THE LIBRARY OF THE UNIVERSITY OF CALIFORNIA LOS ANGELES
TEXT-BOOK

OF

MEDICAL JURISPRUDEENCE

AND

TOXICOLOGY.

By JOHN J. REESE, M.D.,

PROFESSOR OF MEDICAL JURISPRUDEENCE AND TOXICOLOGY IN THE UNIVERSITY OF PENNSYLVANIA; VICE-PRESIDENT OF THE MEDICAL JURISPRUDEENCE SOCIETY OF PHILADELPHIA; PHYSICIAN TO ST. JOSEPH'S HOSPITAL; MEMBER OF THE COLLEGE OF PHYSICIANS OF PHILADELPHIA; CORRESPONDING MEMBER OF THE NEW YORK MEDICO-LEGAL SOCIETY.

PHILADELPHIA:
P. BLAKISTON, SON & CO.,
1012 WALNUT STREET,
1884.
Entered, according to Act of Congress, in the year 1884, by

P. BLAKISTON, SON & CO.,

in the Office of the Librarian of Congress, at Washington, D. C.
PREFACE.

This Text-book has been written more particularly to meet the wants of students of Legal Medicine. The author is aware that the field has already been occupied by able and popular treatises on medical jurisprudence, well known to the professions of medicine and law; but an experience of over twenty years, as a public teacher of this branch of science, has convinced him that students in both these professions, who desire to acquire a knowledge of medical jurisprudence, are too often deterred from their purpose by being confronted by the ponderous works of recognized masters, extending to three, and even six large octavo volumes.

To avoid the above objection, the author of the present work has endeavored to condense in a handy volume all the essentials of the science, and to present the various topics in a simple and familiar style, giving greater prominence, of course, to those of the greatest practical importance.

The subject of Toxicology occupies, as was proper, a considerable space, and has been carefully prepared; special attention being bestowed upon the most important poisons—such as Arsenic, Strychnine, Opium, Prussic Acid, etc.

The chapter on Insanity is as full as the size of the volume would justify, and will be found, it is hoped, to
contain all the essential medico-legal points pertaining to this subject.

The author has not hesitated to avail himself freely of the materials so abundantly presented in the elaborate and classic works of Casper, Taylor, Beck, Wharton and Stillé, Tidy, Guy, Tardieu, and others; always desiring to give due credit to the authority quoted, and usually doing so at the time; and he would embrace this opportunity to express his obligations to these authorities. He is sincerely desirous to encourage an increasing interest in the students of both medicine and law for that most important, but too-much neglected, subject—Forensic Medicine; and he would indulge the hope that the present treatise, in its unpretentious size and style, may aid in so doing.

*Philadelphia, September, 1884.*
# TABLE OF CONTENTS.

## CHAPTER I.

### Introduction.

Definition.—Importance to Students of Medicine and Law.—Order of Proceeding in a Medico-legal Case.—The Coroner's Inquest.—The Criminal Court.—Medical Evidence.—Medical Experts.—Dying Declarations.  .......................................................... 9

## CHAPTER II.

### Phenomena and Signs of Death.

**Section I.**—Molecular and Somatic Death.—The Immediate Cause of Death to be found in either the Heart, Lungs or Brain.—Post-mortem Evidences of these.—The "Signs of Death."—Cessation of Respiration and Circulation.—Changes in the Eyes.—Pallor of the Body. .................................................. 25

**Section II.**—Signs of Death continued.—Loss of Animal Heat.—Post-mortem Caloricity.—Rigor Mortis.—Post-mortem Lividity, or Suggillation 32

**Section III.**—Signs of Death continued.—Putrefaction.—External and Internal Signs.—Adipocere.—Mummification.—How long since the Death? ........................................................................................................ 41

## CHAPTER III.

### Medico-legal Investigations.—The Post-mortem.

Responsibility involved.—Accuracy and Method necessary.—Examination of the surroundings.—External examination of the Body.—Internal examination.—Details of the Examination.—Measurements and Weights.—Notes. ........................................................................................................ 56

## CHAPTER IV.

### Presumption of Death and of Survivorship.

Cases involving Presumption of Death and of Survivorship.—French Law of Survivorship.—Probabilities afforded by Age, Sex, and Mode of Death. 63

## CHAPTER V.

### Personal Identity.

**Section I.**—Important Medico-legal bearings.—I. Identification of the Living.—Personal Appearance.—Peculiar bodily marks.—Remarkable Cases. ........................................................................................................ 69
TABLE OF CONTENTS.

SECTION II.—II. Identification of the Dead.—Mutilated Remains.—Identification by means of the Skeleton, as to Age, Sex, and Stature.—Rules of Proportion.—Fractures, Deformities, and Callus.—Age of Bones.—Examination of Hairs and Fibres.

CHAPTER VI.
The Causes Producing Violent Death.

SECTION I.—Death from Wounds.—Definition of a Wound.—Danger of.—Examination of the Body.—Absence of external marks of violence.—Wounds made before and after death.—Hemorrhage.—Ecchymoses.—Classification.—Homicidal, Suicidal and Accidental Wounds.

SECTION II.—Gunshot Wounds.—Differ from other wounds.—Deflection of the Ball.—Wounds made by Shot, Wadding and Powder.—Wounds of the Head, Neck, Spine, Chest and Abdomen.

CHAPTER VII.
Examination of Blood Stains.

Importance of their identification.—Three methods of identifying.—1. The Chemical Tests.—2. The Microscopic Test.—3. The Spectroscopic Test.—Blood Crystals.

CHAPTER VIII.
Burns and Scalds.

Definition.—Classification.—Symptoms.—Cause of Death.—Post-mortem appearances.—Burns made before and after death.—Accidental, Suicidal and Homicidal Burns.—Spontaneous Combustion.

CHAPTER IX.
Death from Different Forms of Asphyxia.

SECTION I.—Death from Suffocation.—Accidental, Suicidal and Homicidal Suffocation.—Post-mortem signs.

SECTION II.—Death by Strangulation.—Modes of Strangulation.—External signs.—Internal lesions.—Accidental, Suicidal and Homicidal Strangulation.—Mark of the Cord.

SECTION III.—Death by Hanging.—Cause of Death.—Post-mortem appearances.—Cord marks.—Generally suicidal.

SECTION IV.—Death by Drowning.—Mode of Death.—Time required.—Signs of Death, external and internal.—Accidental, Suicidal and Homicidal Drowning.

CHAPTER X.
Death by Lightning.

Medico-legal relations.—Mode of Death.—Post-mortem signs.
TABLE OF CONTENTS.

CHAPTER XI.

Death from Heat and Cold.
Diverse effects of Heat upon the body.—Post mortem appearances.—Effects of Cold.—Post mortem signs, . . . . . . . 170

CHAPTER XII.

Death by Starvation.
Accidental, Homicidal and Suicidal Starvation.— Pretended cases of voluntary starvation.— Symptoms, and Post-mortem signs.— Medico-legal relations, . . . . . . . . . . . . . 175

CHAPTER XIII.

Death from Poisoning.—Toxicology.

SECTION I.— Definition of a Poison.— Effects.— Proofs of Absorption.—Subsequent disposition of the Poison.— Elimination.— Cause of Death.—Circumstances modifying their action.—Antagonism of Poisons, . . 178

SECTION II.— Evidences of Poisoning.— 1. Evidences from Symptoms.— 2. From post-mortem lesions.— 3. Chemical Analysis.—Post-mortem Imbibition of Poisons.—Rules in performing a toxicological analysis.— 4. Physiological experiments.— 5. Circumstantial Evidence.— Medico-legal conclusions.— Classification, . . . . . . . . . . . . . 186

CHAPTER XIV.

Poisoning by the Mineral Acids.
Certain common symptoms.— Post-mortem appearances.— Treatment.— Chemical analysis.— Toxicological examination for the different Acids, 202

CHAPTER XV.

Poisoning by the Alkalies and their Salts.

SECTION I.— Potassa, Soda, and Ammonia.— Similarity of effects.— Symptoms.— Fatal Dose.— Treatment.— Post-mortem appearances.— Chemical analysis, . . . . . . . . . . . . . 215

SECTION II.— Poisoning by the Alkaline and Earthy Salts.— Nitrate of Potassium.— Bitartrate of Potassium.— Sulphate of Potassium.— Alum.— Chlorinated Potassium and Sodium.— Salts of Barium, . . . . . 220

CHAPTER XVI.

Irritants Possessing Remote Specific Properties.

SECTION I.— Poisoning by Phosphorus.— Symptoms.— Fatal Dose.— Treatment.— Morbid appearances.— Diagnosis.— Chemical analysis.— Amorphous Phosphorus, . . . . . . . . . . . . . 224

SECTION II.— Poisoning by Iodine, Bromine, and Chlorine, . . . . . 234
TABLE OF CONTENTS.

CHAPTER XVII.
Poisoning by Arsenic.
Metabolic Arsenic.—Arsenious Acid.—Properties.—Symptoms.—Chronic Poisoning.—Fatal Dose.—Treatment.—Post-mortem signs.—Chemical analysis.—Toxicological examination.—Other Preparations of Arsenic. 238

CHAPTER XVIII.
Poisoning by Antimony (Tartar Emetic).
Properties of Tartar Emetic.—Symptoms.—Fatal Dose.—Post-mortem appearances.—Slow Poisoning.—Chemical analysis.—Toxicological examination. 262

CHAPTER XIX.
Poisoning by Mercury (Corrosive Sublimate).
Corrosive Sublimate.—Properties.—Symptoms.—Fatal Dose.—Antidotes.—Chemical analysis.—Toxicological examination.—Salivation. 273

CHAPTER XX.
Poisoning by Lead.
Frequency of Chronic Poisoning.—Acetate of Lead.—Symptoms.—Treatment.—Post-mortem appearances.—Painters’ Colic.—Lead Palsy.—Toxicological examination. 285

CHAPTER XXI.
Poisoning by Copper.
Accidental Poisoning.—Salts of Copper.—Fatal Dose.—Treatment.—Morbid appearances.—Chemical analysis.—Toxicological examination. 294

CHAPTER XXII.
Poisoning by Zinc, Bismuth, Tin, Iron and Chromium.
Section I.—Poisoning by Zinc. 301
Section II.—Poisoning by Bismuth. 305
Section III.—Poisoning by Tin, Iron and Chromium. 306

CHAPTER XXIII.
Vegetable and Animal Irritants.
Section I.—Poisoning by Oxalic Acid.—Sources of the Acid in Nature.—Symptoms.—Fatal Dose.—Treatment.—Morbid appearances.—Chemical analysis.—Toxicological examination.—Binoxalate of Potassium. 308
Section II.—Poisoning by Carbolic Acid. 315
TABLE OF CONTENTS.

SECTION III.—Poisoning by Croton Oil, Elaterium, Castor Oil Beans, Colchicum and Savin. 317
SECTION IV.—Poisoning by the Hellebores.—Veratria.—Yellow Jessamine. —Poisonous Mushrooms. 320

CHAPTER XXIV.
Animal Irritants.
Poisoning by Cantharides.—Poisonous Animal Food.—Sausage Poison.—Trichinosis.—Cheese Poison.—Poisonous Fish.—Putrescent Food.—Poisoned Flesh. 325

CHAPTER XXV.
Class II. Neurotic Poisons.
SECTION I.—Narcotics.—Poisoning by Opium.—Nature of Opium.—Poisonous Symptoms.—Fatal Period and Dose.—Treatment.—Morphia.—Properties.—Tests.—Meconic Acid.—Tests.—Toxicological examination. 330
SECTION II.—Poisoning by Alcohol.—Acute Alcoholism.—Symptoms.—Post-mortem lesions.—Chemical analysis.—Detection in the Tissues. 341

CHAPTER XXVI.
2. Anæsthetics.
Poisoning by Ether, Chloroform, and Chloral Hydrate. 345

CHAPTER XXVII.
Spinal Neurotics.—Poisoning by Nux Vomica.—Strychnia.
Nux Vomica.—Strychnia.—Effects.—Fatal Dose.—Treatment.—Post-mortem lesions.—Diagnosis.—Chemical analysis.—Interferences.—Physiological Test.—Toxicological examination. 351

CHAPTER XXVIII.
Cerebro-spinal Neurotics.—(1) Deliriants.
SECTION I.—Poisoning by Belladonna.—Symptoms.—Atropia.—Fatal Dose, —Treatment.—Post-mortem appearances.—Chemical analysis.—Toxicological examination. 373
SECTION II.—Poisoning by Stramonium, Hyoscyamus and Solanum. 376

CHAPTER XXIX.
2. Depressants.
SECTION I.—Poisoning by Tobacco and Lobelia.—Effects of Tobacco.—Post-mortem lesions.—Nicotina.—Properties.—Chemical reactions.—Toxicological examination.—Lobelia. 380
SECTION II.—Poisoning by Hemlock—Conia.—Symptoms.—Post-mortem appearances.—Conia.—Chemical reactions.—Toxicological examination.—Other poisonous Hemlocks................................................. 386

SECTION III.—Poisoning by Aconite and Calabar Bean.—Properties of the Plant.—Effects.—Post-mortem appearances.—Aconitine.—Fatal Dose.—Treatment.—Chemical analysis.—Toxicological examination.—Calabar Bean.—Eserine................................................................. 390

CHAPTER XXX.
(3) Asthenics.

SECTION I.—Poisoning by Hydrocyanic Acid.—Natural Occurrence in Vegetables.—Pure and Official Acids.—Symptoms.—Fatal Period and Dose.—Treatment.—Post-mortem appearances.—Tests.—Toxicological examination.—Cyanide of Potassium.—Oil of Bitter Almonds.—Cherry-laural Water.—Nitro-Benzole........................................... 397

SECTION II.—Poisoning by Digitalis.—Symptoms.—Post-mortem lesions.—Fatal Dose.—Digitaline.—Chemical analysis.—Toxicological examination.—Case of De la Pomerais.—Poisoning by Cocculus Indicus.......................... 409

CHAPTER XXXI.
The Alkaloids of Putrefaction.—Ptomaines.
TEXT-BOOK
OF
MEDICAL JURISPRUDENCE
AND
TOXICOLOGY.

CHAPTER I.

DEFINITION.—IMPORTANCE TO STUDENTS OF MEDICINE AND LAW.—ORDER OF PROCEEDING IN A MEDICO-LEGAL CASE.—THE CORONER'S INQUEST.—THE CRIMINAL COURT.—MEDICAL EVIDENCE.—MEDICAL EXPERTS.—DYING DECLARATIONS.

Medical Jurisprudence, or Legal or Forensic Medicine, as it is sometimes named, may be defined to be the science which applies the knowledge of Medicine to the requirements of Law. To aid in the discovery of truth, which is the great purpose of the Law, every department of human knowledge should be made to contribute, and Medicine among the rest. When certain cases are presented for legal decision, affecting the life or property of an individual, and these cases require for their proper elucidation an appeal to medical knowledge, they are termed medico-legal cases, and the science on which they are based is named Medical Jurisprudence.

It should require no argument to show the importance of the knowledge of this science to the student and practitioner of both the professions of medicine and law. The former cannot entirely evade its claims, although he may
seek to do so, since the very nature of his profession, together with his assumed public position as an accredited physician, renders him liable at any moment to be confronted with a case involving the intricate questions of homicidal, suicidal, or accidental death; of infanticide; of criminal abortion; of rape; of drowning, and of numerous other similar cases, all of which, when they become the subjects of legal investigation, must necessarily depend for their proper elucidation, chiefly, if not solely, upon the physician who had previously given his professional attendance in the case, and who must subsequently give his evidence before the court and jury. How important, then, that the practitioner of medicine, even though he may have no special leaning towards legal medicine, should become acquainted at least with the general principles and leading facts of this science. A proper regard, both for his own professional reputation and the general interests of the community, should constrain him not to neglect so important and practical a branch of knowledge. And as regards the profession of the law, it is difficult to understand how a criminal cause of any kind can be satisfactorily conducted without some knowledge of medical jurisprudence, inasmuch as criminal law is indissolubly connected with legal medicine. It is much to be regretted that many of the present generation of lawyers in this country, graduate in their profession without the slightest knowledge of medical jurisprudence. Indeed, strange as it may appear, it is entirely ignored in some of our law schools, whilst in others, although professedly taught, it is not made obligatory upon the student, and therefore is virtually disregarded.

The consequence of such neglect must be either, that criminal law is entirely abandoned by many distinguished practitioners, causing such cases to fall into the hands of
inferior persons, who are not always the best qualified to conduct them; or else, when a really important criminal case is undertaken, the lawyer finds himself compelled to cram for the occasion, and is subjected to no little annoyance, and loss of time, in preparing for the impending trial. In truth, in such cases he is necessarily obliged to depend almost exclusively upon his medical witnesses, both as to the general mode of developing his case, and especially as to the manner of questioning the expert witnesses on either side.

The superficial observer is often surprised at the intimate relationship subsisting between the two great sciences of medicine and law. As has just been shown, numerous cases brought before a court and jury can only be settled by an appeal to medical knowledge; sometimes it is to one department of medicine, and sometimes to another; and it not infrequently happens that several branches of medical science may be simultaneously called into requisition, in order to aid the law in arriving at a proper decision. For example,—is the case one of suspected homicide, where a dead body has been discovered under suspicious circumstances? Who but the skillful anatomist and pathologist, by a carefully conducted autopsy, can shed the requisite light upon it? Is it a case of alleged rape, or criminal abortion, or infanticide? Who so well qualified to conduct the investigation as the well trained obstetrician? Or is it a case involving the dark suspicion of criminal poisoning? Who shall impart the necessary information upon which may hang suspended the life or death of the accused, save he who is thoroughly acquainted with the mysteries of toxicology? Thus it may happen that, in a multitude of cases, the well instructed practitioner of medicine, when called upon to act the part of the legal physician, may be obliged to bring into requisition all the various departments of his science.
It is, perhaps, as a medical witness, that medico-legal knowledge is of the most important service to the physician. After he has accomplished the investigation of the case before him by a carefully conducted autopsy, or by a critical toxicological examination, with perhaps the aid of microcopy, and by other methods of scientific research, there yet remains the all-essential duty of giving the results of his investigations to the court and jury, in the form of evidence; and to be prepared to do this in the proper manner, constitutes the chief acquirement of the medical jurist.

The Coroner's Inquest.—The first public duty imposed upon the legal physician is to testify before the Coroner's Inquest. In all civilized countries a special officer, named the coroner, is appointed to investigate the unknown, or unexplained causes of sudden death, whenever this occurs under suspicious circumstances. For example, a dead body is discovered on the highway or in some sequestered spot, with, or without marks of external violence, or it may have been dragged out of the river; the body of a new-born child has been found in a well or cesspool, or discovered floating in the water; a stranger is discovered, in the morning, dead in bed, at some hotel, far distant from his home; or a person in apparent sound health suddenly drops dead in the street, or in his room. In all these, and analogous cases, the law very wisely provides that a careful investigation shall be instituted, so as to ascertain whether the death was due to natural causes, or whether it was the result of violence. If the latter, then the nature of this violence, e.g. whether from a wound (gunshot or otherwise), a bludgeon, an axe, hammer, or other blunt weapon; or whether occasioned by a fall. In the absence of all external marks of violence,—then, might the death not have been pro-
duced by poison? In each one of these cases, the farther question must be solved—was the death homicidal, suicidal, or accidental?

