. Monatsh., 1892, vi., 549; Ung. Arch. f. Med., 1893, ii., 12; Centbl. f. med. Wissensch., 1894, xxxi., 219

oxid,[1] or a mixture of ferrous and ferric salts (ferrous sulfate, or copperas, and ferric chlorid).[2] When bitter almonds or other kernels containing amygdalin have been eaten in large quantity an emetic of zinc sulfate by the mouth or of apomorphin hypodermically should be given. Inhalations of chlorin or of ammonia have been frequently recommended. They do no exert any truly antidotal action, and can serve only as respiratory stimulants. They are themselves dangerous if incautiously used, and are better replaced by artificial respiration, cold affusion to the back while the patient is in a warm bath, or the faradic current. Hypodermics of ether, or camphor dissolved in ether, or strychnin, or whiskey are indicated to combat the collapse.

Atropin hypodermically administered was considered by Preyer[3] as the physiological antidote of hydrocyanic acid, but, although his experiments have shown that animals already under the influence of atropin are more or less protected from the action of hydrocyanic acid, the use of atropin in cases of poisoning by hydrocyanic acid does not seem to be of much benefit.[4]

Post-Mortem Appearances.—Rigor mortis usually sets in early, and persists sometimes for days, particularly in the lower jaw. As in sudden death from any cause, putrefaction is rather more rapid than usual. The face is usually placid, sometimes bloated, the eyes are prominent and glassy, the pupils dilated, the mouth is more or less covered with foam. The veins everywhere are distended with dark, fluid blood; or the blood, while fluid, is bright red in color, and on spectroscopic examination shows the spectrum of cyanmethæmoglobin, a single band similar to that of (reduced) hæmoglobin but somewhat

[1] Krohl: Diss., Dorpat, 1891. Kobert: "Intoxikationen," 516.

[2] J. and J. H. Smith: Lancet, 1844, ii., 41.

[3] "Die Blausäure," Bonn, 1868, 1870. See also Arch. f. exp. Path. u. Ph., 1875, iii., 381.

[4] For experiments upon animals relating to the method of action and treatment in hydrocyanic poisoning see Antal: Ungar. Arch. f. med., 1894, iii., 117. Becker: Diss., Berl., 1893. Gaethgens: Hoppe-Seyler, "Med.-chem. Unt.," 1868, 325. Geppert: Ztsch. f. kl. Med., 1888–89, xv., 208, 307 (one plate). Gréhant: Tribune méd., Paris, 1889, 2 s., xxi., 491; *Id.*, C. rend. soc. d. biol., Paris, 1890, 9 s., xi., 64. Hoppe-Seyler: "Med.-ch. Unt.," 1871, 258; Knie (Boehm): Arch. f. exp. Path. u. Pharm., ii., 1874, 129. Preyer: "Die Blausäure," Berlin, 1868, 1870; *Id.*, Arch. f. exp. Path. u. Pharm., 1875, iii., 381. Tolmatscheff: Hoppe-Seyler, "Med.-ch. Unt.," 1871, 285. Wagner: Diss. Berlin, 1880. Zillessen: Ztsch. f. physiol. Ch., 1890–91, xv., 387.

more faint and somewhat broader.[1] The left ventricle of the heart is usually firmly contracted and empty, the right heart full of dark, uncoagulated blood. The larynx and trachea contain a bloody foam, and the lungs are highly congested. The stomach frequently presents no abnormal appearance, and may be paler than usual, but usually it is red and inflamed, either over its entire extent or in patches, and its blood-vessels are filled with fluid blood. This is particularly marked when potassium cyanid has been taken, when the injection extends to the duodenum, and ecchymoses are frequently present.[2] One of the most distinctive characters is the odor of bitter almonds, or peach kernels. This may frequently be observed before any incision is made, sometimes only on opening the abdomen, the stomach, the pericardium, or the cranial cavity. In some cases of undoubted cyanic poisoning this odor has not been detected, and it is also present after death from nitrobenzene.

Analysis.—Owing to the great volatility and instability of hydrocyanic acid, the probability of its detection in the cadaver diminishes rapidly as time elapses, particularly if the conditions favoring putrefaction obtain. The analysis, therefore, should be made as soon as practicable after death. Nevertheless instances are not wanting in which the presence of the poison has been demonstrated weeks after death. Thus Reichardt has detected it after eight weeks of putrefaction,[3] and Ludwig after four months in a body which had been desiccated by lying in a cellar for one hundred and fifteen days at temperatures varying from 8°–10° C. (46°.4 to 50° F.).[4] In eight cases of death from potassium cyanid examined by Fagerlund,[5] affirmative results were obtained after two, eight, ten, thirteen, and sixteen days (during the winter in Finland in the last two), and in three instances the results were negative after nine, twenty-seven, and twenty-eight days.

After death from cyanic poisoning hydrocyanic acid has been detected and quantitatively estimated, not only in the stomach contents but also in the blood, liver, spleen, and brain.

[1] See tests 5, 6, 7, pp. 622, 623.

[2] For colored plates see Lesser: "Atl. d. ger. Med.," Pl. ix.; Pl. x., f. 3; Pl. xiv., f. 1; Pl. xv., f. 3; Pl. xviii., ff. 8, 9, 10.

[3] Arch. f. Pharm., 1881, 3 R., xix., 204.

[4] Zillner: Vrtljschr. f. ger. Med., 1881, n. F., xxxv., 193.

[5] *Ibid.*, 1894, 3 F., viii., Supphlft., 85.

In the kidneys and muscular tissue it is present only in traces or is absent, and it does not appear in the urine.[1] The quantity remaining in the body after death may be quite considerable, particularly when death has been caused rapidly by potassium cyanid or by oil of bitter almonds. Thus in a case in which death by potassium cyanid followed the ingestion in four to five minutes, 0.2 gm. (3.2 grains) of anhydrous acid were obtained on analysis, and in another case, in which death from oil of bitter almonds followed in a like period, 0.4967 gm. (7.68 grains) were separated.[2] In a case of death in a few minutes from oil of bitter almonds, Redwood found 2.268 gm. (35 grains) of anhydrous acid in the ten drachms of stomach contents.[3]

In a systematic analysis for all poisons hydrocyanic acid would appear, if present either free or in combination, in the first distillates obtained in the search for volatile poisons (see p. 127), to which the tests described below may be applied. This method should not be followed, however, when the circumstances point clearly to cyanic poisoning, as metallocyanids such as potassium ferrocyanid, and the thiocyanates normally present in the body may yield free hydrocyanic acid on distillation with a mineral acid. The clinical history of a cyanic poisoning, the odor of the poison in most cases observable, and the appearance of the blood (see below) are usually sufficiently marked to warrant a special examination for cyanic poisons and their distinction and separation from non-poisonous or normal cyanogen compounds, and this is particularly necessary in view of the existence of a thiocyanate in the saliva, urine, milk, etc.,[4] and the frequent presence of ferrocyanid as an impurity of potassium cyanid.

A preliminary examination of a portion of the material is therefore necessary to determine whether double cyanids are present or not. To this end a portion of the material, liquefied with water if necessary, is dialyzed, and the dialyzate is concentrated by evaporation and divided into two parts, both of which are slightly acidulated with hydrochloric acid, and ferric chlorid solution is added to one. If a ferrocyanid be present a deep blue

[1] Bischoff: Berichte, Berlin, 1883, xvi., 1354.

[2] Frank: Vrtljschr. f. ger. Med., 1868, n. F., ix., 183.

[3] Ph. J. and Tr., 1856, xv., 377.

[4] See Bruylants: J. d. Pharm., 1888, 5 s., xviii., 104, 153; abst. J. Ch. Soc., London, 1888, liv., 1324.

precipitate (Prussian blue) will be formed, and if a thiocyanate be present the liquid will assume a deep red color, which is discharged on addition of mercuric chlorid solution (see meconic acid). To the other portion of the concentrated dialyzate sodium acetate and then cupric sulfate solution are added, when, if a ferrocyanid be present, a red-brown precipitate or color is produced.

Occasionally the cyanids of mercury, gold, or silver must be taken into account. Mercuric cyanid may be separated by Barfoed's method, by agitation with ether, which dissolves out the mercuric salt and free hydrocyanic acid. The ethereal solutions are rendered alkaline with alcoholic potash and evaporated to dryness, the residue, dissolved in water and acidulated with tartaric acid, is distilled for hydrocyanic acid in the manner described below, and mercury is estimated in the residue. The cyanids of silver and gold are only of interest when a silvering or gilding solution, which always contains potassium cyanid also, has been taken. In that event the quantitative relations of the hydrocyanic acid found in the distillate and the silver or gold and potassium determined in the residue will permit of proper distribution of the acid.

Two conditions are possible: either, A, the preliminary examination has shown the absence of double cyanids; or B, they are present.

A. *In the absence of double cyanids.* The apparatus used is the same as that shown in Fig. 2 (p. 127) except that the Liebig's condenser is to be connected by an air-tight joint with a small tubulated receiver, from the tubulus of which a tube bent twice at suitable angles is in air-tight communication with one limb of a bulbed U tube. About 20 c.c. of a strong solution of silver nitrate are placed in the receiver and U tube. The apparatus having been mounted the flask A is disconnected, charged with the substances to be examined and sufficient aqueous solution of tartaric acid to render the whole distinctly acid, and immediately replaced. After a slow current of carbon dioxid has been established through the apparatus, the flask A, which has in the mean time been brought into an oil or paraffin bath, is gradually heated and the distillation continued so long as the drops of distillate falling into the silver solution produce any precipitation.

B. *If double cyanids are present.* The same apparatus is used as in A, but in place of tartaric acid solution, sodium carbonate solution is added to alkaline reaction, and the process continued as above described.[1]

Whether A or B has been followed, the hydrocyanic acid is quantitatively determined by collecting the silver cyanid from the receiver and U tube upon a weighed filter, washing, drying at 100° (212° F.), and weighing. The last weight obtained, minus the weight of the filter, and multiplied by 0.20194, gives the weight of hydrocyanic acid (HCN) in the amount of material distilled.

If the precipitate of silver cyanid be not perfectly white, silver sulfid, resulting from the liberation of hydrogen sulfid, is probably present. In that event the silver cyanid is dissolved from the filter with ammonium hydroxid, the filter and remaining silver sulfid are dried at 100° (212° F.), weighed, and this weight is used in the above calculation in place of that of the empty filter. The silver cyanid is regained from the ammoniacal solution by addition of dilute nitric acid to faintly acid reaction, collection and washing of the precipitate, which is then treated as below.

Having determined the quantity, the filter with its adherent precipitate is placed in a flask, moistened with water, and treated with a quantity of decinormal hydrochloric acid solution such that 0.74 c.c. of decinormal acid solution (containing 3.64 gm. HCl in 1000 c.c.) is added for every 0.01 gm. of silver cyanid found; the flask is then immediately corked, strongly agitated, and set aside until the precipitate of silver chlorid has subsided. The clear liquid, decanted through a small filter, is subjected to the following tests:

1. *Prussian Blue Test.*[2]—Render a portion of the distillate alkaline with dilute caustic potash solution, add a few drops of a solution of ferrous sulfate which has become yellow by exposure to air, agitate, and let stand ten minutes. In the presence of a sufficient quantity of hydrocyanic acid a dirty greenish precipitate will have formed. Now add hydrochloric acid in slight excess, warm slightly, and, having noted whether any blue or green color is immediately formed, examine again after several

[1] See Jacquemin: Ann. d. chim. et de phys., 1876, 5 s., iv., 135. Otto: "Ausmitt. d. Gifte," 6te Aufl., 34.

[2] Ittner, 1809.

hours' standing. In the presence of small quantities of hydrocyanic acid the liquid is colored green, and only deposits Prussian blue after long standing; with larger quantities the liquid is colored blue, and immediately, or after a short time, deposits a flocculent, dark blue precipitate, while the color of the liquid gradually changes to yellow.

According to Link and Möckel[1] the test is doubtful with dilutions beyond 1 : 50,000; according to Wormley[2] it is sensitive up to a dilution of 1 : 25,000.

2. *Thiocyanate Test.*[3]—Place a portion of the distillate in a porcelain capsule, add a few drops of a dilute solution of sodium hydroxid and a few drops of a dilute solution of yellow ammonium sulfid; evaporate to dryness over the water-bath; add water; acidulate with hydrochloric acid and then add two or three drops of dilute ferric chlorid solution. If the liquid contained hydrocyanic acid it will have been converted by this treatment into sodium thiocyanate, which, with the ferric salt, gives a red color.

Link and Möckel[4] consider this the most delicate of the tests for hydrocyanic acid, and place its limit at 1 : 4,000,000. Wormley,[5] on the other hand, places its delicacy as equal to that of the Prussian blue reaction: 1 : 25,000.

3. *Guaiac Test.*[6]—Moisten a piece of filter paper with a freshly prepared three-per-cent. alcoholic solution of guaiac, dry, moisten with a drop of a 0.05-per-cent. solution of cupric sulfate, and then with a drop or two of the suspected solution. In the presence of hydrocyanic acid a beautiful blue color is produced. With very concentrated solutions the blue color is produced by mere exposure to the vapor which they emit; with highly dilute solutions a larger quantity of the suspected liquid is to be brought upon the paper by slow dropping from a pipette. The paper may be used as a preliminary test, and possibly may be useful to distinguish whether the odor of bitter almonds in the breath is due to a cyanic poison or to nitrobenzene, although it did not prove of value in that manner in the hands of Bellini.[7]

Although this reaction, when used with solutions, is very

[1] Zeitsch. f. an. Ch., 1878, xvii., 455.
[2] "Micro-Chem. of Poisons," 2d ed., 183.
[3] Liebig, 1847.
[4] *Loc. cit.*
[5] *Loc. cit.*
[6] Schönbein, 1868.
[7] Speriment., Firenze, 1876, xxxviii., 313.

delicate, its limits being placed by Link and Möckel[1] at 1:3,000,000, it is only of confirmative value, as it is produced by ammonia, volatile ammoniacal compounds, by hydrochloric acid, tobacco smoke,[2] ozone and chlorin, as well as by hydrocyanic acid.[3]

4. *Nitroprussid Test.*[4]—Add a few drops of potassium nitrate solution to a portion of the distillate, then two to four drops of ferric chlorid solution, and, finally, sufficient dilute sulfuric acid to cause the brown-yellow color to just turn to light yellow; heat to incipient boiling, cool, add a few drops of ammonium hydroxid solution, filter, and add to the filtrate a drop or two of a very dilute colorless ammonium hydrosulfid solution. If hydrocyanic acid was present in the original liquid a fine violet color is produced, which passes into blue in a few minutes, then into green, and finally to yellow. With very small quantities the color is at first bluish-green, soon passing to greenish-yellow. The limit of the reaction is placed at a dilution of 1:312,500.

5. *Methæmoglobin Test.*[5]—Human or mammalian blood is diluted with distilled water to a one to four per cent. solution, filtered, and shaken with a very minute crystal of potassium ferricyanid only until the red color has changed to yellow, when it is poured off from the undissolved salt. The solution is placed in a cell with parallel sides and a portion of the neutralized distillate[6] floated upon its surface: the yellow color of the lower solution changes from yellow to a beautiful bright red. The cell is best placed in position for spectroscopic examination and the spectrum observed before and during the reaction: at first the methæmoglobin band in the red is observed,[7] this disappears as the red color appears, as the cyanmethæmoglobin which is formed does not possess it. According to Kobert[8] the

[1] *Loc. cit.*

[2] According to Vogel (Sitzber. d. Ak. d. Wissensch. z. München, 1884, 286) tobacco smoke and illuminating gas contain hydrocyanic acid.

[3] See Hilger and Tamba: Mitth. a. d. ph. Inst. u. Lab. f. ang. Ch. d. Univ. Erlangen, 1889, Hft. ii., 286.

[4] Vortmann: Ztsch. f. an. Ch., 1887, xxvi., 642.

[5] Kobert: "Ueber Cyanmethämoglobin," etc., Stuttgart, 1891, also "Intoxikationen," 518.

[6] The reaction of either liquid must not be alkaline, that of the methæmoglobin solution neutral or at most very faintly acid.

[7] See spectrum 10, Plate iii. (p. 30), Vol. II.

[8] *Op. cit.*, p. 193.

spectrum of cyanmethæmoglobin is very similar to that of (reduced) hæmoglobin. But Richter in two cases failed to observe any distinctive cyanmethæmoglobin band.[1]

This test may be used during the lifetime of the patient to establish a positive diagnosis. For this purpose samples of blood are taken (by slight puncture of the finger tip with a needle and subsequent pressure) from the patient and from some healthy person, and received in test tubes containing distilled water in such quantity that the two liquids are of equal intensity of color. To each a freshly prepared one-per-cent. solution of potassium ferricyanid is added drop by drop. On shaking the normal blood it immediately becomes brown and shows the methæmoglobin band, while if the blood contain hydrocyanic acid in not too small amount it remains red with the same or even a somewhat greater quantity of ferricyanid, and only exhibits the cyanmethæmoglobin spectrum.

6. Normal blood has the property of reducing itself with time, which does not occur if the blood contain hydrocyanic acid. If two samples of blood, one from the person supposed to have been poisoned (taken during life or shortly after death and not exposed to the air) and one from a healthy human being, be so far diluted with distilled water as to form one-per-cent. solutions, placed in air-tight cells with parallel walls and examined with the spectroscope, both will show the oxyhæmoglobin bands. The two cells are then set in a warm place in the dark and their color and spectra observed from time to time. In from six to twenty-four hours the normal blood becomes darker and the oxyhæmoglobin spectrum is replaced by that of (reduced) hæmoglobin.[2] If the blood contain hydrocyanic acid it remains bright red and still shows the oxyhæmoglobin bands.[3]

7. *Hydrogen Peroxid.*[4]—Two cells are prepared with one-per-cent. solutions of blood as in No. 6, and dilute, *neutral* solution of hydrogen peroxid is carefully added to each in equal amounts. If the blood contain hydrocyanic acid the color changes from red to brown-red, brown, yellow, and finally becomes colorless. At the same time the oxyhæmoglobin bands

[1] Prag. med. Wochenschr., 1894, xix., 105, 120, 132.

[2] Spectrum No. 5, Pl. iii., Vol. II.

[3] See also Buchner: Sitzber. d. K. b. Ak. d. Wissensch. zu München, 1868, ii., 591.

[4] Schönbein: Zeitschr. f. Biol., 1867, iii., 325.

disappear and the methæmoglobin band appears, and this finally vanishes also without the appearance of any further band. In the absence of prussic acid, blood decomposes hydrogen peroxid but retains its own color and continues to exhibit the spectrum of oxyhæmoglobin.

It is essential to the success of the reaction that the hydrogen peroxid used shall be strictly neutral and that it shall not be added in too large amount, else solutions of normal blood will undergo the changes described.

Like the methæmoglobin test this reaction may be applied during the life of the patient, if a spectroscope and the reagent be available.

Other tests have been suggested: With starch and potassium iodid (Schönbein); with cupric sulfate alone (Lassaigne); with picric acid (Braun); with cobaltous chlorid (Braun); with mercurous oxid (Henry and Humbert), and with uranic nitrate (C. Lea). These are, however, all inferior to those given above, which are sufficient to characterize hydrocyanic acid with certainty.

Forensic Questions.—The following points have arisen in trials for murder by prussic acid, and have been the questions upon which the case mainly turned:

1. *Was death due to prussic acid or to apoplexy?* This was the principal question in a classic French case, tried at Cambery in 1841, in which the prisoner was sentenced, and would have been executed had not Orfila shown from the *facts* of the evidence that the death of the deceased, Pralet, was due to apoplexy, and that the *opinions* of the chemical and medical experts upon which the conviction was based were of the most flimsy character even for that date.[1]

At the present time an autopsy, if one be possible, may be depended upon to determine the question, negatively or affirmatively, of the existence of intracranial hemorrhage. While after death from hydrocyanic acid the meningeal vessels and cerebral sinuses are found gorged with blood, there is never extravasation into the ventricles, the membranes, or substance of the brain, nor are there clots.

2. *Are certain symptoms and appearances necessarily present if death has been due to cyanic poisoning?* The ar-

[1] See p. 608.

gument has been formerly advanced that death could not have been due to a cyanic poisoning because the conditions were such that some symptom, alleged to be characteristic, could not have been manifested.

When animals are poisoned by hydrocyanic acid the *inspirations* are deep and convulsive, and are accompanied by a loud cry.[1] In some of the earlier trials for poisoning by this substance the defence has claimed that death was due to other causes because this cry had not been uttered. In the human subject it is the expiration which is convulsively forcible and deep, and we fail to find in medical literature a distinct description of such a cry as is uttered by animals,[2] except in two instances: one of these was in a case, referred to by Taylor,[3] of "a man, æt. twenty," who "swallowed about two ounces of the oil of bitter almonds. A person present saw him fall suddenly while in the act of swallowing, he made a loud cry, gave one deep expiration, and died." Hunt[4] reported the case of a child of three years who was poisoned by eating bitter almonds, but recovered. "The mother said that the child had given a cry when the symptoms began, but it was difficult to determine from her statement whether it was the characteristic cry or not." In both of these cases it is quite possible that the utterance of the cry was a voluntary act.

Although in most cases of cyanic poisoning there are violent tetanic spasms, many instances of death, unquestionably due to this cause, have been reported in which no convulsions occurred. The absence of convulsions is most frequently observed when death follows rapidly after large doses have been taken.[5]

[1] Preyer: *Op. cit.*

[2] In but few cases is any noise said to have emanated from the patient. In Hick's case (Lancet, 1845, i., 559) a "gasping noise" was heard at first, afterward a "moaning noise." In Tardieu and Roussin's case (Ann. d'hyg., 1868, 2 s., xxix., 358) the patient is described as falling to the ground "en poussant quelques cris sourds." Mueller-Warnek (Berl. kl. Wochenschr., 1878, xv., 57), Carmichael (M. Times and Gazette, 1865, i., 647), and Baker (Brit. M. J., 1881, ii., 12) heard loud tracheal râles accompanying the acts of inspiration and expiration in patients seen after having taken potassium cyanid. In Bishop's case (Lancet, 1845, 315) the respiration is qualified as "hissing;" in one of Shapleigh's cases (Tr. Coll. Phys., Phila., 1869, n. s., 291) a "loud mucous rattle" was heard.

[3] "Poisons," 3d Am. ed., 585.

[4] Med. Times and Gaz., 1878, i., 37.

[5] Pooley: Lancet, 1845, i., 559. Leithead: *Ibid.*, 640. Taylor: "Pr. and Pr. Med. Jur.," 2d Am. ed., i., 363. *Id.*, "Poisons," 3d Am. ed., 571, 585. Sewell: Boston M. and S. Jour., 1848, xxxvii., 322.

On the other hand, a man who died in a few minutes from the effects of a large dose of potassium cyanid had general convulsions so violent as to throw him out of bed.[1] The fact that in many instances where the dead body is found the face bears a calm and placid expression is not positive proof that convulsive movements did not occur previous to death.[2] Even in the absence of general convulsions the mouth is tightly closed and opened with difficulty in most instances. Yet cases are not wanting in which the mouth was wide open.[3] Usually the pupils are widely dilated. But Baker[4] and Osthoff[5] have reported cases in which they were contracted; Thomson[6] a case in which they were at first a little contracted, afterward dilated; and Armstrong[7] one in which the pupils became dilated only after washing out the stomach, subsequently they became contracted and again dilated about a quarter of an hour before death.

Involuntary evacuations of fæces or urine or of both have occurred in about one-half of the recorded cases.

The *odor* of prussic acid, which may be described as that of bitter almonds or of peach kernels, is almost invariably present in the breath, and sometimes pervades the entire room. Yet when very small, lethal doses have been taken it may be absent.[8] It is also usually exhaled from the bodies of those who have died of cyanic poisoning, or, if not perceived before, is apparent when the cadaver is opened. Its absence under these circumstances is, however, by no means proof of the absence of cyanic poison.[9] In the case of Twitchell, who committed suicide in a cell, and whose dead body was found within six hours

Hickman: Lancet, 1866, i., 310. Thomas: *Ibid.*, 1873, ii., 522. Hinds: M. Times and Gaz., 1850, n. s., i., 482. Harley: Tr. Path. Soc., London, 1862, xiii., 95. Christison: Monthly J. M. Sc., 1850, x., 97.

[1] Auer: Friedreich's Bl. f. ger. M., 1878, xxix., 436.

[2] Ware: Boston M. and S. J., 1856, lv., 387.

[3] Haskins: Boston M. and S. J., 1870, lxxxii., 21. Tripe: Brit. M. Jour., 1877, i., 11. Ellis: Lancet, 1863, ii., 447.

[4] Brit. M. Jour., 1881, ii., 12.—M., 38, two handsful of bitter almonds, R.

[5] Friedreich's Bl. f. ger. Med., 1881, xxxii., 194—M., 36, KCN, D. in fifty minutes.

[6] Assoc. M. Jour., 1856, iv., 1055—F., 46, S., oil bitter almonds, D. in one hour.

[7] Austral. M. Jour., 1880, n. s., ii., 202—3 yr., A., oil bitter almonds, D. in two and a half hours.

[8] Taylor: London M. Gaz., 1845, xxxvi., 103—M., A., 20 m., R. Lancet, 1845, ii., 656—M., 19, about 2 grains, S., D. in three hours.

[9] See Clark: Am. J. M. Sc., 1854, n. s., xxviii., 103. Rennard: Ph. Ztsch. f. Russland, 1873, viii., 230. Struve: Ztsch. f. an. Ch., 1873, xii., 14. Griffin: M. Times and Gaz., 1864, ii., 434.

after he was known to have been alive, no odor was observed by the watchman a few feet away in the corridor, toward which the wind was blowing from an open window in the cell, nor did the cell or the body exhale any odor, although it was quite marked on opening the body.[1] In six of Fagerlund's seven cases of cyanic poisoning[2] the odor was distinct at the autopsy, and in one (in May) although distinct at that time, it had disappeared three days later, when the analysis also gave negative results. In one case the odor was distinct in the stomach eight days after death.[3] The odor of bitter almonds is possessed by but two other substances: benzoic aldehyd (C_6H_5,COH), which is the chief constituent of oil of bitter almonds, and nitrobenzene (C_6H_5,N_2O). The latter is actively poisonous, and although the symptoms which it provokes in many respects resemble those of cyanic poisoning in character, their course is much less sudden in its onset and slower in development. Indeed, poisoning by "artificial oil of bitter almonds" has been confounded with cyanic poisoning.[4] The analytical characters of the three substances are quite distinct.

3. *Was a person killed by hydrocyanic acid capable of performing certain voluntary acts?* In the early case of Rex *v.* Freeman[5] (1829) the medical evidence was strongly against the possibility that the deceased could have herself corked the phial, wrapped it in paper and adjusted the bed clothes, all of which could have been accomplished in less than ten seconds. That this view is unsound has been proved by many instances in which considerable voluntary movement has been performed by persons after having taken a lethal dose of a cyanic poison which afterward caused death.[6]

4. *Was the hydrocyanic acid found produced by the analytical process from normal constituents of the body or from articles of food?* The only substances normal to the body which are capable of yielding hydrocyanic acid under the condi-

[1] Shapleigh: Tr. Coll. Phys., Phila., 1869, n. s., iv., 290. We are at a loss to understand why "not a trace of poison could be found in the stomach" on analysis, although every part of the body gave off a distinct odor of hydrocyanic acid, and that substance was detected in the contents of a small phial found in the cell.

[2] Vrtljschr. f. ger. Med., 1894, 3 F., viii., supplh., 86.

[3] Griffith: Lancet, 1864, i., 52.

[4] Maser: Med. Rec., N. Y., 1884, xxv., 711.

[5] London M. Gaz., 1831, viii., 759, 795.

[6] See cases cited in note 3, p. 611.

tions of any usual system of analysis are the thiocyanates already referred to, which, when distilled in the presence of a mineral acid, may yield hydrocyanic acid, but do not do so in the presence of excess of a bicarbonate as in the method of analysis above directed (see also No. 5).

If any of the vegetable products containing amygdalin be present in the food and remain in the stomach contents after death they will yield hydrocyanic acid on analysis. The quantity, however, will be porportionate to that of the amygdaloid vegetable taken, and if this be sufficient to furnish any notable quantity of prussic acid, the death will have been due to cyanic poisoning quite as truly as if hydrocyanic acid had been taken. A microscopic examination of the stomach contents will also reveal the presence of the vegetable tissues.

5. *Is hydrocyanic acid a normal or morbid product of the animal body, or may it be produced during putrefaction?* Husemann[1] cites several ancient authors who have claimed to have observed the presence of hydrocyanic acid in the urine and in certain pathological fluids. In the entire absence of modern confirmation of these observations it is probable that they resulted from faulty methods and the presence of thiocyanates.

In two English cases cited by Taylor[2] the argument was advanced that prussic acid might have been formed post mortem in the stomach contents, and Orfila[3] admitted the *possibility* of its production during putrefaction.

At the present time we think it highly probable that a putrefaction enthusiast might be found to advocate the theory of the formation of hydrocyanic acid in the body in favor of the defence on a trial for murder by cyanic poisoning, basing his opinions upon the constitution of the adenyl or nuclein bases, which are normal products of the animal body. These bases form a series of five: uric acid ($C_5H_4N_4O_3$), xanthin ($C_5H_4N_4O_2$), hypoxanthin ($C_5H_4N_4O$), guanin ($C_5H_5N_5O$), and adenin ($C_5H_5N_5$), the last-named being the first formed in the body as a product of decomposition of phosphorized bodies, the nucleins, existing in tissues and organs rich in nucleated cells. Adenin is a polymere of hydrocyanic acid (CHN), and on de-

[1] "Handb. d. Tox.," 1862, 721.
[2] "Poisons," 3d Am. ed., 579.
[3] "Tox. gén.," 5ème ed., ii., 405.

composition might yield that substance. According to Kobert,[1] Bruylants has advanced the theory that the cyanogen group is actually produced in this manner in the body, and, combining with unoxidized sulfur, produces the thiocyanates of the saliva, milk, urine, etc. Vaughan and Novy[2] also refer to the relationship of adenin and hydrocyanic acid and cite the poisonous qualities of some of the leucomains as being "of great significance" in connection with the presence in them of "the hydrocyanic acid molecule." The statement just quoted is misleading, "the hydrocyanic molecule" is no more "contained in" adenin than is that of acetylene (C_2H_2) in that of benzene (C_6H_6). The two polymeres differ entirely in structure and the conversion of one into another requires a powerful agency, as when acetylene yields benzene under the influence of a temperature approaching that of melting glass, or when adenin is split up into hydrocyanic acid by caustic potash at 200° (392° F.). In the normal metabolism hydrocyanic acid is not produced, and from the nucleins the changes take place through adenin, guanin, hypoxanthin, xanthin, uric acid, and urea. The formation of hydrocyanic acid would involve not only a change in the normal process but a reversal. That this retrograde change does not occur during putrefaction has been experimentally demonstrated by Schindler,[3] who has shown that in putrid material adenin is converted into hypoxanthin (adenin + O − NH = hypoxanthin) and guanin into xanthin (guanin + O − NH = xanthin).

Apart from theoretical considerations two facts may be cited in support of a negative answer to this question: 1. We find no mention by any modern writer of the detection of hydrocyanic acid as such or as simple cyanid in an animal body higher in the scale of nature than the myriapods, except it has been introduced from without.[4] 2. Numberless analyses for volatile poisons of portions of the body in all stages of preservation and decay have been made with negative results as to the reactions for hydrocyanic acid.

[1] "Intoxikationen," 736.

[2] "Ptomains," etc., 2d ed., 283, 354.

[3] Zeitsch. f. phys. Ch., 1889, xiii., 432.

[4] Hydrocyanic acid is one of the products of the decomposition of albuminoid substances effected by the action of powerful reagents, such as a mixture of sulfuric acid and potassium dichromate, or by fusion with caustic potash.

OXALIC ACID AND OXALATES.

The salt obtained by the evaporation of the juice of *Oxalis acetosella*, and now called *salt of sorrel* or *binoxalate of potash*, was known as early as the middle of the seventeenth century, as Duclos makes mention of it in the Memoirs of the Academy for 1668. A century later (1773) oxalic acid was obtained from this salt by Savary. Subsequently Scheele showed the oxalic acid obtained from sorrel to be identical with the *acid of sugar* obtained by Bergmann in 1776 by the action of nitric acid on sugar.[1]

Oxalic Acid—$C_2O_4H_2$, 2Aq.—125.7—is prepared industrially by the action of nitric acid upon sugar or starch, or by the action of an alkaline hydroxid in fusion upon sawdust. It crystallizes in transparent prisms, which effloresce on exposure to air and lose their Aq., slowly but completely, at 100° (212° F.) or in a dry vacuum. It fuses at 98° (208°.4 F.) and between 110°–132° (230°–269°.6 F.) it partly sublimes in the anhydrous form and is partly decomposed. At a higher temperature it is completely decomposed into water and the two oxids of carbon, or into carbon dioxid and formic acid. It dissolves in 15.5 parts of water at 10° (50° F.), and is quite soluble in alcohol. Its solutions are strongly acid in taste and reaction.

Oxalic acid is extensively used in the arts in dyeing and calico printing, for cleaning leather, in straw bleaching, etc., and in the household for whitening woodwork, scouring brass and copper, removing ink stains from fabrics, and in the composition of certain "liquid blues."

Monopotassic Oxalate—KHC_2O_4—127.82—also known as *hydropotassic oxalate*, or *binoxalate of potash*, exists in the juices of many acid plants, such as sorrel and rhubarb, and in many other vegetables. The *essential salt of lemon* or *salt of sorrel* used in the household for the removal of ink or fruit stains from clothing, etc., and in straw bleaching, is a mixture of this salt with the "quadroxalate," KHC_2O_4, $H_2C_2O_4 + 2$ Aq. It is a white, crystalline salt, closely resembling Epsom salt in appearance, and very soluble in water, forming an acid solution; but insoluble in alcohol.

[1] Bergmann's "Essays," Cullen's Transl., London, 1788, i., 305. Scheele: "Werke," Ed. Hermbstädt, Berlin, 1793, ii., 369.

Calcium Oxalate, CaC_2O_4—127.69—is a white, crystalline powder, soluble in the presence of sufficient mineral acid, but insoluble in water, in alkaline solutions, or in those owing their acidity to acetic acid. This salt is a normal constituent in small amount (about 0.02 gm. = $\frac{3}{10}$ grain in twenty-four hours) of the urine, in which it is held in solution by the acid phosphates. Under certain conditions it is increased in amount (oxaluria) and is deposited from the urine on standing in "letter-envelope" or "dumb-bell" crystals, sometimes in relatively large octahedra. Occasionally it forms vesical calculi (mulberry calculus).

Statistics and Origin.—The earliest case of oxalic-acid poisoning of which we find record occurred in England in 1814,[1] and five other cases occurred in the same country during the years 1815–19.[2] Among these are the earliest of the great number of instances in which the acid was taken or dispensed in mistake for Epsom salt.

Poisoning by oxalic acid and the oxalates is of very rare occurrence in France, while in England, Germany, and the United States several cases occur annually. Briand[3] gives a table of 280 poisonings in France from 1825 to 1857; Flandin[4] one of 200 cases, in the years 1841–1844; and Tardieu[5] one of 793 criminal poisonings 1851–1871. No case of oxalic acid or oxalate poisoning is mentioned in any of these tables. In England, according to Taylor,[6] there were 527 poisonings, in which the nature of the poison was known, during the years 1837–38, of which 19, or 3.6 per cent., were by oxalic acid. According to the Reports of the Register-General of Great Britain there were in the year 1840 138 suicides by poison, of which 2 were by oxalic acid; and during the years 1863 to 1867, 453 suicides by identified poisons, of which 58, or 12.8 per cent., were by oxalic acid. During the same years (1863–67) the total identified poisonings numbered 1,625, of which 66, or 4.06 per cent., were by oxalic acid. In the years 1871–80 of 1,000 suicides by poison in England and Wales 159, or 15.9 per cent., chose ox-

[1] Royston: London M. S. and Phys. Reposit., 1814, i., 382.

[2] Roberts: *Ibid.*, 1815, iii., 380. Johnson: *Ibid.*, 1816, vi., 474. Smith: *Ibid.*, 1819, xii., 18. Williams: *Ibid.*, 1819, xi., 20. Cooper: "Tracts on Med. Jur." (1818), 449.

[3] "Man. d. méd. lég.," 6ème ed., 1858, p. 414.

[4] "Tr. des poisons," 1846, p. 449.

[5] "Étude . . . sur l'empois.," 2ème ed., 164.

[6] "Poisons," 3d Am. ed., 176.

alic acid. Lesser reports the statistics of poisonings treated in the large Berlin hospitals in 1876–78. The total number was 432, and 19, or 4.4 per cent., were by oxalic poisons.[1] Munzer[2] gives abstracts of the appearances observed in 13 autopsies after death by oxalic poisoning made in Berlin in 1885–86. The reports of the New York City Board of Health from 1866 to 1880 refer to 16 oxalic poisonings in a total of 872.

The explanation of the relative frequency of oxalic poisoning in England and America and its rarity in France is undoubtedly in the common domestic use of oxalic acid and salt of sorrel as a cleansing agent in the former, and the non-prevalence of such use in the latter. The great majority of oxalic poisonings have been either accidental, from mistaking the acid or its potassium salt for Epsom salt or for other medicine, or suicidal; and a great proportion of the cases have occurred in domestic servants. In one instance a boy of five years died the day after eating a large quantity of sorrel (*Rumex acetosa*).[3] In another a woman died in three days from oxalic acid used in making a "lemonade."[4] Of 169 cases which we find reported in medical literature previous to 1895,[5] 83 were suicides, of whom 54 were women, and 41 were accidental. In one instance a discharged servant took an ounce of the acid in a pint of whiskey, more to annoy her mistress than to destroy herself;[6] in another it was not determinable whether a woman had committed suicide or had been poisoned by her husband.[7] The earliest of twelve British cases in which a homicidal intent was alleged was in a trial for assault in 1827, referred to by Christison, in which it was shown that the defendant had previously attempted to poison his wife with oxalic acid in gin.[8] Taylor[9] also incidentally alludes to a trial for murder by oxalic acid which took place in 1832. The same author refers to the case of Reg. *v.* Dickman, in 1845.[10] Two cases are reported in 1857, one that of Reg. *v.* Cochrane,[11] in which there was strong evidence that a woman had poisoned

[1] "Atl. d. ger. Med.," p. 1.
[2] Diss., Berlin, 1887.
[3] Suckling: Lancet, 1886, ii., 227. Also *edit.*, *ibid.*, i., 1229.
[4] White: Boston M. and S. Jour., 1870, lxxxii., 57.
[5] The small proportion of cases so reported is evidenced by the fact that 159 suicides by oxalic poisons occurred in ten years in England.
[6] Dabbs: Brit. M. Jour., 1886, i., 442.
[7] Lancet, 1839-40, ii., 29.
[8] "Poisons," Amer. ed., 167.
[9] "Poisons," 2d ed., 310.
[10] "Poisons," 3d Am. ed., 224.
[11] *Ibid.*

her two children, aged four and six years. The other was that of a woman who attempted to destroy herself and child. The latter recovered, but the woman was found dead.[1] Two years later the result was the reverse in another case:[2] an infant of twelve days succumbed; its mother recovered and was sentenced to fifteen years penal servitude. A suspected homicide in the same year is reported by Skinner.[3] In 1862 a woman was tried for the murder of her father by repeated administration of salt of sorrel. The verdict of "not proven" was found, as, although it was shown that she had administered the poison, an interval of fifteen days elapsed between her last opportunity to do so and the fatal termination.[4] In 1866 a woman as convicted of attempting to forcibly administer oxalic acid in solution to her six-year-old daughter.[5] In 1869 a coroner's jury found a verdict of "wilful murder" against a woman who had destroyed her child and attempted suicide with salt of lemon.[6] In 1872 a woman in Liverpool was charged with attempting to poison her brother with oxalic acid.[7] In 1877 an alleged attempt to poison with salt of lemon was the subject of a trial resulting in acquittal.[8] In a German case a man killed his bride by cutting her throat, having previously either administered to her or witnessed her taking a quantity of potassium quadroxalate, and after her death made a feint at suicide with the same salt.[9]

Lethal Dose.—It would appear from an examination of the reported cases in which the dose taken is numerically stated, that the most dangerous quantity is from 15 to 30 gm. (half an ounce to an ounce), and that quantities in excess of these are less liable to cause death. Thus of 49 cases in which the quantity taken was numerically reported it was 15.5 gm. (½ ℥) in 14 cases, with 6 deaths; one ounce (30 gm.) was taken in 18 cases, with 8 deaths; while in the 3 instances in which greater

[1] Ph. J. and Tr., 1857–58, xvii., 384.

[2] Reg. *v.* Marg. Macdonald, 1859. Littlejohn: Edinb. M. Jour., 1861, vii., 13.

[3] Brit. M. Jour., 1060, i., 107, 186. See also Thudichum: *Ibid.*, Febr. 18th.

[4] Reg. *v.* Mary Struth, Conway: Edinb. M. Jour., 1862, viii., 93.

[5] Reg. *v.* Morris, Taylor: *Loc. cit.*, 239.

[6] Ph. J. and Tr., 1869–70, n. s., xi., 369.

[7] Case of Jane Sutton, *ibid.*, 1872, 3 s., ii., 675. We find no reference to the further disposal of either of these two cases.

[8] *Ibid.*, 1877–78, 3 s., viii., 37.

[9] Casper-Liman: "Handb. d. ger. Med.," 8te Aufl., ii., 543. In the same place are reports of several other cases which contain no allusion to motive.

quantities were taken (39, 16.5, and 62.2 gm.) the persons recovered.[1] It would seem, therefore, that, owing to the early vomiting provoked by the poison itself, excessive doses are less liable to prove fatal than smaller toxic quantities. Yet in some cases in which death has occurred shortly after the ingestion the dose was probably large, although the amount is not definitely stated in the reports.[2] The smallest quantity which has been alleged to have proved fatal was about 4 gm. (¼ of ʒ ss.), which is said to have caused the death of a boy of sixteen years.[3] Double that amount (two drachms = 7.8 gm.) proved fatal in 3 instances.[4]

Duration.—The duration of a case of oxalic poisoning is usually short if it terminate in death, but in a certain class of cases the victim dies, usually suddenly, after an illness of from 2 to 14 days. Of 74 fatal cases 62 lasted less than 24 hours; in 12 the victim was found dead, in 8 death occurred "soon," in 5 in less than 10 minutes, in 15 in from 10 to 30 minutes, in 9 in from one-half to 1 hour, in 2 in 2 hours, in 1 in 3 hours, in 1 in 4 hours, in 2 in 5 hours, in 1 in 6 hours, in 1 in 9 hours, in 1 in 10 hours, in 3 in 13 hours, and in 1 in 21 hours. Of the 12 cases of longer duration, 1 died in 36 hours, 2 in 2 days, 2 in 3 days, 2 in 5 days, 1 in 6 days, 1 in 7 days, 1 in 8 days, 1 in 10 days, and 1 in 14 days. The most rapidly fatal case was that of a woman of 43 years who took from one to two ounces (31–62 gm.) of the acid, and died without pain and in collapse in 3 minutes. "The stomach was rotten" and contained altered blood.[5] In one of Munzer's cases, also a female suicide of 37 years, death occurred in 5 minutes.[6] In 3 instances the duration was about 10 minutes.[7] The most protracted case was one of the earliest reported: that of a man who took half an ounce (15.5 gm.) of the acid in mistake for

[1] Wharton and Stillé: "Med. Jur.," 1st ed., 496—F., A., 1¼ drachms. Johnson (Silk): Brit. M. Jour., 1881, i., 640—M., 30, S., ʒ iss. Tapson: London M. Gaz., 142, xxxi., 491—F., 50, S., ʒ ij.

[2] Wood: Monthly J. M. Sc., 1852, xiv., 227. Lesser: "Atl. d. ger. Med.," i., 46, 49.

[3] Barker: Assoc. M. J., 1855, ii., 1073; Lancet, Dec. 1st, 1855.

[4] Bayley: Lancet, 1883, i., 406—M., 47, S.; Ph. J. and Tr., 1877–78, 3 s., viii., 1016—M., ad., S. Cooper: Lancet, 1883, i., 406.

[5] Ogilvie: Lancet, 1845, ii., 205.

[6] *Op. cit.*, p. 24, Fall 11.

[7] Herapath: Med. Times and Gaz., 1868, ii., 456—F., ad., S., ʒ vi. (23 gm.). Reincke: Pharm. Ztg., 1882, xxvii., 757—M., ad. Lesser: "Atl. d. ger. Med," i., 46, 49—F., 38, S. A large dose of salt of sorrel on an empty stomach.

Epsom salt. He appeared to improve, but later became delirious and died in 14 days after the accident.[1] Another man fell a victim to the same mistake after lingering for 10 days. The dose was one ounce (31 gm.).[2] In one of the few French cases a woman of 74 years died from the effects of oxalic acid in 8 days, with symptoms resembling those of cholera.[3]

Prognosis.—So far as can be judged from statistics, the prognosis as to the final result is unfavorable. There were 104 deaths in 163 reported cases, a mortality of 63.8 per cent. Nor is the outlook for complete recovery in non-fatal cases quite favorable if the poisoning have been at all serious. While the corrosive power of oxalic acid is inferior to that of the mineral acids, and while we know of no instance in which death has been ascribed to the secondary effects at a later period, as so frequently happens with the mineral corrosives, the possibility of such an occurrence must be recognized in view of cases in which the patient has come under observation many months after the poisoning, suffering from great gastric irritability, dyspepsia, and constriction of the œsophagus. Probably an autopsy in such cases would reveal the existence of œsophageal, cardiac, or pyloric stricture and disorganization of the gastric mucous membrane.[4] On the other hand, in Silk's case,[5] there were no signs of œsophageal stricture twenty-five days after the poisoning, although the large dose of one and one-half ounces (47 gm.) had been taken in solution; and in Barham's case of a woman who took an ounce in solution (31 gm.), no secondary effects were observable when she was seen seven years later.[6] Although a few instances are recorded in which patients have been discharged from hospital in two or three days after a serious oxalic poisoning,[7] in most cases the renal disturbance continues for from two to four weeks, and in some instances other symptoms, such as local anæsthesia,[8] spasmodic twitchings,[9]

[1] Fraser: Edinb. M. and S. J., 1818, xiv., 260.

[2] Jackson: Boston M. and S. Jour., 1844, xxx., 17.

[3] Montagum: Lyon méd., 1886, li., 45.

[4] See Danev: Transylvania J. M., 1835, viii., 594. O'Shea: Lancet, 1845, ii., 293.

[5] Brit. M. J., 1881, i., 640.

[6] Prov. M. and S. J., 1847, 544.

[7] Gray: Med. Press and Circ., 1882, n. s., xxxiv., 47—F., 30, A., ʒ ss. (15.6 gm.). Tolafree: Boston M. and S. Jour., 1835, xii., 158—F., ad., A., ʒ ij. (7.8 gm.). Deane: Prov. M. and S. Jour., 1851, 344—M., 22, A., ʒ i. (31 gm.).

[8] Sarganek: Schmidt's Jahrb., 1884, ccii., 18, Case VI.

[9] Bourne: Lancet, 1851, i., 329.

vomiting,[1] and alteration of voice,[2] persist for several weeks. A man who attempted suicide with three teaspoonfuls of oxalic acid had a violent attack of vomiting and hæmatemesis in the second week and only recovered a month later.[3]

Symptoms.—Oxalic acid is both a corrosive and a true poison, one or the other action predominating according to the size of the dose and the degree of concentration of the solution. If it be taken in the solid form or in concentrated solution, as is usually the case, the symptoms of corrosion are the first to appear and may be the only ones observed. But if the poison be taken in dilute solution the symptoms of corrosion may be entirely absent.

In a typical case of oxalic poisoning, the dose being in the neighborhood of 15 gm. (℥ ss.), taken in concentrated solution, the patient experiences the first effects either immediately, during the act of swallowing, or within a few moments. In but three instances does the report convey the impression that from ten minutes to half an hour elapsed before the manifestation of symptoms, and in those it seems probable that the interval referred to is that preceding the observation rather than that preceding the sensations of the patient.[4]

The strongly acid taste is followed by a sense of heat in the mouth, throat, and stomach. This rapidly increases in intensity until it becomes a violent, burning pain. In some cases the pain is accompanied by a sense of constriction of the throat and of impending suffocation. The act of swallowing is performed with difficulty, and later the voice becomes fainter and husky, and sometimes is completely extinguished. Within ten or fifteen minutes violent and persistent vomiting begins in almost every case. The vomited matters are most frequently of "a coffee-ground" character, and separate on standing into two layers: the upper a clear, yellowish, and strongly acid liquid, the lower a thick red-brown sediment of altered blood. Often

[1] Sarganek: *Loc. cit.*

[2] Bradley: Med. Times, London, 1850, n. s., i., 293.

[3] Sleman: Lancet, 1891, 192.

[4] Fraenkel: Ztschr. f. kl. Med., 1880-81, ii., 664—M., 48, S., R. (ten minutes); St. Geo. Hosp. Rep., 1877-78—M., 51, S., R., ℥ i. (one-quarter hour). Ellis: Lancet, 1864, ii., 265—F., 50, S., R., ℥ i. (one-half hour). Didama's case, reported by Wharton and Stillé, "Med. Jur.," 4th ed., ii., 88, departs in so many respects from the usual course of an oxalic poisoning that, in the absence of anything beyond the mere statement, it is questionable whether the substance mistaken for Epsom salt was really oxalic acid.

the vomited matters are black or become so on standing, and produce a black stain upon a board floor. Occasionally true hæmatemesis is observed. When the acid has been taken in small quantity or in dilute solution the vomit may be colorless and free from blood, as it may also be in rapidly fatal cases, even when the stomach is found greatly inflamed at the autopsy.[1] In some cases persistent vomiting and pain, and later persistent purging of a bloody material, are the only marked symptoms, and they may continue with or without intermission for from five to ten days, terminating in death from exhaustion.

When very large doses are taken (30–60 gm. = ℥ i.–℥ ij.), the patient, after vomiting, may go into a state of collapse, and die within five minutes. The lips, mouth, and fauces are, shortly after the poison has been taken in solution, reddened, swollen, and painful. Later they become paler, and finally, sometimes within an hour, of a dirty, ashen-white hue, either throughout or in patches. The tonsils and uvula are much swollen, and there is severe thirst.

Soon the symptoms due to the truly poisonous action of the oxalate are added to those caused by its immediate corrosive action upon the alimentary canal. The countenance is pale, anxious, and haggard, the upper lip trembling, the lower jaw relaxed. The surface is bathed in a cold, clammy perspiration. The fingers are semiflexed and rigid, the nails blue. The eyes are glazed and the pupils contracted. There is sometimes persistent hiccough. The pulse is small and thready, sometimes imperceptible. There is general numbness and a sense of tingling and cramps in the upper and lower extremities. Abdominal pain is no longer complained of, although the abdomen may remain tender to pressure; but the patient suffers violent lumbar pains, shooting down into the lower extremities. The respiration is quick and labored. The skin in some cases is marked with a peculiar exanthem resembling roseola. The urine is at first diminished in quantity and may be completely suppressed for two or three days. Later the quantity rises much beyond the normal; its specific gravity is subnormal throughout, and the elimination of urea and of chlorids, at first deficient, is afterward greatly increased. Albumin is present in considerable quantity and the sediment contains epithelium,

[1] Geoghegan : Dublin M. Press, 1846, xv., 209.

granular or hyaline casts, and calcium oxalate, both crystalline and amorphous. After removal of albumin the urine contains some substance, not sugar, capable of reducing Fehling's solution. The elimination of oxalic acid by the urine continues for two weeks, at least in non-fatal cases.[1] In some instances there have been violent spasms of a tetanic character,[2] in others delirium.[3] In some cases the patient has rapidly become stupid, somnolent, unconscious, and has died comatose.[4] In one instance the frequency of epileptic seizures to which the patient was subject was much increased.[5]

Treatment.—The first indication is to neutralize the acid, without formation of a soluble oxalate, or to convert a soluble into an insoluble oxalate. For this purpose compounds of calcium or magnesium should be given, preferably the former, as magnesium oxalate is slowly formed and is not so insoluble as the calcium compound. The best preparation for the purpose is *syrup of lime*. But this is rarely available and time should not be wasted in searching for it. The most serviceable preparation is calcium carbonate in the shape of finely divided (precipitated) chalk, suspended in a small quantity of water or of chalk mixture. The quantity given should be sufficient not only to convert all oxalic acid present into calcium oxalate, but also to render the stomach contents neutral. For this purpose 50 gm. of chalk will suffice for a dose of 30 gm. of oxalic acid. The use of a carbonate is attended with the inconvenience of the liberation of gas which attends its neutralization of the acid, but the danger from this cause is by no means as great as when the mineral acids have been swallowed. Of the other available calcium preparations, lime water is too dilute and milk of lime is possibly too caustic. The antidote should be given with the least possible delay, and after a few minutes the stomach should be washed out, at first with lime water, afterward with water. The carbonates of potassium and sodium are useless, as the oxalates of those metals are soluble and quite

[1] For examinations of urine see Fraenkel: Ztschf. f. kl. Med., 1880–81, ii., 664. Sarganek: Diss., Berlin, 1883. Didier: J. d. Sc. m. d. Lille, 1894, i., 497. Hood: Lancet, 1886, i., 347.

[2] Bourne: Lancet, 1851, i., 329.

[3] Jackson: Boston M. and S. Jour., 1875, n. s., xiv., 445; *Id.*, 1844, xxx., 17.

[4] Perry: Glasgow M. Jour., 1870–71, iii., 120. Tidy: Lancet, 1872, ii., 41.

[5] McDaniell: Montreal M. Jour., 1890–91, xix., 104.

as poisonous, although not so corrosive as the acid. Tendency to collapse is to be combated by stimulants; the nephritic symptoms by diuretics and abundance of liquid (which should be avoided until the stomach has been washed out), and the acidism by the administration of alkalies.

Post-Mortem Appearances.—The lips, tongue, mouth, and œsophagus are of an opaque, yellowish-white color, sometimes marked with patches of a reddish hue. The stomach is contracted and in many cases contains a thick, gelatinous, reddish-brown and acid liquid, somewhat similar to the "coffee-ground" material vomited during life. The peritoneal surface of the organ, as well as the mesentery and the greater portion of the peritoneal surface of the intestines, are marked by blood-vessels filled with dark, fluid blood. The mucous surface of the stomach is strongly corrugated, and in most cases presents a uniform bright-red color in the elevations and depressions, except in so far as it may have been changed to brown, or even black, by post-mortem action. In some cases the mucous surface is either in part or in whole pale, opaque, or translucent, and marked with a coarse ramiform vascularity of the submucous tissue. Similar appearances are observed in the duodenum and jejunum. The mucous membrane, when it remains, is soft, pulpy, and easily detached. Although perforation has been observed,[1] it is of rare occurrence, and probably post mortem. Crystals of oxalic acid or of monopotassic oxalate are not frequently found in the stomach, although Lesser[2] figures the stomach of a patient who died within ten minutes, which is almost uniformly pale, contracted, and plentifully lined with crystals of monopotassic oxalate. Microscopic crystals of calcium oxalate are, however, found in many cases in the stomach and intestines, particularly when death has followed, not within a few minutes, but in the course of from three to six hours. A microscopic examination of the kidneys shows the presence in the tubules, and sometimes in the tissue, never in the glomeruli, of crystals of calcium oxalate. These are more frequently imperfect octahedra, needles, dumb-bells, whetstone crystals or plates than perfectly formed octahedra. The latter, how-

[1] Wood: Monthly J. M. Sc., 1852, xiv., 227. Littlejohn: Edinb. M. Jour., 1861, vii., 13.

[2] "Atl. d. ger. Med.," i., Pl. viii., f. 1. See also Pl. iv., f. 3; Pl. vi., f. 1; Pl. vii., ff. 1, 2 and 3; Pl. xiv., f. 6.

ever, are found in abundance, and of varying size, in the urine.

Analysis.—The parts to be examined are the stomach and intestines and their contents, the liver, kidneys, and urine; also vomited matters. The contents of the stomach and vomit are strongly acid in reaction unless antidotes have been administered, in which case they may be neutral or even alkaline.

In a systematic analysis oxalic acid is to be looked for in the ethylic ether extract from the acid aqueous liquid (see page 136). If oxalic acid or oxalates alone are to be sought for, the materials are to be treated directly as below.

It must be remembered that the acid sought may be present either in the free state, in combination as a soluble oxalate, or, in consequence of the administration of antidotes, as the insoluble calcium oxalate or the very sparingly soluble magnesium oxalate.

The substance under examination, if acid, is first to be extracted with water, the solution filtered, the filtrate evaporated over the water-bath, the residue extracted with alcohol, the filtered alcoholic solution evaporated, and the residue redissolved in a small quantity of water. The solution so obtained (No. 1) will contain any free oxalic acid which may have been present. The material left undissolved by alcohol in the preparation of solution No. 1 is next to be extracted with alcohol acidulated with hydrochloric acid, the solution filtered and evaporated, and the residue redissolved in a small quantity of water. This solution (No. 2) will contain any oxalic acid which may have been present in the form of a *soluble* oxalate. Lastly, the material left undissolved by water and alcohol in the preparation of solutions Nos. 1 and 2 is to be treated with a sufficient quantity of potassium carbonate (not hydroxid) to render it distinctly alkaline, and boiled for two hours. The solution is filtered and evaporated, the residue extracted with alcohol acidulated with hydrochloric acid, the solution filtered and evaporated, and the residue redissolved in water. This solution (No. 3) will contain oxalic acid, if it were present in the form of an *insoluble* oxalate.

The TESTS for oxalic acid are then to be applied to portions of the three solutions:

1. A solution of a calcium salt produces, in neutral or alka-

line solutions, a white precipitate which redissolves in hydrochloric acid.

2. Silver nitrate solution produces a white precipitate which dissolves in ammonium hydroxid solution and also in nitric acid. If the liquid containing the precipitate be boiled the latter does not darken. If the precipitate be collected, dried, and heated upon a strip of platinum foil it explodes.

3. Lead acetate solution, in solutions of oxalates which are not too dilute, produces a white precipitate which is soluble in nitric acid, but insoluble in acetic acid.

The urine, contents of stomach, and vomited matters should also be examined microscopically for crystals of calcium oxalate.

The detection of a mere trace of oxalic acid or of an oxalate can only be of value as corroborative evidence in a case of suspected oxalic poisoning, owing to the presence of oxalates in articles of food, in medicines, and in the human economy.

TARTARIC ACID.

Although poisonings by tartaric acid are of extreme rarity, that substance has been of forensic interest in two fatal cases. In one (Affaire Weber, 1847) a man and woman were found about five hours after having been seen in health, the latter dead, the former almost dead. According to Devergie, Bayard, and Bouchez tartaric acid was the cause of the woman's death. The facts, however, upon which this opinion was based were shown by Orfila to be insufficient and the man was discharged after an elaborate inquiry.[1] In one English case (Reg. *v.* Watkins, 1845) a druggist was tried for manslaughter for having caused the death of a man of twenty-four years, by giving him one ounce (31 gm.) of tartaric acid in mistake for aperient salts. The man died in nine days.[2]

One other case of alleged tartaric acid poisoning was reported by Trevithick,[3] but the evidence that tartaric acid was the cause of the disorder and death is not conclusive.

[1] See Devergie: "Méd. lég.," 3ème ed., iii., 307; Ann. d'hyg., etc., 1851, xlvi., 432; 1852, xlvii., 383. Orfila: "Tox. gén.," 5ème ed., 977; Ann. d'hyg., 1852, xlvii., 199.

[2] Gill: Lancet, 1845, i., 18; Ph. J. and Tr., 1844–45, iv., 370. Taylor: "Poisons," 3d Am. ed., 239.

[3] Brit. M. Jour., 1893, i., 1321.

Monopotassic tartrate, $C_4H_4O_6HK$, or *cream of tartar*, is extensively used in the household, in the manufacture of baking powders, and as a medicine. Sodiopotassic tartrate, $C_4H_4O_6NaK$, or *Rochelle salt* or *Seignette salt*, is a product of the reaction in the use of tartrate baking powders, and is also used as a medicine.

Cream of tartar has proved fatal in two instances. In one, the servant of an empiric took over four ounces (125 gm.) while drunk and died in four days.[1] In the other, which was the subject of a trial for homicide by negligence in Germany, a man, mistaking the directions of an empiric, took 200 gm. (about ℥ viss.), partly dissolved and partly suspended in water, and died in twelve hours.[2]

Symptoms.—In none of these reports is any detailed account of symptoms given. In Gill's case the man suffered a burning sensation, as if "all on fire," sickness, inflammation of the stomach and intestines, and vomiting until death, which occurred in nine days. In Tyson's case the patient became very weak, could hardly walk in the evening, on the second day after was said to have had several stools during the night, and vomited almost continuously. There was pain at the umbilicus, and great thirst. The tongue was brown and dry, and the pulse feeble. The legs became paralyzed and there was severe lumbar pain.

Post-Mortem Appearances.—These have been more carefully observed in Tyson's and Roger's cases, although in the latter some of those described are probably due to disease. The most notable appearances are in the stomach and intestines, the gastric mucous membrane in Roger's case was of a uniform dark, black-red color over the entire fundus, which changed to a coarse network upward and to an arrangement of parallel lines toward the lesser curvature. Between these markings the mucous membrane was evenly pale red, and marked with a great number of minute punctiform and stellate, bright-red spots. The duodenal mucous membrane was uniformly pale red, with numerous punctiform bright-red spots. A similar appearance was noted in the small intestine, where there were also numerous, almost microscopic white dots.

[1] Tyson: Lancet, 1837–38, i., 162. [2] Roger: Friedreich's Bl. f. ger. Med., 1887, xxviii., 196

ALKALOIDAL POISONS.

The term *alkaloid* is one now undergoing modification in the light of increasing knowledge of the chemical constitution of the vegetable and animal bases. In the wider sense all organic nitrogenized substances, basic in character and capable of neutralizing acids with the formation of salts in the same manner as ammonia, *i.e.*, without the simultaneous liberation of hydrogen or formation of water, are alkaloids. Under this view not only are the vegetable bases, such as morphin, strychnin, etc., alkaloids, but many animal and synthetic substances, such as urea, the amins (including such ptomaïns as putrescin and cadaverin), the hydrazins, etc., also come within this definition.[1] But as those vegetable alkaloids whose chemical constitution has been determined either completely or partially have, with the exception of caffein and theobromin, been found to contain the closed-chain nucleus *pyridin* (C_5H_5N), and consequently belong to the second of the two principal classes of organic compounds, while the other substances above mentioned belong to the first, the present tendency among chemists is to limit the application of the term alkaloid to basic derivatives of *pyridin* (C_5H_5N) and *quinolin* (C_9H_7N).

The number of vegetable alkaloids is large, and among them are some of the most important remedial agents. They differ greatly in their toxic power, some being actively poisonous in minute doses, others comparatively inert even when taken in large amount. Clearly only the former are of direct interest in this work, and of these many are substances difficultly obtainable, and unknown even to medical practitioners. Even the rarest of these may become of forensic interest, but the space at our command will permit of the consideration only of such as are of present interest.[2]

[1] This extended application of the term is accepted in one of the most recent works upon the alkaloids. Guareschi: "Introduzione alle studio degli Alcaloidi," pp. xi., 471, 4to, Torino, 1892.

[2] Information concerning the rarer alkaloids will be found in Guareschi: *Op. cit.* Dupuy: "Alcaloïdes," 2 vols., roy. 8vo, pp. 648, 775, Bruxelles, 1887–89. Pictet: "La constitution chimique des alcaloïdes végétaux," pp. 310, 8vo, Genève et Bale (Paris), 1888. Husemann and Hilger. "Die Pflanzenstoffe," 2te Aufl., 2 vols., 8vo, pp.

The alkaloids are either volatile, oily, pungent liquids (coniin, nicotin, spartein) containing no oxygen, or (the greater number) solid and generally colorless and crystalline bodies composed of carbon, hydrogen, nitrogen, and oxygen. Both fixed and volatile alkaloids combine with acids to form salts, for the most part crystalline, which bear the same relation to the free alkaloid that the ammonium salts do to ammonia gas.[1] Most of the free alkaloids are insoluble or difficultly soluble in water, but more or less readily soluble in alcohol, ether, benzene, chloroform, or other organic liquids. Their salts, on the contrary, are for the most part soluble in water or in alcohol, particularly in the presence of free acid, but are insoluble, with few exceptions, in the other organic liquids referred to. Upon these solubilities the methods for extracting alkaloids from complex mixtures are founded (see pp. 129–138). The reaction of the free bases is differently manifested according as litmus or phenolphthalein is used as an indicator; toward the former they are all alkaline except narcotin, narcein, piperin, and a few rare alkaloids, which are neutral. Toward phenolphthalein all alkaloids except atropin, coniin, homatropin, hyoscin, hyoscyamin, and nicotin, are neutral, and solutions of their salts behave as if the acid were free. Many of the alkaloids as well as their salts are bitter, some intensely so.

Most alkaloids are precipitated from solutions of their salts by the mineral bases and by a number of *general reagents*, and many of them give more or less characteristic *color reactions* with acids and other reagents (see pp. 139–149). Their behavior toward these, along with their *physiological action* (see pp. 161–163) and their physical properties, serve to identify such as are capable of identification by methods at present known.

1571, Berlin, 1882–84. Kobert: "Lehrbuch der Intoxikationen," 8vo, pp. 816, Stuttgart, 1893. Sohn: "Dictionary of the Active Principles of Plants," obl. 8vo, pp. 194, London, 1894.

[1] With due respect to analogy in nomenclature the name of the alkaloid in combination should terminate in *ium* and not in *in* or *ia*. As we speak of *ammonia*, NH_3, and *ammonium chlorid*, NH_4Cl, so we should refer to the compound of morphin (or morphia), ($C_{17}H_{19}NO_3$) and hydrochloric acid as *morphium chlorid* ($C_{17}H_{20}NO_3$, Cl).

ACONITE AND ACONITIN.

The Roman poets used the word *aconitum* in referring to any poisonous plant;[1] and Theophrastus,[2] Dioscorides,[3] and Nicander[4] describe different plants under the name ἀκόνιτον. But according to Fleming[5] all of these plants, as well as those described by Pliny and other early writers, belonged to genera other than aconitum, with the exception of the second of Dioscorides' species, which was probably *A. lycoctonum*.

Aconite was first suggested as a medicine by Störck in 1762, since which time several species have been used (*A. lycoctonum*, *A. ferox*, *A. anthora*, etc.) and found to vary greatly in strength. The species at present in use is *A. napellus*, popularly known as monkshood or wolfsbane. Although aconitin exists in all parts of the plant, the root contains it in greatest abundance and is the part from which the extract and tincture, the pharmaceutical preparations generally used, are prepared.

Aconitum napellus, according to the recent researches of Dunstan *et al.*,[6] contains four or probably five alkaloids, of which three are more abundantly present than the others.

Aconin.—$C_{26}H_{41}NO_{11}$—(or $C_{26}H_{39}NO_{10}$)—a colorless, gum-like, uncrystallizable, very hygroscopic solid, which is very soluble in water or alcohol, slightly so in chloroform, and insoluble in ether and in petroleum ether. Its aqueous solution is distinctly alkaline and dextrogyrous. Its salts crystallize with difficulty, and their solutions are lævogyrous. It is bitter in taste, but does not produce tingling of the tongue, and does not appear to be poisonous in small doses. The *napellin* of Hübschmann is a mixture consisting chiefly of aconin.

Benzoyl-aconin.—$C_{26}H_{40}(C_6H_5, CO)NO_{11}$—(= *isoaconitin*, or *napellin* of Dunstan)—a nearly colorless, varnish-like, amorphous solid, soluble in water, strongly alkaline and basic, forming crystalline salts. It is easily hydrolyzed by alkalies or by superheated water, yielding aconin and benzoic acid. Al-

[1] Juvenal: "Sat.," i., 156. Ovid: "Metam.," i., 147. Virgil: "Geo.," ii., 152.

[2] "Hist. Plant.," ix., 16, Ed. Bodæus, Amst., 1644, p. 1130.

[3] IV., 77, 78, Ed. Saracenus, Frankf., 1598, p. 275.

[4] "Alex.," 13, 42.

[5] "An inquiry into . . . Aconitum napellus," London, 1845, 1.

[6] J. Chem. Soc., London, 1891, lix., 271; 1892, lxi., 385, 395; 1893, lxiii., 443, 491, 991, 994; 1894, lxv., i., 308, Tr. 174, 176, 290; 1895, lxvii., Tr. 459, and authors there quoted.

though poisonous, its toxic power is much inferior to that of aconitin. In experiments upon animals a dose of benzoyl-aconin thirty times as great as the lethal dose of aconitin was required to cause death.

The *picraconitin* of Wright[1] is impure benzoyl-aconin, and the *atisin* of Broughton[2] is identical with it.

ACONITIN—*Acetylbenzoylaconin*—$C_{34}H_{39}(CH_3,CO)(C_6H_5CO)NO_{11}$—*Duquesnel's aconitin*—a faintly yellowish solid, crystallizing in transparent prisms, sometimes a centimetre in length,[3] which fuse at 188°.5 (367°.3 F.). Its solubility in water is one in 4,431 at 22° (71°.6 F.). It is readily soluble in chloroform and in benzene, less readily in alcohol and in ether, and hardly at all in petroleum ether. It forms crystalline salts. Alcoholic solutions of the alkaloid are dextrogyrous; aqueous solutions of its salts are lævogyrous. It is easily hydrolyzed by acids, alkalies, or water at high temperature, with formation of aconin, benzoic acid, and acetic acid. The pure alkaloid gives no known color reactions. The toxic power of aconitin is very high. Cash found the lethal dose for guinea-pigs, administered subcutaneously in solution of chlorid, to be only 0.000064 gm. (0.001 grain) per kilogram of body weight; and frogs of 25 gm. weight are killed by 0.000032 gm. (0.0005 grain).

PSEUDACONITIN—*Nepalin*—*Veratroylaconin*—$C_{36}H_{49}NO_{11}$—exists in very small quantity or not at all in *A. napellus*, but replaces aconitin in *A. ferox* (Indian or Nepaul aconite; Himalaya root). It crystallizes in needles, but is usually obtained as a varnish. It fuses at 104°–105° (219°–221° F.). When hydrolyzed it yields *pseudaconin* and veratic acid. It is highly poisonous.[4]

The JAPACONTIN obtained by Wright[5] from two varieties of Japanese aconite is identical with aconitin.[6]

LYCACONITIN, $C_{44}H_{60}N_2O_{12}$, and MYOCTONIN, $C_{40}H_{56}N_2O_{12}$, are amorphous, very poisonous alkaloids, obtained by Dragen-

[1] J. Chem. Soc., London, 1877, xvi., 143.

[2] Med. Press and Circ., 1874, n. s., xvii., 452.

[3] Tutton: J. Chem. Soc., 1891, lix., 288.

[4] Beckett and Wright: J. Chem. Soc., London, 1875, xxviii., 1265. Wright: *Ibid.*, 1877, xvi., 143; 1878, xxxiii., 151, 318. Boëhm: Arch. f. exp. Path. u. Pharm., 1873, i., 385.

[5] J. Chem. Soc., 1879, xxxv., 387, 399.

[6] Mandelin: Arch. f. Pharm., 1885, 3 R., xxiii., 97, 129, 161. Lubbe: Diss., Dorpat, 1890.

dorff and Spohn[1] from *A. lycoctonum*, which does not contain aconitin.

Rosendahl, in a valuable contribution to the chemistry and toxicology of the aconite alkaloids,[2] has described three other alkaloids obtained from the northern variety of the same species (*A. septentrionale*): LAPPACONITIN, $C_{34}H_{48}N_2O_8$, which forms large hexagonal prisms, colorless, bitter, fusing at 205°.1 (401°.2 F.); soluble in 330 parts of ether, forming a strongly fluorescent solution. On hydrolyzation it yields a crystalline and an amorphous alkaloid, and a crystalline acid which gives a blue-violet color with ferric chlorid. The alkaloid with sulfovanadic acid gives a yellowish-red color, passing to emerald green. Lethal dose for cats and dogs = 0.005 to 0.01 gm. per kilogram. SEPTENTRIONALIN, $C_{31}H_{45}N_2O_9$—an amorphous, yellowish powder, having a bitter taste and producing local anæsthesia. It fuses at 128°.9 (264° F.), and is soluble in 1.7 parts of alcohol, 2.1 of ether, and 58 parts of water. When hydrolyzed it yields the same acid as lappaconitin, and two similar alkaloids. Lethal dose for cats and dogs = 0.008 to 0.016 gm. per kilogram. CYNOCTONIN, $C_{36}H_{55}N_2O_{13}$, an amorphous, very hygroscopic, unstable substance, which fuses at 137° (278°.6 F.). With concentrated sulfuric acid it gives a deep red color. With fuming nitric acid, evaporation, and addition of alcoholic potash, it becomes blood-reddish, changing to red-brown. Lethal dose for cats, = 0.03 gm. per kilogram.