The coroner's jury usually consists of six men (the number is not definitely fixed in this country), who are mostly selected from the neighborhood where the inquest is held. Their duty consists in (1) viewing the body and establishing its identity, and (2) in holding the inquest, which is a sort of petty court, wherein inquiry is made as to the cause of death, and (in a case of homicide) to ascertain, if possible, the guilty culprit. For this purpose, witnesses duly subpoenaed and sworn are examined, and the medical man who has performed the post-mortem examination (whether he be the coroner's recognized physician, or some other who has been specially appointed for this purpose) now makes his detailed report, and gives his opinion as to the real cause of death. The jury having heard the evidence, and consulted together, bring in their verdict, which is usually in accordance with the report of the medical officer. In some cases, particularly in cities, where the coroner has one or more specially appointed physicians, it is not considered necessary that the jury should personally view the body before holding the inquest; it being regarded as sufficient that the body has been properly identified by the examining physician, and the autopsy carefully made, and the result duly reported.

It will be observed that the special duty of the coroner's inquest is to discover the real cause of death; usually it does not fall within his province to discover the person who caused it. Nevertheless, it does sometimes happen that in the course of the investigation, suspicion may so strongly point to some particular individual, as to warrant the coroner to commit him to prison, to await further investiga-
tion. The usual verdict in cases of violent death (shown to have been neither suicidal nor accidental) is, that of murder, or manslaughter, against some person, known or unknown.

As the post-mortem examination is an inseparable part of the coroner's inquiry, and, in fact, constitutes its most important factor, it is indispensable that it should be performed in the most careful and thorough manner; and no one is fit to undertake it but a skilled anatomist and pathologist. The medical man should never permit himself to be hurried in this work; he should allow himself ample time, and always perform the autopsy by daylight, if possible, inasmuch as he might fail to distinguish certain alterations of color by artificial light. When it is remembered that the most serious issues may be at stake—even those of life and death—it will be acknowledged that the utmost caution should be exercised in conducting the autopsy. There may be cases, involving strong suspicion of murder, where it might be deemed advisable that the post-mortem examination should be performed by two independent experts, in order to avoid the imputation of ex parte influence. One of these medical examiners should represent the State, and the other the accused.

In giving his evidence before the coroner's jury the medical man should not undervalue the situation. Although the inquest be held in some remote hovel or barn, and the jury be composed of rude, illiterate persons, he should not fail to remember that his testimony is all taken down by the proper officer, and that it will surely confront him at the approaching trial. This fact should alone be sufficient to impress upon him the seriousness of the occasion, and remind him of the importance of drawing up his report with due care and accuracy.
The Criminal Court.—After the coroner's inquest the case (if a criminal one) is sent to the Grand Jury, who institute a preliminary examination, and either ignore it altogether, or else find a true bill. In the latter event, the case next comes before the judge and petty jury, for trial. To this trial the medical witness is summoned by a subpoena, which he cannot evade, but which it is his duty to obey. Before this court and jury, he will undergo a most strict and impartial examination as to his opinion of the cause of death, and the reasons on which this opinion is founded. He will be questioned most closely, in the cross-examination, as to his professional knowledge and acquirements, the extent of his opportunities for making such investigations as the one now pending, the accuracy of his post-mortem, or toxicological examination, the modes of distinguishing between wounds made before and after death, the method of discriminating between the effects of poisons and of disease, the danger of confounding these latter together, the liability to mistake in chemical results, and a hundred other matters, which will annoy and confuse the medical witness to no small degree, unless he be prepared beforehand, by his thorough medico-legal knowledge and training. A medical witness so properly fortified, need have no fear for himself; for, as he goes upon the stand honestly, to testify to the truth, "he need only," in the language of the late Professor Taylor, "bear in mind two considerations: first, that he should be thoroughly prepared on all points of the subject on which he is to give evidence; and secondly, that his demeanor should be that of an educated gentleman, and suited to the serious occasion on which he appears."

In the matter of Medical Evidence there are several points that require a brief notice here. After opening the case before the court, the prosecuting attorney, or some one
associated with him in the prosecution, calls the witnesses and examines them "according to the rules of evidence;" this is technically called the examination in chief. The "rules of evidence" prohibit counsel, in the examination in chief, from putting leading questions to the witness, i.e., questions that suggest their own answers; and for the reason that the witness may be supposed to be willing to say anything favorable suggested to him by his own counsel, and to repress anything unfavorable. The replies of the witness, however, should always be given with equal clearness and precision, to both the counsel for the defence, and for the prosecution.

The cross-examination next follows: this is conducted by the counsel for the prisoner, and is especially aimed at contradicting and overthrowing, if possible, the witness's previous testimony. To this end, the counsel plies the witness with questions which are strongly "leading," and such as may most strongly suggest any facts or circumstances which he had previously withheld, and which may appear favorable to his client. A counsel for the defence is allowed very considerable latitude in the cross-examination of the witness, and the latter should always be well prepared to meet the attack, with conscious strength, and calm and dignified composure. If, however, the advocate transcends, as he is sometimes tempted to do, the bounds of propriety and decorum, the witness has always the right to appeal to the court for protection. It is far preferable to adopt the latter course, than to attempt to argue, or recriminate with counsel, since the latter has the witness always at disadvantage on the stand.

The re-examination sometimes follows the cross-examination of the witness, when it becomes necessary to clear up, or explain any matter that may have been obscured by the cross-examination.
After the examination of the State's witnesses, that of the witnesses for the defence follows. And here the same general line of examination is pursued as in the former case. When the medical witness for the defence is put upon the stand, a most unpleasant exhibition is, not unfrequently, made, of one expert directly contradicting another expert on the opposite side, and both of them medical men of equal standing and worth in the profession, and in the community. Such professional tilting is sometimes sneeringly designated as the "war of the experts," and is certainly deeply to be regretted, as it tends greatly to prejudice both the court and the public against expert testimony in general; and this, of course, to the detriment of justice.

It is proper to clearly understand the difference between an ordinary witness, and an expert witness. The former testifies only to facts which he has seen, or heard, or learned from personal observation. The "expert" or skilled witness (expertus) does not necessarily testify to facts, but gives his opinion on facts observed by himself, or testified to by others. An expert witness is supposed to be specially skilled in the matter on which he is to testify. It is just here, we believe, that the real cause of difficulty lies in medico-legal cases involving expert evidence. The so-called "experts" are usually taken from the ranks of the medical profession. Such persons, because they are doctors, sometimes, unfortunately, imagine that they are therefore qualified to act as experts, without any previous medico-legal knowledge, or training. Of course, there must necessarily be a conflict of opinion where such an improvised witness is opposed to the genuine expert, on the witness stand. We believe that if all the experts were equally skilled, and equally qualified, and equally honest, there rarely could occur any conflict of opinions between the opposite sides, since both are
equally desirous of discovering and testifying to the truth, and truth is always undivided.

No one should presume to assume the position of an expert witness who has not devoted his special attention to the matter under consideration, and who is, therefore, able really to enlighten the court and jury.

The expert witness has his rights as well as his duties and responsibilities. One of these rights is his compensation. It has often been made a subject of just complaint that an expert witness is placed upon the stand, and his professional opinions, which may be of the utmost value in the pending case, extorted from him piecemeal, by the questionings of counsel, and yet he receives as his compensation merely the pittance of one dollar and a half a day—the pay of an ordinary witness! Some high legal authorities contend, very justly, that the expert is not bound to submit to this imposition, and that he is entitled to an adequate fee for his services, for which, however, he should arrange with the party calling him, before he gives his evidence. The English courts, we believe, have not yet definitely settled this matter. We are glad to see that a recent authority* uses the following pointed language concerning this matter: "No witness can be compelled to give his opinion in the witness box. Further, no one is bound to accept a subpoena merely to state opinions. The witness that can speak to any actual fact connected with the case must attend the trial, if required to do so, but the expert, however wide his experience, cannot be forced to give the court the value of his general or special knowledge." There can be no question as to the propriety and justice of this position; but in this country, the practice of the courts in relation to the compensation of medical experts, is by no means settled. In the great majority of

*Tidy's Legal Medicine. Part I. Lond. 1882; p. 17.
our States, the law allows no additional compensation to the expert; and it is not an unfrequent practice to subpoena him as an ordinary witness, and when in the witness box, to use him as an expert. What shall the expert do in such a case? How shall he conduct himself consistently with his own dignity and proper rights? Certainly, the court would not use a man's private property—the work of his hands, his skilled manual labor, or the product of his farm or merchandise, without adequate compensation; why, then, should they exact from him that which is the result of the labor of his brain, than which nothing can be more exclusively and definitely a man's own private property? It is to be regretted that so few of our American courts and legislatures have appeared to recognize the true bearings of this subject, so that with us the old practice still prevails, of affording no legal protection to the medical expert, in the matter of fees.*

In point of fact, however, it rarely happens, in important criminal cases, especially in poison cases, that either the prosecution or the defence would venture to trust their interests to a reluctant witness; and certainly he would be a reluctant witness who had been dragged, perhaps hundreds of miles from his home and business, by a subpoena, which the law forces him to obey, and who, after spending, it may be, days in attendance upon the court, is compelled to give, for the paltry pittance of the wages of a day-laborer, that which has caused him years of labor and study to acquire, in the shape of an opinion, on which may turn the question of life or death to the prisoner! In all such cases, the ordinary practice is to arrange beforehand with the expert for his proper fee; and the witness should be admonished to look carefully about his interests in this matter. Let

*As far as can be ascertained, only two of our States have legislated on this subject.
him remember that the district attorney, who usually directs the affair, has no authority to pay his fee; neither does this authority lie with the court. The only responsible parties in the case are the county commissioners, or some other equivalent county authorities. With these alone ought the expert to make his arrangements, and always previously to undertaking the case; and to these alone can he look, legally, for his fee.

As regards the obligation of a witness to obey a subpœna when he is to be questioned only as to his opinion, we think that, in this country, the mandate of the court is obligatory; the witness's duty is to obey it, and then, if not previously, endeavor to arrange about his compensation, before giving his evidence.

We venture a word of caution to the medical witness. Before undertaking any case, or consenting to act as an "expert" therein, be sure to institute a thorough examination of all the bearings of the case,—its pros and cons. If retained by the defence, the expert has the right to examine the report of the State's expert; and this is of special importance in a poison case. A critical examination of this report will enable him to determine whether he can conscientiously, and on scientific grounds, undertake to contradict and oppose the conclusions arrived at in this report. It is a most serious and responsible position for him to assume, and he ought to assume it only after a deliberate study of the case, and a strict consciousness of his ability to grasp it. If he finds nothing in the report that he cannot thoroughly endorse, whether the results of chemical or other experiments, or whether the deductions from these experiments in the shape of opinions, our advice would be for him frankly to decline the case altogether. He should never go upon the witness stand as a mere partisan, nor offer his
professional shrewdness and tact, for pay, to the highest bidder! If, however, a careful scrutiny of the report has satisfied him that the State has not made out its case (so far as the scientific evidence goes), but that serious blunders have been committed in the chemical and other experiments performed, whereby most erroneous conclusions have been reached, and which may vitally affect the result, then we are of the opinion that the expert not only need not hesitate to take the case, but that it is his bounden duty to do so, in order that he may aid in carrying out the ends of justice.

This whole matter of expert testimony has long engaged the attention of some of the ablest minds in both the professions of law and medicine, but with no very definite results. No doubt, our present system of volunteer medical experts is open to serious objections, which, under our present laws, cannot be remedied. The only true and proper system, as it seems to us, is for each State to appoint one or more experts, who shall be State officers, physicians of thorough education and experience, and training in this particular line, who shall devote their time and attention exclusively to this duty, and for which they shall receive an adequate compensation. Such an office, properly filled, and kept aloof from all political considerations, would, we believe, be of real benefit to the State. It would, to a great extent, if not completely, abolish the unseemly contention of the experts in the court-room, inasmuch as the State expert (whose professional ability and moral standing should be absolutely unquestioned) would be present at every important trial, and give to the court and jury the results of his previous investigations in the case; and, moreover, since he is to be presumed to be entirely impartial, without bias to either the prosecution or defence, his
opinion would be generally received as final by both sides, and thus both contention and expense would be avoided.

The above system of medical expert testimony resembles, in many respects, that of Germany, which we regard as superior to that of other countries.

There are a few practical rules relating to the giving of evidence, which it is well the medical expert should observe. The first of these is, that he should prepare himself thoroughly upon all the points bearing on the case, in which he is called to give evidence. This he should do in order to further the ends of justice, and also to avoid personal censure. He should be accurate as to weights, measures, distances, size, relationship of objects, etc.

Secondly. He should maintain a quiet, dignified and composed demeanor on the stand, not exhibiting any irritability of temper, however much he may feel provoked by the rudeness of the opposing counsel. He should beware of any display of arrogance, or assumption of manner, or of stubbornness, or testiness of behavior, which are sure to make him appear to disadvantage in the court room.

Thirdly. The witness should give his answers in a clear and audible tone, addressing himself rather to the jury than to counsel, since the former are specially interested in his replies; and these replies, together with his explanations, should always be given in the simplest possible language; and they should be free from all ambiguity, otherwise they will require explanation, which is apt rather to weaken the testimony.

Fourthly. He should never be afraid to say "I don't know," if he does not know. Nothing is more dangerous than for a witness to attempt to guess, for fear of being thought ignorant.

Fifthly. He should particularly avoid the use of all
technical expressions and learned formulae, in giving his
description of the results of an autopsy, or of a toxicological
examination, etc. For example, instead of saying that "the
integuments of the cranium were reflected back, so as to
expose the calvarium," he should simply announce that "the
scalp was thrown back, and the skull, exposed;" and instead
of telling the jury, in grandiloquent phraseology, that, in a
case of assault and battery, he had found that the prosecutor
"had received a severe contusion over the lower portion of
the frontal bone, producing extensive ecchymosis around the
eye, together with considerable infiltration of the subjacent
areolar tissue," he should clear up the matter at once, by
telling them, in plain English, that the man had gotten "a
black eye" (Taylor). All such pomposity and pedantry will,
of course, be avoided by every sensible and well-bred witness,
since it is certain to expose him to well-merited ridicule and
contempt.

_Dying Declarations._—By this term is understood such
declarations as are made by a dying person, who, at the time,
believed he was in actual danger of death, and that his
recovery was impossible. Such declarations are received in
evidence without being sworn to. The law presumes that
all such declarations, made at so solemn a crisis as at a
dying moment, must be sincere. They may not, however,
necessarily be true, although sincere, _i. e._, believed in, at the
time, by the deceased. Dr. Taylor quotes an instance of a
dying woman in St. Thomas' Hospital, who accused a man
of assaulting her. He was found guilty and executed. A
year after the execution, the real murderers were discovered,
and his innocence established. These declarations, more-
over, must relate to the actual circumstances of the death,
and to nothing else.
A magistrate, if he can be had, is the proper person to take down the dying man's declarations, the physician in attendance merely giving his opinion as to the hopelessness of the case, and the soundness of the man's mind. In the absence of the magistrate, the medical man is the best person to receive the dying declaration, or confession; and he should content himself by simply writing down the exact words of the dying person, without any interpretation of them by himself. He should then, if possible, make him sign the declaration, after first reading it over to him.
CHAPTER II.
PHENOMENA AND SIGNS OF DEATH.

SECTION I.

MOLECULAR AND SOMATIC DEATH.—THE IMMEDIATE CAUSE OF DEATH, IN ALL CASES, TO BE FOUND IN EITHER THE HEART, LUNGS, OR BRAIN.—CHARACTERISTIC POST-MORTEM EVIDENCES OF THESE VARIETIES OF DEATH.—THE "SIGNS OF DEATH."—CESSATION OF THE CIRCULATION AND RESPIRATION.—CHANGES IN THE EYES.—PALLOR OF THE BODY.

By molecular death is to be understood the incessant disintegration of tissue which is going on in the body during the active processes of life; the waste of material thus produced being compensated by the never-ending work of reparation. In youth, the supply is in excess of the waste, and growth is the result. In advanced age, the reverse is the case. Somatic death is the cessation of all the vital functions of the body, or the death of the whole body. The latter is the popular idea of death; and the time when it takes place is generally recognizable. The precise period when universal molecular death occurs cannot be accurately determined. No doubt, molecular life may continue some time after somatic death, as is evidenced by post-mortem caloricity (vid. post.) and by certain acts of nutrition and secretion, such as the growth of the hair and nails.

Although the outlets of human life are so numerous and varied, and the phenomena attending the dissolution of the body are equally diversified, the immediate or actual cause of death, in every instance, must be referred to an arrest of the function of one, or other of the three great centres of life—the heart, the lungs, and the brain. And so intimately
are the functions of these three "centres" connected together, that when one ceases to act, the actions of the other two are speedily brought to a standstill. Each one of these three varieties has its own special phenomena or signs; and each exhibits its own peculiar or characteristic post-mortem appearances. We adopt Bichat's classification of (1) death beginning at the brain, (2) death beginning at the heart, and (3) death beginning at the lungs.

I. Death Beginning at the Brain—Coma.—Symptoms.—Stupor, more or less profound; insensibility to external impressions; loss of consciousness; breathing slow, stertorous, and irregular; respiration gradually ceases, as the medulla oblongata begins to be affected. The chest ceases to expand; the blood is no longer aerated; the pulmonary circulation is arrested; the lungs cease to act, and finally the heart's pulsations are brought to a stop.

Post-mortem appearances.—1. Effusion of blood, or serum in the brain or cavities, caused by (a) apoplexy, (b) rupture of vessels, from injury or fracture of the skull. 2. Embolism. 3. Concussion from a blow or fall. 4. Abscess, tumor, or other organic disturbance. 5. Congestion of the vessels of the brain, caused by (a) disease, (b) narcotic poisons, (c) certain mineral poisons, as barium and arsenic.

II. Death Beginning at the Heart—Syncope.—The heart may cease to act, from two distinct causes: (1) from a deficiency in the quantity of blood, its normal stimulant (anaemia), and (2) from a defect in the quality of the blood, or from a loss of heart-power (asthenia).

Anaemia is produced by sudden loss of blood, (1) disease, as in rupture of an aneurism; (2) uterine and other hemorrhage; (3) sudden discharges, etc.; (4) violence, as from
wounds of heart and large vessels, causing fatal hemor-
rhage.

Symptoms.—A mortal paleness of face; lividity of lips; vertigo; cold sweat; dimness of vision; ringing in the ears; slow, weak and fluttering pulse; gradual insensibility. There may also be nausea and vomiting, hallucinations, delirium, jactitations, irregular breathing, sighing, and convulsions before death. The nervous symptoms are due to want of brain power, in consequence of a deficient supply of blood.

Post-mortem appearances.—Heart contracted and empty (if early inspected). If life has been protracted for several hours, a heart clot may be found.

Asthenia.—Here, the cause of the cessation of the heart's action is either a defect in the quality of the blood, or some disorder of the organ producing a loss of heart power: (1) by disease, as (a) various cardiac disorders, such as fatty degeneration, etc.; (b) all exhausting diseases, as phthisis, cholera, cancer, etc.; (2) starvation; (3) certain injuries, as blows on epigastrium; (4) certain poisons, as digitalis, prussic acid, and upas.

Symptoms.—Coldness of hands and feet; lividity of lips fingers, toes, nose and ears; extreme muscular weakness; feeble pulse; senses and intellect not affected, but preserved to the last. This latter is well seen in the collapse of Asiatic cholera.

Post-mortem appearances.—The heart not contracted; its cavities contain more or less blood, or else are dilated and flabby.