The commercial *aconitines* differ greatly in composition and in therapeutic and toxic activity. The name properly applies only to the crystalline alkaloid fusing at 183°–189° (370°.4–372°.2 F.). Of seventeen samples of French, German, and English "aconitines" examined by Dunstan and Carr[3] only two approached this standard of purity: one, an English product containing about three per cent. of benzoyl-aconin, and one a German preparation which was almost pure aconitin. The others contained from "very little" to seventy per cent. The crystalline character of the *salts* is no guar-

[1] Ph. Ztschr. f. Russland, 1884, xxiii., 313, 329, 345, 361, 377. See also van der Bellen: Diss., Dorpat, 1890; Dohrmann: Diss., Dorpat, 1888; Salamonovitz: Diss., Dorpat, 1884; Jacubowsky: Diss., Dorpat, 1884; Ejnberg: Diss., Dorpat, 1887.

[2] Arb. d. pharm. Inst. z. Dorpat, 1895, xi., 1–118.

[3] J. Chem. Soc., London, 1893, lxiii., 491.

anty of purity, as those of benzoyl-aconin and aconin are also crystalline.

ACONITIC POISONING.

Statistics.—Matthiolus relates two experiments upon criminals, one at Rome in 1524, the other at Prague, under his own direction, in 1561, in both of which one of the two subjects died and the other recovered under treatment.[1] Wibmer also cites several early accidental poisonings by aconite;[2] and Valentini[3] reports the murder of a man by his wife in Copenhagen in 1657 by "monk sroot" or "napellus."

In more recent times aconitic poisonings are of rare occurrence in France and Germany, although, since the introduction of "aconitin" they have increased in number; more frequent in England and in the United States, and much more frequent in India.[4]

Most of the aconitic poisonings reported in medical literature are ACCIDENTAL: 144 out of 173 by aconite and its preparations, and 13 out of 20 by "aconitin." In 57 instances the tincture or fluid extract was taken by the victim either without medical advice or through mistake in the directions; in 20 cases the liniment,[5] intended for external use, was swallowed; in 14 instances the poisonings were caused by errors of pharmacists in compounding medicines.[6] In 18 poisonings the root itself or

[1] "Comment. in Dioscorides," Ed. Frankf., 1598, 767–768.

[2] "Wirk. d. Arznmitt. u. Gifte," i., 29.

[3] "Pandect. med.-leg.," i., 3, c. 28; Ed. Frankf., 1722, p. 141.

[4] The chemical examiner for Bengal reported thirty-six cases in the ten years 1860–69. According to Irving, aconite (*A. ferox*, called *bish*) is extensively used, as well as dhatura, arsenic, opium, hemp, and oleander by the professional poisoners of India. (See Med. Times and Gaz., 1882, i., 63.)

[5] *Linim. aconiti*, Br. Ph., 1874: 20 ℥ aconite root are extracted to produce a pint; while in the tincture 2½ ℥ are used for the same quantity. Not officinal in the U. S. Ph.

[6] Once a druggist used the 0.05 gm. weight in compounding a prescription containing aconitin in place of the 0.005 gm. piece, causing the death of the patient (Vidal: Thèse, Lyon, 1893). Two men died apparently from the effects of powdered aconite used in making up a prescription in mistake for ipecacuanha (Boutmy and Gallard, Biéchy: Thèse, Paris, 1881). Six persons were poisoned, three fatally, by "vin de quinquina," in whose preparation tincture of aconite was used in mistake for tincture of cinchona (Lhôte and Vibert: Ann. d'hyg., etc., 1892, 3 s., xxvii., 344, 385). Twelve persons were poisoned, three fatally, by the expressed juice of aconite root in mistake for cochlearia (Ballardini: Ann. univ. d. Med., Milano, 1840, iii., 635).

a decoction of it was taken in mistake for some other root, usually for horseradish, which is somewhat similar in appearance. The mistake is most frequently made in the winter and early spring, when the leaves are absent. The distinctive

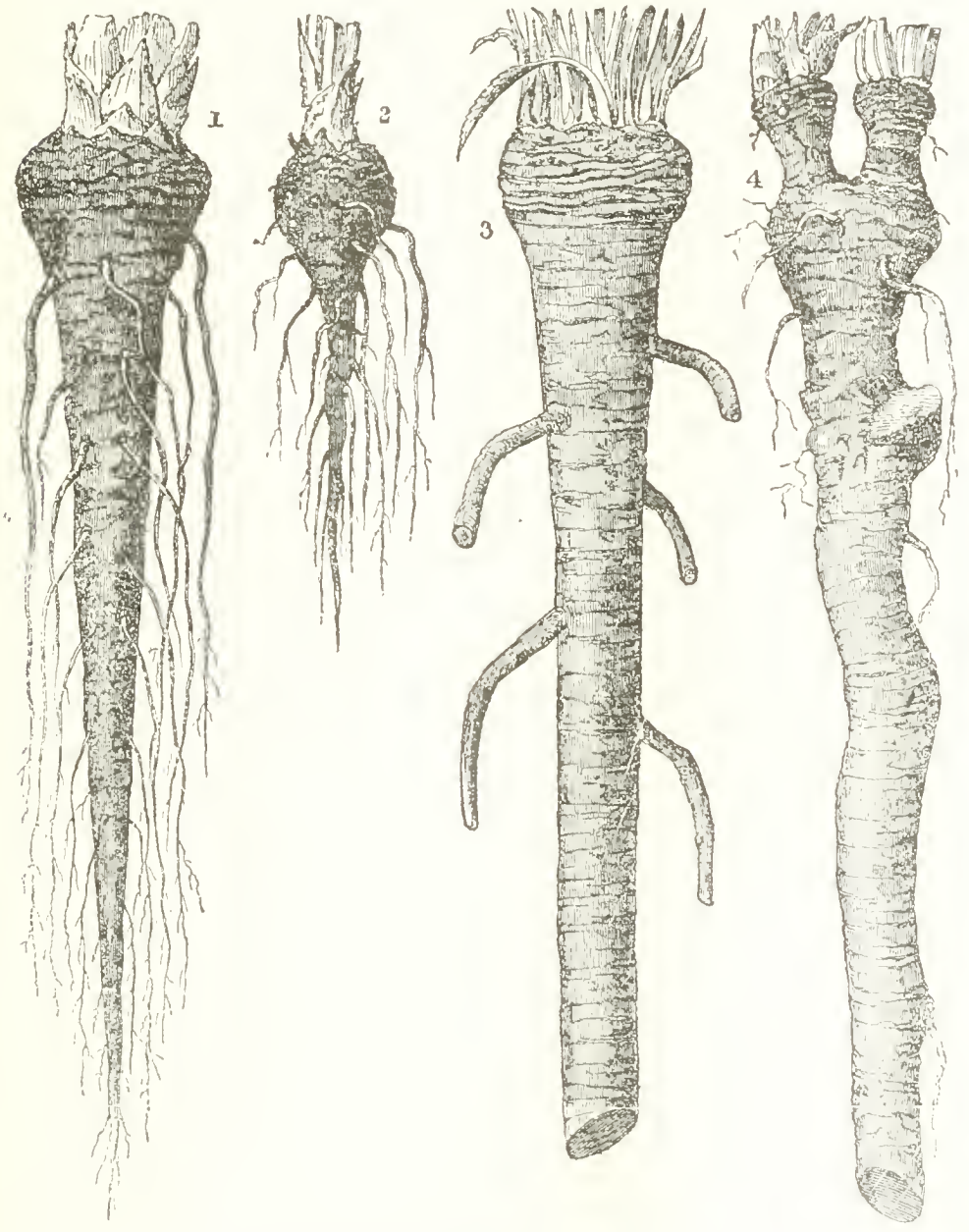

FIG. 30.—Roots of Monkshood and Horseradish. 1 and 2, Aconitum napellus; 3 and 4, Horseradish (*Cochlearia armoracia*).

characters of the roots of Aconitum napellus and horseradish are well described by Bentley,[1] from whose paper the accompanying

[1] Ph. J. and Tr., 1856. xv., 449.

ent is taken (Fig. 30). The leaves and flowers have also been taken by children, or accidentally used in salads, or given in jest. Six were non-fatal poisonings following medicinal administration. On one occasion 4 patients in a hospital were poisoned, and 2 died in consequence of the administration of a stronger preparation than had been previously used,[1] and in a recent case a woman died from the effects of a "nitrate of aconitine" of greater purity than samples previously used.[2] The variation in the purity of commercial "aconitines" was also the indirect cause of the death of Dr. Meyer, of Winschoten, who took a large dose of a prescription containing "nitrate of aconitine" which had caused severe symptoms in two patients, to prove its safety. He died in five hours.[3] Another instance of self-poisoning with the same object, but under different circumstances, was that of an herbalist in England, also in 1880, who took a quantity of his preparation and gave some to his servant, to prove its innocence. Both men died.[4] Toxic effects have also resulted from the use of the homœopathic tincture,[5] and from administration by "medical botanists"[6] and spiritualists.[7] The external application of the liniment,[8] or tincture,[9] or ointment containing aconitin,[10] or even contact of the root with the skin during hot weather,[11] or inhalation of its dust while powdering it,[12] have caused more or less serious poisoning.

According to Boutmy[13] a girl was destroyed by a mixture of aconitin and veratrin taken to provoke abortion.

Of the 26 SUICIDES by aconitic poisoning 18 were males, mostly physicians and druggists, and 8 females; 9 took the tincture, 4 took Fleming's tincture, 4 the root, 3 "aconitine,"

[1] Pereyra and Perrin: Gaz. d. hóp., 1839. 2 s., i., 145.

[2] Garaud: Loire méd., 1892. xi., 149. Ducher: *Ibid.*, 208. Brouardel, Crolas and Lépine: Arch. d. l'anth. crim., 1892. vii., 179.

[3] Busscher: Berl. kl. Wochenschr., 1880, xvii., 337, 356. Tresling: Schmidt's Jahrb., 1881, clxxxix., No. 2. Husemann: Pharm. Ztg., 1880, xxv., 445, 453, 459.

[4] Ph. J. and Tr., 1880-81, 3 s., xi., 423.

[5] Thompson: Cinc. Lanc. and Clin., 1874, xvii., 206; Ph. J. and Tr., 1873-74, 3 s., iv. 598.

[6] Ph. J. and Tr., 1873-74, 3 s., iv., 865.

[7] Channing: Boston M. and S. J., 1857, lvi., 453.

[8] Keene: Boston M. and S. J., 1872, lxxxvi., 74.

[9] Larison: Tr. M. Soc. N. J., 1874, 185. Belcher: Dublin Q. J. M. Sc., 1862, xxxiv., 476.

[10] Bassot: "Étude méd.-lég. sur l'aconitine," Lyon, 1889, p. 30.

[11] [Sherwill]: Med. Times and Gaz., 1882, i.. 63.

[12] Isaacs: N. Y. J. of M., 1859, 3 s., vii., 191.

[13] Ann. d'hyg., 1880, 3 s., iv., 207.

and 1 each the liniment, an aqueous extract, an infusion, and "neuraline."[1] Of the suicides by aconitin one was an early case in which the product taken must have been quite impure,[2] another was the case of an analytical chemist who took eight grains (0.5 gm.) of "German aconitine,"[3] and the third[4] that of a woman who destroyed herself with 0.00175 gm. (.027 grain) of pure aconitin. The sudden death of the Baron de Reinach in 1893 was supposed to have been caused by aconitin taken with suicidal intent.[5]

Homicidal aconitic poisonings are, apparently, of rare occurrence. Excluding the ancient case above referred to and certain imperfectly reported Indian poisonings, we find mention of but six cases. In one a woman in Ireland was convicted of the murder of her husband by mixing "blue rocket" with greens prepared for him.[6] In another a man was convicted of the murder of his wife by aconite in New York State in 1853.[7] The death of a woman in England in 1863 appears to have been due to aconite criminally administered, although the coroner's jury did not so find.[8] In 1867 a physician in England was accused of the murder of his wife by aconite, and committed suicide by hydrocyanic acid.[9] In 1882 an American physician, resident in England, was convicted of the murder of a relative by administration of "aconitine."[10] A number of patients in an insane asylum in Virginia were poisoned by "aconitine" introduced into their medicine from some source other than the asylum dispensary, in 1882. An insane woman in New Hampshire attempted to destroy her two children and did destroy herself with aconite in 1892.

[1] According to Harley, neuraline is a preparation of aconite mixed with chloroform and rose water. (See Lancet, 1875, ii., 454.)

[2] Bird: Lancet, 1848, i., 14.

[3] Springmühl: Med. Press and Circ., 1882, xxxiii., 439.

[4] Lacassagne, *ex* Vidal: Thèse, Paris, 1893, p. 64.

[5] See Brouardel, Richardière, Ogier, Schützenberger, and Villiers: Ann. d'hyg, 1893, 3 s., xxix., 161–198.

[6] Reg. *v.* McConkey, 1841. Geoghegan: Dublin J. M., 1841, xix., 403.

[7] Peo. *v.* Hendrickson: "Report of Trial," Barnes and Hevenor, Albany, 1853. Swinburne: M. and S. Reptr., Phila., 1862, vii., 361. See also Detection, below.

[8] Case of Eliz. Morris: Ph. J. and Tr., 1863–64, n. s., v., 379.

[9] *Ibid.*, 1866–67, n. s., viii., 93. Case of Dr. Warder.

[10] Reg. *v.* Lamson, Centr. Crim. Crt., "Sessions Paper," xcv., pt. 569, pp. 547–590. Browne and Stewart: "Trials for Murder by Poison," 514–567. Dupré: Westm. Hosp. Rep., i., 1885, 105; Lancet, 1882, i., 455. Stevenson: Guy's Hosp. Rep., 1883, 3 s., xxvi., 307.

Lethal Dose.—The lethal dose of pure, crystallized aconitin is very small. A woman of thirty-seven years was found dead after having taken 0.00175 gm. ($\frac{1}{38}$ grain) of Duquesnel's preparation.[1] But in Veil's case[2] a pharmacist took 0.012 gm. (nearly 1 grain) of crystallized aconitin and recovered.

In considering the lethal dose of the preparations of aconite regard must be had to the variations in strength in different pharmacopœias, and to the possibility that in the preparation of samples which have been taken in large doses without causing death some species of aconite other than *A. napellus* (the only one recognized in the United States and British pharmacopœias) may have been used. Of the liquid preparations of the root 100 c.c. contain the active principles of the following quantities (in grams) of the dry drug: Tincture, Br. Ph., 12.5; tincture, U. S. Ph., 40; tincture, Fleming's, 66.6; fluid extract, U. S. Ph., 100; liniment, Br. Ph., 100.[3] Moreover, the proportion of aconitin in the plant varies with the season at which it is collected.

Although the maximum medicinal dose is usually fixed at 0.1 gm. (gr. iss.) of aconite, the smallest quantity which we can find referred to as having caused death was one-third of a teaspoonful (= ℥xx. = 0.5 gm. aconite) of the U. S. Ph. tincture. This quantity caused the death of a sick man of thirty-four years in eighteen hours after having been given him in mistake for other medicine.[4] Roth[5] has reported the fatal poisoning of a man by 6 to 7 gm. of the Ph. Germanica tincture (ten per cent. aconite), corresponding to 0.6 to 0.7 gm. of aconite, and considers this as a dose more than sufficient to destroy life. One death

[1] Lacassagne: *Loc. cit.*

[2] France méd., 1893, xl., 610.

[3] It is also not certain that these proportions have been adhered to in the preparation of a given "tincture," etc. Thus in Topham's case (Lancet, 1851, ii., 56) the tincture was made with ℥iv. of aconite and one pound of spirit = 25.7 to 100 c.c.

[4] Waddell: Cinc. Lancet and Clin., 1875, xvii., 427. In the report of the case of Reg. *v.* Noakes (Ph. J. and Tr., 1865-66, n. s., vii., 243, 489, 527) it does not appear whether the ℥xxx. of tincture which caused the death of a man of eighty years, suffering from disease of the heart, in a few hours was the Br. Ph. tincture, which would have corresponded to 0.46 gm. aconite, or Fleming's tincture, =2.46 gms. aconite. Taylor: ("Poisons," 3d Am. ed., 722) relates that "an excise officer lost his life by merely tasting Fleming's tincture of aconite, under the supposition that it was flavored spirit." The quantity required for an excise officer's taste is, however, not fixed.

[5] Vrtljschr. f. ger. Med., 1883, n. F., xxxix., 76.

from three drachms of the Br. Ph. tincture (= 1.39 gm. aconite)[1] and two from two drachms of the U. S. Ph. tincture (= 2.95 gm. aconite)[2] are reported. Falck[3] gives the minimum lethal dose as 3.75 gm. of the tincture (Ph. Germ.?). But several cases have been observed in which persons have recovered from the effects of much larger doses. In two instances one ounce of the U. S. Ph. tincture (= 11.83 gm. aconite) failed to cause death;[4] in another four drachms of the fluid extract (= 14.79 gm. aconite),[5] in another one ounce of Fleming's tincture (= 19.69 gm. aconite),[6] in another one ounce of the liniment (= 29.57 gm. aconite),[7] and in still another two ounces of the liniment (= 59 gm. aconite)[8] were recovered from.

The poisoning of Dr. Male in 1845 would seem to indicate the possibility of a cumulative action. He was taking the tincture in five-drop doses for rheumatism, without effect. The day after having taken a dose of ten drops he was seized with the symptoms of aconite poisoning, and died the following evening. The total quantity taken during four days was eighty drops (= 0.3 aconite if the Br. Ph., or 1.64 gm. if Fleming's).[9]

Duration.—The usual duration of a fatal aconitic poisoning is from 1 to 5 hours, in whatever form it may be taken, except that in the two cases of fatal poisoning by the leaves death occurred in 20 and 24 hours.[10] The lethal action of the root is practically as rapid as that of the tincture: the average duration of sixteen cases of death from the tincture was 4 hours; that of nine instances in which the root was taken was 4½ hours. Nor is the average duration shorter with aconitin than with the crude drug, so far as can be learned from the small

[1] Seager: Brit. M. J., 1883, ii., 1067.

[2] Jones: Northwestern Lancet, 1884-85. iv., 83. Edson: Med. Rec., N. Y., 1890, xxxviii., 1890, 365.

[3] "Lehrb. d. prakt. Tox.," 1880, 234.

[4] Tuttle (Boston M. and S. Jour., 1891, cxxv., 678), M. ad., A. Cox (Phila. M. Times, 1872, iii., 74), F., 65, A. In Darby's case (M. and S. Reptr., Phila., 1859, i., 399) a liniment containing ℥ i. āā of laudanum, tincture of aconite, and olive oil was swallowed by a woman of forty-nine years, who recovered.

[5] Altenloh (N. Y. M. Jour., 1893, lvii., 358), F., 30, S.

[6] Bradley: Med. Rec., N. Y., 1887, xxxii., 155.

[7] Elliott (Lancet, 1878, ii., 917), F., 21, A.

[8] O'Connor (Dublin J. M. Sc., 1857, xxiii., 224), F., 70, A.

[9] Prov. M. and S. Jour., 1845, 535; Ph. J. and Tr., 1845–46, v., 140.

[10] Lancet, 1856, i., 715. Willis, quoted by Wibmer: "Wirkung," etc., i., 29.

number of fatal cases recorded: In Lacassagne's case[1] the woman was found dead 3 hours after having been seen well; in another case, noticed by Vidal[2] a man died in an hour and a quarter; in Garaud's case,[3] in which three doses were taken during one hour, death followed in 3 hours and 5 minutes after the first; Percy John died in 4½,[4] Dr. Meyer in 5 hours,[5] and Springmühl's patient in 12 hours.[6] The average duration of these six cases is 4 hours and 40 minutes. In the meagre account of the Staunton poisonings it is stated that "within 10 minutes several had died, two died after 2 hours, and another after 2 days."[7] There probably existed other causes of death in the case last referred to.

The most rapidly fatal case is that of a man of thirty-five years who accidently swallowed an unmentioned quantity of the liniment and died in less than 7 minutes.[8] A man died in half an hour after having taken an ounce of the Br. Ph. tincture.[9] In two cases the same tincture caused death in 1¾ hours,[10] and in one the liniment was fatal in an hour.[11] The root, eaten in mistake for horseradish, killed a man in 1 hour.[12]

Excluding the three cases above referred to, the most protracted case was that of a man who died in 18 hours after having taken ℥xx. of the U. S. Ph. tincture.[13] In Hatfield's case[14] a man of sixty-four years died in 13 hours after having eaten of the root.

In non-fatal cases the severe symptoms usually disappear in a few hours, although the numbness and tingling may persist for several days. Thus a girl suffering from hysterical coxalgia recovered from the effects of five granules of Duquesnel's aconitin, but the tingling persisted in both legs for several days, and in the one affected for 15 days.[15] Sometimes headache, anorexia, and depression of spirits remain for 2 or 3 days.

[1] *Loc. cit.*
[2] Thèse, Lyon, 1893, 59.
[3] Loire méd., 1892, xi., 149. Ducher: *Ibid.*, 208. Brouardel, Crolas, and Lépine: Arch. d. l'anthr. crim., 1892, vii., 179.
[4] Reg. *v.* Lamson: *Loc. cit.*
[5] Busscher: *Loc. cit.*
[6] *Loc. cit.*
[7] Ph. J. and Tr., 1883, xiii., 901.
[8] Bennet: Monthly J. M. Sc., 1852, xv., 69.
[9] Ph. J. and Tr., 1873–74, 3 s., iv., 218.
[10] Seager: *Loc. cit.* Taylor: Guy's Hosp. Rep., 1864, 3 s., x., 187.
[11] Ph. J. and Tr., 1878–79, 3 s., ix., 607.
[12] *Ibid*, 1853, xiii., 294.
[13] Waddell: *Loc. cit.*
[14] Med. Times and Gaz., 1857, xiv., 228.
[15] Arnozan: J. d. méd. d. Bordeaux, *ex* Vidal: Thèse, Lyon, 1893.

Symptoms.—The first effects of aconite or aconitin, when taken by the mouth, are usually manifested immediately, or within a few minutes. In some instances, however, the symptoms have been delayed. An alcoholic took an ounce of the U. S. Ph. tincture by mistake at 10:30; at 10:50 he felt sick, and vomited at 11:15 for the first time.[1] A woman took between 0.0025 and 0.005 gm. of "aconitin" for violent neuralgia, and only commenced to experience nausea and vertigo an hour later.[2] A physician took about 2 gm. of the "alcoolature" during three-quarters of an hour, and in two hours was attacked with faintness and vertigo.[3] A woman took three drachms of the U. S. Ph. tincture in mistake on going to bed, and awoke three and a half hours later with general distress and tingling.[4] In the single case of hypodermic administration which we find recorded, in which 0.002 gm. (.03 gr.) were injected in mistake for morphin, the patient immediately experienced a sensation of burning and prickling at the seat of the injection, and the usual tingling, etc., beginning at the lips, in a quarter of an hour.[5] In one non-fatal poisoning by external application of the tinctures of opium and aconite the symptoms began to appear in half an hour.[6]

A peculiar tingling or prickling sensation, or formication, is felt, first at the lips, then in the tongue and mouth, and is accompanied by a feeling of numbness, both sensations extending until the entire surface, particularly that of the extremities, is affected. There is a horrible strangling or clutching sensation in the throat, deglutition is difficult, and, later, attempts to swallow provoke spasms which may be violent and hydrophobic in character. The teeth feel as if loose, the throat feels dry, and there are sensations of swelling or enlargement of the face and forehead, headache, and twitching of the facial muscles, and sometimes lachrymation. There is a sense of sinking or pain, which in some instances is quite violent, in the epigastrium. Nausea is felt early, is accompanied by salivation, and is rapidly followed by the most violent and persistent retching and

[1] Tuttle: Boston M. and S. Jour., 1891, cxxv., 678.

[2] Chandelux, in Bassot: *Op. cit.*, p. 45.

[3] Morel-Lavallée: France méd., 1887, i., 753.

[4] Carlton: Boston M. and S. Jour., 1879, ci., 544.

[5] Franceschini: Thèse, Paris, 1875.

[6] Keene: Boston M. and S. Jour., 1872, lxxxvi., 74.

vomiting. The act of vomiting is a convulsive spasm of the abdominal muscles and diaphragm, by which the fluid is, as it were, "jerked out." After a very brief period of cardiac stimulation, the pulse soon becomes slower, more feeble, irregular, and dichrotic, then flickering and finally imperceptible. There is great weakness, and slight exertion provokes syncope, and in some instances cardiac pain. There is marked dyspnœa, and the respiration is usually shallow and hurried, or slow and irregular. Complete apparent cessation of the respiratory and cardiac actions have been observed in cases which have recovered under treatment.[1] The patient is greatly prostrated, weak, in fear of death, restless, with pale face, blue lips, and surface covered with cold perspiration. The extremities are cold, sometimes paralyzed, and sometimes affected with lancinating pains in the joints. There are chills and a subnormal temperature. The eyes are staring, glistening, and the pupils usually dilate, with more or less complete loss of sight or diplopia. In some instances there are hallucinations, sleep is interrupted by dreams, or the patient may become violently maniacal.[2] In some instances there has been copious purging, the stools being passed unconsciously and involuntarily.[3] Death is usually from syncope. In most instances the patient is perfectly conscious to the last; but in some there is unconsciousness;[4] or the patient may become comatose;[5] or convulsions, even of great violence,[6] with trismus[7] and other tetanic manifestations,[8] and cramps in the extremities[9] have been observed. The urine is retained in most cases, although in Garaud's case[10] micturition and de-

[1] Meldon: M. Pr. and Circ., 1878, xxv., 232. Richardson: M. Times and Gaz., 1869, ii., 709.

[2] Barnett: Med. Rec., N.Y., 1887, xxxii., 761. Valentine: N. Y. M. J., 1888, xlviii., 659. Jones: Northwestern Lancet, 1884–85, iv., 83.

[3] Jones: *Loc. cit.* Altenloh: N. Y. M. J., 1893, lvii., 358. Waddell: *Loc. cit.* Garaud: *Loc. cit.*

[4] Topham: Lancet, 1851, ii., 56. Brick: J. Am. M. Assoc., 1887, viii., 567. Tuttle: Boston M. and S. Jour., 1891, cxxv., 678. Robinson: *Ibid*, 1892, cxxvii., 192.

[5] Bradley: Med. Rec., N. Y., 1887, xxxii., 155. Valentine: N. Y. M. Jour., 1888, xlviii., 659. Springmühl: M. Press and Circ., 1882, n. s., xxxiii., 439.

[6] Wood: Austral. M. J., 1879, n. s., i., 283. Busscher: Berl. kl. Wochenschr., 1880, xvii., 337, 356. Springmühl: *Loc. cit.* Clark: Maryland M. J., 1882–83, ix., 423. Seager: Brit. M. Jour., 1883, ii., 1067. Bradley: *Loc. cit.*

[7] Busscher. *Loc. cit.* Veil: France méd., 1893, xl., 610. Altenloh: N. Y. M. J., 1893, lvii., 358. Baker: Am. Pract. and News, 1887, n. s., iv., 122.

[8] Springmühl: *Loc. cit.*

[9] Veil: *Loc. cit.* Franceschini: *Loc. cit.*

[10] *Loc. cit.*

fecation were abundant; and in Stewart's case[1] there was great incontinence of urine. A peculiar plaintive cry, not loud but prolonged, is sometimes uttered.[2]

Among other unusual manifestations the following have been observed: In Dr. Meyer's patients there was no vertigo and no tingling sensation, and Dr. Meyer himself, who took aconitin nitrate on a full stomach and died in five hours, did not vomit.[3] Altenloh's patient,[4] who took four drachms of the fluid extract and recovered, did not vomit at any time; and Morell-Lavallée's patient,[5] who also recovered from the effects of a large dose, neither vomited nor experienced nausea, but suffered a sensation of epigastric pressure (*barre epigastrique*). Although the respiration is usually shallow and hurried, Bradley,[6] Valentine,[6] McWhannell,[7] and Thach[8] have reported cases in which it was slow and stertorous. In the case of a man who died from the effects of the tincture, taken by mistake, the pulse was described as "rapid and bounding" three-quarters of an hour after the poison had been taken.[9] Although consciousness is usually perfect, in Topham's case[10] the patient was entirely unconscious when seen by the physician, only spoke intelligently twice, and after recovery had no remembrance of anything which had occurred for two hours previous to the time when she was seen. In two instances the patients became very sleepy, the condition amounting to profound stupor in a physician who had himself taken an overdose.[11] There are rarely intermissions in the symptoms, but in a woman who recovered from the effects of two ounces of the U. S. Ph. tincture, the respiration had stopped and all hope was given up, when suddenly the pulse could be felt and rapidly climbed up to 40; very soon the cheeks became flushed and the extremities warm, but in half an hour the pallor returned and the pulse began to fail. The improvement is attributed to the remedies applied.[12] A dilated condition of the pupils is not constant; instances are reported in which

[1] M. and S. Reptr., Phila., 1886, liv., 583.
[2] Topham: Lancet, 1851, ii., 56. Garaud: *Loc. cit.*
[3] Busscher: *Loc. cit.*
[4] *Loc. cit.*
[5] *Loc. cit.*
[6] *Loc. cit.*
[7] Brit. M. Jour., 1890, ii., 791.
[8] Nashville J. M. and S., 1891, lxx., 49.
[9] Eade: Lancet, 1882, i., 478.
[10] *Loc. cit.*
[11] Baker (T. H.): *Loc. cit.* Baker (F. G.): Brit. M. Jour., 1882, ii., 1039.
[12] O'Brien: Med. Rec., N. Y., 1879, xv., 128.

they were contracted.[1] In McWhannell's case[2] they only became dilated immediately before death. In Thach's case[2] they contracted and dilated alternately. In Dr. Meyer's patient,[3] and in a girl of sixteen years, they were at first contracted and afterward widely dilated,[4] in the latter case remaining so for twelve hours. In Isaacs' case of poisoning by inhalation of the dust aphonia was a prominent symptom, and persisted for two weeks.[5]

Treatment.—The stomach is to be washed out as soon as possible, preferably with water containing a solution of iodin in potassium iodid (*Liquor iodi compositus*), which forms a crystalline, insoluble compound with aconitin. In the absence of the necessary instruments an emetic of zinc sulfate or of apomorphin may be given, notwithstanding the tendency to emesis caused by aconite. Respiration is to be maintained artificially if necessary. Diffusible stimulants, ether, ammonia, brandy, etc., should be given by hypodermic or intravenous injection or by enemata. Digitalis, strychnin, and atropin have been given hypodermically to counteract the cardiac effects of aconitin, and amyl nitrite by inhalation to control the spasms. The diminution of temperature is opposed by warmth externally applied (hot bottles, hot baths).

Post-Mortem Appearances.—These are in no way characteristic. If the autopsy be made soon after death the pupils are found widely dilated.[6] The gastric mucous membrane is reddened, hyperæmic, and marked with spots or islands of more intense injection of the vessels. The duodenum and small intestine present similar appearances. There is also marked congestion of the liver, spleen, kidneys, lungs, and brain. The right ventricle contains much dark fluid blood. In the pericardium, peritoneum, and meninges are numerous punctiform or small extravasations of blood, which are also found in the cerebral substance. There are effusions in the pericardium and in the ventricles of the brain.

Detection.—In the exceptional cases in which portions of

[1] Topham: *Loc. cit.* Robinson: M. and S. Reptr., Phila., 1885, liii., 192. Carlton: Boston M. and S Jour., 1879, ci., 544.

[2] *Loc. cit.*

[3] Busscher: *Loc. cit.*

[4] Larison: Tr. M. Soc., N. J., 1874, 185.

[5] N. Y. J. of M., 1859, 3 s., vii., 191.

[6] In the Lamson case sixty-four hours after death.

the plant have been taken, these may be recognized in the stomach contents. In a systematic analysis aconitin, if present, will be extracted by benzene from the alkaline aqueous solution (see p. 136), and, under favorable conditions, is left as a crystalline residue.

Aconitin does not produce any color reaction, nor is there any special chemical test by which it can be identified. The color reactions described in older works are not produced by aconitin itself but by impurities which sometimes accompany it in the commercial samples. It forms precipitates, however, with several of the general reagents (see pp. 139 *et seq.*), notably with iodin in potassium iodid, with which .005 mgm. still forms a crystalline precipitate, and with potassium iodhydrargyrate, bromin water, phosphomolybdic acid, etc.[1]

The most sensitive reagents for aconitin are the human tongue and the mouse or frog. Minute quantities placed upon the tongue produce the peculiar tingling or prickling sensation; or cause the death of frogs or mice when injected hypodermically. A cardiographic tracing of the heart's action of a frog after poisoning by aconitin shows a distinct tetanization of the heart muscle.

In the present condition of our knowledge the most that the toxicologist may say is that the conditions observed are the same as would exist if aconitin were present. The lethal dose being very small, and this being disseminated through the body and in large part removed by vomiting and elimination, chemical tests usually fail. The physiological action, although distinct, has not been sufficiently studied with other substances to be considered as characteristic.[2]

THE ATROPA GROUP.

This group includes the *mydriatic alkaloids: Atropin*, *hyoscyamin*, and *hyoscin*, and the plants which contain them: *Atropa belladonna* (deadly nightshade); *Scopolia japonica*

[1] See Jürgens, Diss., St. Petersb., 1885.

[2] See further in this connection and concerning the toxicology of aconite in general: Stevenson and Dupré: Lamson case, *loc. cit.*; Wagner: Diss., Dorpat, 1887; Adelheim: Diss., Dorpat, 1869; Jürgens: Diss., St. Peterb., 1885; Ewers: Diss., Dorpat, 1873; Steckhan: Diss., Kiel, 1891; Vidal: Thèse, Lyon, 1893; Barsot: "Étude méd.-lég. s. l'Emp. p. l'Aconitine," Lyon, 1889; Laborde and Duques-

(Japanese belladonna), *S. carniolica*, *S. Hlardnackiana*, *Hyoscyamus niger* (henbane), *Datura stramonium* (thorn-apple), and *Duboisia Hopwoodii*, *D. myoporoïdes*, and *Mandragora officinale* (mandrake).

Atropin=*Tropin tropate*—$C_{17}H_{23}NO_3$ or $C_5H_8(CH_2.O.CO.CH.[C_6H_5]CH_2OH)NCH_3$—was discovered by Mein in 1831 and independently by Geiger and Hesse in 1833 in belladonna. The alkaloid described by the latter under the name *daturin*, and obtained from datura, is, when pure, identical with atropin. Commercial samples of "daturine" are often mixtures of atropin and hyoscyamin, and sometimes contain the latter alkaloid only. Atropin exists in atropa and in datura, but not in the other plants above mentioned.

Atropin crystallizes in prisms or silky needles, odorless, but having a disagreeable, bitter, and somewhat metallic taste, which is quite persistent. It dissolves in 300 parts of cold water and in 58 of boiling water, in 3 parts of chloroform, 30 of cold and 6 of boiling ethylic ether, and in 40 of benzene. It dissolves in almost any proportion in alcohol, and is also very soluble in amylic alcohol. It dissolves in glycerol, but very sparingly in petroleum ether or in carbon bisulfid. Benzene extracts it from alkaline solutions. Its solutions are alkaline and, contrary to the action of most alkaloids, redden phenolphthalein. It is readily decomposed by strong acids and by alkalies other than ammonia into *tropin*, $C_5H_8.CH_2.CHOH.NCH_3$, and *tropic acid*, $C_6H_5.CH[CH_2OH]COOH$. It may be obtained synthetically from the same substances, and is one of the class of *tropeins* formed by the union of tropin with acids.[1]

Natural atropin is either optically inactive or very faintly lævogyrous. Ladenburg and Hundt[2] have, however, produced two synthetic atropins: *dextro-atropin*, whose rotary power is $[a]=+10°.02$, and *lævo-atropin*, whose rotary power is $[a]=-9°.22$.[3]

Homatropin, $C_{16}H_{21}NO_3$ is a tropein obtained by heating together tropin and oxytoluic acid. It crystallizes in prisms which, although sparingly soluble in water, are hygroscopic.

nel: "Aconits et Aconitines," Paris, 1883; Cassariny: Thèse, Paris, 1891; Lhôte and Vibert: Ann. d'hyg., 1892, 3 s., xxvii., 344, 385.

[1] See Ladenburg: Ann. d. chem., 1883, ccxvii., 74–149.

[2] Berichte, Berl., 1889, 2590.

[3] For the reactions of atropin, etc., see below, Detection.

It dissolves readily in ether and in chloroform. It is somewhat less poisonous than atropin, and its bromid has been used in ophthalmic practice. Two cases of accidental poisoning from its use have been reported.[1]

Atropin is used medicinally as the free alkaloid and as the sulfate.

Apoatropin (*Atropamin ?*), $C_{17}H_{21}NO_2$, is a non-mydriatic base, derived from atropin by the action of nitric acid.

Hyoscyamin, $C_{17}H_{23}NO_3$, is isomeric with and closely related to, although not identical with, atropin. It crystallizes in needles (from benzene) or in plates (from chloroform) or is amorphous (from ethylic ether or amylic alcohol). It is difficultly soluble in cold water, more readily in hot water (particularly if impure); readily soluble in alcohol, ethylic ether, amylic alcohol, chloroform, and benzene. Its solutions are alkaline to phenolphthalein as well as to litmus. *Duboisin* is either a mixture of hyoscyamin and hyoscin, or the latter alone.

Hyoscyamin exists in all of the plants above mentioned and also in traces in *Lactuca sativa* (lettuce). It is readily converted into atropin by heat approaching its temperature of fusion (108° C.), or by heating with dilute alkalies. Consequently when, in the extraction of the alkaloids, heat is applied after the liberation of the bases by alkalies hyoscyamin, if present, is converted into atropin.

Hyoscin, $C_{17}H_{23}NO_3$ (*Sikeranin*, *Scopolamin*) is another isomere of atropin, which exists in belladonna, stramonium, and hyoscyamus. It is amorphous and semifluid, but forms crystalline salts, of which the bromid and chlorid have been used in medicine. It is difficultly soluble in water, but soluble in alcohol, ether, chloroform, and benzene, and is extracted from alkaline solutions by the last-named solvent.

Besides the above, the following bases have been obtained but imperfectly studied: *pseudo-hyoscyamin*, from Duboisia myoporoïdes; isomeric with atropin; *belladonin*, $C_{17}H_{21}NO_2$, from belladonna; *stramonin*, from stramonium; and *mandragorin*, isomeric with atropin, from mandrake.

[1] Gould: Med. News, Phila, 1893, lxii., 78. Hehir: Ind. M. Rec., Calcutta, 1894, vii., 42.

ATROPIC POISONING.

Statistics.—Accidental and usually non-fatal intoxications by the atropic poisons are of quite frequent occurrence; suicidal or homicidal poisonings by them are very rare. The following tabulation of cases which we have found reported in medical literature will indicate the relative frequency of poisonings by the members of this group:

	Cause uncertain[1]	Accidents.	Suicides.	Homicides.	Total.	Fatal.	Non-fatal.
Belladonna	62	164	6	5	247	24	155
Datura.....	72	105	3	6	186	21	90
Hyoscyamus	23	16	0	2	39	2	15
Atropin	25	170	19	5	219	11	184
Hyoscyamin	..	4	0	0	4	0	4
Hyoscin	..	8	1	0	9	0	9
"Duboisin"	..	29	0	0	29	0	29

The great majority of "accidental" poisonings by belladonna have resulted from taking the pharmacopœial preparations, the extract, tincture, fluid extract, or liniment internally, either in mistake for another medicine or by "error" on the part of the druggist. Many cases, several of which have proved fatal,[2] have resulted from eating the berries, the plant being frequently cultivated in gardens. In 1846 a fruit peddler in London, who was also an herbalist, and therefore considered by the magistrate as cognizant of the poisonous qualities of belladonna berries, was committed on a charge of "wilful murder" for having sold them as food and thus caused the poisoning of several persons, of whom two died.[3] Gaultier de Claubry[4] has left an account of the poisoning of a detachment of 150 soldiers by belladonna berries; and it seems probable that the poisoning of the army of Antony in Parthia, related by Plutarch, was caused by belladonna or datura.[5] In one instance a woman and her two

[1] Including cases reported in journals inaccessible to us.

[2] McNab: Brit. M. Jour., 1882, ii., 579; Lancet, 1846, ii., 251. Abel: Tr. M. Soc. N. Jersey, 1874, 182. Bauer: Med. Corr.-Bl. Würt. aerztl. Ver., 1873, xliii., 113. Rosenberger: Oesterr. med. Wochenschr., 1843, 565. Otto: Vrtljschr. f. ger. Med., 1866, n. F., v. 157; Ph. J. and Tr., 1858–59, xviii., 235, 243.

[3] Ph. J. and Tr., 1846, vi., 174.

[4] J. gén. d. méd., etc., 1813, xlviii., 355.

[5] Plutarch: "Vit. Ant." See also

daughters were seriously poisoned by a proprietary medicine containing belladonna.[1] This was a "*brustthee.*" Proprietary asthma cures usually contain stramonium and belladonna leaves. A poisoning by "Himrod's powder" (lobelia, stramonium, etc.) is reported by Thorpe,[2] and another by an "opium cure" by Philpot.[3]

The external application of belladonna plaster or ointment has in several instances been followed by serious poisoning, and the death of an infant was in all probability caused by such an application to the mother's breast.[4] A few cases, two of which were fatal,[5] resulted from the use of belladonna enemata or suppositories. Almost all the accidental stramonium poisonings have resulted from eating the seeds or other parts of the common Jimson, or Jamestown weed, either by children or in mistake for caraway seeds, greens, sage, or "dried herbs." Accidents from the alkaloids have been almost exclusively medicinal, chiefly non-fatal, poisonings arising in eye practice from the use of atropin or of "duboisin" as mydriatics. In not a few, solutions intended for such use have been swallowed in mistake for other medicine. In some twenty non-fatal cases toxic symptoms followed hypodermic injections of atropin.

Seeley[6] has recently reported an instance in which fifteen young ladies in a boarding-school exhibited the symptoms of atropic poisoning after eating turkey, and suggests as a cause that the bird had probably fed upon mydriatics; in the absence of proof of such being actually the cause it is quite as probable that these poisonings were caused by ptomatropins. A similar origin of atropic poisoning, which terminated fatally, after indulgence in rabbit pie was claimed by the defence in a criminal trial.[7]

Only one of the five attempts at suicide by belladonna was successful, that of a dissipated young man of twenty five years, who destroyed himself with "pommade belladonnée."[8] Of the 19

Imbert-Gourbeyre: "Recherches s. l. solanum d. anciens," Paris, 1884, 117.

[1] Friedreich's Bl. f. ger. Med., 1887, xxxviii., 130.

[2] Lancet, 1888, i., 973.

[3] Tr. S. Carolina M. Assoc., 1882, xxxii., 127.

[4] Goodwin: M. and S. Reptr., Phila., 1871, xxiv., 346.

[5] Mavel: Gaz. d. hôp., 1854, xxvii., 399. Scharf: Wochenschr. f. d. ges. Heilk., 1845, 101.

[6] Med. Rec., N. Y., 1894, xlv., 14.

[7] Reg. *v.* Sprague, 1865; Ph. J. and Tr., 1865, n. s., vii., 72.

[8] Diday: Lyon méd., 1869, ii., 547.

persons who attempted suicide by atropin 11 were men and 6 women, while one case was a double suicide. All but 3 recovered: a man of sixty years who died in 15 hours after having taken six grains (0.39 gm.) of the sulfate;[1] another man who died in 12 hours from a dose supposed to have been gr. ij.;[2] and another man of seventy-four years who died in 20 hours.[3] A woman to whom hyoscin was administered medicinally, intentionally took the whole quantity of the solution. She recovered, and afterward destroyed herself by suspension.[4] Of the 3 suicides who took datura 2 were Hindus, one of whom died in about 7½ hours.

Of the 5 homicidal poisonings by belladonna, 2 occurred in Germany,[5] and 2 in the United States.[6] In one of the German cases, a woman was accused of an attempt to murder her husband, who had exhibited the symptoms of atropic poisoning. Although belladonna seeds were found in the soup and vomit, the woman was acquitted, there being strong ground for the belief that the man had poisoned himself in order to accuse the defendant. In the other a woman removed her husband by repeated administrations of pounded belladonna root mixed with his food. The more recent of the American cases was an unsuccessful attempt in which belladonna leaves were mixed with coffee. In the earlier one, also an unsuccessful attempt, four distinct acts of administration were alleged, and, after two mistrials, the defendant was convicted.

Of the homicidal poisonings by stramonium, one was an attempt to murder a priest in Dantzig, by an unknown person who sent him a bottle of poisoned wine.[7] A servant in Germany confessed that she had been hired by her mistress to mix seeds of stramonium and hyoscyamus with the food of the latter's husband, who died in 14½ hours, having manifested the usual symptoms, and in whose stomach and intestines the seeds were found. Both women were convicted.[8] An Austrian

[1] Pollak: Wien. med. Presse, 1870, xi., 565.

[2] Taylor: "Poisons," 3d Am. ed., 732.

[3] Kanders: Wien. med. Wochenschr., 1881, 1253.

[4] Githens: Therap. Gaz., Detroit, 1887, 3 s., iii., 811.

[5] Buchner: Friedreich's Bl. f. ger. Med., 1887, xxxviii., 134, 136.

[6] Nichols: Phila. M. Times, 1882–83, xiii., 859. Peo. *v.* Northrup, Westchester Co., N. Y., 1868. Lee: Quart. J. Psych. Med., 1868, ii., 28. The fifth is the case of the peddler above mentioned.

[7] J. de chim. méd., etc., 1847, 3 s., iii., 201.

[8] Vrtljschr. f. ger. Med., 1858, xiv., 139.

woman was convicted in 1870 of an attempt to murder her husband by administration of a decoction of stramonium leaves in coffee.[1] A young man in Mississippi died from the effects of datura, administered, it was alleged, by a negro and negress. It is said that the poisonous quality of stramonium is well known to the Southern negroes.[2] The fifth case may be taken as the type of poisonings for the purpose of robbery, which appear to be of frequent occurrence in India.[3] A Hindu and his wife set up a feast at which they dosed their seven guests with datura and robbed them when they had become insensible.[4]

Both of the two hyoscyamus poisonings were German. In 1850 a man died from the effects of henbane administered by his wife and son-in-law. The seeds were found in the cadaver, exhumed $2\frac{3}{4}$ years after death.[5] In 1859 another woman was tried for husband murder by henbane, and acquitted.[6]

Besides the case of Reg. *v.* Sprague, above referred to, one other homicide by atropin is reported in England: Reg. *v.* Steele, 1872, in which a hospital nurse was acquitted upon a trial for the murder of the senior surgeon by atropin, given in milk, from lack of evidence connecting her with the administration.[7] A sixteen-year-old boy was also acquitted, for the same reason, in Germany in 1890 of an attempt to poison his parents, and grandmother, and a servant.[8] Marie Jeanneret was a nurse in an establishment at Lausanne, who, in the years 1867 to 1869, poisoned nine persons, of whom five, a man and four women, died. The agent which she used was the solution of atropin used as a collyrium, although she also tried morphin

[1] Hashek: Wien. med. Presse, 1870, xi., 859, 883, 912, 932, 987, 1007, 1063. The defence appears to have claimed that the poison was given in jest and not with intent to kill.

[2] Mallet: N. Orl. J. M., 1868, xxi., 550.

[3] Matthiolus and others relate instances in which belladonna root was administered by practical jokers, and to facilitate robbery, etc., in Europe in the fourteenth century. See Gmelin: "Geschichte d. Pflanzengifte," Nürnb., 1803, 527, *note*.

[4] Tyrrell, S. M.: Madras Q. J. M. Sc., 1867, xxi., 167. See also Banerjee: Ind. M. Gaz., Calcutta, 1885, xx., 209; Irving: Ind. Annals M. Sc., 1864, xvii.; Med. Times and Gaz., 1882, i., 63; Cheever's "Med. Jur. for India," Calc., 1870; Gribble and Hehir: "Med. Jur. for India," Madras, 1892.

[5] Gossow: Vrtljschr. f. ger. Med., 1856, x., 216.

[6] Möller: *Ibid.*, 1860, xviii., 78.

[7] Taylor: "Poisons," 3d Am. ed., 733; Ph. J. and Tr., 1871–72, 3 s., ii., 596, 617, 636. Calvert: Med. Times and Gaz., 1872, i., 598.

[8] Rauscher: Friedreich's Bl. f. ger. Med., 1891, xlii., 400.

and antimony.[1] Bentzen reported an attempt on the part of a woman's lover to poison her and her two-year-old child, she being at the time pregnant, by the administration of an atropin solution which he had received from an ophthalmic surgeon. The dose administered was considered by the medical faculty of Christiania as being sufficient to cause serious poisoning but hardly death.[2] Schauenstein[3] refers to the Fall Simére, in Vienna in 1878, in which atropin was given to a maidservant in a liqueur to stupefy her in order to facilitate robbery.

In two American cases it was supposed that the medical poisoners had administered atropin and afterward morphin with the object of preventing the contraction of the pupils and otherwise masking the symptoms of morphin poisoning.[4]

Duration.—Fatal cases of atropic poisoning usually terminate within 24 hours. The average duration of 14 poisonings by *belladonna* was 14 hours, leaving out of the calculation 2 cases, one that of a man who is said to have survived 7 days,[5] the other that of a suicide who died in "3 or 4 days,"[6] the reports of which are not sufficiently full to exclude the possibility of other causes of death. Death has occurred in $3\frac{1}{2}$,[7] 6,[8] and 7[9] hours in the most rapidly fatal cases, and in 17 (2 cases),[10] 18,[11] 21,[12] and 29[13] hours in the most prolonged.

The average duration of 12 fatal poisonings by datura was 13 hours. The extremes were 7 to $7\frac{1}{2}$ hours[14] as the shortest and 24 hours[15] as the longest.

[1] "Procès criminel contre Marie Jeanneret, huit empoisonnements," Lausanne, 1869. (The ninth was that of a young girl to whom she gave atropin in candy.) Chatelain: J. d. méd. ment., Paris, 1869, ix., 113–120.

[2] Norsk. Mag. f. Läg., 1885, xv., 497, *ex* Schmidt's Jahrb., 1885, ccviii., 131.

[3] Maschka: "Handb. d. ger. Med.," ii., 651.

[4] Ruth-Medlicott case, Saunders: Mich. Univ. M. J., 1872–73, 641. Peo. *v.* Buchanan, New York, 1894.

[5] Bayard and Chevallier: Ann. d'hyg., 1847, xxxviii., 413.

[6] Diday: Lyon méd., 1869, ii., 547.

[7] Molyneux (Lancet, 1869, ii., 327), M., 16, A., ℥ ij. extract; also Taylor: Brit. M. J., 1869, ii., 555.

[8] Tanner (Brit. M. J., 1886, i., 589), F., 16, A. (?), liniment.

[9] Hudson (Austral. M. J., 1861, vi., 54), M., 47, A., ℥ ss. liniment.

[10] Jackson (Lancet, ii., 1848, 318), M., 75, A., gr. iv.–v. (?) extract. MacNab (Brit. M. J., 1882, ii., 579), M., 5, A., berries.

[11] Mitchell (Canada Pract., 1883, viii., 225), M., 53, A., herb.

[12] Otto: Vrltjschr. f. ger. Med., 1866, n. F., 157.

[13] Ph. J., 1846, vi., 174.

[14] Droste (Ztschr. f. d. Staatsarznk., 1837, xxxiii., 129), F., 63, A., decoction of seeds. Allan (Lancet, ii., 1847, 298), M. (Hindu), 45, S., plant.

[15] Duffin (London M. Gaz., 1834–35, xv., 194), F., $2\frac{1}{4}$, A., about 100 seeds.

So far as can be judged from the small number of fatal cases in which the duration has been reported, atropin differs from other alkaloids in being slower in its action than the crude drugs from which it is obtained. The fact is, however, more probably apparent than real. The average duration of 8 cases was 18.7 hours; the extremes being 10½ [1] and 41 [2] hours. Greenway has reported the case of a man of forty-five years who died in 3½ days from the effects of half a grain of the sulfate.[3]

Lethal Dose.—The smallest quantity of *atropin* which has been known to cause death was 0.0004 gm. (about gr. .006). This quantity, dropped into the eye of a child of four years during four days, caused its death, after characteristic symptoms. The child had previously manifested great sensitiveness to atropin.[4] Jaenicke [5] has reported the death of a woman of fifty-seven years from the effects of from 0.005 to 0.007 gm. (.006 to 0.1 gr.) of the sulfate taken in solution during the night. In Dunlap's case, above cited, the dose was 0.004 gm. (1/16 gr.), and in Greenway's case[6] it was 0.065 gm. (1 gr.). But very much larger doses have been recovered from. Travers [7] has reported the recovery of a woman who had swallowed 0.226 gm. (3½ gr.) in solution; Bucquoy [8] that of two suicides who took 0.25 gm. (3.9 gr.) each; Kernig[9] that of a girl of fourteen years who took about 0.26 gm. (4 gr.) in mistake for quinin; Borden[10] that of a man who attempted suicide by swallowing an injection, prescribed for gonorrhœa, containing one drachm of zinc sulfate and 0.324 gm. (5 gr.) of atropin sulfate; and Macchiavelli[11] that of a man who attempted suicide with the enormous dose of 0.5 gm. (7.2 gr.) of the sulfate. Instances of recovery after having taken 0.065 gm. (1 gr.) have been quite numerous. The amounts of the lethal and maximum medicinal doses constituted an important factor in a prosecution for

[1] Boston M. and S. Jour., 1869, lxxix., 148; M., 3, A., 1½ gr.
[2] Dunlap (Am. Pract. and News, Louisv., 1887, n. s., iii., 230), F., 1/16 gr.
[3] Brit. M. Jour., 1878, ii., 515.
[4] Burrenich: Ann. et bull., Soc. méd. d. Gand., 1891, 288.
[5] Deut. Arch. f. klin. Med., 1877, xx., 617.
[6] *Loc. cit.*
[7] Brit. M. J., 1889, i., 1051.
[8] Gaz. d. hôp., 1878, li., 43.
[9] St. Pet. med. Wochenschr., 1883, viii., 238. See also Mackenzie: Cincin. Lancet and Obs., 1878, xxi., 148.
[10] Alabama M. and S. Age, vi., 1893-94, 413.
[11] Gazz. med. it. lomb., 1880, 8 s., ii., 339.

homicide by negligence, reported by Pouchet,[4] in which a man died from the effects of 0.045 gm. (0.7 gr.).

We find but four fatal poisonings by *belladonna* in which the doses have been accurately reported. These were 3.7 c.c. (fl. ʒ i.)[5] and 14.8 c.c. (fl. ʒ ss.)[6] of the British Pharmacopœia liniment; 7.77 gm. (ʒ ij.) of the British Pharmacopœia extract,[4] and 7.65 gm. of the crude drug.[5] Persons have recovered after having taken 15.6 gm. (ʒ ss.) of the extract,[6] 14.8 c.c. (fl. ʒ ss.) of the tincture,[7] and 19.6 c.c. (fl. ʒ i.) of the liniment.[8]

No sufficient statistics are available to serve as a basis for even an approximate estimate of the lethal dose of stramonium, beyond the statements that a man of twenty-seven years recovered from the effects of 71 c.c. (half a gill) of the tincture,[9] and a child of two and a quarter years died after having swallowed about sixteen grains (1.04 gm.) of the seeds.[10]

The lethal doses of hyoscyamus, hyoscyamin, and hyoscin are undetermined.

Symptoms.—The symptoms produced by the individual members of this group of poisons resemble each other closely. The action takes place in two stages: first a period of delirium, and second a period of coma, the former merging into the latter, while the patient, still delirious, becomes comatose, but may be aroused. There is little delay in the appearance of the symptoms, which may become menacing in a few moments, particularly when atropin or belladonna has been taken. In a few instances the patient has gone to sleep shortly after having taken the poison, and has awakened in from one to several hours afterwards in delirium,[11] or has been found comatose.[12] In some

[1] Ann. d'hyg., etc., 1889, 3 s., xxi., 139.

[2] Beddoe (Lancet, 1870, ii., 83), F., 66, A.

[3] Hudson (Austral. M. J., 1861, vi., 54), M., 47, A.

[4] Molyneux: Taylor, *loc. cit.*

[5] Bull. gén. d. thérap., 1832, i., 102. In this case the pharmacist dispensed 2 "gros" in mistake for 2 "grains" (0.106 gm.).

[6] Castaldi (Gaz. méd. d. l'Orient, Constant., 1860, iv., 79, 102, 117), child, A. (in liniment). Duffin (Brit. M. J., 1881, i., 639), M., 2½, A. (in liniment). Oliver (Lancet, 1891, ii., 929), M., 43, A.

[7] Girdlestone (Austral. M. J., 1870, xv., 204), F., ad., S.

[8] Williams (*ibid.*, 1879, n. s., i., 590), M., 48, S. A(?). Wood (Brit. M. Jour., 1885, i., 377), F. ad., A.

[9] Williams: N. England J. M. and S., 1823, xii., 253.

[10] Duffin: London M. Gaz., 1834–35, xv., 194.

[11] Huber: Ztchr. f. kl. Med., 1888, xiv., 511. Terry: Boston M. and S. Jour., 1882, cvi., 123.

[12] Diday: Lyon méd., 1869, ii., 547. Macchiavelli: Gazz. med. it. lomb., 1880, 8 s., ii., 339.

instances also, where large doses have been taken, the sequence of the stages is reversed; the patient becomes unconscious and comatose in a few moments, and may either die in convulsions in eight or ten hours,[1] or only become delirious when aroused from the coma later.[2]

Usually the patient becomes giddy and "feels queer," yawns, staggers, and loses the power of standing. The mouth and throat are dry and parched, and sometimes a bitter taste is observed, and a burning sensation is experienced. The secretion of saliva is completely arrested, the voice becomes husky or extinct, there is a sense of constriction of the throat and one of impending suffocation, and deglutition becomes difficult or impossible. Although there is thirst, attempts to swallow provoke spasms of the throat and pharynx, which may be hydrophobic in intensity. The face may be pallid at first,[3] but soon becomes red or even purple, with the veins of the forehead greatly distended. The color of the face resembles that of scarlatina, and a scarlatinal rash, accompanied by itching, makes its appearance upon the skin of the body and extremities as well. The pulse is at first full, hard, bounding, and greatly increased in frequency, up to 190–200 to the minute,[4] afterward it becomes irregular, intermittent, fainter, and even imperceptible at the radial. The skin is dry and hot, although at a later stage the extremities become cold and, in two cases reported by Carroll,[5] the entire surface was cool one hour after the poisoning. The eyes are somewhat prominent, brilliant, wild, and staring straight forward, or restless, but exceptionally there is either converging[6] or diverging[7] strabismus in the later stages. There are disturbances of vision, diplopia, amblyopia, chromatopsia, or total blindness. The pupils are widely dilated, the iris is a mere ring a millimetre in width, the retina is insensible to light, the accommodation is completely paralyzed, and the conjunc-

[1] Mitchell (Canada Pract., 1883, viii., 225), F., 26, A., D., eight hours, C—; Boston M. and S. Jour., 1869, lxxix., 148, M., 3, A., D., ten hours thirty-five minutes.

[2] Ringer (Lancet, 1876, i., 346), F., 4. McLain: Va. M. Monthly, 1891–92, xviii., 832.

[3] Loomis: Med. Rec., N. Y., 1885, xxvii., 235. Kjellberg: Arch. f. Kinderhlk., 1881–82, iii., 435.

[4] Link: Memorab., Heilbronn, 1883, n. F., iii., 327. Hedler: Berl. kl. Wochenschr., 1875, xii., 471.

[5] M. and S. Reptr., Phila., 1882, xlvii., 76.

[6] Smith: Med. Times and Gaz., 1883, ii., 373.

[7] Mitchell: *Loc. cit.*

tivæ are injected. A few cases have, however, been reported in which the pupils were said to have been of normal diameter[1] or even contracted,[2] and one in which the contraction was unsymmetrical.[3] Attacks of giddiness and vertigo and severe headache are soon followed by delirium, which may, exceptionally, be silent or muttering, but is usually violent, noisy, and in some cases as active as and resembling that of delirium tremens. The efforts of several persons are required to restrain the patient, who, if left to himself, will throw about all articles within reach, and has been known to jump out of a window. There are the most fantastic hallucinations and delusions, the patient clutches at imaginary objects, sees snakes, animals, etc., has outbursts of laughter and weeping, while the expression of the countenance alternates between that of the most abject terror to that of extreme hilarity. He chatters incessantly, talks utter nonsense, mistakes one person for another, mistakes the names of objects, and says one thing when he intends to say another. According to Banerjee[4] the alteration of voice and the talk of those under the influence of the Indian dhatura are peculiar and pathognomonic; when recovering the patient answers questions in an undertone, shortly and quickly, and all of a sudden he appears to lose the thread of thought, looks in another direction, and wanders away as if thinking of something else. In a small proportion of cases there are convulsions of a tetanic character, most frequently of the upper part of the body, or limited to trismus. The delirium gradually subsides, or during its continuance the patient becomes somnolent and passes into a state of profound coma, with slow, stertorous respiration, and failing and finally imperceptible pulse, and death occurs from respiratory or cardiac paralysis. Or death may occur, during apparent amelioration, by syncope.[5] In the great majority of cases there is retention of urine, but occasional instances have occurred in which there has been involuntary,[6]

[1] Wood: Brit. M. J., 1885, i., 377. Nisbet: Austral. M. Gaz., 1889–90. ix., 114. McCartney: Phila. M. News, 1892, lxi., 306.

[2] Blake: St. Geo. Hosp. Rep., 1878. iii., 159 (Tr. stramonium).

[3] Golding: Lancet, 1859, ii., 560.

[4] Ind. M. Gaz., Calcutta, 1885, xx., 209.

[5] Dunlap: Am. Pract. and News, Louisv., 1887, n. s., iii., 230.

[6] Wood: *Loc. cit.* Smith: *Loc. cit.* Wilson: Lancet, 1878, i., 165. Greenway: Brit. M. J., 1878, ii., 515. Murrell: M. and S. Reptr., Phila., 1876, xxxv., 269. Pollak: Wien. med. Presse, 1870, i., 565.

frequent,[1] or free[2] micturition. In one case there was polyuria and albuminuria, although the urine examined the day before had been found to be normal.[3] Usually no pain is complained of except that due to the dryness of the mouth and throat, but in some instances there has been epigastric pain, and more rarely pain in the region of the kidneys. Nausea is most rarely experienced, and spontaneous vomiting is equally exceptional.[4]

The statement of Boehm,[5] Schauenstien,[6] Kobert[7] and others, following Schroff,[8] that there is always a constant diminution in the body temperature in atropin poisoning, is not in accordance with the recorded observations in the human subject. It must, however, be remembered, that owing to the character of the effects of atropin, buccal temperatures cannot be taken, and consequently the observations are of axillary temperature, and that possibly a reverse of the usual condition may exist, the surface temperature being higher than the internal. But we know of no clinical evidence that such is the case. In all of the nineteen reports of poisoning by atropin or belladonna in which we find the temperature recorded it was, during the severity of the poisoning, above the normal of 98°.4 (axillary). The lowest temperature observed was in a case reported by Sauberzweig[9] in which, after a notable elevation, there was a fall below the normal on the second day, which increased until the fifth day, when the patient was discharged cured. The readings in this case were: 102°.3, 103°1., 104°.4, 102°.5, 102°.5, 98°.2, 96°.6, 96°.4, 95°.4, 96°.8, 95°.4, 91°.4. In but one other case did the temperature fall below the normal at any time after the establishment of the poisoning; in Macchiavelli's case[10] during two days the observations were 98.9°, 98°.6, 96°.8, 100°.4. In all the others it was supernormal, and in one patient, who died in six hours, it was 105°.6, 107°.4, and, just before death, 108°.6,[11] while in another fatal case a temperature of 110° was observed.[12] In a poisoning by tincture of

[1] Lancet, 1885, i., 1047.
[2] Bond: Brit. M. J., 1881, i., 639.
[3] Mills: Canada M. and S. J., 1880–81, ix., 19.
[4] Mitchell: *Loc. cit.* Huber: Zeitschr f. kl. Med., 1888, xiv., 511. Fraser: Lancet, 1865, ii., 536.
[5] Ziemssen: "Handb.," 2te Aufl., 1880, xv., 363.
[6] Maschka: "Handb. d. ger. Med.," 1882, ii., 655.
[7] "Intoxikationen," 1893, 609.
[8] Zeitschr. Wien. Aerzte, 1852, 211.
[9] Diss., Halle, 1892, p. 9.
[10] *Loc. cit.*
[11] Tanner: Brit. M. Jour., 1886, i., 589.
[12] MacNab: *Ibid.*, 1882, ii., 579.

hyoscyamus the thermometer indicated 104°,[1] and in datura poisoning, where the temperature has been taken, it has been found to be as high as 104°,[2] and 108°–109°.[3] Kratter,[4] in his excellent discussion of atropic poisoning, gives six cases in which the temperature was supernormal.[5]

In non-fatal cases, although the severe symptoms disappear in a few hours, or, in severe cases, within forty-eight hours, disturbances of vision and incoherency have been known to continue for weeks or months.[6] In one of Ringer's cases[7] the patient was delirious for four days, and there was retention of urine for several days.

After recovery the patient has usually no remembrance of anything that has transpired while he was under the influence of the poison, although he may recall certain of his delusions, which he still considers to have been realities, or may remember that while endeavoring to make certain remarks he was irresistibly driven to say something else. This remembrance has been particularly noted in hyoscyamin and hyoscin poisoning.

Treatment.—The stomach should be washed out and, unless the case is seen quite early, the intestine also. Mechanical removal is particularly preferable in atropic poisoning, as even powerful emetics given in large doses frequently fail to act. During the period of delirium morphin may be given hypodermically in small and repeated doses; tendency to coma may be combated by the ambulatory treatment (see Morphin, Treatment), flagellation and stimulation, and during coma injections of ether or brandy, inhalation of amyl nitrite, and artificial res-

[1] White: Lancet, 1873, ii., 8.

[2] Newton: Med. Rec., N. Y., 1880, xviii., 289.

[3] De Lantour: N. Zeal. M. Jour., 1893, vi., 84.

[4] Vrtljschr. f. ger. Med., 1886, xliv., 52.

[5] The other reports referred to are Huber: *Loc. cit.* (98°.6–102°.2). Davy: Lancet, 1882, i., 345 (101°.4). Cooper: Brit. M. J., 1877, i., 164 (98°.5). Nisbet: Austral. M. Gaz., 1889–90, ix., 114 (98°.6, 102°). Oliver: Lancet, 1889, ii., 1003 (101°, 103°, 102°, 101°, 99°.8). Greenway: Brit. M. J., 1878, ii., 515 (98°, 99°, 102°, 102°.4, 103°, 102°.6, 100°.4, 102°.2, 102°.4, 100°.6, 100°.2). Thompson: Austral. M. Gaz., 1890–91, x., 298 (Case II., 99°.8; Case IV., 100°.6; Case VI., 99°.6; Case I., 108°). Cantlie: M. Pr. and Circ., London, 1890, n. s., ii., 88 (99°.3, 99°.7, 100°, 100°.2, 99°.4). Westcott: Chicago M. J. and Exam., 1888, lvi., 138 (101°). Link: *Loc. cit.* (101°.1, 100°.8). Reisz: Wien. med. Wochenschr., 1881, xxxi., 97 (99°.7). Siegmund: Arch. f. path. Anat., etc., 1869, xlviii., 1888 (102°, 99°.5).

[6] Mitchell: *Loc. cit.* Cotter: Med. Times and Gaz., 1870, i., 564.

[7] Lancet, 1876, i., 346.

piration may be resorted to. Hypodermics of pilocarpin, while they do not counteract the poisonous action of atropin, may serve to establish the arrested salivary secretion and relieve a troublesome symptom. Caution should be exercised that excessive use of pilocarpin and, particularly, of morphin does not increase the tendency to coma.[1]

Post-Mortem Appearances.—The number of autopsies after death from atropic poisoning is small, and the lesions observed are not characteristic. In the case of a man who died in three and a half hours after having taken two drachms of the extract of belladonna[2] the pupils were found dilated eighteen hours after death, as they were also in Greenway's case, in which the autopsy was made sixty-four and a quarter hours after death. The lungs are highly congested or œdematous, and the pleuræ contain notable quantities of bloody serum.[3] The left heart has been found empty, and with more or less coagulated or dark fluid blood in the right side. In all cases in which the brain was examined it and the meninges have been found greatly congested, with bloody serum in the ventricles, and the brain substance studded with punctiform blood spots on section. The trachea and œsophagus have been found strongly hyperæmic after death from atropin,[4] and the mucous membrane of the stomach and upper small intestine congested, or even marked with scattered ecchymoses, after death from stramonium.[5] When death has been caused by belladonna berries the bucca, œsophageal, gastric, and intestinal mucous membrane may be colored dark blue by their juice.

Detection.—If the seeds of datura or of hyoscyamus have been taken they may be found in the stomach, intestines, vomit, or stomach washings and fæces. The datura seeds are hard, flattened, kidney-shaped, dull black or brownish-black in color, about 3 mm. long and 2.5 mm. wide, and marked externally with a network enclosing punctiform depressions (Fig. 31, *B*). They have a somewhat sharp, bitter taste. The seeds of hyoscyamus are smaller than those of stramonium, about a milli-

[1] See Husemann, in Penzoldt and Stintzing: "Handb. d. spec. Therap.," 1895, ii., 258..

[2] Molyneux: Taylor, *loc. cit.*

[3] Molyneux: *Loc. cit.* Beddoe: Lancet, 1870, ii., 83. Hudson: Austral. M. Jour., 1861, v., 54. Newton: Med. Rec., N. Y., 1880, xviii., 289.

[4] Jarnicke: Deut. Arch. f. kl. Med., 1877, xx., 617.

[5] Allan: Lancet, 1847, ii., 298.

meter long, almost kidney-shaped, flattened, roughened, gray or grayish-brown externally, white within, odorless, and having a sharp, bitter taste (Fig. 31, *C*). The seeds of belladonna, which will usually be found in the stomach when the berries have been eaten, are about 3 mm. long, kidney-shaped, brown or gray in color, roughened exteriorly, but not so markedly as those of hyoscyamus, which they also resemble in the shape of the embryo (Fig. 31, *A*).

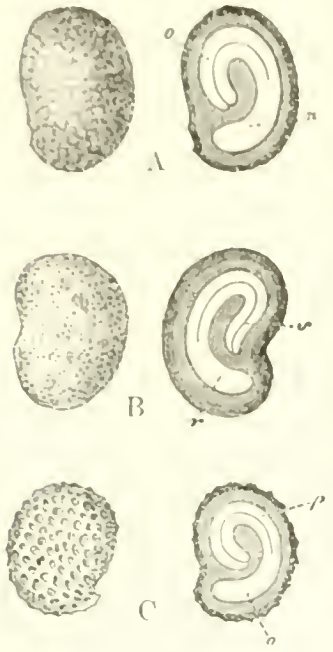

FIG. 31.—Seeds of Belladonna, Stramonium, and Hyoscyamus. External appearance and section. (Dragendorff.) *A*, Belladonna. *B*, Datura. *C*, Hyoscyamus.

THE CHEMICAL IDENTIFICATION of atropin, hyoscyamin, and hyoscin in the small quantities in which they may be expected to be present in a cadaver may easily fail, although in some cases the statement of Kratter[1] that the separation of atropin from the cadaver and its identification can be accomplished with certainty is true, with the reservation that hyoscin, hyoscyamin, and atropin cannot be distinguished from each other. Under the most favorable conditions, therefore, it can only be asserted that one of the three is present, while in many cases the toxicologist will not be warranted in making any statement more positive than that the appearances observed are such as would be produced by atropin, while certain other tests of atropin have failed (see below).

The most favorable situation for the search for atropin is the urine, by which it is eliminated, and in which it may be detected even after it has been completely absorbed from the stomach, or after introduction by another channel, and in non-fatal as well as in fatal cases.