III. Death from the Lungs—Apnea—(Asphyxia).—Respiration may be arrested (1) by any mechanical impediment to the ingress of air (oxygen) into the lungs, as (a) pressure on the thorax; (b) tetanic spasm of the muscles of
respiration, as in tetanus and strychnia poison; (c) paralysis of the pneumogastric, or phrenic nerves; (d) exhaustion of muscular power from debility, or cold; (e) foreign bodies in the air passages; (f) compression of the throat, as in hanging and strangling; (g) suffocation; (h) drowning. 2. By disease, as pneumonia, phthisis, etc., spasm of the glottis, oedema of the glottis, pharyngeal abscess, and embolism of the pulmonary artery. (Strictly speaking, most of these diseases cause death, through mechanical interference with breathing.)

Symptoms.—Great dyspnœa, lividity of the face, loss of consciousness, vertigo, and convulsions.

Post-mortem appearances.—The right side of the heart and the whole venous system are usually filled with dark blood; the left side, together with the arteries, is generally empty. Cases are however reported where the right cavities of the heart were found empty. The lungs themselves are nearly always gorged with dark blood; but there are some exceptions to this, which will be noticed hereafter.

By keeping in mind the foregoing varieties of somatic death, together with the characteristic post-mortem appearances attendant on each, the examiner will be considerably aided in arriving at a definite conclusion, as to the real cause of death, in any particular case.

In every inquest over a dead body four important questions will present themselves for solution: 1. The reality of the death. 2. The cause of the death. 3. The time that has elapsed since the death. 4. In the case of the body of a new-born infant,—was it born alive?

I. The first of these questions comprises the phenomena and signs of death. How can we distinguish a case of real from one of apparent death? In the great majority of
instances, of course, there is no practical difficulty; but exceptional cases do, at times, present themselves in persons recently dead, where the corpse still retains so much the appearance of life, as to occasion some doubts about the reality of dissolution. The natural horror of being buried alive also suggests the most scrupulous caution in the matter, although we rarely, if ever, hear of cases of premature burial in civilized countries; yet instances are not wanting to show that such may have actually occurred, in places where a fatal pestilence has prevailed to such a degree as to produce a panic, and demoralize the community. Dr. Tidy (Legal Med. p. 30) informs us that Professor Nussbaum, of Munich, states "that he believes many to have been buried during the war (Franco-German) that were not really dead, but merely suffering from an extreme lethargy arising from loss of blood, exhaustion, hunger, cold and fear" (Jour. de Med. de Bruxelles, February, 1871).

The following may be regarded as the Signs of Death. We are, however, of the opinion that no single sign should be relied upon exclusively, but that several "signs" should always be present in determining the question.

I. The complete and continuous cessation of the functions of circulation and respiration. In some cases of apparent death these two functions seem to be suspended for a time, as in syncope, trance, catalepsy, etc.; but the suspension is not absolute, but only apparent. The absence of the pulse at the wrist is no criterion of the suspension of the circulation, as this may be going on so feebly, as only to be detected by a very close stethoscopic examination of the heart, which should never be omitted in cases of doubt. The condition of both the circulation and respiration, in such cases of apparent death simply resembles that of certain animals in
the state of hibernation. Thus, M. Bouchut informs us that in the marmot, while the heart-beats during its state of activity amount to 80 or 90 a minute, they are reduced down to 8 or 9 very feeble pulsations during the period of hibernation. Instances are recorded (like that of Colonel Townshend, by Dr. Cheyne) of a voluntary suspension of the heart's action; but as these cases occurred many years ago, before the discovery of auscultation, it is, we think, highly probable that the suspension was not absolute, but only reduced down to so fine a point as to have escaped notice. It is certainly contrary to all scientific reasoning that life can continue many minutes without the circulation of the blood; therefore we need have no hesitation as to the reality of death, if we can be positively certain of the continuous arrest of this function, say for one hour. The converse of this proposition, however, is not always true; that is, the pulsation of the heart may continue for a brief space of time after actual death. Duval mentions having seen the heart of a criminal beat fifteen minutes after decapitation, the left auricle pulsating for an hour. This same phenomenon, as is well known, is witnessed still more remarkably in the heart of the turtle, and also of the shark, which will continue to pulsate many hours after removal from the body.

This question of the beating of the heart in a still-born infant being regarded as a valid sign of life, will be discussed hereafter, under the title of Live Birth.

The same remarks may be made with regard to the function of respiration. The absolute and continuous cessation of breathing—say for one hour—may be regarded as a positive sign of death. In cases of apparent death, as already remarked, this function may apparently be suspended; but it is in reality only reduced down to its minimum of action.
This likewise should be verified by careful and repeated auscultation. The common practice of holding a feather near the nose or mouth may serve, by its movements, to indicate breathing. So likewise the deposit of moisture on a mirror, held in the same position, will indicate the feeblest respiration. But neither of these is an absolutely positive sign, since they both fail when applied in the case of the hibernating animal, which we know is really alive.

Another method is to place a small vessel containing mercury on the thorax of the body lying on its back; the slightest respiratory action will be indicated by the movements of a reflected image, made to fall on the surface of the bright metal.

It may be remarked that in cases of trance, catalepsy, and other instances of suspended animation, the body never exhibits either the pallor, or coldness of real death. Moreover, if a ligature be applied around the finger of a corpse, no change of color will be observed; but if the experiment be made on a living body, the tip of the finger will become of a deep red or purple color, in consequence of the arrest of the capillary circulation at that spot.

II. The Condition of the Eyes.—The changes produced in the eyes by death consist (1) in the entire loss of sensibility to light: the pupils neither contract nor expand under this stimulus. This, however, cannot be regarded as a positive sign, since the same insensibility to light is witnessed in certain cerebral affections during life; it is also the result of the action of certain poisons. (2) The action of atropia and other mydriatics to expand the pupil, and of calabar bean to contract it during life, is lost within a few hours after death. These agents do, however, produce a visible effect if applied very soon after the cessation of life, and
before the body has become cold, and all muscular irritability has ceased. (3) The cornea loses its transparency, and the eyeball its elasticity, very speedily after dissolution. But these conditions may likewise exist before death, as the effects of disease. In apparent death, the cornea retains its translucency; the papilla of the retina is of a rose-red color; and the fundus of the eye is furrowed by the arteries and veins of the retina. At the moment of death, the papilla of the optic nerve becomes quite pale, and the central artery of the retina disappears (M. Bouchut, *La Tribune Médicale*, No. 47, 1868). It should also be remarked that the eye sometimes retains its lustre after death, as is witnessed after poisoning by prussic acid and carbon dioxide.

III. *The Ashy Pallor of the Body.*—This sign is very uniform, though not without some exceptions, as in the case of persons of very florid complexions, and in exceptional instances where the cheeks retain their pink color for some days after death, so as to occasion some uncertainty as to the actual fact of death in the minds of relatives. It is also wanting in cases of death from yellow fever and jaundice; moreover, the red, inflammatory zones around ulcers, tattoo marks, the spots of purpura, and ecchymoses or bruises, do not disappear after death. It must also not be forgotten that a death-like pallor is seen in cases of swooning, and sometimes in the cold stage of ague and in collapse.

**SECTION II.**

**SIGNS OF DEATH CONTINUED.—LOSS OF ANIMAL HEAT.—POST-MORTEM CALORICITY.—RIGOR MORTIS.—POST-MORTEM LIVIDITY, OR SUGILLATION.**

IV. *Loss of Animal Heat.*—During life the animal body possesses the wonderful faculty of maintaining its own normal temperature (about 98° F.) independently of the
surrounding medium. This is effected as the result of certain vital processes. When these cease at the moment of dissolution, the temperature of the body immediately begins to decline, and it continues so to do progressively until it attains that of the surrounding medium. It never gets lower than the latter, unless the temperature of this medium becomes suddenly increased; then, for a while, the body will be really colder than the temperature of the atmosphere. The sense of touch does not convey an accurate idea of the actual coldness of the dead body, since the conducting power of the tissues varies materially. The direct application of the thermometer to the body is the only safe criterion.

The time when the cooling of the body is completed may be stated to be, on the average, fifteen to twenty-four hours. Prof. Casper makes it from eight to twelve hours. But it varies very considerably, according to the condition of the body itself, according to the medium in which it is kept after death, and also according to the manner of death. Thus, fat bodies retain heat longer than lean ones; the bodies of young children and of old persons cool more rapidly than those of adults; whilst the bodies of those who die from lightning, or suffocation are said to retain heat longer than others.

The body cools more rapidly if exposed to the air, unclothed, than if covered up in the bed clothes; also in a large, airy apartment, than in a small, close room. It will cool more rapidly in water than in the air. In death from chronic wasting diseases, and also in cholera, the body cools very rapidly. According to Dr. Taylor, loss of blood does not hasten the cooling process.

The interior of the body retains its heat considerably longer than the surface, so that if an autopsy be made
twenty-four hours after death, or when its exterior feels perfectly cold, the abdominal viscera may exhibit a temperature twenty degrees, or more, higher than that of the surface.

It should not be forgotten that coldness of the body is a frequent phenomenon of sickness; it is witnessed in hysteria and ague, also in cholera. Its value as a sign of death consists in the fact that it is progressive, and continuous, whilst the coldness of disease is sudden, and not permanent. Hence, the degree of coldness of the body will often be a good indication of the time that has elapsed since death.

The singular phenomenon is sometimes exhibited of a rise of temperature after death, instead of a fall. This exceptional condition occurs in the bodies of persons who have died from yellow fever, cholera, tetanus, smallpox, and some other acute disorders. The precise cause of this singular rise of temperature (post-mortem caloricity) is not clearly understood. In some instances the increase of heat amounted to nine degrees F. Dr. Davy records a post-mortem temperature of 113° F. in the pericardium. We must suppose in these cases that, after general or somatic death, there still lingers some remnant of vitality in the tissues, or rather that molecular life has continued after the cessation of somatic life. We know that muscular irritability and contractility continue for many hours (under certain conditions) after death, and this undoubtedly indicates the continuance of their molecular activity, up to a certain point.

Another fact to be here noticed is that the rate of cooling after death, although progressive, is not uniform; it is much more rapid during the earlier hours, than later. Dr. Goodhart's observations show that during the first three hours after death the loss of heat, per hour, amounted, in the robust, to 3.5°, in the emaciated, to 4.7°; whilst, when the body was nearly cold, the loss, per hour, was, in the
emaciated, 1.12° and in the robust, 1.26° (Tidy's *Leg. Med.*., p. 49).

V. *Cadaveric Rigidity, or Rigor Mortis.*—By this is understood the stiffening of the body, so generally observed after death. It usually occurs simultaneously with the cooling process. It may be stated to be universal in death from any cause, and to be present in the lower animals, as well as in man. In some instances, however, it is so transient as to escape notice. It comes on at very variable periods, from a few moments, to eighteen to twenty hours after death. This remarkable variation in its approach is chiefly due to the condition of the muscular system at the time of death. Its duration is equally variable, lasting from a few moments, to many hours, or even weeks. After the rigidity passes off, the body regains its original pliancy, and decomposition immediately commences. As a general rule, the putrefaction of the body is retarded until the rigor mortis has passed off.

It commences usually in the muscles of the eye, which often become rigid within a few minutes after death; next in the muscles of the neck and lower jaw; then in the chest and upper extremities; afterwards in the muscles of the abdomen and lower limbs. The rigidity generally passes off in the same order; thus the legs frequently remain quite rigid, after the upper portion of the body has regained its suppleness.

The *seat* of the rigor mortis is undoubtedly the muscular system. That it is in no wise dependent upon the nervous system, is proven by the fact that all the nerves supplying a muscle may be divided, and yet the muscle will continue to act, contracting under the galvanic stimulus. But it ceases immediately on division of the muscle. Even the removal of
the brain and spiral marrow has no effect in preventing the muscular contraction. Again, the muscles of a paralyzed limb become equally rigid with those in sound health. The cause of the contraction is usually ascribed to the coagulation of the muscular plasma (myosin), an albuminous principle possessing the property of coagulation to a high degree. The chemical action of a muscle in rigor mortis is acid (reddens blue litmus), but it becomes alkaline after the rigidity passes off. While in the state of rigor mortis, the muscle is opaque; before this, it is partially translucent. Brown-Séquard has shown that a current of arterial blood will restore muscular contractility to a rigid limb.

The duration of rigor mortis is one of its most important features. As already observed, this is extremely variable, although, as a rule, it does not set in until the body has begun to cool; still, in some of the lower animals, and notably in birds, it often manifests itself while the body is still warm. From the observations of Brown-Séquard and others, it appears that the period after death when the rigor mortis manifests itself, together with its duration, is dependent chiefly, if not altogether, upon the previous degree of muscular exhaustion. To properly understand this, it should be remembered that immediately after death the muscles are in a state of complete relaxation, giving to the body perfect pliancy. This condition may last for so brief a space of time as not to be noticed, though usually it continues for three or four hours, when rigidity commences. During this period of relaxation, the muscles have not yet lost their molecular life, so that they will respond to galvanic and other stimuli. Hence, although the contraction of a muscle by electricity is no positive sign of somatic life, still it will enable us to conclude either that the person is yet alive, or more probably, that death has very recently
occurred. The cessation of all muscular contractility under galvanic stimulus is a proof, not only of the death of the individual, but it also indicates that the death was not very recent—hardly within three, or four hours. So long as the muscles retain their contractility, the rigor mortis is postponed.

It can now be understood that whatever produces exhaustion of the muscular system, must thereby hasten the approach of cadaveric rigidity. Thus, in death from exhausting diseases, as in phthisis or after protracted convulsions, or when the muscular system becomes exhausted by over-exertion and fatigue, as is seen in over-driven cattle, or animals hunted in the chase, the rigor mortis shows itself early, and lasts but a short time; whereas, if death occurs suddenly, in a previously healthy person, the rigidity is postponed for many hours, but when once established, it continues for a much longer period. Thus, according to Brown-Séquard, the bodies of decapitated healthy criminals were observed not to become rigid until after the lapse of ten to twelve hours, and the rigidity lasted over a week, even in warm weather. An experiment of the above named physiologist very satisfactorily proves this statement. Three dogs of equal size were poisoned with strychnia in different doses. One took two grains, and died almost immediately. The second took half a grain, and died in twelve minutes. The third took one-fourth of a grain, and died, after protracted convulsions, in twenty-one minutes. In the first animal, whose muscular system had been least exhausted by the spasms, rigor mortis did not set in before the lapse of eight hours, and the duration was nineteen to twenty days. In the second, where the muscular exhaustion was greater, the rigidity appeared after two and a half hours; and lasted five days. In the third, in which the muscular exhaustion was the most protracted, the rigor
mortis was developed as early as thirty minutes, and lasted less than a day.

It has been supposed by some, that the rigor mortis does not occur in the bodies of persons killed by lightning; this, however, is a mistake, as experience abundantly proves. Neither is it interfered with by the previous loss of blood by hemorrhage. It is, however, dependent on temperature, at least, so far as regards the duration, which is shortened by heat and prolonged by cold. Bodies submerged in cold water retain their rigidity for a considerable length of time.

When a joint or articulation stiffened by rigor mortis (if this be complete), is forcibly bent, the rigidity is destroyed. If, however, the rigidity is incomplete, it will be resumed afterwards. This may serve to distinguish real death from certain cases of catalepsy, tetanus, and hysteria, accompanied by rigidity. In all these latter cases, the stiffness will return, on removal of the opposing force.

Cadaveric rigidity is not so strong as voluntary muscular contraction. As a rule, the flexors are more affected than the extensors, so that the limbs are generally found to be slightly bent after death.

The fact that the involuntary muscles are likewise subject to rigor mortis should not be lost sight of, as it might lead to an error as to the true pathological state of an organ on making an autopsy. The heart, for instance, may be found very firmly contracted after death by rigor mortis; this might be mistaken by the inexperienced, for a true contraction of the organ, the result of previous disease.

Closely connected with rigor mortis, if not indeed a modification of this very state, is the condition described as cadaveric spasm. This is exhibited in the bodies of persons who have died by sudden and violent deaths, in whom there seems to be present a strong will-power just prior to the
death, and producing strong muscular contraction at the moment of dissolution. This spasmodic contraction, moreover, appears to pass at once, after death, into the usual rigor mortis. The best illustrations of this peculiar condition are afforded in those cases of determined suicides, who have taken their lives by shooting themselves with a pistol. Very commonly in such cases, the lethal weapon is found so tightly grasped in the dead man’s hand, as to require considerable force to remove it. The same thing is sometimes witnessed in the bodies of drowned persons; fragments of wood, grass and weeds, or other objects which had been convulsively seized in the water before death, being found tightly grasped in the hands; and where two persons have perished together by drowning, it is not uncommon to find them, after death, convulsively clasped in each other’s arms. To a similar reason, doubtless, is to be ascribed the singular and striking posture which the bodies of soldiers, on a field of battle, killed in conflict, are noticed to have assumed in the act of dying. Thus, the attitude of one is described as “resting on one knee, with the arms extended, in the act of taking aim; the brow compressed, the lips clenched—the very expression of firing at an enemy stamped upon his face, and fixed there by death. A ball had struck this man in the neck. Another was lying on his back, with the same expression, with his arms raised in a similar attitude, the minnie musket still grasped in his hands undischarged” (Taylor).

VI. Cadaveric Lividity, or Suggillation.—This term is applied to those livid, or violet-colored patches, or discolorations, which are observed upon the body at variable periods after death, usually after several hours. It is the result of the settling of the blood in the capillaries by gravitation.
Hence it is noticed in the most dependent parts of the body, such as (supposing it to be lying on the back) the back, sides, and under surface of the neck, calves of the leg, and under portions of the thighs. These patches, at first isolated, gradually increase in size, and coalesce, so as to cover a larger surface of the body. Cadaveric lividity is an unquestionable "sign" of death. It makes its appearance sometimes much earlier than at others; and for this variation no very satisfactory reason can be assigned.

The most important point connected with cadaveric lividity is not to confound it with ecchymosis, or bruising, to which it bears a considerable resemblance. Several cases are recorded where a body has been found covered with these death spots, and the mistake has been made of supposing them to be bruises, and consequently attributing the death to violence inflicted during life. The medical examiner should be particularly cautious not to confound them. Fortunately, a very simple test will settle the question. If the scalpel be drawn through a suggillation, no blood will flow; the most that will be observed will be a few bloody points or specks, arising from the division of small veins of the skin. If, however, the patch be ecchymosis (where the effused blood has been infiltrated into the cellular tissue), the incision will either be followed by a flow of blood, or else a coagulum will be seen. Moreover, whilst the ecchymosis is sometimes raised above the level of the surrounding skin, the cadaveric stain never is. These spots are not affected by age, sex, or constitution; and they follow upon all kinds of death, not excepting that caused by hemorrhage.

Suggillation takes place in the internal organs as well as upon the surface of the body, producing in the former, appearances strongly resembling true congestion and inflammation, for which they are without doubt frequently
mistaken by the inexperienced; and as it may be a matter of considerable consequence, in a post-mortem examination, not to confound these two conditions, the examiner should be very cautious as to his pathological inferences. These internal suggillations are also termed hypostatic congestions; they appear chiefly in the lungs, brain, kidneys and intestines. The fact that they invariably occur in the most dependent portions of these organs should be suggestive of their true origin, since a real congestion or inflammation exhibits itself either throughout the whole organ, or else upon its upper surface equally with the lower one. Certainly, it is not confined exclusively to the under portion, as is the suggillation. When it occurs in the intestines, it may readily be distinguished from true inflammation, by simply lifting up several folds of the bowels, when the horizontal line, which previously had marked the hypostatic settling of the blood, becomes immediately broken and disjointed, whereas, if it had been a real congestion or inflammation, the redness would have involved the whole circumference of the intestines, and there would have been no broken line of separation.