Atropin apparently withstands putrefaction well. Dragendorff[2] states that atropin was separated after two and a half months from an organic mixture which had stood in a warm room and was in advanced putrefaction.

[1] Vrtljschr. f. ger. Med., 1886, xliv. 95

[2] "Ermittelung von Giften," 4te Aufl., 1895, 214.

Atropin is extracted from alkaline solutions by benzene and by chloroform.[1] The quantity of benzene used should be sufficient, it should not be allowed to become cold, and should be removed and evaporated spontaneously or at a slightly elevated temperature as soon as possible, as concentrated benzene solutions deposit crystals of the alkaloid when allowed to stand in the cold. The residue should be purified by dissolving it in water faintly acidulated with sulfuric acid, filtering the solution if necessary, washing the acid aqueous solution by agitation with chloroform or ether, alkalizing it with slight excess of ammonia, extracting the alkaloid by agitation with chloroform, washing the chloroform solution with distilled water, and allowing it to evaporate spontaneously. The residue so obtained is amorphous, and may be converted into crystals of the alkaloid by solution in alcohol, or of the sulfate by solution in alcohol containing a minute quantity of dilute sulfuric acid, and spontaneous evaporation of the solution. Excess of alkali is to be avoided during the extraction and purification, and the alkaline liquid should not be heated, lest the alkaloid be decomposed.

1. Kratter[2] attaches great importance to the appearance of the crystals, particularly when they are observed with polarized light, as one of the factors in the evidence of the presence of atropin. When it is possible the crystals should be obtained and photographed before proceeding to the chemical or physiological tests.

2. The sensitiveness of solutions of atropin salts to the general reagents is given by Dragendorff as follows: Iodin in potassium iodid gives a precipitate in solutions of 1:8000; phosphomolybdic acid, a cloudiness in 1:4000; phosphoantimonic acid, a faint cloud in 1:5000; potassium-bismuth iodid, 1:4000 distinctly; potassium-cadmium iodid, 1:500 distinctly; potassium-mercuric iodid, 1:4000 faintly; auric chlorid, 1:100 faintly; picric acid, 1:200 distinctly, 1:500 fails.

3. According to Wormley[3] an aqueous solution of hydrobromic acid, saturated with bromin, produces in solutions of the salts of atropin and of the free alkaloid, even when highly

[1] Also by amylic alcohol, with which, however, a portion of the alkaloid volatilizes on evaporation.

[2] *Loc. cit.*, 86, Plate ii.

[3] "Micro-Chemistry of Poisons," 2d ed., 641.

diluted, a yellow, amorphous precipitate, which in a little time becomes crystalline (Figs. 32, 33). The precipitate from somewhat strong solutions of the alkaloid after a time disappears, but is immediately reproduced upon the further addition of the reagent. The precipitate is insoluble in acetic acid, and only very sparingly soluble in large excess of hydrochloric, nitric, and sulfuric acids, and in the fixed caustic alkalies; it is even produced from solutions of the alkaloid in concentrated sulfuric acid. Wormley regards this reaction as characteristic of atropin (and hyoscyamin and hyoscin), as, although most of the al-

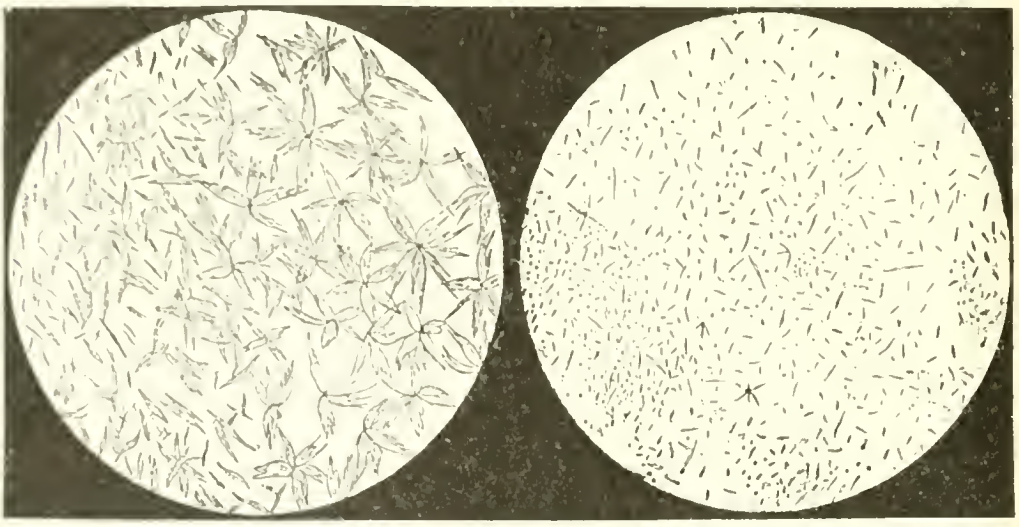

FIG. 32.—Gr. $\frac{1}{150}$ Atropin + Bromin in Hydrobromic Acid. × 75. (Wormley.)

FIG. 33.—Gr. $\frac{1}{1000}$ Atropin + Bromin in Hydrobromic Acid. × 125. (Wormley.)

kaloids and certain other organic substances form yellow precipitates with the reagent, these do not become crystalline except that from opianyl (meconin), which differs from that produced by atropin in its crystalline form. Nor does opianyl respond to the physiological test or to Vitali's reaction.

Although recent European writers (Dragendorff, Otto, Baumert, Guareschi, Dupuy, Kobert) make no mention of this reaction, we have found it to be quite distinct, and have obtained it with the suitable residue in a case of alleged poisoning by atropin and morphin in which the physiological test gave affirmative results, but the Vitali reaction failed. Although this is the condition of affairs which one would expect in searching for atropin when that alkaloid is present in organic mixtures (see Vitali reaction and physiological test below), we

had not sufficient faith in the diagnostic certainty of this test to assert positively the presence of atropin, particularly as the result of the physiological test, although distinct, was not intense.

A solution of bromin in hydrobromic acid was used as a reagent by Selmi in his researches upon the ptomaïns, many of which gave with it yellowish, yellow, or orange precipitates, which are, however, not described as crystalline.[1]

4. Atropin (and hyoscyamin, hyoscin, and homatropin) redden phenolphthalein paper or solution; which other vegetable alkaloids, except coniin and nicotin, do not do.[2]

Methylamin, cadaverin, putrescin, and other ptomaïns which are amins also redden phenolphthalein.

5. The most reliable of the tests for atropin (as well as hyoscyamin and hyoscin) is that suggested by VITALI:[3] the solid alkaloid, or any of its salts except the chlorid, is treated with fuming nitric acid, heated to boiling, and evaporated to dryness on the water-bath. On moistening the cooled colorless or yellowish residue with a freshly prepared alcoholic solution of potassium hydroxid a purple color is produced, which changes to violet and red, and finally disappears. The reaction is sensitive to 0.000001 gm. with the pure alkaloid or the sulfate. If a solution is to be tested it must be evaporated, and the test applied to the solid residue. The test should not be applied to the chlorid of the alkaloid, because, as Chapuis has shown,[4] the reaction usually fails when applied to that salt. We have also found the reaction to fail in a residue, even after the addition of atropin sulfate in amount sufficient to give the appearances distinctly. The residue had been purified as far as possible by repeated washing of the acid solution with benzene and chloroform, yet only a deep orange color, without any trace of violet, was obtained.

Two other alkaloids give results with the Vitali reaction, which are in some respects similar to those produced by atropin.

Beckmann[5] stated that *veratrin* behaves like atropin to this

[1] "Memorie sopra Argomenti tossicologici," Bologna, 1878, 143 *seq.*, 167 *seq.*

[2] Plugge: Arch. d. Pharm., 3 R., xxv., 45.

[3] J. d. méd., chir., et pharm., Bruxelles, 1880, lxxi., 66, 161, 167, 279, 382; *ex* l'Orosi, 1880, No. 8.

[4] "Précis de Tox.," 2ème ed., 631.

[5] Arch. f. Pharm., 1886, 3 R., xxiv., 481.

test as well as to Flückiger's modification, but that if a nitrite be used in place of a nitrate, and aqueous potash substituted for alcoholic, atropin alone gives a violet color. Moreover, veratrin does not behave like atropin with Gerrard's reaction, and does not redden phenolphthalein. Nor does atropin, mixed with sugar and moistened with sulfuric acid, turn green and blue as does veratrin, but yellow and brown; or form a red solution, like veratrin, when heated with hydrochloric acid. Thoms[1] has pointed out these differences: that the residue after the action of nitric acid is "brownish-yellow" with veratrin and "hardly yellowish" with atropin, and that on addition of alcoholic potash veratrin is only slowly colored red-violet, while with atropin the color is immediately pure violet. Fabris[2] was the first to call attention to the fact that *strychnin* interferes with the Vitali reaction, unless the atropin is in great excess. Later Mengazzi,[3] having obtained both the Vitali reaction of atropin and the color reaction of strychnin, but only the physiological action of the latter alkaloid in a toxicological investigation, experimented with strychnin alone and found that even in the amount of 0.0001 gm. strychnin produces with the Vitali reaction a violet color, which is, however, transient and soon passes into orange and brownish-red. The color is only produced when the potash solution is quite fresh and colorless, and of about four-per-cent. strength. Vitali[4] in a further study of his reaction points out the following differences between atropin and strychnin: (1) During the evaporation of the nitric acid, particularly on approaching dryness, atropin gives off an agreeable odor, resembling that of hawthorn blossoms; strychnin does not. (2) Strychnin moistened with nitric acid is colored yellow, which on heating turns to orange-red, and back to yellow, leaving a yellow residue; atropin is not colored, and leaves a colorless residue. (3) If the mixture be evaporated to dryness after addition of the potash solution a residue remains which is violet with atropin, and yellow or reddish-yellow with strychnin; and which, on addition of further alcoholic potash, becomes more intensely violet with atropin, but red-violet, and then reddish-brown with strychnin. (4) The red or red-violet

[1] Ph. Centralh., 1890, xxxi., 559.
[2] Berichte, Berlin, 1892, xxv., *c.*, 643.
[3] Boll. Chim. Farm., 1894, iv., 103.
[4] *Ibid.*, 449.

color produced with strychnin passes immediately to yellow on addition of water, but on similar treatment the violet of atropin is completely discharged. Either color is restored on addition of further alcoholic potash. (5) If an aqueous solution be used in place of an alcoholic, atropin gives a yellow color and the mixture, when stirred, deposits brownish flocks, which dissolve in alcohol with a fine violet color. Under like treatment strychnin gives a reddish-brown, almost black, color which changes to red-violet on addition of alcohol. (6) If the residue of evaporation of nitric acid in the presence of atropin be treated with ammonium hydroxid, pale yellow drops separate, which dissolve in alcoholic potash with a violet color. Or if the ammoniacal liquid be extracted with chloroform, this, on evaporation, leaves an almost colorless residue, which gives a very beautiful and enduring violet color with alcoholic potash. In the presence of strychnin, in place of atropin, ammonium hydroxid causes an orange-red color, and, on addition of alcoholic potash, a transient violet is produced, which suddenly passes to a deep blood-red. Or chloroform agitated with the ammoniacal liquid is colored light yellow and leaves a yellow residue, which gives an orange-red color, increasing in intensity, with alcoholic potash. (7) If the residue of evaporation of nitric acid and atropin be ground up with a little potassium cyanid, a yellow color is produced which, on addition of alcoholic potash, passes into an intense but rapidly transient violet, or on agitation passes to a dirty greenish-blue. If the mixture be allowed to evaporate spontaneously yellow rings are formed in the capsule, and sky-blue drops separate, which are dissolved by ether. Under like treatment with strychnin the mixture with potassium cyanid is reddish-brown, and a fine red-violet color, passing to blood-red, is produced by alcoholic potash. If the mixture be agitated with a little water and ether, the ether assumes a yellow color, and the water is colored reddish-yellow or red.

Arnold[1] has modified the reaction in that he heats the residue with concentrated sulfuric acid and a minute fragment of sodium nitrite, when a dark yellow or orange residue remains, which on addition of alcoholic potash gives a beautiful

[1] Ztschr. f. an. Chem., 1884, xxiii., 231; *ex* Arch. d. Pharm., 3 R., xx., 561.

red-violet, soon changing to red. FLÜCKIGER[1] mixes the alkaloid with sodium nitrate, adds concentrated sulfuric acid, and finally stirs in an alcoholic solution of sodium hydroxid.

6. GERRARD'S REACTION[2] consists in adding to a small portion of the substance in a test tube about 2 c.c. of a five-per-cent. solution of mercuric chlorid in fifty-per-cent. alcohol and warming gently: a yellow precipitate of mercuric oxid is at once formed, and turns brick-red. The atropin chlorid produced in this reaction combines with a further quantity of the mercuric salt, to form a definite compound which crystallizes out in tufts as the liquid cools. Schweissinger[3] has further studied this reaction and found it reliable; also that if 0.001 gm. of hyoscyamin be treated with 2 c.c. of the reagent the precipitate is not formed even on heating or after long standing; but that if a fragment of hyoscyamin be treated with only one or two drops of the solution, the precipitate appears immediately on warming. He therefore recommends a one-per-cent. solution of mercuric chlorid in place of a five-per-cent., to differentiate hyoscyamin from atropin. Homatropin does not respond to this test, whereas it does to Arnold's modification of the Vitali reaction. According to Schweissinger a combination of the Gerrard and Arnold tests permits of a distinction between atropin, hyoscyamin, and homatropin.

7. GULIELMO'S REACTION[4] consists in moistening the residue with concentrated sulfuric acid, warming until the mixture turns brown and begins to give off fumes, and adding a few drops of distilled water, when an odor is developed which is between that of orange blossoms and that of the wild sloe (*Prunus spinosa*). Brunner[5] modified the reaction, and made it more certain by using chromic anhydrid in place of sulfuric acid. The solid under examination is added to a few crystals of chromic anhydrid in a porcelain capsule, which is then heated until a green color is produced. According to Sohn[6] the test is applied as follows: If atropin sulfate be heated first alone until white fumes appear, then sulfuric acid (1.5 gm.) be added, and further heated until browned, an agreeable odor is produced on

[1] Ph. J. and Tr., 1886, 3 s., xvi., 601.

[2] Ph. J. and Tr., 1883–84, 3 s., xiv., 718, 729.

[3] Pharm. Ztg., 1884, xxix., 683.

[4] Schweiz. Wochenschr. f. Pharm., 1863, i., 146.

[5] Berichte, Berlin, 1873, vi., 98.

[6] "Dict. of the Act. Princ. of Plants," London, 1894, 15.

subsequent addition of 2 c.c. water. On then adding potassium permanganate (a fragment), an odor of bitter almond oil will be perceived.

An agreeable odor, accompanied by that of acetic acid, is also developed when atropin is boiled with a mixture of equal volumes of concentrated sulfuric acid and glacial acetic acid and allowed to cool. The mixture is also fluorescent.

It must not be forgotten that many ptomaïns exhale odors resembling those of certain flowers.

8. THE PHYSIOLOGICAL TEST consists in applying a drop or two of the neutral solution to the corner (inner canthus) of the eye and observing the dilatation of the pupil which is produced by atropin, hyoscyamin, and hyoscin. Of the lower animals the cat should be selected for the experiment, or, if the alkaloid or its sulfate has been separated in a crystalline and pure condition, the application may be made to the human eye, which is more sensitive. In any event no excess of acid should be present. According to Donders[1] dilatation is produced by one drop of a solution of 1 : 130,000. Fedderson,[2] from the results of experiments with seventy-six individuals, fixes the minimum quantity of atropin sulfate which will distinctly dilate the healthy pupil of a human adult at 0.0002 mgm. (= 000003 gr.), while half that amount caused dilatation in forty-two per cent. of the subjects.

Mydriasis is also produced by cocain, aconitin, and certain ptomaïns.

Ptomatropin.—Toward the end of the eighteenth century it was observed that in persons suffering from botulism (sausage poisoning) the pupils were dilated, frequently unsymmetrically, and the upper lid fell over the eye (blepharoptosis).[3] In 1869 Zülzer and Sonnenschein[4] obtained from putrid muscular tissue, after five to eight weeks, a crystalline substance responding to several of the general reagents for alkaloids, which was extracted by ether from alkaline aqueous solutions, and which produced mydriasis in cats and rabbits. This alkaloid is not identical with another ptomaïn, also possessed of mydriatic action, obtained by Brieger, and called by him *my-*

[1] Otto: "Ausmittelung d. Gifte," 6te Aufl., 73.

[2] Diss., Berlin, 1884, p. 38.

[3] Kerner: "Das Fettgift." 1822, xix., 108 and *passim*.

[4] Berl. kl. Wochenschr., 1869, vi., 121.

dalein.[1] It may, however, have been identical with one of those obtained by Selmi which caused a dilatation of the pupil of much shorter duration than that caused by atropin.[2] It is plain therefore that the physiological test, unsupported by the evidence of other tests, is not sufficient to distinguish atropin from a ptomaïn.

All of the putrid products, however, which have been described as presenting resemblances to atropin in some respects differ from it in others. Thus Zülzer and Sonnenschein's alkaloid, while crystalline and mydriatic, gave a heavy white cheesy precipitate with mercuric chlorid, but the same reagent in the cold fails to precipitate even concentrated solutions of atropin salts, while in alcoholic solution it gives Gerrard's reaction, to which the putrid alkaloids do not appear to respond.[3] The chloroplatinate of Brieger's mydalein is crystalline and its chlorid crystallizes with great difficulty and is deliquescent; while atropin chloroplatinate is amorphous and its chlorid, although not crystalline, is stable. The behavior of neither Zülzer and Sonnenschein's base nor that of mydalein toward Vitali's reaction is indicated. The only putrid product said to react with Vitali's test is one referred to by Giotto and Spica,[4] which, however, gave negative results with the Gulielmo reaction, and had no mydriatic action. Selmi's base, which is not only mydriatic but also gives off a fragrant odor, does so spontaneously in aqueous solution after a few days, or when treated with acids in the cold, while atropin only does so on being heated with an acid.

COCAIN.

Although the properties of coca (*Erythroxylon coca*) were well known to the Peruvians in the time of Pizarro (1532),[5] and Monardes, in 1582, gave an account of its use and effects which reminds one of the cocain *habitués* of to-day,[6] the plant appears

[1] "Ptomaine," ii., 48.

[2] "Sulle Ptomaine," Bologna, 1878, 10, 35, 105.

[3] Brieger, who used an alcoholic solution of mercuric chlorid as a precipitant for ptomaïns, notes no such appearance as that produced by atropin with Gerrard's test.

[4] L'Orosi, 1890, No. 10, *ex* Ph. Centralh., 1891, xxxii, 26.

[5] Acosta: "Hist. nat. y moral. d. l. Indias," 1590, lib. iv., cap. 22. A royal order of 1601 regulated the employment of Indians upon coca plantations (Helps: "Span. Conq. of America," iv., 363).

[6] "Simplicium medicamentorum ex novo orbe," etc., Antwerp, 1582. The account is not found in the edition of Antw., 1579.

to have been forgotten among Europeans until Montegazza again called attention to its medicinal value in 1859.[1] In the same year a quantity of the leaves was brought to Europe by the Austrian frigate *Novara* and submitted to Professor Woehler for examination. Under his direction Niemann isolated the alkaloid for the first time.[2] During the succeeding twenty-five years numerous observations of the therapeutic action of cocain were reported,[3] but it was only in 1884 that its value as a local anæsthetic was simultaneously recognized by Koller[4] and Jelinek in Vienna,[5] although the temporary insensibility which it produces had already been noticed by von Anrep,[6] and even by Niemann.[7]

Cocain, $C_{17}H_{21}NO_4$, whose partial synthesis from ecgonin[8] has been accomplished,[9] is benzoyl-methyl-ecgonin $[C_5H_7.NCH_3.CH]OC_7H_5O,CH_2.COOCH_3$. It crystallizes in monoclinic prisms, which fuse at 98° (208°.4 F.), and at higher temperatures sublimes partly and is partly decomposed. It dissolves in 704 parts of cold water, more readily but with liability to decomposition in hot water, readily soluble in ether, alcohol, and benzene. Chloroform, methyl alcohol, acetone, and petroleum ether dissolve it when hot, but deposit it in crystals on cooling. It is lævogyrous ($[\alpha]_D = -15°.8$). Its solutions are alkaline, have a slightly bitter taste, and cause loss of taste and local anæsthesia.

Cocain is an ethereal base which is readily saponified. Even on contact with boiling water it is decomposed into methylic alcohol and *benzoyl-ecgonin*, and by concentrated acids or baryta water into methylic alcohol, benzoic acid, and ecgonin. This decomposition also takes place rapidly in the living body, and when cocain is taken in non-fatal doses the urine does not contain it, but contains ecgonin. Glasenap,[10] experimenting with dogs, found cocain as such when death was caused in one or two hours, but only ecgonin after less rapidly fatal action. The

[1] Prize essay. Milan, 1859. *ex* Ph. J. and Tr., 1860, n. s., i., 616.

[2] Ann. d. Ch. u. Pharm., 1860, cxiv., 213.

[3] See Index Cat. Libr. Surg.-Gen. U. S. A., Washington, 1883, iv., 352.

[4] Wien. med. Bl., 1884; Wien. med. Wochenschr., 1884.

[5] Wien. med. Bl., 1884; Wien. med. Wochenschr., 1884.

[6] Arch. d. ges. Physiol., 1870, xxi., 38.

[7] *Loc. cit.*

[8] See below.

[9] Merck: Berichte, Berlin, 1885, xviii., 2264, 2952.

[10] Diss., St. Petersburg, 1894.

same author confirms the statement of Vitali,[1] that the decomposition of cocain, rapid during life, progresses only very slowly after death in the presence of putrefying material.[2]

Cocain is used medicinally in the form of its chlorid: *Cocainum chlorid, hydrochlorate of cocain*, which crystallizes in anhydrous colorless prisms, plates, or scales, fusible at 181°.5 (358°.7 F.); soluble in 0.75 part of water, difficulty soluble in absolute alcohol, insoluble in ether, benzene, petroleum ether, and soluble in acetone and in chloroform. Its solutions are neutral, and it decomposes in a short time when in solution. (For reactions see p. 690.)

Benzoyl-ecgonin accompanies cocain in coca leaves, or at least in the product therefrom. It crystallizes in prisms with 4 Aq., or in long anhydrous prisms from chloroform. The hydrated crystals fuse at 90°–92° (194°–197°.6 F.) the anhydrous at 189°–193° (372°.2–379°.4 F.). It is neutral in reaction, and has but slight anæsthetic action. It is difficultly soluble in cold water and in ether, but soluble in alcohol, methylic alcohol, and in hot water, acetone, or chloroform.

Ecgonin, $C_9H_{15}NO_5$, is a substance possessed of both basic and acid properties, whose constitution has been partially established,[3] which occurs in the amorphous bases obtained after the separation of cocain, and is one of the products of decomposition of that alkaloid. It crystallizes, with 1 Aq., in oblique rhombic prisms, fusible at 198° (388°.4 F.). It is neutral in reaction, bitter-sweet in taste, and very soluble in water, soluble with difficulty in absolute alcohol or chloroform, and insoluble in ether and carbon disulfid.

The chemical relationship between atropin and cocain is close. Both are ether bases, yielding an acid and a pyridin derivative on saponification. Ecgonin on decomposition of its barium compound by heat yields a liquid base, boiling between 210°–230° (410°–446° F.), isomeric with tropin (see p. 660),

[1] "Manuale di chim. toss.," 1893, 403.

[2] See also Sonnié-Moret . J. de ph. et de chim., 1893, 5 s., xxviii., 390.

[3] For the chemistry of ecgonin and cocain see the researches of Einhorn: Berichte, Berlin, 1887, xx., 1221; 1888, xxi., 47; 1889, xxii., 399, 1362, 1495, 1890, xxiii., 468, 979, 1338; 1893, xxvi., 324, 962, 1482; 1894, xxvii., 1874, 2447; and those of C. Liebermann: *Ibid.*, 1888, xxi., 2342, 3196; 1889, xxii., 130, 672, 675, 680, 2661; 1890, xxiii., 141, 508, 512, 926, 2518; 1891, xxiv., 407, 606, 1101, 2336, 2587; 1892, xxv., 927; 1893, xxvi., 834; 1894, xxvii., 1416, 2037, 2051.

isotropin or *pseudotropin*, $C_8H_{15}NO$, a derivative of which, *benzoyl-pseudotropin*, $C_8H_{14}NO(C_6H_5CO)$, has been obtained in colorless, crystalline plates from Java coca leaves. As atropin is but one of a series of *tropeins* produced by the union of tropin with acid radicals, so cocain is the representative of similar compounds of ecgonin with acid and alcoholic radicals; one of which, *cinnamyl-methyl-ecgonin*, or *cinnamyl-cocain*, $C_9H_{17}NO_3,CH_3(C_9H_7O)$, occurs naturally in Java coca.

Cocamin—*Truxillin*—$C_{19}H_{23}NO_4$—is another of the amorphous bases of the coca leaves, which is neutral in reaction, bitter, soluble in alcohol, ether, chloroform, benzene, and hot water, but sparingly soluble in cold water and in petroleum ether. It has no anæsthetic action, but is intensely poisonous.[1]

Hygrin.—As early as 1862 Lossen[2] described a liquid, volatile alkaloid which accompanies cocain. The cocain mother liquors from Truxillo coca were further examined by Liebermann,[3] who found two volatile alkaloids, *α hygrin*, $C_8H_{15}NO$, and *β hygrin*, $C_{14}H_{24}N_2O$, which are strongly alkaline liquids, having a quinolin-like odor and forming crystalline chlorids.

Statistics.—In the brief period during which cocain has been in general use instances of poisoning by it have accumulated rapidly. The earliest case reported was that of a pharmacist, who took a large quantity of a crystalline extract, obtained from two pounds of the leaves, in 1863. He went to sleep, but in a few hours awoke and manifested the symptoms since so familiar, with suppression of urine during twenty-four hours, but ultimately recovered.[4]

We have collated the reports of 273 cases up to 1895.[5] All were accidental poisonings resulting from the use of cocain in surgical practice, except in one instance. Fager-

[1] Liebermann: Berichte, Berlin, 1888, xxi., 2342.

[2] Ann. d. Ch. u. Ph., 1862, cxxi., 374.

[3] Berichte, Berlin, 1888, xxii., 675; 1891, xxiv., 407.

[4] Plass: Ztschr. f. Med., Chir., u. Gebh., 1863, n. F., ii., (5), 222.

[5] For good bibliographies of the earlier cases see Latte: Diss., Berlin, 1888. Mannheim: Ztschr. f. kl. Med., 1890, 380, and Falck: Therap. Monatshft., 1890, iv., 511, 564. Also Mattison's papers, Quart. J. Inebr., 1888, x., 57; M. and S. Reptr., 1891, lxv., 645. We have not access to Viau's paper: "Étude critique des intoxications par la cocain," Odontologie, Paris, 1893, xiii., 97. Other recent cases not mentioned by the above are reported by Glück: Med. Rec., N. Y., 1890, xxxvii., 709; Anon., Odontologie, Paris, 1890, x., 356. Hueber: Deut. mil.-ärztl. Ztschr., 1890, xix., 160. Legg: Br. M. Jour., 1890, ii., 732 [coroner's inq.], Lancet, 1891, ii., 849. Berger: Bull.

lund's case[1] was that of a young woman who swallowed a teaspoonful of cocain in a glass of beer with suicidal intent, and died in half an hour.

Up to the present time we find record of 20 fatal cases.

1. Man, 33; small quantity of a two-per-cent. solution brushed into the larynx. Died in three hours.[2]

2. Woman, 23; twenty-four "gran" (1.5 gm.) administered in enema. Death in three hours. The surgeon, Professor Kolomnin, killed himself in consequence.[3]

3. Girl, 11; six to twelve drops of a four-per-cent. solution injected over the deltoid. In less than forty seconds took a deep breath, became deathly pale, dropped unconscious, and died one minute later.[4]

4. Woman, 39; used a four-per-cent. solution for toothache. "Seen at 5 A.M., died apparently from complete exhaustion 3:30 A.M." *(sic!)*[5]

5. Man, 29; one drachm of a twenty-per cent. solution injected into the urethra. Death in twenty-five minutes.[6]

6. Female, 71: subconjunctival injection of 0.04 gm. ($\frac{6}{10}$ grain). Death in five hours.[7]

7. Woman; hypodermic injection in breast of 0.225 gm. ($3\frac{1}{2}$ grains). Immediate epileptiform convulsions, which were repeated until death.[8]

8. Man; twenty grains (1.3 gm.) given in solution by nurse to patient in hospital, in consequence of error in delivering medicines. Death in one hour.[9]

9. A pharmacist, who sprayed his own throat with a solution of cocain, had a series of syncopes and died in seven to eight hours.[10]

10. "Death at a dentist's in consequence of injections of cocain."[11]

11. Woman, 29; subgingival injection of 0.06 gm. ($\frac{9}{10}$ grain). Became motionless and died soon.[12]

et mém. Soc. d. chir., Paris, 1891, n. s., xvii., 751. Also Gaz. d. hôp., 1891, lxiv., 1367. Cobb: South Clin., Richm., 1891, xiv., 7. Eliot: Virg. M. Monthly, 1891–92, xviii., 947. Lorentz: Centlb. f. Gyn., 1891, xv., 1033. Trzebicky: Wien. med. Wochenschr., 1891, xli., 1521. Chobant: Lyon méd., 1892, lxx., 73. McReynolds: Tex. Cour.-Rec. M., 1892–93, x., 252. Lafevre: Kansas M. J., 1893, v., 357. Baker: Am. J. Ophth., 1893, x., 342 (two cases). Redus: Bull. et mém. Soc. d. Chir., Paris, 1894, n. s., xx., 276. Haynes: M. News, Phila., 1894, lxv., 144. Fagerlund: Vrtljschr. f. ger. Med., 1894, 3 R., viii., Supplh. 91 (two cases).

[1] *Loc. cit.*

[2] Long: Amer. Lancet, 1886, n. s., x., 404.

[3] Therap. Monatshft., 1888, ii., 393, *ex* Vrach, St. Pet., 1886.

[4] Knabe: Berlin, cited by Mattison, Med. Age, 1887, v., 129.

[5] Thomas: *Ibid.*

[6] Simes: M. News, Phila., 1888, liii., 70.

[7] Abadie, Latte: Diss., Berlin, *ex* Intern. kl. Rundschau, 1888.

[8] Zambianchi: Gazz. d. Osp., 1888, 93.

[9] Lancet, 1889, i., 292.

[10] Baratoux, quoted by Reclus and Wall: Rev. de Chir., 1889, ix., 167.

[11] Odontologie, Paris, 1890, x., 356. Journal inaccessible to us.

[12] "Liller Zahnarzt," J. d. Zahnhlk., 1890, 25 Sept.

12. Man, 45; an application of five drops of a five-per-cent. solution to the ear.[1]

13. Man, 29 (physician); found dead in a lavatory. Had probably taken an overdose of cocain to relieve pain, and had recourse to apomorphin.[2]

14. Man, 40; about 0.4 gm. (6 grains) injected in solution into tunica vaginalis. Death in twenty minutes.[3]

15. Man, 44; about one drachm of a four-per-cent. solution (= 0.154 gm.) injected into urethra. Convulsions, great cerebral excitement, pulse imperceptible, death in four to five minutes.[4]

16. Girl, 12; a solution sprayed into the nose. Suddenly became faint and died in a few minutes.[5]

17. Young woman; a teaspoonful in beer. Suicide; death in half an hour.[6]

18. Boy, 2½; half a grain (0.03 gm.) injected into the urethra. Death in three days.

19. "A friend;" subgingival injection of four grains (0.26 gm.) Death in convulsions in one hour.[7]

20. Man, 72; 1 gm. (15.3 grains) injected into urethra. Death in a few minutes.[8]

In the two cases in which cocain was swallowed (8, 17) the dose taken was large, and probably notably in excess of that capable of causing death. Much smaller quantities otherwise administered caused death in Nos. 3, 6, 11, 12, 14, 15, 18. In the earlier cases, however, the purity of the cocain used is not beyond question. Mannheim[9] considers 1 gm. as the lethal dose, whether it be taken by the mouth or otherwise.

When death has followed the introduction of cocain into the general circulation it has done so rapidly, within a few minutes, or in less than an hour. In a few instances (1, 2, 6) life has been prolonged for from three to five hours, and in one exceptional case for three days (18). When taken by the stomach cocain acts somewhat more slowly (8, 17) than when injected or absorbed from the urethra.

Symptoms—Non-fatal cases.—Immediately after the administration the patient "feels queer" and soon becomes very restless, with a wild look in the eyes, is frequently very loqua-

[1] Schwabach: Therap. Monatshft., 1890, iv., 149.
[2] Lancet, 1891, ii., 849.
[3] Berger: *Loc. cit.*
[4] Gwyer: Unpublished; Bellevue Hosp. Records, 1892, p. 553.
[5] Baker: *Loc. cit.*
[6] Fagerlund: *Loc. cit.*
[7] Haynes: *Loc. cit.*
[8] Redus: *Loc. cit.* In the light of previous experience this case can hardly be considered as "accidental."
[9] *Loc. cit.*

cious, utters foolish remarks, and makes chorea-like movements with the arms and legs. Frequently the mental disturbance progresses to delirium, with hallucinations of sensation and of vision, or a maniacal condition in which the efforts of several attendants are required to control the patient. There is a sense of dryness and of scratching in the mouth and throat, difficulty of deglutition, and a feeling of constriction and impending suffocation, anæsthesia at the seat of application, and a sensation of tingling, prickling, or numbness of the hands and feet. There is an early inclination to vomit, which later develops into extreme nausea with eructations, although actual vomiting rarely occurs until after administration of brandy, which frequently provokes it. There are cramps, or a burning pain in the stomach. Attacks of giddiness, with loss of power in the legs, staggering, and a disposition to faint are early manifestations. The speech is incoherent, although the person is aware of what is transpiring around him.[1] There are spasmodic contractions and rigidity of the fingers, arms, and legs, with a tendency to opisthotonus, and frequently violent tetanic or epileptiform attacks. The face at first is usually extremely pallid, and the forehead covered with profuse perspiration, although in several instances there has been marked redness, or even cyanosis of the face in the early stages.[2] The pupils are in most cases dilated and insensible to light. In some instances, however, they have been of normal size and sensitive,[3] in others unsymmetrically dilated,[4] or contracted,[5] or contracted at first and afterward dilated.[6] The lids are widely opened, the eyeballs protruding, and there are disturbances of vision, paralysis of accommodation, and even complete amaurosis. The pulse is at first rapid, feeble, and frequently hardly perceptible at the wrist, although the carotids may pulsate strongly, and the heart's action is tumultuous, bounding, and thumping, with

[1] For a good account of the sensations see Way: Med. News, Phila., 1887, l., 486.

[2] Ashworth: Lancet, 1889, i., 273. Baker: Am. J. Ophth., 1893, x., 342. Bullock: Boston M. and S. J., 1887, cxvi., 575. Blodgett: *Ibid.*, 1887, cxvii., 282. Wagner: Centlb. f. Nervenhlk., 1887, x., 518.

[3] Ashworth: *Loc. cit.* Heymann: Berl. kl. Wochenschr., 1885, xxii., 795. Beck: Deut. med. Wochenschr., 1886, xii., 92.

[4] Ricci (Deut. med. Wochenschr., 1887, xiii., 894) administered hypodermically in the leg. Thomas: Med. Age, 1887, v., 129.

[5] Spear: Med. Rec., N. Y., 1885, xxviii., 536.

[6] Mannheim: Berl. kl. Wochenschr., 1886, xxiii., 583.

severe palpitation and pain in the cardiac region. The respiration is short, convulsive, gasping, panting, or Cheyne-Stokes.[1] There are severe pain and a bursting sensation in the head. The patient rapidly becomes prostrated, suffers short periods of collapse and unconsciousness, or may become deeply comatose. The surface is cold, there are attacks of shivering, coldness, and copious general perspiration. In exceptional instances, however, the skin is dry.[2] In the later stages the surface becomes cyanosed, the heart's action irregular, the pulse slow, weak, intermittent, and imperceptible, and the respiration slow, shallow, irregular, and tending to arrest. Frequently when the symptoms have ameliorated they return with marked severity on moving the patient. Recovery is usually rapid and complete, although in some cases great muscular weakness, prostration, and inability to perform certain acts persist for several weeks or longer.[3]

Fatal Cases.—The small number of fatal cases in which the symptoms have been reported are divisible into two groups: Those in which death has been caused rapidly, within a few minutes, and those in which the patient has survived a few hours. In Knabe's case (3)[4] the girl took a deep breath in less than forty seconds, turned deathly pale, dropped unconscious, and was dead one minute later. In a case of subgingival injection (11) the patient became motionless and soon died. In the case referred to by Redus (20) the patient, who was suffering from cardiac disease and angina pectoris, and had sudden suppression of urine, became pale immediately after the injection, was seized with nausea and trembling, the face became livid, and he died in a few minutes in an attack of syncope. In Baker's case (16) the girl died of failure of respiration in a few minutes in spite of active treatment.[5] In less rapidly fatal cases the symptomatology is that of non-fatal cases, death occurring either from failure of respiration or of the heart's action.

Treatment.—Removal of unabsorbed cocain will be rarely

[1] Mayerhausen: Wien. med. Presse, 1885, xxvi., 707.

[2] Finlay: Austral. M. Gaz., 1887–88, vii., 249. Knocke: Kansas City Med. Rec., 1886, iii., 400.

[3] Fox: Brit. M. Jour., 1888, i., 349. Kilham: Lancet, 1887 i., 17. Hallopeau: Bull. gén. de thérap., 1891, cxx., 481.

[4] The numbers refer to the list of cases above.

[5] See also Gwyer's and Zambianchi's cases above.

possible. If, however, it have been administered by the mouth, rectum, etc., these should be washed out as expeditiously as possible. The tendency to collapse may be combated by inhalation of amyl nitrite and hypodermics of ether. The internal administration of alcoholic stimulants is also indicated for the same object. For the control of spasms chloroform or ether by inhalation, and chloral hydrate internally may be resorted to, and artificial respiration to supplement failure of that function.

Post-Mortem Appearances.—In the few autopsies after death from cocain marked hyperæmia of the liver, spleen, kidneys, lungs, and particularly of the brain and cord, have been uniformly observed, and also injection and ecchymoses of the gastric mucous membrane.[1]

Detection.—Cocain is extracted from ammoniacal aqueous liquids by petroleum ether or, better, by benzene, and is consequently to be sought for in residues V. and VI. (p. 136). The tests for its identification are, however, as yet unsatisfactory:

1. Probably the most characteristic reaction is its physiological action in producing local anæsthesia at the point of application. This may be tested either by evaporating the solution upon a strip of filter paper, which is then placed upon the tongue (previously rinsed clean), and after a minute or two testing the sensibility with a needle; or by applying the neutral solution to the eye of a cat, and examining the cornea for sensibility and noting the effect upon the pupil.

2. *Giesel's Reaction.*[2]—A solution of potassium permanganate produces in solutions of pure cocaium chlorid a fine bright-violet crystalline precipitate of cocaium permanganate, in rhombic plates, frequently arranged in rosettes. The presence of impurities interferes with the reaction.

3. *Greitherr's Reaction.*[3]—Add 2–3 c.c. chlorin water to a solution of cocain and then two to three drops of a five-percent. solution of palladious chlorid: a fine red precipitate is produced which is gradually decomposed by water, is insoluble in alcohol and in ether, but soluble in sodium thiosulfate solu-

[1] Montalti: Sperimentali, Firenze, 1888, lxii., 294. Fagerlund: Vrtljschr. f. ger. Med., 1894, 3 R., viii., Supplhft., 98. Simes: Med. News, Phila., 1888, liii., 70. Berger: Bull. et mém. Soc. d. chir., Paris, 1891, n. s., xvii., 751.

[2] Pharm. Ztg., 1886, xxxi., 132.

[3] *Ibid.*, 1889, xxxiv., 617.

tion. Other alkaloids do not give the reaction, which, however, lacks in sensitiveness.

4. *Metzger's Reaction.*[1]—On addition of a five-per-cent. solution of chromic acid to an aqueous solution of cocaium chlorid a distinct precipitate is formed with each drop, which immediately redissolves. On the addition of concentrated hydrochloric acid a permanent orange-colored precipitate is formed.

5. *Vitali's Reaction.*[2]—If a minute quantity (0.00005 gm.) of cocain or of its chlorid be moistened with sulfuric acid, and a fragment of iodic acid, or of potassium or sodium iodate be added after solution, and the mixture warmed over the water-bath, light-green streaks of color are produced, which change to dark green and then to deep blue. On stronger heating the liquid becomes violet and gives off violet vapors.

6. *Schell's Reaction.*[3]—If cocaium chlorid be mixed dry with calomel the mixture on slight moistening with water (by breathing upon it) or with alcohol becomes blackened. The reaction is not produced by the free alkaloid. It is also produced more intensely by pilocarpin.

7. *Lerch and Schärge's Reaction.*[4]—Add a drop of a solution of ferric chlorid to a solution of the cocain salt: a yellow solution is formed which turns orange and red on heating.

8. *Biel's Reaction.*[5]—If a test tube containing a solution of cocain in concentrated sulfuric acid be immersed in boiling water for one or two minutes, and the solution diluted with three volumes of water after cooling, crystals of benzoic acid separate and the odor of the acid is given off.

9. *Da Silva's Reaction*[6] is the Vitali test for atropin: the material is moistened with fuming nitric acid, the solution evaporated to dryness on the water-bath, and the residue moistened with alcoholic potash; a peculiar odor, resembling that of peppermint, is given off, but no red color is produced.

Most of these chemical reactions are produced by benzoic acid, liberated by the decomposition of cocain.

10. Solutions of cocaium chlorid produce crystalline precipitates with platinic chlorid, auric chlorid, and picric acid. The

[1] Pharm. Ztg., 1889, xxxiv., 697.
[2] L'Orosi, 1891, *ex* Ph. Jahrb., 1891, 527.
[3] Ph. Ztg., 1891, xxxiv., 55.
[4] Arch. f. Pharm., 1889, 994.
[5] Ph. Ztg., 1886, xxxi., 132.
[6] J. d. ph. et de chim., 1890, 5 s., xxii., 345.

platinochlorid crystallizes in peculiar T-shaped crystals, the chloraurate in fern-like groups.

Chronic Poisoning — Cocainophagia. — The cocain habit, which bids fair to equal morphin addiction in the number of its victims, while of great hygienic importance and of forensic interest in relation to the mental condition of the cocain *habitués*, does not properly come within the limits of forensic toxicology. We therefore content ourselves with referring to a few of the more important articles upon the subject.[1]

COLCHICIN—COLCHICUM.

Two alkaloids have been obtained from *Colchicum autumnale* (meadow saffron):

Colchicein — $C_{21}H_{23}NO_6$—which crystallizes in needles, difficultly soluble in cold water but readily soluble in alcohol, chloroform, benzene, and amylic alcohol. It is feebly acid and also basic. According to Paschkis[2] colchicein is almost inert.

Colchicin—$C_{21}H_{22}NO_6$, CH_3—is the methyl ether of colchicein, and is usually amorphous, but may be obtained in prismatic crystals.[3] It is faintly basic in character, and is decomposed by hot acids into colchicein and a yellowish-green resin. It is soluble in the solvents of colchicein and is also soluble in all proportions in water. Petroleum ether not only does not dissolve it but precipitates it from its chloroform solution.

Statistics, Prognosis, Duration.—We find record of but 3 serious poisonings by colchicin. In one a woman of forty-three years received 1.2 gm. (18½ grains) dispensed in mistake for cotoin, and died from its effects in 31 hours.[4] The others were non-fatal poisonings; one that of a girl of twenty years who received 0.045 gm. (0.7 grain) of the alkaloid, and recovered from its effects.[5] The other that of a young woman who died in 33

[1] Erlenmeyer, in Penzoldt and Stintzing's "Handb. d. spec. Therap.," 1895, ii., 367, and the literature there referred to. Krebs: "Ein Fall von reinem Cocainismus," Diss., Königsb., 1892. Bauer: Med. and Surg. Reptr., Phila., 1885, liii., 365. Heiman: Deut. med. Wochenschr., 1889, xv., 232. Mattison: Quart. J. Inebr., 1888, x., 57.

[2] Med. Jahrb., Wien, 1883, 257; 1888, 569.

[3] Laborde and Hondé: "Le colchique et la colchicine," Paris, 1887.

[4] Albertoni and Casali: Boll. d. sc. med. d. Bologna, 1890, 9 s., i., 38.

[5] Koller: Ber. . . . Rudolph Stift., Wien (1867), 1868, 227.

hours from the effects of 0.0104 gm. ($\frac{1}{6}$ grain, Russian) administered in mistake for cocain in consequence of a pharmacist's error.[1] Laborde and Hondé[2] report 4 not severe accidental poisonings from 0.01 gm. or less. In a French case colchicin was alleged to have been given with homicidal intent.[3]

In other colchicum poisonings some part of the plant itself, or one of its medicinal preparations, was taken. Thus the leaves have been eaten in mistake in a salad,[4] or cooked,[5] and in several instances the capsules or seeds have been eaten by children with fatal results. Ratti[6] attributed the cause of an epidemic of gastro-intestinal irritation in a district in Rome to the use of the milk of goats whose pasture contained notable quantities of colchicum, along with other poisonous plants, and he extracted a yellow material which gave the reactions of colchicin from the milk and from the vomit of the patients. Böttern[7] has given an account of the non-fatal poisoning of five persons by beer. The symptoms were those of colchicin poisoning and the reactions of that alkaloid were obtained with an extract from the remaining beer. He refers to a similar poisoning reported by Warncke.[8] Dannenberg, has, however, shown that a substance may be extracted by the Stas or Dragendorff methods from normal beer, which gives the colchicin reactions, but which may be separated from that alkaloid by the process of purification described below.[9]

The form in which the poison was taken in most of the cases was either the wine of colchicum (that from the seeds or that from the root or bulb) or the tincture (in a few instances made from the flowers), swallowed in mistake for wine or spirits, or taken in mistake or overdose as medicine. Several proprietary gout remedies contain colchicum,[10] and an accidental death from

[1] Von Maydell: St. Petersb. med. Wochenschr., 1881, vi., 166.

[2] C. rend. soc. de biol.. Paris, 1885, 8 s., ii., 66.

[3] Affaire R——, see below.

[4] Tartarin: Gaz. d. hôp., 1881, liv., 427.

[5] Bleifus: Med. cor. Bl. würt. aerztl. Ver., 1839, ix., 409.

[6] Ph. J. and Tr., 1875, 3 s., vi., 47.

[7] Hosp. Tid., Kjøbenh., 1874, 2 R., i., 161.

[8] See also v. Gelder: Deut. Klin., 1875, xxvii., 92, and Jundzitt: "Ermittelung einiger Bitterstoffe in Bier," Diss., Dorpat, 1873, 44.

[9] See Pharm. Jahresb., 1875, 484; 1876, 633; 1877, 560.

[10] See Hahn and Holfert: "Specialitäten u. Geheimmittel," Berlin, 1893, Nos. 662, 668, 670, 671, 677, 686, 687, 703.

"Laville"[1] is included among the poisonings reported by the New York City Board of Health in 1877.[2]

We have collated the reports of 122 colchicum poisonings. Of these the great majority (108) were accidental, 6 were suicidal, and 5 homicidal. In one instance a woman took an infusion of colchicum to induce abortion. She miscarried the next day and died a few hours afterward.[3] One death from colchicin was due to gross negligence on the part of a pharmacist;[4] another was caused by the administration of colchicum by an empiric;[5] 2 fatal poisonings were the subject of the prosecution of a physician for homicide by negligence in Alsace;[6] and the method of administration of the seeds to 2 children who died therefrom remained undetermined.[7] Of the 6 attempts at suicide 2 were unsuccessful.[8] Two of the fatal suicides were those of two sisters who destroyed themselves, one about a year after the other, with wine of colchicum used as a medicine by their adoptive father.[9]

None of the 5 cases of alleged homicide by colchicum was demonstrated to be such by expert evidence which would be satisfactory at the present time. The earliest was a French case in 1832 in which a man attempted to poison his wife, but, by chance, the poisoned food was given to a visitor, who died in three days from its effects.[10] Albert[11] has reported an instance in which a woman died from the effects of colchicum seeds which another woman had induced her to take on the pretence that they would cure her of a dropsy, but with the intent apparently of causing her death. Taylor refers to the

[1] According to Hahn and Holfert (*op. cit.*, No. 697), the original French "Laville" does not contain colchicum.

[2] Five accidental deaths from "colchicum" are also included in the reports for 1871–77.

[3] Dillon, quoted by Beck: "Med. Jur.," 12th ed., ii., 891.

[4] v. Maydell: *Loc. cit.*

[5] Reg. *v.* Markuss: Med. Times and Gaz., 1864, i., 288. The defendant was acquitted.

[6] Process Flocken. Strasburg, 1887. Epagnon Dézille: Thèse, Paris, 1889 (No. 248), 38; "Neue Pitaval," 1890, n. s., xxiii.

[7] Porter and Tidy: Med. Times and Gaz., 1874, ii., 723.

[8] Fifield: Extr. Rec. Soc. Med. Impr., Boston, 1859, iii., 70. Henderson: London Med. Gaz., 1839, n. s., ii. [o. s., xxiv.], 763.

[9] Ollivier: Arch. gén. de méd., 1836, 2 s., xi., 429; Ann. d'hyg., etc., 1836, xvi., 394. Coffe: J. hebd. de progr. méd., etc., 1835, iv., 49. The journal in which the other two suicides are reported is inaccessible to us. Schmid: "Zwei Selbstvergiftungen mit tödtlichem Ausgang durch Herbstzeitlosensamen," Schweiz. Ztschr. f. Med., Ch. u. Geb., 1846, ii., 327.

[10] Chevallier: J. d. chim. méd., etc., 1832, viii., 351.

[11] Ztschr. f. Staatsarznk., 1862, lxxxiv., 202.

case of Reg. *v.* Wilson, also in 1862, in which it appears probable that at least one of the woman's victims was destroyed by colchicum.[1] Hecker[2] has published a review of the evidence in the case of a man who was found dying from a wound in the throat, and who, it was alleged, had also been poisoned by colchicum, and concludes that the evidence of poisoning was not satisfactory. A somewhat similar conclusion was reached after a careful examination in a more recent French case of alleged poisoning by colchicin.[3]

The **prognosis**, so far as can be judged from reported cases, is very unfavorable. Of 122 poisonings 90, or 73.7 per cent., were fatal.

In 52 of 71 fatal cases of which the **duration** is reported, death occurred in less than 48 hours; in 13 cases in from 2 to 7 days, and in 6 after illness of longer duration. In one group of 5 fatal poisonings the deaths occurred in from 19 to 26 hours,[4] and in another of 17 cases, 7 of which were fatal, in from 19 to 29 hours.[5] The most rapidly fatal case was one mentioned by Taylor,[6] of a man who died in 7 hours from the effects of an ounce and a half of the wine. In some instances the fatal termination has been delayed for several weeks. McPhaill[7] has reported the poisoning of 3 soldiers by wine of colchicum, 1 died in 2 days, but the other 2 survived for "a few weeks" in a condition resembling that of chronic dysentery. In Schilling's case[8] a boy of 6 years died in 50 days after having eaten a quantity of the leaves and seeds.

Lethal Dose.—Sufficient data for the determination even approximately of the minimum lethal dose of colchicin are still lacking. The smallest dose of the wine which is stated to have caused death is three and a half drachms, which quantity, given medicinally in divided doses, is said to have proved fatal on the fourth day.[9]

[1] "Pr. and Pr. of Med. Jur.," 3d ed., i., 341; "Poisons," 3d Am. ed., 493.

[2] Friedreich's Bl. f. ger. Med., 1868, xix., 321.

[3] Brouardel *et al.*: Ann. d'hyg., etc., 1886, 3 s., v., 230. See also below, "Detection."

[4] Roux: Union méd., Paris, 1855, ix., 145.

[5] Major: Med. Times and Gaz., 1874, 275.

[6] "Pr. and Pr. Med. Jur.," 3d ed., i., 340.

[7] Am. Med. Intelligencer, 1838, ii., 312.

[8] Med. Ann., Heidelberg, 1840, vi., 591.

[9] Mann: Taylor, "Poisons," 3d Am. ed., 492. No particulars of

In three instances persons have recovered after having taken about an ounce (30 c.c.) of the tincture.[1] A woman recovered from the effects of an ounce of the wine, taken during twelve hours;[2] and a man from the effects of two ounces (60 c.c.) of a tincture of the flowers.[3]

Symptoms.—The symptoms are those of intense gastro-enteritis, followed by collapse, and in fatal cases death from exhaustion. In Maydell's case[4] (the only one of fatal colchicin poisoning of which even a brief account of the symptoms has been published), the girl experienced burning pain in the stomach, and nausea in fifteen minutes after having taken the powder, and vomited in one hour. When seen by Maydell, four hours after the ingestion, she was vomiting constantly, purging, suffered severe colicky abdominal pain, and great thirst. The skin was somewhat anæsthetized, and the urine was diminished. She soon became collapsed. On the next day the pulse was filiform and the heart's action very faint. On the morning of the second day she was unconscious, cyanosed, and pulseless. Trismus occurred half an hour before death, thirty-three hours after the poisoning.

An interval of from three-quarters of an hour to two or three hours elapses between the ingestion and the first active symptoms in all cases of colchicum or colchicin poisoning. The cause of the delay is attributed by Jacobi[5] to the comparatively non-toxic quality of colchicin itself, and to its transformation in the system into a colored and actively toxic product of oxidation, which he has called oxydicolchicin, and to which he assigns the formula ($C_{22}H_{25}NO_6O_2$.)

A burning sensation is experienced in the mouth and throat which, as well as the tongue, afterward become dry, leathery or parchment-like, so that deglutition becomes difficult or impossible, and thirst is intense. Burning epigastric pain is also present and, later, severe abdominal colic and pain in the lum-

the case are given, however. See also Jacobi: Arch. f. exp. Path. u. Ph., 1890, xxvii., 124.

[1] Henderson (London Med. Gaz., 1839, n. s., ii., 763), F., 33, S. Leroy des Barres (Bull. Ac. de méd., Paris, 1847–48, xiii., 1013), F., 57, A. Warncke (*Loc. cit.*), M., 17, A. (32–40 gm. = .056–.068 colchicin).

[2] Kennard: Am. J. Med. Sc., 1857, n. s., xxxiii., 69.

[3] Kuhn: Bull. gén. de thérap., 1835, ix, 144.

[4] *Loc. cit.*

[5] Arch. f. exp. Path. u. Ph., 1890, xxvii., 129.

bar region. Nausea and retching are followed by vomiting, which is incessant, and is soon accompanied by purging of watery, rice-water discharges, containing shreds of mucous membrane and frequently blood, passed involuntarily and attended with tenesmus. The pulse is small and irregular, and later imperceptible. There is præcordial anxiety, and sometimes attacks of vertigo, or delirium. The patient becomes greatly prostrated, cold, cyanosed, and collapsed. Toward the end there is continual jactitation, with more or less violent convulsions, dilated pupils, tetanic spasms of the arms and legs, and trismus. The secretion of urine is diminished and the elimination of the poison is slow, so that the repeated administration of small doses may cause poisoning if the total quantity administered be sufficient.[1]

In protracted cases remissions of the symptoms with subsequent sudden and violent aggravation occur.[2] Or the first manifestations of violent symptoms are succeeded by a condition resembling that of chronic dysentery and terminating in death.[3] In Schilling's case[4] of death in fifty days the patient had tetanic convulsions in the night following the taking of the poison (seeds and leaves), and two days later hemiplegia, convulsions, and constant diuresis. Diabetes began twenty-four hours after the ingestion and continued until death.

Treatment.—The slowness of the action of this poison will afford opportunity for the prevention of its effects by prompt treatment, particularly when portions of the plant have been taken and its identity is known. The stomach should be washed out, as well as the large intestine, with water containing tannic acid in solution. The acid, or an astringent aqueous preparation containing it, has the advantage of forming a precipitate with colchicin which is insoluble in excess of the precipitant or in the dilute hydrochloric acid of the gastric secretion. As, however, it is soluble in alcohol (and in acetic acid) an astringent tincture should not be used. Moreover, tannic acid serves to control the dysenteric symptoms, for which purpose it may also be given in powder by the mouth, and in

[1] Budd: Lancet, 1881, i., 368 (see, however, Gould: *ibid.*, 439). Mann's case, cited above. De Mussy, Bucquoy, and Moutard-Martin: Bull. et mém. Soc. de thérap., 1868, ii., 170.

[2] Tartarin: *Loc. cit.*

[3] McPhaill: *Loc. cit.*

[4] *Loc. cit.*

enema in later stages of the poisoning. Opium is indicated to control the pain, vomiting, and purging, and warm mucilaginous drinks to stimulate the diminished secretion of urine, and thus favor elimination. Warm compresses applied to the abdomen diminish the colic. Hypodermics of stimulants are indicated in the stage of collapse; and artificial respiration may be called for by failure of that function.

Post-Mortem Appearances.—These are generally negative in character, except more or less inflammation of the intestinal mucous membrane, and sometimes that of the stomach. In Ware's case[1] the colon was swollen and inflamed throughout, with patches of lymph upon its mucous surface. Hemorrhagic stains have been observed upon the surface of the heart, and slight effusions in the heart muscle.[2] Casper noted a dark cherry-red and thick condition of the blood in his four cases.[3] In Neubrandt's case[4] the pupils were found widely dilated twenty-three hours after death, but in Ollivier's two cases[5] they were not dilated when observed five and forty-three hours respectively after death.

Analytical.—Colchicin and oxydicolchicin are extracted from acid aqueous liquids by chloroform and by amylic alcohol. When the search is particularly directed to these alkaloids the acid solution should be agitated, first with petroleum ether to remove impurities, and then repeatedly with chloroform. The residue is usually yellowish and amorphous. It may be purified by solution in water, filtration, precipitation of the alkaloid with tannic acid, decomposition of the washed precipitate by lead oxid, which is to be mixed with it, and the mixture boiled with dilute alcohol, filtered, and evaporated to recover the alkaloid. Colchicein is extracted from acid solutions by benzene.

1. The behavior of colchicin toward general reagents is given by Dragendorff[6] as follows: *Tannic acid* precipitates 0.2 mgm. in solution of 1 : 2500, the precipitate being readily soluble in acetic acid. *Auric chlorid* precipitates 0.5 mgm. 1 : 1000.

[1] Extr. Rec. Soc. Med. Impr., Boston, 1859, iii., 71.

[2] Mayer: Med. News, Phila., 1894, lxiv., 457.

[3] Vrtljschr. f. ger. Med., 1855, vii., 12. See also Roux: Union méd., 1855, ix., 145 (five cases).

[4] Med. Cor.-Bl. würt. aerztl. Ver., 1840, x., 17. Also reported by Blumhardt (see Christison: "Poisons," Am. ed., 676).

[5] *Loc. cit.*

[6] "Ermittelung von Giften," 4te Aufl., 1895, 275.

Platinic chlorid forms no precipitate in solution 1:55.[1] *Iodin in potassium iodid* precipitates 1:2500. *Bismuth potassium iodid* and *phosphomolybdic acid* precipitate 0.15 mgm. 3:10,000. The other general reagents precipitate only very concentrated or strongly acid solutions. *Chlorin water* forms a yellow precipitate in aqueous solutions of colchicin, which forms an orange-colored solution with ammonium hydroxid.

2. Concentrated sulfuric acid forms a yellow solution with colchicin (.05 mgm.) On addition of nitric acid or sodium nitrate to the solution the color changes to green, blue, violet, and, finally, to pale yellow; and if concentrated potassium hydroxid solution be then added a brick-red color is produced. If the addition of nitric acid or of nitre be postponed for eighteen hours the yellow color changes to red-violet, blue, and, finally, nut-brown.

3. Nitric acid (sp. gr. 1.4) forms a violet solution which changes to green and then to yellow (0.2 mgm.). The yellow solution turns red on addition of potash solution. Fuming nitric acid colors colchicin violet to indigo.

4. Ferric chlorid colors colchicin solutions dark green. If a hydrochloric-acid solution of the alkaloid be boiled with ferric chlorid it is colored greenish-black; and chloroform agitated with this is colored garnet-red or brown.

5. Erdmann's reagent forms a blue solution.

6. Sulfovanadic acid forms a bluish-green solution.

7. Fröhde's reagent forms a yellow solution, changing to yellowish-green and back to yellow in twenty-four hours.

Colchicein and oxydicolchicin give the same color reactions.

Experiments with colchicin upon animals have not shown that it produces effects which are sufficiently characteristic, nor that any species experimented upon is sufficiently sensitive to its action for the purposes of a life test.

Baumert[2] obtained a putrid product, probably a peptone, from a cadaver twenty-two months after death which resembled colchicin in that it passed from acid solutions into chloroform and into amylic alcohol (and also into benzene), which left it as a yellow residue, and in its behavior toward the general reagents, but which did not give the reaction with ferric chlorid,

[1] A precipitate is formed after twenty-four hours in 1 : 3000.

[2] Arch. d. Pharm., 1887, ccxxv., 911.

and also differed from colchicin in forming precipitates with platinic chlorid and with picric acid. Liebermann[1] also obtained a putrid product which passed into ether from acid as well as from alkaline liquids, and which was yellow and amorphous. It differed from colchicin, however, in giving white precipitates with chlorin water, with mercuric chlorid, and with potassium iodhydrargyrate; in giving a reddish-violet color after standing with sulfuric acid, and in failing to give the nitric-acid reaction. Indeed, Liebermann considered this ptomain as resembling coniin, except in that it was not volatile and had a different odor.

In view of the small number of the not very characteristic reactions of colchicin the analyst would not at present be warranted under any circumstances in expressing a stronger opinion than that of the experts in the Affaire R—— :[2] that the results are in accordance with the hypothesis of colchicin poisoning, but that they do not demonstrate that such hypothesis is correct.

According to Oboloonski[3] colchicin is not readily decomposed by contact with putrefying material.

CONIIN AND CONIUM.

Hemlock (*Conium maculatum*) is a native of Europe, but is also common in the older portions of the United States, where it grows on roadsides and in waste places. Its toxic character was recognized by the ancients, and its juice or an infusion of the leaves was used by the Athenians (κώνειον) as a judicial poison. Hemlock was administered to Socrates, Phocion, and Theramenes.[4]

Five alkaloids have been obtained from conium.

Coniin—*Conicin*, *Cicutin*—is, chemically, *alpha-propyl-piperidin*, $C_5H_9(C_3H_7\alpha)NH$, and is the most simply constituted of the vegetable alkaloids, as well as the earliest to be obtained by synthesis.[5] The natural alkaloid was first obtained by Giesecke in 1827.[6] It is a colorless liquid, having a burning

[1] Berichte, Berlin, 1876, ix., 151.

[2] Ann. d'hyg., etc., 1886, 3 s., xv., 262, 278.

[3] Chem. Centbl., 1888, 3 R., xix., 429.

[4] For a discussion of the identity of hemlock and the Athenian poison see Imbert-Gourbeyre: "De la mort de Socrate," etc., Paris, 1875.

[5] Ladenburg: Berichte, Berlin, 1884, xvii., 772, 1121, 1676; 1885, xxviii., 47, 913, 1587, 2961.

[6] Arch. d. Pharm., xx., 97.

taste, and a penetrating, disagreeable odor, sp. gr. 0.878 to 0.886. Volatile to some extent at ordinary temperatures, it distils with alcohol or water. Its boiling point is variously stated between 150° and 212° (302°–414° F.). It dissolves in 100 parts of cold water and is less soluble in hot water, soluble in all proportions in alcohol, and in 6 parts of ether; and is also dissolved by chloroform, benzene, petroleum ether, amylic alcohol, acetone, and ethereal oils. Its solutions are strongly alkaline and dextrogyrous $[\alpha]_D = +13.8$. With acids it forms crystalline salts, soluble in water and in alcohol, which are partly decomposed, with loss of coniin, on evaporation of the solutions. Its vapor produces a white cloud with acid vapors or gases. The free base on exposure to air becomes oxidized and resinous. It exists in the greatest proportion (0.05 to 0.09 per cent.) in the leaves at the time of flowering, and in the seed when almost ripe (0.7 per cent.).

Conhydrin—derived from coniin by substitution of OH for H in one of the remaining alpha positions, $C_5H_8(OH,C_3H_7\ \alpha)$-NH—exists in very small proportion in hemlock, from which it was obtained by Wertheim in 1856.[1] It separates from commercial coniin when this is cooled to 5° (41° F.). It crystallizes in pearly plates, which fuse at 126°.6 (260° F.) to a liquid which boils at 226°.3 (439° F.). The solid also sublimes in part at 100° (212° F.). It volatilizes without decomposition. Its odor resembles that of coniin. It is soluble in water, readily soluble in alcohol and in ether, and somewhat soluble in petroleum ether. It is strongly alkaline and dextrogyrous. It is highly poisonous.

Pseudo-conhydrin—$C_8H_{17}NO$—isomeric with conhydrin, a base existing in conium in small quantity, discovered by Merck,[2] is a light, white, crystalline solid, having the odor of conhydrin, readily soluble in alcohol, ether, and chloroform. It fuses at 100°–102° (212°–215°.6 F.) and boils at 229°–231° (444°.2–448° F.), is dextrogyrous $[\alpha]_D = +4°.3$, and is a strong base, having an alkaline reaction.

Methyl coniin—$C_5H_9(C_3H_7)N,CH_3$—and **ethyl piperidin**—$C_5H_{10}N,C_2H_5$—are volatile, oily, alkaline bases, lighter than water, which accompany coniin in small quantity.[3]

[1] Ann. d. Ch. u. Ph., c., 329; cxxiii., 157; cxxvii., 75; cxxx., 269.

[2] Ladenburg and Adam: Berichte, Berlin, 1891, xxiv., 1671.

[3] For the physiological action of

Statistics.—In recent times coniin or conium poisonings are of rare occurrence. We have collated the reports of 33 cases from medical and pharmaceutical literature.[1] Almost all were accidental, many caused by persons eating the root in mistake for parsnips, etc., or the leaves. In one instance 50 persons are said to have died from the use of hemlock in the kitchen in mistake for parsley.[2] Poisonings have also resulted from the accidental mixing of hemlock seeds with those of anise;[3] and 3 children are said to have been poisoned, 1 fatally, by blowing whistles made of hemlock stems.[4] Only 6 cases, 3 fatal, were caused by the tincture or fluid extract taken in overdose.[5] In one instance a man of 22 years was severely poisoned by inhaling the vapor of coniin in small amount.[6] Although Schauenstein[7] refers to 2 suicides by conium, we find no record of an instance of intentional self-destruction by this poison. One of the 2 cases here referred to is that of Walker, who died in New York in 1875 from the effects of 150 minims of Squibb's fluid extract, taken in three doses during an hour. But this case, frequently referred to as suicidal, was due to a misunderstanding of the physician's directions.[8] Four alleged homicides by this poison have been investigated. In Reg. *v.* Bowyer, in 1848, a woman was accused of the murder of her child by a decoction of hemlock, but acquitted from lack of evidence.[9] Mitscherlich and Casper[10] reviewed the evidence as to the cause of the death of a man alleged to have been poisoned by conium, reaching the

methyl-coniin see Brown and Fraser: Tr. roy. Soc., Edinb., 1869, xxv., 893. Hope: Diss., Kiel, 1893.

[1] Not including Oria y Cruz: Anfiteatro anat., Madrid, 1878, vi., 103, and Reichardt: Jenaisch. Ztsch. f. Med., etc., 1865, ii., 340, which are inaccessible to us.

[2] Med. Times and Gaz., 1855, ii., 51.

[3] Galtier: "Tox.," ii., 206. Schauenstein: Maschka's "Handb. d. ger. Med.," ii., 574.

[4] Lancet, 1851, ii., 276.

[5] Stillé ("Mat. Med.," ii., 268) (Hunter), M., ad., ʒx. extract, D. two hours. Fountain (Am. J. M. Sc., 1846, n. s., xi., 123), M., ad., gr. xij., extract. R. Pereira ("Mat. Med.," ii., 732), M., ad., "overdose," R. Pepper (Med.-leg. J., N. Y., 1885–86, iii., 179), eight months, gr. v., extract, D. seven hours. Langdon (Cincin. M. and Dent. J., 1885–86., i., 138), F., 35, about ʒ vij. tinct., R.; the Walker case below.

[6] Schulz: Deut. med. Wochenschr., 1887, xiii., 495.

[7] *Op. cit.*, p. 573.

[8] Wharton and Stillé: "Med. Jur.," 4th ed., ii., 613. Bell: Sanitarian, 1875, iii., 117, and Tr. Am. Med. Assoc., 1875, xxvi., 345; Med. Times and Gaz., 1875, i., 473.

[9] London Med. Times, 1848, xviii., 244. Taylor: "Poisons," 3d Am. ed., 700.

[10] Vrtljschr. f. ger. Med., 1859, xv., 193.

conclusions that death was due to mechanical suffocation, and that the facts were insufficient to constitute positive proof that poisoning by conium had also been attempted. The same negative conclusion as to proof of hemlock poisoning as the cause of death was reached in the case of a man of seventy years who was alleged to have been destroyed by conium, administered in soup by his son.[1] In 1861 Louise Berger died in a few minutes from the effects of ten to fifteen drops of coniin, administered by her lover, Dr. Jahn.[2]

Lethal Dose—Duration—Prognosis.—The minimum lethal dose of pure coniin for the human subject is undetermined. Probably that supposed to have caused death in a few minutes in Dr. Jahn's case, ten to fifteen drops (about 0.42 to 0.63 gm.)[3] was greatly in excess of that sufficient to cause death, and probably 0.15 gm. might cause the death of a human adult. The experiments of Schroff[4] made upon twenty-seven physicians showed 0.003 to 0.085 gm. of the pure alkaloid, dissolved in alcohol, to constitute a toxic dose. Van Praag[5] states the minimum lethal dose for cats and dogs as 0.122 gm., for rabbits 0.0163 gm., for small birds 0.243 gm., for frogs 0.004 gm., and for fishes 0.0732 gm. Ihmsen[6] found that thirty drops (1.26 gm.) given to a horse by the mouth remained without effect, and that five drops (0.2 gm.) injected into the jugular, although a toxic dose, was not lethal. Stienhäuslin[7] found the lethal dose for rabbits to be 45.75 mgm. per kilogram. Martens[8] found the minimum lethal dose for white mice to be 1.54 mgm. (75.05 mgm. per kilogram), and for pigeons 11.4 mgm. (40.3 mgm. per kilogram). The investigations of Brown and Frazer,[9] and of Tiryakian[10] have shown that commercial samples of coniin differ materially in their activity with the degree to which they have become altered by exposure to air.

The parts of the plant and the pharmaceutical preparations

[1] Hofmann: Vrtljschr. f. ger. Med., 1870, n. F., xiii., 1.

[2] Arch. d. Pharm., 1861, 2 R., cvii., 257, 360. Dupuy: "Alcaloides," i., 465. Husemann u. Hilger: "Pflanzenstoffe," 2te Aufl., 923.

[3] Schroff gives the weight of a drop of coniin as 0.042 gm.

[4] Wochenbl. d. k. k. Gesellsch. d. Aerzte in Wien, 1856, ii., 33, 49, 73, 102.

[5] Nederl. Lancet, 1855, 3 s., iv., 672.

[6] Husemann u. Hilger: "Pflanzenstoffe," 2te Aufl., 921.

[7] Diss., Berne, 1887.

[8] Diss., Kiel, 1893.

[9] Tr. Roy. Soc., Edinb., 1869, xxv., 893.

[10] Thèse, Paris, 1878.

made therefrom vary still more in strength according to the season at which the plant is gathered and their degree of freshness.

The **duration** of a fatal poisoning by coniin is compassed within a few minutes; and even when death is caused by the leaves, or by a decoction, or by the fluid extract the duration of the poisoning has only exceeded three hours in two instances: a man of forty-three years died in three and a half hours after eating a quantity of the leaves;[1] and in Pepper's case[2] a child died in seven hours from the effects of five grains of the extract. An exceptional case is reported by Armstrong[3] of a woman of twenty years who is said to have died in fifty-two hours from the effects of the root of Conium maculatum. The case was, however, peculiar in other respects, and so far as the imperfect report indicates, the symptoms were not those of conium poisoning, but rather those of a cicuta poisoning of prolonged duration.

The **prognosis** will depend upon the quantity of coniin absorbed, and consequently with the amount of the alkaloid present in the plant or preparation swallowed, and with the length of time it is allowed to remain in the stomach. In cases not rapidly fatal the condition of the respiration is the best indication of the probable result. Owing to the imperfect character of many of the reports and the uncertainty of the quantities of coniin present, an attempt to indicate the fatality by a comparison of the numbers of deaths and recoveries in reported cases would be misleading.

Symptoms.—The action of coniin is that of a powerful paralyzant. A burning sensation in the mouth, with a sense of scratching in the throat, and dizziness may be perceived upon, or shortly after, swallowing the poison, but the first noticeable effect is a prickling sensation and loss of power in the lower extremities, the paralysis beginning at the feet and extending upward. Dr. Earle, however, who was on horseback, perceived the loss of power first in the upper extremities.[4] The person staggers as if intoxicated on attempting to walk, or falls. Consciousness is, however, perfect, and the intellect re-

[1] Bennett: Edinb. M. and S. Jour., 1845, lxiv., 169.
[2] *Loc. cit.*
[3] Tr. M. Soc. N. Jersey, 1880, cxiv., 249.
[4] Fountain: *Loc. cit.*

mains clear even after the powers of motion and of speech are lost. There is some nausea, but no vomiting. Frontal headache, and a peculiar sensation as if of an obstruction between the eyelids are experienced. The speech becomes thick and finally lost, and deglutition difficult, then impossible. The pupils are dilated, and the pulse and temperature are normal until toward the end. The arms become paralyzed, and the paralysis extends to the muscles of respiration, causing death by asphyxia.

The cases frequently quoted from Orfila of two persons who manifested the symptoms of atropic poisoning, and whose poisoning is ascribed to conium, were probably caused by hyoscyamus.[1]

Treatment.—This should consist in the removal of unabsorbed poison by mechanical means or by the administration of emetics and purgatives; and in the performance of artificial respiration when the natural function fails.

Post-Mortem Appearances.—These are not characteristic, and are mainly those found after death from asphyxia by other causes: hyperæmia of the brain, meninges, and lungs, dark, difficultly coagulating blood, which becomes brighter on exposure to air, and sometimes œdema of the lungs. The gastric mucous membrane is somewhat reddened, and, after death from coniin, may be ecchymosed.

Detection.—Coniin is abstracted from alkaline aqueous liquids by petroleum ether, and will consequently be found in residue V. in the systematic process for alkaloids (see p. 136). In the process for volatile poisons it will also appear in the distillate from alkaline solution. Indeed, the best method for the separation of the alkaloid consists in adding magnesia and distilling nearly to dryness. The distillate is neutralized with oxalic acid and evaporated to dryness, and the residue extracted with alcohol, which dissolves conium oxalate, but not ammonium oxalate. After filtration the alcohol is evaporated, and the alkaloid liberated with caustic soda. The free alkaloid presents the following reactions:

[1] These cases, cited as conium poisonings by most writers (as late as Kobert, 1893), are quoted by Orfila ("Tox. gèn.," 5ème ed., ii., 539) from Vicat ("Hist. d. plantes vénéneuses de la Suisse," 1776, p. 274), who in turn took them from Matthiolus (Lib. vi., cap., xi., ed. Venet., 1554, p. 663), who says that the root was cooked in mistake for

1. It is oily (oily drops or streaks), has a peculiar odor, resembling that of the urine of mice, is alkaline in reaction, and reddens phenolphthalein. According to Heut,[1] coniin and nicotin may be distinguished from each other by the fact that an alcoholic solution of the former reddens an alcoholic solution of phenolphthalein, while an alcoholic solution of the latter does not.

2. A strong cold solution of the alkaloid in water becomes cloudy when heated, and clears up on cooling.

3. An aqueous solution of the alkaloid is rendered milky by chlorin water. Nicotin does not behave similarly with 2 and 3.

4. A glass rod, moistened with hydrochloric acid, when approached to the alkaloid, is surrounded by a white cloud, while the coniin becomes converted into a crystalline mass, which, with a magnifying power of 180–250 is seen to consist of doubly refracting needles arranged in stellate bundles or in dendritic forms, or irregularly interlaced. After a time yellowish amorphous masses and cubical or octahedral crystals, having no action on polarized light, and sometimes arranged in cross or dagger shapes, are produced from the needles (Erhard).

5. The bromid also forms similar crystals, which may be produced by placing a watch-glass containing bromin water over one containing an ethereal solution of the alkaloid, and covering both with an inverted beaker. A similar crystallization is obtained if a solution of bromin in hydrobromic acid be used in place of bromin water.[2] Nicotin does not form crystals under these conditions.

6. If an aqueous solution of coniin containing sulfuric acid be evaporated at the ordinary temperature, needle-shaped crystals are first formed, and later large crystalline plates. Similar crystals are produced by the alkaloid with phosphoric acid.[3]

7. Dragendorff[4] gives the behavior of coniin in sulfuric aqueous solution toward the general precipitants as follows: with *potassium bismuth iodid*, an orange-red precipitate up to 1 : 6000; *phosphomolybdic acid*, a yellowish precipitate 1 : 1000;

parsnips. Hyoscyamus has been frequently eaten in consequence of a like mistake.

[1] Arch. d. Pharm., 1893, ccxxxi., 376.

[2] Wormley: "Micro-Chem. of Poisons," 2 ed., 461.

[3] Erhard: "Die giftig. Alkaloide," etc., Pl. i., f. 4; Pl. ii., f. 1.

[4] "Ermitt. von Giften," 4te Aufl., 284.

potassium mercury iodid, a cloudiness 1:800; *potassium cadmium iodid*, slight cloud 1:800; *iodo-potassium iodid*, kermes-colored precipitate 1:10,000.

Auric and *platinic chlorids*, which precipitate nicotin in dilutions of 1:10,000 and 1:5000 respectively, do not precipitate coniin in solutions more dilute than 1:100.[1]

8. Color reagents for the most part give negative results. According to Sohn,[2] fuming nitric acid colors coniin bluish, changing to orange, and dry hydrochloric acid gas forms a purple-red color, changing to indigo.[3] According to Kundrát,[4] sulfovanadic acid gives an intense green color, gradually becoming brownish.

9. When injected into the hind leg of a frog coniin, or its salts, produces paralysis of that member, which becomes limp and is dragged around by the animal in moving. Injected into animals in larger amount it produces paralysis, beginning with the posterior extremities, very much like that caused by curare.[5]

The different species of *Lupine* (*Lupinus albus*, *luteus*, etc.), whose seeds, after suitable preparation, are used as food in the countries bordering the Mediterranean, have been found by Baumert to contain at least two alkaloids, one of which, **lupinidin**, $C_8H_{15}N$, resembles coniin in being liquid, oily, and volatile, in its odor, in being more soluble in cold than in hot water, and in its physiological action. Its *platinochlorid* is, however, insoluble in water.[6] According to the more recent investigation of Soldaini,[7] *lupinus albus* contains two alkaloids, one solid and crystalline, the other oily, both having the formula $C_{15}H_{24}N_2O$. He makes no mention of an alkaloid having the characters of lupinidin, unless a product of the action of alkalies upon his fluid alkaloid, to whose chloraurate he assigns the formula $C_{15}H_{22}N_2$. $HCl.AuCl_3$, be such.

[1] See also Zalewski: Diss., Dorpat, 1869.

[2] "Dictionary of Active Principles of Plants," 43.

[3] Dragendorff does not mention the nitric-acid reaction, and considers that with hydrochloric acid to be due to impurities ("Ermitt. von Giften," 4te Aufl., 284).

[4] Ztschr. f. an. Chem., 1889, xxviii., 710.

[5] See Dupuy: "Alcaloides," i., 449. Blyth: "Poisons," 253.

[6] Baumert: Ann. d. Ch. u. Ph., 1884, ccxxiv., 321; ccxv., 365. Loewenthal: Diss., Königsberg, 1888. Husemann: Ph. Ztg., 1890, 611.

[7] Arch. d. Pharm., 1893, 3 R., xliii., 321, 481.

Coniin-like Ptomains.—Volatile, liquid, alkaline bases, some of which have a more or less distinct mousey odor, are frequently produced during putrefaction, and in the present condition of our knowledge the analyst will not be warranted in asserting the existence of coniin in putrefying material from the affirmative results of the reactions above mentioned alone, and it must also be remembered that similar substances may be introduced into the living body in decomposed food articles.[1] Most of the liquid putrid products have been identified as monamins (trimethylamin, ethylamin, etc., or diamins (cadaverin, putrescin, etc.), which are extracted by petroleum ether or pass over on distillation from alkaline solutions. These bases have not the physiological action of coniin, however.

Many of the volatile ptomaïns obtained by Selmi were in all probability amins, but some of them presented such similarity to coniin in odor, volatility, and alkalinity that Selmi expressed the opinion that coniin itself is produced during putrefaction.[2] Whether this product had a physiological action resembling that of coniin or not was not determined. Schwanert[3] had in 1874 obtained a volatile, alkaline base from putrefying material, which had the odor of propylamin and was probably a monamin. He remarks that "from its ready volatility and its peculiar odor it was not coniin or nicotin." In a noteworthy criminal case in Germany in 1874 (Process Brandes-Krebs), Otto showed that the liquid, volatile, alkaline base, separated from the cadaver and said by two analysts to be coniin, could not be either that alkaloid or nicotin, and was not identical with any vegetable base known to him. While presenting many points of resemblance to coniin and to nicotin it had not the odor of coniin, was intensely bitter and highly poisonous, and its aqueous solution did not cloud on heating. It resembled nicotin in that it formed precipitates with platinic, auric, and mercuric chlorids, but differed from it in the crystalline and doubly refractive characters of its chlorid, and in failing to give the Roussin reaction.[4] In 1878 Brouardel and

[1] See Brouardel and Boutmy's case below.

[2] "Sulle Ptomaine," Bologna, 1878, 22–30.

[3] Berichte, Berlin, 1874, vii., 1332.

[4] "Ausmittelung d. Gifte," 6te Aufl., 2te Abd., 1892, 93–95.

Boutmy [1] obtained from the cadaver of a woman who had died from the effects of eating a portion of a decomposed stuffed goose, as well as from the remains of the food, a liquid, alkaline base which had the odor of coniin, and bore some resemblance to that alkaloid in its behavior toward the general reagents, but which differed from it in its action upon frogs. They attributed the death to ptomaïn poisoning. Otto [2] also refers to an instance in which Sonnenschein mistook a volatile putrid alkaloid for coniin in a case of poisoning by water hemlock (*Cicuta virosa*). The body had been buried three months.

OTHER POISONOUS UMBELLIFEROUS PLANTS.

Water Hemlock (Cicuta Virosa, vel Aquatica).

The water hemlock, or cowbane, which grows abundantly in the north of continental Europe and less commonly in the south of England, has been frequently confounded with conium, which it resembles in appearance, but from which it differs markedly in its physiological action. Early medical writers refer frequently to poisonings caused by the root having been eaten in mistake for parsnips or celery root, or by the herb having been used in the kitchen in mistake for parsley,[3] and one of the earliest of toxicological monographs is that of Wepfer, relating to the poisoning of eight children by the root.[4] Similar accidents are of occasional occurrence at the present time, although they are rarely reported in medical literature.[5] In the United States similar accidental poisonings by the closely related *Cicuta maculata*, which is widely known as *water hemlock*, *spotted cowbane*, *beaver poison*, or *musquash root*,[6]

[1] Ann. d'hyg., etc., 1880, 3 s., iv., 352.

[2] *Op. cit.*, 96.

[3] See Wibmer: "Wirkung," etc., ii., 113.

[4] "Cicutæ aquaticæ historia et noxa," Basilæ, 1679; *ibid.*, 1713; Lugd. Bat., 1733; Venet., 1759.

[5] Ph. J. and Tr., 1872, 3 s., ii., 1063. Lender: Vrtljschr. f. ger. Med., 1865, n. F., iii., 126. Herzog: Deut. Ztschr. f. d. Staatsarznk., 1868, n. F., xxvi., 92. Kelp: Vrtljschr. f. d. ger. Med., 1879, n. F., xxx., 380. Breternitz: Berl. kl. Wochenschr., 1885, xxii., 540. Kobert: "Intoxikationen," 629. Lüdtke: Arch. d. Pharm., 1893, ccxxxi., 34.

[6] Stockbridge: New England J. M. and S., 1814, iii., 334. Hazeltine: *Ibid.*, 1818, vii., 219. Truesdale: Brit. Am. J., 1862, iii., 37. Dupuis: Canada M. J., 1865, i., 416. Matchett: Cincin. Lancet and Obs., 1870, n. s., xiii., 462. Folk: Tr. S. Car. M. Assoc., 1882, xxxii., 69. Armstrong: Tr. M. Soc. N. Jersey, 1880, cxiv., 249 (reported as conium).

are not of unusual occurrence. Two homicidal poisonings by cicuta virosa have been subjects of investigation in Germany. In one a young woman of seventeen years was convicted of the murder of her husband, aged sixty, by this plant, administered partly in coffee, and partly mixed with parsley.[1] The other was an accusation of poisoning by cicuta referred to by Otto.[2] A poisoning by the herb, purchased from an herbalist, by a man of fifty-five years, reported by Caillard,[3] appears to have been the result of an attempt at suicide.

Cicuta virosa contains no coniin;[4] but owes its toxic action to a neutral, resinous body, first separated by Boehm,[5] and called by him *cicutoxin*, which is amorphous, gummy, light yellow, acid in reaction, rather soluble in hot water and in dilute alkaline solutions, soluble in alcohol, ether, and chloroform, but not soluble in petroleum ether, which precipitates it from its ethereal solution. Nor is coniin or another volatile alkaloid present in the American Cicuta maculata, whose poisonous action, similar to that of Cicuta virosa, is probably due to the action of a substance similar to or identical with cicutoxin.[6]

The **symptoms** of poisoning by cicuta resemble those caused by *Œnanthe crocata* (see below) more closely than they do those of conium poisoning. The onset is sometimes rapid, sometimes delayed; there are nausea, vomiting, colicky pains, staggering, palpitation, and loss of consciousness, with, at first, ptosis, dropping of the lower jaw, and pale face and lips. Soon there are twitchings of the facial muscles; the patient becomes uneasy, the respiration more accelerated; the paleness gives place to cyanosis, and the patient is attacked with the most violent convulsions, at first tonic in character, becoming clonic, of long duration (one to five minutes), during which there is marked opisthotonos of the head, neck, and trunk, and the legs are drawn up; there is gnashing of the teeth, and the lips are covered with a reddish-brown froth. During the spasm the fæces

[1] Friedreich's Bl. f. ger. Med., 1850, i., Hft. 1, 39-68.

[2] "Ausmitt. d. Gifte," 6te Aufl., 96.

[3] Clin. d. hôp., 1829, iv., 33.

[4] Dragendorff: "Ermitt. von Giften," 4te Aufl., 279. Otto: *Op. cit.*, 71. Boehm: Arch. f. exp. Path. u. Ph., 1876, v., 279. Van Ankum: J. f. prakt. Chem., 1868, cv., 151.

[5] *Loc. cit.* See also Wikszemski: Diss., Dorpat, 1875. Pohl: Arch. f. exp. Path. u. Ph., 1894, xxxiv., 265.

[6] Blacksmann: Amer. J. Pharm., 1893, 4; Pharm. Ztg., 1893, 148. See also Maisch: Am. J. Ph., 1891, 328.

are passed involuntarily, and the urine is expelled in a strong stream; and at its height the respiration, and apparently the heart also, is completely arrested. During the intervals, which are of about ten minutes' duration, the face is pale, the saliva flows from the angles of the mouth, the extremities are cold, though the rectal temperature is normal, the respiration is accelerated and stertorous; the heart's action is irregular, with pauses after every second, third, or fourth beat; the radial pulse is imperceptible, the pupils are widely dilated,[1] the corneal reflex is absent and the patient is entirely unconscious. In fatal cases death occurs during a spasm, from asphyxia, or the severity of the spasms abates, and the patient remains unconscious and expires in coma. In non-fatal cases recovery is rapid after cessation of the convulsions.

The **treatment** of cicuta poisoning should consist in the evacuation and washing out of the stomach, whether the patient has vomited or not, and even if considerable time has elapsed since the poison was swallowed. If emetics be given, apomorphin is to be preferred to those given by the mouth. To control the convulsions, chloral hydrate should be resorted to if possible, and chloroform and ether by inhalation.

The **post-mortem appearances** are for the most part negative. There are evidences of gastric irritation, and sometimes ecchymoses are produced in various organs during the spasms. If death have occurred from asphyxia the usual evidences of that mode of death will be present.

The chemical **detection** of cicutoxin is, in the present condition of our knowledge, impossible. In cases of death from portions of the plant, evidences of value may be obtained from the botanic character of portions remaining in the stomach;[2] or the action of cicutoxin upon frogs may be utilized for its identification under favorable conditions. Animals recently captured are more sensitive than those which have been kept in confinement. The action of cicutoxin is divided into three periods: 1st, A period of latency, during which the animal assumes a peculiar position; the hind legs are spread out, with the legs making a right angle to the thighs and the toes spread

[1] In one of Dupuis' cases the pupils were said to be contracted during the spasm, and dilated during the interval (Canada M. J., 1865, i., 416).

[2] Lüdtke: Arch. d. Ph., 1893, ccxxxi., 34.

wide apart. The animal moves only when strongly irritated, and then walks with legs wide apart. 2d, The animal becomes very uneasy, with accelerated respiration, then for a few minutes there are rapidly successive extensions and flexions of the hind legs, a few strong jumps, accompanied by shrill cries, and powerful tetanic convulsions, with opisthotonos or emprosthotonos. 3d, After some clonic convulsions the animal remains quiet and apparently exhausted, and when irritated moves slowly, with limbs stiff and widely spread out.[1]

Œnanthe Crocata.

The *hemlock water-dropwort*, *five-finger root*, or *dead tongue* (Œnanthe crocata) grows abundantly near water in the south of England and in France and Holland, where it has frequently caused poisoning, notably in sailors from Mediterranean ports, who have eaten the root in mistake for wild celery, which grows in similar situations in those parts. Frequently several individuals have partaken of the root and have all been more or less severely poisoned. Thus we have collated the reports of 26 poisonings in more than 120 individuals, of which 19 were multiple.[2] Another European species, *Œnanthe fistulosa*, is poisonous, and its expressed juice was the agent employed in one of the two recorded instances of homicidal œnanthe poisoning, in which a gardener destroyed a rival by the administration of some ounces of the juice in wine.[3] A woman was convicted of an attempt to murder her husband by preparing for him a soup containing slices of the root.[4]

[1] See Böhm: Arch. f. exp. Path. u. Ph., 1875, iii., 224, 1876, v., 289. Wikszemski: Diss., Dorpat, 1875. Compare with nicotin.

[2] Wibmer: "Wirkung," etc., iv., 3; 2 men, both died (Vanderwiel); 3 French prisoners, 2 died; 36 soldiers, 1 died (Rochard); a family, all recovered (Charles); 3 soldiers, all died (Dunal); 3 sailors, 1 died; 5 soldiers, 3 died (Reveillé-Parise). Bossey (London M. Gaz., 1844, xxxiv., 288), 21 convicts, 6 died. Taylor ("Pr. and Pr. M. Jur.," 3d ed., i., 450), 2 men, both died. Nevins (Assoc. M. J., 1853, 1069), 4 children, 1 died. Grahame (Med. Times and Gaz., 1858, xvi., 241), several persons, 1 died. Appleton (Guppy) (Brit. M. J., 1861, i., 293), 4 sailors, 2 died. Baume (Med. Times and Gaz., 1862, ii., 263), 8 persons, 1 died. Bloc (Montpelier méd., 1872, 343), several ladies, 2 died. Foss (Practit., London, 1876, xvii., 248), 9 children, 3 died. Bampton (Lancet, 1881, i., 823), four instances, sailors: 27, 4 died; a crew, 5 died; 8, 2 died; and 3, 1 died.

[3] Friedreich's Bl. f. ger. Med., 1856, vii., 5 Hft., 77.

[4] Toulmouche: Gaz. méd. de Paris, 1846, i., 18.

Gerding in 1848[1] obtained from *Œnanthe fistulosa* a resinoid substance to which he gave the name *œnanthin*, which was probably a less perfectly purified form of the *œnanthotoxin* obtained by Pohl from *Œnanthe crocata*,[2] which is a dark-brown, semi-solid resin, soluble in methylic, ethylic, and amylic alcohols, chloroform, ether, and benzene, but insoluble in petroleum ether, water, or cold, dilute acids or alkalies, a few milligrams of which cause severe spasms and death in frogs, and similar manifestations in rabbits in doses of 0.02 gm.

The **symptoms** of œnanthe poisoning as described by Bampton in his account of three cases of varying intensity[3] are as follows: All three continued their usual avocation without inconvenience for from three-quarters of an hour to an hour. One man experienced a burning sensation in the nose from constant eructation and spitting of thick stuff, the limbs became weak, and there were muscular contractions, with tremors in the limbs and joints. He drank some rum, after which he felt better and experienced no further inconvenience. The second man became giddy and cold, had tremors in the limbs, could not see, although he could hear, and was unconscious for half an hour. On regaining consciousness he suffered from pain in the head, and a twisting, grinding pain in the stomach. A thick phlegm accumulated at the back of the throat. He did not vomit until melted butter was given him, two hours after the ingestion. Three hours later the pupils were normal, the conjunctivæ highly injected, the pulse fair, and the extremities cold. He moaned and expectorated constantly. The next day he was drowsy and suffered no pain except in the eyeballs; the corneæ were dull, the conjunctivæ injected, and objects appeared less than their natural size. During the next two days he expectorated rusty, viscid sputa, and was discharged after three days. The third man, while walking, fell as if shot, with sufficient violence to cause a scalp wound, beneath which, however, there was no injury to the skull. He cried out as he fell, immediately vomited a quantity of green stuff, and became green in the face. A quantity of thick mucus produced foam at the mouth, and the man was unconscious and unable to

[1] J. f. prakt. Chem., 1848, xliv., 175.

[2] Arch. f. exp. Path. u. Ph., 1894, xxxiv., 259.

[3] Lancet, 1881, i., 823.

swallow. The hands were clenched, and there were spasmodic contractions of the muscles of the legs and face. When seen by Bampton, five hours after the ingestion, he was unconscious, with widely dilated pupils, the pulse perceptible, and the respiration slow and impeded by the accumulation of thick mucus, which filled the mouth and throat and covered the beard. The muscular system was relaxed, with occasional spasmodic twitchings. He died fifteen minutes later of asphyxia. In other reported cases the convulsions appear to have been more violent.

The most noteworthy **post-mortem appearance** is the accumulation of thick, viscid mucus which fills not only the pharynx and larynx but also the finer bronchi.

Æthusa Cynapium.

It is doubtful whether this plant (fool's parsley) is poisonous. Thomas[1] relates the poisoning of three children, one fatally, said to have been caused by their eating the bulbs in mistake for turnips. Gmelin[2] refers to the death of a boy of six years who died after having eaten the plant in mistake for parsley. Stevenson[3] attributes symptoms of poisoning in two ladies to their having eaten fool's parsley in a salad. Tott[4] practically supposes that the non-fatal poisoning of a woman was caused by this plant. On the other hand, Harley[5] concludes from his experiments and observations that this plant is absolutely free from the noxious properties attributed to it.

Sium Latifolium.

The common *water-parsnip*, which grows in Europe and the United States, as well as other species of the same genus, *S. lineare*, *Carsonii*, *angustifolium*, are poisonous. Little[6] has reported the fatal poisoning of a boy of sixteen years who ate a piece of the root of the size of a large almond. In one hour he

[1] London Med. Times, 1845, xii., 408.

[2] "Allg. Gesch. d. Pflanzengifte," 1803, 571.

[3] London M. and Phys. J., 1833, xiv., 425.

[4] Jahrb. d. ges. Staatsarznk., 1839, v., 183.

[5] St. Thomas Hosp. Rep., 1879, n. s., x., 257.

[6] Clinic, Cincin., 1874, vii., 49.

experienced nausea, vomited, and had a convulsion, after which he never spoke nor showed any signs of consciousness. When seen by Little he had had three convulsions of about three minutes' duration each, was wet with perspiration, the head hot, the body cold, the pulse 60, full and strong, the respiration stertorous, the pupils dilated and responding slowly to light. There were feeble efforts of deglutition on touching the fauces, and the muscles were in tonic contraction with the arms drawn toward the middle of the body. Spasms succeeded each other every few minutes, at first violently clonic, diminishing in violence with each convulsion, until they became mere tremors. The last spasm, the twelfth, was purely tonic, and during its continuance the pulse and respiration ceased and the patient died, two and a half hours after the ingestion. Two other boys who merely masticated the root without swallowing it suffered from sore mouth for four days.

Phillips[1] has reported the poisoning of three children, one fatally, apparently by *Sium aquaticum*, in which the symptoms were similar to those described above.

GELSEMIUM—GELSEMIN—GELSEMININ.

That the root of the *yellow* or *Carolina jessamine* (or *jasmine*)[2] yielded to alcohol a principle having medicinal virtues was accidentally discovered by a Mississippi planter. A few years later (1852) Proctor, who obtained most of his information from "eclectic" publications, published an account of its therapeutic effects.[3] In 1855 Kollock separated an alkaloid in a partially purified condition, which he called *gelseminia*.[4] In 1870 Wormley[5] obtained from gelsemium an acid, *gelsemic acid*, which is non-poisonous, and an *amorphous* alkaloidal product, *gelsemin*. Gerrard[6] in 1882, showed that the gelsemin obtained by his predecessors was not a pure product, and separated the substance to which the name *gelsemin* now properly applies, as a crystalline alkaloid forming crystalline salts.

[1] Tr. Maine Med. Assoc., 1889, x., 147.

[2] For an excellent account of this drug see Holmes: Ph. J. and Tr., 1876, 3 s., vi., 481, 521, 561, 601.

[3] Am. J. Pharm., 1852, xviii., 307.

[4] *Ibid.*, xxvii., 203.

[5] *Ibid.*, 1870, 3 s., xviii., 1.

[6] Ph. J. and Tr., 1882, 3 s., xiii., 502, 641.

Thompson[1] verified the work of Gerrard, and separated a second alkaloid, *gelseminin*, which is amorphous and forms amorphous salts.

Gelsemin—$C_{84}H_{60}N_4O_{12}$ (Thompson), $C_{40}H_{63}N_5O_{14}$ (Cushny[2]) forms a white, dry, amorphous, highly alkaline, bitter mass, insoluble in water, or small white crystals fusible at 154°–155° (309°–311° F.), easily soluble in alcohol, chloroform, and ether,[3] most of whose salts are crystalline, the chlorid and bromid difficultly soluble in water. The alkaloid is known pharmaceutically in England as "Gelsemin (Gerrard)" and in Germany as "Kristallisirtes Gelseminin," "Gelseminum puriss. cryst., Merck." The analyses of Spiegel[4] do not agree with those of Cushny. They were made from crystalline compounds of a non-crystalline base or bases obtained from Trommsdorff's "gelseminin."

Gelseminin—$C_{42}H_{17}N_3O_4$? (Cushny), a colorless, amorphous base, which turns light yellow on contact with acids. It is sparingly soluble in water, easily soluble in alcohol, ether, and chloroform. Its salts are amorphous, yellowish, and very soluble in water and in alcohol.

Cushny's experiments show gelseminin to be much more active physiologically than gelsemin. Both alkaloids exist in the pharmaceutical preparations.

Ford and Crow[5] have investigated the *Gelsemium elegans*, which is said to be frequently used in China as an agent of criminal poisoning, and have found it to contain a tetanizing alkaloid, which differs, however, from gelsemin in its color reactions.

Gelsemic Acid (Æsculin?)—(Æsculin = $C_{15}H_{16}O_9$, $1\frac{1}{2}$ H_2O, Liebermann).—In 1876 Robbins[6] and Sonnenschein[7] obtained from gelsemium products similar to those previously obtained by Wormley, but considered gelsemic acid to be identical with *Æsculin*, a glucosid obtained from horse chestnut, and showed many points of similarity between the two, including similar chemical composition. This view was also entertained

[1] *Ibid.*, 1887, 3 s., xvii., 606, 805.
[2] Arch. f. exp. Path. u. Ph., 1892, xxxi., 53.
[3] Merck: Bericht, 1891, *ex* Ph. Jhrber., 1891, 539.
[4] Berichte, Berlin, 1893, xxvi., 1054.
[5] Ph. J. and Tr., 1887, 3 s., xvii. 924.
[6] Diss., Berlin, 1876; Ph. Jahrb., 1876, 152.
[7] Berichte, Berlin, 1876, ix., 1182.

by Schwarz.[1] Wormley[2] has, however pointed out certain differences, which indicate that the view of Dragendorff, implied if not expressed,[3] that the two substances, although closely related, are not identical, is the correct one.

According to Wormley gelsemic acid is a colorless, odorless, nearly tasteless solid, which readily crystallizes either in groups of prisms or in tufts and single needles, or in minute plates and scales. It has only a feebly acid reaction, and forms definite salts with but few metals. When gradually heated to about 163° (325° F.) it fuses to a clear liquid, which may be vaporized without change of color or composition. [Æsculin is decomposed by heat into æsculetin and sugar.] If its vapors be received upon a warm glass slide, they condense to brilliant crystals, which may also be obtained by heating a small portion of the acid in a reduction tube. Gelsemic acid is readily soluble both in ether and in chloroform. In ether of sp. gr. 0.728 one part of the acid quickly dissolves in 300 parts of the fluid. [Æsculin does not dissolve at all in absolute ether, and only very sparingly in ordinary ether. Æsculetin is insoluble in ether.] It is freely soluble in alcohol. Water at the ordinary temperature dissolves one part of the acid in 2,912 of the solvent. Its solubility is greatly increased by the presence of traces of coloring matters or of gelsemin. It is more soluble in hot water, from which it is deposited in crystals on cooling. [Æsculin dissolves in 9 parts cold alcohol (sp. gr. 0.798), in 672 parts of cold water, and in 12½ of boiling water.] (For reactions of the alkaloids and acid see below.)

Almost all of the gelsemium poisonings which have been reported have been accidental. Of thirty-eight cases which have been reported there are but four in which the responsibility for administration has been questioned. In two instances the poisoning (in one case fatal) was caused by the use of gelsemium preparations by empirics.[4] A man of thirty-five years was fatally poisoned by a glassful of the tincture, dispensed in mistake for whiskey at a botanic drugstore where both commodities were sold.[5] A physician was tried for manslaughter in

[1] Diss., Dorpat, 1882.

[2] Am. J. Ph., 1870, 3 s., xviii., 1; *ibid.*, 1882, 4 s., xii., 337; "Micro-Chem. Poisons," 2d ed., 691.

[3] "Ermitt. v. Giften," 4te Aufl., 183.

[4] Pinkham: Boston M. and S. J., 1871, lxxxiv., 89. Hall: Med. Rec., N. Y., 1882, xxi., 65.

[5] Merrill: Memphis M. Recdr., 1856, iv., 38; 1858, vi., 197.

1862, he having administered thirty-five drops of the tincture to a negress of twenty-five to thirty years, with her knowledge and consent, "with a view to investigating its physiological action." She died within two hours.[1]

Symptoms.—An instance of almost fatal poisoning by gelsemin (gelsem. sulf., Merck) is reported by Fronmüller:[2] A boy of fifteen years, suffering from chronic nervous laryngitis, received 0.36 gm. in divided doses. The pupils dilated widely, the vision became indistinct, and the patient became weak and suffered from frontal headache. In going to the closet he fell unconscious, with pale face, glassy, fixed eyes, slow, snorting respiration, and trismus. The pulse rose from 80 to 130, and the skin was cold. In spite of treatment the face became paler, the extremities colder, the pulse slower and weaker, and the respiration more and more infrequent. On resort being had to artificial respiration he gradually improved and finally recovered. Parsons[3] has given a description of the effects of accidentally swallowing a drachm of the fluid extract in himself: in a few minutes there were giddiness, nausea, strabismus, paralysis of the muscles of the mouth and throat, and muffled speech. The eyelids dropped, and deglutition became impossible. The voluntary muscles were entirely unimpaired, and sensation and consciousness were perfect. Then there were præcordial oppression and difficulty of respiration, which latter increased rapidly, the inspiration being short and rapid, followed by three or four long gasps. The respiration apparently ceased, he became livid, rolled in agony, felt himself becoming stiff, and then lost consciousness entirely. As he regained consciousness he saw myriads of stars, and, although he recovered gradually, the paralysis of the mouth, the muffled speech, and the dropping of the eyelids continued for some hours. Up to loss of consciousness the pulse had remained regular and full. The symptoms were greatly aggravated by movement or touching of the head, or by any application of the lips, which caused spasms almost hydrophobic in intensity. There was no vomiting and little loss of voluntary motion. The left side was more paralyzed than the right. Recovery was rapid after return of con-

[1] "The legality of drug provings recognized." [Report of the trial of Dr. E. A. Lodge, etc., Detroit, 1862.] Also Lancet, 1878, i., 892.

[2] Memorab., Heilbronn, 1878, xxiii., 195.

[3] Lancet, 1878, i., 953.

sciousness (two and a half hours after the accident), and the most marked symptom was a persistent and depressing numbness in the occipital region, which lasted for some hours. The muscles of respiration seemed to have been paralyzed, but the epiglottis was the part most affected. In Davis' fatal case[1] of a man who died in two and a half hours after having taken a tablespoonful of Tilden's fluid extract, the face became congested, the pupils dilated but sensitive to light, the lids half closed, with inability to move them, the lower jaw drooping, the tongue thickened, the skin warm and moist, the pulse small and feeble, and the respiration diminished in frequency. There was neither vomiting nor purging. Later the respiration became spasmodic, the surface cold, and the pulse imperceptible. In Harris' case[2] a boy of eighteen months retched and vomited about fifteen minutes after having taken about thirty drops of the fluid extract. He was difficult to arouse, and entered into profound stupor, with dilated pupils, which continued until he died, three hours after the accident. An infant of two years was given twenty-one drops of the fluid extract by mistake, and soon sank into a deep slumber from which it never awoke.[3] Seymour has reported the fatal poisoning of an alcoholic by repeated doses of the tincture, taken to quiet his nerves after drinking.[4]

The average **duration** of 13 fatal cases was $3\frac{1}{6}$ hours; the extremes being 1 hour (Freeman, Hatfield), and $7\frac{1}{2}$ hours (Wormley), or $6\frac{1}{2}$ hours (Seymour). The **mortality** of reported cases is somewhat less than fifty per cent., 15 deaths in 37 cases.

Treatment.—The stomach should be promptly evacuated and washed. A tendency to arrest of respiration is to be met

[1] Am. J. M. Sc., 1867, n. s., liii., 271.

[2] Chicago M. Jour., 1868, xxv., 760.

[3] N. Car. M. J., 1879, iii., 67.

[4] Boston M. and S. J., 1881, cv., 590. For other cases not referred to above see Hall: M. and S. Reptr., Phila., 1861, n. s., v., 366. Main: Boston M. and S. J., 1869, lxxx., 185. Boutelle: *Ibid.*, 1874, xci., 321. Goss: *Ibid.*, 1879, ci., 16. Hardin: Richm. and Louisville M. J., 1873, xv., 621. Freeman: Lancet, 1873, ii., 475. Hills: *Ibid.*, 1878, i., 858. Blake: N. York M. Jour., 1875, xxxi., 366. Courtright: Cinc. Lancet and Clin., 1876, xix., 961. Sinkler: Phila. M. Times, 1877–78, viii., 150; *ibid.*, 1881, xi., 382. Testi: Raccog. lit. med., Forli, 1883, 4 s., xx., 301 (white jasmine). Jepson: Brit. M. J., 1891, ii., 644. Wormley: "Micro-Chem. Poisons," 2d ed., 687, 688. See also Raimondi: Salute, Ital. med., Genova, 1885, xix., 37, 86, and Rehfuss: Therap. Gaz., 1885, 3 s., i., 655. Davison: Eclectic M. J., Cinc., 1879, xxxix., 222, is questionable.

by hypodermics of stimulants and by inhalation of pure oxygen if it be available. The patient's head should on no account be thrown back, nor should he be placed in the prone position, to avoid asphyxia by the paralzyed epiglottis (Parsons). If resort to artificial respiration become necessary the Michigan method[1] should be selected. The supposed antagonism between gelsemin and strychnin does not exist, and atropin and physostigmin hasten the advent of paralysis. Even alcohol and ammonia are said to be of no avail in the treatment.[2]

Post-Mortem Appearances.—These have been observed, so far as we can learn, in but one case within a reasonable period after death.[3] The cadaver of a man of twenty-four years, five and a half hours after death. Blood dark and fluid, without tendency to coagulate or to become brighter on exposure to air. Heart, spleen, lungs, kidneys normal. Liver dark and congested. Stomach contains four ounces of light-colored liquid and mucus, its surface marked with numerous injected vessels. Intestines normal. Brain pale, sinuses not congested; no fluid in ventricles, a few punctiform red dots on section of the brain substance. These appearances show nothing characteristic.

Detection.—In the systematic process for vegetable poisons gelsemic acid (and æsculin) pass from the acid aqueous solution into chloroform, and also to some extent into benzene; and gelsemin (a mixture of gelsemin and gelseminin) from the alkaline aqueous solution into benzene and chloroform, and in minute quantity into petroleum ether. In cases of supposed poisoning by gelsemium the alkaloid is consequently to be sought in residues V. and VI.,[4] and the acid in residue III.

Reactions of Gelsemin.—1. Concentrated sulfuric acid dissolves gelsemin, forming a solution which is either colorless or yellowish, reddish or brownish, according to the degree of purity of the alkaloid. If now a fragment of ceric oxid or manganic oxid be drawn slowly through the liquid, *after complete solution*, a reddish-purple or cherry red color is produced, while the liquid becomes green or bluish-green. Potassium dichromate or lead peroxid may be used in place of either of the oxids

[1] "Wood's Ref. Handb. Med. Sc.," i., 379.

[2] See Husemann: Penzoldt and Stintzing. "Handb. d. sp. Ther., ii., 285.

[3] Boutelle: *Loc. cit.*

[4] See p. 136.

mentioned. If the sulfuric-acid solution have been heated the reaction is not produced on the addition of the oxidant; but if the acid be neutralized with barium hydroxid and the mixture extracted with ether, this on evaporation leaves the alkaloid, which responds to the reaction. (See Strychnin, Detection.)

2. If the residue be mixed with strong sugar solution, and sulfuric acid be then added, a red color is produced. Strychnin is not so colored.

3. Nitric acid dissolves the alkaloid and its colorless salts with little or no color, but the solution, on spontaneous evaporation, leaves a permanently bluish-green residue.

4. According to Dragendorff, *iodo-potassium iodid*, *bromo-potassium bromid*, *phosphomolybdic acid*, *phospho-tungstic acid*, *potassium iodhydrargyrate*, and *potassium cadmium iodid* produce precipitates in solution of 0.00025 gm. of the alkaloid in 1 c.c. of acidulated water; *auric chlorid* and *mercuric chlorid* in similar solutions of 0.005, and *picric acid* in 0.001 per c.c.

5. With the Brouardel-Boutmy reaction (ferric chlorid and ferricyanid) gelsemin gives an intense green color. Strychnin does not react.

6. Sulfovanadic acid produces a purple or red-violet color.

7. With iodic acid in sulfuric acid gelsemin (strychnin also) gives a rose red color.

8. If a preparation of gelsemium, or "gelsemin" prepared by Wormley's or Robbins' methods (gelsemin plus gelseminin) be injected into a frog, loss of sensibility, want of co-ordination, paralysis of motor power, and finally death result. The sensory ganglia are first attacked, then the motor ganglia, but there is no action upon the muscular system. Therefore loss of sensation always precedes that of motion. But with warm-blooded animals the reverse order is observed; the motor ganglia are first attacked and the sensory afterward. Tetanic manifestations are not produced.[1] Bordoni[2] gives respiration tracings showing that in the frog "gelsemin" and scillain similarly produce, after a period of irregular action, a periodically interrupted respiration, without any fixed relation between the dura-

[1] Ott: Phila. Med. Times, 1874–75, v., 689. Moritz: Arch. f. exp. Path. u. Ph., 1879, xi., 299. Schwarz: Diss., Dorpat, 1882.

[2] Boll. d. Soc. tra i cult. d. sc. med., Sienna, 1886, iv., 1.

tions of the respiratory and apneic periods. The frog is, however, not remarkably susceptible to the action of "gelsemin": 0.0005 gm. causes respiratory paralysis, but even with 0.001 gm. cardiac paralysis is not produced (Dragendorff).

Cushny[1] has found that gelsemin and gelseminin differ markedly in the intensity and nature of their action upon frogs. The former, in doses of 10 and 20 mgm., produces marked increase in reflex sensibility, opisthotonus, and intense tonic spasms, but not death. Gelseminin causes paralysis of the central nervous system, which begins without any previous excitement and progresses from the brain to the cord, and a curare-like paralysis of the motor nerves. The respiration is slow and shallow and is arrested, the head trembles with every movement, and there is muscular relaxation. Marked effects are produced with 0.001 gm., and death with 0.002 gm.

Reactions of Gelsemic Acid.—These, as given by Wormley, are substantially as follows: 1. *Nitric acid* dissolves it, forming a yellow or reddish solution, which, on addition of *ammonium hydroxid* in excess, acquires a permanent deep blood-red color. Æsculin gives the same reaction.

2. *Sulfuric acid* slowly dissolves pure gelsemic acid, forming a yellow solution, which is unchanged by a moderate heat. If the acid be impure the solution may have a reddish color, which is changed to deep brown by a moderate heat. Æsculin, when pure, quickly dissolves to a yellow solution in sulfuric acid, and soon becomes brown and charred when moderately heated. If a fragment of potassium dichromate be added to the sulfuric solution of gelsemic acid, green oxid of chromium quickly appears.

3. If a drop of aqueous ammonia be allowed to flow into a drop of the sulfuric-acid solution of gelsemic acid, the latter immediately separates as a mass of crystalline needles. Æsculin does not respond to this test.

4. Hydrochloric acid fails to dissolve or act upon gelsemic acid, even when heated to 100° (212° F.). Æsculin is readily soluble in this acid.

5. *Ammonia* and *the fixed alkalies* cause gelsemic acid to assume an intense yellow color, and quicky dissolve it to solutions having very striking fluorescent properties, even when

[1] Arch. f. exp. Path. u. Ph., 1892, xxxi., 55.

highly diluted. When the solution is examined by transmitted light, it has a yellow color; under reflected light a deep bluish appearance, and under condensed sunlight an intense blue color along the path of the condensed rays. The fluorescence is still manifest in solutions containing 1 : 100,000 of the acid. It is quickly destroyed by free acids. Æsculin behaves in the same manner. Solutions of quinin salts are colorless and fluorescent in the presence of free acid, but their fluorescence is destroyed by alkalies.

6. Solutions of gelsemic acid form precipitates with *lead acetate*, dirty yellow, amorphous, soluble in acetic acid, soon replaced by crystalline needles, 1:1000; *mercuric chlorid*, yellowish-white, from which crystals of the acid separate, 1:1000; *silver nitrate*, brownish-yellow, soon becomes bluish-black or purplish, 1:50,000; *copper sulfate*, dirty-brown precipitate in tolerably strong solutions, soon becomes dull red, crystals of the acid separate after a time; *auric chlorid*, strong solutions, a deep blue deposit which soon becomes green.

7. When the acid is cautiously heated in a subliming cell it condenses in the form of brilliant transparent crystals. Only a minute quantity of the acid should be used, and the condensing surface should be warm. The sublimate may be dissolved in nitric acid and excess of ammonia added, when an orange or red color is developed.[1] Æsculin behaves similarly.

According to Schwarz,[2] gelsemin is not decomposed during putrefaction, and gelsemic acid (æsculin) only when the reaction is alkaline. Gelsemin is detectable in the urine several days after the ingestion of a single dose.

MORPHIN AND OPIUM.

Opium is obtained by making incisions in the unripe heads of the poppy (*Papaver somniferum*), and drying the juice which exudes. It is a highly complex body, containing some twenty or thirty constituents, among which are a number of alkaloids, combined with acetic, lactic, and meconic acids. Of the twenty-one alkaloids which have been obtained from opium

[1] Holmes: Ph. J. and Tr., 1876, 3 s., vi., 601. Wormley: *Op. cit.*, Pl. xv., f. 3.

[2] *Loc. cit.*

some are probably produced by the processes of extraction, and only three: *morphin*, *codein*, and *thebain*, are directly of toxicological interest. The remainder are either comparatively inert or occur in extremely minute quantity. Opium yields about 10 per cent. of morphin, 0.25 to 0.5 per cent. of codein, and 0.15 to 1 per cent. of thebain.

MORPHIN—$C_{17}H_{19}NO_3 + H_2O$—was the first alkaloid dis-

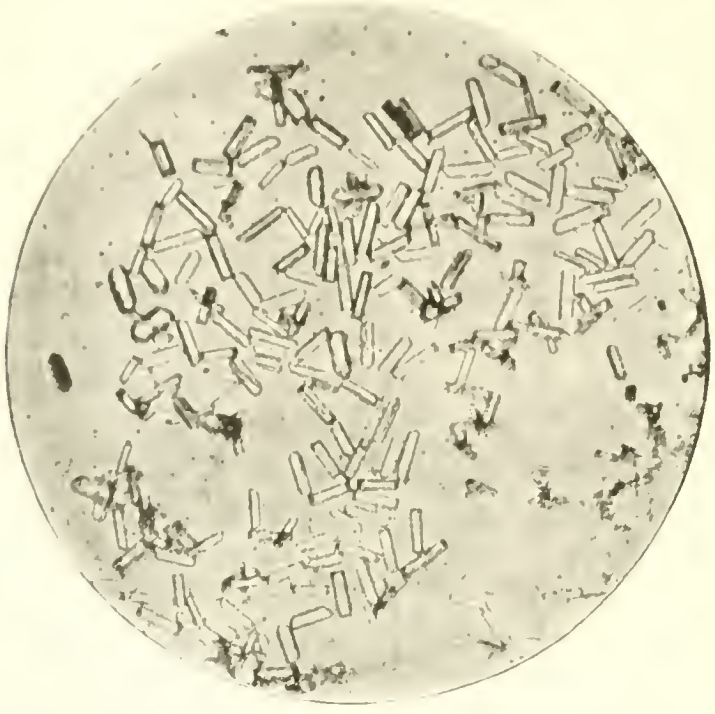

FIG. 34.—Crystals of Morphin. From Amylic Alcohol Solution, × 75.

covered, by Sertürner in 1806. It crystallizes in colorless, rhombic prisms (Fig. 34), which lose their water of crystallization at 100° (212° F.), turn brown at about 190° to 200° (374°–392° F.), and fuse, with partial decomposition, at about 230° (446° F.). Morphin is very sparingly soluble in cold water, and soluble in 400 to 500 parts of boiling water. Its solubility in organic solvents is given by Florio[1] as follows, in amounts soluble in 100 parts of the solvent: Absolute alcohol, at 10°.6, (57°.1 F.)—1.132; at 78° (172°.4 F.)—8.623; alcohol of 90 per cent. at 10°.6 (51°.1 F.)—0.377; at 78° (172.4° F.)—2.991; alcohol of 75 per cent. at 10°.8 (51°.4 F.)—0.223; at 78° (172°.4 F.)—1.985; amylic alcohol at 11° (51°.8 F.)—0.268; at 178° (72°.4

[1] Beilstein: "Handb. d. org. Chem.," iii., 550.

F.)—2.247; benzene at 9°.4 (48°.9 F.)—0.029; chloroform at 9°.4 (48°.9 F.)—0.040; at 56° (132°.8 F.)—2.247; ether (absolute) at 10° (50° F.)—0.023. It is less soluble in aqueous ether, but more soluble in ether-alcohol. It dissolves in acetic ether to the extent of 0.213 parts in 100. All the solvents dissolve morphin more readily when the alkaloid is freshly precipitated than after it has assumed the crystalline form. Solutions of morphin are lævogyrous, alkaline, and bitter. It dissolves readily in dilute acids, from which solutions it is deposited on evaporation as the corresponding salts. Solutions of the salts of morphin are decomposed by the hydroxids of sodium, potassium, ammonium, barium, and calcium, and by magnesia; the precipitated alkaloid being, however, partly or completely redissolved by excess of the precipitant, unless it be magnesia.

Morphin is a monacid base, possessing the functions of an alcohol and those of a phenol, and forms crystalline salts, of which the sulfate, chlorid, and acetate are used in medicine. These salts are readily soluble in water. It is a strong reducing agent and reduces the salts of gold and silver in the cold. Most of the reactions of morphin are based upon this property. It is oxidized by atmospheric oxygen when it is in alkaline solution, as well as by nitrous acid, potassium permanganate, potassium ferricyanid, or ammoniacal cupric sulfate, with formation of a non-toxic derivative which has received the names *oxymorphin*, *oxydimorphin*, *dehydromorphin*, and *pseudomorphin* $(C_{17}H_{18}NO_3)_2$. When heated to 140° (284° F.) with hydrochloric acid or with dehydrating agents it is converted into *apomorphin*, $C_{17}H_{17}NO_2$, by loss of the elements of a molecule of water.

Codein, $C_{17}H_{18}NO_3,CH_3 + H_2O$,—or *methyl-morphin*, crystallizes in large rhombic prisms, or from ether, without water of crystallization, in octahedra. It is bitter, soluble in 80 parts of cold water and in 17 parts of boiling water; very soluble in alcohol, amylic alcohol, ether, chloroform, and benzene, and almost insoluble in petroleum ether.

Thebain = *paramorphin*, $C_{19}H_{21}NO_3$, crystallizes with difficulty in prisms or needles. It is lævogyrous, alkaline, has a sharp taste, and is very poisonous, exerting a tetanic action. It is insoluble in water and in petroleum ether, soluble in 10

parts of cold alcohol, in 60 parts of amylic alcohol, in 18 of chloroform, in 20 of benzene, and in 140 of cold ether.

Preparations Containing Morphin or Opium.

Officinal U. S. Ph.—*Acetum opii* = vinegar of opium = black drop, contains 100 gm. of opium in 1,000. *Emplastrum opii* = opium plaster, contains 60 gm. of opium in 1,000. *Extractum opii*, contains 18 per cent. of morphin. *Opium*, containing not less than 9 per cent. of morphin. *Opium deodoratum* = denarcotized opium, is opium washed with ether, and the original weight restored by addition of milk-sugar. *Pilulæ opii* = opium pills, containing 6.5 gm. opium in 100. *Pulvis ipecacuanhæ et opii* = Dover's power, contains 10 gm. opium in 100. *Pulvis morphinæ compositus* = Tully's powder, contains 1 gm. morphium sulfate in 60. *Tinctura ipecacuanhæ et opii*, of the same strength as the simple tincture. *Tinctura opii* = laudanum, made from 100 gm. of opium for 1,000 of tincture and contains 1.3 to 1.5 gm. morphin in 100 c.c. *Tinctura opii camphorata* = paregoric, made from 4 gm. of opium for 1,000. *Tinctura opii deodorata*, of the same strength as the simple tincture. *Trochisci glycyrrhizæ et opii*, contain 0.5 gm. opium in 100. *Trochisci morphinæ et ipecacuanhæ*, contain 0.16 gm. morphin in 100. *Vinum opii*, of the same strength as the tincture. The *acetate*, *chlorid*, and *sulfate* of morphium, and alkaloidal *codein* are also officinal.

A great number of **proprietory nostrums** contain morphin or opium: Soothing syrups, pectoral syrups, bronchial troches, cough remedies, opium cures, cholera remedies, pectorals, polyforms, chlorodyne, etc.

Opium Poisoning.

Statistics.—The poisonous nature of opium and of the poppy was known to the ancients. Nicander (185–135 B.C.) gives a clear description of the symptoms caused by a poisonous dose of a "drink prepared from the tears which exude from poppyheads." Dioscorides, three centuries later, remarks of the poppy that "if it be taken in larger quantity, it is capable of causing death." Pliny (A.D. 70) speaks of the toxic power of

opium, and cites the instance of Post. Licinius Cæcina, who, disgusted with life by protracted illness, terminated his existence by opium, an instance which does not seem to have been singular, as the narrative concludes with "item plerosque alios."

At the present time, so far as can be judged from the few available tables of statistics, from 35 to 40 per cent. of all accidental and suicidal poisonings are by opiates. The high percentage of mortality among infants and young children, as compared with that among older children, is unquestionably due in large measure to the very general use of opiates, either in the form of paregoric or laudanum or of various proprietary nostrums, by ignorant and indolent mothers and nurses. That the practice of "soothing" infants with opiates is not of recent origin is shown by Pyl and Engel's report,[1] in 1793, of the case of a child of four weeks which died during the night from the effects of a dose of "*Requies Nicolai*," administered by its nurse in the evening. Whether such infant deaths are truly accidental, or are due to a degree of negligence amounting to manslaughter, or are even deliberate infanticides is a question of intent which is only indirectly within the province of the expert. It cannot be doubted, however, that overdoses are frequently intentionally given, particularly to illegitimate children; and modern imitators of that worthy precursor of Mrs. Winslow who, as Fodéré relates,[2] disposed of the children confided to her care by means of a decoction of poppies in the latter part of the eighteenth century, have plied the same trade in more modern times.

Homicidal poisonings by the opiates or by morphin have rarely been the subject of investigation, a fact for which we can assign no adequate cause. The deterrent effect of the bitter taste of morphin, to which Kobert refers,[3] cannot be the true reason for the infrequency of homicidal morphin poisoning, as strychnin, which is much more intensely bitter, has been resorted to by the poisoner in certainly one hundred instances.

We find reference to but two homicidal poisonings of adults by opiates other than morphin: In one a man was tried for an attempt to murder his wife by strychnin in December, 1860, and acquitted, but in the following March was convicted of a

[1] Pyl: "Aufsätze u. Beobacht.," 1793, viii., 92.

[2] "Méd. lég.," iv., 20.

[3] "Intoxikationen," 552.

similar attempt with laudanum.[1] In 1871 a man and a woman were committed for trial in England for the murder of the wife of the former by laudanum.[2]

Of ten homicidal *morphin* poisonings of adults, the earliest was also the first instance in which an alkaloidal poison was used, and the last four have occurred since 1890:[3]

1. Affaire Castaing, 1823. A French physician was convicted of the murder of his friend by morphium acetate. The conviction was entirely upon lay evidence, the post-mortem and analysis having furnished no positive indications.[4]

2. In 1871 Dr. Medlicott was convicted of murder by morphin and atropin in Kansas. An analysis was made by the Stas method and affirmative results were obtained with the nitric acid, ferric chlorid, Fröhde, and iodic acid tests for morphin, as well as with the sulfuric acid and physiological tests for atropin. The deceased was found dead in bed, and the autopsy was made the same day.[5]

3. In November, 1875, the case of the Peo. *v.* Eliza A. Stone was tried in Columbia County, N. Y., and the defendant was acquitted. Dr. John T. Wheeler, who made the autopsy, informs us that the "autopsy showed absence of disease and presence of venous congestion. But chemical examination showed no morphine in the stomach." We find no published account of this case.

4. Dr. Krauss, of Tübingen, reports the poisoning of a woman of eighty-two years, in 1878 in a German village. The poison was administered in coffee, which the deceased drank, notwithstanding its bitter taste. The symptoms were those of morphin poisoning, and death occurred in thirty-six hours. The analysis (?) was limited to an application of the iodic acid test.[6]

5. The same author relates at length the singular case of Anna Thornählen, who was tried in 1876 and acquitted, although, nine weeks after the death of her husband, she accused herself of having poisoned him with morphin, and gave a circumstantial account of the manner

[1] Reg. *v.* Buckle, Ph. J. and Tr., 1860–61, n. s., iii., 629.

[2] *Ibid.*, 1870–71, 3 s., i., 714. We find no further reference to this case. A laudanum poisoning reported by Letheby (Lancet, 1844, ii., 317) was apparently homicidal.

[3] Lee ("Homicide and Suicide in the City of Philadelphia during a Decade, 1871–81") refers to one homicide by morphin and three by opium. No particulars are given. Casper ("Klin. Novellen," 391) relates a case of supposed poisoning by alkaloidal morphin, in which he considers the evidence insufficient. The case quoted by Husemann (Maschka's "Handb. d. ger. Med.," ii., 402) as related by Casper, we do not find at the place indicated or elsewhere.

[4] Tardieu: "Empoisonnement," 2ème ed., 1058. "Causes criminelles célèbres du XIX. Siècle," Paris, 1828, iv., 1–103. "Neue Pitaval," v., 331–446.

[5] Saunders: Mich. Univ. M. J., 1871–72, 641.

[6] Friedreich's Bl. f. ger. Med., 1883, xxxiv., 370.

of administration. The body was exhumed seventy-one days after death, and the results of the autopsy and analysis were negative. The case is of psychological rather than toxicological interest.[1]

6. In 1879 Joseph and Mary Volckmer were sentenced in New York City to twelve years' imprisonment for having administered morphin to a rural visitor, with the intent, apparently, of facilitating robbery.

7. Peo. *v.* Carlyle W. Harris. The defendant was convicted in New York City in 1892 of the murder of his unrecognized wife by morphin, which had been administered by substituting morphin for quinin and a medicinal dose of morphin in a capsule. The symptoms were those of opium poisoning, and death occurred in twelve hours. The analysis showed the presence of morphin and the absence of quinin in the cadaver.[2]

8. Peo. *v.* Walter J. Buchanan. The defendant was convicted in New York City in 1893 of the murder of his wife. The symptoms were such as would be produced by atropin, followed by morphin. The analysis showed the presence of morphin, but the presence of atropin was not demonstrated with certainty.

9. Caso Urbino de Freitas, Portugal, 1891–93. The defendant, a physician, was accused of having poisoned three of his wife's nephews, and of having caused the death of one of them by poison administered in enemata. The symptoms resembled those caused by opium in part only. The analysts claimed to have detected morphin, narcotin, and delphinin in the urine and viscera; but based their conclusion as to morphin on the insufficient evidence furnished by the iodic acid, Fröhde's, and Lafon's reactions. (See Detection.)[3]

10. Affaire Joniaux. A lady moving in aristocratic circles in Belgian society was accused in 1894 of having poisoned three of her relatives with morphin, for the purpose of collecting large insurances upon their lives. The viscera of the alleged victims were analyzed by Druyts and Bruylants, who found morphin in the stomach, liver, kidneys, spleen, lungs, blood, intestine, and urine of the brother who had died March 6th, 1894, and whose body was exhumed nine days later, but none in the cadavers of the other victims who had died March 17th, 1893, and February 24th, 1892.[4]

Duration.—The duration of a fatal poisoning by morphin is not as a rule shorter than when laudanum or other opiates are the cause of death. The usual duration is from 6 to 24 hours, the greater proportion of deaths occurring in from 12 to 24 hours. Instances are recorded of death in less than

[1] *Ibid.*, 1887, xxxviii., 153.

[2] Witthaus: "Researches Loomis Lab.," 1892, ii., 1, 91.

[3] Souto, Azeredo, Silva-Pinto and Silva: "Relation méd.-lég. d. l'Aff. Urbino de Freitas," Porto, 1893.

[4] Bruylants et Druyts: "Affaire J—— Rapports d'expertises chimiques," Charleroi, 1895.

1 hour. Thus Eberts[1] reports the death of a woman in from 10 to 50 minutes from the effects of 0.25 gm. (3.85 grains) of morphium chlorid dispensed in mistake for quinin. Lyman[2] and Coale[3] have reported deaths from laudanum within 45 minutes. A similar case is quoted by Taylor.[4] The most prolonged fatal case of which we find record was that of a child of three months, which died in 56 hours after the administration to it of an opiate.[5] Maschka[6] reports a suicide, probably by morphin, of a man who was found unconscious in a railway carriage, and who died on the third day thereafter. Penny[7] makes mention of the case of a girl of nineteen years who died in 3 days after having taken ten grains of morphin with suicidal intent. When death is delayed for a longer period it is not due directly to the opiate, although that may have been contributory to the generation of the proximate cause.[8]

Lethal Dose.—The effects of a given dose of an opiate will vary within wide limits under different conditions of age and physical condition of the individual, and particularly with the extent of previous use. The minimum lethal dose for a healthy adult, not habituated to opiates, is probably from 0.2 to 0.26 gm. (3 to 4 grains) of morphin, or an equivalent quantity of a liquid opiate. These quantities have proved fatal in several reported instances. Hartley[9] has reported the case of a man who died in eighteen hours after having swallowed half an ounce of laudanum (= 0.2 gm. = 3 grains morphin) with suicidal intent. Taylor[10] refers to the death of a woman of fifty-six years from the effects of a like quantity of laudanum administered in mistake for tincture of rhubarb. Ebertz[11] relates the case of a woman who died in fifty minutes from the effects of 0.25 gm. (3.9 grains) of morphium chlorid dispensed in mistake for quinin. Anstie[12]

[1] Vrtljschr. f. ger. Med., 1873, n. F., xviii., 280.

[2] Amer. J. M. Sc., 1854, xxviii., 384.

[3] *Ibid.*, 1850, n. s., xix., 71.

[4] "Poisons," 3d Am. ed., 537.

[5] Mushet: Med. Times and Gaz., 1858, n. s., xvi., 292.

[6] Prag. med. Wochenschr., 1887, xii., 437.

[7] Med. Sentinel, Portland, Ore., 1893, i., 299.

[8] See Parham and Lamb: N. Or. M. and S. J., 1886–87, n. s., xiv., 601 (Case I.). Isham: Med. News, Phila, 1882, 32 (Case II.). Ramich: Allg. Wien. med. Ztg., 1879, 350. Riddell: South. Clin., Richm., 1879–80, ii., 437. Fell: J. Am. Med. Assoc., 1892. xix., 130. Lewin: Berl. kl. Wochenschr., 1893, xxx., 993.

[9] Lancet, 1873, ii., 684.

[10] "Poisons," 3d Am. ed., 533, 534, 537, 560.

[11] Vrtljschr. f. ger. Med., 1873, n. F., xviii., 280.

[12] Med. Times and Gaz., 1863, i.,

records the case of a man of forty years who died in sixteen hours from the effects of an enema containing three grains (0.19 gm.) of morphin, administered to relieve the pain caused by a fistula. Boeck[1] refers to the death of a maniacal woman caused by the hypodermic injection of 0.18 gm. (2.8 grains) of morphin in two hours. Smaller quantities have proved fatal to persons in advanced age, or have been a contributing cause of death in those suffering from kidney diseases, or debilitated from other illness. The following is a list of such cases as well as of others in which death has been ascribed to small quantities of morphin upon insufficient data:

1. *Gilliam* (*Med. Rec., N. Y.*, 1885, *xxvii.*, 679): F., 66; about $\frac{1}{8}$ grain (0.008 gm.) morphium sulfate hypodermically in the afternoon, after several doses (how many not stated) of ten to fifteen grains of chloral hydrate during the forenoon, to relieve neuralgia. Died at about nightfall from exhaustion.

2. *Toogood* (*Prov. M. and S. J.*, 1841, *iii.*, 129): F., 80; was given seven drops of liquor morphiæ acetatis. The next day she was in a deep sleep, from which she could not be aroused, and died in a few hours. The dose administered would contain about $\frac{1}{6}$ grain of the acetate (= 0.011 gm.).

3. *Reamy* (*Cinc. Lanc. and Clin.*, 1885, *n. s.*, *xiv.*, 1): F., 28; $\frac{1}{4}$ grain (0.016 gm.) morphium sulfate and $\frac{1}{96}$ atropin hypodermically, after perineorrhaphy and removal of a cicatrix of laceration of the cervix. Death in profound coma in eight and one-half hours, notwithstanding treatment. Said to have been previously poisoned by a minute dose of morphin. No autopsy. No examination of urine.

4. *A North Carolina Physician* (*N. Car. M. J.*, 1879, *iii.*, 228): F., 40; hypodermic of $\frac{1}{2}$ grain (0.032 gm.) morphium acetate to control pain in a violent epileptic seizure occurring at the menstrual period. Urine highly albuminous during the seizure, not at other times. In treatment she was given $\frac{11}{20}$ grain (0.036 gm.) atropium sulfate in divided doses. No autopsy. [Death probably from uræmic and atropic poisoning.[2]]

5. *Account of Inquest* (*Lancet*, 1838–39, *i.*, 276): F., ad.; physician stated that he gave half a grain (0.032 gm.) of the acetate in one-half of a mixture compounded by himself. The analysis of the remaining half showed it to contain two grains (0.13 gm.). The woman had been ill (from what disease is not stated), and the accounts of the symptoms were conflicting. Death in from six to seven hours.

134; Ph. J. and Tr., 1862–63. n. s., iv., 330.

[1] Ziemssen: "Handbuch," etc., xv., 541.

[2] See N. Car. M. J., iii., 228; iv., 31.

6. *Wormley* ("*Micro-Chemistry of Poisons*," *2d ed.*, 479): "Thus an instance is related in which half a grain employed in this manner (hypodermically) for the relief of sciatica proved fatal to a woman aged fifty years." [1]

7. *Ingalls* (*Chic. M. J. and Exam.*, 1878, *xxxvi.*, 493): M., ad.; suffering from myalgia of the back, received $\frac{1}{60}$ grain atropin hypodermically, then ¼ grain (0.016 gm.) morphin by the mouth, and three-quarters of an hour later ¼ grain morphin hypodermically. A further quantity of atropin was administered to counteract the effects of the morphin—how much is not stated. No autopsy.[2]

8. *Souchon* (*N. Orl. M. and S. J.*, 1886–87, *n. s.*, *xiv.*, 437, *Case II.*): M., 24; half a grain (0.032 gm.) morphium sulfate hypodermically to relieve pain in a colloid cancer into which pure zinc chlorid had been injected. Collapse complete in one and one-half hours, but regained consciousness under treatment. Remained in this improved condition and took nourishment for four hours when he sank gradually, and died eleven and a half hours after the administration of morphin. No autopsy.

9. *Sieveking* (*Lancet*, 1861, *i.*, 575): M., 45; one grain (0.065 gm.) of the acetate in pill form. Out-patient in St. Mary's Hospital, London. Died in thirteen hours. The urine was albuminous and the kidneys were highly diseased.[3]

10. *Account of Inquest* (*Pharm. J. and Tr.*, 1863, *n. s.*, *iv.*, 532): M., 52; died in ten hours after having taken a mixture which, according to the attending physician, contained half a grain of morphium chlorid. No statement of the nature of the illness, nor of the results of the autopsy.[4]

11. *Report of Trial* (*Buff. M. and S. J.*, 1867–68, *vii.*, 121): M., ad.; death in seven hours. This is an account of the trial of a suit for damages against the attending physician who stated that he administered hypodermically "half a grain as near as I (he) can judge."

12. *Paterson* (*Edinb. Mthly. J.*, 1845, 195): F., 19; one grain in divided doses during six hours. Death in seven hours. [5]

13. *Morgan* (*Taylor:* "*Poisons*," *3d Am. ed.*, 549): M., ad.; one grain (0.065 gm.) hypodermically in twelve hours. Died in six hours. Patient in Middlesex Hospital. No particulars as to the nature of the

[1] No reference to the place where this case is related is given, and in answer to a communication Professor Wormley states his inability to find the reference.

[2] This is not an observation of Ingalls', but a case communicated to him in response to a circular letter.

[3] See also Ph. J. and Tr., 1862, n. s., iii., 45.

[4] Taylor ("Poisons," 3d Am. ed., 549) refers to this case as that of a "healthy man," yet he was under treatment by a physician. Taylor also states the dose to have been one grain: on what authority does not appear.

[5] We have been unable to consult the original report of this case, which is quoted by Taylor: "Poisons," 3d Am. ed., 549.

disease. No autopsy. Guy and Ferrier ("Forensic Med.," 6th ed., 473) in citing this case use the words "appears to have proved fatal."

14. *Johnson* (*Lancet*, 1872, *ii.*, 24): F., 62; afflicted with large abdominal tumors weighing thirty to forty pounds, and with dropsy; had been an invalid for twenty years. She received one grain of the acetate in a pill and died in less than twelve hours.[1]

15. *Bossart* (*Rev. méd. d. l. Suisse rom.*, 1885, *v.*, 679): F., 32; died the day after she had taken 0.06 to 0.07 gm. (about one grain) of morphin and 6 to 7 gm. (93 to 108 grains) of chloral.

16. *Wharton and Stillé* ("*Med. Jur.*," 3*d ed.*, *ii.*, 337, 348): M., ad.; one and one-third grains in four pills at intervals of one hour. The patient had acute rheumatism.[2]

17. *Denig* (*Wormley*: "*Micro-Chemistry of Poisons*," 2*d ed.*, 470): F., 17; robust and healthy. Dose given as ʒij. of laudanum. This should contain at least 1.6 grains morphin, but the druggist testified that it contained only the soluble portions of seven grains of opium (= 0.7 gm. morphin).

18. (*Lancet*, 1857, *i.*, 435): M., ad.; a patient received an injection containing 10 gm. of laudanum (= 1.6 grains morphin), became comatose, was completely roused, remained quite free from narcotic symptoms for twenty hours, became suddenly worse and died in two days. No statement of the nature of the disease or of the results of the autopsy.

19. *Fagerlund* (*Vrtljschr. f. ger. Med.*, 1894, 3 *s.*, *viii.*, *Supplhft.*, 86): M., 70; took 10 gm. of laudanum (= 0.15 gm. = 2.3 grains morphin) with suicidal intent, and was found dead next morning.

20. *Med. Times*, *Lond.*, 1846, 114): F., 66; had gangrene of right foot. Three grains (0.2 gm.) of the chlorid administered by mistake. Died in nine hours.

Other early reports in which the accuracy of the dose stated is questionable are those of Sharkey (*Lond. M. Gaz.*, 1846, II., 235, Case II.; Toogood (*Prov. M. and S. J.*, 1841, III., 129; Vermandois (Alibert) *Bibl. Med.*, 1806, quoted by Ortila: "Tox. gén.," 5ème ed., II., 254.

Infants and young children are very susceptible to the toxic action of the opiates: An infant died from the effects of $\frac{1}{12}$ gr. (0.005 gm.) of the chlorid administered in mistake for dill water;[3] and a child of eleven months was destroyed by $\frac{1}{15}$ gr. (0.004 gm.), also given by mistake.[4] Cheatham[5] has reported the case of a boy of eight years, suffering from acute basilar menin-

[1] See also Ph. J. and Tr., 1872–73, 3 s., iii., 14.

[2] This case is quoted from Med. Times and Gaz., June, 1860, 254. We fail to find it in that journal.

[3] Chem. News, 1863, 98; Ph. J., and Tr., 1863, n. s., v., 138.

[4] *Ibid.*, 1870–71, 3 s., i., 615.

[5] N. Car. M. J., July, 1886 (Reprint), p. 8.

gitis, whose death was hastened by the hypodermic administration of $\frac{1}{8}$ gr. (0.008 gm.) of morphin combined with atropin.[1] Infants of a few days have died from the effects of three,[2] two,[3] and even a single[4] drop of laudanum (= 1.26, 0.84, 0.42 mgm. morphin); and children of eighteen months or less from five,[5] four,[6] and three[7] drops (= 2.1, 1.68, 1.26 mgm. morphin). Taylor[8] states that a case was communicated to him by Mr. Edwards of an infant of four weeks which died from the effects of a quantity of paregoric containing $\frac{1}{90}$ gr. of opium (= 0.0007 gm. = 0.00007 gm. morphin). No particulars are given, however, concerning the child's condition of health or disease.[9] Two instances are reported in which young children have been fatally poisoned by the milk of the mother to whom opiates had been administered: in one the infant was three days old, and the opiate had been administered to the mother internally.[10] In the other the child was six weeks old; laudanum had been administered to the mother internally and a fomentation of gin and laudanum had been applied to her side.[11]

On the other hand even non-habituated adults have recovered from the effects of very large doses. The largest quantity of morphin certainly thus recovered from was 60 grains (3.89 gm.) of the acetate, which was taken with suicidal intent by a man of forty years, who had never been an opium eater.[12] Other recoveries from large doses of morphin have been reported by Norris (75 gr. = 4.86 gm.),[13] Watkins (61 gr. = 3.95 gm. taken in one hour),[14] Bonjean (55 gr. = 3.56 gm. acetate),[15] Castara (50

[1] See also Ph. J. and Tr., 1873–74, 3 s., iv., 99.

[2] Ph. J. and Tr., 1870–71, 3 s., i., 798, æt. seventeen days. Everest. Lancet, 1841–42, 758, æt. two days.