In the brain, hypostatic congestion might be mistaken, by the inexperienced, for one form of apoplexy; and in the spinal cord it might be confounded with spinal meningitis. In the heart, true suggillation is not believed to occur; but this is replaced by the formation of post-mortem clots, called polypi of the heart.

SECTION III.

SIGNS OF DEATH CONTINUED.—PUTREFACTION—EXTERNAL AND INTERNAL SIGNS.—ADIPOCERE.—MUMMIFICATION.—HOW LONG SINCE THE DEATH?

VII. Putrefaction.—This is usually regarded as the most unequivocal "sign" of death. By this term is understood those spontaneous chemical changes undergone by all dead
animal bodies, resulting in the elimination of fetid gases. The period after death when putrefaction first manifests itself varies considerably, being dependent upon several conditions, some of which are connected with the body itself, and others extraneous to the body.

Among the conditions inherent to the body itself are:
1. *Corpulence.* Fat and flabby bodies undergo putrefaction more speedily than thin and lean ones, doubtless on account of the preponderance of fluids in the former. 2. *Age* and *Sex.* For the same reason the bodies of new-born children, and of women dying in child-bed (according to Casper), decompose more rapidly than others, especially the aged. 3. *The manner of death.* The bodies of persons dying after an exhausting disease, especially if the vitality of the blood has been impaired, as in typhus fever, undergo rapid putrefaction; also after death from certain poisons, and especially poisonous gases, as coal gas and sulphuretted hydrogen; also from suffocation from smoke, and, indeed, from suffocation generally. Putrefaction is also accelerated in bodies that have been much bruised and mangled by machinery, or railway and other accidents; but we must except those cases where the body remains protected from atmospheric influences, as when buried beneath ruins, etc. On the other hand, the process is retarded in death by alcohol, phosphorus, sulphuric acid, arsenic, and some narcotic poisons. The antiseptic properties of alcohol and arsenic are well understood. The action of sulphuric, and doubtless the other mineral acids, is probably to neutralize the ammonia as fast as it is formed, rather than actually to retard the process of putrefaction. Admitting all the above conditions, and giving them due allowance, there are doubtless other causes, as yet unknown to us, which influence the rapidity of putrefaction. Casper adduces the instances of
four men, all of about the same age and general physique, and all suddenly killed in a riot. They were all buried at the same time, and in precisely similar coffins and graves; yet, on subsequent examination, the progress of decomposition in the several bodies was found to vary very considerably.

The external or objective conditions influencing putrefaction are *air, moisture* and *temperature*. The influence of the atmosphere upon animal decomposition is well understood, and is familiarly witnessed in the preservation of meats and other articles of food in hermetically sealed cans, for an indefinite length of time. It is undoubtedly the oxygen of the atmosphere that is the destructive agent, since flesh may be preserved in nitrogen (the other constituent of air) for a long period. Moreover, the oxygen must be in a free state, as it exists in the atmosphere, and not in a compound, as in carbonic acid gas, or nitrous and nitric oxide. These gases do not act as decomposing agents. The influence of atmospheric air is not limited to the mere supply of oxygen, but it modifies putrefaction according to the amount of moisture it contains. For this reason, perfectly dry air, such as that of the arid deserts of Arabia and Africa, by its rapid desiccating properties, arrests putrefaction; the body speedily losing its fluids by evaporation, dries, and shrivels up into a sort of mummy. The effects of an entire exclusion of air in retarding the process of decomposition in a human body are witnessed in the burial of royal personages in leaden coffins hermetically sealed, and these afterwards enclosed in marble sarcophagi. When these have been opened, hundreds of years subsequently, the remains have been found in a remarkable state of preservation. On the other hand, bodies naked, or but slightly clothed, and buried in pine coffins, which soon decay, and
in shallow graves, to which the air has easy access, will undergo very speedy decomposition. The nature of the soil, and the depth of the grave also materially influence this process. Thus, a loose, sandy soil and a shallow grave favor it, by the ready admission of air, whilst one of a stiff, clayey nature, and a deep grave would retard it, for the opposite reason. From recent observations, it is highly probable that the real cause of atmospheric influence upon decomposition is the presence of the bacteria or baccilli which float in such myriads in the air, and which find their peculiar habitat in dead animal matter.

The effect of moisture as an agent in animal putrefaction is to aid it, by favoring solution. The different tissues and organs of the body undergo decomposition just in proportion to the amount of fluids they contain. In this respect the brain of the young infant, and the eye contrast widely with the bones and teeth. The human body contains eight-tenths of its whole weight in fluids; hence its great tendency to putrefy after death. The bodies of drowned persons undergo rapid decomposition, unless the water be extremely cold. In the latter case, the low temperature acts as a preservative. Likewise, bodies thrown into dungheaps and cesspools speedily putrefy, from a similar cause, although the process may also be aided by the warmth of these media. If a body be completely deprived of its fluids by drying, putrefaction is arrested, as was remarked under the preceding head.

The influence of temperature as an agent in putrefaction is very manifest. The temperature most favorable to this process is that between 70° and 100° F. It commences, however, as low as 50°, but it is completely arrested at 32°, below which the body becomes frozen, and also at 212°, when it becomes desiccated by complete loss of its fluids,
through evaporation. As is well known, an animal body may be preserved for an indefinite period if completely frozen in snow or ice. It is recorded that the body of a Russian nobleman that had been buried in the frozen soil of Siberia, on being exhumed, after a period of ninety-two years, was found in a state of almost perfect preservation. On the other hand, the effects of a high temperature as a preservative are witnessed in the mummies of Egypt and adjacent countries. In this case, however, the dryness of the atmosphere as well as the high temperature assists in the preservation.

The effect of temperature in the process of putrefaction is familiarly shown in the influence of the seasons. Thus, in summer a body will decompose very much sooner than in winter—a circumstance that should not be forgotten when giving an opinion respecting the date of death in an unknown case. According to Casper, the relative rapidity of decomposition in bodies exposed to the air, kept in cold water, and buried in the earth, is in the ratio of one, two and eight; that is, putrefaction advances as rapidly in one week in the open air, as in two weeks in the water, and in eight weeks in the earth (average). It may here be remarked that a body floating near the top of the water will decompose more rapidly than when at the bottom; and when taken out of the water and exposed to the air, the putrefaction will be far more rapid than if left in the water.

External signs of Putrefaction.—The following is the order generally observed, externally, in the progress of putrefaction of bodies exposed to the open air. In one to three days in summer (three to six in winter), there first appears a greenish, or yellowish-green spot upon the abdomen, three or four inches in diameter, accompanied with the peculiar odor of putrefaction. The eyeball becomes soft and yielding within the same period. In a few days
more, this greenish discoloration has spread generally over the whole body, first in spots, which subsequently gradually coalesce. Dirty red streaks now show themselves throughout the surface, marking the course of the blood vessels. In ten or fifteen days (in warm weather), the epidermis begins to loosen, forming blebs or blisters containing fluid. Gases now begin to form in the chest and abdomen, causing these cavities to swell out greatly. The eyeballs protrude, from the same cause; the face is swollen; the features so much bloated as no longer to be recognizable. In two or three weeks, the blebs of the cuticle may have burst open; maggots appear; the formation of gases increases, so that the body is enormously swollen. If it be now punctured, the gas which is emitted will frequently take fire on the approach of a flame (carburetted hydrogen). Other gases are likewise formed, the result of animal decomposition, as carbonic acid, sulphuretted hydrogen, phosphoretted hydrogen, nitrogen, and ammonia. The nails now loosen; and in the further progress of putrefaction the cavities burst open, and discharge their contents; the softened flesh dissolves off from the bones, which now become exposed, and ultimately fall apart from the skeleton. The sexes cease to be distinguishable, except perhaps by the discovery of a uterus, which appears to be the very last organ to yield to putrefaction.

The above description is only a very general and average one, since the process of the external putrefaction of the body is so very variable, and is influenced by so many circumstances, all of which are not yet fully understood.

*Internal signs of Putrefaction.*—The order in which the internal organs of the body undergo decomposition, being more regular as to time, affords a rather better criterion as to the time of death. The first organ of the body that
shows signs of decomposition after death is the lining membrane of the windpipe (larynx and trachea); this assumes a dirty red coloration simultaneously with the appearance of the greenish spot upon the abdomen. That this is not the result of injection of the blood vessels is proven by the microscope. In the earliest stage of death, this membrane is always very pale, except when the death has been caused by laryngitis, or suffocation. The examiner should be cautioned not to mistake this mark of putrefaction for congestion. Very soon after this stage of redness, it becomes of an olive-green color, and the rings of the trachea separate, and it all falls to pieces and disappears.

The next (2) organ to decompose is the brain of young infants. The reason of this lies, of course, in the fact that this organ at such an early age is so very delicate, and is so little protected, by its bony covering, from the outer air. When decomposing, it changes into a soft, rosy, pulpeceous mass, and flows away out of the smallest openings.

Then follows (3) the stomach. This organ is among the earliest to putrefy after death. The decomposition first manifests itself in discolorations of the fundus, together with the formation of dirty red spots in the posterior portion of the fundus, owing to hypostatic congestion. These spots soon ramify, and cover the whole lining membrane. There is great risk of mistaking these spots for signs of congestion or inflammation due to irritant poisoning. The examiner should be specially cautioned on this point, as it is often impossible to distinguish them apart by a merely ocular inspection. It is quite certain that a post-mortem redness of the mucous membrane of the stomach cannot of itself prove a case of poisoning. In the further progress of putrefaction, the stomach softens, the spots
become greenish and gray; then black, with dark red streaks (veins) running through them. It is finally converted into a pulpaceous mass, and ceases to be recognized.

Next to the stomach, the intestines (4) follow in the process of decomposition. They become discolored very much as in the case of the stomach; then they burst open, and discharge their contents, forming a greasy mass which finally disappears.

The spleen (5) comes next in the order of putrefaction. If not diseased at the time of death, it may retain its integrity for two or three weeks. It first assumes a dark red color, then a greenish-blue, then becomes soft and pulpy, so that its substance can be rubbed down with the handle of the scalpel.

Following the spleen, the omentum and mesentery (6) are the organs next to decay. If there is not much fat connected with them, they will rapidly dry up, and disappear.

The liver (7) resists putrefaction for a considerable time after death—in adults for several weeks. In infants it decomposes earlier. It first becomes of a green color, then black; then softens, shrivels, and finally disappears. In case of death by arsenic poisoning, the liver would be likely to resist decomposition for a very considerable time, in consequence of the affinity of that organ for arsenic, which would exercise its preservative influence over it.

The brain of adults (8) does not begin to show signs of putrefaction until the end of the fourth or fifth week, and sometimes even later. The process commences at the base, which softens and becomes bluish-green, and gradually progresses upward, and then inward. If the brain has been injured, as by a depressed bone, or by a gunshot wound, it is affected earlier.

Next in order is the heart (9). This is one of the toughest
of all the organs. The softening here begins in the columnae carneae, and progresses outward toward the walls of the organ, which finally deliquesce into an unrecognizable mass.

It is remarkable that the lungs (10), which are very soft organs, and are so nearly connected with the outward air, should resist putrefaction so long. These organs are often found quite sound for weeks after death, provided they were healthy and uninjured at dissolution. The first evidence of their decomposition is the formation of little bladders of air in the sulci, between the lobes, on the under surface, looking like a string of beads. These increase rapidly, the lung structure turning first green, then black, and finally softening and disappearing.

The kidneys (11) follow the lungs. They become reddish-brown, and soften; then they assume a greenish-black color, and soften and disappear. Next in order (12) follow the urinary bladder and œsophagus. Next (13) the pancreas, which, though a soft organ, and located near the stomach, is among the last to decompose. Then follow (14) the diaphragm and the arteries; the tissue of the latter resisting putrefaction while everything else around them has fallen into a shapeless mass.

Last of all, according to Casper, is the uterus (15), which has been found to retain its identity at the end of seven months after death. This fact is of great medico-legal importance, where the question arises of the possibility of pregnancy.

The above description of the progress of putrefaction, both external and internal, of the human body is taken chiefly from Prof. Casper's work on Forensic Medicine, translated and published by the Sydenham Society. It is intended to represent the average, both as regards appearance and time.
As already stated, there may be considerable deviations from the order laid down, depending upon a variety of circumstances.

*Saponification*, or *Adipocere.*—It sometimes happens, in the course of the putrefaction of the body, that this process is interfered with under peculiar circumstances, and gives place to a new condition, known as the saponification of the body, or the production of adipocere. This remarkable condition was first observed by Fouroroy, who discovered, during the removal of human remains from one of the public cemeteries of Paris, that a number of the bodies, instead of undergoing ordinary putrefaction, had been converted into a new substance, which he styled *adipocere*, from its resemblance to a combination of fat (*adeps*) and wax (*cera*).

This adipocere has an unctuous feel, somewhat like spermaceti, and is of a whitish, discolored appearance. By the analysis of M. Chevreul, it was found to be an ammoniacal soap—a compound of stearic and oleic acids, united with ammonium. In the course of putrefaction, the fatty acids of the body combine with the ammonia, which is the result of the decomposition of the nitrogenized tissues. It is interesting to inquire what are the conditions under which this singular process of saponification replaces the ordinary decomposition of the body. The presence of water is essential to it. It only occurs in bodies that have been buried in wet, or very moist soil. It never happens to those interred in a loose, or sandy soil. It is frequently the case that when the grave, after burial, fills with water, the contained body is converted into adipocere. The same thing takes place in bodies which remain in the water for a certain length of time.
The composition of adipocere is not always precisely the same. Its base may consist of either ammonia or lime. The latter takes the place of the former, whenever the saponified substance remains for any considerable time in water containing any salt of lime. This was determined experimentally by Orfila, who placed an ammonium adipocere in a solution of sulphate of lime; he found that after a time it had been changed into the oleo-stearate of lime. Adipocere is insoluble in water, but partially soluble in alcohol. It takes fire and burns at a temperature of about 212° F., emitting a greasy smell. It contains a coloring matter, and an odorous and a bitter principle. Its odor resembles somewhat that of musty cheese.

From the fact that if a body remain immersed in the water for any length of time it is likely to be changed into adipocere, it becomes an important medico-legal question to establish the period necessary for this conversion. Devergie ascertained that the body of a new-born child was more or less changed into adipocere after remaining in the water for five or six weeks. We see at once the value of this knowledge to the legal physician, since the bodies of new-born infants are frequently thrown into wells, privies and cesspools, by their unnatural mothers. If such a body be found under such circumstances, with the process of saponification only just begun, it is tolerably certain that it could not have been long in the water, and vice versa. According to the same distinguished authority, an adult body requires an immersion in water for one year, before the conversion is complete; and when it is buried in wet earth, a period of three years may elapse before the change is completely effected.

An adipocerous body is always heavier than an ordinary one, because the adipocere is more weighty than the original fat.
Mummification.—This constitutes another process by which the ordinary putrefaction of the body is interfered with. By mummification we understand the complete desiccation, or drying up of the body. A mummified body is the result either of burial in an arid and sandy soil of hot countries, such as those of Arabia and Egypt, or of the exposure of the body to a constantly cold and dry atmosphere—where, for instance, it is placed in a vault, through which a constant stream of dry, cold air is pouring. Such a condition of things is found at the Hospice of St. Bernard, in Switzerland. In the charnel house attached to this establishment, the bodies of those who have perished in the snows are placed. The atmosphere is so constantly cold and dry, that the flesh and fat completely dry up. It is quite impossible, from the mere inspection of a mummy, to venture an opinion as to the length of time that has elapsed since death. Some of the Egyptian mummies are from two to three thousand years old, as is shown by the inscriptions upon their burial cases.

There are certain agents which retard, and others which promote, decomposition. The former comprise the various antiseptics. Lime, although popularly supposed to hasten putrefaction, in reality retards it, as is shown by a simple experiment of Dr. John Davy, who buried a piece of raw flesh that had been first covered over with powdered lime. It continued sound much longer than another piece that was buried without the lime. No doubt, the lime here served the purpose of excluding the atmospheric air. The strong acids and alkalies, although they do not hasten putrefaction, promote dissolution through chemical action, and in this way they aid in the removal of a body.

The period and method of Interment very materially influence the rapidity of putrefaction. Thus, if decomposition
has already set in *before* burial, this action will progress far
more rapidly afterwards, than in a body which was interred
before putrefaction was begun. Again, the depth of the
grave and the nature of the soil exercise a very marked
influence on the subsequent decomposition of the body, as
already pointed out. Finally, the cause of death—as from
a wasting disease, more especially when the blood has been
deteriorated, as in typhus fever, etc.—will materially influ-
ence the subsequent rapidity of the decomposition of the
body.

II. Having disposed of the first medico-legal question—
is the death real or apparent? we may consider the second
important query—how long a time has elapsed since the
death? This is to be determined, in the absence of direct
evidence, solely by attending to the different "signs" or
phenomena of death already described. The inferences may
be drawn, first, from the signs occurring before putrefaction,
secondly, from those occurring after it.

(1) *Inferences from the signs exhibited before putrefaction.*—
If the body is only slightly cold, and rigidity is just com-
mencing about the jaws, the eyes glazed, and the eyeballs
sunken; death has occurred, most probably, from a quarter
of an hour to four or five hours. (The inference can never
be more than approximative.)

Suppose the body to be perfectly cold (externally) and
rigid throughout: it has probably been dead from twelve
hours, to three or four days. If rigidity is complete over
the body, and cadaveric lividity (suggillation) is manifested
over the surface, death has probably occurred from one to
four days.

The importance of attending to the above phenomena, by
the medical jurist, is shown by a case mentioned by Taylor,
which occurred in London some years ago. A man named Gardiner was convicted and transported for killing his wife. The woman was discovered, with her throat cut, at 8 o'clock in the morning. She was very rigid throughout the upper part of her body, and the whole body was cold. The prisoner was able to prove an alibi between the hours of 4 and 8 A.M., and his counsel endeavored to show that the post-mortem coldness and the partial rigidity might have developed within four hours, which, if true, would have exculpated the accused. But this point was very properly overruled by the mass of medical testimony to the contrary.

(2) Inferences after putrefaction.—Suppose the body exhibits the greenish discoloration on the abdomen, the peculiar odor of putrefaction; the rigor mortis has passed off, and the body is cold but pliant: death has occurred from one to three days, in summer, and from three to six or eight days, in winter.

If the greenish-yellow discoloration extends more or less over the whole surface, together with greenish-brown stains, and dark red lines over various parts, along with relaxation of the sphincter ani muscle, it must have been dead from eight to ten days, in summer, and from ten to twenty days, in winter.

If blebs are found over the skin, and some of them opened, with maggots in the muscles; if the body is green all over, and the chest and abdomen are enormously distended; the nails loose or falling out; the color of the eyes not recognizable; the features very much swollen, then the death must have occurred from two to three weeks, in summer, or from four to five weeks, in winter.

If the chest and abdomen have burst open and discharged their contents, and some of the bones are denuded of their
fleshy coverings; the eyes enormously swollen; the body has been dead, probably, from two to four months.