[3] Prov. M. and S. Jour., 1846, 519, æt. five days. Smith: Lancet, 1854, i., 419, æt. seven days, ♏ i. Balfour: Edinb. M. J., 1856–57, ii., 146, æt. four days.

[4] Woolen: South. M. and S. Jour., July, 1849, quoted by Beck: "Infant Therap.," 145.

[5] Jackson: Am. J. M. Sc., 1854, xxviii., 384; Ph. J. and Tr., 1879–71, 3 s., i., 742. Fagerlund: Vrtljschr. f. ger. Med., 1894, 3 s., viii., Supplh., 86, gtt. v. or more. Homicide; by mother in teaspoonful of her own milk.

[6] Kelso: Lancet, 1837–38, i., 304; Lancet, 1869, ii., 187. Alison: Christison, "Poisons," Am. ed., 550.

[7] Simsen. Christison: *Loc. cit.*

[8] "Poisons," 3d Am. ed., 536, 542.

[9] See also Ph. J. and Tr., 1872–73, 3 s., iii., 1047.

[10] Evans: Br. M. J., 1885, ii., 1159.

[11] Med. Times and Gaz., 1861, 70; Lancet, 1861, i., 93.

[12] Wood: Boston M. and S. J., July, 1876, 82.

[13] Am. J. M. Sc., 1862, n. s., xliv., 395 (Case I.).

[14] Richm. and Louisv. M. J., 1870, x., 453.

[15] J. d. chim. méd., etc., 1844, x., 692.

gr. = 3.24 gm. acetate),[1] Morse (38 gr. = 2.48 gm. sulfate),[2] and Lemen (36 gr. = 2.33 gm. sulfate).[3] Two cases are reported of recovery from the effects of eight ounces of laudanum (= 51 gr. = 3.31 gm. morphin),[4] one from six ounces (= 38.3 gr. = 2.48 gm. morphin),[5] and many from doses of from two to five and a half ounces (12.8–35 gr. = 0.83–2.28 gm. morphin). Wormley[6] cites the case of a pregnant female who swallowed between seven and eight ounces (220–250 gm.) of solid opium, yet recovered under treatment, after having exhibited well-marked symptoms of narcotism. Children have also recovered from relatively large doses. Thus Callender has reported the recovery of a child of seven years after it had received, through mistake of the nurse, two drachms of laudanum (= 7.6 gr. = 0.1 gm. morphin);[7] Dow[8] has reported the case of a child of four years which withstood the effects of 3 grains (= 0.19 gm.) of morphium sulfate given in mistake for quinin; and Hadden[9] refers to the case of a youthful suicide of seven years who recovered from the effects of a dose of half an ounce of laudanum (= 3.2 gr. = 0.2 gm. morphin).

The poisonous action of morphin is very greatly diminished by habit, probably more than that of any other poison. The amounts taken by adult opium eaters, laudanum drinkers, and morphin injectors are sometimes enormous. Cases in which the consumption reaches 2 to 3.5 gm. (30–54 gr.) of morphin daily have been observed,[10] and De Quincy relates that his daily allowance of laudanum at one time reached nine ounces (= 57.5 gr. = 3.7 gm. morphin). Chisolm[11] refers to an opium eater who took 120 grains (7.8 gm.) of morphin at a single dose with suicidal intent, but without any ill effects. This tolerance may also be

[1] J. de chim. méd., etc., 1831, vii., 135.

[2] Boston M. and S. Jour., 1887, cxvi., 603.

[3] *Ibid.*, 1887, cxvi., 443. The report by Du Bois (Western Lancet, i., 1872, 336) of a case in which 80 grains (5.18 gm.) are said to have been taken contains internal evidence of inaccuracy.

[4] Barstead: Lancet, 1873, i., 468. Harvey, cited by Taylor: "Poisons," 3d Am. ed., 535.

[5] O'Callachan: Canada M. and S. Jour., 1881–82, x., 6 (Case I.). See also Marcet: Med.-Chir. Tr., 1806, i., 77.

[6] "Micro-Chem. of Poisons," 2d ed., 472.

[7] Med. Rec., N. Y., 1894, xlvi., 345.

[8] Va. Med. Monthly, 1877–78, iv., 670.

[9] Phys. and Bull. Med.-leg. Soc., N. Y., 1880, xiii., 8.

[10] Regnier: "Intoxication chronique par la morphine," Paris, 1890, 101, 107, 114, 123, 124. Groom: Med. Rec., N. Y., 1894, xlvi., 340.

[11] Maryland M. J., 1878, iii., 109.

established in children. Thus Little[1] has reported the case of a male infant, suffering from acute inflammation of the knee-joint, to whom opiates were given in gradually increasing amount until, when eight months of age, it took two fluidounces of Magendie's solution (= 32 gr. = 2.07 gm. sulfate) in one day with no toxic effects. The tolerance of morphin resulting from habituation does not amount to immunity, and cases are by no means rare of morphin *habitués* who have died from the effects of overdoses.[2]

Symptoms.—The symptoms produced by opium and its preparations and by morphin—acute meconism—are practically the same whatever preparations of opium or salt of morphin may have been used.

The *time* when symptoms begin is usually from half an hour to an hour after the poison has been taken. Frequently this period is much shortened. In children which have received relatively large doses the poison sometimes begins to produce its effects within a few minutes or almost immediately. When the poison has been introduced hypodermically its action is more rapid than when taken by the stomach, and opiates administered by the rectum are more prompt in their action than when they are swallowed. In exceptional instances the interval between the taking of the poison and the manifestation of symptoms is much shortened, even in adults. Many instances are reported in which the patient became unconscious in ten or fifteen minutes and several in which the action was "almost immediate."[3] Or the appearance of symptoms may be delayed for several hours. Thus Boeck[4] cites the case of a maniacal woman who received 0.18 gm. (3 gr.) of morphin hypodermically during two hours; she was not quieted, but in two hours went to sleep, and afterward died in collapse. Channing[5] reports the fatal poisoning of a man who took laudanum at 6:30 A.M., and, although in bed all day and attending to some business, only became drowsy between 5 and 6 P.M. Christison[6] states, on the

[1] Am. J. Obst., 1878, xi.

[2] Gombault: Maschka, "Handb. d. ger. Med.," ii., 449.

[3] Davy: Cinc. Lancet and Cl., 1880, n. s., iv., 101. Nutt: Louisville M. News, 1881, xi., 267. Prentiss: Am. J. M. Sc., 1867, n. s., liii., 562. Trask: N. Y. M. J., 1874, xx., 165. Woodbury: Phila. Coll. and Cl. Rec., 1882, iii., 265. Hennessy: Albany Med. Ann., 1889, x., 247 (Case II.).

[4] Ziemssen: "Handb.," etc., xv., 541.

[5] Boston M. and S.J., 1857, lvi., 449.

[6] "Poisons," Am. ed., 544.

authority of Hende, that a man who took ℥ iss. of laudanum and ʒ vi. more an hour afterward was perfectly sensible seven hours later and only became unconscious in eighteen hours.

The clinical history of morphin poisoning may be divided into three stages:

The Period of Excitement.—This stage is marked by restlessness, great physical activity, loquacity, greatly increased imaginative power, frequently to the extent of hallucination, always of a pleasing character, and by increased cardiac action. This stage is of short duration, or absent in those not habituated to morphin, particularly when large doses are taken. Sometimes, without any previous excitement, the patient becomes suddenly narcotized in fifteen or twenty minutes.[1] In children the onset of the poisoning is frequently marked with convulsions, which gradually give place to narcosis. In a few instances adults have become wildly excited, and even maniacal, at this stage.[2]

The Period of Sopor.—The condition of excitement passes, sometimes rather suddenly, into an intermediate stage of diminished sensibility. The patient becomes weary, incapable of physical exertion, dull, and drowsy. He experiences a sense of weight in the extremities, and an irresistible desire to sleep, to which he finally yields if not kept awake. The sleep is at first seemingly normal, though profound. The pulse and respiration are normal. The patient may be aroused and sometimes kept awake by shaking him, by the infliction of pain or by loud calling. The face is pale, the lips are somewhat livid, the surface is covered with a cold perspiration, and the pupils are contracted. During this period the patient frequently experiences a violent itching of the skin, which is sometimes followed or accompanied by the appearance of an exanthem which may be papular, red, bluish, or almost colorless, or resembling that of urticaria or that of scarlatina. This stage in severe poisonings is always of short duration, and merges insensibly into the third:

The Period of Narcosis.—The patient can no longer be

[1] Salviat: Union méd. de la Gironde, 1859, iv., 314. Keith: Chicago M. J. and Exam., 1879, xxxix., 276.

[2] Lee: N. Y. M. and Phys. J., 1828, viii., 518. Foulke: Am. J. M. Sc., 184, n. s., xv., 568. Turchetti: Gaz. med. it. prov. Venet., 1860, iii., 226. Bergsten: Upsala Läk. För., 1872, vii., 645.

aroused, even by the most violent means. He lies motionless and senseless, with eyelids closed, or partly closed. The surface is bathed in profuse perspiration, which exhales the odor of opium in opium cases. The face is pale or lead-colored, the lips are blue or purple, the lower jaw is pendulous, the muscular system is completely relaxed, and the reflexes are abolished. The pupils are contracted to the size of pinheads, or even obliterated, and do not react to light; and the conjunctivæ are injected. At first the superficial arteries, temporals and carotids, pulsate fully, strongly, and rapidly, while the respiration is slow and shallow (80 pulsations to 4 to 8 respirations in the minute). Later the pulse becomes feeble, slow, irregular, and easily compressible, and the respiration becomes more infrequent, shallow, stertorous, accompanied by mucous râles and impeded by the tendency of the tongue to fall back. Retention of urine occurs early in the history, and continues until death or recovery.

In this period, if the case do not yield to treatment, the poisoning usually proceeds rapidly to a fatal termination. The surface of the body and even the expired air become cold. The skin is cyanosed, and covered with a cold, clammy perspiration. The pulse becomes slower, more feeble, and gradually imperceptible. The respiration is more shallow and more feeble, sometimes Cheyne-Stokes, while the râles become more pronounced. Individual muscles or groups of muscles are agitated by short clonic twitchings, and occasionally convulsions and tetanus, with trismus and opisthotonus or emprosthotonus occur. Later the muscles become completely paralyzed, the respiratory movements are made at longer intervals and finally cease, the circulation continuing for a time after the cessation of respiration. Finally the action of the heart is arrested, and death comes quietly. Sometimes epistaxis and other hemorrhages occur toward the end.

Should recovery follow after the stage of narcosis has been established, the respiration gradually becomes more frequent and more natural, the pulse becomes first perceptible and then gradually passes toward the normal, while the condition of coma passes into one of deep sleep, which may continue for twenty-four or thirty-six hours longer, although in this condition the patient may be aroused. In the great majority of cases in

which recovery has progressed so far that the patient may be aroused it will be complete. Nevertheless occasional instances have occurred in which the victim has relapsed into a deeply comatose condition and has finally died.[1]

In cases of recovery the patient, on awakening, is weary, giddy, and uncertain in his movements. He may also suffer for some hours from nausea and headache, and for a longer time from loss of appetite and derangement of digestion. If atropin have been administered as an antidote, the delirium, hallucinations, disturbance of vision, and dryness of the fauces which it causes may continue for several hours or days.

Unusual Symptoms.—Usually tetanic symptoms appear only late in the history, are not severe, and are sometimes limited to trismus. Cases have, however, occurred in which they set in early and were prominent. Thus Pellacani[2] has reported the case of a man of twenty-one years who received morphin and atropin hypodermically and whose first symptoms resembled those produced by strychnin, were violent in degree, and subsequently merged into a condition of somnolence and sopor. An analysis of the solution injected showed it to contain morphin, and to be free from strychnin and brucin.[3] Similar cases have been reported by Courtenay,[4] Anderson,[5] Shearman,[6] Tupper,[7] Parsons,[8] and Davidson.[9] When strychnin and morphin are taken together in quantities approaching equality the action of the strychnin predominates, but is mitigated by the antidotal action of the morphin.[10]

The mouth is usually dry and the patient sometimes complains of thirst, but cases have occurred in which saliva flowed from the mouth.[11]

[1] Hoffmann: Wien. med. Presse, 1877, xviii., 73. Souchon: N. Orl. M. and S. J., 1886–87, n. s., xiv., 437 (Case II.); Lancet, 1851, i., 435. Taylor: "Poisons," 3d Am. ed., 530 (two cases). Hartley: Brit. M. J., 1892, ii., 460. See also Silberstein: Med.-Ch. Centbl., Wien, 1882, xvii., 184.

[2] Riv. sper. d. freniat., etc., 1885, xi., 83.

[3] The tetanic symptoms are sometimes ascribed to thebain, which accompanies morphin in opium; but the cases here cited are all morphin cases.

[4] Brit. M. J., 1879, ii., 615.

[5] Am. J. M. Sc., 1848, n. s., xvi., 347.

[6] Med. Times and Gaz., 1857, xiv., 235.

[7] Boston M. and S. J., 1879, ci., 619.

[8] Med. Pr. and Circ., London, 1881, n. s., 507.

[9] Atlanta M. and S. J., 1885, n. s., ii., 595.

[10] Stadeler: N. Orl. M. and S. J., 1887–88, n. s., xv., 200. Marvin: Med. Herald, Louisville, 1879, i., 4.

[11] Alexander: Wien. med. Presse, 1865, vi., 754. Hennessy: Albany

Vomiting is frequently provoked only with difficulty, but in a few instances it has been known to occur spontaneously,[1] in two of which the reporter suspects that some of the morphin had been converted into apomorphin.[2]

Contraction of the pupils is one of the most constant symptoms of opium poisoning, yet in a few reports they have been described as dilated. Two of these are early cases in which the symptoms were otherwise those of opium poisoning.[3] In Brochin's case[4] the patient was in a condition of general contraction at the time of the observation; and in Cartaz' case[5] the patient, who had received the morphin hypodermically in the course of medical treatment in which habituation had been established, had hallucinations, and fell to the floor in syncope, with staring eyes and pupils widely dilated. In Salviat's case[6] the symptoms were of a tetanic type, and the pupils were "not contracted." An observation of Cheever's[7] shows that the dilatation of the pupils observed at autopsies takes place at the time of death. The pupils were under examination and "just at the instant of dissolution the pupils became largely dilated, as if moved by a spring, and remained in that condition."

The symmetrical contraction of the pupils in opium poisoning has been relied upon as an important factor in the differential diagnosis of opium poisoning from hemorrhage in the pons Varolii, which causes unsymmetrical contraction of the pupils. Two cases have been reported in which in opium poisoning the pupils were unsymmetrically contracted.[8] Nothing is said of the position of the patient in Caston's case. Stonehouse observed that the patient was lying upon the right side, and the right pupil was the smaller. Thinking that the position might be the cause of the inequality, he put the patient squarely upon his back, when the pupils became equal and remained extremely contracted.[9]

Med Ann., 1889, x., 247 (Case II.). Hawkes: Lancet, 1881, i., 209.

[1] Davy: Cincin. Lancet and Cl., 1880, n. s., iv., 101; Ph. J. and Tr., 1880–81, 3 s., x., 598.

[2] Fenyköry: Wien. med. Presse, 1883, xxiv., 208.

[3] Ann. d'hyg., etc., 1845, 212. Toogood: Prov. M. and S. J., 1841, iii., 129.

[4] Gaz. d. hôp., 1877, l., 227.

[5] France méd., 1878, xxv., 593.

[6] Union méd. de la Gironde, 1859, iv., 314.

[7] Med. Times and Gaz., 1882, i., 63.

[8] Caston: Tex. Cour. Rec. of M., 1884–85, ii., 616. Stonehouse: Albany Med. Ann., 1882, iii., 241.

[9] Taylor ("Poisons," 3d Am. ed., 529) says: "In a case referred to me in 1846 one pupil was contracted and

Six cases of **Poisoning by codein** have been reported.[1] The symptoms differed materially from those caused by morphin. There were nausea, vomiting, and abdominal pain. Although somnolent, the patients were very uneasy, with more or less redness and itching of the skin, and the sensibility, at first diminished, afterward became greatly increased. Later the pulse became weaker, and there was tendency to asphyxia. In Medvei's case the eyes were prominent, the conjunctivæ greatly injected, and the pupils somewhat dilated. In the other cases the pupils were markedly contracted. The patients were not narcotized.

Diagnosis.—*Can a diagnosis of opium poisoning be made from the symptoms alone?* The answer of this question will depend upon the force attached to the word "diagnosis" (see p. 101). While a "working diagnosis," amounting to an extremely strong probability, almost a certainty, may frequently be made from observation of the patient and his surroundings, including the history of the attack, a "positive diagnosis" cannot be made from the appearances and statements of the patient alone, and can be reached only when these are supplemented by the results of the autopsy and the detection of morphin by chemical methods, or evidence from the history of the attack and proof of the presence of morphin or opium in remains of the material which has caused the poisoning.

The conditions which resemble opium poisoning in their symptomatology are poisoning by chloral, acute alcoholism, apoplexy, uræmic coma, acute hydrocephalus in children, and in the tetanic form, strychnin poisoning. It is clear that opium poisoning may coexist with any of these. Numerous cases of combined morphin and chloral poisoning have been reported. Suicides have frequently taken large doses of opiates while under the influence of alcohol. Cerebral congestion is always observed in opium poisoning, and in persons whose arteries are degenerated cerebral extravasations or hemorrhages may be provoked by large doses of opiates. Persons suffering from Bright's

the other dilated." Apparently Dr. Taylor did not see this patient himself and he does not state the conditions under which the observation was made.

[1] Myrtle: Br. M. J., 1874, i., 478. Ambrosoli: Gazz. med. it. lomb., 1875, xxxv., 41. Walsh: Brit. M. J., 1889, ii., 718. Mettenheimer: Memorab., Heilbr., 1891–92, n. F., xi., 136. Medvei: Intern. kl. Rundschau, 1892, vi., 1457. Spratling: Med. Rec., N. Y., 1893, xliv., 81.

disease are peculiarly susceptible to the action of morphin because of deficient elimination. We have known children suffering from acute hydrocephalus to recover from severe opium poisoning and to die of the disease in a short time thereafter. Finally we have seen above that strychnin and morphin have been taken in combination. In such cases the difficulty of determining which of the two factors, if either, was the predominating cause of death is sometimes insuperable. Indeed it is, as it was in Fisk's case, sometimes difficult to determine positively whether death was finally due to a gunshot wound or to morphin subsequently administered.

Treatment.—The treatment in cases of acute opium or morphin poisoning should be directed first to removal of any of the poison still remaining in the stomach, and second, to the prevention of coma or cessation of respiration until the processes of elimination have removed that portion of the poison which has been absorbed.

The first object is best attained by thoroughly washing out the stomach with a solution of potassium permanganate, as suggested by Moor,[1] by means of the stomach tube or siphon, and leaving some of the same solution in the stomach at the end of the washing. This should be done in any case, even at the risk of temporarily arresting the respiration. The mechanical removal in this manner is preferable to the exhibition of emetics, as these frequently fail to act. It is only when solid opium has been swallowed in masses too large to enter the pipe (a most exceptional condition) that an emetic of zinc sulfate or of apomorphin is to be preferred. The washing out of the stomach should not be omitted even if a poisonous dose has been hypodermically administered, as Alt[2] has shown that morphin so administered is eliminated to a notable extent by the stomach, and that lethal doses administered hypodermically are rendered harmless by protracted washing out of the stomach. If the case be seen in its earlier stages, before the sopor has become deep, the patient should be aroused if somnolent by cold effusions to the head and spine, while the feet are immersed in hot water, followed by friction to avoid undue diminution of body temperature, by flagellation to the palms and soles, or to the back with damp

[1] Med. Rec., N. Y., 1894, xlv., 200.

[2] Berl. kl. Wochenschr., 1889, 560.

towels, or by the use of the faradic current. The patient having been sufficiently aroused is to be kept awake by the "ambulatory treatment:" walked between two persons, preferably in the open air if the weather permit, but not in the direct sunlight. There should, however, be a limit to the efforts to arouse the patient and to keep him awake. The brutal treatment of patients described in some reports is neither necessary, serviceable, nor excusable. In the use of the ambulatory treatment it must not be forgotten that in some cases the patient cannot be kept awake, and that in these a persistence in "walking" him only results in exhaustion which intensifies the subsequent coma. In such cases as well as in those in which there is threatened or actual failure of respiration, artificial respiration is to be resorted to, aided by application of the faradic current (see Treatment, p. 92). Artificial respiration, to be of service, must be persisted in, sometimes for several hours, so long as the heart's action continues. It may be aided by inhalation of pure oxygen, and is to be considered as the main reliance in opium poisoning. The use of the catheter should not be neglected, as the bladder is liable to become greatly distended by the retained urine.

Other mechanical operations have been resorted to, but are of questionable value; tracheotomy,[1] forced respiration,[2] transfusion of milk,[3] anal dilatation,[4] and massage.[5]

Of the so-called antidotes strong coffee by the mouth or rectum, or caffein hypodermically, and ether or ammonia by the same method are certainly of value. The utility, indeed the freedom from danger of atropin is questionable. While its use has been advised in recent years by Krafft-Ebing,[6] Husemann,[7] Sticker,[8] Kobert[9] and others, it has also been condemned as not only useless but dangerous by Unverricht,[10] Lenhartz,[11] Knapstein,[12] and Orlowski.[13] Strychnin and picro-

[1] Lewis: Weekly M. Rev., St. Louis, 1888, xvii., 412.

[2] Fell: Buff. M. and S. J., 1887–88, xxvii., 145; J. Am. M. Assoc., 1892, xix., 130.

[3] Gilliam: Med. Rec., N. Y., 1885, xxvii., 679.

[4] Pyle: Med. News,, Phila., 1894, lxiv., 514.

[5] Gorton: *Ibid.*, 1893, lxiii., 266.

[6] Wien. med. Presse, 1891, xxxii., 746.

[7] Penzoldt u. Stintzing: "Handb. d. spec. Therap.," 1895, ii., 306.

[8] Centbl. f. kl. Med., 1891, xii., 969; 1892, xiii., 82.

[9] "Intoxikationen," 557.

[10] Centbl. f. kl. Med., 1891, xii., 849; 1892, xiii., 49.

[11] Arch. f. exp. Path. u. Pharm., 1887, xxii., 337; Deut. Arch. f. kl. Med., 1886–87, xl., 574.

[12] "Sind Atropin u. Morphin Antidote?" Diss., Bonn, 1879.

[13] "Exper. Beitr. z. Kenntn. d.

toxin have also been recommended as antidotes; and jaborandi to favor elimination by stimulating the secretion of the perspiration.

Post-Mortem Appearances.—The autopsy reveals no lesions which are characteristic of opium poisoning. The surface of the body is frequently livid, and rigor mortis is said to be of shorter duration. The appearances are such as are usually observed after death from asphyxia: The blood is fluid and dark. The vessels of the brain and meninges are gorged with blood, and the cut surfaces of the brain substance present numerous dark-red spots. The veins of the scalp are also filled with blood. Serous effusions are often met with between the membranes, more rarely in the ventricles. The lungs are usually congested. The stomach and other viscera are normal, except that, if a preparation of opium have been taken, the odor of that drug may be observed on opening the stomach. The bladder is usually full of urine. The appearances, when present, are only confirmatory, and the autopsy is principally of interest to determine the presence or absence of other causes of death.

Action of the Animal Economy upon Absorbed Morphin.—As late as 1886 Donath[1] has reiterated the opinion, expressed by several of the earlier toxicologists, that morphin disappears completely in the organism, and is converted into no other alkaloid; consequently that neither morphin nor oxydimorphin can under any circumstances be detected in the urine. That this view is erroneous has been shown by numerous observations. Not to mention the results of earlier analyses, Dragendorff and Kauzmann,[2] Marmé,[3] Eliassaw,[4] Nolta and Lugan,[5] and Wormley[6] have obtained positive evidence of the presence of morphin in the blood and urine both after administration of notable doses by the mouth, and after hypodermic administration. Marmé also showed that morphin is converted

Einwirk. d. Atropins," etc., Diss., Dorpat, 1891.

[1] Arch. f. d. ges. Physiol., 1886, xxxviii., 528.

[2] "Gerichtl.-Chem. Nachweis des Morphins," etc., Diss., Dorpat, 1868. Dragendorff: "Beiträge z. ger. Chemie," etc., 121–139.

[3] Deut. med. Wochenschr., 1883, ix., 197.

[4] "Zur Lehre v. Schicksal d. Morphins im lebenden Organismus, Diss., Königsberg, 1882.

[5] J. de pharm. et de chim., 1884, 6 s., x., 462.

[6] Univ. M. Mag., May, 1890, reprint.

in the system, in part at least, into oxydimorphin (pseudomorphin or dehydromorphin), and that this mixture of morphin and oxydimorphin is eliminated, partly by the urine and partly by the alimentary canal. The elimination by the stomach has been further shown to occur by Alt,[1] who found by washing the stomach after hypodermic injection of morphin that about one-half of the amount injected is eliminated by the stomach, and that the elimination begins in from two to three minutes, continues for half an hour, then becomes less active, and finally ceases completely in from fifty to sixty minutes. Tauber[2] has experimentally proved the elimination of morphin by the intestine and its presence in the fæces after hypodermic administration.

It follows that in cases of poisoning by morphin, by whatever channel administered, the chances for its detection are the best in the alimentary canal, and that under favorable conditions it may also be detected in the blood, urine, and liver.

Detection.—Of the many constituents of opium a few only are of practical interest in this connection: morphin, oxydimorphin, narcotin, meconic acid, and codein. Oxydimorphin resembles morphin in many of its reactions and is of interest in view of its formation from morphin in the system, referred to above. The reactions of narcotin and of meconic acid may afford evidence that morphin found in the body or elsewhere exists there as a constituent of some preparation of opium, and not as a salt of morphin—a differentiation which may be of importance collaterally. Codein is in use as a medicine and may itself cause poisoning.

In the systematic method for the extraction of vegetable poisons (pp. 133–138) narcotin and codein pass from the alkaline aqueous liquid into benzene, and are found in residue VI.; meconic acid is extracted from the acid aqueous solution by amylic alcohol, and is found in residue VIII.; and morphin and oxydimorphin are extracted by hot amylic alcohol from the alkaline aqueous liquid, and are found in residue IX.

Or morphin may be extracted from urine or blood by the Marmé-Warnecke method,[3] or by that of Tauber.[4] In the

[1] Berl. klin. Wochenschr., 1889, 560.

[2] Arch. f. exp. Path. u. Ph., 1890, xxvii., 336.

[3] Ztschr. f. an. Chem., 1883, xxii., 634.

[4] Arch. f. exp. Path. u. Pharm., 1890, xxvii., 353.

latter method the blood, diluted with four volumes of water, or the urine if albuminous, is first freed from albumin by addition of acetic acid to faint but distinct acid reaction, boiling until the albumin is coagulated in flocks, filtering through muslin from the coagulum, and washing of the latter. Basic lead acetate is added to the mixed filtrate and washings so long as a precipitate is produced; when this has deposited, the liquid is filtered off and the precipitate washed first with water, then with ninety-five-per-cent. alcohol. The alcohol is expelled and the aqueous liquid freed from lead by hydrogen sulfid, and filtration from the precipitated lead sulfid, which is then washed with water. The filtrate and washings, freed from hydrogen sulfid by a current of air, are evaporated nearly to dryness on the water-bath. The residue is extracted with alcohol by maceration for several hours with repeated warming, filtration, and washing of the residue. The alcoholic liquid is evaporated at the ordinary temperature. If the residue be not sufficiently pure it should be redissolved in alcohol, and the solution treated with an alcoholic solution of basic lead acetate. The alcoholic liquid is then filtered from the precipitate, which is washed with alcohol, and the alcoholic solution and washings are again evaporated. The residue is extracted with water, filtered, and concentrated to a few cubic centimetres. The acid aqueous liquid is warmed, treated with finely powdered monosodic carbonate to neutralization, and allowed to cool. Under favorable conditions the morphin separates in crystals, but if the quantity present be not large, several hours or even a day elapse before any appreciable separation occurs. The filtrate from this deposit is received in a graduated cylinder, in quantitative determinations, and an addition of 1 mgm. of morphin for each cubic centimetre is allowed for the morphin remaining dissolved. The deposit may be collected upon a weighed filter, dried and weighed. With this method Tauber recovered from 93.3 to 97.3 per cent. of morphin added to blood.

The precipitation of foreign substances by basic lead acetate in aqueous and alcoholic solution as above described may be also utilized to purify residues obtained by the extraction method. It must not be forgotten, however, that this entails certain loss of morphin by its partial solubility in the alkaline liquid, and that very small quantities may fail of precipitation completely.

TESTS FOR MORPHIN.—A great number of tests for morphin has been proposed, most of them depending upon its reducing action. Of these tests six: the ferric chlorid, the Fröhde, the Pellagri, the Husemann, the nitric acid, and the iodic acid, are the most prominent, and are sufficient, we believe, when yielding the reactions distinctly and unmistakably, to identify morphin. When it is possible it is also advisable to cause the crystallization of the alkaloid (see Fig. 34, p. 724), but the failure to obtain crystals is by no means proof that the reactions obtained are due to some substance other than morphin (or oxydimorphin). While there is no difficulty in obtaining the crystals from a simple solution of the alkaloid in pure amylic alcohol, particularly if the amylic solution is not allowed to stand, but is evaporated immediately at a moderately elevated temperature, the presence of minute quantities of impurities is sufficient to prevent crystallization absolutely, while not interfering seriously with the reactions. We have repeatedly found that known solutions of morphin in insufficiently purified amylic alcohol obstinately refused to crystallize, while the residues gave the reactions perfectly; a fact which has been shown by Udránszky[1] to be due to the presence of traces of furfurol in the alcohol. Stolnikow[2] and Wormley[3] in their experiments with urine obtained residues which gave the color reactions distinctly, but did not succeed in causing them to crystallize.

1. *The Ferric-Chlorid Reaction—Robiquet's Test.*—If a fragment of morphin be moistened with a solution of neutral ferric chlorid a fine dark-blue color is immediately produced, which becomes lighter, and finally fades out. The color is discharged by the addition of alcohol, free acids, or caustic alkalies and by heat. The reaction is not so delicate as some of the others, but is more delicate with a solid residue than with a solution. According to Wormley the reaction is not satisfactory with a solution containing 0.13 mgm. of morphin, but is distinct with a solid fragment of 0.0065 mgm. According to Dragendorff the presence of strychnin interferes with the reaction; and in solutions the limit of the reaction is at 1 : 5000.

The following precautions are to be observed in the applica-

[1] Ztschr. f. physiol. Chem., 1889, xiii., 258.

[2] *Ibid.*, 1884, viii., 235.

[3] Univ. med. Mag., 1890, reprint, p. 9.

tion of the test: 1. The morphin residue is to be as free from impurity as possible. Absolute purity is not, however, essential if the alkaloid be present in sufficient quantity. Frequently the reaction will not be satisfactory with the residue as at first extracted, but will subsequently become distinctly manifest as a state more nearly approaching, but still by no means reaching, that of "isolation" is attained by purification. 2. No free acid should be present either in the residue or the reagent. The latter should be freshly prepared from the sublimed chlorid, and not from that made by the wet method. The reagent should be dilute, and only a minute drop should be added at first. 3. The reaction is not satisfactory unless the color is distinctly blue—not green or bluish-green.[1]

Oxydimorphin behaves like morphin with this test.

The following substances also give a blue color with ferric chlorid: *Arbutin*[2] gives a shade of color very similar to that obtained with morphin, but tending somewhat more to violet. Arbutin is, however, extracted from acid aqueous solution. Moreover, it gives a bright gamboge color with nitric acid; it does not respond to the Pellagri test; it does not color the chloroform purple in the iodic-acid test; it is colored almost black on being heated with sulfuric acid in the Husemann test; it gives a greenish-brown color, changing to black with sulfuric acid and potassium dichromate; and it gives a green color with chlorin water and ammonia (see below), in all of which respects it differs from morphin. *Gentianic acid* (oxysalicylic acid) gives a fine blue color with ferric chlorid; but the color is changed to dirty red on the addition of soda solution. An alkaline aqueous solution of this acid when exposed to air soon becomes fiery red, changing to brown. Sulfuric acid forms with it a yellow solution. Nitric acid forms a dark-green solution from which water separates a green powder. *Geissospermin*[3] is extracted from alkaline solutions by amylic alcohol, but also from acid solutions by benzene or chloroform. Nitric acid colors it purple; with Fröhde's reagent it gives a persistent

[1] If a reagent containing too much ferric chlorid be used in the presence of a minute quantity of morphin the color may at first have a greenish tinge, or become greenish and finally brown on standing. Should this be the case a more dilute solution of the reagent should be used.

[2] A glucosid existing in the bearberry (uva ursi) and in wintergreen.

[3] An alkaloid of Pareira brava.

blue; and it behaves like strychnin toward sulfuric acid and potassium dichromate. *Saligenin*,[1] which also gives an intense red with sulfuric acid, also gives a blue with ferric chlorid. *α Naphthylamin* with ferric chlorid is at first colorless, but soon gives a steel-blue color, gradually turning to dark violet-blue. It differs from morphin in being colored black, changing to brown, by nitric acid; in giving only a faint green, which becomes darker, with Fröhde's reagent; in coloring the chloroform amber, and forming a brownish-red liquid above with the iodic test; and in turning pink on being heated with sulfuric acid and black on addition of nitric acid with the Husemann reaction. *Alumnol*[2] gives with ferric chlorid a violet-blue color, similar to that produced by arbutin, but which does not fade. With Fröhde's reagent it assumes a faint yellowish-green color; with the iodic-acid test the chloroform remains colorless for thirty minutes, and no dark band is produced on floating on ammonium hydroxid solution; with the Husemann reaction it gives a reddish-brown, changing to cherry-red, which becomes lighter. With sulfuric acid and potassium dichromate alumnol gives a brown color. Alkalies decompose it with precipitation of aluminium hydroxid. *Phenol* (carbolic acid) gives a permanent violet-blue color with ferric chlorid. It is, however, extracted from acid aqueous solutions by benzene and to some extent by petroleum ether; it gives no color with either the Pellagri or the Husemann test, and exhibits special color reactions to which morphin does not respond. Other phenols give blue or bluish colorations with ferric chlorid, indeed morphin is itself a phenol: *Resorcinol* not only gives a violet-blue with ferric chlorid, but also resembles morphin in its behavior in the first steps of the Pellagri (*q. v.*). It does not respond to the iodic-acid test and is only faintly colored (yellow) by nitric acid. It is also extracted from acid solution by benzene.

Other substances have been referred to as resembling morphin in their behavior toward this test; salicylic acid, tannins, gallic acid, phloroglucin, vanillin, orcin, and hydroquinone (quinol), but the colors which these produce are distinctly

[1] A glucosid derived from salicin, a constituent of willow bark.

[2] Aluminium β-naphtholdisulfonate ($C_{10}H_5,OH,(SO_3)_2)_3Al_2$, used in medicine as an astringent and antiseptic.

different from that developed by morphin (see Ptomaïns below).

2. *Fröhde's Reaction.*—Fröhde's reagent[1] dissolves morphin, forming a purple[2] solution, which in a minute or two turns blue, beginning at the margin of the drop, and the color then slowly changes to dirty green, then to yellow, and finally to faint pink. The changes in color are the more rapid the smaller the quantity of morphin present. With a large amount of the alkaloid the blue color may persist for hours or even for days. The reaction is more distinct with the free alkaloid (when the quantities are minute) than with its salts, although these react also, the sulfate better than the chlorid or acetate.

The reaction, although not nearly so characteristic of morphin as the ferric chlorid reaction, is much more delicate. Dragendorff places the limit of the reaction at 0.000005 gm. with the Fröhde, and at 0.000001 gm. with Buckingham's modification. Wormley states the smallest quantity with which he obtained the blue color was 0.0001 gr. = 0.0000065 gm. With one-tenth that amount he obtained the purple only, which is not distinctive.[3] The color is discharged by hydrochloric acid, or even by dilution with water. Nitric acid changes the blue to orange-red if the quantity of morphin be not too small.

This test depends upon the reduction of the molybdenum compound by the morphin and the formation of a compound or compounds of lower oxidation, purple, blue, green, yellow, or brown in color. Indeed among the most sensitive reactions for molybdic acid is the formation of a blue color by the action of reducing agents such as sulfurous acid, or metallic zinc in acid solution. Consequently the presence of other reducing agents

[1] The reagent, *which must be freshly prepared*, is made by dissolving 0.1 gm. of sodium molybdate in 10 c.c. of concentrated sulfuric acid with the aid of heat, and cooling (Ann. d. Chem., 1861, cxx., 188). Buckingham's reagent is made in the same way, with the substitution of ammonium molybdate. The heat applied to cause solution should be moderate, else the molybdate will be reduced.

[2] The color is frequently designated as "violet." This not because of any variation in color, but from the use of the word *violet* to apply to two different tints; as there are violets having a bluish tint and others inclining to red, we use the word "violet" to apply to the shades of color, sometimes designated as "blue violet," which are found in the solar spectrum beyond the blue; and the word "purple" to apply to "red-violet," a color which is not found in the spectrum, but is a combination of red and blue.

[3] Wormley does not mention the colors succeeding the blue, although they are distinct if sufficient time be allowed for their development.

must be excluded in this as in other reduction tests. A number of other alkaloids, glucosids, etc., give violet or purple coloration with Fröhde's reagent: Apomorphin, papaverin, sabadillin, sabatrin, populin, salicin, corydalin (Aderman's), fumarin, hypoquebrachin, porphyrin, taxin, oxyacanthin, and loxopterygin. All of these, however, save the last mentioned are distinctly colored red, purple, or violet by sulfuric acid alone in the cold, which does not color morphin. The red color produced by sabatrin is formed slowly. Oxyacanthin,[1] according to Hesse, remains colorless or yellow. It is, however, extracted from alkaline solutions by ether and by chloroform. Loxopterygin, one of the two alkaloids obtained by Hesse from the red quebracho bark[2] is said by him to be colored violet (purple?) changing to blue in Fröhde's reaction, in which it closely resembles morphin, from which it differs, however, in being readily soluble in ether, chloroform, and benzene, in giving no color with ferric chlorid, and in being colored blood-red by nitric acid. Narcein is colored violet, but that color appears only as the last of a series: brown, green, red, violet. Absinthin also is colored first brown, changing to violet.

Other substances giving a blue color with Fröhde's reagent, but differing in other respects from morphin, are: Aricin, blue changing to green with Fröhde, is colored intensely green by sulfuric acid or nitric acid alone; codein, at first dirty green, only becoming blue after several hours with Fröhde, gives no blue with ferric chlorid (see reactions below); ergotinin, violet changing to blue with Fröhde, is removed from acid solution by ether or chloroform; laurotetanin, indigo blue with Fröhde, pale rose red with sulfuric acid, dirty brown with nitric acid, physiological action like that of strychnin; pareirin, blue with Fröhde, violet with sulfuric acid, blood-red with nitric acid, extracted from alkaline solution by benzene or chloroform; ceanothin, blue with Fröhde, brown with sulfuric acid, yellow with nitric acid; phlorrhizin, blue lasting for some minutes with Fröhde, yellow with sulfuric acid, changing to red on slight warming, dissolves in ammonia, the solution turning yellow, to red, to blue on exposure to air; aspidospermin and quebrachin, blue with Fröhde, but also blue with sulfuric acid and potassium dichromate, with which morphin gives a green color.

[1] One of the berberis alkaloids. [2] Ann. d. Chem., 1882, ccxi., 278.

Oxydimorphin behaves toward Fröhde's reagent in the same manner as morphin.

Bruylants[1] has recently suggested another method of using Fröhde's reagent: If morphin or one of its salts be heated with concentrated sulfuric acid as in the Husemann test (*q. v.*) and a drop of Fröhde-Buckingham reagent be added, a splendid green color is produced which persists for some time and then disappears. The same color is produced with apomorphin, oxydimorphin, codein, and narcotin, and, somewhat modified, with narcein, papaverin, and meconin.

3. *Pellagri's Reaction.*[2]—This reaction has not received at the hands of toxicologists other than the Italian the recognition which it deserves.[3] Being based upon the conversion of morphin into apomorphin, it affords, along with the ferric chlorid reaction, one of the most reliable distinctions between morphin and possible ptomaïns, whose chemical constitution, so far as determined, is so far removed from that of morphin that their conversion into apomorphin by this or by any other treatment is, to say the least, highly improbable.[4]

The test is best applied as follows: The solid substance is dissolved in fuming hydrochloric acid to which a small quantity of concentrated sulfuric acid has been added, and the mixture evaporated at a temperature of 100°–120° (212°–248° F.) until the hydrochloric acid is expelled. In the presence of morphin (or codein or apomorphin) the residue is light purple (red-violet), and even in the presence of other material carbonized by the acid, the edges of the char have a purple tinge. The watch glass is then allowed to cool, and hydrochloric acid (two drops of dilute 1 : 5) is added, when a brilliant cherry-red color is instantly produced. Solid monosodic carbonate in fine powder is then gradually added, with a drop or so of water occasionally if required, and the mixture stirred from time to time until effervescence ceases and a slight excess of the bicarbonate has been added. The red color persists for a time during neutralization, being particularly marked at the outlines of the bubbles; but as

[1] Ann. de Pharm., 1895, reprint.

[2] Gazz. chim. ital., 1877, vii., 297.

[3] It is not mentioned by Wormley (1885), Prescott (1887), Sohn (1894), or Dragendorff (1895), although it is given as one of the principal tests by Ludwig (1885), Guareschi (1892), Otto, (1893), Baumert (1893), and Vitali (1893).

[4] See Selmi: "Ptomaine," Bologna, 1881, 34, and below, pp. 760 ff.

the point of neutralization is approached it gives place to a smoky green (similar to that observed sometimes in crystals of apomorphium chlorid). If now a drop or two of very dilute alcoholic solution of iodin be added a brilliant green color is produced. This pigment is soluble in ether, forming a purple solution, which may be produced by transferring the material from the watch glass in which the reaction was performed to a test tube and strongly agitating with ether.

The evaporation at the first stage of the reaction may be effected on a water-bath, but the colors are more brilliant if it be performed in an air oven at about 115° (239° F.) In either case the reaction fails entirely if the evaporation be not continued until the hydrochloric acid is *completely* expelled, which usually requires fifteen to twenty minutes' heating.

4. *The Husemann Reaction.*[1]—This is a modification of and improvement upon the reaction previously suggested by Erdmann,[2] and depends upon the formation of a color by the action of a trace of nitric acid upon a product (sulfomorphid) resulting from the action of concentrated sulfuric acid upon morphin, either by prolonged contact at the ordinary temperature (twelve to twenty-four hours) or more rapidly at temperatures between 100° and 150° (212°–302° F.).

Morphin with pure concentrated sulfuric acid forms a colorless solution, which remains so for days, but if the acid contain traces of nitrogen compounds (which it invariably does unless purified by a tedious process) the solution, colorless at first, after a time assumes a red or violet tinge. In Husemann's test the solid material under examination is moistened with concentrated sulfuric acid (as pure as obtainable) and either allowed to stand twenty-four hours or placed in an air-bath at 100°–105° for about five minutes.[3] After the watch glass and its contents (which may have a violet or red tinge) have cooled, a pointed glass rod moistened with dilute nitric acid is brought into the centre of the drop. An intense dark violet color is immediately

[1] Ann. d. Chem. u. Pharm., 1863, cxxviii., 305.

[2] *Ibid.*, 1861, cxx., 188. Erdmann's reagent was made by dissolving six drops of nitric acid of sp. gr. 1.25 in 100 c.c. water and adding ten drops of this to 20 gm. of pure concentrated sulfuric acid.

[3] Husemann directed a heating of half an hour; this is unnecessary and the sulfuric-acid solution is more likely to be colored with the longer heating, even with a relatively pure acid.

produced, which persists for some minutes, gradually changing to blood-red from the centre outward and then becoming paler and changing to orange. The same colors are observed when nitric acid is similarly added to a solution of morphin in concentrated sulfuric acid, which has stood at the ordinary temperature for twenty-four hours. If in applying the test with the aid of heat the temperature indicated be surpassed, as when the watch glass is heated until the acid just begins to give off white fumes, and the drop cooled before addition of nitric acid, the violet color is not observed but a brilliant red color (like that of red onions) is immediately produced and persists for some time, gradually becoming paler. When nitric acid is added in the manner described to a freshly prepared and unheated mixture of morphin and sulfuric acid a faint rose color only is produced, which in a few seconds passes to yellow. Husemann examined into the behavior of this reaction with other alkaloids of forensic interest, and found none which could be mistaken for morphin. Only two, brucin and narcotin (for reactions see pp. 759 and 812), presented distant resemblances, but can be easily distinguished by other characters. According to Dragendorff,[1] Farre and Hasselden obtained this reaction with certain constituents of cloves and allspice, which are, however, extracted by immiscible solvents from acid aqueous solutions.

Husemann places the limit of delicacy of this reaction at 0.01 mgm. or 1 : 40,000.

Oxydimorphin behaves like morphin with this reaction, except that it forms a yellow solution with sulfuric acid alone.

Many variations of or additions to this sulfomorphid reaction have been suggested. Husemann himself stated that an aqueous solution of sodium hypochlorite, chlorin water, or a fragment of chloride of lime, of potassium nitrate, or of potassium chlorate might be substituted for nitric acid without changing the result.

In the first edition of his work Otto[2] stated that when morphin is heated with concentrated sulfuric acid, the solution diluted with water, and a fragment of potassium chlorate added an intense mahogany-brown color is produced.

Nadler[3] took advantage of the solubility of sulfomorphid in chlor-

[1] "Ermitt. v. Giften," 4te Aufl., 231.

[2] "Ausmitt. d. Gifte," 1856, 92.

[3] Pharm. Centralhalle, 1873, xiv., 346.

oform, with which it forms a rose-red solution. The substance under examination is boiled in a test tube with a mixture of two volumes of concentrated sulfuric acid and one volume of water, the solution cooled, supersaturated with ammonium hydroxid, cooled, and agitated with chloroform; this is colored bright rose-red. Codein gives the same reaction.

Jorissen's modification [1] differs from Nadler's principally in the degree of heat applied: the dry residue in dissolved in concentrated sulfuric acid, the solution transferred to a test tube and heated to 190°–200° (374°–392° F.) until the mass has become opaque and green-black. The product of this action is added in drops to 10 c.c. of water in another test tube, when the mixture becomes bluish. The solution is divided into two parts, one of which is shaken with ether, which assumes a purple color; the other with chloroform, which is colored blue. The reaction is not very delicate, the limit being stated by Jorissen to be 0.4 mgm. With another method Jorissen stated the limit to be 0.6 mgm., but Donny found it to be sensitive to 0.006 mgm. The residue is heated in a steam bath with concentrated sulfuric acid, a very small crystal of ferrous sulfate is added and crushed, the mixture is again heated for a minute and added to about 2–3 c.c. of ammonium hydroxid solution in a porcelain capsule. The acid solution underruns and forms a red color which is violet at the borders, while the ammoniacal solution becomes blue. If the liquids be mixed the blue alone remains; and with very small quantities of morphin only the blue is observed.

Donath [2] describes a modification of the Husemann which he claims serves to distinguish morphin from oxydimorphin: The residue is heated with eight drops of a mixture of two volumes of concentrated sulfuric acid and one of water, over a small flame, until white fumes begin to be evolved. With oxydimorphin the solution becomes bluish-green, with morphin rose-red and soon dirty. If diluted cautiously with water the oxydimorphin solution becomes rose-red and the morphin reddish. If now an oxidizing agent, nitric acid, sodium nitrite, etc., be added, the oxydimorphin yields a fine deep-violet, and the morphin a beautiful raspberry-red.

Another modification is that described in three forms by Arnold.[3] The residue is warmed with concentrated sulfuric acid, or syrupy phosphoric acid, and an aqueous or alcoholic solution of caustic potash is added, with and without previous addition of sodium nitrite.

Siebold and Gron [4] at about the same period described a test consisting in warming the residue with concentrated sulfuric acid and

[1] Ztschr. f. anal. Chem., 1881, xx., 423.
[2] *Ibid.*, 1887, xxiv., 744.
[3] Arch. d. Pharm., 1882, 3 R., xx., 561; Ztschr. f. anal. Chem., 1884, xxiii., 228.
[4] Ztschr. f. an. Chem., 1874, xiii., 236.

adding a fragment of potassium perchlorate (free from chlorate) when morphin produces a dark brown color.

Tattersall's reaction[1] consists in the formation of a dirty violet color, changing to dark sea-green when morphin is mixed with sulfuric acid and sodium arsenate. The color becomes transitorily dark gray when the mixture is heated until white fumes are evolved.

According to Donath,[2] if morphin be heated with concentrated sulfuric acid in the cold, and then a drop of a solution of potassium chlorate (1:50 of concentrated sulfuric acid [!]) added, a fine and long enduring grass-green color is produced, while the borders of the liquid are faintly rose-red.

5. *The Nitric-Acid Reaction.*—A fragment of morphin moistened with concentrated nitric acid assumes an orange-red color, changing to yellow. The color is not changed to violet on addition of stannous chlorid or of ammonium sulfid.

Many other substances give a *yellow* color with nitric acid. Only a few give orange tints: hydrastin, laudanin, physostigmin, piperin, papaverin, arbutin, alumnol. The last two have been discussed in connection with the ferric-chlorid test, and papaverin with the Fröhde. The orange solution of hydrastin is fluorescent when diluted, and the alkaloid gives a green color, changing to brown, with the Fröhde. Laudanin is colored emerald-green by ferric chlorid, and is extracted by chloroform from alkaline aqueous liquids. With physostigmin alkaline hydroxids produce a color reaction beginning with an intense red, changing successively to yellow, green, and blue. Piperin is removed from acid solutions by benzene and by petroleum ether; and with Fröhde's reagent is colored yellow, turning to brown and nearly black. Ditamin gives a series of colors: yellow, dark green, orange.

6. *The Iodic-Acid Reaction—Le Fort's Test.*—This, one of the earliest known of the morphin reactions, depends upon the reducing power of the alkaloid; and it consequently is of no value in the presence of other reducing agents, among which many animal bases are included. Under any circumstances its chief value is for differentiation between morphin and other alkaloids which are not reducing agents. It is best performed as follows: Take two small test tubes. Into one (*a*) put the solution under examination, into the other (*b*) an equal bulk of water. Add to each a granule of iodic acid and agitate; in the

[1] Chem. News, 1880, xli., 63. [2] *Loc. cit.*

presence of morphin *a* becomes yellow or brown. To each test tube add a drop of chloroform (or carbon disulfid) and agitate: the chloroform in *a* is colored red or purple. Float some very dilute ammonium hydroxid on the surface of the liquid: a brown band is formed at the junction of the layers. None of the colors mentioned should be observed in *b*, whose purpose it is to guard against the use of an iodic acid containing free iodin. Or in place of chloroform or carbon disulfid, starch paste may be used, which is colored violet or almost black by the liberated iodin. The reaction is sensitive to a solution containing 1 : 10,000, except as to the first yellow color, which is not observed beyond a dilution of 1 : 2000. Oxydimorphin behaves like morphin.

The following are the most important of the other morphin reactions:

7. Mixed with cane sugar and concentrated sulfuric acid : a wine-red color. Under like conditions oxydimorphin is colored green. This, the *furfurol* or *Pettenkofer* reaction, is also produced by codein, biliary acids, albumin, etc. Udránsky [1] has improved this reaction by substituting furfurol for sugar. A drop of a 0.5-per-cent aqueous solution of furfurol is mixed with 1 c.c. of concentrated sulfuric acid, and a drop or two of the mixture is applied to the solid residue.

8. A solution of iodic acid in sulfuric acid colors morphin dark violet, then brown (Selmi).

Morphin dissolved in glacial acetic acid, mixed with methylenaceto-chlorhydrin, and then with excess of sulfuric acid, is colored rose color and then violet. On addition of water the rose color is restored. If, after twenty-four hours, the liquid be diluted with water and ammonia added, a brown precipitate is produced which forms a purple solution with sulfuric acid (Grimaux).

10. If solution of sodium sulfid be added to a solution of morphin in sulfuric acid and the mixture warmed, a flesh color is developed which changes to violet and then to dark green. If after the addition of the sulfid a solution of potassium chlorate in sulfuric acid (!) be added, a green color, changing to violet and yellow, is produced (Vitali).

11. If a mixture of 1 part morphin, 1 part sodium nitrite, and 2 parts mercuric chlorid in 300 parts water be heated, with stirring, on the water-bath for half an hour, the mixture is colored blue, and a dark-blue deposit is formed which turns brown (Flückiger).

12. Morphin boiled with a mixture of dilute hydrochloric acid and potassium chlorate gives a colorless solution. If this be cooled and reduced with zinc for two minutes, and ammonium hydroxid added a carnation-red color is produced (Bloxam).

[1] Ztschr. f. anal. Chem., 1888, xii., 355.

13. Chlorin water colors morphin yellowish, or gaseous chlorin deep yellow. Ammonia changes the color to red or brown (Fairthorne).

14. Silver-nitrate solution is reduced by morphin in the cold. Nitric acid causes a blood-red color in the filtrate (Horsley).

15. Morphin causes a precipitate of Prussian blue in mixed solutions of ferric chlorid and potassium ferricyanid (free from ferrocyanid). This reaction, first suggested for morphin by Hager in 1873, was afterward recommended by Brouardel and Boutmy as one indicating the presence of ptomaïns. It is clearly quite distinct from the ferric-chlorid reaction above described.

16. A solution of sodium selenate, or of ammonium selenite in concentrated sulfuric acid, gives a green color which slowly changes to brown. According to Lafon, the only alkaloids which give this reaction are morphin and codein.[1]

Dragendorff gives the sensitiveness of the general reagents to morphin as follows: With 0.2 c.c. of morphium sulfate solution 1 : 5000, phosphomolybdic acid gives a faint cloudiness slowly; potassium-bismuth iodid a recognizable precipitate; auric chlorid a faint cloudiness, difficultly soluble in hydrochloric acid, rapidly becoming dark; with iodin in potassium iodid, and with bromin in potassium bromid, distinct precipitates. The same quantity of a solution 1 : 1000 gave a distinct precipitate with phosphomolybdic acid; a copious precipitate, remaining amorphous, with potassium iodhydrargrate; a crystalline precipitate after two hours with potassium-cadmium iodid; a faint precipitate with tannic acid; a slight reduction after fifteen minutes with silver nitrate. The same quantity of a solution 1 : 100 gave a slight precipitate with platinic chlorid; a crystalline precipitate with mercuric chlorid; hardly any cloudiness with potassium dichromate, and a strong precipitate with picric acid.

Reactions of Oxydimorphin.—The following means of distinction between oxydimorphin and morphin, other than those already referred to, have been described:

1. The chlorid, warmed to 100° with concentrated sulfuric acid for five minutes, is colored green, then, on addition of nitric acid, violet (see Husemann reaction above) (Boedeker).

2. When dissolved in cold concentrated sulfuric acid and then warmed after addition of a drop of a solution of ferric chlorid, 4 : 100, morphin is colored blue, oxydimorphin yellow (Lindo).

3. If oxydimorphin be dissolved in lime water and chlorin water added, a greenish-yellow color is produced, which is not

[1] C. rend. Ac. Sc., Paris, 1886, c., 1543.

changed by warming. The greenish-yellow color produced by morphin changes to red on the application of heat.

4. Oxydimorphin, when treated with solution of sodium hypochlorite (two drops) and then with sulfuric acid (eight drops) is colored emerald green; morphin dark yellow.

5. Lafon's reagent (solution of sodium selenate in sulfuric acid) gives a brownish-violet color, changing to violet with oxydimorphin; morphin is colored bright green.

Reactions of Narcotin.—Narcotin is extracted from alkaline aqueous liquids by benzene (see p. 136).

1. Fröhde's reagent dissolves narcotin, forming a green solution. If a solution containing more molybdate (0.05 gm. to 1 c.c.) be used the color is green, changing to a fine cherry-red.

2. With Husemann's reaction narcotin, on being heated with sulfuric acid, is colored a brilliant red with violet streaks, the color passing to distinct violet on addition of the nitric acid.

3. If bromin water be added in drops to a solution of narcotin in hydrochloric acid, and the liquid boiled after each addition, a rose-red liquid is produced. Bromin water added at once in excess causes a yellow precipitate (Bloxam).[1]

Reactions of Meconic Acid.—Meconic acid is extracted by amylic alcohol from acid solutions, or it may in some instances be directly extracted by alcohol containing hydrochloric acid. The alcoholic extract is distilled until about one-sixth remains, cooled, filtered, and the filtrate evaporated to dryness on the water-bath. The dry residue is dissolved in boiling water, filtered and freed from coloring matters by agitation of the filtrate with benzene. The watery solution is then heated to dryness, neutralized with magnesia, filtered boiling hot, and concentrated over the water-bath. The residue of evaporation contains magnesium meconate.

The characteristic reaction of meconic acid is that with ferric chlorid, with which it gives a blood-red color, which is not discharged by addition of either hydrochloric acid, of mercuric chlorid, or of auric chlorid solution, nor on the application of heat. It is, however, decolorized by stannous chlorid, but the color returns on addition of nitrous acid.

[1] For other reactions see Dragendorff: "Ermitt. v. Giften," 4te Aufl., 237.

Meconic acid also forms white precipitates with lead acetate, silver nitrate, and mercurous nitrate.

Comenic acid, which may be produced from meconic acid on evaporation of the hydrochloric acid solution, also gives the ferric-chlorid reaction.

Reactions of Codein.—Codein is extracted from alkaline solutions by benzene. Being closely related, chemically, to morphin, of which it is the methyl ether, it responds to several of the tests for morphin, yielding similar products of decomposition (see Morphin tests). It does not, however, give a blue color with ferric chlorid, except in the presence of concentrated sulfuric acid. With Fröhde's reagent it gives a dirty green, changing to indigo in twenty-four hours. It dissolves in concentrated sulfuric acid, forming a colorless solution, which becomes blue after twenty-four hours, or on the application of heat; if nitric acid be added the color changes to greenish, cherry red, blood red, and orange.

Can Morphin be Distinguished from Ptomains?—In all the recent trials for murder by morphin poisoning the theory that the reactions observed by the State's analysts have been, or may have been, caused by ptomaïns and not by morphin has been advanced with more or less vigor on the part of the defence. As Professor Vaughan has been one of the most vehement champions of the ptomaïn theory, and as his views upon this subject have been most recently published,[1] these may properly serve as a text for the discussion of this question, with such commentary as is furnished by the evidence in the Buchanan case.

It is conceded that no substance is known which gives the morphin reactions with the six tests above described in the same manner as pure morphin,[2] yet it has been claimed that the evidence of these tests is insufficient to demonstrate the presence of morphin unless it be supported by the further evidence of the

[1] Vaughan and Novy: "Ptomains, Leucomains," etc., 3d ed., April, 1896.

[2] Q. (To Professor Vaughan.) Do you know of any one substance which will give the color tests of the ferric chloride, the nitric acid, the Huseman, the Pelagri, the Froeder and the iodic acid?

A. I know of no substance, I know of no substance which will give these tests as pure morphine would give them. (Appeal Book, Buchanan Case, fol. 8209.) Future references to this evidence will be designated by "Buch.," the numbers referring to the folios, and excerpts made with all errors of orthography, etc.

isolation of morphin in the crystalline form. We quote: "Many of the tests for morphin employed by toxicologists are fallacious. In the examination of a stomach, and part of a liver sent from Lincoln, Neb., VAUGHAN, following the method of Dragendorff, obtained in the amylic alcohol extract from alkaline solution a residue that gave with more or less distinctness all of the principal color tests for morphin; but failing to obtain crystals that could be identified as those of this alkaloid, the absence of morphin was reported. HAINES, working with the same material, obtained similar reactions, but he also was unable to secure the crystals, and made a negative report. Afterward it was quite positively shown that death had been caused in this case by a blow on the back of the head with a heavy piece of iron.[1] It is clear that the assertion that this individual was killed by a blow upon the head is utterly irrelevant to the question of the presence or absence of morphin in his body. In the Stokes case the point of greatest contest was whether Fisk died of a gunshot wound or from the effects of morphin known to have been administered in excess, and it is patent that evidence of the presence of the bullet would not be accepted as evidence of the absence of morphin. Eliminating this factor, and if in the above quotation we take the words "all of the principal color tests" literally, and if we further interpret "more or less distinctness" to be equivalent to "clearness and certainty," we have all of the conditions which obtained in the analysis in the Buchanan case, and which would obtain in any analysis made by the Dragendorff or Erdmann-Uslar methods or their modifications in a case of known morphin poisoning, if an amylic alcohol had been used which had not been purified to almost complete elimination of furfurol, or if the amylic alcohol extract had not been promptly evaporated (see p. 747). If, on the other hand, "more or less distinctness" in the above quotation be equivalent to "more or less indistinctness" the entire statement is clearly irrelevant.

The next paragraph in our text would, if it were accurate, render this discussion unnecessary: "In the Buchanan case in New York, the symptoms as sworn to by the attending physician clearly were not those of morphin, and *all the tests obtained by the experts were duplicated with putrefactive prod-*

[1] Vaughan and Novy: *Op. cit.*, p. 285.

ucts."[1] The symptoms in this connection are only of remote interest, like the piece of iron in the case above quoted. While they were certainly not those of pure morphin poisoning they were such as would be produced by the administration of atropin at first and morphin afterward. The statement referring to the "duplication" of the tests is disingenuous.

The tests relied upon by the analysts were the ferric chlorid, the Husemann, the Pellagri, the Fröhde, the nitric acid, and the iodic acid, all of which had given the morphin reactions clearly and distinctly. These the expert for the defence *endeavored to reproduce* at the trial with a material which he said was an extract from a decomposing pancreas and with the same extract to which a tenth of a grain of morphin had been added.[2] But how, and with what results?

First the ferric-chlorid reaction. With this a brilliant grass-green color was produced, not a blue; a color as distinct from that of the morphin reaction as that of the grass is from that of the Neapolitan sky.[3] Next the Husemann reaction. In the pretence of applying this test sulfuric acid was added, and then immediately nitric acid, without the application of heat, which this witness said was "not absolutely essential." Yet

[1] The italics are ours.

[2] Buch., fol. 8085.

[3] Buch., 8092 (Professor Vaughan): I think the ferric chloride has been used (the witness mixes in one of the evaporating dishes and explains as follows): I put into the dish one or two drops from bottle A, and in the other dish we will put the same amount of B; this is the ferric-chloride test; I take a small amount of ferric-chloride and apply it to this fluid and take another rod, so that there will be no question about getting the impurities in it, and there it is.

(8093) Q. What is the result?

A. One of these contains morphin and the other does not, and now the question is which contains the morphin, and whether it is a green or a blue color.

MR. WELLMAN: Won't you state what the color is in those two dishes?

A. I would say they are green.

MR. WELLMAN: Both green?

(8094) A. Yes, sir.

(8116) THE WITNESS (displaying dishes to the jury): "These two are the ferric-chloride tests, this has been standing ever since it was passed around, you see; some authors say that the colors should be blue, others say that it should be green, you can decide as to which it is for yourself; this contains morphine, this does not (indicating the two plates)." The remainder of the answer refers to another test and is quoted below. We have sought diligently through chemical and toxicological literature, and find but one writer who describes the initial color produced on adding ferric chlorid to morphin as other than blue. Dupuy ("Alcaloides," 1889, ii., 128) calls it "a handsome blue coloration or sometimes greenish blue, when the salt of iron is relatively in excess." Yet subsequently this witness, applying the ferric-chloride reaction to pure morphin designated the color as green (fol. 8135, 8136).

the reaction is produced by the action of nitric acid upon a product which sulfuric acid only produces from morphin by contact of twelve to twenty-four hours or under the influence of heat. Therefore the conditions under which this test was here applied insured its failure.[1] (See p. 753.) Then the Fröhde, which was passed over with great celerity. In this the color was not purple (or red-violet) changing to blue and then slowly through dirty green and yellow to pink; but it was a distinct and palpable orange, changing to yellow.[2] The iodic-acid test was next per-

[1] Buch., 8094. THE WITNESS: Yes, sir. [See note, p. 762] (the witness makes a new test taking two dishes and marking them "A" and "B" respectively, and tests with the following explanation): I will put one or two drops of solution A into dish A, and the same quantity of solution B into the dish B, two drops of each; I then add a drop of sulphuric acid to each (witness does so)—now, the test is, this should be warmed, though that is not absolutely essential, and as we have not any means of doing it here we will add the nitric acid direct; a drop of nitric acid is added to each (witness does so).

(8095) MR. WELLMAN: Is that a violet gray; won't you give us the color?

THE WITNESS: I should say that it was violet; those colors are evanescent and it will disappear before it can be passed to the jury.

[2] Buch. 8097. THE WITNESS (testing). I put some molybdic acid in a test tube here and will dissolve it in a little sulphuric acid, forming what is called Froeder's reagent (witness does so); I will put the same amount of solution A in another (8098) dish (which has just been marked "C"), and the same amount of solution B in this dish (a dish just marked "D"); then we will add a drop of Froeder's reagent to each; now (passing to jury), this will have to be passed quick as it is an evanescent color; I will now make the iodic acid test; I put some iodic acid," etc.

(8139) The witness accordingly makes test. [In cross examination, applying Fröhde's reagent to pure morphin.]

Q. Is that a violet color?

A. That is a violet color (passing dish to the jury).

(8142) Q. Have you got the one before that, the purple one?

(8143) A. Of course these colors are all changed.

Q. The one you did for me just now, the Froeder test, which you did for me just now?

A. Here it is (handing).

Q. Will you show us what you got from your residue taken from the pancreas?

A. (Handing dishes.) That is after standing, of course, the colors change. The jury understand that.

Q. Did it ever have that color, the purple color which the morphine gave?

(8144) A. The color was not exactly as it is with pure morphine.

Q. It never had anything like it, did it? It was yellow from the start?

A. Yes, sir; it had a color very much like it.

Q. Well, you have got your stuff there; do it over again. Do the Froeder test with your residue that you have got there?

THE COURT: Suppose we exhaust one test before we get to another? You had already got down to the nitric acid test.

(8145) MR. WELLMAN: The nitric acid test has been passed upon and finished.

Q. Now, if you will go back a second to the Froeder test, we have got the morphine reaction; give us the reaction from your ptomains there that you have got.

A. Please understand that I do not claim that these tests are identical with those of pure morphine

formed and gave a very faint reaction with both samples. The Pellagri came next in order, or what was called the Pellagri. In it hydrochloric acid alone was used, in place of hydrochloric acid and sulfuric acid, and the mixture was not heated, although heat is absolutely necessary to the proper application of the test. (See p. 752.[1]) Finally the nitric-acid test gave not an orange-red but a yellow color.[2] It is apparent, therefore, that far from having "duplicated" these six tests, the witness misapplied two, obtained results different from these which are produced by morphin in three, and only secured a faint reaction with the iodic-acid test.

It has been claimed that indol and skatol are produced during anaërobic[3] putrefaction; that they are extracted from putrid material by the Dragendorff and Stas-Otto methods, passing into the same final residue as morphin; that they interfere with the morphin reactions when they are present; that they themselves give the morphin reactions; and that a buried cadaver is under such conditions that the putrefaction is necessarily anaërobic, and that consequently, by reason of the generation of indol and skatol under such conditions, evidence of the presence of morphin furnished by a method of extraction in which amylic alcohol is used, and by the morphin reactions is *a priori* fallacious.

Considering these propositions *seriatim:* That indol and skatol are produced, along with other substances, during strictly anaërobic putrefaction is conceded. That they pass into the amylic alcohol extract from alkaline solutions in the Dragendorff method is apparently proven by Vaughan's experiments.[4]

(the witness makes the test asked for).

(8146) Q. Your ptomains give a yellow, don't it?

A. It gives a purple, a violet color.

Q. My eyes must be rather bad. I must be becoming color blind.

A. Well, it is a violet.

[1] Buch. (8101), THE WITNESS (testing): I take some of solution B and put it in tube C, and some hydrochloric acid. Then I take some of solution A and place it in the tube marked D, with some hydrochloric acid (displaying the tubes to the jury in the light). This is A and B with hydrochloric acid, one with morphine and one without.

[2] Buch. 8120, THE WITNESS: In G I put the solution A, and in H I put the solution B. I add nitric acid to each (passing glasses to the jury) that contains morphine, and the other does not (indicating).

(8121) THE COURT, Doctor, what ought they to show, any particular color?

THE WITNESS: It is not a characteristic test, it shows a yellowish color.

[3] In absence of air.

[4] *Op. cit.*, 285–289.

That they interfere with the morphin reactions when both are present is also shown by the same experiments, if we concede the accuracy of the observer's color perception. But if this only be the case their presence would operate to conceal that of morphin, not to simulate it. They might cause a negative error but not an affirmative one. That this could occur would require the truth of the next proposition. But that neither indol nor skatol gives the morphin reactions is demonstrated by Vaughan's experiments as well as by our own. In the table on pages 766, 767, Nos. 1 to 4, 8, and 9 are Vaughan's results obtained from: 1. "A sample of indol prepared by" himself "from decomposing pancreas. It is a brown, granular substance, and is probably not chemically pure. This fact, however, does not unfit this sample for experiments on the point under consideration, because any impurities which it may contain originated in the decomposing tissue, and may be present in the same substance obtained from like tissue. No. 2. Obtained from Merck. The order was simply for 'indol,' without any specifications whether it should be synthetic or putrefactive. This sample is white and in flakes. No. 4. Obtained from Kahlbaum. Putrefactive indol was ordered, and the label is simply 'indol.' This sample consists of white flakes." No. 8. Skatol from Schuchardt, No. 9. Skatol from Kahlbaum. No. 5. Is impure indol prepared by Professor Sickels in my laboratory by anaërobic putrefaction of ox-blood fibrin following accurately the method of Salkowski.[1] It is in nacreous scales which become reddish-brown on exposure, and fuses at 67°. As indol fuses at 52° and skatol at 95°, this is probably a mixture of the two. No. 6. Indol, from Schuchardt, in white pearly scales. No. 7. Indol, from Merck, in smaller, slightly brownish scales. No. 10. Skatol, from Schuchardt, in small brownish scales. No. 11. Morphin. A comparison of the reactions observed with these products with those given by morphin (as indicated in No. 11) shows a decided lack of similarity.

Nor is the *a priori* supposition which constitutes the last of the series of propositions under discussion borne out by practical experience. We do not pretend to state at present whether or no the putrefaction in buried bodies is always as strictly anaërobic as it is assumed to be. But whatever the cause, the

[1] "Practicum d. physiol. u. path. Chem.," Berlin, 1893, 291 ff.

Reagent.	No. 1.	No. 2.	No. 3.	No. 4.	No. 5.	No. 6
Nitric acid. (See page 756.)	Bluish-black with violet border.	Reddish-brown..	Reddish-brown..	Reddish-brown..	Reddish-brown	Reddish-brown.
Sulfuric acid	Yellowish-green.	Brown	Greenish-yellow..	Brownish-red....	Light reddish-brown	Yellow.
Sulfuric acid with nitric acid.	Same as with nitric acid alone.				Reddish-brown	Reddish-brown.
Ferric chlorid. (See page 747.)	No change at first but all become greenish-blue.				No color except reddish-brown for one hour.	With strong solution Fe_2Cl_6 a slight green in 20 minutes, turning darker. With dilute Fe_2Cl_6 no green until dry, when faint green.
Iodic acid. (See page 756.)	No reduction				No reduction	No reduction.
Fröhde's reagent. (See page 750.)	Reddish, then dark blue.	Reddish, then greenish-blue.	Reddish, then greenish-blue.	Reddish, then greenish-blue.	All immediate yellowish-green, rapidly becomes intense dark green, changing after 5 minutes to blue on the borders, centre becomes darker, almost black.	
Husemann. (See page 753.)	Not given.	Not given..	Not given.	Not given.	After heating, light reddish-brown. With HNO_3 no change; fades slowly.	After heating, orange. With HNO_3 no change.
Pellagri. (See page 752.)	Not given..	Not given.	Not given..	Not given.	With cold $HCl + H_2SO_4$ faint pink. Heated to 115°, 20 minutes, brown orange-red, and light purple streaks; + 2 gtt. HCl dil. dirty light-brownish; + $NaHCO_3$ dirty dark yellow; + Tr. I. dirty yellowish.	

Reagent.	No. 7.	No. 8.	No. 9.	No. 10.	No. 11.
Nitric acid. (See page 756.)	Reddish-brown.	All become lemon-yellow.		Reddish-brown	Orange-red, changing to yellow.
Sulfuric acid	Yellow.	All become very faintly yellow.		Yellow	Colorless.
Sulfuric acid with nitric acid.	Brown.	All become more of a red than with nitric acid alone.		Orange-yellow	At first colorless, slowly purplish red.
Ferric chlorid. (See page 747.)	Faint greenish, beginning in 25 minutes.	No change.	No change.	No color except reddish-brown for an hour.	Pure blue, fading slowly. If Fe_2Cl_6 solution concentrated, greenish after a time, then brown.
Iodic acid. (See page 756.)	No reduction.	All promptly reduce the acid.		Reduces	Reduces. (See p. 756.)
Frölide's reagent. (See page 750.)	All immediate yellowish-green; rapidly becomes intense dark green, changing after 5 minutes to blue on the borders, centre becomes darker, almost black.	Green to blue.	Green to blue.	Same as Nos. 5-7	Purple. In a minute or two blue, then slowly dirty green, yellow, pink.
Husemann. (See page 753.)	After heating, dirty orange. With HNO_3 no change; after 10 minutes darker.	Not given.	Not given	After heating, orange and light purple streaks. With HNO_3 purple discharged. Orange fades in few minutes to yellow with brownish border.	After heating, very pale pink. With HNO_3 immediate brilliant reddish-purple, then red, to orange, to pale yellow.
Pellagri. (See page 752.)	With cold $HCl + H_2SO_4$ faint pink. Heated to 115°, 20 minutes brown, orange-red, and light purple streaks; + 2 gtt. HCl dil. dirty-light-brownish; + $NaHCO_3$ dirty dark-yellow; + Tr. I. dirty yellowish.	Not given.	Not given.	$HCl + H_2SO_4$ = faint yellow. Heat = light purple with brown streaks. HCl = orange, yellow. $NaHCO_3$ = dirty greenish-yellow. I. = dirty brownish.	$HCl + H_2SO_4$ = colorless. Heat = uniform pale purple. HCl = intense cherry-red. $NaHCO_3$ = light red to light greenish. I = brilliant green.

fact remains that residues have been obtained by processes involving extraction by amylic alcohol from cadavers which have been buried, residues which have not consisted of absolutely pure morphin and which have nevertheless exhibited the morphin reactions with a distinctness and purity equal to those of pure morphin. Such was the fact in the analyses in the Harris and Buchanan cases, in both of which the bodies had been buried for over a month, but by reason of adequate embalming were in an excellent condition of preservation. In the Affaire Joniaux, Bruylants obtained clear morphin reactions from such a residue obtained from a cadaver exhumed nine days after death.[1] Further, Davoll, experimenting with the cadaver of a dog buried for forty-five days, and extracting by a method involving the use of amylic alcohol, obtained residues from the viscera which failed to give either the ferric chlorid, Fröhde's, or Lefort's reaction, but did give those reactions distinctly after the addition of traces of morphin.[2]

Other putrid products have been referred to as giving reactions resembling that of morphin with ferric chlorid: Skatolcarbonic acid, in solutions of a certain degree of concentration (1 : 10,000), when treated with a few drops of hydrochloric acid and two to three drops of very dilute ferric-chlorid solution and heated, gives an intense *violet*. But solutions of greater or less concentration give a cherry color.[3] Moreover, the *blue* color which the same reagent gives with morphin is destroyed by free acids or by heat. Hydro-para-cumaric acid (para-oxyphenyl-propionic acid) gives with ferric chlorid a pale violet color. It is, however, readily soluble in ether.[4] Para-oxyphenyl-acetic acid, which is easily soluble in water, gives with ferric chlorid a faint color, at first grayish-violet, then dirty green.[5] Parakresol gives with ferric chlorid a reaction which is not blue, but dirty bluish-gray.[6] Indeed we fail to find in the voluminous literature of putrid products mention of a single substance, which could possibly find its way into the amylic extract from an alkaline aqueous solution, described as giving a blue color with ferric chlorid.[7]

[1] Bruylants: "Affaire J —— Rapports," etc., Charleroi, 1895, 12 ff.
[2] J. Am. Chem. Soc., 1894, xvi., 799.
[3] Salkowski: Ztchr. f. anal. Chem., 1885, ix., 25.
[4] Baumann. Berichte, Berlin, 1880, xiii., 281.
[5] Salkowski: "Practicum," 301.
[6] *Ibid.*
[7] Prescott ("Organic Analysis,"

In view of all the evidence we feel justified in answering the question at the head of this section with a distinct affirmative, and in holding that if a residue of evaporation of an amylic extract from an alkaline liquid, obtained by the Dragendorff or a similar method, give the six reactions of morphin described in the previous section with distinctness and purity, the proof of the presence of morphin is quite as complete as it is in the case of nitric acid, whose presence is unhesitatingly asserted by chemists upon the evidence of color reactions.

That instances have occurred in which chemists have predicated the presence of a vegetable alkaloid upon insufficient evidence is unquestioned. The Sonzogno case, in Cremona in 1873–74, so frequently quoted in this connection, in which the experts reported the presence of morphin upon the evidence of reactions in all probability due to ptomaïns, far from invalidating the position which we have taken, supports it. For Selmi, who showed the errors in the original report, did so by pointing out that the substance mistaken for morphin did not give either the Pellagri reaction, the ferric-chlorid reaction, the nitric-acid reaction, or the Erdmann reaction; and that it only resembled morphin in that it behaved as a reducing agent toward iodic acid, auric chlorid, and certain other reducible substances.[1] In a recent Portuguese case (1890) also, which has been the subject of wide comment, the experts failed to apply a sufficient number of tests, and erred in asserting the presence of morphin in a cadaver upon the evidence of a not entirely satisfactory Fröhde reaction, the iodic-acid reaction, and the formation of a green color with the Lafon test,—the last a reagent of comparatively recent (1885) introduction, whose merits have been insufficiently tested.[2]

1887, p. 369) states that "According to Selmi (1876) certain cadaver alkaloids give the blue color to ferric salts, as well as reduce iodic acid," without, however, citing the reference. The statement is probably based upon a letter from Schiff, dated Florence, Jan. 24th, 1876 (Berichte, Berl., 1876, ix., 195–96), referring to certain researches of Selmi, and stating that a ptomaïn soluble in amylic alcohol reduced iodic acid and was colored *bluish* (bläulich) by ferric chlorid. The paper here referred was published by Selmi in the Mem. Ac., Bologna, 1876, Ser. iii., vol. vi., 189–200, and is reprinted in his "Mem. sopra argomenti tossicol," Bol., 1878 (p. 12), where the words are "in verde più o meno manifesto col percloruro di ferro neutro" (in a more or less distinct *green* with neutral perchlorid of iron).

[1] "Sulle Ptomaine," Bologna, 1878, 67 ff.

[2] "Relation médico-légale de

Influence of Putrefaction on Morphin.—Morphin withstands decomposition under the influence of putrefactive changes in organic material with which it is mixed for a longer period than was formerly supposed. In the Buchanan case positive evidence of the presence of morphin was obtained after a burial of 43 days, and in the Harris case after a burial of 53 days. But in both of these cases putrefaction had been practically arrested by embalming and the bodies were well preserved. That of Helen Potts was not even discolored; the only external marks of post-mortem change were mould upon the eyes, mouth, and one arm, and the internal organs were all perfectly firm. Marmé[1] obtained certain evidence of the presence of morphin in a mass of 100 gm. of liver to which 0.01 gm. of the chlorid had been added and which had putrefied in an incubator for eight weeks.

NICOTIN—TOBACCO.

Nicotin—$C_{10}H_{14}N_2$—is the only alkaloid existing in ordinary tobacco, *Nicotiana tabacum*, which is included, along with the plants yielding the atropic poisons, in the order Solanaceæ. The alkaloid exists in all parts of the plant, but notably in the leaves, which contain from 2 to 8 per cent., the proportion being the greater in the less esteemed varieties. *Piturin*, a liquid alkaloid obtained from *Duboisia Hopwoodii* (see Atropic Poisons) is probably identical with nicotin.

The free alkaloid is, when pure, a colorless, oily liquid, which rapidly becomes brown, and finally resinifies on exposure to air. It boils at 240°–250° (464°–482° F.), suffering partial decomposition when boiled in air, but capable of distillation in vapor of water, with which it distils at lower temperatures. Its specific gravity is 1.027 at 15° (49° F.), its reaction is intensely alkaline, its odor is penetrating and peculiar, and its taste burning and caustic. It is lævogyrous $[a]_D = 161°.55$ It dissolves in all proportions in water, alcohol, and ether, and is also soluble in petroleum ether, benzene, chloroform, and oils. It forms salts which crystallize with difficulty, are deliquescent, and lose nicotin on evaporation of their solutions.

l'affaire Urbino de Freitas," Porto, 1893, 122, 208.

[1] Ztschr. f. anal. Chem., 1883, xxii., 635.

NICOTIN POISONING.

It is remarkable in view of the large quantities of tobacco consumed, its wide-spread use, and the toxic powers of the plant, that serious poisonings by tobacco are of exceptional occurrence. Less severe poisonings are common enough and are experienced by most persons upon beginning the use of tobacco, and chronic poisonings are frequently observed in those using it in excess as well as in operatives in tobacco factories.

With very few exceptions, reported tobacco poisonings have been accidental, and have for the most part arisen from the medicinal use of infusions or decoctions of the leaves, generally as popular remedies. Tobacco enemata were formerly frequently resorted to and have caused a number of poisonings, several of which have terminated fatally.[1] Severe and even fatal poisonings have also been caused by the administration of decoctions or infusions of tobacco or "pipe oil" by the mouth, as popular remedies for various disorders[2] or to provoke abortion;[3] by the use of food articles accidentally contaminated with tobacco;[4] by water saturated with tobacco smoke given to a child "to induce quietness;"[5] by chewers or "dippers" swallowing the juice;[6] by children sucking foul tobacco pipes;[7] by the application of tobacco to abraded sur-

[1] Ansiaux: J. d. chim. méd., etc., 1827, iii., 23. Grahl: J. d. prakt. Heilk., 1830, lxxi., 4 St., 100. Eade: Lancet, 1849, ii., 480. Paris and Fonblanque: "Med. Jur.," ii., 418. Pereira: "Mat. Med.," ii., 494, 579 (4 cases). Christison: "Poisons," Am. ed., 650–51 (7 cases). Beck: "Med. Jur.," 12th ed., ii., 869. Tavignot: Gaz. méd. de Paris, 1840, 2 s., viii., 763.

[2] Shaw: Phila. Med. Times, 1878, viii., 528; Ph. J. and Tr., 1864–65, n. s., vi., 91. Turchetti: Gazz. med. it. prov. venet., 1860, iii., 226. Hjorth: Gaz. d. hôp., 1852, xxv., 362. Evans: Lancet, 1869, i., 843.

[3] Landerer: Schweiz. Ztschr. f. Pharm., 1868, No. 11, 72.

[4] Posner: Alg. med. Centr. Ztg., 1894, xviii., 481. Lion: *Ibid.*, 233. Morgan: Brit. M. J., 1875, ii., 487 (in well water, one death). Barkhausen: Pr. Ver.-Ztg., 1836, vii., 33.

[5] Weaks: Bost. M. and S. J., 1853, xlvii., 461.

[6] Skae: Edinb. Med. J., 1855–56, i., 643 (an insane man); Ph. J. and Tr., 1864–65, n. s., vi., 341. Hardman: Atlanta M. and S. J., 1884–85, n. s., i., 648. Oppolzer: Wien. med. Presse, 1866, viii., 1151. Brit. M. J., 1873, ii., 520. Wormley: "Micro-Chem. of Poisons," 2d ed., 436.

[7] Brit. M. J., 1877, ii., 389; Med. Chir. Centbl., Wien, 1882, xvii., 1429.

faces, and even to the uninjured skin;[1] and by excessive smoking.[2]

We find no record of a tobacco poisoning distinctly suicidal in origin. In Skae's[3] case tobacco was intentionally swallowed by a lunatic, and another lunatic was severely poisoned by snuff, swallowed "during a lucid interval."[4] A fatal poisoning reported by Rabot[5] appears to have been suicidal, although the account does not so state.

Taylor refers to two poisonings by tobacco in which there was suspicion of homicidal intent;[6] and Christison,[7] upon the authority of Ogston, relates the case of a man fatally poisoned by snuff in a brothel. The poet Santeul is said to have died from the effects of snuff put into his wine at the dinner table of the Prince de Condé.[8]

We find record of six instances of *poisoning by nicotin* in the human subject,[9] all of which were fatal. Of these four were suicides,[10] and the account of one refers only to the analysis.[11] The remaining case (Affaire Bocarmé) was the *cause célèbre* in which Stas first used his process for the separation of alkaloids from organic mixtures. In this instance the poison was administered by force.[12]

Duration.—The action of nicotin may be quite as rapid as that of hydrocyanic acid. One suicide fell dead with the vial from which he had taken the poison still in his

[1] O'Niell: Lancet, 1879, i., 296. v. Meyern: Pr. Ver.-Ztg., 1844, xiii., 33. Gallavardin: Gaz. d. hôp., 1864, xxxvii., 387. Coletti: Gazz. med. it. prov. venet., 1864, vii., 309. Allan: Lancet, 1871, ii., 663. Blanchard: France méd., 1869, xvi., 128.

[2] Dow: Pac. M. and S. J., 1880–81, xxiii., 308; Lancet, 1885, ii., 1114. Broomhead: Med. Chron., Manchester, 1888–89, ix., 473. Richardson: Asclepiad, London, 1889, vi., 132. Marshall Hall: Edinb. M. and S. J., 1816, xii., 11. Gmelin: "Pflanzengifte," 550.

[3] *Loc. cit.*

[4] Christison: *Op. cit.*, 651 (Caillard).

[5] Pharm. Jahresb., 1883–84, 1149.

[6] "Poisons," 3d Am. ed., 770.

[7] "Poisons," Am. ed., 651.

[8] Julia Fontenelle: J. d. chim. méd., etc., 1836, 2 s., ii., 652, *ex* "Mém. d. Saint Simon."

[9] We find reference to two others: Borsarelli and Bruno: Giorn. d. r. Ac. d. med. d. Torino, 1867, 3 s., iv., 745, and Simons: Nederl. Tijdschr. v. Geneesk., 1877, 2 R., xiii., 233, to which we have not access.

[10] Ph. J. and Tr., 1858–59, xviii., 46. *Ibid.*, 1859–60, n. s., i., 195. Fonssagrives and Besnon: Ann. d'hyg., etc., 1861, 2 s., xv., 404. Johnson: Lancet, 1890, ii., 337.

[11] Boutmy: Ann. d'hyg., etc., 1880, 3 s., iv., 201.

[12] "Procès du comte et de la comtesse de Bocarmé devant la cour d'assises de Hainault," Mons., 1851. Stas: Bull. ac. roy. d. méd. d. Belg., Brux., 1851–52, xi., 202–312. Tardieu: "Empoisonnement," 2ème ed., 945.

hand;[1] another died in a few moments, and another was found dead. In Johnson's case[2] death did not occur so rapidly. The patient was found upon a doorstep, bleeding from a severed jugular vein, and having vomited. The vein was ligated and he was taken to the hospital, where he died suddenly, without convulsions. The amount of blood lost had been insufficient to account for the death, and 0.191 gm. (2.95 grains) of nicotin was separated from the stomach and the alcohol in which it had been preserved. In the Bocarmé case death occurred within five minutes.

The duration of fatal tobacco poisoning varies within wider limits. Ansiaux[3] states that a woman of twenty-eight years was given an enema prepared by boiling ℥ ij. of the leaves in water, whereupon she cried out that she felt as if drunk, and immediately expired. Tobacco enemata have caused death in 15 minutes in two instances,[4] and in Rabot's case[5] a preparation of tobacco used by gardeners to destroy insects proved fatal within the same time. In Tavignot's case[6] an enema caused the death of a man in 18 minutes, and in one of Christison's cases the administration of a tobacco enema was followed by death in 35 minutes.

Skae's patient died in 7 hours after admission. A child whose death was attributed to its use of a foul tobacco pipe in blowing soap bubbles died in 4 days,[7] and one of Morgan's[8] patients died in 5 days.

Lethal Dose.—The smallest quantity of nicotin capable of causing the death of a human being is not known. In the reported cases large doses were probably taken, as in Johnson's case above cited, and in the Bocarmé case, in which 0.4 gm. (6.2 grains) was obtained from the stomach. It is probable, however, that as small a quantity as two or three drops of the freshly prepared and pure alkaloid might cause death. As the proportion of nicotin in different varieties of tobacco, raw and prepared, varies from two to eight per cent., the effects of a given dose must also vary. Pereira[9] cites a case in which

[1] Ph. J. and Tr., 1859–60, n. s., i., 195, *ex* Lancet, Aug. 23d, 1859.
[2] *Loc. cit.*
[3] *Loc. cit.*
[4] Grahl: *Loc. cit.* Beck: *Loc. cit.*
[5] *Loc. cit.*
[6] *Loc. cit.*
[7] Brit. M. J., 1877, ii., 389.
[8] *Ibid.*, 1875, ii., 487.
[9] "Elem. Mat. Med.," 1872, 619.

death was caused by an infusion of 30 grains (2 gm.) of dry tobacco leaves, given as an enema.

Symptoms.—Nicotin, like hydrocyanic acid, may cause death without any subjective symptoms. In one case "the deceased stared wildly; there were no convulsions, and he died quietly, heaving a deep sigh in expiring."[1] In Johnson's case, complicated with an incised wound, severing the jugular, the patient had vomited when he was found, resisted all attempts to introduce the stomach tube, and died suddenly without convulsions. Smaller doses of nicotin produce unpleasant sensations in the mouth and throat, salivation, headache, vertigo, dulness, sleepiness, dimness of vision, photophobia, deafness, increased frequency of respiration, weakness, prostration, paleness, cold extremities, attacks of syncope, vomiting, diarrhœa, trembling of the extremities, and stupor.[2]

In grave cases of tobacco poisoning the same symptoms are observed, accompanied by severe chills, abdominal pain, præcordial anxiety, and deep stupor or violent convulsions, with both tonic and clonic spasms. The pupils are generally contracted in the beginning and subsequently dilated, frequently with alternations of contraction and dilatation. In fatal cases death is due to apnœa. In non-fatal cases great weakness, trembling, and giddiness persist for days and even for weeks.

The toxic effects caused by excessive smoking are the same as those caused by absorption of nicotin from the alimentary canal. In some cases the symptoms have begun suddenly with violent convulsions, and in some death has occurred rapidly during an acute attack in the course of a chronic poisoning.[3] Batten[4] has reported the case of a man of thirty-nine years in whom smoking caused a disposition to fight. When the smoke is not inhaled the poisoning is due to nicotin only, which is partly dissolved by the saliva and probably also absorbed by the buccal mucous membrane. The alkaloid is volatilized by the heat of the combustion, and according to Loewenthal[5] fifty to sixty per cent. of that contained in the tobacco passes into the

[1] Taylor: "Poisons," 3d Am. ed., 771.

[2] Boutron-Charlard: J. de pharm., 1836, xxii., 689. Dworzak u. Heinrich: Husemann u. Hilger, "Pflanzenstoffe," 2te Aufl., 1175.

[3] See cases referred to on p. 772, and Bigelow: New York M. J., 1876, xxiii., 399. Bryden: Lancet, 1879. i., 334.

[4] J. Am. Med. Assoc., 1887, ix., 415.

[5] "Nikotingehalt des Tabakrauchs," Diss., Würzb., 1892.

smoke. When the smoke is inhaled, not only is the absorption of nicotin more complete, but, as Gautier has shown,[1] the carbon monoxid, also produced during the combustion, is absorbed to some extent from the lungs, but not from the mouth.

Treatment.—The stomach (or rectum) should be washed out with water to which tannin or a solution of iodin in potassium iodid has been added. Cold affusions to the head while the body and extremities are kept warm are indicated to overcome stupor, and the administration of stimulants, ether, whiskey, etc., hypodermically, inhalation of ammonia, and coffee internally. Electricity, inhalations of oxygen, or even artificial respiration should be resorted to to supplement the failing respiration.

Post-Mortem Appearances.—These are not characteristic except in certain cases when the odor of tobacco is detected in the stomach, or even pervades the entire body, or when tobacco in substance is found in the stomach contents. The appearances of death from apnœa are present—*i.e.*, a dark, fluid condition of the blood, a dark color and hyperæmic condition of the lungs and liver, and congestion of the vessels of the brain and meninges. If death have not occurred rapidly the appearances of a more or less intense gastro-enteritis are observed.

Analysis.—The separation of nicotin from the stomach contents and from the viscera has been accomplished in several instances. Stas obtained the alkaloid not only from the stomach of Fougnies, the victim of Bocarmé, but also from the tongue, liver, and lungs, and even from the floor, upon which a portion had been spilled, and which had been washed with hot water shortly after the murder. Lehmann[2] in 1853 obtained nicotin from the stomach of a youth who had died in one hour after he had swallowed "sauce" used in preparing chewing tobacco. Ogston[3] in 1834 separated nicotin from the stomach contents in a case of poisoning by snuff. Taylor,[4] Fonssagrives and Besnon,[5] and Boutmy[6] separated nicotin from the stomach contents in cases of poisoning by the alkaloid. In Johnson's case of poisoning by nicotin[7] 0.03 gm. (0.46 grain) of the alkaloid was obtained from the stomach contents, and 0.188 gm. (3.9 grains)

[1] "Poisons de l'air," 229–237.
[2] Arch. d. Ph., 1853, lxxvi., 144.
[3] Christison: "Poisons," Am. ed., 648, 651.
[4] "Poisons," 3d Am. ed., 772.
[5] Ann. d'hyg., 1861, 2 s., xv., 404.
[6] *Ibid.*, 1880, 3 s., iv., 201.
[7] Lancet, 1890, ii., 337.

from the alcohol in which the stomach had been preserved. In Rabot's case of poisoning by tobacco extract,[1] 2.25 gm. (34.7 grains) of (impure?) nicotin were separated from the stomach contents, and evidences of its presence in the liver and urine were obtained.

In the process of extraction of alkaloids by immiscible solvents nicotin, like coniin, passes from the alkaline aqueous liquid into petroleum ether, and is consequently to be looked for in residue V. (see p. 136). It may also be separated by distillation, and in the process for volatile poisons it passes into the distillate from the alkaline liquid (see p. 129). The residue may be purified by conversion into chlorid or oxalate, extraction with alcohol, decomposition of the salt with caustic soda, and extraction with petroleum ether.

The characters by which nicotin may be recognized are the following:

1. The residue is oily, colorless or yellowish, strongly alkaline, reddens phenolphthalein,[2] and gives off a peculiar acid odor, somewhat resembling that of stale tobacco smoke, and producing irritation of the nasal mucous membrane and conjunctiva. If left exposed to light the oil becomes yellow or brown and thicker, and finally resinous. When heated it volatilizes completely, giving off white fumes.

2. If a glass rod moistened with hydrochloric acid be held over the watch glass containing the alkaloid, or if drops of the alkaloid and acid be placed upon two watch glasses and one inverted over the other, a white cloud is produced, which is not so dense as that produced by coniin under like conditions, but no crystals are produced as with coniin.[3]

3. *Auric chlorid* produces a yellow or brown, amorphous precipitate in neutral solutions of nicotin salts, which is nearly insoluble in acetic or hydrochloric acid, but readily soluble in caustic alkalies. A cloudiness is produced slowly in a solution of 1 : 10,000.

4. *Platinic chlorid* in rather strong solutions of the alkaloid or of its salts produces immediately a yellowish precipitate which is amorphous at first and afterwards becomes crystalline,

[1] Ph. Jahresb., 1883–84, 1149.

[2] According to Heut nicotin in alcoholic solution does not redden phenolphthalein (see p. 706).

[3] For this and other distinctions between nicotin and coniin see p. 706.

or in solutions of the chlorid is crystalline immediately. With more dilute solutions, up to 1:5000 of the chlorid, the precipitate is more slowly formed and is crystalline. When amorphous and small in amount the precipitate is somewhat soluble in hydrochloric acid, when crystalline it is much less soluble. It dissolves on heating the liquid and slowly reappears in the crystalline form. Wormley figures this crystalline precipitate, and considers its formation, along with the odor and physical state of the nicotin, sufficient "to distinguish this alkaloid from all other substances."[1]

5. *Mercuric chlorid* gives a white precipitate with free nicotin, which is at first white and curdy, afterward crystalline. The precipitate, which is formed in solutions up to 1:3000 (slowly) dissolves in a solution of ammonium chlorid, from which it is redeposited after a time; and is also soluble in hydrochloric or acetic acid. *Mercuric cyanid* does not precipitate with the free alkaloid; but a crystalline deposit is formed when a neutral solution of the chlorid of the alkaloid is added to a saturated solution of the cyanid.

6. *Roussin's Reaction.*—If an ethereal solution of iodin be added to a solution of nicotin, likewise in ether, a brownish precipitate is produced in solutions of 1:100, which, after some hours, is converted into long needle-shaped crystals. With more dilute solutions, to 1:500, the crystalline deposit is more slowly formed, without the previous production of an amorphous precipitate.

7. Chlorin gas colors the free alkaloid brown or blood-red. The substance formed dissolves in alcohol, from which it crystallizes on evaporation.

8. The behavior of nicotin toward other general reagents is as follows: *Potassium-cadmium iodid*, in acid solution a cloudiness up to 1:10,000 (Sohn). *Phosphomolybdic acid*, cloud up to 1:40,000 in acid solution. *Picric acid*, added in great excess in alcoholic solution, an amorphous precipitate which subsequently becomes crystalline, 1:5000 (Dragendorff), 1:40,000 (Wormley). *Iodin in potassium iodid*, in acid solution, a reddish-brown precipitate, 1:250,000 (Wormley), 1:1000 (Guareschi). *Bismuth-potassium iodid*, in acid solution cloud at 1:40,000 (Dragendorff). *Mercuric-potassium iodid*,

[1] "Micro-Chem. of Poisons," 2d ed., 442, Pl. vi., Fig. 1.

in neutral solution a white, amorphous, resinous precipitate, 1 : 15,000 (Dragendorff), 1 : 25,000 (Sohn). *Tannic acid*, a white precipitate soluble in warm hydrochloric acid, and reappearing as the solution cools, 1 : 500 (Dragendorff), 1 : 10,000, (Wormley). *Potassium thiocyano-platinate*,[1] forms a yellow, crystalline precipitate in solutions of nicotin salts, 1 : 3000 (Guareschi). *Platino-potassium iodid*, in a solution of nicotin containing excess of acetic acid, produces an immediate black precipitate, while the reagent is decolorized. With coniin a very faint turbidity at most is produced and the red color of the reagent remains (Selmi).[2]

9. *Physiological Test.*—The frog is most susceptible to the action of nicotin. With a dose of $\frac{1}{200}$ to $\frac{1}{100}$ of a drop the animal is at first uneasy and, in about five minutes or less, becomes apathetic and assumes a peculiar position, with the forelegs pressed backward against the sides, and the hind legs drawn up so that the thighs stand at right angles to the body, and the legs fixed so that the feet rest upon the back. If the extremities be moved from this position they immediately resume it on liberation. The respiration, at first accelerated, becomes slow. There are slight muscular spasms, particularly in the hind legs. With larger doses, $\frac{1}{60}$ to $\frac{1}{10}$ drop, the injection is immediately followed by a severe clonic convulsion, followed by immobility in the position described; diminution in frequency or arrest of respiration, without previous acceleration, strong fibrillar contractions, marked diminution of reflex irritability, and general muscular relaxation, which persists for twenty to forty hours. As the animal recovers the respiration is first re-established, at first with difficulty and at long intervals; later there are slight muscular movements and a return of reflex irritability. The heart's action continues later than the functions of the nervous centres, even when the animal finally dies; the frequency of the beats being at first diminished, then increased, and again diminished.[3] Small birds are killed by doses of $\frac{1}{4}$ to $\frac{1}{2}$ a drop of nicotin. The respiration becomes panting, the bird falls upon

[1] Obtained by adding 4–5 gm. chloroplatinic acid to a solution of 40 gm. potassium thiocyanate in 400 c.c. water, heating to 70°–80°, agitating, cooling, and recrystallizing the crystals (Guareschi: "Alcaloidi," 26, 218).

[2] "Memorie sopra argomenti tossicologici," Bologna, 1878, 61. See also p. 64.

[3] Rosenthal u. v. Anrep: Arch. f. Anat., Physiol., etc., Phys. Abth., 1879, Suppl. Bd. 167; 1880, 209. Compare with cicuta, p. 711.

its side in a strong convulsion, the body arched backward, and the heart continues to beat, although more slowly, for a time after apparent death, which occurs in ten minutes or less.

PTOMAINS.—Oily, alkaline, basic substances are frequently produced during putrefaction. Those described bear a closer resemblance to coniin than to nicotin,[1] and, like Schwanert's base,[2] differ from both nicotin and coniin in their odor, which is frequently rather pleasant than disagreeable, and in being much more volatile. Wolckenhaar obtained from a highly putrid intestine, six weeks after death, a liquid base, extracted by ether from alkaline solution, which was at first yellow, and soon became brownish-yellow, and which volatilized completely without becoming resinous. It was strongly alkaline, and emitted an odor similar to that of nicotin, but more powerful and resembling that of fresh poppy heads, and which was not destroyed by neutralization with oxalic acid. It also differed from nicotin in its behavior toward bismuth-potassium iodid, auric chlorid, iodin solution, mercuric chlorid, and platinic chlorid, in yielding no crystals, even after long standing, with iodin in ethereal solution, and in having no poisonous action.[3]

QUANTITATIVE DETERMINATION.—The complete recovery of nicotin from organic mixtures is not accomplished either by distillation or by extraction by immiscible solvents. To determine the quantity of the alkaloid contained in the residue of evaporation of the petroleum-ether extract, or in the distillate, the alkaline material is neutralized with oxalic-acid solution, concentrated to a small bulk at a low temperature, and the residue extracted with alcohol. The alcohol is evaporated at a low temperature and the residue extracted with ether, after having been rendered alkaline with caustic soda. From this point either Kissling's or Papovici's method may be followed. In both the alkaloid is brought into aqueous solution, and is determined in the former by titration with standard acid, and in the latter by the polariscope.[4]

[1] See p. 708.

[2] Berichte, Berl., 1874, vii., 1332.

[3] Otto: "Ausmittel. d. Gifte," 6te Aufl. 96, *ex* Corr.-Bl. d. Ver. anal. Chem., 1878, i., 33, 37. Husemann (Maschka: "Handb. d. ger. Med.," ii., 463) refers to Otto and Selmi as having obtained ptomaïns closely resembling nicotin. With the exception of Otto's reference to Wolckenhaar we find no reference to nicotin-like ptomaïns in the writings of either author, although both discuss coniin-like ptomaïns in the places referred to.

[4] For the details of the methods

STRYCHNIN—NUX VOMICA, ETC.

The species of *Strychnos* and other members of the *Loganaceæ* contain the two tetanizing alkaloids *strychnin* and *brucin* or other allied bases. The Carolina jasmine (*Gelsemium sempervirens*) is included in this order and has been already considered.[1] Brucin and strychnin always accompany each other, and occur principally in the seeds of *Strychnos Ignatia* (St. Ignatius' beans), and of *S. nux vomica* (nux vomica), in the bark of the same tree (false Angostura bark), in the root bark of *S. colubrina*, and in the *S. tieuté* (Upas tieuté or radja), the most deadly of arrow poisons, Java). The South American arrow poison *curare*, *woorara*, or *woorali*, from *S. Guianensis*, contains the alkaloid *curarin;* and an arrow or ordeal poison from the west coast of Africa, known to the natives as *akasga*, *m'boundu*, *ikaja*, and *quai*, is probably derived from a species of strychnos, and contains an alkaloid, *akazgin*. A third alkaloid, *igasurin*, was described by Desnoix[2] as existing in nux vomica, but Shenstone[3] has demonstrated its non-existence.

The proportion of strychnin and brucin in nux vomica appears to vary within wide limits. Pelletier and Caventou[4] obtained from 0.28 to 0.4 per cent. of strychnin. The maximum percentage of strychnin obtained has been about half a per cent.[5] The percentage of both alkaloids was found to be 2.3 per cent. by Dragendorff, 2.56 to 3.57 per cent. by Dunstan and Short,[6] and 2.25 per cent. by Shenstone.[7] While brucin thus predominates over strychnin in nux vomica, the reverse is the case in St. Ignatius' beans, and in upas tieuté, which contains a mere trace of brucin and nearly 1.5 per cent. of strychnin.[8]

Strychnin—$C_{21}H_{22}N_2O_2$—crystallizes from alcohol in an-

see Kissling: Ztschr. f. an. Chem., 1882, xxi., 64; 1883, xxii., 199. Papovici: Ztschr. f. physiol. Chem., 1889, and xiii., 445. Also Lowenthal: *Op. cit.*

[1] See p. 715.

[2] J. de pharm., 1854, 3 s., xxv., 202.

[3] J. chem. Soc., London., 1881, xxxix., 457.

[4] Ann. d. chim. et de phys., 2 s., 1819, x., 142; 1824, xxvi., 44.

[5] Husemann u. Hilger: "Pflanzenstoffe," 2te Aufl., 1284.

[6] *Ibid.*

[7] *Loc. cit.*

[8] Moens: Ztsch. f. Chem., 1866, 2 s., ii., 288.

hydrous four-sided prisms with four-sided pyramidal points, or from benzene in octahedra (Fig. 35). It may also form crystalline scales, or a granular powder by rapid evaporation. Ammonium hydroxid precipitates it from dilute aqueous solutions of its salts in delicate needles. It is colorless and odorless, but intensely bitter in taste, a quality which is recognizable in a solution as dilute as 1 : 100,000. Its salts are also bitter. It is alkaline in reaction. It is lævogyrous; $[\alpha]_r = 132°.08$ to $136°.78$ in alcoholic solution, but acid solutions

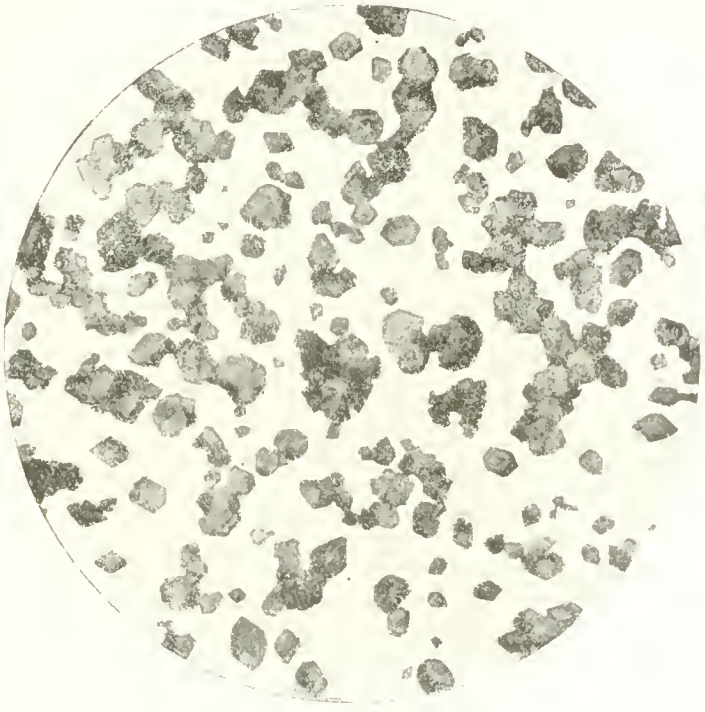

FIG. 35.—Crystals of Strychnin, from Solution in Benzene. × 75.

have a much lower rotary power. Strychnin dissolves in 6,667 parts of cold water and in 2,500 parts of boiling water, in 120 parts of cold alcohol of sp. gr. 0.863, less soluble in more aqueous alcohol, in 200 parts of amylic alcohol, in 5 to 7 parts of chloroform, in 160 parts of benzene, in 1,250 parts of commercial ether, and in 12,500 parts of petroleum ether. Absolute ether does not dissolve it. Most of the salts of strychnin are soluble in water: the nitrate in 60 parts of cold and in 2 parts of boiling water; the sulfate (neutral) in 50 parts of cold water; the chlorid in 50 parts of cold water; the acetate, which crystallizes with difficulty, in 96 parts of cold water. Strychnin is extracted from aqueous solutions of its salts by agitation, immediately after

addition of slight excess of ammonium hydroxid, with chloroform, benzene, or a mixture of equal measures of chloroform and ether.

Strychnin is a powerful monacid base; it not only neutralizes the strongest acids with the formation of salts, but decomposes solutions of many metallic salts with separation of the oxids, or formation of double salts. (For reactions see p. 806.)

Brucin—$C_{23}H_{26}N_2O_4$—crystallizes from aqueous alcohol in transparent, four-sided prisms, containing 4 Aq., which are lost *in vacuo* over sulfuric acid; or, by more rapid evaporation, it forms plates resembling those of boric acid, or cauliflower-like tufts. It is alkaline, lævogyrous, and very bitter in taste. The crystalline alkaloid dissolves in 320 parts of cold water and in 150 of boiling water; that which has lost its water of crystallization requires 850 parts of cold, and 500 parts of boiling water for solution. It dissolves in 1.5 parts of alcohol, in 2 parts of chloroform, and in 64 parts of benzene. Amylic alcohol dissolves it readily, petroleum ether and oils with difficulty, and ether not at all. It is extracted from aqueous solutions of its salts, after addition of excess of ammonium hydroxid, by chloroform, or benzene. It is a monacid base, forming salts which are bitter and, for the most part, soluble in water and crystalline. (For reactions see p. 812.)

Curarin—$C_{10}H_{15}N$($C_{18}H_{35}N$, Sachs)—crystallizes from its chloroform solution in four-sided prisms which are very hygroscopic, very bitter, faintly alkaline, and extremely poisonous. It is soluble in water and alcohol in all proportions, difficultly soluble in amylic alcohol and in chloroform; insoluble in ether, benzene, petroleum ether, carbon bisulfid, or turpentine. It is consequently not removed from aqueous alkaline solutions by agitation with immiscible solvents. Its salts are very soluble in water, crystallizable with difficulty, bitter and poisonous.

Boëhm has described a second alkaloid, *curin*, which exists in many samples of curare, but which is non-toxic.[1]

Akazgin is a white amorphous substance, which crystallizes with difficulty in small prisms from its alcoholic solution, obtained by Frazer[2] from a West African arrow poison. It is alkaline, bitter, and tetanizing; soluble in 13,000 parts of cold

[1] Berichte, Berl., 1887, xx., c. 143. [2] Brit. and For. Med.-Chir. Rev., 1867, lxxix., 210.

water, in 60 of absolute alcohol, in 16 of eighty-five-per-cent. alcohol, in 120 of absolute ether, and also soluble in chloroform, benzene, and carbon bisulfid. It causes tetanus, although somewhat more slowly than strychnin, and, like curarin, behaves in the same manner as strychnin towards the sulfuric-acid and dichromate test.

STRYCHNIN POISONING.

Historical and Statistical.—An Arabian, Serapion the younger, was probably the earliest medical writer who referred to nux vomica.[1] Wepfer describes experiments performed by himself upon animals with nux vomica in 1676–77.[2] Valentine in 1680, in an expert opinion declared seeds of nux vomica seized upon a prisoner to be poisonous.[3]

Strychnin, discovered in 1818 by Pelletier and Caventou, remained a "rare chemical" for many years. Christison, in his first edition (1829), states that "except the hydrocyanic acid, no poison is endowed with such destructive energy as the strychnia." He also describes the effects of the alkaloid upon animals, but in this as well as in the second (1832) and third (1836) editions he is silent concerning its action upon the human subject. At about this time, probably, Thomas Griffiths Wainwright was making use of the alkaloid to expedite the collection of life insurances. His uncle, George Griffiths, died in 1828, from the effects of strychnin it was subsequently supposed. In 1830 his sister-in-law expired after a short illness, caused by the administration apparently of tartar emetic at first and strychnin afterward. Her mother had died previously in the same year with similar manifestations. A Norfolk gentleman, with whom Wainwright travelled upon the Continent, died in convulsions at Boulogne in 1831. All of these lives were insured for the benefit of Wainwright, who returned to England in 1837, was afterward convicted of forgery and transported to Tasmania, where he died in 1852.[4]

Richter, in 1834, related a case of dangerous, although not

[1] About the end of the eleventh Century, "De Simpl. Med.," Argentorat., 1531, clxiii., p. 115.

[2] "De Cicuta Aquatica," Basil., 1679, p. 194.

[3] "Corpus juris med.-leg.," Francof., 1722, p. 219.

[4] Life of Wainwright, A. S. Allen, in "Lives of Twelve Bad Men," Th. Succombe, 1894. Taylor: "Poisons," 3d Am. ed., 103.

fatal, poisoning caused by the medicinal use of strychnin.[1] Blumhardt[2] was probably the first to record the death of a human being caused by strychnin: a youth, aged 17, who committed suicide by taking two scruples (!) of the alkaloid.

A collation of 529 cases of strychnin poisoning shows a relatively high proportion of suicidal and homicidal poisonings and a correspondingly small number of accidental cases. Of the whole number 264, or 49.9 per cent., were suicidal; 150, or 28.3 per cent., were accidental; and 100, or 18.9 per cent., homicidal. Another peculiarity is that the great majority of the victims of strychnin have been adults. Of the 528 cases only 44 were in children less than 10 years of age.

Accidental poisonings have for the most part arisen from mistakes in medication, either administration of overdoses or of the wrong medicine, but most frequently by pharmacists' errors in compounding. Strychnin has been thus dispensed in place of calomel, morphin, zinc valerianate, caffein, quinin, James' powder, santonin, and salicin. Proprietary medicines have caused strychnin poisoning in a few instances. Children have been poisoned by eating strychnin pills; and several poisonings in the human subject have resulted from the use of strychnin to destroy foxes, squirrels, wolves, or dogs. In one instance a man was severely poisoned by the last dose of a medicine containing strychnin, which tasted unusually bitter.[3] Three instances are referred to of infants having suffered from the effects of strychnin absorbed in their mother's milk, in one case fatally.[4]

Suicidal strychnin poisonings are of much more frequent occurrence in England than elsewhere, owing to the general use of vermin-killers (Battle's, Board's, Gibson's, Adshead's, Barber's, Hunter's, Butler's, Marsden's) containing strychnin, packages of which containing more than enough strychnin to destroy an adult are sold for three pence.[5]

Excluding the Wainwright poisonings above referred to, the earliest homicidal poisoning by strychnin to which we find reference in medical literature occurred in Canada in 1851.

[1] Wibmer: "Wirking," etc., v., 250.

[2] Med. Corr.-Bl. d. Würtemb. aerz. Ver., 1837, vii., 1.

[3] Thomson: Brit. M. J., 1893, i., 406.

[4] Lancet, 1869, ii., 241; i., 733; i., 872.

[5] Letheby found the threepenny package of Battle's vermin-killer, that most extensively used, to weigh 14 grains and contain 3.22 grains (0.21 gm.) strychnin.

Of the 100 homicides of which we have information as having occurred since that date 8 appeared in 1850–59, 19 in 1860–69, 24 in 1870–79, 40 in 1880–89, and 9 in 1890–95. Forty-five occurred in the United States, 24 in England, 9 in Finland, 7 in Germany, 7 in France, 3 in Canada, 3 in Australia, 1 in Italy, and 1 in Java.[1]

Homicides by Strychnin.—1. (1851) Report of trial of Azenath Smith for the murder John Freeman, *alias* Elijah Pease. Defendant lived with deceased as his wife. Symptoms characteristic. The poison was probably twice administered in pills. Strychnin found on analysis (Dupson). Acquitted.[2]

2. (1855) Peo. *v.* Geo. W. Green, Chicago, Ill. Convicted of the murder of his wife. Strychnin found (Blomey).[3]

3. (1856) Reg. *v.* William Palmer. Convicted of the murder of John Parsons Cook, at Rugeley, November 21st, 1855. Symptoms characteristic. Analysis failed (Taylor). The principal contention was concerning the chemical evidence.[4]

4. (1856) Reg. *v.* William Dove. Convicted of the murder of his wife at Leeds, March 1st, 1856—a crime no doubt suggested by the newspaper accounts of the Palmer investigation. Defence insanity. Symptoms characteristic. Strychnin found (Nunneley).[5]

5. (1858) A woman in Louisiana confessed to an attempt to poison her husband by strychnin in a cup of coffee, of which he drank half.[6]

6. (1858) A mere mention of the fatal poisoning of a man in a Southern State. Strychnin found.[7]

7. (1858) State *v.* Edward Robbins, tried in Ohio for the murder of Nancy Holly. Strychnin detected (Cassels).[8]

[1] As strychnin poisonings are now of frequent occurrence, and as the symptomatology rarely departs from the normal type, the cases reported in medical literature are but a small proportion of those which actually occur.

[2] Brit. Am. J., 1851, vii., 102. Testimony in full.

[3] Northwest M. and S. J., 1855, xii., 75. Taylor: "Poisons," 3d Am. ed., 679.

[4] Barnet and Buckler: "Queen *v.* Wm. Palmer, official report of minutes," etc., Lond., 1856. "The Times Report," etc, Lond., 1856. Gilbert: "Report," etc., Lond., 1856. Bennett: "Verbatim Report," etc., Lond., 1856. "The Most Extraordinary Trial of," etc., Lond., 1856. "Letter to Lord Chief-Justice Campbell by the Rev. Thomas Palmer," etc., Lond., 1856. Taylor: "On Poisoning by Strychnine," Lond., 1856. Browne and Stewart: "Reports of Trials for Murder by Poison," Lond, 1883, 85–232. Also the English medical journals of that year, *passim*, and all works on toxicology subsequent to that date.

[5] Assoc. M. J., 1856, ii., 637. Ph. J. and Tr., 1856–57, xvi., 115. Browne and Stewart: *Op. cit.*, 233–268.

[6] Elmer: N. Orl. M. News and Hosp. Gaz., 1858–59, v., 313.

[7] Crawcour: *Ibid.*, 223.

[8] Lawrence and Wormley: Ohio M. and S. J., 1864, xvi., 10–27, Robbins *v.* State, 8 Ohio, 131.

8. (1859) A man poisoned by strychnin in liquor at San Antonio, Texas.[1]

9. (1860) A man was tried in Perry County, Pa., for the murder of his wife. The body was exhumed six weeks after death. Strychnin was not detected (Reese).[2]

10. (1860) A girl of eighteen years died in England from the effects of Battle's Vermin-Killer purchased by herself. Strychnin found in the stomach (Letheby). In 1854 an infant of the same family had died suddenly in convulsions; in 1859 another child with similar symptoms, and a few days before the girl's death a boy had been attacked with similar symptoms, but recovered under prompt treatment.[3]

11. (1860) State *v.* Rich. S. Richardson, New Hampshire, indicted with Sarah Ann Healey for the murder of Stephen Healey, March 6th, 1860. No symptoms described. Strychnin detected; 2.48 grains extracted from stomach contents (Hayes). Richardson convicted of murder, second degree; Healey acquitted.[4]

12. (1861) Reg. *v.* Buckle. A man attempted to poison his wife with strychnin mixed with sugar. She was suspicious and did not take it. Acquitted. Subsequently convicted of attempting to murder her with laudanum.[5]

13. (1861) Reg. *v.* Vyse. A woman poisoned her two children with Battle's Vermin-Killer and attempted suicide by cutting her throat. Strychnin found in both stomachs and livers (Attfield). Defence insanity. Acquitted.[6]

14. (1862) Reg. *v.* Vamplew. A girl of thirteen years convicted of manslaughter of an infant in her charge by giving it Battle's Vermin-Killer. There was reason to believe that she had destroyed two other infants by similar means in two other families.[7]

15. (1862) Com. *v.* Hersey. Tried in Massachusetts for the murder of Betsy Tirrol.[8]

16. (1862) Reg. *v.* Burke. Convicted of the murder of his wife. Strychnin administered mixed with Epsom salt. Strychnin detected: more than 3 grains separated from stomach contents (Blyth).[9]

17. (1863) Peo. *v.* Dan'l E. Salisbury, tried in Cortland County, N. Y., for the murder of his wife.[10]

[1] King: Am. J. M Sc., 1859, n. s., xxxvii., 573.

[2] Am. J. M. Sc., 1861, n. s., xlii., 409.

[3] Ph. J. and Tr., 1859–60, n. s., i., 621. Paley: Brit. M. J., 1860, ii., 604.

[4] Bost. M. and S. J., 1861, lxiv., 101, 131, 181, 269, 283.

[5] Ph. J. and Tr., 1861–62, n.s., iii., 390, 629.

[6] *Ibid.*, 1861–62, n. s., iii., 627; 1862–63, iv., 43, 89.

[7] *Ibid.*, 1862–63, n. s., iv., 139. Taylor: "Poisons," 3d Am. ed., 683.

[8] Yerrinton: "Report of the Case of Geo. C. Hersey," etc., Boston, 1862. We have not seen this report.

[9] Ph. J. and Tr., 1862–63, n. s., iv., 90; Med. Times, Aug. 2d, 1862. Taylor: "Poisons," 3d Am. ed., 690.

[10] Am. J. M. Sc., 1864, n. s., xlviii., 399–418.

18. (1863) State *v.* Mary Freet and L. Converse, tried in Ohio for the murder of Wm. Freet. Strychnin detected (Wormley).[1]

19. (1864) Case of Capt. Jarrey, tried at Dunedin, New Zealand, for the murder of his wife. Strychnin detected in stomach.[2]

20. (1864) Affaire Grisard. Convicted at Dieppe of the murder of a woman (Pégard). Strychnin found in the viscera (Tardieu, Lorrain, and Roussin).[3]

21. (1865) A girl of nineteen years died from the effects of pills containing strychnin, apparently given her to produce abortion. Coroner's jury found verdict of manslaughter.[4]

22. (1865) Fall Demme-Trümpy. Dr. Demme and Frau Trümpy were accused of the murder of the latter's husband. After the trial of Demme, in which he was acquitted, he ran off with Flora Trümpy, the daughter of the deceased and of his former paramour. A false report that the two were drowned in the Lake of Geneva was followed by the finding of their dead bodies, three weeks after Demme's acquittal, in bed in a hotel at Nervi, Italy, poisoned by morphin. Strychnin was found in the stomach and intestines (Schwarzenbach and Flückiger).[5]

23. (1867) A woman was fatally poisoned in Austria by strychnin dissolved in wine, sent by a relative under the pretence that it was a cure for epilepsy, from which she suffered. Three bottles were sent, only one of which contained the poison. Analysis of the viscera failed (Maschka).[6]

24. (1867) A man in Austria poisoned himself and his two children. All died. Strychnin in crystals found in all three stomachs.[7]

25. (1869) Reg. *v.* Langford. A man and an infant were fatally poisoned at different times by the wife and mother. Strychnin found in the viscera of the child, not in that of the man (Letheby). Acquitted. Defence insanity.[8]

26. (1869) A questionable case.[9]

27. (1870) Reg. *v.* Callandine. Woman in England administered strychnin to her husband upon two occasions, to which she confessed. He died some time after in an infirmary. Sentenced to fourteen years penal servitude.[10]

[1] Lawrence and Wormley: Ohio M. and S. J., 1864, xvi., 95, 190, 377.

[2] Austral. M. J., 1865, x., 321.

[3] Tardieu: "Empoisonnement," 2ème ed., 1125. Chapuis: "Précis de Tox.," 591.

[4] Ph. J. and Tr., 1865–66, n. s., vii., 240.

[5] Emmert: "Der Criminal-Process Demme-Trümpy," Wien, 1866. Wien. med. Halle, 1864, v., 480, 503, 525, 536, 550. Espagne: Montpel. méd., 1865, xiv., 54, 119. Maschka: Vrtljschr. f. d. prakt. Heilk., 1865, lxxxvi., 69–112.

[6] Maschka. Samml. Gutacht. Prag. med. Fak., 1867, 3 F., 280.

[7] Maschka: Viertljschr. f. prkt. Heilk., 1868, iv., 19. Friedreich's Bl. f. ger. Med., 1868, xix., 440.

[8] Ph. J. and Tr., 1869–70, n. s., xi., 159.

[9] *Ibid.*, p. 370.

[10] Brit. M. J., 1870, ii., 270.

28. (1870) A man was poisoned by strychnin placed in a sack of flour by an unknown enemy. He recovered.[1]

29. (1870 ?) A woman in Germany was poisoned by her husband by strychnin in coffee. He had made previous attempts. She was acquainted with the bitter taste of the poison and perceived it in the coffee yet drank of it. In the treatment morphin was given. The spasms became less intense and she died comatose. Two dogs ate of coffee and died with strychnin symptoms. Four contradictory "Gutachten." Strychnin detected in the viscera and coffee. Defendant convicted.[2]

30. (1871) Reg. *v.* Edmunds. A woman convicted of the murder of a boy of four years by strychnin administered in "chocolate creams."[3]

31. (1871) A man accused of having murdered his wife. Strychnin detected in the stomach (Calvert).[4]

32. (1871) Case of Mrs. Groocock. Poisoned her infant child and attempted suicide with Battle's Vermin-Killer.[5]

33. (1871) Case of Blackburn (Ohio ?). The accused and his mistress resolved upon mutual suicide. He handed her strychnin, which she took eagerly and died. He either repented of his resolve or never intended to take the poison himself.[6]

34. (1872) Two hunters in Germany were in the habit of using strychnin to kill foxes, etc. One was accused of having attempted to destroy the other by mixing the same poison with his food. Acquitted. Apparently the alleged victim had put the poison into the food himself to support a false accusation.[7]

35. (1873) Reg. *v.* King. Defendant twice tried for the murder of of his father in Dublin. Mistrial and acquittal. It was admitted that the deceased had died of strychnin poisoning.[8]

36. (1873) Edward McCormick was found dead February 3d, near Redwood Falls, Minn. Was last seen alive January 31st. His brother was charged with his murder. Body exhumed February 12th. Strychnin but no brucin found in the stomach.[9]

37. (1873) A woman in a Western State killed her three children and attempted to kill herself with strychnin. She recovered and was sentenced to life imprisonment.[10]

[1] Bard: Phila. M. Times, 1870–71, i., 316.

[2] Preitner: "Ueber Strychninvergiftung," Diss., Würzb., 1870, 6.

[3] Ph. J. and Tr., 1871–72, 3 s., ii., 176, 196, 215, 236, 597.

[4] *Ibid.*, 916.

[5] *Ibid.*, 1872–73, 3 s., iii. 94.

[6] Bartholow and Whittaker: Cincin. Clin., 1871, i., 196, 208.

[7] Schumacher: Friedreich's Bl. f. ger. Med., 1873, xxiv., 36.

[8] Med. Press and Circ., 1874, n. s., xvii., 153.

[9] Hand and Boardman: Northwest. M. and S. J., 1873–74, iv., 364.

[10] Hays: Pac. M. and S. J., 1874–75, n. s., viii., 389.

38. (1874) Reg. *v.* Chas. Stansfield. Convicted of having attempted to murder his wife by vermin-killer in beer. She did not take it. A sediment in the cup was strychnin.[1]

39. (1874) State *v.* Major. Convicted of the murder of his wife in New Hampshire, December 20th, 1874. Analysis showed the presence of 1.72 grains of strychnin in the stomach contents, 0.51 in the stomach, and 0.23 in the liver (Wood).[2]

40. (1874) State *v.* Magoon. Tried for the murder of his wife in New Hampshire, August 31st, 1874. Body exhumed August 12th, 1875; 0.15 grains of strychnin separated from the stomach and 0.23 grains from part of the liver; it was also detected in the intestine. The accused confessed to the crime after a disagreement of the jury and died, probably a suicide, before the second trial.[3]

41. (1875) State *v.* Stickles. A woman in Iowa tried for the murder of her husband by strychnin. Mistrial. Strychnin detected in the liver (Hatfield). There was evidence that the deceased had taken a medicine containing tincture of nux vomica and liquor potassæ. This would cause precipitation of the strychnin, nearly all of which may have been taken in the last dose.[4]

42. (1876) Reg. *v.* Silas Barlow. Convicted of the murder of Eliza Soper, who lived with him as his wife, by Battle's vermin-killer. Traces of strychnin found in the stomach (Bernays).[5]

43. (1876) State *v.* Hendricks. A woman in South Carolina tried for the murder of her husband by strychnin in whiskey. Strychnin found in stomach contents (Baruch). The possibility of strychnin being an impurity of whiskey was discussed during and subsequent to the trial. Acquittal.[6]

44. (1877) Affaire Toulza *dit* Rapala. A man in France was accused of the murder of his wife (æt. twenty-eight), who came home at 10:30, was heard to cry out at 10:45, had frightful spasms and died at 11 o'clock. Autopsy three months after death. Analysis negative as to strychnin. An alkaloid was found which did not correspond in reaction with any known alkaloid (Filhol). Defence apoplexy.[7]

45. (1878) An examination as to the cause of death of V—— A——, supposed to have been murdered by strychnin. Ciotto obtained the color reaction of strychnin from extracts from the cadaver, exhumed twenty-five days after death. This Italian case appears to have been

[1] Ph. J. and Tr., 1874–75, 3 s., v., 96.

[2] Hayes: Amer. Chemist, 1875, vi., 202. Wharton and Stillé: Med. Jur., 4th ed., ii., 625.

[3] Hayes: *Loc. cit.* Wharton and Stillé: *Op. cit.*, 203.

[4] Chicago M. Times, 1876, 7, viii., 389.

[5] Browne and Stewart: *Op. cit.*, 268–275.

[6] Baruch: Tr. So. Car. M. Assoc., 1877, 84–94.

[7] Caussé and Bergeron. Ann. d'hyg., etc., 1878, 2 s., l., 272.

the earliest strychnin poisoning in which the question of ptomaïns was raised (see p. 817).[1]

46. (1879) State *v.* T. P. Bowman, North Carolina. Convicted of the murder of his wife. Strychnin found in the cadaver (Redd).[2]

47. (1879) Peo. *v.* Harriet Merrihew, Lewis County, N. Y. Convicted of murder in the second degree for the killing of her husband. A cousin, Winthrop M., was also indicted.[3]

48. (1879) Elijah Smith, New Haven, Conn., attempted to poison a family by strychnin in coffee. Confessed.[4]

49. (1875–79) In June, 1875, the wife of S——, a Bavarian veterinary surgeon, died in two hours after having received medicine at the hands of her husband, and having suffered violent tetanic spasms. The body was exhumed four months later and an analysis failed to show the presence of strychnin. S——, who had fled to England, returned, was tried and aquitted. This case was made the subject of discussion as to the possibilities of detecting strychnin in a cadaver four months after death.[5]

50. (1880) A woman of seventy years and her son, aged forty, were poisoned (in Pennsylvania?) by strychnin, "given with criminal intent" in whiskey, but they recovered.[6]

51. (1880) State *v.* Lucey E. Meeker and Almon Meeker. Convicted in Vermont of the murder of Alice Meeker. Strychnin detected in viscera (Witthaus).[7]

52. (1880) State *v.* John Dyer, DeKalb County, Ind. Convicted of the murder of his wife. Strychnin detected.[8]

53. (1880) Affaire Delaporte. An attempt at Paris to poison four persons by strychnin in coffee.[9]

54. (1880) An attempt by a servant to poison his master. Incidental mention only.[10]

55. (1881) Peo. *v.* T. N. Cream. "Dr." Cream convicted at Belvidere, Ill., for the murder Dan'l Stott. Stott's wife was also charged with the crime jointly with Cream. Nearly four grains of strychnin were found on analysis (Haines). Cream was sentenced to life imprisonment. (See Case No. 87.)[11]

[1] Ciotto: "Parte chimica di un caso di Perizia," etc., Padova, 1880. Selmi: "Ptomaine," etc., Bologna, 1881, 187–236.

[2] N. Y. Herald, Aug. 30th, 1879.

[3] *Ibid.*, May 12th, 13th, 14th, 1879; Aug. 23d, 25th, 29th, 1880.

[4] *Ibid.*, June 8th, 1879.

[5] Ranke (*et al.*): Arch. f. path. Anat., etc., 1879, lxxv., 1–23. Ann. d'hyg., etc., 1881, 4 s., v., 385–389 (abst.).

[6] Roberts: Med. Times., Phila., 1880, x., 323.

[7] Case unpublished, see note, p. 164.

[8] N. Y. Herald, Jan. 19th, 1880.

[9] Thibaut: "Des alcaloides des strychnées," Thèse, Lille, 1886, 108.

[10] Austral. M. Gaz., 1881–82, i., 105.

[11] Texas M. and S. Rec., 1881, i., 418.

56. (1882) May Booth, a negress of fourteen years was convicted in Surrey County, Va., of the murder of two persons by strychnin mixed with their food.

57. (1882) Mrs. Mary Martin was convicted of murder in the first degree, at Emporia, Kans. for the killing of a woman whose life was insured in her favor.

58. (1882) Sally Story was charged at Paterson, N. J., with the murder of Mrs. Feest, by strychnin administered as quinin.

59. (1882) A demented girl poisoned an entire family in Tennessee. The father died.[1]

60. (1882) Affaire Martiné. A man convicted at Brugéres (France) of the murder of his niece, æt. twenty-four, to obtain insurances upon her life. His younger brother had died two years previously under similar conditions. Strychnin was detected in the stomach and brain of the woman (Garnier and Schlagdenhauffen). The defence attributed the reactions to ptomaïns.[2]

61. (1882) Case mentioned by Blackwell.[3]

62. (1882) A woman in England killed her infant and herself with Adshead's vermin-killer.[4]

63. (1882) An attempt to poison a man by strychnin in a cake sent through the mail from Louisville, Ky., to Nashville, Tenn.[5]

64. (1882) A woman in Germany poisoned her seven-year-old daughter and herself. Strychnin was determined quantitatively in the stomach and liver of the child. The cadaver of the woman had been buried and was exhumed in about eleven months. Strychnin was detected in the stomach and liver of this also (Bischoff).[6]

65. (1882) A woman named Seybolt poisoned herself and four children in Chicago. All died.[7]

66. (1883) Four prisoners in Java were poisoned by strychnin, apparently administered by their jailer in coffee. They recovered.[8]

67. (1883) A woman near Quebec was charged with the murder of her husband by strychnin. Case of Emma C. Coates.[9]

[1] Cases 56 to 59 are taken from the secular press.

[2] Garnier and Schlagdenhauffen: J. d. ph. et de chim., 1882, 5 s., vi., 342. Tourdes: Méhauté, "De l'empois. p. l. strychnine," Lyon-Paris, 1888, 40. Garnier: Rev. méd. de l'Est, 1883, xv., 40.

[3] Med. and S. Reptr., Phila., 1883, xlviii., 671

[4] Lancet, 1882, i., 537.

[5] In the Graves case death was caused by arsenic in whiskey sent through the mail from Providence, R. I., to Denver, Col. We have found a sample of sherry wine, alleged to have been sent to a person for the purpose of causing his death, to contain a large quantity of strychnin.

[6] Wolff: "Einige Fälle von Strychninvergiftung," Diss., Halle, 1887.

[7] N. Y. Herald, June 11th, 1882.

[8] Bolhalter: Rev. d. sc. méd., 1884, xxiv., 94.

[9] N. Y. Herald, Sept. 3d, 12th, 1883.

68. (1883) Cook Teat was convicted of the murder of his wife by strychnin. Prov. Ontario.[1]

69. (1884) Case mentioned in *Lancet*, 1884, I., 581.

70. (1885) State *v.* A. B. Morse, Nebraska. Tried for murder of his wife. Mistrial. Strychnin found in the stomach (Clark).[2]

71. (1885) Com. *v.* Wm. Reed, Virginia. Tried for the murder of his wife. Analysis failed. Autopsy five months after death.[3]

72. (1886) Case reported by Marston. Administration in porter. Strychnin found in stomach and washings (Blunt).[4]

73. (1886) Peo. *v.* Isaac Griffin, Alleghany County, N. Y. Convicted of murder in the second degree for the killing of Christ. Fuller. Strychnin found in the stomach (Witthaus).[5]

74. (1886) Affaire Rouan. Trial for wife murder. Strychnin found in the viscera of the deceased, and in those of a hog which had eaten of the poisoned soup.[6]

75. (1888) A family at Windsor, Mo., was poisoned by strychnin put into the coffee by a negress, aged twelve. The girl was compelled to drink of the coffee and died.[7]

76. (1889) A woman in Germany destroyed her illegitimate child by "Weizen" (wheat impregnated with strychnin, used to destroy vermin). She confessed.[8]

77. (1889) Affaire Hoyet. A pregnant girl of sixteen years destroyed by strychnin, administered in three pills. Strychnin obtained on analysis, 0.25 gm. from 80 gm. stomach and 90 gm. contents (Garnier).[9]

78–86. (1880–93) Nine cases of homicide by strychnin reported to have occurred in Finland during these years. Seven of these victims were females, two males. One of these cases, that of a schoolmaster poisoned by his wife, is probably that of Aina Sainio. Strychnin was found on analysis in all cases save one.[10]

87. (1892) Reg. *v.* Thos. Neill. Executed in England for the murder of Matilda Clover by strychnin. This defendant had been previously convicted of murder by strychnin in Illinois in 1881 (see No. 55 above).[11]

88–89. (1893) Two women were poisoned by strychnin administered in capsules by an unknown man, in England.[12]

[1] N. Y. Herald, Nov. 16th, 1883, Nov. 6th, 1884.

[2] Clark: Omaha Clin., 1893–94, vi., 83.

[3] Va. M. Mthly., 1885–86, xii., 659.

[4] Lancet, Lond., 1886, ii., 442.

[5] Not reported.

[6] LHôte et Pouchet: Ann. d'hyg., 1888, 3 s., xx., 319. Chapuis: "Précis de Tox.," 2ème ed., 600.

[7] N. Y. Times, June 4th, 1888.

[8] Mittenzweig: Ztschr. f. med. Beamte, 1889 ii., 257.

[9] Ann. d'hyg., 1890, 3 s., xxiv., 501.

[10] Fagerlund: Vrtljschr. f. ger. Med., 1894, 3 R., viii., Supplhft. 92. See also N. Y. Herald, Sept. 23d, 1892; N. Y. Evening Post, Sept. 22d, 1892.

[11] See daily papers of the period—N. Y. Herald, June 21st, 24th, 25th, 28th, 29th; July 1st; Oct. 18th, 19th; Nov. 16th, 1892.

[12] Harley: Lancet, 1893, i., 83.

90. (1893) Two persons were tried for murder by strychnin poisoning at Meadville, Pa. The death occurred in July, 1892. An expert testified that strychnin could not be detected after a year's burial. Acquittal.[1]

91. (1895) State *v.* Maggie Kuhn. Tried at Greensburg, Ind., for the murder of her husband, June 9th, 1894. Mistrial. The deceased had taken a medicine containing tincture of nux vomica four or five days before his death. The defence invoked this as an explanation of the presence of strychnin, and also attacked the reliability of the chemical tests. There was also question of the number of spasms which may occur in strychnin poisoning.[2]

The remaining nine cases are briefly referred to in secular journals. In one a woman met her death by swallowing a poisoned lemonade which she had prepared for her husband.

Lethal Dose.—The minimum lethal dose of strychnin for adults may be placed at about 0.03 gm. (half a grain). That quantity has proved fatal in two instances, in both of which the amount of the dose was accurately fixed. In one a woman of twenty-two years died in five hours from the effects of half a grain of strychnin administered through mistake of a hospital nurse.[3] The other was the case of Dr. W. C. Warner, at the time a member of the Vermont Legislature, who took half a grain of strychnin dispensed in mistake for morphin to the use of which the deceased was habituated. The first spasm came on in about five minutes and lasted about five minutes. After an interval of the same duration he had a second spasm, during which he died, in about 14 minutes after having taken the poison.[4] Two deaths are reported from three-quarters of a grain (0.048 gm.), one that of a girl of thirteen years,[5] the other that of a man of twenty-eight years.[6] In the latter, however, Marsden's vermin-killer was taken by a suicide, and the dose is consequently estimated. In two instances adults have died from the effects of one grain (0.065 gm.).[7] The minimum lethal dose for young children may be placed at 0.004 gm. ($\frac{1}{16}$ grain) which quantity is said by Taylor, upon the authority of Blacklock, to have caused the death of a child of two to three years.[8] This

[1] Communication of Dr. J. P. Hassler, Meadville, Pa.

[2] Communication of Dr. Saml. Kennedy, Shelbyville, Ind.

[3] Lancet, 1856, ii., 291, 302.

[4] Bost. M. and S. J., 1847, xxxvi., 209. Brit. Am. J., 1847, iii., 105.

[5] Watson: Lancet, 1846, i., 73.

[6] Ogston: *Ibid.*, 1856, i., 428.

[7] Lancet, 1873, ii., 533. De Courcillon: Med. Arch., St. Louis, 1869, iii., 31.

[8] "Poisoning by Strychnine," 138.

must, however, be considered as exceptional, as we find no record of another instance in which the death of a child has been caused by less than six times that quantity.[1]

Adults have recovered after having taken very large doses, as much as 0.97,[2] 1.3,[3] 1.75[4] and even 2.6[5] gm. (15, 20, 27, and 40 grains). Some cases have been reported which seem to indicate a cumulative action by repeated doses of strychnin, or, at all events, an excessive action of relatively small doses in persons who have previously taken smaller doses, or have even established a certain tolerance. Thus Booth reported the death of a man of forty-six years, which followed in two and a half hours after his having taken one and a half grains, he having previously taken repeated smaller doses.[6] Pereira refers to the case of a paralytic who, while under treatment by strychnin, the dose having been increased from $\frac{1}{8}$ grain to $\frac{1}{3}$ grain and then to $\frac{1}{2}$ grain, died rather suddenly in a tetanic spasm.[7]

Duration.—Usually in fatal strychnin poisonings death occurs within one hour, and is rarely delayed beyond 2 hours. In 77 of 143 fatal cases in the reports of which the duration is stated, it was less than 1 hour, in 32 it was from 1 to 2 hours, in 10 from 2 to 3 hours, in 9 from 3 to 4 hours, and in 15 longer than 4 hours. If the patient survive for 2 hours the probability of recovery is great.

In a number of reports death is said to have occurred "soon," or "suddenly." In one instance a man took $1\frac{3}{4}$ grains of strychnin and an equal quantity of nux vomica in consequence of a druggist's error. "Very soon" afterward he com-

[1] Gallard: Ann. d'hyg., et ., 1865, 2 s., xxiii., 386. Two deaths: boy of 11 years, 0.03 gm.; girl of 5 years, 0.025 gm.

[2] Lee: Med. Bull., Philadel., 1883, v., 82. Parker: Med.-leg. J., New York, 1884–85, ii., 375. Berry: Phila. M. Reg., 1887, i., 566. Gobrecht: Tr. M. Soc. Pa., 1870, 70, with ʒi. chloroform.

[3] Conner: Ohio M. Recdr., 1879, iv., 12. Gray: Brit. M. J., 1880, i., 486. Seargent: Louisv. M. News, 1883, xv., 225. Wallace and McRae: Brit. M. J., 1892, ii., 179. Tschepke: Am. J. M. Sc., 1863, n. s., xlv., 29, *ex* Deut. Klin. Terry: Med. Indep., Detroit, 1857, iii., 259. Atlee: Med. Times and Gaz., 1871, i., 283.

[4] Davis: Cinc. M. and Dent. J., 1886–87, ii., 65, in divided doses between morning and noon.

[5] Wilson: Am. J. M. Sc., 1864, n. s., xlviii., 70.

[6] Med. Times and Gaz., 1856, xiii., 35.