The above "inferences," it will be remembered, are only approximative, as already stated. They cannot be positive under any circumstances; and, moreover, they are predicated on the supposition that the body under examination has not been buried, but exposed to the action of the atmosphere. It is important that the legal physician should avoid giving a very positive opinion on this question, as it must, at best, be but conjectural, and dependent on so many contingencies.
CHAPTER III.
MEDICO-LEGAL INVESTIGATIONS.—THE POST-MORTEM.

Responsibility Involved in Making the Post-Mortem.—General Accuracy and Method Necessary.—Examination of the Surroundings of the Body.—External Examination of the Body.—Internal Examination of the Body.—Details of the Examination.—Measurements and Weights.—Notes.

The physician who undertakes to make a post-mortem examination in a medico-legal case, assumes a very serious responsibility. He should, therefore, be fully prepared to meet the various contingencies that may present themselves, and he should execute his work so thoroughly as to leave no cause for subsequent regret. He should, moreover, perform his duty with strict impartiality, unbiassed by prejudice, and untrammeled by fear or favor. Not only should the examiner be an expert anatomist and pathologist, but he should also be a close and careful observer of all the surrounding circumstances that might throw light upon the case.

In all cases where dispute is likely to arise, it is advisable to have two examiners, so as to avoid an ex parte appearance; and the suspected person should be represented by a friend of his own selection. The examination should always, if possible, be made by daylight, since artificial light might conceal certain shades of color which it might be important to recognize, such as the stains of nitric acid.

Where a chemical or microscopical investigation becomes necessary, the parts required should be carefully put aside for as early a subsequent examination as possible.

The examination should always be exhaustive—leaving
nothing undone—so that the examiner may be able to testify accurately as to the cause of death. For example, the discovery of a disease of the heart (a sufficient cause of death) should not preclude an examination of the lungs and brain, in each of which the real cause of the death may be found located. So, also, the finding of poison in the stomach may co-exist with a ruptured aneurism, or a clot in the brain. The examination, moreover, should be conducted according to method, and all the details carefully recorded in a notebook.

The post-mortem should be made as early as possible after the first day subsequent to the death. But it should never be declined on account of the interval that may have elapsed, nor even if the body be in a state of putrefaction.

The surroundings should first claim attention, such as the locality where the body was discovered, as this may afford a clue to the criminal, especially in a case of infanticide. Sometimes the body has been dragged by the murderer to a distant spot, or the victim may have followed his assailant after receiving the blow, and died at a distance. The presence of footprints near by should be noted, together with their direction; evidences of struggling, as denoted by the condition of the grass, or dust, or mud in the road; the presence of any weapon, or other missile. If in a room, the position of the body in reference to articles of furniture, to any weapon, to glasses, cups, bottles, etc., from which poison may have been taken. It is also advisable to make a sketch, or rough drawing of the locality.

As regards the body itself, the examiner should note its exact position when found: this is especially important if death was caused by a wound. The clothes should be carefully examined, whether torn or cut; whether marked by blood-stains, or by any acid; if stabbed, whether the cuts
correspond with the wounds on the body. The clothes should then be removed, and the whole body minutely inspected. It should also now be identified, if possible. Notes should be made of the sex, height, weight, age and general development; of scars and other marks; abnormalities; blood, seminal and other stains; the color of the skin, and condition of the eyes and teeth; the temperature and rigidity of the body; the degree of putrefaction; lividity and ecchymoses; matters flowing from the nose and mouth; state of the tongue; expression of the countenance. The hands should be inspected, to ascertain if they hold a weapon—whether loosely or tightly grasped; or if portions of hair or clothing are firmly held (denoting a struggle); whether stained with blood, or blackened by powder (the latter indicating firearms); the presence or absence of foreign bodies in the nose, mouth, anus and vagina should also be noticed.

All wounds should be carefully examined, as to depth, extent and direction, and whether they suit the weapon that may be found near by; the condition of their edges, as indicating whether recent or not; marks of inflammation, suppuration or gangrene; whether any foreign body be present, as a ball, fragments of clothing etc. The scalpel may be used, if necessary, to enlarge the wound, with care not to interfere with its original character. If there is contusion without solution of continuity, the examiner should not fail to look for internal injuries.

In fractures and luxations, notice their condition, and that of the surrounding parts. In case of burns, observe their degree and extent; whether merely inflamed or vesicated, and the state of the adjacent parts.

In females, examine the genital organs, in cases of rape, pregnancy, and recent delivery.

In newborn children, ascertain their length and weight,
condition of the hair, nails, membrana pupillaris and genital organs, condition of the umbilical cord. The question of a live-birth will be a subject for future investigation.

The Internal Examination of the Body.—The following order should be observed:

(t) The Head.—After a careful external examination for wounds or injuries (for which the hair may have to be removed), the scalp should be separated by an incision made across it from ear to ear, down to the bone; it should then be everted in both directions, so as to expose the skull. Now look for fractures, and do not mistake irregular sutures for these. Notice any unusual thinness of bone; follow out any fracture to its whole extent; observe any extravasation of blood under the scalp.

The skull should now be carefully sawed around, about half an inch above the opening of the ear, the calvarium removed, and the condition of the dura mater noticed. This membrane should be carefully cut around with a probe-pointed scissors, and the arachnoid and pia mater closely inspected.

The upper part of the brain can now be examined before removal—as to congestion of its vessels, laceration, or extravasation of blood upon its surface. (Remember this latter is often seen on the side opposite to the external injury.) The brain is now to be carefully removed, by inserting the fingers beneath it, and dividing the medulla oblongata.

The base of the skull should be carefully inspected for fractures, which otherwise might escape notice.

The brain should now be examined from above, slicing it horizontally; regarding specially its consistence, color, presence of extravasated blood or serum, of tumors or abscesses, disease of blood vessels, or of the membranes.
In opening the skull of very young children, a pair of strong scissors may be used instead of a saw.

(2) The Spinal Column should be opened through its whole extent, by sawing through on each side of the spinous processes. The cord, together with the dura mater, should then be removed and examined. The presence of fracture, dislocation, or contusion, should be carefully noticed.

(3) The Neck should be carefully inspected for marks of violence by the fingers (garrotting); by a cord (strangling or hanging); ecchymoses; the great vessels, whether full or empty; the nerves, whether in their natural state. The cavity of the mouth and nose. The condition of the larynx, trachea, pharynx and oesophagus.

The thorax and abdomen may be opened together, by a single incision, extending from the root of the neck to the pubes, and a transverse one across the umbilicus. The thorax should be first examined, except in the bodies of new-born children, where it is important to observe the condition of the diaphragm; in this case the abdomen should first be opened. Moreover, in cases of death from asphyxia, it is recommended by Dr. Tidy to examine the condition of the heart before opening the head, because the blood is apt to escape from the right side of the heart if the head is opened first.

(4) The Thorax.—The cartilages of the ribs, together with the sterno-clavicular ligaments, should be carefully divided, avoiding wounding the large veins of the neck, and the sternum should be reflected. Notice the condition of the lungs, whether adherent, collapsed, or emphysematous. Record immediately the position and color of the thoracic viscera; also the presence, and amount, and nature of any fluid in the pleural cavity. Open the pericardium, and note the presence and amount of any contained fluid.
The lungs are removed by passing the hand beneath them (noticing any adhesions), and cutting through the bronchi and vessels at their roots. They should be inspected as to their color, density, etc., as indicating disease; the condition of the bronchial tubes and pulmonary artery (embolism); and the presence of foreign matters in the air passages (in case of drowning). If blood has escaped into the thorax, it should be removed by a sponge, so as to ascertain the color of the parts. The hydrostatic examination of the lungs in new-born children will be considered farther on.

The Heart should be examined in situ, before removal, as to its size, and the fullness of the coronary vessels; the cavities should now also be opened, and the amount of their contained blood noted, together with any clots or polypi. The organ may next be removed by cutting through the vessels at its base, and examined as to its weight, condition of its walls and tissue, and state of its valves. Sometimes a microscopic investigation may be required. The aorta should likewise be examined, for atheroma and aneurism.

(5) The Abdomen.—On removing the integuments, the examiner should carefully note all signs of peritonitis, and of swelling, extravasation, strangulation, or twist of the intestines, and hernia; likewise the condition of the liver, spleen, kidneys, bladder; and, in the female, the state of the vagina, uterus and ovaries, the uterus especially, for evidences of pregnancy, abortion, and delivery.

The Stomach should be examined by first ligating it at the cardiac extremity, and then by applying two ligatures at the pyloric end, and cutting between them. Note the general external appearance, and then open it along its lesser curvature. Examine the contents, as to quantity, character and odor, and reaction. Carefully inspect the lining membrane with a lens, for solid particles of phosphorus, crystals, or
patches of arsenic, or other mineral poisons, fragments of leaves or seeds, or other foreign matters. Note any evidences of inflammation, or ulceration.

The Intestines should next be examined, by removing them from their attachments, and slitting them throughout with an enterotome; looking for inflammation and ulceration of the glands, and for any foreign matters; also noting the condition of the appendix vermiformis. In cases of poisoning, the stomach and its contents should be preserved for chemical examination, in a separate jar. The intestines also (at least a portion of the small and large bowel, together with the rectum), and portions of the liver, kidney and spleen, should be kept for a similar purpose. The jars containing the viscera should be securely stoppered, and sealed with the private seal of the examiner, with a label affixed, stating the name of the deceased person, the date of death and of the autopsy. They should then be delivered personally, by him, to a responsible party, from whom he should always take a written receipt.

In case of disinterment of a body, the inspectors should always view it before it is removed from the coffin; at which time, also, it should be properly identified by the friends or relatives of the deceased.

In a medico-legal examination of a body, such as above described, there should always be present either a second inspector, or a clerk, to take down the notes as the autopsy progresses. These notes should comprise the appearances presented by the different organs, stating only facts, but no opinions. The notes should be carefully read over by both examiners before sewing up the body, and, if necessary, corrected. A report should then be carefully drawn up, containing the opinion of the case, as deduced from the ascertained facts, with the reasons therefor, clearly and succinctly stated, but avoiding all theorizing on the subject.
CHAPTER IV.

PRESUMPTION OF DEATH, AND OF SURVIVORSHIP.

CASES INVOLVING PRESUMPTION OF DEATH, AND OF SURVIVORSHIP.
—FRENCH LAW OF SURVIVORSHIP.—PROBABILITIES AFFORDED BY AGE, SEX, AND MODE OF DEATH.

Presumption of Death.—This question may be raised when a person goes away from home, and is not heard of for many continuous years. The law will, in that event, regard him as dead, or presume his death, and his administrator or executor may proceed to settle his estate. The question under this form is not unfrequently raised in life insurance companies, where the party insured has not been heard of for many years, and his lawful heirs demand the payment of his policy. It must also be considered in cases where a husband deserts his wife, or vice versa; or where either married person leaves the other, and remains continuously away; or where the party going away without the intention of remaining, is not afterwards heard of for a succession of years.

The length of time usually regarded as legally warranting a presumption of death, in any of the above cases, is seven years from the time the person was last heard from; so that in the case of married persons, it is not regarded as bigamy if the other party should marry again after the expiration of the seven years of continuous absence, without being heard from, or being known to be alive. In cases of heirship and property, and in some cases of life insurance, it is often not considered necessary to wait the whole seven years, but a settlement has been made by the courts or company in two years.
The presumption of death must depend on general evidence, being a presumption of fact to be determined by a jury. There are cases, however, of a special character, where the courts have decided the presumption of death to be sooner or later than the period of seven years, as, e. g., if the individual concerned was in feeble health when he, or she, was last heard from. This question would involve medical evidence as to the probabilities of life, in such a case.

Presumption of Survivorship.—Questions relating to presumption of survivorship are much more frequently discussed in the courts than those pertaining to presumption of death. There is, however, no general law upon the subject, either in this country, or Great Britain, every case in which the question is involved being decided according to its individual merits. When two or more persons perish by the same calamity, in the absence of all testimony, the courts frequently refuse to assume that one survived the others, but have decided that all perished together. Yet, very momentous questions may be dependent upon a legal decision of the question of survivorship; as when the parties dying are a father and a son; if the son survive but for a moment, "his wife shall have dower, for the lands descended the instant the father died." So, in the case of a testator and legatee; if the latter dies first, the legacy lapses; but if he survives the testator, for ever so short a time, his executors can claim. So, again, the husband of a woman possessed of freehold property (not specially settled), has a life interest in her estate, provided she has issue by him, born during the life of the mother, and which survives her even for a moment of time (tenancy by courtesy). The old Roman law upon this subject, upon which are based most of our
modern decisions, enacted that when persons of different ages perished in battle, those under puberty were deemed to have died first; but if the son was above the age of puberty, and both died together, the son was presumed to have survived the parent. In the case of husband and wife, the husband was presumed to be the survivor (Beck).

According to Foderé and Beck, the French law, as contained in the Code Napoléon, is as follows:

"I. If several persons, naturally heirs of each other, perish by the same event, without the possibility of knowing which died first, the presumption as to survivorship shall be determined by the circumstances of the case; and in default thereof, by strength of age and sex.

"II. If those who perished together were under fifteen years, the oldest shall be presumed the survivor.

"III. If they were all above sixty years of age, then the youngest shall be presumed the survivor.

"IV. If some were under fifteen, and others above sixty, the former shall be presumed the survivors.

"V. If those who perished together were over the age of fifteen, but under sixty, the males shall be presumed the survivors, where the ages are equal, or the difference does not exceed one year.

"VI. If they were of the same sex, that presumption shall be admitted which opens the succession in the order of nature. Of course, the younger shall be considered to have survived the elder."

According to Section IV in the above Code, no distinction is made between an infant and a man of sixty years; yet certainly, it may fairly be supposed (as remarked by Dr. Tidy) that the latter had a better chance of life than the former. The Prussian law on this question is about identical with the Code Napoléon.
Although our laws are not decisive on questions of presumption of survivorship, but treat them as questions of fact depending wholly on evidence, and, in the absence of all evidence, regarding them as matters incapable of being determined, still, there are certain matters of importance connected with each case as it presents itself, which deserve the consideration of the court and jury, in influencing their decision. These points may be considered under the following heads:

(1) **Probabilities afforded by the Age.**—Between a father, and a child under puberty, the English civil law decides the father to be the survivor. Between the ages of fifteen and sixty, there is no probability. Between a middle-aged man, and one under fifteen and over sixty, the probabilities are in favor of the former. Between one under fifteen and one over sixty, the former is deemed the survivor; but the same exception might be taken here as in the case of Sect. IV of the Code Napoléon (supra). Between two under fifteen, the oldest is considered the survivor. If the question is between a mother and infant, both dying in childbed, without assistance, the presumption of survivorship is in favor of the mother, because the child might be still-born, and also because, if large, its life might be endangered by delay, and it would be more exposed to danger without assistance, such as strangulation by the cord, or suffocation in the discharges of the mother.

(2) **Presumption afforded by the Sex.**—The presumption is in favor of the male, when it is a question of physical strength and courage, as when a man and woman perish together by drowning, or some other casualty. But in particular cases, the question of the respective health of the two persons might have to be considered. When, however, it is a question of passive endurance, especially where insen-
sibility supervenes, then the presumption is in favor of the female.

(3) The *Cause of Death*, as affording a presumption of survivorship. In death from asphyxia (apnœa), as in smothering, or breathing noxious gases, as women require less oxygen than men, the probabilities are in favor of the former, other things being equal. Thus, it is stated that, in Paris, in one year, there occurred three hundred and sixty cases of poisoning by charcoal vapors; of this number there were nineteen instances where a man and a woman were exposed together, and of these only three survived, and all were females. Dr. Beck relates the case of a man, wife and child, who were all asphyxiated while sleeping in a room which was exposed to the vapors of a coal stove. In the morning, the man was found dead, the child dying, but the woman recovered.

In *drowning*, or *shipwreck*, the question becomes very complicated, having to take into the account age, sex, strength and opportunity. Thus, men, being stronger, more likely to be able to swim, and, in case of shipwreck, being more apt to be on deck, and, therefore, in a better position to escape, have the best probabilities for survival; but, on the other hand, the buoyancy of a woman's clothes might support her in the water, and thus save her life, under possible circumstances. In case of two or more persons, all males, equally exposed, a presumption of survivorship can only be entertained by searching for bodily injuries, or other weakening causes, which would necessarily interfere with the individual's exertions to save his life. Here, also, their respective swimming capacities would have to be considered.

If the question be on the survivorship, in the case of several persons exposed to excessive *cold*, the amount of
clothing, the physical condition, and the immoderate use of alcohol, must all be considered before arriving at a conclusion. The probabilities would here be in favor of the strong adult over the very young, or very old person, and of males over females. The debilitating effects of poverty, entailing a bad nutrition, and also of intoxication, as being especially obnoxious to the effects of cold, should not be overlooked. The perishing of drunken people on a cold winter's night is a too familiar occurrence. In relation to the effects of heat, it may be remarked that, while the young and old suffer more from cold than adults, they seem able to withstand a greater amount of heat than the latter.

In death by starvation, the general principle that the young require more food than the aged, will determine the presumption of survivorship to be in favor of the latter; also for the female, rather than the male. Certain circumstances, however, should here be considered, such as proximity to water, which would aid in sustaining life for some time, even without food.
CHAPTER V.
PERSONAL IDENTITY.

SECTION I.
IMPORTANT MEDICO-LEGAL BEARINGS.—I. IDENTIFICATION OF THE LIVING.—PERSONAL APPEARANCE.—PECULIAR BODILY MARKS.—REMARKABLE CASES.

The medico-legal consideration of the subject of personal identity is much more important than it may appear at first sight. The question is often raised in trials, both of a civil and criminal character, and it may constitute the chief link in the whole chain of evidence. Cases of mistaken identity are constantly occurring, and proofs abundant might be adduced to show that innocent persons have frequently been made to suffer the penalty of death judicially, instead of the guilty, simply through an error of this nature. Should an alleged child, or other claimant present his claim to an inheritance, he must first establish his identity before taking further steps in the suit. Is an individual assaulted, or robbed? he will be required to identify his assailant before he can successfully prosecute him. Or, again, a person, after many years' absence in foreign climes, returns home to claim his rightful property or title, but he is so changed as to be unrecognized by his nearest relatives; he must be able to prove his identity before the courts, before his claim can be sustained. And then, in relation to persons found dead—whether in cases of recent death, where the body has undergone but little change, or years after the decease, where nothing remains of the body from which to glean the important information but the bare skeleton—the question of
personal identity acquires the most intense interest, more especially in a trial for murder, where it becomes essential to establish the identity of the alleged victim as the *corpus delicti*.

It is true that the aid of the physician is not so frequently invoked for proving the identity of the *living*, since this can generally be established as satisfactorily by friends and neighbors, as by medical men. Still, there may be occasions of unusual complexity, in which a professional opinion may become requisite, as, for example, to verify certain deformities, fractures, scars, and other marks about the person, when these constitute the evidences on which the identification may be dependent.

The subject will be considered under the two divisions of (1) The Identity of the Living, and (2) The Identity of the Dead.