[7] "Mat. Med.," 1872, 656. See also in this connection Leach: M. Times and Gaz., 1863, ii., 487. Dutgher: M. and S. Reptr., Phila., 1865, xiii., 2. Forman: Tr. M. Soc. N. Jersey, 1871, 314.

plained of some extraordinary sensations and almost immediately expired.[1] Terry[2] also reported the case of a suicide who died "very soon" after having taken 20 grains (1.3 gm.) of strychnin. Lefort[3] refers to the poisoning of three children by strychnin dispensed in mistake for santonin. Two of them are said to have fallen as if struck by lightning, and to have died in a few minutes. In another case of mistake for santonin a child is said to have screamed violently within four minutes after the administration, and to have died in a few minutes.[4] MacLagan found $\frac{1}{3}$ grain of strychnin in the stomach. Gray[5] refers to a death in 5 minutes. Taylor[6] refers to the deaths of two adults which followed one in 10, the other in 15 minutes after their having each taken about $7\frac{1}{2}$ grains of strychnin in mistake for quinin. A child is said to have died in 10 minutes from the effects of strychnin given by its father by mistake.[7] Deaths in 15 minutes have been reported by Blackwell,[8] and Ogston;[9] and in 20 minutes by Adams,[10] Allen,[11] Heward,[12] Geoghegan,[13] and Lacassagne.[14] The supposed duration of the poisonings of Cook (Reg. *v.* Palmer) and of Matilda Clover (Reg. *v.* Neill) was 20 minutes. Almost all of the small number of fatal poisonings whose duration has exceeded 2 hours have not exceeded 9 hours. Cases whose duration has been from 4 to 9 hours have been reported by Tarchini-Bonfanti,[15] Lancereaux,[16] Blacklock,[17] Jones,[18] Stewart,[19] Pilkington,[20] Reese,[21]

[1] Am. J. M. Sc., 1854, n. s., xxvii., 537.
[2] Med. Independ., Detroit, 1857, iii., 259.
[3] Bull. ac. de méd., Par., 1881, 2 s., x., 460.
[4] Ph. J. and Tr., 1874–75, 3 s., v., 757, 879.
[5] Glasgow M. J., 1870–71, iii., 167.
[6] "Poisons," 3d Am. ed., 682.
[7] Ph. J. and Tr., 1871–72, 3 s., ii., 298. See also Med. Times and Gaz., 1869, 605.
[8] M. and S. Reptr., Phila., 1883, xlviii., 671.
[9] Lancet, 1856, i., 428. Also another in Ph. J. and Tr., 1863–64, n. s., v., 586.
[10] Brit. M. J., 1894, i., 300.
[11] Bost. M. and S. J., 1847, xxxvi., 209 (case of Dr. W. C. Warner).
[12] Brit. M. J., 1869, ii., 322.
[13] Dublin M. Press, 1856, xxxv., 401.
[14] Arch. de l'anthrop. crim., 1888, iii., 503. (Aff. de Pont de Beauvoisin, 1877.) Other cases also referred to in Ph. J. and Tr., 1866–67, n. s., viii., 308. Lancet, 1866, ii., 395. Brit. M. J., 1885, i., 449.
[15] Gazz. med. it. lomb., 1856, 4 s., i., 147 (4 hours).
[16] France méd., 1877, xxiv., 17 (4 hours).
[17] Taylor: "Poisoning by Strychnia," 138 (4 hours).
[18] Lancet, 1856, ii., 291, 302 (5 hours).
[19] Mthly. Stethosc. and M. Reptr., 1856–57, i., 787 (5 hours).
[20] Lancet, 1893, i., 1513 (6 hours).
[21] Am. J. M. Sc., 1861, n. s., xlii., 409 (6 hours).

McClintock,[1] Wilkins,[2] Thibault,[3] and Henry.[4] In a case referred to by Wolff,[5] involving a life-insurance litigation, the duration could not have been less than 7 hours; it was a question whether the death was due to strychnin poisoning or to tetanus resulting from an injury, apparently self-inflicted.

In a few instances the duration in fatal cases has been even greater. Tardieu and Roussin[6] describe the case of a young girl who died in 18 hours after having taken more than a grain of strychnin with suicidal intent. A homicidal case, in which the question of duration was important, and in which it could not have been less than 20 hours, is reported by Preitner.[7] In June, 1879, Edward Parr was convicted in Philadelphia of the brutal murder of his daughter. While the sentence of death was being pronounced he succeeded in taking strychnin unobserved: at the termination of the passage of sentence he was attacked with spasms and died between 17 and 18 hours afterward, although the stomach was washed out in a short time. As he was unconscious during much of this time it was thought possible that his death had been caused by the large quantity of morphin administered by the physicians. The terminal symptoms were, however, distinctly those of strychnin. Probably death was delayed by the morphin.[8]

The action of nux vomica is as rapid as that of strychnin. The longest duration of a fatal poisoning was 7 hours, in a girl of eighteen years, who took half an ounce (15.6 gm.) of the powder with suicidal intent. A man died in a few minutes after having taken pills in the composition of which extract of nux vomica had been used in mistake for walnut extract.[9] A man died in 10 minutes,[10] and another in 15 minutes.[11] Hender-

[1] Dubl. Q. J. M. Sc., 1857, xxiv., 214 (6 hours, also jumped out of a window 35 to 40 feet above the ground).

[2] Lancet, 1857, i., 551 (6½ hours).

[3] "Alcaloides des strychn.," Diss. Lille, 1886, 109 (7 to 8 hours, Aff. Remillerot).

[4] Austral. M. Gaz., 1893, xii., 73.

[5] "Einige Fälle v. Strychninvergift.," Diss., Halle, 1887, 23.

[6] "Empoisonnement," 2d ed., 1173.

[7] "Ueb. Strychninvergift.," Diss., Würzb., 1870.

[8] Leffmann: Med. Bull., Phila., 1879, i., 58. Mendenhall (Cinc. Lanc. and Obs., 1864, n. s., vii., 26) reports a death which he attributes to strychnin taken four days before. That it was a strychnin poisoning is doubtful. Apart from the symptoms the only evidence that it was are the statement of the deceased to a neighbor that she had taken strychnin, and the results of a very crude and unsatisfactory analysis.

[9] Perini and Tarchini-Bonfanti: Gazz. med. ital., 1864, 5 s., 111, 430.

[10] Pellarin: Ann. d'hyg., 1860, 2 s., xiv., 431.

[11] Gorré: Bull. gén. de thérap., 1853, xliv., 266.

son[1] has reported the death of a girl of ten years in 15 minutes after having taken a prescription containing tincture of nux vomica and antipyrin, and also relates the serious poisoning of a young lady by the same combination.

Symptoms.—The bitter and somewhat hot taste of strychnin may be observed at the time of swallowing the poison. It has been observed, however, that the taste of this intensely bitter substance is sometimes not perceived until some little time after it has been taken.[2] A man affected with loss of function of the gustatory nerves has been known to unconsciously chew tobacco containing sufficient strychnin to produce poisonous effects.[3]

The symptoms proper, particularly when large doses have been taken, usually make their appearance promptly; sometimes in five minutes or less after the poison has reached the stomach, more usually in about ten or fifteen minutes. But cases are not wanting in which the appearance of the symptoms has been delayed. A woman of seventeen years who took ten grains (0.65 gm.) of dry strychnin showed no symptoms for fifty minutes.[4] A man showed no uneasiness for an hour after having taken the poison. He was seen by a physician in half an hour.[5] Another man took twenty grains (1.3 gm.) in a glass of whiskey at 11:30. At 12:30 he felt sick and vomited, when he had the first spasm.[6] Another man was only attacked with spasms one hour after having taken twenty grains (1.3 gm.) of the crystallized alkaloid.[7] Another man took strychnin at 11:10 P.M. At 11:40 he was rational, his face slightly flushed, the odor of alcohol in his breath, the pupils dilated, and the limbs lax. There were no other symptoms until 12:30 A.M., when he had opisthotonus.[8] Still another man exhibited no symptoms for one and three-quarter hours.[9] A man took five grains (0.32 gm.) of strychnin by mistake, immediately retired, went to sleep, and awoke in two hours, feeling "as if something unusual was going to happen." Shortly he was in a violent tet-

[1] Med. Rec., N. Y., 1887, xxxi., 95.
[2] See Bennett: Austral. M. Gaz., 1883–84, iii., 209.
[3] Kersch: Richm. and Louisv. M. J., 1871, xi., 502.
[4] Fleming: Austral. M. J., 1893, n. s., xv., 529.
[5] Adams: Med. Times and Gaz., 1856, n. s., xiii., 165.
[6] Gray: Brit. M. J., 1880, i., 476.
[7] Seargent: Louisv. M. News, 1883, xv., 225.
[8] Cooke: Lancet, 1890, ii., 972.
[9] Thomas: Cincin. Lanc. and Obs., 1860, n. s., iii., 577.

anic spasm.[1] A suicide also slept for two hours after having taken five grains (0.32 gm.) in brandy and awoke in a convulsion.[2] A man took three and a half grains of strychnin in mistake for morphin. No effects were observed for two and a half hours when he fell suddenly backward, but walked home, feeling better, and in five hours repeated the dose. In ten minutes thereafter he had a violent tetanic seizure.[3]

If the dose be relatively small, yet sufficient to cause death, there is frequently an initiatory stage of nervous exaltation without any violent symptoms, whose duration varies inversely with the magnitude of the dose and the rapidity of absorption. The special senses are much more acute than normally, the mental functions are active, the patient is restless, and experiences a sensation of itching. But with large doses the onset is frequently very sudden. A pronounced example of the suddenness of the attack is in a case cited by Wormley[4] of a woman who while about her ordinary work suddenly exclaimed "Oh!" and quickly fell, saying her feet had given out. Violent tetanic spasms followed. Or with excessive doses death has been known to occur almost immediately, with no tetanic convulsions, from sudden general paralysis, as in one of Wolff's cases[5] in which a man was found seated on a bench, who, as the watchman approached him, fell backward with a rattle in his throat and was dead. The sensation of the onset of the spasm has been described as a peculiar feeling beginning at a point about two and a half inches above and behind the eye, passing rapidly backward through the brain, and down the back and legs, to the toes, resembling an electrical discharge.[6]

Soon twitchings of individual groups of muscles occur, followed by violent tetanic convulsions. During the spasms there is marked opisthotonus. Every muscle is in rigid contraction. The head is thrown sharply back, the body bent backward, resting (on a flat surface) on the balls of the heels and the occiput, the abdominal and the thoracic muscles are firmly contracted, the lower extremities rigid, sometimes pressed closely

[1] Hewlett: New York M. J., 1871, xiii., 297.
[2] Dodge: Pac. M. and S. J., 1860, iii., 189.
[3] Anderson: Mthly. J. M. Sc., 1848, viii., 566.
[4] "Micro-Chemistry of Poisons," 2d ed., 545.
[5] *Op. cit.*
[6] Mead: National M. J., Wash., D. C., 1870–71, i., 406.

together, in other cases widely separated, and the soles of the feet bent inward and strongly arched. The arms are strongly flexed over the chest, or as rigidly extended along the sides. Or both legs and hands are spread out to their widest limits.[1] The lower jaw closes with a snap and remains firmly clenched; the eyeballs protrude; the pupils are widely dilated; the lips and even the entire face are cyanosed; the mouth is covered with froth, frequently bloody from the tongue being caught between the teeth (speech is sometimes impeded by injury to the tongue in this manner); the neck is swollen, and the expression of the countenance is hideously distorted (risus sardonicus). Occasionally opisthotonus is replaced by emprosthotonus,[2] or by pleurosthotonus;[3] and in a fatal poisoning by repeated doses of liquor strychniæ in which death occurred in ten minutes after the last dose of $\frac{1}{6}$ grain, there was no opisthotonus.[4] In a woman who poisoned herself with five grains of strychnin the muscles of the lower jaw were not affected.[5] In Claiborne's case, above referred to, in place of the usual condition of trismus, the mouth gaped wide open as if the jaws were kept asunder by a gag.[6]

The spasm gradually passes off, the muscles relax, the eyes and pupils become normal, and respiration is resumed. The patient speaks, usually calls for air, desires to be held, is perfectly conscious (unless unconscious from the action of remedies), and is in dread of impending death. Indeed consciousness and intellectual activity do not seem to be impaired even during the spasms, which are attended with severe pain, causing the patient to cry out or scream. The patient suffers from thirst, but an attempt to drink is liable to provoke a spasm.

After the first convulsion others, similar in character, occur, either spontaneously or in consequence of any, even very slight, unexpected excitation. An attempt to move the patient, a slight jarring of the floor or bed, a sudden noise, a slight draught of air, or even a flash of light, is sufficient to provoke a spasm if the patient do not expect it. On the other hand, much more

[1] Claiborne: Virginia M. J., 1857, ix., 460.

[2] The body bent forward. Wilson: M. and S. Reptr., Phila., 1880, xliii., 415.

[3] The body bent to one side. Faucan: Arch. gén. de méd., 1883, i., 74.

[4] Hunter: M. Times and Gaz., 1867, ii., 5.

[5] McWilliams: Chicago M. Exam., 1866, vii., 726.

[6] *Loc. cit.*

active excitation fails to call forth a spasm if the patient be not taken unawares; and he frequently asks to be rubbed, held down, or moved. When the spasm recurs spontaneously, the patient usually announces its coming some seconds in advance, and asks to be held. The number of spasms usually varies from three to ten; though death may occur during the first spasm, or in prolonged cases, whether fatal or non-fatal, this number may be greatly exceeded.[1] The duration of the spasm varies from half a minute to as long as fifteen minutes. In Faucan's case some of the spasms are said to have lasted as long as an hour and a quarter.[2] In fatal cases death most frequently occurs during the fourth or fifth tetanic seizure. The intervals vary in duration from forty-five seconds to an hour, or even to one and a half hours; usually from five to fifteen minutes. In cases terminating in recovery the interval between the spasms increases in length, and the convulsions become less active and shorter in duration, and finally cease, leaving the patient in a condition of great muscular fatigue and with increased reflex irritability. In fatal cases death results from one or two causes. In some cases death is due to asphyxia, caused by fixation of the muscles of respiration during a protracted spasm; in others it is due to exhaustion, and occurs during the non-tetanic period. Usually the body remains rigid when death occurs during a spasm, but it has been known to relax at the instant of death.[3]

The symptoms produced by **brucin** are the same in kind as those caused by strychnin, but much larger quantities of the former alkaloid are required to produce the same degree of action.

Among unusual occurrences not referred to above the following may be noted: Consciousness is usually perfect throughout, but periods of unconsciousness have been known to occur apart from that produced by treatment.[4] The pupils are usually dilated during the spasms, and normal or even contracted during the periods of relaxation. Two instances of unequal dilatation

[1] Surbadhiccary: Ind. M. Gaz., 1894, xxix., 270. Faucan: Arch. gén. de méd., 1883, i., 74. 153.

[2] *Loc. cit.*

[3] Henry: Austral. M. Gaz., 1893, xii., 73.

[4] Parker: Med.-leg. J., N. Y., 1884–85, ii., 375. Pilkington: Lancet, 1893, i., 1513. Durian: Ann. d'hyg., 1862, 2 s., xvii., 428. Brown: Brit. M. J., 1886, ii., 1030. Berry: Phila. M. Reg., 1887, i., 566.

of the two pupils have been reported,[1] in the most recent of which the inequality alternated.

The spasms are usually distinctly tonic in character, and the relaxation complete or almost so during the intervals. In some instances the spasms have been clonic at their beginning, afterward becoming tonic in character.[2] In one case of unquestionable strychnin poisoning the spasms were clonic in character, following each other at intervals of a few seconds, and lasting from two to three minutes, with no complete muscular relaxation, even during the intervals.[3] Epigastric pain, or a burning sensation in the stomach, is sometimes complained of.[4] In one instance there was retention of urine, and ninety ounces (2,660 c.c.) were drawn off with the catheter.[5]

A man who recovered from the effects of 0.019 gm. ($\frac{1}{4}$ grain) of strychnium arsenate taken in mistake for caffein, experienced frightful hallucinations of sight and hearing on the following day.[6]

A pregnant woman who recovered from the effects of 0.1 gm. ($1\frac{1}{2}$ grains) aborted.[7] Another woman who in her fifth month of pregnancy attempted suicide with Battle's vermin-killer recovered and did not abort.[8]

Diagnosis.—The disease bearing the closest resemblance to strychnin poisoning is tetanus, whether traumatic or symptomatic ("idiopathic"). In poisoning by strychnin the attack is more sudden than in tetanus, and the entire history of the case is compassed within a few hours, instead of lasting for days. The spasms follow each other at shorter intervals, and are of less duration in strychnin poisoning than in tetanus. In the latter trismus is one of the earliest and most prominent of the symptoms, while in the former it occurs later, if any succession of the factors of the spasm be observable, and may be insignificant as compared with the violent tetanic rigidity of the respiratory muscles. During the intervals between the con-

[1] Rowbotham: M. Times and Gaz., 1879, i., 591. Ott: Med. News, Phila., 1894, lxv., 270.

[2] Alverson: M. and S. Reptr., Phila., 1880, xliv., 416. Faucan: *Loc. cit.*

[3] Surbadhicarry: *Loc. cit.*

[4] Marston: Lancet, 1886, ii., 442 (Case I.). McWilliams: *Loc. cit.* Lejeune: Gaz. d. hôp., 1891, lxiv., 973.

[5] Charteris: Lancet, 1875, i., 510.

[6] Bock: J. de chir., ph., etc., Bruxelles, 1893, xcv., 97.

[7] Lancet, 1856, i., 671.

[8] *Ibid.*, 1881, i., 578.

vulsions due to strychnin the muscles are usually relaxed,[1] while in tetanus they remain more or less rigid, particularly those moving the lower jaw. The chief points of distinction are in the much more rapid progress of strychnin poisoning, and, in the great majority of cases, in the history of the onset, which in the poisoning follows, with very little warning, within a few minutes or possibly an hour or two, the ingestion of some bitter substance, but in tetanus is gradually developed several hours or days after an injury, which may have been trivial or serious. The distinction is most certainly made in doubtful cases by the detection or non-detection of strychnin in the urine or stomach washings.[2]

Uncertainty concerning the diagnosis between epilepsy and strychnin poisoning can only occur in the very exceptional case of an unknown person dying during a single convulsion. In such an event chemical analysis would decide the question definitely. In other cases the history of the patient and of the attack, the much longer interval between the paroxysms in epilepsy, and the more distinctly tonic character of the spasms in strychnin poisoning, are sufficient to establish the distinction.

In cases of poisoning of pregnant women by strychnin[3] the distinctions between the effects of the poison and puerperal convulsions are of importance. The principal diagnostic point is the fact that in puerperal convulsions the patient is entirely unconscious of what occurs, either during or between the convulsions, while in strychnin poisoning consciousness usually remains unimpaired, except immediately before death.[4] Here again chemical analysis may be depended upon to remove all doubt.

Treatment.—The ends to be attained are, first, the prevention of further poisoning by removal of unabsorbed poison from the stomach; and, second, the prevention or mitigation of the spasms which are the cause of death. The first end is best attained by the hypodermic administration of apomorphin, followed by washing out the stomach with a decoction of tannin containing sodium bicarbonate. While the washing is more effi-

[1] See, however, Surbadhiccary's case above.

[2] The time required for this examination is such as to render it valueless for clinical purposes.

[3] Two such are referred to by Wharton and Stillé ("Med. Jur.," 4th ed., ii., 443, 625).

[4] See, however, cases cited above, p. 800.

cacious than the action of the emetic, and should not be omitted when large doses have been taken, the introduction of the tube is certain to provoke a severe and possibly fatal spasm if it be attempted before the irritability is at least partially controlled, and therefore the evacuation of the stomach by this means is to be deferred until after the patient has come under the influence of the physiological antidotes.[1] These are more truly antidotal than is usual with so-called physiological antidotes, serving as they do to hold the spasms in check and thus to prevent death by asphyxia or by exhaustion until the poison has been removed by evacuation and elimination. The most serviceable agents for this purpose are chloroform by inhalation and chloral by the mouth, both of which should be resorted to, the former first, if at hand, until anæsthesia is established, then chloral by enema or hypodermically, if not by the mouth, after evacuation of the stomach by the siphon. In an emergency whiskey to alcoholic intoxication may be resorted to. The use of potassium bromid, tobacco, etc., in conjunction with chloral is not to be recommended; that of opium or morphin, while it was well enough before the discovery of chloral, and might be still resorted to in an emergency, is attended with the greater danger of narcotic poisoning. Artificial respiration may in exceptional cases become of service, but only when the natural respiration is not resumed at the cessation of a spasm. It is obvious that mechanical respiration cannot be performed during rigidity of the muscles. The patient should be kept as quiet as possible, and all noises, jars, or sudden movements on the part of those in attendance carefully avoided. The patient may be rubbed, moved, or held at his request, or if he be warned that it is to be done.

Post-Mortem Appearances.—There are no peculiarities discoverable, on external or internal examination, which are characteristic of this form of poisoning. Rigor mortis is more rapidly established and continues for a longer period in most cases. According to Wharton and Stillé, rigor mortis was very marked in the body of a woman exhumed two weeks after death;[2] and Taylor[3] states that cadaveric rigidity was well

[1] In any event the introduction of the pipe should not be attempted without the use of a gag.

[2] "Med. Jur.," 4th ed., ii., 445.

[3] "Poisons," 3d Am. ed., 676.

marked in the body of Cook two months after death. Usually the muscles are relaxed at death and soon stiffen, but in some cases the tetanic spasm merges into rigor mortis. But instances are met with in which cadaveric rigidity has disappeared in from fourteen to forty-eight hours. In some cases the hands are firmly clenched and the soles arched after the other muscles have become relaxed. Rigidity is of shorter duration in the bodies of those in whom the spasms have been more or less controlled by treatment during life than in those who have died without medical interference. The surface is usually livid, but not in all cases. Sometimes lividity is confined to the fingers, and in some cases the inner surfaces of the thighs and arms assume a red color.

The internal appearances are still less characteristic. The blood is usually fluid and dark. The vessels of the scalp, the brain and its coverings, and of the spinal cord, as well as the lungs, are congested when death has been due to asphyxia. The heart is usually empty and sometimes firmly contracted, the right side being less so than the left, and sometimes distended with dark fluid blood. The bladder is usually empty, though in some cases it has been found to be nearly full of urine. Occasionally ecchymotic spots or patches of congestion are observed in the stomach.

Detection.—Owing to the stability and non-volatility of strychnin, the rapidity with which it causes death, and the fact that it does not usually provoke vomiting, its detection in the cadaver rarely fails. Its presence has been demonstrated in the stomach in numerous cases, sometimes in notable quantity, and it has even been found in the solid and crystalline form.[1] Pouchet and Magnier de la Source obtained 0.148 gm. (= 2.28 grains) from 510 gm. (about 1¼ pounds) of viscera.[2] Blyth separated 3 grains (0.195 gm.) of strychnin from the stomach contents of a woman poisoned by strychnin;[3] and Sonnenschein obtained the same quantity (3.123 "gran" = 0.194 gm.) from the stomach contents of a man who had died in three and a quarter hours after having swallowed about five or six "gran."[4] Bischoff separated the enormous quantity of 0.732 gm. (11.3

[1] Von Maschka: Vrtljschr. f. d. prakt. Heilk., 1867, xcvi., 19.

[2] Thibault: "Alcal. d. strychnées," p. 111.

[3] Reg. v. Burke, 1862, Ph. J. and Tr., 1862–63, n. s., iv., 90.

[4] Casper: Vrtljschr. f. ger. Med., 1864, n. F., i., 1.

grains) from the viscera of a man who had died suddenly from the effects of strychnin.[1] (See Failure to Detect and Elimination.)

In the systematic process for alkaloids (see p. 133) strychnin and brucin are extracted by benzene from alkaline aqueous liquids.

An abbreviated process may be used when strychnin only is to be sought for. The substances to be examined, finely divided and suspended in water if solid, are rendered distinctly acid with sulfuric acid and macerated at 40°–50° (104°–122° F.) for six or eight hours, after which the liquid is filtered off. A fresh portion of dilute acid is added to the residue, and the extraction repeated two or three times. The united acid filtrates are evaporated to the consistence of a thin syrup on the water-bath. The residue is mixed with four volumes of strong alcohol, *gradually* added during stirring, and allowed to macerate twenty-four hours. The alcoholic liquid is decanted and filtered, the residue is warmed with strong alcohol, filtered after cooling, the residue washed with strong alcohol, and the united alcoholic liquids evaporated on the water-bath until free from alcohol. The residue, thinned with water if necessary,[2] is transferred to a separator (see p. 135) without filtration, and agitated with successive portions of benzene until a sample of the separated benzene leaves no residue on evaporation. The aqueous liquid is then rendered alkaline with slight excess of ammonium hydroxid and at once agitated with benzene, and the extraction with fresh portions of that solvent repeated several times.[3] The benzene layers from the alkaline aqueous liquid are evaporated spontaneously in a glass dish. The residue contains crystals of strychnin, but it usually requires purification. To this end the deposit is extracted by warming with water acidulated with acetic acid. The acid aqueous solution is filtered into a small separator and the insoluble part washed with acidulated water. The acid filtrate and washings

[1] Wolff: "Einige Fälle von Strychninvergiftung," Diss., Halle, 1887, p. 7. From 477 gm. stomach, intestine, and contents 0.68 gm., and from 568 gm. spleen, liver, kidneys, and heart, 0.052 gm. Also traces in the brain; none in the cord or urine.

[2] The bulk should be kept as small as possible. The quantity of each portion of benzene, etc., used should equal that of the aqueous liquid.

[3] For qualitative testing one extraction is sufficient; for quantitative determination as many as ten or twelve may be necessary.

are agitated with a mixture of equal volumes of chloroform and ether. After separation the ether-chloroform is drawn off, another portion of ether-chloroform and a slight excess of ammonium hydroxid are added, and the two liquids immediately agitated together. After separation the lower layer is drawn off into a clean glass dish and allowed to evaporate spontaneously, and the extraction with ether-chloroform repeated so long as the glass capsule and its contents, after evaporation of the solvent and drying in a desiccator, continue to gain weight.

The process is quantitative. The residue may, however, contain brucin, which, if present, is to be separated as directed below (see p. 812).

Tests for Strychnin.—1. *The Crystalline Form.*—As there is practically but one color reaction for strychnin known, the crystalline form is important; and as the alkaloid crystallizes readily by slow evaporation of its solvents there is no difficulty in obtaining the crystals, if the methods described have been followed and if the alkaloid be present in quantity greater than "traces." It must not be assumed, however, that affirmative results of the color and physiological tests are necessarily due to a substance other than strychnin if the crystals be not obtained, as those reactions are distinctly obtained with quantities altogether unweighable and insufficient to yield a crystalline deposit. To obtain well-formed crystals the solution should be allowed to evaporate spontaneously in a round-bottomed glass dish (see p. 135). Strychnin crystallizes from benzene or alcohol in small, four-sided, orthorhombic prisms, terminating in four-sided pyramids (Fig. 35, p. 781); sometimes also in plates. Precipitated by ammonium hydroxid from solutions of its salts it forms slender, needle-like, four-sided prisms; in which form it is also left by evaporation of its solution in ether-chloroform.

2. *The Taste.*—The taste of strychnin is intensely and persistently bitter, with a faintly metallic after-taste, and is still perceptible (faintly) with a solution of 1 : 100,000.[1] With dilute solutions the taste is not perceived immediately the drop is placed upon the tongue, but only after a short time. Of course,

[1] Prescott ("Organic Analysis," 448) gives the limit as 1 : 600,000 or 1 : 700,000. This may be possible with a sense sufficiently acute.

there being many other bitter substances, this quality is only confirmatory.

3. *The Color Reaction.*—Strychnin dissolves in concentrated sulfuric acid, forming a colorless solution of the sulfate.[1] If, now, nascent oxygen be generated in the solution, a peculiar play of colors is produced; at first, and but for an instant, blue (this color is sometimes absent), then dark violet, which gradually becomes more reddish and changes to red, and then to yellow.

This test, which is the most delicate and characteristic, may be produced in a variety of ways, but the strychnin should be dissolved in the acid before the next step of the reaction is proceeded to.

The sulfuric acid solution may be placed upon a strip of platinum foil connected with the positive (platinum) pole of a single Grove cell, and a platinum wire, connected with the negative (zinc) pole, brought into contact with the upper surface of the drop of liquid. The nascent oxygen liberated at the foil produces a dark-violet blotch, which on breaking the contact gradually suffers the changes of color mentioned.

The sulfuric acid solution may be placed in a watch glass upon a white background, and a minute fragment of some solid substance capable of yielding oxygen by contact with the acid drawn through it with a pointed stirring rod. The path of the fragment is marked by a streak of color passing through the changes above mentioned. Either black oxid of manganese, oxid of cerium, potassium dichromate, potassium ferrocyanid, or lead peroxid may be used. Black oxid of manganese and potassium dichromate are preferable to the other substances named, so far as the strychnin reaction alone is concerned, but with cerium oxid the differences between the reaction with strychnin and that produced by gelsemium are more marked (see below). The test should therefore be repeated with all three oxidants if the amount of material be sufficient. Potassium permanganate should not be used in this test as, while it gives the reaction with strychnin quite as well as the other oxidants, it also gives with certain constituents of eupatorium (boneset) as well as with traces of other organic substances, a

[1] This mixture, in the absence of other substances, does not blacken even when it is heated.

reaction closely simulating that of strychnin, which is not produced when potassium dichromate or manganese oxid is used.[1] The blue color is most persistent with the manganic oxid, which, being the least soluble, develops the reaction most slowly. Sulfovanadic acid (see p. 147) gives the same color changes; and if water be added after the yellow has become pale, a red color is produced.

As an additional precaution against ptomaïns the test may be repeated, heating the residue to 120° (248° F.) with sulfuric acid and cooling it again, before adding the oxidant—a treatment which the vegetable alkaloid withstands (if the quantity be not extremely small) but which destroys the animal alkaloids. If the material darken too much (from charring of foreign substances) the acid is to be neutralized, the mass extracted with chloroform, and the test applied to the residue of evaporation of this solution.

The following substances give this reaction in a manner more or less closely resembling that observed with strychnin but with certain differences: *Curarin* (see p. 782) gives the same play of colors, but they are developed much more slowly than with strychnin. But curarin is colored red by sulfuric acid alone; or blue at first, changing to red after some hours, or on being heated to about 90° (194° F.), and is colored purple by sulfuric and nitric acids. Moreover, curarin is not extracted by benzene or by the other immiscible solvents from either acid or alkaline aqueous liquids (see p. 138). *Gelsemin* (see p. 720) if pure, forms a colorless solution with concentrated sulfuric acid (yellow or brown if impure); with potassium dichromate or ceric oxid a reddish-purple or cherry-red color is produced, without the initial blue or blue-violet of the strychnin, and the liquid assumes a green or bluish-green color, which is not observed with strychnin. Gelsemin produces motor paralysis, not tetanic spasms, in frogs.[2] Most of the alkaloids from quebracho bark give somewhat similar reactions: *Aspidospermin* gives a faint brown color with sulfuric acid alone; and on addition of potassium dichromate a purple color, which in one minute turns brown, the brown color persisting. This alkaloid

[1] Sedgwick: Amer. Chem. J., 1879–80, i., 369.

[2] See Schwarz: "Forens.-chem. Nachweis d. Gelseminums," Diss., Dorpat, 1882, also p. 721 *ante*.

gives a red color with nitric acid, and the same color when warmed with hydrochloric acid. *Aspidosamin*, *quebrachin*, and *quebrachamin* give a blue color with sulfuric acid and potassium dichromate. They also give a blue color with Fröhde's reagent, which is not affected by strychnin. An alkaloid of Pareira brava, *geissospermin*, behaves like strychnin with sulfuric acid and potassium dichromate. But nitric acid colors it purple, ferric chlorid colors it blue, and with Fröhde's reagent it gives a persistent blue color. Moreover, it is extracted by benzene and by chloroform from acid solutions.[1] *Anilin* also gives a blue-violet color with potassium dichromate and dilute sulfuric acid, but this color does not change to red and yellow, but to black, while a peculiar odor, somewhat resembling that of bitter almonds, is given off, which is not observed with strychnin (see *Lauro-tetanin*, under test No. 5). Many alkaloids and other substances are colored by sulfuric acid *alone*, which is not the case with strychnin.

The reaction is interfered with to a greater or less extent by the presence of sugar, morphin or other reducing agents, brucin, and other substances. Therefore the alkaloid should be separated in as pure a form as possible before the application of the test.

The color reaction is distinct with 0.001 mgm. ($\frac{1}{60000}$ grain) of strychnin.

4. Solution of potassium dichromate produces a yellow precipitate in solutions of strychnin or of its salts, which is very sparingly soluble in cold water. This precipitate when moistened with sulfuric acid gives the color reaction. Brucin forms a similar precipitate, but only slowly in dilute solutions.

5. *The Physiological Test.*—This consists in the production of tetanic spasms in an animal by injection of strychnin in solution. As Marshall Hall was the first to suggest the use of the frog (whose susceptibility to the effects of strychnin he first observed) for this purpose,[2] the test is known by his name. The frog is the animal still most frequently used. Rautenfeld[3] called attention to the fact that the two common European species of frog are not equally susceptible to the action of strychnin.

[1] See Czerniewski: "D. forens.-chem. Nachw. d. Quebracho u. Pareiro-alcaloide," Diss. Dorpat, 1882.

[2] Lancet, 1856, i., 36, 335.

[3] "Ausscheidung d. Strychnins," Diss., Dorpat, 1884, 39.

Falck,[1] experimenting with *Rana esculenta*,[2] found that 0.005 mgm. of strychnium nitrate caused tetanic spasms in eighteen minutes in a frog weighing 2.1 gm., 0.02 mgm. in thirty-one minutes in one weighing 13 gm., and 0.05 mgm. in forty-nine minutes in one weighing 53 gm. Rantenfeld, using *Rana temporaria*, found that 0.03 mgm. did not produce tetanus in a frog weighing 20.5 gm., that 0.04 mgm. caused spasms in one weighing 15 gm. in twenty-one minutes, and 0.16 mgm. in one weighing 46 gm. in twenty-two minutes. The frogs selected for experiment should be as small and as lively as can be obtained. They should not be dried as has been recommended,[3] but, on the contrary, the skin should be kept moist; for, while a frog whose surface is dry is certainly more susceptible to the action of strychnin, the cutaneous respiration is impeded, and the animal is not in a physiological condition. The solution to be tested is to be injected with a hypodermic syringe either into the abdominal cavity or into the lymph heart immediately beneath the skin of the back at the root of the hind legs. The animal is then placed in a moist chamber under a bell glass and observed. It will become rigid, with a general tetanic spasm of all of the muscles, within a period varying from two minutes to an hour, according to the magnitude of the dose, if the liquid injected contained strychnin; and similar spasms may be provoked by striking upon the table, or by pinching a foot, or by touching or blowing upon the animal. The appearance of tetanus is preceded by a period of uneasiness during which the respiration is accelerated. If the amount of material available be sufficient, a second experiment should be made with a frog, one of whose gastrocnemii has been previously attached to a myograph, when the characteristic tracing of tetanus will be obtained.[4]

Falck[5] has shown that young rabbits and young white mice are even more susceptible to strychnin than frogs. Tetanus is produced in a rabbit a day old in from ten to fifteen minutes by 0.02 mgm. of strychnium nitrate, and in one eleven days old

[1] Vrtljschr. f. ger. Med., 1884, n. F., xli., 345.

[2] *Rana esculenta* is not found in America, where, however, a closely allied species, *R. palustris* occurs, as well as *R. temporaria* and *R. mugiens* (bullfrog). *R. esculenta* is also unusual in England, although occasionally met with there.

[3] Gray: Glasgow Med. Journ., 1870–71, iii., 167.

[4] See Garnier: Ann. d'hyg., etc., 1890, 3 s., xxiv., 508.

[5] *Loc. cit.*

by 0.026 mgm. Mice must be from fourteen to sixteen days old. If younger they are too small, if older not sufficiently sensitive. Tetanus was produced in mice fifteen days old and weighing from 3 to 5 gm. in less than ten minutes by as little as 0.0012 mgm. of strychnium nitrate.

Tetanus is also produced by brucin, by thebain, and certain other opium alkaloids, and by laurotetanin.[1] Falck,[2] having experimented with rabbits, gives the lethal doses per kilogram of body weight in milligrams of the nitrate as for strychnin 0.6, thebain 14.4, brucin 23.0, laudanin 29.6, codeïn 51.2, and hydrocotarnin 203.8. These alkaloids are, however, readily distinguished from strychnin by their failure to give the reaction with sulfuric acid and potassium dichromate, and by the special reactions of each, not given by strychnin. Laurotetanin gives a violet color with sulfuric acid and weak oxidants, and it is bitter. But it only becomes crystalline after a few days; it gives an indigo blue color with Fröhde's reagent, is colored pale rose color by sulfuric acid alone, and brown by nitric acid.

6. A solution of iodic acid in concentrated sulfuric acid colors strychnin yellow, changing to brick-red, and to violet.[3]

7. Aqueous solution of periodic acid colors strychnin red, and red crystals separate on evaporation of the solution.

8. Strychnin, when heated with an aqueous solution of perchloric acid sp. gr. 1.13 forms a reddish-yellow solution. Under similar treatment aspidospermin gives a fuchsin-red color and brucin a madeira color. The same reaction is produced by boiling sulfuric acid solutions of the alkaloids with a minute quantity of potassium chlorate.[4]

9. Another form of the "euchlorin reaction"[5] consists in dissolving the alkaloid in a drop of dilute nitric acid, warming, and adding a minute quantity of potassium chlorate to the warm mixture, when a scarlet-red color is produced. A drop or two of ammonia changes this to brown. On evaporation to dryness a green residue remains, which forms a green solution

[1] Gresshoff: Pharm. Jahresb., 1890, 13.

[2] Vrtljschr. f. ger. Med., 1875, n. F., xxiii., 78. See also Bratz: "Ueber Strychnin u. Brucin," Diss., Kiel, 1891.

[3] Selmi: Berichte, Berl., 1878, xi., 1692.

[4] Fraude: Berichte, Ber., 1879, xii., 1558.

[5] See p. 148.

in water, changes to orange-brown with caustic potash, and returns to green with nitric acid.[1]

10. The delicacy of the general reagents with strychnin is given by Dragendorff as follows for 1 c.c. solution: Phosphomolybdic acid, 0.07 mgm.; phosphotungstic acid, 0.0001 mgm.; phosphoantimonic acid, 0.02 mgm.; potassium-bismuth iodid, 0.0002 mgm.; potassium-cadmium iodid, 0.01 mgm. Potassium ferrocyanid precipitates colorless, four-sided prisms from a solution of 1:1000. Potassium ferricyanid, a greenish-yellow, crystalline precipitate from 1:250, which gives the color reaction with sulfuric acid. Mercuric chlorid and mercuric cyanid form white, crystalline precipitates in 1:500, which become yellow with potassium chromate. Potassium thiocyanate precipitates in white crystals (0.02 mgm.). Sodium nitroprussid a light-brown, crystalline precipitate (1:5000, according to Helwig). Potassium perchlorate, white crystals (1:100). Ferric chlorid, brownish-yellow crystals (not in 1:100). Potassium-iridium chlorid (fresh solution) at first a dark-brown precipitate which disappears on agitation, and afterward deposits in crystals (1:500). Potassium-mercuric iodid, 1:8000. Platinic chlorid 1:1000, yellowish-white, sparingly soluble in hot alcohol, and deposited from the solution on cooling in crystalline bundles resembling crystalline stannic sulfid. Auric chlorid, 1:1000, soluble in alcohol, from which it crystallizes in orange-yellow crystals. Picric acid, greenish-yellow crystals, 0.05 mgm. Tannic acid, 0.04 mgm. Iodin-potassium iodid, brown precipitate, from hydrochloric solution 0.0012 mgm., soluble in boiling alcohol, from which it is deposited in reddish-brown, doubly refracting prisms.

Separation of Strychnin and Brucin.—This may be effected by taking advantage of the greater solubility of brucin (1:38) than strychnin (1:2617) in dilute alcohol of sp. gr. 0.970. The mixture of alkaloids is digested for one hour with 100 parts of alcohol of the gravity mentioned at the ordinary temperature, filtered, and the undissolved alkaloid washed with 100 parts of alcohol of the same gravity. The undissolved alkaloid contains no brucin, but the residue of evaporation of the alcohol contains a trace of strychnin (Prescott).

Tests for Brucin.—1. Nitric acid or sulfuric acid colors

[1] Bloxam: Chem. News, 1887, lv., 155.

brucin a fine red, which fades to orange and red. Pure sulfuric acid does not color it.

2. The solution from 1, after having become yellow, is colored bright purple by stannous chlorid, ammonium sulfid, or sodium sulfid.[1]

Failure of Detection.—The reactions for strychnin are delicate and well defined, and it is difficult to suppose a case of death from the effects of this poison in which a properly conducted analysis would not reveal the presence of the alkaloid in the stomach and contents, except, possibly, under the exceptional circumstance of the stomach having been washed out shortly before death and too late to save the life of the patient; but even under these circumstances the alkaloid would be detectable in the stomach washings, liver, and urine. The duration of a fatal strychnin poisoning is so short (rarely exceeding six hours) that it is highly improbable that a person should die from the effects of this poison and no trace of it remain in the body.

That absorption takes place rapidly is true, and consequently when but small quantities have been taken, yet sufficient to cause death, and life has been prolonged for more than two hours, very little will remain in the stomach. Mann[2] has given the results of three analyses which demonstrate the rapidity of absorption, and also the fact that when, under such conditions, only a trace remains in the stomach notable amounts are still to be found in the liver and urine. In the first of Mann's cases a girl died in three hours from the effects of a threepenny package of Battle's vermin-killer, the stomach having been washed out. Only a trace of strychnin was found in the stomach, but 0.013 gm. was obtained from the liver, 0.005 gm. from 278 c.c. of urine, and a trace from the kidney. In the second case the conditions were precisely the same, except that the patient was a woman, and that the stomach had not been evacuated, and here again the amount in the stomach contents was just sufficient to demonstrate its presence, no more. In the third case double the dose had been taken by a girl who died in two hours without evacuation of the stomach. In this instance 0.078 gm. (of 0.182 gm. ingested) was recovered from the

[1] For further reactions see Dragendorff: "Ermittel. von Giften," 4te Aufl., 185.

[2] Med. Chron., Manchester, 1889–90, x., 113.

stomach contents. These cases, as well as many others, show that elimination is also rapid. But death may occur before elimination has apparently begun, as in the case of a man who fell over and died without having a single tetanic seizure and in 187 c.c. of whose urine Bischoff found no evidence of strychnin, although he separated the large quantity of 0.68 gm. from the alimentary canal and its contents.[1]

Yet cases have occurred in which death has in all probability been due to strychnin, and an analysis has nevertheless failed to reveal the presence of the alkaloid in the cadaver. An historical instance is the case of Palmer, tried in London in 1856, in which Professor Taylor, who made the analysis, did not obtain chemical evidence of the presence of strychnin, although the deceased, Cook, was seized with violent tetanic convulsions fifty-five minutes after having taken the poison, and died in about fifteen minutes thereafter. The failure of this analysis (for such it must be considered, as complete elimination or even absorption in so short a period is not possible) was the subject of much bitter controversy, and seems to have permanently warped the judgment of Dr. Taylor on the subject of chemico-legal evidence. It was due to two causes, both avoidable. The autopsy was conducted without the commonest precautions necessary in such cases. The defendant was present, and accidentally (?) caused the loss of the contents of the stomach; and the solid viscera were only obtained for analysis at a later date. But the loss of the stomach contents, although they probably contained strychnin in larger quantities than the tissues, would not have caused complete failure of the analysis had the analytical processes been properly conducted, as strychnin has been detected in the liver, kidneys, tissue of the stomach, and urine in many instances. Maschka also reports a case[2] in which an analysis failed to give satisfactory evidence of the presence of strychnin in the stomach or its contents, or in the blood or liver, although there is little room to doubt that death was rapidly caused by that alkaloid. A woman of thirty-six years had died in about fifteen minutes (during which she manifested distinct symptoms of strychnin poisoning) after having

[1] Wolff: "Einige Fälle von Strychnin-vergiftung," Diss., Halle, 1887, 7.

[2] "Samml. Gutacht. Prag. med. Fak.," 1867, 3 F., 280.

taken about one-third of the liquid contents of a bottle the remaining two-thirds of which were found to contain six grains ("gran") of strychnin. In the absence of all details as to the methods of analysis followed, it appears more probable that these were defective than that, as Maschka says, the strychnin had become undetectable by dissemination and alteration in the body or by putrefaction.

Indeed, strychnin withstands decomposition under the influence of putrefactive changes for a long period. Bischoff obtained distinct evidence of the presence of strychnin in the stomach, spleen, liver, and kidneys after a burial of six weeks.[1] Cloetta obtained positive reactions from viscera containing strychnin which had been buried three, six, and eleven and a half months.[2] Noyes recovered about 2 mgm. of strychnin from the cadaver of a child which had been buried 308 days.[3] Heintz detected strychnin with certainty in some meat to which the nitrate had been added three years before, and which had undergone putrefaction.[4] Rieckher demonstrated the presence of strychnin in a mass of heart, lungs, and liver which had been exposed to the ordinary variations of temperature, with which it had been mixed eleven years previously.[5] The question of the power of strychnin to withstand putrefactive changes was an important subject of debate in a criminal case in Germany in 1876, in which a woman had died in two hours after the alleged administration, and in which an analysis, made after four months' burial, had failed to demonstrate the presence of any vegetable poison in the liver, stomach, or intestine. In experiments made in connection with this case strychnin was detected in the bodies of seventeen dogs poisoned by it and buried for periods varying from 70 to 330 days.[6]

In an elaborate series of experiments Falck and Otto have shown that strychnin is decomposed by filtration of its solution through a certain thickness of soil, varying with the nature of the soil and the presence or absence of bacteria.[7]

Strychnin or Ptomain.—Baumert states that "in one

[1] Wolff: *Op. cit.*, p. 16.
[2] Arch. f. path. Anat., 1866, xxxv., 369.
[3] J. Amer. Chem. Soc., 1894, xvi., 108.
[4] Arch. d. Pharm., 1871, cxcvi., 126.
[5] Ztschr. f. anal. Chem., 1868, vii., 400.
[6] Ranke, Buchner, Gorup-Besanez u. Wislicenus: Arch. f. path. Anat., etc., 1879, 7 F., v., 1–23.
[7] Vrtljschr. f. ger. Med., 1891–93, 3 F., ii., 171; iii., 269; iv., 165.

case the ptomaïn in question not only gave various chemical reactions, including the identifying reactions of strychnin, but also possessed the tetanizing action of that alkaloid."[1] He, however, gives no further particulars and no reference to where such may be found, nor the name of the observer, although he discusses Amthor's base (see below) at length. Nor can we find any verification of the statement elsewhere.

Bacterial products have, however, been obtained which either possess a tetanizing action, or, being without such action, give a reaction with sulfuric acid and oxidants which in some respects resembles that produced by strychnin, yet differing in others.

Brieger and his followers[2] have obtained from the culture media of Nicolaier's tetanus bacillus three or more basic substances and a toxalbumin which cause tetanus in varying degrees of intensity. Of the bases one, tetanin, is not crystalline, but is a volatile, highly alkaline yellowish oil, and another, tetanotoxin, crystallizes in flat, pointed plates. These bases are not described as giving the color reaction of strychnin or as being bitter in taste, and have not its crystalline form.

Amthor[3] obtained a ptomaïn by the Stas-Otto method, which passed from alkaline solution into benzene and into amylic alcohol, which was not crystalline, only faintly bitter, and with sulfuric acid and potassium dichromate gave a green color. And yet Baumert refers to it as a "cadaveric strychnin!"

In investigating the cause of the "pellagra" or "maïdism" prevalent in Italy and traceable to the use of spoiled Indian corn (*zea mais*) Lombroso and Dupré,[4] and others, have obtained a base, or mixture of bases (pellagrozein), which is bitter in taste, causes tetanus in frogs, and is said to give the color reaction of strychnin, but whose reaction with sulfuric acid and oxidants only resembles that of strychnin in its initial stage—it gives a blue color which passes to a dirty yellow, without any appearance of red-violet and red, as is observed with strychnin.

[1] "Lehrb. d. gerichtl. Chem.," 353.

[2] "Ptomaine," 1886, iii., 89. Berichte, Berl., 1886, xix., 3119. Arch. f. path. Anat., 1888, cxii., 549. Berl. klin. Wchnschr., 1887, xiii., 303; *ibid.*, 1888, xxv., 329. Weyl and Kitasato: Ztschr. f. Hyg., 890, viii., 404. Kitasato: *Ibid.*, 1891, x., 267. Tizzoni and Cattani: Arch. f. exp. Path. u. Pharm., 1890, xxvii., 432.

[3] Baumert: *Op. cit.*

[4] "Rendiconti Ist. Lomb.," 1872. Lombroso: Lo Sperment., Firenze, 1876, 353, 385, 516.

Moreover, it has not the crystalline form of strychnin, and differs from that alkaloid also in that its sulfuric-acid solution assumes a permanent violet color when exposed to vapor of bromin.[1]

In an Italian criminal case in 1878 Ciotto obtained the color reaction from residues extracted from a cadaver after five days' burial, as well as a crystalline precipitate with iodin in hydriodic acid. The taste was, however, not intensely bitter, and the physiological tests appear to have been unsatisfactory.[2] Nor could the alkaloid be separated in a state of sufficient purity to distinguish with certainty whether it exhibited an alkaline reaction, or to obtain it in crystals. In view of these facts Ciotto took the ground that "a substance had been found in the viscera of A—— V——, which gave chemical reactions corresponding with the substance known as strychnin, and which consequently probably was strychnin."[3] Selmi, in criticising the results of the analysis for the defence, did so on the ground that the evidence was insufficient to positively prove the presence of strychnin; and, while he had much to say with regard to ptomaïns, he did not contend that the color reaction was produced by a ptomaïn. Indeed he distinctly says that no ptomaïn known at that time gave the sulfuric-acid-dichromate reaction.[4]

Evidence of the presence of strychnin is conclusive when the bitter taste, the color reaction, and the physiological action are all distinctly observed. The further evidence of the crystalline form, the alkalinity of the alcoholic solution, and the reaction by precipitation with potassium dichromate and the color reaction with the precipitate should be obtained if the

[1] For a full account of the products of putrid maize see Husemann: Arch. f. exp. Path. u. Pharm., 1878, ix., 226–288. H. incidentally remarks (p. 267) that no alkaloid separated from cadaveric material is known to him to which a tetanizing action is to be attributed.

[2] Ciotto says in this connection: "The results of the experiments on frogs, if indeed they do not prove distinctly that the substance was strychnin, were far from proving or causing suspicion to the contrary; they even corresponded with those which were obtained in control experiments with minimal doses of strychnin; enough to warrant the supposition that they were in great probability due to that alkaloid" ("Parte chimica di un Caso di Perizia," etc., Padova, 1880, 39). Selmi, in criticising the results of the physiological test, says that they were "uncertain to that degree that the experts themselves could not draw any certain conclusion in favor of strychnin" ("Sulle Ptomaine," etc., Bologna, 1881, 224).

[3] Ciotto: *Op. cit.*, p. 10.

[4] Selmi: *Op. cit.*, p. 232.

quantity of strychnin present be sufficient, but is not to be expected from very minute quantities.

VERATRUM—VERATRIN, ETC.

Three species of *veratrum* are of toxicological interest: *V. album* (white hellebore) a native of continental Europe, which contains *jervin* and lesser quantities of other alkaloids, but no veratrin; *V. viride* (green, or American, or swamp hellebore, Indian poke, poke[1]) which contains, besides jervin, *veratrin*, *pseudojervin*, and *veratroidin;* and *Veratrum officinale* (sabadilla) which contains veratrin, *sabadillin*, and *sabadin*, but not jervin.[2] Of these that which is the most generally used as a medicine and possesses the greatest toxic power is *veratrum viride.*

Veratrin[3]—*Cevadin*—$C_{32}H_{49}NO_{11}$—crystallizes in needles from alcohol, and forms a varnish on evaporation of its ethereal solution. It is soluble in alcohol, ether, chloroform, amylic alcohol, and benzene, sparingly soluble in petroleum ether and in boiling water (1 : 1000 H_2O), insoluble in cold water. Its solutions are alkaline and fluorescent, but have no action on polarized light. It has a burning taste, but no odor, although it provokes violent sneezing.[4] Its salts are for the most part amorphous, the picrate, chloroplatinate, and chloraurate being crystalline.

[1] The name "poke" is more generally applied in the United States to *Phytolacca decandra*, the root and berries of which are used as medicines, and which contain a poisonous principle which does not appear to be an alkaloid. Cases of poisoning by phytolacca, some of them fatal, have been reported by Terrill: Stethoscope and Va. M. Gaz., 1852, ii., 134; Dutcher: Cincin. Lanc. and Obs., 1859, ii., 350; Flumiani: Gazz. med. it. lomb., 1857, 4 s., ii., 251; Garnett. Louisv. M. News, 1881, xii., 65; Gibbs: Cinc. Lanc. and Obs., 1859, ii., 594; Griggs: Atlanta M. and S. J., 1866–67, vii., 193; Morris: Med. and S. Reptr., Phila., 1880, xlii., 505; Musgrove: Southern M. and S. J., 1858, n.s., xiv., 230; Guthrie: J. Am. M. Assoc., 1887, ix., 125; Paresi: Independente, Torino, 1875, xxvi, 487; Rampazzo: Gazz. med. it. prov. Venet., 1867, x., 221; Toldo: *Ibid.*, 1875, xviii., 85; and Pugliesi: Gazz. d. osp., Napoli, 1891, xii., 435.

[2] Black hellebore (*Helleborus niger*) is not a veratrum and contains no alkaloid, but owes its medicinal and toxic powers to four glucosids, principally *helleborein.*

[3] Sometimes designated as "Merck's veratrin." "Conerbe's veratrin" is not this alkaloid, but is an amorphous base obtained from sabadilla, existing only in traces in *V. album* and not at all in *V. viride.*

[4] Hence the German name of veratrum, *Nieswurz*, sneeze-herb.

Jervin—$C_{14}H_{22}NO_2$—crystallizes readily from alcohol in needles. It dissolves readily in alcohol (1 : 16.8) and chloroform (1 : 60), less in ether (1 : 268), sparingly in benzene (1 : 1625), and slightly in water and acetic ether; insoluble in petroleum ether. Its sulfate is very difficultly soluble. It does not possess the sternutatory power of veratrin but has an acrid, bitter taste.

VERATRIC POISONING.

Poisonings by veratrum or veratrin are of exceptional occurrence. We find reports of 28 cases, almost all accidental, either medicinal poisonings from overdoses of the tincture (Norwood's tincture) or the fluid extract (Tilden's extract), or from eating portions of the plant. In all of these poisonings by veratrin the alkaloid has either been combined with other substances (in a liniment) or was probably impure.[1]

In an early case two men were seriously poisoned by white hellebore mixed with brandy as a practical joke.[2]

All of three alleged homicides by veratrum occurred in France. In one a girl was convicted of having caused the death of her two brothers and the serious poisoning of her mother by repeated doses of veratrum album mixed with their food (Affaire Journy, 1860).[3] In another a child was found dying in the street in Paris; and evidences of the presence of veratrin in the cadaver were obtained by Boutmy.[4] About three months after this case the same analyst is said to have obtained the reactions for veratrin and aconitin on analysis of the viscera of a young woman who was supposed to have taken the plants containing those alkaloids to provoke abortion.[5]

Lethal Dose.—The lethal dose of veratrin for man is not known. It is probably relatively high. In two of the three reported veratrin poisonings, in which 0.3 and 0.2 gm. (4 and 3 grains) were recovered from, the action of the alkaloid was complicated by that of the other ingredients of the liniment.

[1] Grenander: Hygiea, Stockholm, 1885, 9 s., xlvii., 510. Blake: St. Geo. Hosp. Rep. (1870), 1871, v., 69; Pharm. J., 1851, x., 521.

[2] Martini: Mag. f. d. Staatsarznk., 1845, iii., 394.

[3] Nivet and Girard: Gaz. hebd. d. méd., 1861, viii., 499.

[4] Ann. d'hyg., 1880, 3 s., iv., 211.

[5] Hougouneng: "Traité des poisons," 493. N.Y. Herald (Aug. 15th, 1882) mentions the conviction of a priest at Perpignan, France, of the murder of two women, one by "hellebore root," the other by hydrocyanic acid.

In the third (in 1851) the veratria, of which 29 grains (1.9 gm.) in alcohol failed to cause the death of a man, was probably impure.

Excluding the homicidal cases above mentioned we find reference to but five fatal veratric poisonings. A child of one and a half years died from the effects of thirty-five drops of the tincture, given in divided doses.[1] A woman died from the effects of a teaspoonful of the tincture, given by mistake of the nurse.[2] The same quantity of the tincture, similarly administered to a man suffering from typhoid fever, was a contributory cause of his death.[3] A woman of fifty years is said to have died in four weeks from the effects of seventy drops of Tilden's fluid extract.[4] In the remaining fatal case the patient, a man, took "a drink" of the tincture in mistake for whiskey.[5]

On the other hand, large doses have been taken without fatal result. A man took four teaspoonfuls of Norwood's tincture in two doses, having mistaken the directions. He began to vomit and became very weak in half an hour after the second dose, but recovered without any unusual symptoms.[6] A man recovered from the effects of half an ounce of powdered white hellebore taken in mistake for cream of tartar, although emetics and enemata were only administered four hours later.[7] Wood and Westmoreland[8] cite several instances of tolerance of large doses.

Duration.—Death from veratrum viride may occur in less than an hour, as in Walmsley's case above referred to. Usually it is delayed for a longer period. In Kirk's case a man died in four hours from the effects of "a drink" of the tincture (which may have been a mouthful or a tumblerful). In Harris' case a young child died in thirteen hours. In Johnson's case a woman died after four weeks of "continuous vomiting," following the ingestion of a dose of seventy drops of the fluid extract.

Symptoms.—These usually appear within twenty minutes,

[1] Harris: Bost. M. and S. J., 1865, lxxii., 249.
[2] Walmsley: N. Orl. M. and S. J., 1884–85, n. s., xii., 256.
[3] Horwitz: Phila. M. Times, 1883–84, xiv., 863.
[4] Johnson: Buff. M. and S. J., 1866, Nov., 133.
[5] Kirk: M. and S. Reptr., Phila., 1879, xl., 372, xli., 63.
[6] Tuttle: New York M. Journ., 1892, lv., 691.
[7] Giles: Lancet, 1857, ii., 9.
[8] Atlanta M. and S. J., 1866–67, vii., 251.

but may be delayed for an hour or more, and are referable partly to the action of the poison upon the terminations of motor, sensory, and secretory nerves, and partly to its action upon the central nervous system. There is severe burning pain in the stomach, and a sensation in the bowels as if they were being tied with a cord, continually tightening; the tongue is swollen, the throat feels as if scalded with hot water, deglutition is difficult, and the patient suffers from thirst. Nausea is intense, vomiting is severe and persistent, and the purging which occurs later is attended with tenesmus. The skin is reddened and affected with an unbearable itching and continual tingling sensation. Sneezing is said to occur, although in Blake's case it was absent, nor do we find it referred to in other reports, and also salivation, lachrymation, and running from the nose. The patient is restless, in fear of impending death, becomes greatly prostrated, has attacks of vertigo and of severe dyspnœa, during which he gasps for breath, and the respiration, which is shallow and superficial, sometimes fails entirely. The pupils are dilated,[1] but not widely, and at times there is entire loss of perception of light. The temperature is subnormal. The pulse becomes feeble, small, thready, and rapid, and imperceptible at the radial. Death may occur in collapse, suddenly from heart failure, as in Walmsley's case, in which the patient, while apparently improving, suddenly called out "I cannot breathe," and lost consciousness. No pulse was perceptible at the radial, and no heart sound was audible, and after one or two slight efforts at respiration the patient was dead in spite of intravenous injection of ammonia. Or death may be due to exhaustion from persistent and long-continued vomiting and prostration, as in Johnson's case.[2]

[1] In Blake's case they were contracted, but the liniment also contained opium.

[2] For cases of veratrin poisoning not referred to see Wagner: J. d. prakt. Heilk., 1827, lxiv., 5 Stück, 42 (family of eight, V. album, A., R.). McIntyre: Chicago M. Exam., 1861, ii., 200 (F., 3, A., R., V. viride). Blas: Verh. d. naturf. Ges. z. Freiburg, 1861, 173 (veratrin, not seen). Edwards: M. Times and Gaz., 1863, n. s., i., 5 (M., ad., experiment, R., V. viride). Buckingham: Am. J. M. Sc., 1865, n. s., i., 563 (2 cases, R., fl. extr. V. viride). Craig: Richm. and Louisv. M. J., 1869, viii., 237 (Indian poke, not seen). Mason: Tr. M. Assoc. Ala., 1877, 180 (F., 16, A., R., Norwood's tr.). Bailey: N. Orl. M. and S. J., 1877–78, n. s., v., 38 (M., 30, A., R., 3 iiss. Tr. V. viride in 7 hours). Greenwell: Cinc. Lanc. and Cl., 1885, n. s., xv., 736 (F., ad., A., R., ɱ80, Tr. V. viride). Pedigo: Va. M. Mthly, 1889–90, xvi., 426 (M., 4, A., R., V. viride, plant).

Treatment.—Evacuation of the stomach is usually not called for, but if emesis have not occurred spontaneously it should be provoked by tepid water in large amount, or the stomach should be washed out. Aromatic spirits of ammonia by the mouth serves the double purpose of a stimulant and to prevent the formation of the readily soluble chlorids of the alkaloids. Warm drinks and warmth externally are indicated to raise the body temperature. Stimulants should be given and the patient kept flat on the back. Should the respiration fail, artificial respiration should be resorted to. Opium may be given to quiet the pain and vomiting.

Post-Mortem Appearances.—These have been entirely negative in such observations as have been made in the human subject.

Detection.—Veratrin and jervin pass from acid aqueous liquids into chloroform, and in the systematic process are found in residue III. (p. 135). The former is also extracted from alkaline liquids by benzene and in traces by petroleum ether.

The residue first obtained may be purified if necessary by solution in dilute acetic acid, filtration, alkalinization of the filtrate, and extraction with benzene.

Tests for Veratrin. — 1. Veratrin dissolves in concentrated sulfuric acid, forming a yellow solution, which gradually reddens through orange, cherry red to dark carmine-red. If the yellow solution be heated, or if bromin water be added to it, the red color is immediately developed. Similar colors are produced by veratrin with Erdmann's reagent (p. 147), Fröhde's reagent, and sulfovanadic acid. The reaction is sensitive to 0.1 mgm.

Many other substances give a red color *immediately* with concentrated sulfuric acid. The following give a yellow color, changing to red with sulfuric acid: *grandiflorin*,[1] which differs from veratrin in being colored purplish by nitric acid, and in not giving the hydrochloric-acid reaction (No. 2). *Heliotropin* (*cynoglossin*) is not only colored yellow, changing very slowly to red by sulfuric acid, but also gives a lilac color with hydrochloric acid. It, however, exerts a curare-

[1] An alkaloid of *Solanum grandiflora*, the "wolf-fruit" of Brazil.

like action on frogs, causing arrest of respiration, while the heart continues to beat, and muscular paralysis (see No. 5 below). *Sabadin*,[1] which also possesses the sternutatory power of veratrin, but is not colored by nitric acid and does not give the hydrochloric-acid reaction (No. 2). The red color with sulfuric acid subsequently changes to violet. *Phlorrhizin*,[2] which is also colored dark-red when heated alone to 200° (392° F.), is without physiological action, and does not give the hydrochloric acid reaction. *Atisin*[3] is colored yellow by sulfuric acid, changing to a brilliant purple, which lasts for several days. It is colored yellow, changing to reddish and then to carmine by sulfuric acid and sugar. It remains colorless with hydrochloric acid.

2. Veratrin dissolves in concentrated hydrochloric acid, forming a colorless solution, but if this be boiled for a minute or two it forms a beautiful red liquid which retains its color for weeks. Sensitive to 0.17 mgm. According to Dragendorff *syringin*, *sanguinarin*, and *rhœadin* give red solutions with hydrochloric acid, but in the cold, and the color of the first two is discharged by heat.

3. If veratrin be mixed with three to four times its weight of cane sugar and the mixture moistened with concentrated sulfuric acid, it is colored first yellow, then after a time dark green, then blue, and finally dirty violet (Weppen's reaction).

Or this (furfurol) reaction may be obtained by dissolving the alkaloid in concentrated sulfuric acid, and adding a drop of furfurol solution (2 drops furfurol to 1 c.c. water), when a dark green color, changing to dark blue-violet, is produced (Brasche).

4. Veratrin behaves like atropin with Vitali's reaction (see p. 677).

5. If a solution of veratrin be injected into the abdominal cavity or into a lymph-heart of the frog, the heart's action rapidly diminishes in frequency to 10 or 15 per minute, and spasmodic contractions of the muscles are observed.

Tests for Jervin. — Jervin behaves like veratrin with Weppen's reaction (see No. 3 above). It is colored red by sul-

[1] An alkaloid accompanying veratrin in sabadilla seeds, but not in veratrum.

[2] A glucosid existing in the bark of apple, pear, cherry trees, etc.

[3] An alkaloid from Atis root (aconitum).

furic acid, and wine-red by hydrochloric acid. It may be distinguished and separated from veratrin by taking advantage of the fact that its sulfate and nitrate are very sparingly soluble in water containing an excess of acid (sulfate in 427 parts, nitrate in 1200 parts).

NON-ALKALOIDAL VEGETABLE POISONS.

Poisonous plants other than those owing their activity to acids or to alkaloids usually contain a toxic *glucosid*[1] (black hellebore, daphne, jalap, etc.), or an *indifferent body* (cocculus indicus, etc.), or a *volatile oil* or *camphor* (savin, laurus, etc.). Of these substances, however, but few are of forensic interest.

DIGITALIS.

The investigations of many chemists and pharmacologists, Homolle, Quevenne, Nativelle, Walz, Schmiedeberg, Kiliani, Amand, and others, have failed to render the chemistry of the active principles of foxglove (*digitalis purpura*) entirely satisfactory. This much seems to be established, however, that it contains at least three glucosids, and a fourth substance not a glucosid, which exist in varying proportion in different samples of commercial "digitaline." Considerable confusion is also caused by application of the same name to substances apparently different.

Digitonin crystallizes from eighty-five-per-cent. alcohol, but separates as an amorphous solid from its solution in more concentrated alcohol. In the amorphous condition it is very soluble in water, less so when crystalline; sparingly soluble in alcohol, not soluble in ether, chloroform, or benzene. It is decomposed by heating with dilute hydrochloric acid into a crystalline derivative, *digitonin*, galactose, and dextrose. According to Kiliani and others digitonin is valueless medicinally. It is the chief constituent of "amorphous" or "soluble digitaline" and of the German "digitalinum crystallisatum" or "digitalin verum."

[1] Glucosids are vegetable compounds which on being heated with a dilute acid are decomposed into glucose, or some related sugar, and another substance.

Digitalin (Schmiedeberg's) is a colorless or yellowish, amorphous solid, or separating from alcoholic solution in nodular masses; almost insoluble in cold water, somewhat more soluble in warm water, easily soluble in chloroform and in a mixture of chloroform and absolute alcohol, sparingly soluble in ether; readily soluble in warm, dilute acetic acid. This glucosid, which possesses the action of digitalis upon the heart, is, according to Schmiedeberg, the principal constituent of "Homolle's digitaline."

Digitoxin (Schmiedeberg) is the *digitalin* of Amand and of Nativelle. It crystallizes in fine needles, which fuse at 243°–246°, and is soluble in water to the extent of 0.65 part in 100, and is also dissolved by boiling benzene. It dissolves sparingly in cold benzene, ether, and chloroform, by which it is extracted from acid aqueous liquids. It is the most active of the digitalis glucosids, is poisonous in minute dose, and causes dilatation of the pupil. It is the chief constituent of "Nativelle's digitaline."[1]

DIGITALIS POISONING.

Poisonings by digitalis are not of frequent occurrence, and reported cases are for the most part non-fatal medicinal poisonings. We find mention of eight attempts at suicide, all of which were unsuccessful. In four of these "digitaline granules" were taken,[2] two drank of the tincture,[3] and one swallowed a preparation intended for external use.[4]

Excluding the case of a quack, tried for manslaughter for having caused the death of a boy by giving him seven ounces of a strong decoction of digitalis,[5] we find record of but one homicidal poisoning by digitalis, that, however, a *cause célèbre*. In 1864 a homœopathic physician in Paris, La Pommerais, was convicted of the murder of a widow, de Pauw, in whose death he had a pecuniary interest. It was proven that

[1] For examinations of commercial "digitalines" see Lafon: Ann. d'hyg., 1886, 3 s., xv., 527; xvi., 429, 506.

[2] Heer: J. d. chim. méd., etc., 1858, 4 s., iv., 85. Mawer: Lancet, 1880, i., 166. Bérenger: France méd., 1878, xxv., 233. Dubuc: Gaz. d. hôp., 1865, xxxviii., 238.

[3] Jeanton: Gaz. d. hôp., 1885, lviii., 441. Darezac: J. d. méd. d. Bordeaux, 1882–83, xii., 567.

[4] Bull. gén. d. thérap., 1848, xxxv., 418. In the remaining case the preparation is not stated. Reid: Mass. Eccl. M. J., 1883, iii., 166.

[5] Edinb. M. and S. J., xxvii., 223.

the defendant had purchased and used an inordinate quantity of digitaline; the deceased had died after an illness marked with symptoms such as are produced by that poison; no natural cause of death was found at the autopsy; and extracts from the viscera of the deceased and from scrapings of the floor where she had vomited, when administered to animals, caused vomiting, diminution in the rapidity of and intermittence in the heart's action, marked prostration, and death.[1]

Lethal Dose.—It is not possible at present to fix the minimum lethal dose of digitalis or even of digitalin. This is partly because of the great variations in the strength of the pharmacopœial preparations of the former and in the composition of commercial samples of the latter; and partly because of the peculiar cumulative action of this poison, and the tendency of even slight causes to precipitate a fatal termination in patients under its influence. The danger of a sudden outbreak of toxic symptoms during repeated administrations is well known to physicians, and death has resulted suddenly from the effects of half an ounce of an infusion administered in repeated doses during three days.[2] A recruit also died suddenly from the effects of repeated doses of digitalis which he had taken to simulate illness and thus avoid military service.[3]

Duration.—Digitalis is rarely rapidly fatal. Yet Viallet[4] has reported the death of a woman of seventy-nine years, which occurred suddenly in forty-five minutes after she had taken 20 to 25 gm. of tincture of squills and digitalis. In two other cases death has followed the ingestion in less than twenty-four hours.[5] In other fatal cases the duration has been from five to twelve or sixteen days.[6]

Symptoms.—The symptoms produced by digitalis are referable partly to its action upon the heart, and partly to that

[1] Tardieu and Roussin: "Relation méd.-lég. de l'Aff. Couty de la Pommerais," Paris, 1864, also "Empoisonnement," 2ème ed., 809, Ann. d'hyg., 1864, 2 s., xxii., 80. Gallard: Union méd., 1864, xxii., 263, 296, 307, 322, 347.

[2] Armstrong: Med. Times and Gaz., 1860, Oct., 417.

[3] Köhnhorn: Vrtljschr. f. ger. Med., 1876, n. F., xxiv., 278.

[4] Bull. Soc. d'anat., Paris, 1849, xxiv., 89.

[5] Hall: Canada M. J., 1865, i., 353. Edin. M. and S. J., xxvii., 223.

[6] Edward: Lancet. ii., 1849, 31 (5 days). Mazel: Gaz. d. hôp., 1864, xxxvii., 301 (5 days). Caussé: Ann. d'hyg., 1859, 2 s., xi., 464 (12 days). Hirn: "Expérience," Paris, 1839, iii., 393 (12 days). Duroziez: Union méd., 1879, 3 s., xxvii., 991 (16 days. Death from digitalis or uræmia).

upon the stomach and bowels, one or another phase predominating at different periods of the poisoning and in different cases.

Within a quarter to half an hour after a single toxic dose has been taken a sense of stiffness, fatigue, sleepiness, and pain in the head are experienced. Soon there is violent vomiting, accompanied by pain in the stomach, nausea, attacks of vertigo and syncope, while the headache becomes more violent. The heart beats rapidly and violently, and there is a sense of suffocation. The pulse, however, rapidly diminishes in frequency, and becomes intermittent, irregular, small, and difficult to count. Visual disturbances begin early in the case; dimness of vision is usual, and oscillation and apparent inclination of surrounding objects, and modifications of color perception have been experienced. The pupils are usually dilated. There may be violent delirium; and epileptiform convulsions have occurred. In some instances there has been suppression of urine. Death may occur in coma or in convulsions, but more usually suddenly, from syncope provoked by some slight movement.

Treatment.—If the case be seen early the stomach is to be evacuated or emesis favored by large draughts of tepid water. In a more advanced stage, however, the persistent vomiting should be checked by avoidance of too much drink, by ice fragments, or by small quantities of opium or cocain. The tendency to fatal syncope is to be met by administration of whiskey or nitroglycerin internally, and by inhalation of amyl-nitrite. The patient should be kept perfectly quiet in bed, and should avoid all movements.

Post-Mortem Appearances are entirely negative.