I. *The Identity of the Living*.—This may usually be established by the direct evidence of witnesses who have known the individual sufficiently long to have a distinct recollection of his personal appearance; such is the testimony of relatives, friends and acquaintances. Although among the myriads of the human family it is very rare to find two persons exactly alike in all points, yet remarkable instances do occasionally occur where the personal resemblance is so striking as to baffle even the skill of the detective; and this resemblance has been made still stronger by the existence of similar marks, cicatrices, or certain peculiarities of structure, in both individuals. Some striking illustrations might be given of the extreme difficulty—amounting, at times, to an impossibility—of deciding the question, which also go to show how easily witnesses may be mistaken in their evidence on this subject. Only two will be here referred to.
In the year 1560 the celebrated case of Martin Guerre and Armand du Tilh was tried before the Parliament of Toulouse. Martin had been absent from his home for eight years, when the person named du Tilh appeared, and represented himself as the long absent man. So strong was the resemblance, that his statement was universally accepted by all of Guerre's family, including his wife, four sisters and two brothers-in-law, among whom he lived unsuspected for three years. About this time, however, something occurred to excite suspicions as to the true character of the supposed husband and brother, when he was arrested, and brought before the tribunal, on a charge of fraud. Upon his examination he gave satisfactory answers to the most minute questions in relation to Guerre's former life. Some one hundred and fifty witnesses were examined during the investigation, of whom between thirty and forty testified, from a life-long acquaintance, that the prisoner was Martin Guerre; while about the same number swore positively that he was Armand du Tilh, whom they well knew; and over sixty, who knew them both, declared that they were unable to say which the prisoner was. Finally, however, the real Martin appeared upon the scene, when immediately he was recognized. The four sisters who had previously testified that du Tilh was their real brother, now admitted their error, and acknowledged the distinction. There being now no doubt of the guilt of the prisoner, he was condemned, and afterwards executed. (Wharton and Stillé's Med. Jurisp. Vol. II, p. 1092.)

The other instance is afforded in the recent famous Tichborne case, in which a person named Orton, with various aliases, undertook to personate an English baronet, heir to a large entailed estate. So successful was his scheme that "he was sworn to be Sir Roger Tichborne by eighty-
five witnesses, among whom were Sir Roger's mother, the family solicitor, one baronet, six magistrates, one general, three colonels, one major, two captains, thirty-two non-commissioned officers and privates of the army, four clergy-men, seven tenants of the Tichborne estates, and seventeen servants of the family." The claimant also gave proof of "a fish-hook wound on the eye, of a mark of bleeding on the ankle, and of a peculiar scar on the head," all of which the genuine Sir Roger possessed. The case, however, broke down on cross-examination, many circumstances being proven against the claimant, which need not be here enumerated. Suffice it to say that a verdict was taken against him, and that an indictment was since found against him for perjury.

Now, as a fair inference from the above two instances, and other remarkable cases, we may assume, that appearances are not conclusive evidences of personal identity; and, as a sufficient reason for this we must admit the fact that "a large proportion of ordinary persons are very untrustworthy witnesses to identity, when dependent on appearances alone. They are, from nature or habit, incapable of appreciating form, and form alone is the unerring proof of personal identity. The difficulties in the way of identification, more especially of the dead, are to them insuperable" (Lond. Spectator). To this inherent difficulty on the part of the witnesses, may be added, their want of previous training as minute observers; and also, the well-known fact of the adroitness of criminals at personal disguisement.

A second means of establishing the identity of the living, especially in a criminal, is by certain peculiarities in the appearance, which are noticed at the time of the commission of the crime, and which are, therefore, apt to leave a strong impression on the senses,—such as (a) size, when the indi-
individual is very tall or very short, very corpulent or very slim; whether lame, or otherwise deformed; (b) dress, when a portion—sometimes a mere shred—of the prisoner's dress is discovered near the seat of the crime, which exactly corresponds with the rest of the garment found on his person, or in his own house.

A third means of identification is afforded by the voice. Peculiarity of the voice (such as depth or shrillness, lisping or stammering) always makes a strong impression upon those who hear it, and constitutes a valuable aid in personal identification.

Fourthly, the presence of certain peculiar marks, either natural or acquired, about the person, often affords material aid in establishing identity. These marks comprise moles, nævi, scars, cicatrices, deformities, fractures, tattoo-marks, etc. Such marks are usually well known, and remembered by relatives and friends of the individual, who can usually identify them. Some of these remain upon the body during life; others gradually decline and fade away. In relation to tattoo-marks, Prof. Casper's experience leads to the inference that some of them (the red ones) are gradually obliterated by time, while the black and purple ones are more permanent. A cicatrix is permanent during life, if there has been any original loss of substance. It may not always be distinguished from the surrounding skin, unless the part be smartly rubbed, when the white scar is immediately manifested on the red surrounding surface. Caution should be given against too strong a reliance upon scars as a means of identity, since these may, at times, be discovered upon another, precisely similar, both as to form and situation.

Under this head may be mentioned the appearance of the hands (whether hard and horny, or soft and pliant, or
whether stained in a peculiar manner), as often indicating the nature of the occupation of the individual.

Photographs and other portraits of the suspected person are sometimes useful aids in the identification of the living, as well as of the dead; but caution is requisite here, since the art of the photographer in the touching up of the picture frequently makes it an unfaithful representative of the negative.

As bearing upon this subject, it may be proper to say a few words upon vision and hearing. The following remarks are abridged from Woodman and Tidy's Forensic Medicine. The limits of normal vision or healthy sight, unassisted by instruments, in a perfectly clear atmosphere, are as follows:

<table>
<thead>
<tr>
<th>Height (feet)</th>
<th>Range of distance (miles)</th>
</tr>
</thead>
<tbody>
<tr>
<td>5</td>
<td>2.96</td>
</tr>
<tr>
<td>20</td>
<td>5.91</td>
</tr>
<tr>
<td>50</td>
<td>9.35</td>
</tr>
<tr>
<td>100</td>
<td>13.2</td>
</tr>
<tr>
<td>500</td>
<td>29.5</td>
</tr>
<tr>
<td>1000</td>
<td>41.8</td>
</tr>
<tr>
<td>5000</td>
<td>94</td>
</tr>
</tbody>
</table>

It follows from this, that a man of ordinary height may be seen on level ground at a distance of two or three miles, on a clear day; but this is very different from recognition of the person, so as to identify him. The effects of age upon the acuteness of vision is considered by Dr. De Guéret to be as follows:

At fifty years it is diminished one-fifth; at sixty years one-fourth; at seventy years, one-third; at eighty years, one-half. In other words, if a man of thirty or forty could distinguish an object at one hundred feet distance, at sixty years of age he could not recognize it further off than seventy-five feet, or, at eighty years, at fifty feet.

The recognition of persons at a nearer or greater distance, is afforded by their stature, gait, complexion, color of the hair
and eyes, and peculiarities of appearance. According to the above authority, the best-known persons can be recognized often with difficulty, in broad daylight, at one hundred metres, or about one hundred and nine yards. Less known persons may be recognized, in broad daylight, at sixty to one hundred yards; and people who are almost strangers, and who have no personal peculiarities, at twenty-seven to thirty-three yards. By the clearest moonlight, the best-known persons cannot be recognized further off than sixteen or seventeen yards. By starlight, recognition cannot be effected beyond ten to thirteen feet. The light of a flash of lightning enabled a lady, on her passage home from India, to see distinctly the features of a man who was robbing her trunk in the cabin of a vessel, on a very dark night; and authentic instances are given where, by the flash of a pistol or gun, sufficient light was momentarily afforded to enable not only an assailant to be recognized, but likewise the color and appearance of his horse. The subjective sensation of flashes of light or sparks, produced by a blow upon the eyeball, has no effect whatever in aiding recognition; since the same sensation is often experienced by those who are totally blind.

The distance at which sounds (such as the report of a gun or pistol) continue to be audible cannot be determined with accuracy, since it depends upon the direction of the wind, the condition of the atmosphere as to moisture, and other disturbing sounds. The velocity of sound may be stated to be, on an average, 1135 feet per second, which is about 13 miles a minute, or one mile in about 4½ seconds.

The Identification of the Dead.—This may have reference (1) to the body recently dead; and (2) when the soft parts have disappeared by putrefaction, and the skeleton only remains, or where detached bones merely have been discovered.

When the death has but recently occurred, and the body is unmutilated, most of the same general methods of establishing identity are available as have already been mentioned in the case of the living,—such as the testimony of relatives and acquaintances as to the personal appearance of the deceased, certain marks upon the person, as nævi, moles, cicatrices, tattoo-marks, fractures, deformities, etc. Photographs and other portraits are here also admissible, although by no means reliable proofs.

If the body has been subjected to mutilation after death, and the severed portions removed to a distance from one another, and some of them even destroyed, as is sometimes done by a murderer with a view to escape detection, the difficulty of identification is, of course, very much increased. Nevertheless, if the disconnected parts can be recovered, or even a portion of them, it will always be possible for a skilled anatomist so to readjust them, as to build up again the body, so to speak, by making the proper allowance for the missing parts, and comparing these with other average specimens of a similar kind. Several striking examples of this character are given in the books. One of these is the well-known case of Dr. Parkman, who was murdered by Dr. Webster, in Boston, Mass., about thirty years ago.
After the death of his victim, Dr. Webster attempted to destroy all evidences of the deed by cutting up the body into fragments, some of which were burned in a grate, some immersed in chemicals and others packed away in boxes in distant parts of the building. On the discovery of these remains, a week after the murder, the portions of the body were accurately examined by a skilled anatomist. It was proved that they were human remains, belonging to one and the same body; of the male sex; and that they had not been dissected for anatomical purposes, but cut and hacked in different directions, for the object, evidently, of mutilation. On restoring these disjointed parts *in situ*, and supplying the deficient portions, it was found that the proper measurements agreed closely with those of the missing Dr. Parkman. This circumstance, together with the discovery of certain marks of identity about the teeth and jaws (the head had been almost completely destroyed by fire), afforded sufficient evidence of the personal identity of the missing gentleman to enable the jury, on the trial of Dr. Webster, to find a verdict of guilty. Another instance of a somewhat similar nature is recorded by Professor Taylor, in his Medical Jurisprudence. A number of years ago a murder was committed in London, on the river Thames, and shortly afterward a package containing mutilated human remains was discovered on one of the abutments of Waterloo bridge. The murderer had, no doubt, intended to throw the bundle into the river, but it had lodged on the projection in its descent. Dr. Taylor was requested to examine and identify these mutilated remains; and when, after great difficulty, the parts were brought together and found to fit, the body was identified as that of a man who had recently disappeared from a vessel on the river—a Swedish sailor.

When the question of identity relates to the skeleton
merely, or only to portions thereof, the answer cannot be always satisfactory, and the medical jurist has need of much caution and reserve before giving a positive opinion.

The very first thing for him to determine is, whether the bones submitted to his inspection are human bones, or those of some of the inferior animals. Doubtless, if the entire skeleton be discovered, there need be no uncertainty about the matter; but if only a single bone or two be found, a mistake may easily be made, except by a practiced anatomist and osteologist. Indeed, many ludicrous blunders are recorded, of persons of otherwise good medical education mistaking the bones of the ox, horse, dog, pig and goat for those of the human subject. But may not something be learned by the aid of chemistry, or the microscope? The reply must be, generally, in the negative. Certainly the bones of the aged do contain more calcareous matter than those of the young, and consequently present a somewhat different appearance under the microscope. But human bones have the same general chemical composition as those of the lower animals. It is also true that the bone-cells or corpuscles vary somewhat in size in the different orders of animals, being largest in reptiles, smallest in birds and mammals, and intermediate in fishes. In this respect there is an analogy with the size of the blood-corpuscles in these different orders. But these are only generalizations, and would be of little practical use in individual instances. To be sure, the microscope will enable us to determine the fact of any specimen submitted being bone, or not, by the presence or absence of the bone-cells; but it can go no further, inasmuch as it cannot distinguish the bone-cell of a man from that of a mouse, or of an elephant.

If the skull is the only portion of the skeleton submitted for examination, there can usually be no difficulty in recog-
nizing it as human; the only doubt that might arise would be the possibility of its belonging to one of the higher order of (anthropoid) apes; but even here, there are important differences which would not be overlooked by one skilled in comparative anatomy and osteology. The further question, whether from the examination of a skull simply, it is possible to decide to what race the individual belonged—Caucasian or otherwise—we do not think can be answered with absolute certainty. Doubtless, well-marked typical skulls may be identified as belonging to some particular race, e.g., the negro, or Caucasian; but we must remember that the points of distinction, which in well-marked specimens serve to separate these, shade away in many instances, so as to make it extremely difficult, if not impossible, to give a medico-legal opinion in an isolated case.

Another important point is to ascertain whether all the bones submitted for inspection belong to one and the same skeleton. The mere fact of their being discovered together does by no means necessarily prove it, since they might have been so placed either accidentally, or with the design of eluding detection of some crime.

In the identification of the dead by means of the skeleton, or by detached bones, the three leading points to determine are (1) the age, (2) the sex, and (3) the stature.

I. The Age.—This can generally, in young subjects, be pretty accurately determined by the development of the teeth, and by the progress of ossification in the different bones. In the skeletons of new-born children, and before the teeth have appeared, it may become important for the medical jurist to be able to decide upon the age, in order either to rebut or confirm a charge of infanticide. It is authoritatively stated that in the jaws of a child at full term
there will always be found the rudiments of twenty-four teeth—twenty primary teeth, and four permanent molars. Hence, if only the jaws of an infant be discovered, medical evidence of its probable age may be given. The average date of the eruption (cutting) of the teeth is, according to Mr. Bell, as follows: The four central incisors appear from five to eight months after birth; the four lateral incisors, from seven to ten months; the four anterior molars, from twelve to sixteen months; the four cuspidati, from fourteen to twenty months; and the four posterior molars from eighteen months to three years. Between six and seven years the jaws contain forty-eight teeth—twenty temporary ones in a perfect state of development, and twenty-eight permanent ones imperfectly developed, and placed behind the temporary teeth, which they are to replace. According to Mr. Saunders, the order in which the permanent teeth make their appearance is as follows: At seven years, the four anterior molars; at eight years, the four central incisors; at nine years, the four lateral incisors; at ten years, the four anterior bicuspids; at eleven, the four posterior bicuspids; at twelve to twelve and a half years, the four cuspid; and at thirteen to fourteen years, the four second molars—making the whole number of permanent teeth at this period to be twenty-eight. The four remaining (posterior molars) teeth—called dentes sapientiae—do not usually appear until eighteen to twenty-one years of age. As a rule, the teeth of the lower jaw are cut first, but there are many exceptions; nor must it be forgotten that irregularities often occur as to the order of their appearance. The above description is intended to apply only to the average cases.

Let us now take one or two examples to illustrate the medico-legal application of the foregoing rules. Suppose the skull of a child was discovered, in the jaws of which
were twelve permanent teeth—eight incisors and four molars, we should decide the age to be about nine years. If the jaws contained twenty-four permanent teeth—eight incisors, four molars, eight bicuspids and four cuspid, we should conclude the age to be about thirteen years; and so on. It is proper here to remark, that there are two diseases which affect the growth of the teeth, viz., rickets and syphilis. In a rickety child, the first teeth do not usually appear until after the twelfth month, whereas in cases of congenital syphilis, the teeth appear prematurely—before the sixth month; but they present a peculiar notched appearance; and they are apt to be brittle and to crumble away easily.

The progress of ossification in the different bones of the skeleton affords an additional test of its age, especially in early life. According to Béclard, the degree of ossification in the lower epiphysis of the femur affords the most certain criterion of the age of the foetus, and of the new-born child. Thus, if no ossific deposit can be seen in this cartilaginous epiphysis, it is certain that the foetus has not attained to the eighth month of uterine life. If the osseous deposit is as large as a poppy-seed, it is probably in the ninth month of fetal existence; and if it has acquired the diameter of a line and a quarter, to one and a half, it has reached the full period. If the point of ossification measures three lines or more, it may be assumed that the child had survived its birth some little time.

The (average) length of the skeleton of a new-born child is about sixteen inches. At the end of the first year, ossification has commenced at the extremities of most of the long bones; and this progressively advances from year to year, until the whole process is completed; and the epiphyses of all the long bones are united to their shafts at full
maturity, which, in the male, may be considered to be twenty-four years, and in the female, twenty-two years. After this period, or when ossification is once completed, it is difficult to determine the precise age by an examination of the bones of the skeleton. It should, however, be remembered that the different bones of the sternum do not unite until about the fortieth, or forty-fifth year; and union between the sacrum and os coccygis is not usually completed until fifty-five, or sixty years of age.

In old age, the bones become lighter in weight, and more brittle, from the loss of animal matter. They are also darker in color; and the flat bones become thinner, from the absorption of their diploë. In the skull of the aged, the sutures are more or less obliterated; and the remaining teeth present a worn appearance, and a yellowish color. If the teeth have been lost (as is usually the case, at least in this country), the alveolar processes become absorbed, and the lower jaw undergoes a well-marked change in its appearance, consisting of the widening of the angle at its neck, and the shortening of the vertical diameter of its body, or width, which imparts the characteristic senile expression to the mouth of the aged. The discovery of such a jawbone would positively determine the age to be about seventy years, or over.

The presence or absence of certain teeth in the head has frequently been the means of determining the identity of the body. So also, the presence of artificial teeth, with their mechanical appendages, has at times furnished the strongest corroborative evidence in such identification, as in the celebrated Parkman-Webster case, already alluded to, in which the artificial teeth, discovered undestroyed by the fire in the grate, where the head had been burnt up, were positively identified by the dentist, who had manufactured
and fitted them some years before. So, likewise, the remains of the Marchioness of Salisbury, discovered among the burnt ruins of Hatfield House, were identified by the jaw-bone having gold appendages for artificial teeth (Guy). The importance of the teeth as a means of identification is shown in the case of the late French Prince Imperial, whose body had been so much disfigured by his assailants, that its identification would have been extremely difficult but for certain peculiarities about his teeth (Taylor, *Prin. and Prac. Med. Jurisp.*, 1884).

II. *The Sex.*—This can usually be determined from the skeleton, if entire, without much difficulty. The general appearance of the male and female skeleton presents many well defined points of difference, which need not here be enumerated, as they are described in all anatomical works. Suffice it to say that the corresponding bones of the two differ in size, weight, strength, and prominence of their ridges and protuberances which mark the points for the insertion of muscles. There are also certain recognized differences in the head and thorax; but it is in the *pelvis* that the most characteristic distinctions are observed. The male pelvis is narrower and deeper than that of the female. In the latter, the *ossa illii* are more spread out, and flatter, which renders the superior part of the pelvis more capacious; the sacrum is broader, and turned more backward; the arch of the pubis is much wider. The greatest diameter is the bilateral; whereas in the male, the antero-posterior is the greater. The foramen ovale is triangular in the female; in the male, it is more oval. Owing to the greater breadth of the female pelvis, the acetabula are farther apart than in the male. It is to be understood that these peculiarities in the female pelvis are not exhibited until the period of
puberty, and subsequently. From a fragment of a bone, merely, it would certainly be hazardous to undertake to determine the sex; and the medical jurist should exercise much reserve in giving an opinion in such a case.

III. The Stature.—If the whole skeleton has been preserved, and none of the ends of the long bones have been lost by decay, the original height may be calculated with tolerable accuracy by arranging the bones in situ, and adding an inch and a half, to two inches, to the entire length of the skeleton, to supply the loss of the soft parts. But even here perfect accuracy cannot be attained, chiefly on account of variations in the curve of the spinal column in different individuals. Dr. Dwight* assumes, as the result of numerous observations, that the total height of the intervertebral cartilages is 25.6 per cent. of the entire length of the spine. As a collateral aid in estimating the stature, we may regard as correct the generally accepted rule, that the top of the symphysis of the pubes is about the centre of the body in average women; while in men, the centre is a little below the symphysis.

The attempt has frequently been made to estimate the height of the body from a study of the individual long bones of the skeleton; but no reliance can be placed upon such comparisons, inasmuch as there is considerable variation in the length of these bones in skeletons of the same stature. The so-called "rules of proportion" of certain writers cannot be regarded as by any means certain, or authoritative. In case the skull is wanting, the rule laid down by Dr. Gould is "to find the height of the spine of the seventh cervical vertebra from the ground, and add to

this 9.95 inches, which is the average height from this point to the top of the head."* M. de St. Luca (Cosmos, October 2d, 1863, quoted by Professor Taylor) states that an approximative estimate of the stature may be had by measuring the length of the third phalanx of the middle finger, thus: this phalanx is equal in length to one-fourth that of the whole finger, or one-eighth that of the hand, including the carpus. The arm may be divided into five parts, of which two are included in the humerus, two in the fore-arm, and one in the hand. The total length of the hand is, therefore, one-fifth that of the arm. Double the length of the arm (or the two arms stretched out horizontally), added to the length of the two clavicles, together with the transverse diameter of the sternum, is equivalent to the whole length of the body. In applying this rule to practice, however, we must not forget that the length of the hand, and especially that of the fingers, varies materially in persons of the same height; and so trifling a variation in the third phalanx of the middle finger as the one-thirty-second of an inch would, according to this method of calculation, figure up as great a difference in the total result, for the height of the whole body, as two and a half inches.

The existence of fractures, deformities and callus in a skeleton sometimes affords valuable aid in its identification, even many years after death. In relation to the production of callus, it is well understood that this substance is the result of the reparative inflammation of bones, and that its presence on a bone is a certain indication that some time must have elapsed between the injury and the death of the individual. On the other hand, the total absence of callus in a fractured bone, indicating that no time had been given for the process of repair, would be very good evidence that

* Ibid.
the injury was the immediate precursor of death, and if on
the skull, the probable cause of death. An instructive illus-
tration of this is given by Professor Taylor (Med. Jurisp.)
in the case of an Englishman who was tried in India for the
murder of a native, who had been beaten by the former with
a stick, with the allegation that his rib had been broken,
thereby causing his death. To substantiate this charge, a
skeleton was produced which had been dug up three months
subsequent to the decease, which was almost completely
denuded of flesh; the bones clean and dry; one rib frac-
tured, with a deposit of callus around the fracture. The
identity of these bones with those of the missing man was
attempted to be established by the prosecution, but unsuc-
cessfully, in consequence of their dry and denuded state—a
condition altogether incompatible with so short a period of
time as three months since death. Moreover, the amount
of callus thrown out made it evident that more than a week
must have elapsed before death took place, which event was
alleged to have occurred immediately after the injury.

Other notable instances might be mentioned of the iden-
tification of the skeleton by means of the above-mentioned
marks, or peculiarities, and even where it was possible to
determine the actual cause of the violent death. In the
year 1823, a soldier living in the south of France suddenly
disappeared, under suspicious circumstances. Two years,
however, elapsed before any investigation was instituted by
the proper authorities. Some human bones were then dis-
covered in digging in the garden of the deceased soldier.
Of course, it became necessary to identify these remains. It
was remembered that the deceased had a singular personal
deformity, in possessing a sixth finger on the right hand, and
a sixth toe on the left foot. On examination, it was ascer-
tained that the fifth metacarpal bone of the right hand was
shorter and broader than the corresponding bone of the other hand, and further, that there were two articulating surfaces on its digital end, indicating clearly the existence of a supernumerary finger. In the same way, the fifth metatarsal bone of the left foot showed two distinct articulating faces on its digital extremity, indicating the existence of a supernumerary toe. Besides this, the age, sex and stature of the skeleton corresponded with those of the missing man. But even further than this, a close inspection of the skull revealed the distinct marks of a depressed and radiated fracture of the temporal bone, which showed no sign of reparation by the formation of callus. Evidently, then, death had occurred very soon after the fracture of the skull, and in all probability, as the direct result of violence. Upon this evidence, the suspected parties were tried and executed, having previously confessed their crime.

Sometimes, on the exhumation of bones, the medico-legal question arises—how long have they been buried? It is quite impossible to give more than an approximative reply to this question, after all the soft parts have disappeared, which commonly requires about ten years, on an average. In a dry soil, bones will resist decomposition for thirty or forty years after burial. As this process progresses, they become lighter in weight, in consequence of the loss of animal matter, and the color externally grows darker. The ends gradually become brittle, and crumble away, and finally the shaft of the bone undergoes a similar disintegration, the mineral matter alone remaining unaltered, and constituting the "dust" to which the animal body must eventually be reduced. Devergie states that the bones of King Dagobert were found in a state of tolerable preservation, enclosed in a leaden coffin and sarcophagus, at St. Denis, after the lapse of twelve hundred years; and Dr. Taylor mentions that the
skeleton of William Rufus was found in a stone coffin at Winchester, nearly perfect, after seven hundred and eighty years' burial. The bones of Abelard and Héloïse were so well preserved, that after a lapse of five hundred years the female skeleton could readily be distinguished from the male.

Even if the bones have undergone calcination, as when a body has been burned with a view of destroying its identity, especially in cases of infanticide, it may still be possible to determine whether the remains are human, provided the bones preserve their proper form, and have not been reduced to powder. In the latter case, although a chemical analysis of the ash might detect the calcium phosphate, this would not solve the mooted question, since the ash of human and animal bone is chemically identical.

Other means of personal identification are afforded by a microscopic examination of the hair, and the fibres of various sorts of fabrics, such as, cotton, linen, wool and silk. Human hair discovered on a weapon, along with blood stains, affords strong presumptive evidence of murder, or violence. So also, fibres of cotton, or of other material, found on weapons supposed to have caused death, or else on the person of the accused, suggest a strong suspicion, if these fibres correspond to the clothes of the deceased. Thus, a case is mentioned by Prof. Taylor, where the discovery of some cotton fibres, accompanied by a blood-stain, upon the edge of a razor, found near a woman whose throat had been cut while in bed, led to the subsequent detection of the murderer. In the same manner, the discovery of a few hairs upon the handle of a knife, on which also were marks of blood, enabled a London microscopist to declare that these hairs were squirrel hairs; which circumstance further led to the identification of the murderess of a child, whose throat had
been cut with a knife, which, in the death wound, had passed through a victorine made of squirrel fur, worn around the child's neck.

In case of rape the examination of the hair about the female genitals will be likely to show the presence of seminal spots, and consequently of spermatozoa, which cling to them with great tenacity.

In all cases, except when hairs are to be examined for spermatozoa, they should be washed in warm water, and then thoroughly dried, afterwards steeped in turpentine, and finally mounted in Canada balsam. They should then be examined with a magnifying power of about 200 diameters. To examine hairs for spermatozoa, moisten first of all with a drop of ammonia solution, and examine under a microscope after the liquid has evaporated (Tidy).

For the identification of hairs, human or other, it is desirable to have at hand specimens of various kinds of these, properly mounted, for comparison. Hairs resist putrefaction for an indefinite length of time, which fact aids greatly in their examination for medico-legal purposes. It should also be remembered that hair is affected differently by different reagents. Strong alkalies dissolve it; acids roughen it; alcohol causes it to look clearer; chlorine water bleaches, and rots it.

The size of hairs from different parts of the human body, as well as from different individuals, varies considerably; thus, the hairs from the head are finer than the eyelashes, but coarser than the hairs from the arm. There is also considerable difference in the size of the hairs of the various lower animals. The shape and microscopical appearance of human and other hair are figured in some of the larger works.

The main medico-legal questions connected with the
identification of hairs are: (1) Is the hair human, and from what part of the body? (2) Does it correspond with the hair of the murderer, or of the victim? (3) Has its color been naturally, or artificially changed? It should be remembered that gray hair is not unfrequently found on comparatively young persons, and that undoubted instances have occurred of the sudden bleaching of the hair through fright or grief. As regards the artificial coloring of the hair, it is well known that this is one of the means of disguise most commonly adopted by criminals, in order to elude detection.

The common hair dyes for coloring light or red hair black or brown, are composed of the salts of lead, silver, or bismuth. Hair thus colored may easily be detected by soaking it in nitric acid, which dissolves out the mineral, which may then be identified by the appropriate tests. It is more difficult to bleach or whiten the hair, than to darken it. This is usually effected by first washing it in an alkali, to remove the greasy matter, and then soaking it in chlorine water, which will lighten its tint in a few hours; but, at the same time, it will render it very brittle, and impart its peculiar odor to it.

In all artificially colored hair, the fraud can be detected by closely watching the new growth, which will be of a different color from the other portions; and also by chemical tests.

The fibres of cotton, linen, wool and silk all present well-marked differences, when viewed under the microscope. The cotton fibre is in the form of a flattened band, with thickened borders, and is spiral, or twisted upon itself. Linen consists of round fibres, having a firm consistency, with jointed transverse markings at unequal distances, somewhat resembling those on the India cane, and taper-
ing to a point. *Silk* fibre has the appearance of straight, well-defined cylinders, free from all markings, and refracting light powerfully. *Wool* fibre is irregular, wavy, and of unequal thickness. The fibres of *hemp* resemble those of flax (linen), but are coarser; and when boiled in nitric acid they exhibit no spiral streaks, but swell and become brittle.

The identification of *blood stains* and *seminal spots* will be treated of later.
CHAPTER VI.
THE CAUSES PRODUCING VIOLENT DEATH.

These may be considered under the following heads:—

I. Wounds, including Burns.        VI. Lightning.
II. Suffocation.                    VII. Heat and Cold.
III. Strangulation.                 VIII. Starvation.
IV. Hanging.                       IX. Poisoning.
V. Drowning.

SECTION I.

VIOLENT DEATH FROM WOUNDS.

Definition of a wound.—Danger of.—Examination of the body.—Results of the injury.—Absence of external marks of violence.—Wounds made before and after death.—Hemorrhage.—Ecchymoses.—Classification.—Homicidal, suicidal and accidental wounds.

The surgical and the legal definition of a wound are not identical. The former idea of the term is "a solution of continuity of the soft parts, occasioned by external violence." According to this meaning, there must be a rupture of the skin, or the mucous membrane, to constitute a wound. But this would evidently exclude internal injuries, such as rupture of the liver, spleen, or heart, fractures and luxations unaccompanied by external lesion; hence, the legal definition of a wound is more comprehensive; it embraces all injuries of the body, whether external or internal, with or without a solution of continuity of the skin, produced suddenly by external, or mechanical violence. The latter meaning of the term wound is evidently its proper medico-legal application, although it may not strictly accord with the surgical definition.
A distinction is sometimes made between mortal and non-mortal wounds, or between wounds dangerous and not dangerous to life, and the medical witness is asked to give his opinion on this subject. But he should be guarded in his answer, since it is well known that many wounds at first considered as comparatively trivial, subsequently assume a dangerous, and even fatal character. Of course, in many cases there can be no difficulty in pronouncing upon the dangerous or mortal character of a wound, as, for instance, if the heart or the great vessels have been wounded, in compound fracture of the skull, in wounds of the internal viscera, etc. The danger of a wound, it may be remarked, depends upon a variety of circumstances, all of which should be considered, such as its position; its locality in relation to the great vessels and nerves; the kind of weapon by which it was inflicted; the amount of hemorrhage; the age, constitution and general health of the subject; the circumstances (favorable or unfavorable) for treatment, and other considerations, all of which must be taken into account as important factors in the prognosis. Medical testimony is not usually required, except in case of a fatal termination. An exception to this may, however, occur in the case of an assault, where the character of the injury (whether dangerous or trivial) might decide as to the propriety of accepting bail for the prisoner.

In case of death from a wound, the medical examiner should never theorize as to the manner of its causing the death; and he should give his opinion only after a very careful post-mortem examination of the body. Moreover, this examination should not be confined simply to the wounded portion of the body, but all the cavities and organs should be inspected, since it might be affirmed that a natural cause of death might have existed in that very part which
was neglected by the examiner. Such neglect has often been the means of securing the release of the prisoner, inasmuch as it occasioned a doubt as to the real cause of death, in the minds of the jury. It may even be proper to examine the stomach for poison in all doubtful cases; as shown by the oft quoted instance related by Wildberg, of the girl who was beaten by her father for stealing, and who died shortly afterwards, apparently from the effects of the blows, but in whose stomach a considerable quantity of arsenic was found. She had swallowed the poison soon after committing the theft, fearing her father's anger. The man was discharged. In a similar manner, it sometimes happens that a person, after taking poison with suicidal intent, may destroy himself by another means, as by a gunshot wound, by drowning, or by throwing himself from a window or a precipice.

The examination of the wound includes the observation of its situation, extent and direction; the presence or absence of effused blood, whether liquid or coagulated, and the presence of ecchymoses; the condition of the edges of the wound, whether everted or not; whether adhesion has commenced; the presence of granulation, inflammation, suppuration, or gangrene; whether it was inflicted before, or after death; whether there was loss of substance; hernia of the intestinal organs, etc. There should also be an inspection of the clothes of the deceased, to ascertain if the rents or stabs in these correspond with the wounds of the body; and if a weapon be discovered, it should be carefully compared with the wound.

It sometimes happens, in cases of severe injury, that death has resulted from internal lesions, with few or no external marks to indicate them. According to Casper, this is of frequent occurrence in severe internal lacerations occasioned by violence. He cites a case of this character.
A wagoner, in guiding his team with a loaded wagon down a hill, was accidentally crushed against a tree on the road. He was found dead the next morning. The only external injuries were a slight abrasion upon the left arm, and one upon the right temple. On opening the body, however, the most striking evidences of violence were discovered. From the spinal canal, about a quart of blood escaped. The spinous processes of the first thoracic vertebrae were broken off. The left pleural cavity contained about thirty ounces of fluid blood. The pericardium was torn completely across, and the heart, severed from its large vessels, lay almost entirely loose in the cavity of the thorax. The open ends of the aorta and pulmonary artery were distinctly visible. The left lung was entirely torn through its middle portion; and in the right lobe of the liver was a laceration two inches long and half an inch deep (Gericht. Med. 1, 122).

The distinction between wounds made before and after death should be carefully noticed. Wounds inflicted before death may be recognized by the following signs: (1) Incised wounds exhibit everted edges, arising from the elasticity of the skin and subjacent muscles, with considerable hemorrhage, usually of an arterial character; the spots of arterial blood which have spouted on neighboring surfaces are of a peculiar comet-like shape. Coagula are more or less abundant in the wound, and around it. The surrounding tissues are more or less infiltrated with blood. If some days have elapsed before death, evidences of vital reaction will be shown, such as partial healing, granulation, suppuration, or sloughing. If the wound was made immediately after death—within a few minutes—there may be some retraction of the skin, and some slight bleeding, with few or no coagula, which are of loose texture. There is little or no staining of the surrounding tissues, and never any
attempt at repair. If the wound be made ten or twelve hours after death, there will be no eversion of its edges, no hemorrhage, except of a slight venous character, and no surrounding infiltration. The experiments of Professor Taylor and Mr. Aston Key upon amputated limbs confirm the above description. The amount of hemorrhage accompanying an incised wound affords a pretty good criterion as to whether it was inflicted before, or after death. Comparatively little bleeding accompanies wounds made after death, and this is chiefly venous; the arteries yield little or none, while in the living, the hemorrhage is chiefly arterial. In a case of murder reported by Casper, as also in the case of Greenacre, in England in 1837, where the head of the victim was severed from the body, the fact that the head was completely drained of blood led to the conclusion that the decapitation had been done during life, and that there must then have been a copious hemorrhage to account for the absence of the blood after death.

(2) In lacerated and contused wounds, the distinction is not so obvious as in incised wounds. Lacerations are not always accompanied by bleeding, but there will always be more or less coagula present; and if the person survives a few days there will be evidences of vital reaction, such as suppuration and granulation, sloughing or gangrene, all of which are absent in such wounds inflicted after death.

Contused wounds made during life are chiefly distinguished by the amount of effused blood in the cellular tissue under the skin (ecchymosis). This arises from the rupture of small vessels, and is manifested by the well-known "black and blue" discoloration produced. If the effusion of blood is rapid, the spot is of a dark red, at first; if slower, the discoloration is deep blue, or violet. In some cases of even violent contusion, there may be no appearance of external
ecchymoses. Again, it is not always manifested immediately over the seat of the contusion, but at a little distance from it, especially if the surrounding tissue is loose. Familiar illustrations of this are afforded in the case of a blow over the eye, producing an ecchymosis of the lower lid; and of a blow over the lower portion of the abdomen being attended with ecchymosis of the scrotum. The presence of ecchymoses, then, in cases of contused wounds, may be regarded as pretty good evidence of the ante-mortem character of the injury, while its absence is not necessarily an indication that the wound was post-mortem. The experiments of Sir R. Christison upon the dead body go to show that if the contusion be made very soon after death, and while the body is still warm, the resulting appearances strongly resemble those produced by ante-mortem contusion; with this difference, however, that the effusion is usually immediately beneath the skin, and not in the areolar tissue; also, that there is an absence of coagula, and of swelling.

Ecchymosis is usually superficial, and may appear very shortly after the injury; or it may be deep-seated, and not visible at all. In some instances it is not manifested until after death, as in the case of a man who died from rupture of the bladder, resulting from the kick of a horse, thirty-five hours after the injury. No discoloration of the abdomen was observed until after his death. Neither can the quantity of blood effused, nor the extent of the injury be always estimated by the amount of the discoloration. This is well illustrated in the case of the wagoner who was crushed to death, as mentioned by Casper, and which was alluded to above.

Another important fact relative to ecchymoses is the change of color which accompanies them, since this may serve to indicate the probable date of the contusion. In
about twenty-four hours the blue or livid margin of the
bruise becomes lighter, or of a violet color, which gradually
changes to green and yellow. During these alterations of
color, the spot may become larger, but the central portion
remains always darker than the margins. These changes
of color are believed to be due to a dilution of the serum
of the blood by the fluid of the cellular membrane, and its
gradual dispersion throughout the cells. It is finally ab-
sorbed, and the color entirely disappears. In general, it
shows itself within twelve hours after the contusion; the
violet color within three days; the green from the fifth to
sixth day; the yellow from the eighth to tenth day; and in
healthy persons, the complete disappearance of the spot
occurs from the twelfth to fourteenth day. The changes
are more rapid in the young than in the old, and depend
also on the degree of the contusion. The above changes of
color never appear in contusions on the dead, which cir-
cumstance constitutes another diagnostic mark.

It is also important not to mistake the ecchymosis pro-
ceeding from natural causes, such as scurvy, petechiae
and purpura, from that occasioned by blows. The former
may usually be distinguished by being confined to the
superficial layers of the skin, and by their presence also on
the internal mucous membranes, together with the absence
of swelling, and the fluidity of the blood.

According to Devergie, ecchymoses are often concealed
on the bodies of the drowned, when first they are removed
from the water, owing to the sodden state of the skin; they
may become apparent only after the body has been exposed
for some days, and the water has evaporated.

(3) In punctured and penetrating wounds, the diagnosis
between those inflicted before, and those produced after
death, is usually not difficult. The former are attended with
more or less hemorrhage, and often exhibit signs of vital and reparative reaction, such as inflammation and suppuration, or gangrene. The latter are destitute of all these. For example, a stab made into the left ventricle of the heart after death is followed by no hemorrhage.