Detection.—The glucosids of digitalis appear to resist the influence of putrefactive changes at least moderately well. In the La Pommerais case physiological effects were obtained, which were undoubtedly due to these glucosids, with an extract from a cadaver which had been buried thirteen days. Brandt[1] obtained both the physiological and chemical reactions with extracts from animal material mixed with digitalis leaves and with digitalin, which had putrefied for nearly four months. Lafon[2] also obtained his chemical reaction under like conditions

[1] "Exper. Studien u. d. forens. chem. Nachw. d. Digitalins," etc., Diss., Dorpat, 1869, p. 61.

[2] *Loc. cit.*, p. 517.

for periods ranging from seven days to a little over four months. On the other hand he failed to obtain such reaction with the cadavers of dogs that had been buried for six and seven months.[1] He also found that, while evidences of the presence of digitalin could be obtained shortly after death by that poison, from the contents of the alimentary canal and from the vomit, he failed to obtain it from the liver, kidneys, spleen, brain, blood, or urine.

In the systematic method of extraction (see p. 134) "digitalin" is extracted by benzene and by chloroform from acid aqueous solution. The process cannot be abbreviated when digitalin only is to be sought for, but in that event acetic acid should be substituted for tartaric in the first extraction.

Tests for "Digitalin."—The color reactions of the digitalis glucosids are apparently neither very numerous nor distinctly characteristic. The following two reactions are, however, given by a material extracted from the French "digitalines" and from digitalis, by benzene or by chloroform:

1. It dissolves in concentrated sulfuric acid, forming a green solution, which on addition of bromin water turns purple-red, and on dilution with water emerald green or dull green (Grandeau).

2. When mixed with a mixture of equal parts of concentrated sulfuric acid and alcohol, and heated until it turns yellowish, it assumes a fine blue-green color on addition of a drop of dilute ferric-chlorid solution. According to Lafon,[2] who discovered the reaction, it is sensitive to 0.1 mgm. It has only been proved with the French "digitalines" and with digitoxin.

The physiological reaction is more to be depended upon. This should be tried with frogs, and, if the material suffice, with a small dog, not with rabbits. When frogs are used the heart is exposed and the solution injected. If digitalin be present the frequency of the heart beats is much diminished. Three frogs of about equal size should be similarly prepared, two for comparison, into one of which a known solution of digitalin is injected, the third receiving no injection. In the dog digitalis produces vomiting and purging, dilatation of the pupils, slowness and irregularity of the heart's action, and, in sufficient quantity, death.

[1] *Loc. cit.*, p. 443.

[2] *Loc. cit.*, p. 527.

MISCELLANEOUS VEGETABLE POISONS.

A few of the many vegetable poisons not considered in the previous pages have been of interest in legal inquiries in exceptional cases. We will content ourselves with their mere mention and references to the literature relating to them.

Cocculus indicus owes its toxic qualities to the presence of a crystalline indifferent body, *picrotoxin*. In Reg. *v.* Clanderoy, a man was convicted of an attempt to murder a child by administration of two of the whole berries. The case is interesting chiefly with regard to the "intent of the administration."

CASES.—Wharton and Stillé: "Med. Jur.," 4th ed., ii., 500. Clark: *Phila. Polyclin.*, 1895, iv., 72. Dutzmann: *Wien. med. Presse*, 1869, x., 491. Haynes: *Phila. Med. Times*, 1883–84, xiv., 748. Poma: *Gazz. med. ital. lomb.*, 1870, 6 s., iii., 163. Rosenkranz: *Northwest M. and S. J.*, 1848–49, v., 295. Shaw: *Med. News*, Phila., 1891, lix., 38. Sozinskey: *Ibid.*, 1883, xliii., 485. Van Sydow: *Förh. Svensk. Läk-Sällsk. Sam.*, 1862–63, 168. Thompson: *Med. Exam.*, Phila., 1852, n. s., viii., 227. PAPERS.—Barth and Kratschy: *Sitzber. Ak. d. Wissensch.*, Wien, 1880, lxxxi., 7. Cayrade: "Picrotoxin," Par., 1866. Chlopinski: Diss., Dorpat, 1883. Falck: *Deut. Klin.*, 1853, 47. Gaabe: Diss., Dorpat, 1872. Gottlieb: *Arch. f. exp. Path. u. Ph.*, 1892, xxx., 21. Keck: Diss., Kiel, 1891. Köhler: *Berl. klin. Wochensch.*, 1869, No. 47. Köppen: *Arch. f. exp. Path. u. Pharm.*, 1891, xxix., 327. Kossa: *Ungar. Arch. f. Med.*, 1892, i., 1. Loewenhardt (A.): Diss., Halle, 1880. Loewenhardt (E.): *Ann. d. Chem.*, 1884, ccxxii., 353. Palm: *Ztsch. f. anal. Chem.*, 1885, xxiv., 556. Paterno and Oglialoro: *Gazz. chem. ital.*, 1879, ix., 57, 113, 1881, xi., 36. Planat: *Bull. gén. de thér.*, 1876. Plugge: *Arch. f. exp. Path. u. Ph.*, 1893, xxxii., 267. Ricciardi: *Presse méd.*, 1894, 247. Röber: *Arch. f. Anat. u. Phys.*, 1869, 38. Schmidt: *Ann. d. Chem.*, 1884, ccxxii., 313. Siegl: Diss., Kiel, 1891. Tschudi: "Kokkelskörner," St. Gallen, 1847. Valentin: *Ztsch. f. Biol.*, xvii., 113. De Varigny: *J. de l'Anat. et de Physiol.*, xxv., 187.

Croton oil (*oleum tiglii*), a violent gastro-intestinal irritant, which has caused death in a dose of twenty drops. In a French case an unsuccessful attempt was made to poison three persons by dropping croton oil into the cavities left in strawberries by plucking out the stems (Mayet and Hallé). In Reg.

v. Massey and Ferrand the defendants were charged with causing the death of a man by placing jalap and croton oil in food of which the deceased and others partook. A woman, named Phillips, was charged with having attempted to poison her husband with croton oil in Michigan in 1890. In the same year another woman in New Jersey was accused of attempting to murder her son by the same means.

The toxicology and bibliography of croton oil will be found in very complete form in Hirschheydt: *Arb. d. pharm. Inst. zu Dorpat*, 1890, iv., 5–80.

Lobelia.—*Lobelia inflata*, known in the United States as *Indian tobacco*, produces symptoms very similar to those caused by tobacco. Indeed it owes its activity to an oily, yellowish-white substance which is related to nicotin, although it is a glucosid. It has caused poisoning and death in several instances. It is of medico-legal interest chiefly in connection with prosecutions of ignorant empirics for having caused death by its administration (Coffinites, Thomsonians; Peo. *v.* Drake, Broome County, New York, 1843).

Cases.—Barker: *Brit. M. J.*, ii., 799. Brown: *Bost. M. and S. J.*, 1836, xiv., 55; *Brit. M. J.*, 1881, ii., 23; *Lond. M. Gaz.*, 1849, n. s., ix., 383; *Med. Pr. and Circ.*, 1884, n. s., xxxvii., 127; *Lancet*, 1850, ii., 31, 250. Davis: *N. York M. J.*, 1844, iii., 343; *Med. Times*, Lond., 1849, xix., 669, 684. Johnson: *Brit. M. J.*, 1858, 493. Pearson and Curtis: *Med. Times*, Lond., 1849, xx., 147; *Med. Times and Gaz.*, 1853, n. s., vi., 270; *Lancet*, 1862, i., 467. Simmons: *Kansas M. J.*, 1884, ii., 287; *Lond. M. Gaz.*, 1850, n. s., xi., 384. Wood: *West. Lancet*, 1850, xi., 363. Papers.—Curtis: *Lond. M. Gaz.*, 1851, n. s., xiii., 160. Dreser: *Arch. f. exp. Path. u. Ph.*, 1880, xxvi., 237. Lewis: *Ph. J. and Tr.*, 1877–78, 3 s., viii., 561. Mayer: *Vrtljschr. f. prakt. Pharm.*, 1866, xv., 233. Ott: *Bost. M. and S. J.*, 1875, xcii., 124; *Phila. M. Times*, 1875–76, iv., 121; *J. Nerv. and Ment. Dis.*, 1877, n. s., ii., 68. Paschkis and Smita: *Monatsh. f. Chem. etc.*, 1890, xi., 131. Roosen: Diss., Dorpat, 1886. Tidy: *M. Press and Circ.*, 1869, vii., 91.—*Lond. M. Gaz.*, 1850, n. s., xi., 285.

Camphor is widely used to drive off moths, and in alcoholic solution as a household and homœopathic remedy. The habit of chewing camphor is by no means uncommon. When taken in overdose it produces severe gastro-enteric disturbance and violent convulsions, and has proved fatal in several instances. While non-fatal accidental poisonings are of frequent

occurrence, its odor renders it unfit for secret administration. In a German case a pregnant woman and a midwife were convicted on a trial for administering camphor to provoke abortion (Kuby: *Friedr. B. f. ger. Med.*, 1881, xxxii., 310).

Cases.—See Ind. Cat. Surg.-Gen. Libr., ii., 661, to which add: Albutt: *Brit. M. J.*, 1873, ii., 679. Banerjee: *Ind. M. Gaz.*, 1885, xx., 142. Brothers: *Med. Rec.*, N. York, 1887, xxxiii., 734. Chatterji: *Med. Regs.*, Calcutta, 1894, iii., 123. Davis: *Brit. M. J.*, 1887, i., 726. East: *Ibid.*, 1886, i., 542. Finly: *Med. Rec.*, N. York, 1887, xxxi., 125. Hononan: *Austral. M. J.*, 1888, n. s., x., 252. Lesser: "Alt. d. ger. Med.," p. 52, T. viii., f. 2 (col. pl. stomach). Main: *Med. Age*, Detroit, 1883, i., 288. Planat: *Ann. méd.-psych.*, Par., 1885, 7 s., i., 224. Prentiss: *Therap. Gaz.*, 1892, 3 s., viii., 106. Rosenthal: *Wien. med. Blätt.*, 1881, iv., 1353. Ryan: *Austral. M. J.*, 1885, n. s., vii., 433. Socquet: *Ann. d'hyg.*, 1893, 3 s., xxv., 520 Papers.—Gottlieb: *Arch. f. exp. Path. u. Ph.*, 1892, xxx., 31. Harnack u. Witkowski: *ibid.*, 1876, v., 429. Schmiedeberg and Meyer: *Ztschr. f. physiol. Chem.*, 1879, iii., 425. Wiedemann: *Arch. f. exp. Path. u. Ph.*, 1877, vi., 216 (Bibl. of earlier papers).

Savin.—An infusion or decoction of the leaves of *Juniperus sabina*, or the volatile oil to which it owes its odor and toxic quality, has frequently been used to cause abortion. Administered internally it causes severe gastro-intestinal disturbance and stimulation at first, followed by prostration, collapse, and, sometimes, death. An early trial for murder in consequence of death so produced was that of Charles Angus in England in 1808. Taylor refers to a similar case: Reg. *v.* Pascoe, 1852. For bibliography see Ind. Cat. Surg.-Gen. Libr., VII., 310.

Oil of Cedar.—The volatile oil of *Juniperus virginianus* has also been frequently used to provoke abortion. It causes vomiting, vertigo, suppression of urine, delirium, and violent convulsions. It has caused death in a few instances, as in the case of Peo. *v.* Kidder, tried for manslaughter in Niagara County, N. Y., in 1885. For bibliography see Ind. Cat. Surg.-Gen. Libr., VII, 311, to which add Brown: *M. News*, Phila., 1893, lxiii., 15; De Fareri: *Sperimentale*, Firenzi, 1889, lxiii., 284; Ellison: *Therap. Gaz.*, 1890, 3 s., VI., 371.

Tansy.—The herb *Tanacetum vulgare*, and the volatile oil obtained from it by distillation, are reputed abortifacients.

In overdose they are narcotico-irritant poisons, and have been the cause of death in a few cases. Thus a man was charged with having caused the death of a pregnant girl by oil of tansy in Pennsylvania in 1882. For bibliography see Ind. Cat. Surg.-Gen. Libr., XIV., 103.

ANIMAL POISONS.

Poisons generated in animal bodies either during life or after death are of interest in forensic toxicology at the present time in two connections:

1. In relation to resemblances of their properties and reactions to those of vegetable poisons; which may interfere with the certain detection of the latter in cases of supposed poisoning by vegetable alkaloids or glucosids.

2. With regard to the symptoms and effects which they produce when they are taken into the living human body, as constituents of more or less decomposed articles of food, or as natural products of the bodies of certain animals. Symptoms and effects which sometimes simulate those, or some of those, which are produced by certain mineral or vegetable poisons.

In the first of these relations they have already been considered in treating of the vegetable alkaloids.

POISONOUS FOODS.

Sausage Poisoning (*Allantiasis*, *Botulism*).—According to Buchner and Kerner the first published report of a case of sausage poisoning appeared in 1735, and from that time to 1853 Schlossberger estimates that 400 cases occurred in southwestern Germany, of which 150 terminated fatally. It is certain, however, that many of the earlier cases were not true botulism but were caused by the then unrecognized trichina spiralis.

The poisonous sausages are usually liver or blood sausages, sometimes made of materials already tainted, of large size, imperfectly smoked, and more or less softened in the interior. The central portion is in some of a grayish tinge, pulpy, and has a rancid or sharp and somewhat acid taste. The cortical portion is sometimes inert, while the central portion is actively

poisonous. The odor is sometimes musty, but in most cases in no way peculiar. The odor of putrefaction is not observed. The nature of the poison has not been determined; although Gaffky and Paak have obtained from a sausage which had caused numerous poisonings, and one death, a bacillus which produced similar symptoms in animals.[1]

The action of the poison is usually manifested in from twelve to twenty-one hours after it has been taken. The shortest interval was one hour in one of Kaatzer's cases,[2] and in two of Eichenberg's two and three hours.[3] The recorded cases with an interval longer than twenty-four hours are probably cases of parasitic disease.

The total DURATION of fatal cases is very variable, as is shown by the 48 cases cited by Müller,[4] of which 6 died on the first day, 19 from the second to the seventh, 16 from the eighth to the tenth, 4 from the eleventh to the twenty-first day, and 3 after long illness. Recovery is usually slow, and may require several months. Total blindness may remain.

The earliest SYMPTOMS are usually gastro-intestinal: nausea, abdominal pain, not intense, but increased on pressure; vomiting of yellow, pulpy, sour, or bitter masses, and not infrequently purging. These symptoms, which in light cases are sometimes the only ones observed, are never accompanied by the cyanosis and cramps which occur in poisoning by mussels, by certain varieties of fish, and by cheese. Following upon the gastric symptoms, either immediately or after an interval of a day or more, or, in some cases, without the occurrence of gastric symptoms, the truly poisonous action is established. The secretions are much diminished or arrested, the mouth, tongue, and pharynx are dry, and neither saliva, perspiration, nor tears are secreted. The skin is wrinkled and cool. The tongue is covered with a whitish coat, the buccal mucous membrane is marked with aphthous patches, and the pharynx is inflamed or ulcerated, and coated with a grayish-white deposit. The voice is hoarse and in some cases there is a dry cough. The urine seems to be the only secretion which is not diminished. There is diminished power of vision, which may reach

[1] "Arb. a. d. K. Gsdhtsamt.," Berl., 1890, vi., 159.
[2] Deut. med. Wchnschr., 1881, vii., 73.
[3] Diss. Göttingen, 1880.
[4] Deut. Klinik, 1869, 1870, *passim*.

the point of temporary or even permanent blindness; objects appear colored and sometimes double, and the field of vision is traversed by sparks. The muscles moving the globe are partially paralyzed as well as the accommodation, and the pupils are dilated. Paralysis of the levator palpebræ superioris, with consequent ptosis, is characteristic. Interference with deglutition is a marked and constant symptom, and is frequently accompanied by interference with the movements of the tongue and with hoarseness, or even aphonia. The cerebral functions and those of the spinal nerves are but little interfered with. Consciousness is perfect, almost up to the time of death. Sensation remains unimpaired. There is great and progressive muscular weakness, and progressive emaciation, due to complete loss of appetite. The patient complains chiefly of the derangements of vision and of attacks of suffocation.

Death is caused in many cases by marasmus. In some cases suffocation, with or without preceding convulsions, is the cause of death; or sometimes suffocation, due to entrance of particles of food into the air passages.

The symptoms of botulism present some similarities to those of the atropic poisons (see p. 668), which also produce dilatation of the pupil, paralysis of accommodation, marked dysphagia, and diminution of the secretions. But the cerebral symptoms —delirium and hallucinations—of atropic poisoning are absent in botulism, while ptosis does not occur in atropic poisoning. The pulse is also much more rapid in atropic poisoning, and the duration of the poisoning is much shorter than in botulism. The similarity to the action of gelsemium is closer. The effects upon the eye and its muscles, including ptosis, is the same in the two forms of poisoning, which may, however, be distinguished by the much more rapid and, in case of recovery, temporary action of the vegetable poison.

No characteristic POST-MORTEM APPEARANCES are recognizable. The body is emaciated, and the skin is bleached and parchment-like. Rigor mortis begins early and continues longer than usual, and putrefaction is delayed in some cases, but very rapid in others. The mucous membrane of the mouth and pharynx is often white, dry, and parchment-like, or horny. The gastric mucous membrane is frequently injected and sometimes ecchymosed. The spleen is sometimes enlarged, con-

gested, and extremely soft and friable. The blood, as a rule, is dark. The lungs are frequently congested and œdematous.

Bibliography.—See Ind. Cat. Surg.-Gen. Libr., xii., 545, to which add Van den Corput: "Du poison . . . dans les boudins fumés," Bruxelles, 1855. Emmerich: *Ber. ii. d. Versam. d. Ver. bayer. Vort. d. aug. Chem.* (1891), 1892, x., 70. Glass: *Pest. med.-chir. Presse*, 1888, xxiv., 575. Haberda: *Ztschr. f. med. Beamte*, 1893, vi., 601. Jeserich und Niemann: *Hyg. Rundschau*, 1893, iii., 813. Kornfeld: *Friedreich's Bl. f. ger. Med.*, 1892, xliii., 385. McDougall: *Med. Pr. and Circ.*, 1879, n. s., xxviii., 550. Reisz: *Wien. med. Presse*, 1891, xxxii., 1862. Rizzi and Dall' Acqua: *Gior. d. r. soc. ital. d'ig.*, 1893, xv., 5. Schroeder: *Vrtljschr. f. ger. Med.*, 1893, 3 F., vi., Supplhft. 104. Slucha: *Vrach. Vaidom.*, 1880, v., 1500. Spät: *Friedreich's Bl. f. ger. Med.*, 1889, xl., 385, 462. Vipand: *Montreal M. J.*, 1893–94, xxii., 739.

Meat Poisoning.—Apart from the disorders produced in the human subject by eating trichinous or measly pork or beef, which are parasitic in origin, meat poisonings may be divided into two classes: 1, Those which are caused by meat which has undergone putrefactive change, and, 2, Those which are traceable to the use of meat from animals suffering from disease.

Those of the first class are usually produced by bacon, hams, or corned meats. The "Welbeck cases," described by Ballard, afforded a series of examples. Several scores of persons were affected, of whom four died after a few days, while several of those who ate of the ham, to which the poisoning was distinctly traced, escaped altogether. In most of the cases there was a period of incubation of from twenty-four to thirty-six hours. In some the attack began with a chill, in others with giddiness, faintness, or pain in the back, chest, or abdomen; but sooner or later in all there was abdominal pain, with fetid diarrhœa and, usually, vomiting. In most of the cases there were muscular weakness, headache, and thirst. Nervous phenomena were observed in some cases, cramps, twitchings, prickling sensations, numbness of the hands, drowsiness, hallucinations, disturbance of vision, and photophobia. In most cases there was fever after the early stage of the attack, the temperature rising to 101° to 104°. From the ham which was the cause of the poisonings Klein obtained a bacillus, the causative relation of which to the sickness was not, however, demonstrated. In

other reported cases the symptoms have begun shortly after the tainted meat was eaten, and have departed more or less widely from those observed in the Welbeck cases; the abdominal and cephalic pain, high temperature, nervous symptoms, and prostration are, however, tolerably constant. In many cases there was not purging but constipation.

Illness caused by the meat of diseased animals may be due to true poisoning or to what may be more properly considered an infection, transmitted, not by a bacterial product, but by the bacteria themselves. Indeed, Basenau claims the existence of a bacillus which he designates as *bacillus bovis morbificans*, which he considers as causing the disturbances frequently produced by eating the flesh of cows slaughtered soon after calving, and while suffering from septic puerperal fever.

Bibliography.—See Ind. Cat. Surg.-Gen. Libr., viii., 759–762, to which add Abelous: *Arch. d. physiol. norm. et path.*, 1894, 5 s., vi., 433. Ballard: *Tr. VII. Int. Cong. Hyg. and Demog.* (1891), 1892, iii., 102. Barnes: *Practitioner*, Lond., 1887, xxxviii., 302, 385, 463. Basenau: *Arch. f. Hyg.*, 1894, xx., 242. Bouchereau and Noir: *Arch. de méd. et pharm. mil.*, 1889, xiii., 97. Boulengier: *Presse méd. belges*, 1893, xlv., 9. Carsten: *Geneesk. Courant*, 1887, xli., No. 44. Charrin: *Rec. d. trav. Com. consult. d'hyg. publ. de Fr.* (1887), 1888, xvii., 317. Chesney: *Sanitarian*, N. York, 1888, xx., 412. Demuth: *Vereinsbl. d. pfälz. Aerzte*, 1888, iv., 51, 65. Engelbrecht: "Drei Todesfälle durch Schinkenvergiftung," Jena, 1894. Fadeaux: *Arch. méd. belges*, 1887, 3 s., xxxi., 375. Fiquet: Thèse, Par. (No. 184), 1894. Flinzer: *Vrtljschr. f. ger. Med.*, 1887, xlvi., 105. Fröhner u. Kundsen: *Monatsch. f. prakt. Thierhlk.*, 1889–90, i., 529. Gärtner: *Bresl. aerztl. Zeitschr.*, 1888, x., 249, 264, 277, 292. Gemmell: *Liverp. Med.-Chir. J.*, 1888, viii., 94. Gust: "Ueber Wildvergiftung," Berl., 1894. Gutmann: *Berl. kl. Wchnschr.*, 1891, xxviii., 203. Gwynn: *Public Health*, Lond., 1892–93, v., 68. Herzog: *Med. Rec.*, N. York, 1894, xlv., 155. Holst: *Norsk. Mag. f. Läg.*, 1894, lv., 781 (1 pl.). Jeserich u. Niemann: *Hyg. Rundschau*, 1893, iii., 813. Juhel-Rénoy: *Bull. et mém. Soc. méd. des hôp. de Paris*, 1893, 3 s., x., 430; *Ann. d'hyg.*, 1893, 3 s., xxx., 113. Kluyskens: *Ann. Soc. de méd. de Gand.*, 1890, lxix., 176. Levy: *Arch. f. exp. Pharm. u. Path.*, 1894, xxxiv., 342. Lewis: *Chem. News*, 1893, lxvii., 52. Lieventhal: *Ph. Ztschr. f. Russl.*, 1887, xxvi., 305, 321. Lohmeyer: *Berl. kl. Wchnschr.*, 1887, xxiv., 53. Macé: *Ann. d'hyg.*, 1891, 3 s., xxv., 268. Macfadyen and Blyth: *Public Health*, 1892–93, v., 98. Mouginet: Thèse, Par., 1891 (1 pl.). Ostertag: *Ztschr. f. Fleisch- u. Milchhyg.*, 1891–92, ii., 193, 210, 227. Polin et Labit: *Arch. d. méd. et pharm. mil.*, 1889, xiv., 372. Proust:

Bull. méd., Paris, 1888, ii., 939. Von Ratz: *Monatsch. f. prakt. Thierhlk.*, 1893–94, v., 11. Reed: *J. Am. Med. Assoc.*, 1887, viii., 203. Schmit: *Rev. d'hyg.*, 1888, x., 293. Schröder: *Vrtljschr. f. ger. Med.*, 1893, 3 F., vi., Supplhft., 104. Spamer: *Deut. Arch. f. kl. Med.*, 1886–87, xl., 187. Spear: *Rep. Med. Off. Soc. Gov. Brd.* (1887), 1888, xviii., 106 (1 pl.). Ströse: *Ztschr. f. Fleisch- u. Milchhyg.*, 1892–93, iii., 196. Suter: "Die Fleischvergiftung in Andelfingen und Kloten," Zurich, 1889. Tavel: *Cor.-Bl. f. schweiz. Aerzte*, 1887, xvii., 417. Underhill: *Brit. M. J.*, 1892, ii., 631. Van Emergem: *Bull. Ac. roy. de méd. de Belg.*, 1892, 4 s., vi., 1025. Vassale e Rossi: *Riv. sper. di freniat.*, 1893, xix., 676.

Poisonous Fish.—Certain species of fish are always poisonous to the human subject, or become so at times, and sometimes the same fish, eaten with impunity by one person, proves actively poisonous to another. Poisonous fish are usually the inhabitants of warm climates. The use of a Japanese fish, *fugu*, a species of Tetrodon, is interdicted by law in that country. In three cases of poisoning by this fish, one of which terminated in death, the symptoms began in from fifteen to forty-five minutes. There were headache, nausea, great muscular weakness, failure of the pulse and respiration, diminution of temperature, and total insensibility. In southeastern Russia, poisonings by sturgeon are of frequent occurrence. The roe appears to be the most actively poisonous part of the fish.

Edwards reports the poisoning of a man of sixty-eight years by the flesh of the ray. In fifteen minutes the face began to swell; there was a burning sensation in the hands and back of the head; the feet were cold; the tongue swelled until it filled the cavity of the mouth, causing a choking sensation at the root of the tongue; the eyes were swollen and protruded. There was no pain, but great dyspnœa, thirst, and an intense itching of the skin. Consciousness was perfect. The patient vomited freely, and soon recovered. In Sobbe's case there were attacks of syncope, great thirst, and a sense of oppression, and the pulse became as slow as fourteen in the minute.

Tinned salmon, herrings, sardines, etc., have also caused poisoning, probably by the generation in them of putrid poisons. In a case of this kind, reported by Mead, the face had an anxious expression, and the eyeballs were prominent. The temperature was 99° to 101°.5. The tongue was coated. There were great thirst, severe vomiting, copious

diarrhœa, finally of pure blood, without straining, abdominal pain, irregular respiration, and rapid, irregular, and intermittent pulse.

Bibliography.—See Ind. Cat. Surg.-Gen. Libr., iv., 991–992, to which add: Addinsell: *Lancet*, 1884, ii., 540. Alexander: *Bresl. aerztl. Ztschr.*, 1888, x., 25. v. Anrep: *Vrach*, 1885, vi., 213. Arnstamoff: *Centbl. f. Bakter. u. Par.*, 1891, x., 113. Belin: *France méd.*, 1886, ii., 1458. Béranger-Férand: *Ann. d'hyg.*, 1885, 3 s., xiv., 331, 427, 516. Bertherand: *J. de méd. et de pharm. de l'Algerie*, 1884, ix., 3 (1 pl.). Berthier: Thèse (No. 466), Lyon, 1889. Carruccio: *Bull. d. r. Ac. di Roma*, 1886–87, viii., 353. Edwards: *Brit. M. J.*, 1884, i., 40. Fischel and Enoch: *Fortschr. d. Med.*, 1892, x., 277. Grancher: *Rec. d. trav. com. consult. d'hyg.*, etc. (1887), 1888, xvii., 1. Griffiths: *Chem. News*, 1893, lxviii., 45. Heckel: *Rev. san. d. Bordeaux*, 1887, iv., 84; *C. rend. Ac. d. sc.*, Par., 1888, cvi., 220. Hirschfeld: *Vrtljschr. f. ger. Med.*, 1885, n. F., xliii., 283. Inoko: *Tokio med. Wchnschr.*, 1892, No. 754, 1. Kakahashi and Inoko: *Sei-i-Kwai M. J.*, 1892, xi., 46. Klimenko: *Protok. Zasaid. Kavkaszk. med. Obsh.*, Tiflis, 1893–94, xxx., 647. Knoch: *Voyenno-med. J.*, St. Pet., 1888, clxi., pt. 3, 399, clxii., 1, 147, 181 (2 pl.). Lardier: *Bull. méd. d. Vosges*, 1891–92, vi., No. 23, 80, 83. Layet, Artigalas et Ferré: *Rev. san. de Bordeaux*, 1886, iii., 49. Loir: *C. rend. soc. de biol.*, Par., 1894, 9 s., vi., 141. Lusanna: *Bol. de Soc. med. prov. di Bergamo*, 1894, v., 60. Macgowan: *China Imp. Customs Med. Rept.*, Shanghai, 1884, xxvii., 12. Marcacci: *Arch. ital. de biol.*, 1891–92, xvi., 1. Mauriac: *J. de méd. de Bordeaux*, 1885–86, xv., 425, 437, 459, 461. Mead: *Practitioner*, Lond., 1886, xxxvi., 264. Millet: *Arch. de méd. et pharm. mil.*, 1886, viii., 417. Miura u. Takesaki: *Arch. f. path. Anat.*, etc., 1890, cxxii., 92. Mosso: *Arch. ital. de Biol.*, 1888, x., 141. Naunyn: *Ph. Centhalle.*, 1885, xxvi., 234. Osawa Kenji: *Iji Shinbun*, Tokio, 1884, No. 22. Pohl: *Prag. med. Wchnschr.*, 1893, xviii., 31. Popoff: *Voyenno-med. J.*, St. Pet., 1887, clviii., pt. 3, 65. Prochorov: *Vrach*, 1884, v., 54. Rawe: Diss., Dorpat, 1889. Richardson: *Asclepiad*, Lond., 1893, x., 39. Sartschenko: "Atlas des poissons vénéneux," St. Pet., 1886 (10 col. pl.). Schmidt: *Verh. d. X. internat. med. Congr.* (1890), 1891, ii., 4 Abth., 43. Schreiber: *Berl. kl. Wchnschr.*, 1884, xxi., 161, 183. Simsohn: *Polyclinic*, Phila., 1887–88, v., 111. Slumin: *Med. pribav. k. morsk. sborniku.*, St. Pet., 1892, i., 269. v. Sobbe: *Berl. kl. Wchnschr.*, 1889, xxvi., 137. v. Sobierowski: Diss., Strasburg, 1890. Spofforth: *Austral. M. Gaz.*, 1886–87, vi., 266. Stevenson: *Brit. M. J.*, 1892, ii., 1326. Takahashi u. Inoko: *Centbl. f. d. med. Wissensch.*, 1889, xxvii., 529, 881; *Mitth. a. d. med. Fac. d. k. jap. Univ.*, Tokio, 1892, i., No. 5, 375 (3 pl.); *Arch. f. exp. Path. u. Pharm.*, 1889–90, xxvi., 401, 453. Thomson: Diss., Dorpat, 1882.

Vasilev: *Novosti vet. lit.*, St. Pet., 1885, ii., No. 3. Wear: *Lancet*, 1888, ii., 193. Wilson: *Brit. M. J.*, 1887, ii., 992. Wolff: *Vrtljschr. f. ger. Med.*, 1858, xiv., 232. Yamawaki: *Iji Shinbun*, Tokio, May 5th, 1883.

Poisonous Shell-Fish. — The edible mussel (*Mytilis edulis*) frequently becomes actively poisonous, probably by reason of changes in the character of the surrounding water. A case is recorded in which serious symptoms and a scarlatinal eruption were caused by eating two mussels; and another in which similar effects followed the ingestion of a single mussel. Death has resulted in several instances. Farrar reports the case of a strong, temperate, well-built man of sixty who had been in the habit of eating mussels without ill effects. Immediately after eating about twenty, without removing the "moss," or byssus, he felt sick and had griping abdominal pain. In half an hour he vomited and purged severely several times. In two and a half hours he was in a state of extreme collapse. The hands and feet were cold; the radial pulse was almost imperceptible and much increased in frequency. The face was pale, except occasional hectic flushes on the cheeks; the nose was pinched and quite bloodless, the pupils were normal, and the tongue was clean. There was loud wheezing and rattling respiration, with frequent yawning and sighing. The patient fainted frequently in spite of large doses of brandy. There were repeated clonic spasms, apparently implicating all the muscles of the body. He was perfectly calm and conscious, suffering no pain, but great thirst, and itching over the entire surface. There was neither sense of constriction in the throat nor headache. Surrounding objects appeared misty. The chest was covered with an erythematous rash. The next morning recovery was complete.

The mussels which caused the poisonings at Wilhelmshaven in 1885 came from a stagnant basin; but those taken from the neighborhood of the sluices where the water was changed were not poisonous. The poisonous mussels, when transplanted to pure sea water, lost their poisonous quality competely in from two to four weeks; while non-poisonous mussels became actively toxic after two or three weeks' sojourn in the stagnant water of the basin. The poisonous individuals have a somewhat lighter and more striped shell than those which are innoc-

uous. From the poisonous mussels Brieger obtained an alkaloidal poison, *mytilitoxin*.

Poisonings by oysters, lobsters, etc., have also occurred.

Bibliography.—See Ind. Cat. Surg.-Gen. Libr., iv., 991–92, to which add: *Mussels.*—Bardet: *C. rend. d. trav. du lab. de thérap.*, 1889, 106. Cameron: *Lancet*, 1890, ii., 174. Descroizilles: *Rev. mens. d. mal. de l'enf.*, 1885. Dubrisay: *Union méd.*, 1888, 3 s., xlvi., 409. Griffiths: *Chem. News*, 1890, lxii., 17. Jourdain: *C. rend. ac. d. Sc.*, Par., 1891, cxii., 106. Lindner: *Deut. med. Ztg.*, 1888, ix., 585, 597. Lohmeyer: *Arch. f. path. Anat.*, etc., 1886, civ., 169. *Berl. kl. Wochenschr.*, 1886, xxiii., 167. McWeeney: *Brit. M. J.*, 1890, ii., 628. Manicus: *Hosp. Tid.*, Kjøbenh., 1888, 3 R., vi., 889. Möbius: *Schrift. d. naturw. Verh. f. Schlesw.-Holst.*, 1886, vi., 2 Hft., 3. Monod: *Rev. san. de Bordeaux*, 1883–84, i., 27. Permewan: *Lancet*, 1888, ii., 568. Salkowski: *Arch. f. path. Anat.*, etc., 1885, cii., 578. Schmidtmann: *Ztschr. f. med.-Beamte*, 1888, i., 19, 49. Schuster: *Deut. med. Wochenschr.*, 1886, xii., 304. Todd: *Brit. M. J.*, 1891, ii., 749. Virchow: *Deut. med. Wochenschr.*, 1885, xi., 831; *Arch. f. path. Anat.*, etc., 1886, civ., 161. Wolff: *Arch. f. path. Anat.*, etc., 1886, ciii., 187; civ., 180; 1887, cx., 376. *Oysters.*—Cameron: *Brit. M. J.*, 1890, ii., 685. Lankester: *Quart. J. Micr. Sc.*, 1885–86, n. s., xxvi., 71 (pl). Liégois: *J. de méd. de Paris*, 1886, xi., 56. Moody: *Therap. Gaz.*, 1883, n. s., iv., 283. Upshur: *N. York M. J.*, 1890, lii., 583. *Shrimps.*—Lee: *Local Gov. Chron.*, Lond., 1884, xxii., 76. *Periwinkles.*—*Sanit. Rec.*, Lond., 1883–84, n. s., v., 355.

Poisonous Cheese, Milk, etc.—In cheese poisoning the symptoms are chiefly those due to irritation: severe vomiting, first of watery, then of reddish masses, purging, abdominal pain, dizziness, and prostration, beginning in from one to four hours. The face is at first pale, later cyanosed; the pulse is feeble and irregular; and the throat feels dry and constricted. Although the symptoms are frequently very severe, the poisoning very rarely terminates in death. The few fatal cases have almost all been in young children. The poisonous constituent of such cheeses, whether it be the crystalline base of Lepierre or the so-called tyrotoxicon of Vaughan (or tyrotoxin if it be a definite compound), may either be produced by putrefactive changes in the cheese or may pre-exist in the milk from which it was made.

Milk has been known to produce serious outbreaks of poisoning or disease. In many of these the injurious effects are

to be attributed, not to the existence of any poison in the milk, but to the use of impure water as an adulterant. It has also been shown that the milk from diseased cows is capable of communicating similar pathological conditions to the human subject. But apart from such instances of disease transferral, cases have occurred in which true poisoning has been caused by milk. Thus Firth describes the cases of ten soldiers in a military prison. Of these one abstained from the milk in question, and was not affected. The other nine were seized with nausea, vomiting, dryness of the fauces, a sense of constriction of the throat, colic, purging, and a tendency to collapse in some, in others a tendency to stupor. All recovered. A substance, semi-crystalline, mawkish in odor, pungent in taste, extracted from this milk, and called *lactotoxin* by Firth, caused nausea, dryness of the fauces, and headache in the human subject, and purging and vomiting in a dog.

Cases of poisoning by articles of food into whose composition milk enters, such as puddings, ice cream, etc., are of occasional occurrence.

Bibliography.—*Cheese.*—See Ind. Cat. Surg.-Gen. Libr., ii., 866; xiv., 918, to which add: Apéry: *Rev. méd. pharm.*, Constant., 1888, i., 114. Cameron: *Dubl. J. M. Sc.*, 1882, 3 s., lxxii., 116. Ehrhardt: *Vereinsbl. d. pfälz. Aerzte*, 1887, iii., 4. Fulton: *Am. J. Surg. and Gyn.*, Kansas City, 1894–95, v., 90. Glaesel and Holm: *Schmidt's Jahrb.*, clxxxv., 21. Harvey: *Phys. and Surg.*, Ann Arbor, 1894, xvi., 385. Lepierre: *C. rend. ac. Sc.*, Par., 1894, cxviii., 476. Müller: *Jahrb. d. ges. Staatsarznk.*, 1837, ii., 430. Pflüger: *Med. Cor.-Bl. d. würt. aerztl. Ver.*, 1894, cxiv., 145. Prollins: *Ann. d. Staatsarznk.*, 1841, vi., 355. Reed: *Texas Hlth. J.*, 1893–94, vi., 23; *J. Am. M. Assoc.*, 1893, xxi., 354. Vaughan: *M. and S. Reptr.*, Phila., 1890, lxiii., 584. Vaughan and Novy: "Ptomains," etc., 3d ed., 1896, 83. Wallace: *Med. News*, Phila., 1887, li., 69. Weigel: *N. Mag. f. d. ger. Arznk.*, etc. (Pyl), 1786, i., 1. Willich: *Ibid.*, 1786, i., 667. *Milk.*—See Ind. Cat. Surg.-Gen. Libr., ix., 294–295, 307–308, to which add: Anon., *Brit. M. J.*, 1894, i., 1331. Cassebat: *Rev. d'hyg.*, 1893, xiv., No. 9. Corwin: *Phys. and Surg.*, Ann Arbor, 1880, ii., 201. Demme: *Pediatr. Arb.*, Berl., 1890, 412. Gray: *Austral. M. J.*, 1893, n. s., xv., 565; *Practitioner*, Lond., 1894, lii., 67. Mackaye: *R. I. Med. Sc. Monthly*, 1893, i., 571. Winternitz: *Zeitschr. f. physiol. Chem.*, 1892, xvi., 460. *Ice Cream, etc.*—See Ind. Cat. Surg.-Gen. Libr., vi., 771; v., 63–64, to which add: Allaben: *Med. Rec.*, N. York, 1886, xxx., 417. Allen: *San. Rec.*, Lond., 1878, ix., 257 (pudding). Anon., *M. Times*

and Gaz., 1878, ii., 519 (pudding). Blaschko: *Vrtljschr. f. ger. Med.*, 1884, 3 F., vii., 362. Gibson: *Med. Rec.*, N. York, 1886, xxx., 249. Hull: *M. News*, Phila., 1891, lviii., 713. Lombroso and Ottolenghi: *Gior. d. r. Acc. di med. di Torino*, 1890, 3 s., xxxviii., 757 (polenta). Smith: *Rep. Bd. Health, Conn.* (1889), 1890, xii., 232. Vaughan: *M. Rec.*, N. York, 1886, xxx., 306.

Miscellaneous.—Acute and chronic poisonings have been produced by a great variety of food articles. In some instances acute symptoms are produced in certain persons by a particular article of food by reason of an idiosynerasy of the individual. Thus some persons are rendered severely ill by eating eggs,[1] others by strawberries or other berries,[2] etc. In other cases a vegetable food has caused poisoning, either by reason of its retaining a harmful ingredient in consequence of improper preparation, as by eating uncooked potatoes or the ordinary French bean; or because of its having undergone decomposition, as in the numerous instances of poisoning by tomatoes and other "canned goods." Bread and other food articles prepared from cereals have been the cause of both acute and chronic poisoning, either in consequence of the formation of a toxic bacterial product in the grain (maïdism, blue bread), or because of the admixture of a poisonous vegetable by accident (Lolium temulentum, Agrostemma githago; ergotism, lathyrism, etc.).

CANTHARIDES.

The body of the Spanish fly (*Lytta vesicatoria*) and of certain related beetles (*Cantharis*, *Mylabris*, *Meloe*) contain an acrid, irritant substance of acid nature:

Cantharidin — $C_{10}H_{24}O_4$.[3]—It crystallizes in four-sided, rhombic prisms, very sparingly soluble in water, soluble in 800 parts of alcohol, in 80 of chloroform, in 900 of ether, and in 500 of benzene, soluble in oils. Its salts are also sparingly soluble. It is a powerful vesicant.

Poisonings by cantharides appear to have been of more fre-

[1] Hutchinson: Brit. M. J., 1886, i., 1018; Arch. Surg., Lond., 1893–94, v., 363. Orton: Practit., Lond., 1886, xxxvi., 265. Brothers: Med. Rec., N. York, 1887, xxxi., 545. Glassmacher: Berl. klin. Wchnschr., 1886, xxiii., 666.

[2] Amyot: Brit. M. J., 1885, i., 986. Broadbent: *Ibid.*, 1884, i., 267.

[3] Homolka: Berichte, Berl., 1886, xix., 1082, and papers therein referred to.

quent occurrence in France than elsewhere. Of the more recently reported cases the majority have been accidental or medicinal, very few suicidal. Many have resulted from a widespread popular belief in the aphrodisiac power of cantharides;[1] and in a few cases it has been given or taken as an abortive.[2]

Orfila[3] gives the particulars of two homicidal poisonings by cantharides: the Affaire Poirier, in 1846, an unsuccessful attempt in which cantharides plaster was added to soup; and the Affaire J. B——, in 1849, in which a woman repeatedly administered cantharides to her husband in his food and in enemata. In Reg. *v.* Royal, 1859,[4] a woman died in four days, apparently from the effects of cantharides; and in another English case of the same year,[5] a man administered cantharides in food to a female fellow-servant and six others of the household, but was discharged because "there was no criminal law to meet the case." Taylor[6] mentions two prosecutions for administration of cantharides to females, one in 1836, the other in 1861.

Symptoms.—The action of cantharides is that of an intense local irritant, either upon the parts to which it is first applied or upon the urinary organs during their elimination of the poison. Applied to the skin it produces pain, redness, swelling, and, by more prolonged contact, vesication. When taken by the mouth it causes immediate burning pain in the mouth and throat, which extends to the entire alimentary canal and becomes most intense. There are intense thirst, vomiting, purging, frequently of bloody stools, difficulty in swallowing, and pain in the region of the kidneys and bladder. In the male there is painful priapism, and in the pregnant female abortion; in both sexes strangury, with bloody urine, or the voiding of pure blood. The pulse is accelerated, the respiration impeded, and nervous disturbances, headache, dilatation of the pupil, vertigo, delirium, convulsions, trismus, coma, and collapse.

Treatment.—The stomach should be evacuated with the siphon, or by apomorphin, and the intestine by mild purga-

[1] Schauenstein: Maschka's "Handb. der ger. Med.," ii., 738. Orfila: "Tox. gén.," 5ème ed., ii., 174.

[2] Beck: N. Amer. Practit., 1891, iii., 522. Orfila: *Op. cit.*, ii., 180.

[3] *Op. cit.*, ii., 192, 195.

[4] Ph. J. and Tr., 1859–60, n. s., i., 150.

[5] *Ibid.*, p. 388.

[6] "Poisons," 3d Am. ed., 508.

tives. Morphin and mucilaginous drinks should be given to control the pain. Oily foods or medicines are to be avoided as favoring solution.

Post-Mortem Appearances.—The mucous membrane of the entire alimentary tract is found highly inflamed, and sometimes marked with ulcerations and vesications. The kidneys are the seat of parenchymatous inflammation, and the bladder is also inflamed and its blood-vessels injected.

Detection.—When the powdered drug has been taken, the shining, green particles of outer portions of the beetle may be found in the stomach, vomit, or washings. Although cantharidin is sparingly soluble it may be encountered in the benzene extract from the acid aqueous liquid in the systematic process (see p. 135). When the history of the case points toward cantharides poisoning, Dragendorff's direct method should be adopted. The finely divided material is heated with aqueous potash (1 KHO : 12–15 H_2O) until a uniform fluid is obtained. This is cooled, thinned with water if necessary, and shaken with chloroform to free it from other substances soluble in chloroform. After separation of the chloroform the alkaline liquid is supersaturated with sulfuric acid and immediately mixed with four volumes of alcohol. The mixture is boiled for some time, filtered hot, strongly cooled, again filtered, and the filtrate freed from alcohol by distillation. The acid aqueous solution is then extracted by chloroform by agitation, and the residue of evaporation of the chloroform tested as to its irritating and vesicating power, by solution in warm oil and application to the skin of the inside of the arm.

SYNTHETIC POISONS.

In this class are included poisonous organic substances (compounds of carbon) which either have no existence in nature, or occur naturally only in minute quantity; substances which are the products of the chemical laboratory or of industrial processes. The number of such substances known to chemists is daily increasing, and some of them, having been applied to uses in the arts and in medicine, are now within the reach and knowledge of all persons, and even the most uneducated are acquainted with their poisonous qualities.

The toxicology of many of these synthetic products has been studied, and instances of accidental poisoning by them have been observed. Only a few, however, have thus far been subjects of interest in forensic toxicology.

CARBON MONOXID.

Carbon monoxid (carbonic oxid), CO—is a colorless, tasteless gas, practically without odor; sp. gr. 0.9678 A.; very sparingly soluble in water and in alcohol. It burns in air with a blue flame, and forms explosive mixtures with air or oxygen. At high temperatures it is a powerful reducing agent. It is produced whenever carbon (particularly charcoal or anthracite coal) is burnt with a deficient supply of air. Consequently it is a constituent of coal gas (4 to 7.5 per cent.), "water gas" (30 to 35 per cent.), the gases from stoves or furnaces (1 to 17 per cent.), the gases from blast furnaces and copper smelting furnaces (13 to 32 per cent.), the fumes from burning charcoal, etc.

The actively poisonous character of this gas, its lack of odor, and the very common fulfilment of conditions under which it may be discharged into inhabited spaces render it, practically, the most dangerous of toxic gases. In illuminating gas

there is a certain factor of safety in the odor of other gases which accompany it, but even this has not prevented numerous accidental poisonings. In the gases from stoves and furnaces this factor of safety is absent, as, once the smoke produced by the kindling has ceased to be given off, the gases have but a slight and practically unnoticeable odor. The number of accidental and suicidal deaths from carbon monoxid now exceeds that of similar poisonings by any other agent. Nearly one-half of the accidental poisonings in New York City in 1889–92 were by illuminating gas, and the same agent was used by suicides more frequently than any other poison except Paris green and "rat poison." In France a very common method of suicide is by inhalation of the fumes of burning charcoal. We find no mention in toxicological literature of homicidal poisonings, unaccompanied by simultaneous suicide, by gases owing their toxic quality to carbon monoxid, except in one of Liman's cases[1] in which it was questioned whether the death of four persons by illuminating gas was accidental, suicidal and homicidal, or purely homicidal. In a recent American case, however, a man enticed two boys to a room in a hotel, where, when they slept, he turned on the gas and left them, having closed the room, in which they were found dead in the morning.

Carbon monoxid poisoning may be of forensic interest in other connections: Negatively when death has been caused by mechanical suffocation, and illuminating gas is resorted to to conceal a crime. Several such cases are cited by Liman.[2] Or the conditions under which a person is fatally asphyxiated by the gases from a stove, furnace, etc., may amount to criminal negligence on the part of another. Or an insurance policy may be claimed to have been rendered void on the ground of suicide or of "contact with poisonous substances."

Symptoms.—The early symptoms of carbon monoxid poisoning are rarely observed. If the attack be sudden, by entrance into an already contaminated atmosphere, the person may immediately fall unconscious and the heart's action become progressively slower until it stops. More usually the poisonous gas is gradually added to the atmosphere, when the patient experiences a sense of heaviness in the head, severe

[1] Casper-Liman: "Handb. d. ger. Med.," 8te Aufl., ii., 610.

[2] *Op. cit.*, 69, 604, 607.

headache, with throbbing of the temples, vertigo, a sense of oppression in the chest, accompanied by trembling, muscular weakness, and tearing pains in the chest; the heart's action is irregular and tumultuous, and the respiration stertorous; then follows general insensibility and death in coma.

Recovery is slow, and during its progress the patient experiences trembling, pains in the limbs, head, and chest, and obscuration of vision. The memory is usually impaired, and paralysis of the upper extremities has been observed.

The METHOD OF ACTION of carbon monoxid, whatever be its source, is clearly defined. The function of the red pigment of the blood corpuscles (hæmoglobin) is to carry oxygen from the lungs to the tissues, a function which it exerts by virtue of its capacity to become oxidized in the lung and deoxidized in the capillary circulation. Carbon monoxid combines chemically with hæmoglobin, to form a compound more stable than oxyhæmoglobin, and hence, by preventing the absorption of oxygen in the lungs, it produces what may be termed an internal or circulatory asphyxia, and the respiratory function of the hæmoglobin so combined is permanently destroyed.

Treatment.—This should be directed to the supply of pure air and the maintenance or restoration of respiration. The patient should be transferred to a purer atmosphere, or the windows and doors widely opened. In light cases, in which respiration and consciousness are not seriously impaired, this will usually suffice. In more serious cases cold affusions to the head while the body is kept warm, the inhalation of ammonia, external irritation, and artificial respiration with inhalation of pure oxygen should be resorted to. Transfusion of blood, which seems to be directly indicated, has been attempted in several instances without beneficial result. Infusion of alkaline salt solution or of milk has been practised with more favorable effects.

Post-Mortem Appearances and Detection.—The post-mortem appearances are almost diagnostic. The blood everywhere is of a bright red color, and this color is communicated to various parts of the body. The lungs on section are bright crimson in color; patches of skin, particularly on the inner surfaces of the thighs and about the genitals, are often similarly colored; and blood which escapes from the nostrils, and bloody froth found in the larger air passages, have the same hue. The

only other condition in which a similar color of the blood is observed after death is in some cases of hydrocyanic-acid poisoning. The spectroscope may be depended upon to remove all doubt. The spectrum of carbon monoxid hæmoglobin consists of two bands, more nearly equal to each other and more toward the violet end of the spectrum than the two oxyhæmoglobin bands. The addition of a solution of ammonium sulfid does not affect the carbon monoxid spectrum, but converts the oxyhæmoglobin spectrum into that of hæmoglobin. (See Vol. II., p. 31, Pl. III., Nos. 6, 2, 5).

CHLOROFORM.

Chloroform ($CHCl_3$) is a colorless liquid, volatile at all temperatures, boiling at 60°.8 (141°.4 F.), having a burning and sweetish taste, and a characteristic, strong, somewhat ethereal and agreeable odor, very sparingly soluble in water, but miscible in all proportions with alcohol and ether. Sp. gr. 1.497.

Chloroform is poisonous both when swallowed as a liquid and when its vapor is inhaled.

Poisonings by the ingestion of liquid chloroform are of exceptional occurrence, and have been accidental or suicidal. The burning taste and characteristic odor render it unfit for secret administration; although in a sensational English case it was alleged to have been administered with homicidal intent by a woman to her husband under peculiar conditions.[1]

Medico-legal questions relating to the administration of vapor of chloroform relate chiefly to allegations of the commission of other crimes upon persons who are brought under the influence of the anæsthetic, either with the consent of the person said to have been injured, or without such consent. Cases of the first class are usually accusations made by females against physicians or dentists, and involve rather the mental and physical condition of the accusing and accused parties than any question of toxicology.[2]

In cases of the second class the question of the possibility of suddenly or secretly rendering a person incapable of resistance

[1] Leach: Lancet, 1886, i., 968; 1017 (case of Edwin Bartlett).

[2] See articles on "Mental Diseases Vol. III. and "Rape" Vol. II.; also Lyman: "Artificial Anæsthesia and Anæsthetics," N. York, 1881, 93–98.

by inhalation of vapor of chloroform is usually involved. If the person be asleep it is possible, although difficult, to produce anæsthesia and unconsciousness without awakening. Although in the great majority of cases in which this has been attempted by physicians, experimentally or otherwise, the persons have awakened in spite of every precaution to render the conditions such as are most favorable to success, in some instances it has succeeded; and it must consequently be considered as within the range of possibility.[1] The statement that a person while awake was instantly rendered insensible by chloroform vapor alone is not to be accepted. It requires from two to ten minutes to produce anæsthesia by chloroform vapor properly diluted with air, and a successful attempt to suddenly anæsthetize a person with the undiluted vapor would cause death. The chloroforming of an unwilling and waking person by administration extending over any appreciable time involves the use of force or restraint. Consequently, under these circumstances, the determination of the truth or falsity of the accusation depends upon the circumstances of the case and the relative strength of the parties, rather than upon toxicological data.[2]

We find no mention in toxicological literature of administration of vapor of chloroform with intent to murder. Yet it is clear that such cases may occur. It is probable that in the Preller-Maxwell homicide in St. Louis in 1885 chloroform was one of the agents used; and in a more recent case, in Trenton, N. J. (1890), a woman died from the effects of inhaled chloroform, possibly administered by another with homicidal intent.

Lethal Dose.—Liquid: Half an ounce has proved fatal to a woman, apparently affected with epilepsy,[3] and six drachms caused the death of a suicide.[4] Taylor also cites the case of a boy of four years who died from the effects of a drachm. On the other hand, the same author relates an instance in which a man recovered in five days from the effects of four ounces; and Oliver one of recovery from the effects of three ounces.[5]

[1] See Lyman: *Op. cit.*, 90–93. Blyth: "Poisons," 136.

[2] See Wharton and Stillé: "Med. Jur.," 4th ed., ii., 389–395. Tourdes: "Dict. encyclop. d. sc. méd." Rogers: Papers, Med.-Leg. Soc., N. York, 1874, 1 s., 298.

[3] Taylor: "Poisons," 3d Am. ed., 619., xx.

[4] Fox: Tr. Louisiana Med. Soc., 1889, 123.

[5] Brit. M. J., 1882, i., 775.

Vapor: The minimum lethal dose of chloroform thus administered cannot be absolutely stated. Deaths have occurred from the effects of a few drops, and large quantities have been given without ill effects during protracted surgical operations. The effects of a given quantity will depend upon the condition of health of the patient, the degree of dilution of the vapor with air, and the purity of the chloroform.

Duration.—Liquid: The action of chloroform taken by the mouth is not rapid. The average duration of fatal cases is about twelve hours. In the case of the four-year-old boy above referred to death occurred in three hours.

Vapor: When death results from inhalation of chloroform vapor it does so rapidly. Lyman[1] gives a table of 52 cases out of 393 "in which was recorded the time that intervened between the commencement of inhalation and the occurrence of death." In 4 of these the period was less than one minute, in 22 less than five minutes, and in all twenty-five minutes or less. Deaths have occurred at longer periods, but in them causes other than the chloroform have been operative.

Symptoms.—Liquid: The first effects of liquid chloroform when it is swallowed are those referable to its local irritating action; intense burning pain in the mouth, throat, and stomach, usually accompanied by vomiting of matters smelling strongly of chloroform and occasionally tinged with blood. The narcotic effects are soon manifested: the patient becomes drowsy, stupid, insensible, and unconscious; the eyes are fixed; the pupils at first contracted or normal, but insensible, are afterward dilated; the respiration is slow and stertorous; the pulse, at first full and strong, becomes frequent and feeble; and the surface is cyanosed, cold, and covered with profuse perspiration. In prolonged cases blood and bile are passed per anum. Death may occur from asphyxia or cardiac paralysis.

Vapor: The effects of vapor of chloroform when inhaled in overdose may be divided into three periods: 1. A stage of drunkenness, marked by hallucinations, struggling, a flushed face, contracted pupils, and a creeping sensation of the skin. This period lasts for a few minutes, or a minute or less. 2. A stage of depression, during which sensibility is lost, the patient becomes unconscious, the reflexes are abolished, the muscular

[1] *Op. cit.*, 195.

system relaxed, the temperature subnormal, the respiration slow, the pulse full and slow, and the pupils dilated. If the inhalation be discontinued this condition, if established, may continue for from twenty to forty minutes. 3. The stage of paralysis, when the pulse is irregular, the respiration fails, and the surface becomes cyanosed. Death, which results from paralysis of the heart or of the respiratory centres, may occur in any stage. Indeed, 45 per cent. of those recorded have occurred in the first stage, 35 per cent. in the second, and the balance shortly after the beginning of the third.

There is no habituation to the effects of chloroform, and those addicted to the chloroform habit either become insane or die from the effects of an overdose.

Treatment.—If liquid chloroform have been swallowed the stomach should be washed out without delay. The action of the absorbed poison may be combated, before cessation of respiration, by hypodermics of strychnin or of ammonia, by external warmth, and by keeping the patient awake. When respiration or the heart's action stops during inhalation of chloroform vapor, or threatens cessation when the liquid has been swallowed, artificial respiration is to be resorted to immediately, preferably with inhalation of pure oxygen. If the existence of cerebral hyperæmia is not indicated by lividity of the face, the head should be depressed. Falling back of the tongue is to be guarded against. The faradic current and non-depressant external stimulants should be applied.

Post-Mortem Appearances.—When the liquid has been swallowed the mucous membranes of the mouth, stomach, and intestines are found highly reddened and inflamed. Whatever the method of introduction the blood is dark and fluid, the vessels of the brain and meninges are gorged with blood, and there is hyperæmia of the lungs, liver, and kidneys. The odor of chloroform may be perceived (although it is not always) on opening the thoracic or cranial cavity or the stomach.

Detection.—When the liquid has been swallowed the stomach or the washings therefrom are the situations in which the poison will most probably be detected; if the vapor have been inhaled, the lungs and blood.

Chloroform not only withstands but also impedes putrefaction. But it is readily volatile and will escape from the body

by evaporation, particularly after an autopsy has been performed. Luedeking has shown that chloroform may be detected with facility four weeks after death in the bodies of animals asphyxiated with it.[1]

The method for the separation of chloroform from portions of the cadaver, etc., is that followed for volatile poisons, the liquid in the distilling flask being acid (see p. 127). The tests are applied to portions of the distillate, except test No. 4.

Tests for Chloroform.—1. Add a little alcoholic solution of caustic potash and a drop or two of anilin, and warm: a peculiar, disagreeable, and persistent odor, resembling that of witch-hazel, is produced.

2. Dissolve about 0.01 gm. of β-naphthol in a small quantity of caustic potash solution, warm, and add the suspected liquid: a blue color is produced.

3. Add about 0.3 gm. of resorcinol in solution, and three drops of caustic soda solution, and boil strongly: in the presence of chloroform a yellowish-red color is produced, and the liquid exhibits a beautiful yellow-green fluorescence.

4. Disconnect the condenser from the distilling flask (see Fig. 2, p. 128) and substitute for the former a piece of glass tubing having a horizontal section which is heated with a burner, and a vertical limb dipping into a solution of silver nitrate. If the distilled vapors contain chloroform it is decomposed in the heated tube with formation of hydrochloric acid, which produces in the silver nitrate solution a white precipitate of silver chlorid, insoluble in nitric acid, soluble in ammonia. If the material distilled contain chlorids these may be decomposed by a free acid; but if this be the case the white precipitate in the silver solution will be formed even when the vapor is passed through an unheated tube.

CHLORAL.

Pure chloral, CHO,CCl_3, is a colorless, oily, pungent, and highly irritating liquid. This on contact with water produces a solid crystalline compound, $CHOCCl_3,H_2O$, which is *chloral hydrate*, miscalled *chloral* in the United States Pharmacopœia.

Although the use of chloral as a medicine was first sug-

[1] Amer. Chem. Jur., 1886, viii., 358.

gested by Liebreich in 1869, a fatal poisoning by its administration occurred in 1870, and since that time similar cases have been numerous. We have collated the reports of 93 cases of poisoning by chloral hydrate, of which 70 were accidental, 22 suicidal, and 1 homicidal. The earlier accidental poisonings were chiefly medicinal; the more recent due to the taking of overdoses by persons addicted to the chloral habit. Those attempting suicide by chloral have for the most part belonged to the same class; and the attempts have been rarely successful—only five of the twenty-two. The only homicidal case was an early instance of the use of "knock-out drops." A man was found dead in a cab with all of his valuables missing, after having been drugged by a person who had accompanied him, and against whom two other robberies similarly perpetrated, but without fatal effects, were proven.[1]

Lethal Dose.—The effects of a given dose of chloral hydrate vary greatly with different individuals and in the same individuals at different times. Death has in a few instances followed the administration of doses notably less than those which are given medicinally with safety and benefit in certain conditions of disease. Ashbough[2] has reported the death of a woman in fifteen minutes after the last of two doses of 10 grains each (1.3 gm. in all); Fordyce[3] the death of a man suffering with delirium tremens in one and one-half hours after the last of three doses of 15 grains each (2.9 gm. in all) given during seven hours; von Maschka[4] the death of a healthy woman from the effects of one drachm (3.9 gm.) taken at one dose; Ludlow[5] the death of a girl of eighteen years from the effects of two injections containing 40 grains each (5.2 gm. in all), given in puerperal eclampsia; and Stappen and Poirier[6] the death of a girl of four years from the effects of 4 gm. (61 grains).

But very much larger doses have been taken without fatal result. Recoveries have been reported by Stone[7] from 430 grains

[1] Reynolds: Brit. M. J., 1889, ii., 235 (the Manchester cab mystery. Reg. *v.* Parton).

[2] Chicago Med. J., 1877, xxxiv., 234.

[3] Wkly. Med. Rev., Chicago, 1883, viii., 94.

[4] Wien. med. Wchnschr., 1871, xxi., 1163.

[5] M. and S. Reptr., Phila., 1888, lviii., 596.

[6] Ann. Soc. de méd. de Gand, 1876, liv., 85.

[7] Louisville Med. News, 1883, xv., 179.

(28 gm.); by Eschelman[1] from 460 grains (30 gm.); by Springthorpe,[2] Hutchinson,[3] and Madigan[4] from one ounce (31.2 grains); by Anstie[5] from "more than an ounce;" and by Williams[6] from about 600 grains (39 gm.). In Williams' case there was no treatment whatever during the period of coma.

Duration.—The average duration of a fatal chloral poisoning is about ten or twelve hours. Dandridge has reported the death of a man in two and one-half hours after having taken a single dose of 160 grains (10.4 gm.);[7] and Carroll a death in three and one-quarter hours from the effect of a single dose of 480 grains (31.1 gm.).[8] Death has followed more promptly after the last of two or more doses; as in Ashbough's case of death in fifteen minutes,[9] and in Fordyce's case of death in one and one-half hours.[10]

On the other hand, cases have been observed in which life has been prolonged for forty,[11] forty-three,[12] and forty-eight[13] hours, and even for three days after medicinal administration of repeated doses.[14]

Symptoms.—In most cases there is immediate burning pain in the mouth, throat, and stomach, not so intense as when liquid chloroform is swallowed, and not so marked when the poison is swallowed in a mucilaginous liquid. In some instances there have been vertigo, severe dyspnœa, convulsions, and delirium. But usually, after a short period of excitement, the patient sinks into a deep sleep, which in some instances has been quiet and natural, and has continued, with occasional awaking, for as long as seventy-two hours in non-fatal cases. In severe cases the sleep deepens into coma, with stertorous respiration, contracted pupils, cold surface, muscular relaxation, the face pale and afterward cyanosed, failing pulse and respiration, and death from coma or pulmonary œdema. Although the temperature is usually subnormal, this is not always the case. Welch

[1] Med. Times, Phila., 1870, i., 23.
[2] Austral. M. J., 1887, n. s., ix., 499.
[3] Lancet, 1879, ii., 487.
[4] Chicago Med. Rev., 1881, iv., 447.
[5] Practitioner, Lond., 1874, xii., 104.
[6] Baltimore M. J. and Bull., 1871, ii., 84.
[7] Cincin. Lanc. and Obs., 1878, xxi., 385.
[8] Phila. M. Times, 1878–79, ix., 82.
[9] *Loc. cit.*
[10] *Loc. cit.*
[11] Plummer: Lancet, 1894, i., 21.
[12] Craig: Amer. Practit., 1879, xx., 366.
[13] Ludlow: M. and S. Reptr., Phila., 1888, lviii., 596.
[14] Needham: J. Psychol. Med., 1871, v., 93.

reports a non-fatal poisoning from 360 grains (23.4 gm.) in which there was never any fall of temperature; in Plummer's[1] case of suicidal poisoning by about an ounce (31 gm.) of the crystals, fatal in forty hours, the temperature was 37°.8 (100° F.) in seven hours, and rose from that to 40°.3 (104°.6 F.) on the second day. Chouppe[2] also reported a non-fatal case in which the temperature (rectal), starting at 36°.4 (97°.5 F.) fell during an hour to 30°.2 (86.°4 F.), but during the following two hours rose to 37°.4 (99°.3 F.). The skin in many cases is covered with a scarlatinal rash. In some instances the patient before going to sleep is in a peculiar psychic condition; while half-awake he may perform numerous voluntary acts of which he afterward has no remembrance.

Treatment.—The stomach should be promptly washed out. The body temperature should be maintained by external warmth. Hypodermics of ammonia or strychnin are indicated to support the heart, and artificial respiration, with the use of pure oxygen, may be necessary.

Post-Mortem Appearances.—The mucous membrane of the mouth, œsophagus, and stomach are reddened and inflamed, and there may even be ecchymosis and destruction of tissue in the stomach. There is marked engorgement of the vessels of the brain, cord, and lungs.

Detection.—The analysis is conducted in the same manner as that for chloroform (see p. 853). After the distillate from the acid materials has been tested for chloroform the contents of the distilling flask are rendered alkaline and the process is repeated. If chloral be present it is decomposed by the alkali with formation of chloroform, whose presence is indicated by the tests described.

Chloral is possessed of distinct antiseptic powers, and consequently would probably be detectable in the cadaver for a long period after death.

PHENOL—CARBOLIC ACID.

Pure phenol ($C_6H_5.OH$) crystallizes in long, colorless, prismatic needles, fusible at 40°–41° (104°–105°.8 F.), boiling at

[1] *Loc. cit.*

[2] C. rend. Soc. de biol., 1875, 6 s., ii., 45.

181°.5 (258°.7 F.). It has a peculiar, well-known odor, and an acrid, burning taste. It is very sparingly soluble in water, readily soluble in alcohol and in ether; sp. gr. 1.065; neutral in reaction. It absorbs water from the air to form a hydrate, which crystallizes in six-sided prisms, fusible at 16° (60°.8 F.). It is met with commercially as "crude carbolic acid," a reddish liquid, used as a disinfectant, as pure phenol, and as "ninety-five per cent. acid," the hydrate above referred to.

Accidental and suicidal poisonings by carbolic acid are of very frequent occurrence; but the caustic taste and well-known odor of phenol render its secret administration practically impossible. We find, however, reference to two homicidal poisonings of young children by carbolic acid in medical literature: an Indian case in 1882,[1] and a German case in 1896;[2] and in the secular press reference to the poisoning of three soldiers in Naples in 1887, to whom the acid, which they were using as a disinfectant, was forcibly administered by a mob.

Lethal Dose.—The smallest quantity of phenol which has been known to cause death was 1.5 gm. (23 grains) taken with suicidal intent by a woman, who was found dead.[3] One drachm (3.9 gm.) of the concentrated (liquid) acid has proved fatal in two instances.[4] On the other hand, recoveries from the effects of an ounce have been numerous, and Taliaferro[5] reports the recovery of a girl of nineteen years, who aborted, but recovered after having swallowed 1½ ounces (46.7 gm.) of 95 per cent. acid.

Duration.—In the majority of fatal cases death occurs in from two to eight hours, but in the most acute cases the duration does not exceed an hour or two. Marwood[6] reports a death in ten minutes, Friedberg[7] one in twelve minutes, Sigler[8] one in nineteen minutes, and Hagenbach[9] one in less than twenty minutes. Or life may be prolonged for several days. Thus

[1] Empr. *v.* Upendo Kisto Dutt: Ind. M. Gaz., 1883, xviii., 130.

[2] Coester: Vrtljschr. f. ger. Med., 3 F., xi., 303.

[3] De Vries: Allg. med. Centr. Ztg., 1890, lix., 1701.

[4] Wilkinson: Therap. Gaz., Detroit 1892, 3 s., viii., 220. Edmunds: M. and S. Reptr., Phila., 1887, lvii., 345.

[5] Wkly. M. Rev., Chicago, 1883, viii., 55.

[6] Austral. M. Gaz., 1893, xii., 78.

[7] Arch. f. path. Anat., etc., 1881, lxxxiii., 132.

[8] Louisville Med. News, 1880, x., 294.

[9] Chicago M. J. and Exam., 1882, xliv., 278.

Langerhans reports a death in four days,[1] Bertag[2] one in five days, Schleicher[3] one in six days, and De Mahy[4] one on the seventh day. Fatal poisonings of still longer duration have followed the absorption of carbolic acid by channels other than the alimentary canal.

Symptoms.—Carbolic acid is both a corrosive and a poison. The symptoms referable to its local action, of course, begin immediately, and those of true poisoning are not delayed. The patient may be in complete collapse in from three to five minutes. Dilute solutions produce a burning sensation, while the concentrated acid causes intense pain in and cauterization of all parts which it touches. The skin, lips, etc., are marked with white stains, which subsequently turn red, and on separation of the eschar a persistent dark stain remains. Vomiting occurs in some cases, and the vomited matters, as well as the expired air, may or may not have the odor of phenol. The patient, in severe cases, soon becomes unconscious and may die in collapse, or become comatose, with stertorous respiration, at first slow, afterward accelerated, contracted pupils, cold surface, and cyanosis. The urine is of a dark smoky or olive-green color, either when voided, or becoming so in a short time after exposure to air. In some cases there is nephritis or hæmoglobinuria.

Treatment.—The stomach should be washed out. Saccharated lime and alkaline sulfates serve to form insoluble compounds with the poison which may remain. The tendency to collapse may be met by injections of ether.

Post-Mortem Appearances.—The stains upon the skin and the mucous membrane of the mouth and pharynx are dirty white, or, if the case has been one of long duration, red. The mucous membrane of the stomach and intestines is reddened and inflamed.[5] The odor of phenol may be observed on opening the stomach. The brain, lungs, liver, and kidneys are frequently congested.

Detection.—In a systematic analysis phenol may be en-

[1] Deut. med. Wchnschr., 1893, xix., 269.

[2] Berl. kl. Wchnschr., 1883, xx., 415.

[3] Deut. med. Wchnschr., 1891, xvii., 9.

[4] N. Orl. M. and S. J., 1887–88, n. s., xv., 111.

[5] For colored plate see Lesser, "Atl. d. ger. Med.," Taf. i., fig. 1, also Taf. ii., fig. 4.

countered either in the distillate from the acid liquid in the search for volatile poisons (see p. 127), or in the residue of evaporation of the petroleum ether or benzene from acid aqueous solution, in the search for vegetable poisons (see pp. 134, 135).

Tests for Phenol.—1. The peculiar odor.

2. Mix with one-quarter volume of ammonium hydroxid solution; add two drops of sodium hypochlorite solution and warm: a blue or green color. Add hydrochloric acid to acid reaction: turns red.

3. Add two drops of the liquid to a little hydrochloric acid, and then one drop of nitric acid: a purple-red color.

4. Boil with nitric acid so long as red fumes are given off. Neutralize with caustic potash: a yellow, crystalline precipitate.

5. With solution of ferrous sulfate, a lilac color.

6. Float the liquid on sulfuric acid, add powdered potassium nitrate: a violet color.

7. Add excess of bromin-water: a yellowish-white precipitate.

TABLE OF CASES REFERRED TO IN THIS VOLUME.

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