It is not always possible for a medical witness to state positively that a wound was caused by a particular weapon; but it is desirable, if possible, to establish the relation of the injury with its supposed cause; thus, an incised wound would naturally be referred to a cutting weapon; a penetrating wound to a pointed one; and a contused wound to a blunt instrument. But caution should be observed in giving an opinion on this subject, especially in case of contused wounds.

Incised wounds are characterized by the regularity and evenness of the cut. This usually serves to distinguish them from wounds made by glass and crockery ware, or nails, which are generally irregular and uneven. But in some instances, the cuts produced by broken glass or china exactly resemble incised wounds. In stabs, the shape of the wound may often indicate the character of the weapon, whether double-edged or not. But where the weapon has penetrated obliquely through the tissues, and when these have been stretched, the shape of the wound will not exhibit this correspondence. So also, a wound made in parts where the skin is wrinkled may suggest the idea of several distinct wounds, as in the neck. It must not be overlooked that superficial incised wounds may give rise to dangerous, or even fatal hemorrhage. And also, that it is not always possible, in such cases, to determine the direction of the incision, i.e., whether made from right to left, or the reverse. And yet, as remarked by Casper, this fact might have a most important medico-legal significance in determining the ques-
tion whether the wound, as in cutting the throat, was homicidal or self-inflicted. The attendant circumstances, however, might throw some light upon it, such as the presence of blood on the right or left hand, or cuts on certain parts of the clothing of the deceased.

Lacerated and contused wounds do not afford the same facility for identifying the weapon as incised wounds. From simply inspecting them, the medical witness will not generally be able to indicate the precise weapon, or cause. He may, indeed, be able to say that it was not produced by a cutting instrument. But a blow made by a blunt weapon upon the skull, or over the zygoma, may give rise to a cut which strongly resembles an incised wound, though, as a rule, the division of the parts is not as straight and regular as in the latter, and the angles of the wound are less acute. Moreover, in the contused wounds there is more or less swelling, and extravasation of blood into the adjoining parts; and, at times, the existence of irregular fracture and internal hemorrhage.

In the case of a fatal contused wound of the head, it has been judicially decided that it makes no difference as to the guilt of the accused, whether he produced the death of his victim by a direct blow upon the head, or indirectly, by causing him to fall upon a stone, or other hard substance, which produced the fracture, or contusion.

As before mentioned, rupture of the internal organs—the liver, spleen, heart, lungs and kidneys—is a frequent result of contusions. Fracture of the base of the skull is sometimes caused by severe contusion of the head. Wharton and Stillé (Med. Jurisp., Book V, p. 660) allude to the fact that spontaneous wounds sometimes occur in the labia and vagina of pregnant women, which might give rise to suspicion of assault. Also, that in such women accidents of
different kinds are frequently attended with profuse hemorrhage from the pudenda.

It is evident from what has been said in reference to the difficulty of always connecting a contused wound with the precise instrument that caused it, that the witness should avoid committing himself upon the question. In some instances, however, the shape of the contused wound, especially a depressed fracture of the skull, will enable us to come to a correct conclusion on the subject. Some years ago the author was called upon, as an expert, to testify as to the probable cause of a depressed fracture of the temporal bone of a man who had been struck during a mêlée. The question was, whether the injury had been inflicted by the fist merely (as was alleged), or by an instrument like a loaded cane, or billy. There was good reason for believing that the latter instrument, in the hands of another person, was the real cause of his death, owing to the private confession of a comrade of the prisoner. The fractured bone was produced in court. The depression was well-marked, a quarter of an inch deep, exactly corresponding to the loaded end of the billy; no radiating fissures (as would most probably have resulted from a blow of the fist). Although the opinion of the author, founded on the above facts, was, that the fatal blow had not been inflicted by a fist, but by a billy, two physicians on the other side thought differently; and the judge dexterously solved the mooted question by asserting that, in such cases, one expert was about as good as another; and inasmuch as, in the present trial there were two against one, he would decide in favor of the majority! and so he did, and the prisoner (possibly an innocent man) was convicted, and sent to the penitentiary for five years.

The examination of the clothes of the deceased constitutes
an important part of the legal physician’s duty, as this may throw light upon the mode in which the wound had been made, from the character of the cuts, or stabs observed upon them. So likewise, marks of blood, dirt, grass, or other substances, on the clothing may afford valuable indications in the same direction. Contused wounds by bludgeons may, however, occasion considerable laceration of the muscles, or even severe fractures, without tearing the dress.

Professor Taylor (Med. Jurisp.) mentions an instructive case, showing the importance of comparing the articles of dress with the injuries which may have proved fatal. A woman, aged sixty, was found one morning dead in her bed. She had been seen in her usual health on the previous night. On inspection, there were found two indentations in the right parietal bone, and a large clot of blood in this situation, beneath the skin, together with a fracture of the bone, four inches in extent. Beneath the bone, on the dura mater, were found nearly three ounces of clotted blood. On the evening before her death, she had been suddenly knocked down on the public road, by a man accidentally running against her. She fell heavily on the back of her head, appeared stunned, was raised upon her feet, and, after swallowing some brandy, recovered sufficiently to walk home, a mile and a half, and eat her supper. She was found next morning dead in bed. There was a suspicion of murder, in this case, against a fellow lodger; but when the bonnet worn by the woman was produced at the inquest, two indentations were discovered on the back part of it, corresponding to those on the skull of the deceased. The indentations on the bonnet, moreover, contained dust and dirt, thereby confirming the statement of witnesses who had seen her fall, and rendering it highly probable that this fall was the real cause of the fatal fracture, and effusion of blood.
It also illustrates the well-known fact that a person may receive a fracture of the skull ending in effusion, which may not prove fatal for many hours after the accident, and which may not have prevented the individual from walking a considerable distance after the injury.

Was the wound *homicidal*, *suicidal*, or *accidental*? This important medico-legal question cannot always be settled by medical testimony alone, though there are many points in which it is of the greatest aid to the legal authorities. These are as follows:

1. **The Situation of the Wound.**—Suicidal wounds are usually inflicted upon the most accessible parts of the body, such as the head, neck, breast and abdomen. If by firearms, the part usually selected is the head (mouth, forehead or temple), or over the heart; if by a cutting instrument, the throat or heart. The discovery, therefore, of wounds on a part of the body difficult to reach by the individual himself, as the back, would certainly not be suggestive of suicide. But an exception must be made here as regards the insane, who are well known to destroy themselves by self-inflicted wounds of the most extraordinary character, on the back of the head and neck; by striking the head against some solid substance; or precipitating themselves from a height. An insane person has been known to shoot himself with a pistol fired from behind the ear. The situation of the wound is, therefore, only suggestive of its origin, since it is quite possible that an assassin might inflict a death wound upon his victim in such a situation designedly, in order to deceive, and thus elude the suspicion of homicide. Accidental wounds are usually met with on exposed parts of the body.

2. **Nature and Extent of the Wound.**—Suicide is rarely inflicted by contused wounds, but usually by incised or
penetrating ones. Exceptions occur, as when a person throws himself out of a window, or from a height; and in some remarkable instances of self-destruction, by butting the head against a wall, and subsequently chopping it with a hatchet. In the case of the insane, there is no accounting for the variety in the nature, and extent of the wounds inflicted for the purpose of self-destruction. This fact ought to be remembered, since, if the bodies of such persons should afterward be discovered, and nothing be known of their previous histories, serious errors in relation to the real origin of the wounds might result.

Incised Wounds of the Throat are usually regarded as indicating suicide; but it is well-known that murderers frequently destroy their victims by cutting their throats. As to the extent of the wound, it is commonly supposed that a suicidal incision of the throat does not reach as deeply as a homicidal one of the same character; but instances are not wanting where a determined suicide has severed the throat down to the vertebrae. Again, irregularity in the cut of the throat has been deemed by some as indicating homicide rather than suicide, under the idea of resistance on the part of the victim; but it is evident that the irregularity might have resulted equally from nervousness, or indecision in inflicting the wound, on the part of the deceased.

The nature and extent of the wound or injury may serve to distinguish accident from homicide. Thus, if numerous wounds or bruises are discovered in opposite sides of a dead body, the presumption would be in favor of homicide; and when the accused attempts to ascribe the death of his victim to a fall, the nature of the wounds might be such as positively to contradict his assertions.

3. Direction of the Wound.—This will often enable us to distinguish between a homicidal and an accidental wound,
rather than to decide upon its suicidal character. Thus, if death has occurred from a stab, inflicted downward from the upper part of the thorax, and penetrating the heart, and it was attempted on the part of the prisoner to show that the wound had been accidentally occasioned by the deceased falling, while drunk, downward upon the knife which the prisoner had held in his hand sloping upward, the direction of the wound would prove the falsity of the statement. Two other cases are here quoted from Wharton and Stille's *Med. Jurisp.*, of a similar character. One of a man discovered dead, with a deeply-punctured wound of the neck, which, on examination, showed that the weapon had been partially turned and withdrawn, and again plunged into the neck in a different direction, after the manner of the German butchers. This circumstance proved not only that the death was not accidental, nor probably suicidal, but indicated the occupation of the murderer. The other occurred in England, some years since, where a murder was fixed upon a man from the fact that the wound in the neck of the deceased had been evidently made by a knife cutting from within outward, as is done in slaughtering sheep.

In most suicidal wounds of the throat, it is found that the cut has been made from left to right; in punctured wounds the direction is commonly from right to left, and downward. In left-handed persons, the direction would, of course, be the reverse. These facts, however, can only afford moderate presumptive evidence, since it is obvious that a murderer might inflict an incised wound in the throat of his victim from behind, which would exactly resemble that made by the suicide. In all such doubtful cases, particular attention should be directed to the surrounding circumstances, such as the position of the body, and the weapon, the presence
or absence of blood upon the hands and person of the deceased, etc. If the death has been very sudden, from hemorrhage (in a case of suicide), the weapon will most probably have fallen from the hand, on account of the relaxation of the muscles; but if it has been caused by a pistol, the weapon may be found tightly grasped in the hand of the deceased. If the throat has been cut suicidally, blood will be found on one or other of the hands; but if homicidally, and no resistance has been made, the hands will probably be unstained. As regards the position of the body, if the death be very sudden, from loss of blood, the body will be found lying on the back; if less sudden, the face and trunk will be turned toward the ground. If the body be found upon the back, in death from hemorrhage, and the weapon at a distance from it, the act was, in all probability, homicidal.

The position of the weapon in relation to the dead body, although at times strongly suggestive, can never afford absolute evidence as regards the question of homicide or suicide. Thus, Professor Casper mentions the case of a man who cut his throat with a razor, which was found, bloody, and closed, two feet distant from the body. Also, of another suicide by a pistol-shot in the breast, where the pistol was found in the pocket of the deceased, who afterwards terminated his life by drowning himself (Gericht, p. 17).

From what has been said above, it is manifest that the medical jurist can rarely venture to give a positive opinion as to the homicidal, suicidal, or accidental cause of death, apart from a consideration of the circumstances accompanying it. These circumstances constantly vary in almost every case, and they require the utmost experience and tact on the part of the medical examiner to recognize and apply
them in each individual instance. Some of them have already been alluded to: they include the position of the body and the weapon; the condition of the ground where the wound was inflicted; the presence of footprints, of man or horse; the condition of the clothing of the deceased; the condition of the hands, whether showing wounds, or cuts on their palms (indicating resistance), or the hands holding portions of hair or fragments of the assailant's clothes; the adherence of certain fibres to a weapon, such as cotton, woolen, linen, silk or fur; marks of blood upon clothing or furniture; state of the mouth and throat; marks of blood or other matters on the person of the assailant; rifling of the pockets, and tearing of the dress, etc. These cannot be farther enlarged upon here, but their medico-legal importance cannot be too strongly insisted upon.

SECTION II.

GUNSHOT WOUNDS.

DIFFER FROM OTHER WOUNDS.—DEFLECTION OF THE BALL.—WOUNDS MADE BY SHOT, WADDING AND POWDER.—CAUSE OF DEATH IN WOUNDS.—WOUNDS OF THE HEAD, NECK, SPINE, CHEST, ABDOMEN.

Gunshot wounds differ from other wounds chiefly in the fact that the vitality of the part struck is lost, and that there is a consequent slough, or loss of substance. They are essentially contusions. They are dangerous to life on account of their involving vital portions of the body, death occurring either from hemorrhage, or from shock to the nervous system. The hemorrhage is seldom great, except when large vessels are wounded. Often, from the form of the wound, there may be but little external bleeding, while a fatal internal hemorrhage may be going on. They differ much in appearance, according to the distance from which the piece was fired, and the nature of the projectiles. If the
explosion occurs in close contact with the body, the wound is large and circular, the skin denuded, blackened and burned by the half consumed grains of powder. The hair and clothes also in the vicinity of the wound are more or less scorched. The entrance orifice of the ball is livid and depressed, and is larger than the point of exit. When the piece is fired from a distance, the blackened and burned appearance of the skin is not seen, but only the mark of the entrance of the missile, and sometimes that of the exit. The aperture of entrance of the ball when fired from a distance is, according to most authorities, always smaller than that of exit. Nélaton says that when the wound is recent, the entrance orifice is depressed and contused, while the exit aperture is lacerated and everted. In the former, there is an actual loss of substance; in the latter, there is merely a solution of continuity. After some days, however, the contused margins of the entrance wound slough away, thereby enlarging the orifice, while those of the exit partially adhere, causing the latter wound to appear smaller than the former. Professor Casper declares that the entrance aperture is always the larger. Very possibly, this discrepancy of views may arise from not distinguishing between the early and the later stages of the two orifices. If the ball enters a very fat portion of the body, this often protrudes between the edges of the wound, and completely changes its appearance. Again, the character of the entrance will depend very much upon the nature of the projectile, and its velocity, as well as the distance from which it was fired. If the ball is conoidal, as in the minie rifle, and traveling with great speed, the wound is linear, and resembles a puncture, producing little external harm, but causing very considerable internal injury. A rifle ball makes a large and ragged wound, caused by the spiral
direction given to the missile. It is evident that several wounds may be made by a single ball, as this may chance to traverse different parts of the body and limbs. It may also happen that the piece may have been loaded with two or more balls, which may account for the number of the wounds.

The deflection of a ball from its straight, or direct course after entering the body is easily produced by its striking obliquely against any resisting surface, such as a bone, tendon, aponeurosis, or even muscle. In this way it often happens that a ball, striking the chest or abdomen, may be caused to pass almost entirely around the body, and afterwards be extracted close by the entrance point. Wharton and Stillé (Med. Jursp.) relate the case of a German student who was wounded in a duel by a pistol ball striking him on the larynx obliquely, and passing around the neck so as to lodge on the opposite side of the thyroid cartilage. It was thence removed by simply cutting through the skin. It is not uncommon for a ball to travel half way around the chest or abdomen, and lodge in the back, giving the appearance of having passed directly through the lungs, or intestines.

If the wound be caused by a load of shot, its appearance will depend chiefly on the distance from which it was discharged. If fired very near the body, so as to enter it as a single charge before separating, it will produce a single large and ragged wound, much contused and blackened by the powder; and as the shot diverge after entering the body, there will be considerable laceration of the parts beneath. For the opening to be single, the experiments of Dr. Lachèse, of Antwerp, have shown that the charge should not be fired at a greater distance than ten to twelve inches. When the distance is so extended as to allow the scattering of the shot, each grain will make its own individual wound.
It is quite possible for a single shot to cause a mortal wound, as when it happens to strike the heart, or aorta.

Wounds made by the wadding and gunpowder alone may prove serious or fatal, according to the distance of the piece from the body. A pistol thus loaded, at twelve inches distance tore the clothes, and abraded the skin without penetrating it; at half this distance, the wadding penetrated to the depth of half an inch; at two inches it entered to the depth of two inches, causing a ragged and blackened wound; and at one and a half inches, the wadding entered the thorax between the ribs, and in one experiment, carried away a portion of the rib (Phil. Med. Exam., 1846). Taylor mentions an instance of a man sitting in a gallery of a theatre at Brighton, in 1881, who had the upper half of his hand completely blown away by a piece of greased newspaper, tightly rammed, discharged from a small cannon on the stage of the theatre.

Even gunpowder alone is capable of producing very serious wounds, if fired close to an exposed part of the body. The wound will present a lacerated appearance, and be blackened and burned by the partially consumed powder. If the grains of powder be coarse, the wound may have the appearance of having been caused by very small shot.

The question of the homicidal, suicidal, or accidental character of gunshot wounds must generally be settled by the appearance of the wounds, and also by the surrounding circumstances. Thus, if it be on the forehead or temple, in the mouth or over the heart, and if it be blackened and lacerated (indicating the close proximity of the weapon), it may be regarded as a suicidal act. If, on the contrary, the wound be on the back, or side of the head (except in the
case of the insane), or of the body, without the blackened and lacerated appearance above alluded to, it may be considered as the act of a homicide. *Accidental* gunshot wounds bear the marks of near wounds, as they are mostly the result of the accidental discharge of the piece, either in the hands of the deceased at the time, or else in close proximity to his person.

Out of 368 cases of suicide by firearms, 297 were from wounds in the head; of these, 234 were fired into the mouth; only 71 were from wounds inflicted on the chest or abdomen (M. de Boismont, *Du Suicide*, p. 531).

**Cause of Death from Wounds.**—In a medico-legal case it may become important to ascertain the real *cause of death* occasioned by a wound—whether *immediate*, as from hemorrhage or shock, or *remote*, resulting from subsequent complications. In a trial for murder, this question might have an important bearing on the result.

When the death is directly traceable to hemorrhage, its rapidity depends upon the amount and suddenness of the bleeding; and this again is dependent on the size and nature of the vessel wounded. Exhaustion follows much more rapidly from a sudden hemorrhage than from a more copious flow of blood if gradually lost. Again, arterial hemorrhage is more rapidly fatal than venous. It should also be remembered that some persons have a constitutional tendency to bleed very easily, from the slightest superficial wound. Such a tendency is termed a *hemorrhagic diathesis*; this is sometimes hereditary; and where it exists, it exposes the individual to great danger, in case of being wounded. Age and disease also increase the danger of death by hemorrhage from wounds.

*Internal* hemorrhage, as the result of a wound, is often as
fatal as the external; the danger is here further increased by the pressure exerted by the effused blood upon a vital organ, such as the brain, as is witnessed in effusion of blood within the cranium, produced by a fracture of the skull. It is also exemplified in a wound of the intercostal arteries, causing effusion of blood into the chest, and producing fatal pressure on the lungs; and also in wounds of the throat resulting in asphyxia, from the flow of the blood into the windpipe.

*Shock* is the result of a violent impression made on the great nervous centres. It often is the immediate cause of death, after a severe injury, without leaving behind any trace or lesion discoverable on a post-mortem examination. Shock is most apt to follow extensive lacerations of the body, such as result from machinery or railway accidents, or from extensive burns.

The *remote* causes of death from wounds are numerous and varied. The following may be regarded as the most common:

1. *Tetanus or lockjaw.*—This is generally the result of lacerated and punctured wounds, and especially if inflicted on nerves, tendons, aponeuroses and fibrous tissues. A very slight wound in these structures may be followed by fatal tetanus. Tetanus is always a very serious complication, and is mostly fatal. It does not usually appear before the seventh day after the receipt of the wound, though sometimes earlier; and it rarely supervenes after the twentieth day.

2. *Erysipelas* is another complication of wounds, which may give to them a fatal issue. It is particularly apt to accompany wounds of the scalp; and it sometimes assumes an epidemic character, especially in hospitals, where it may occasion great mortality among the wounded patients.
3. *Hospital gangrene* is another occasional result of wounds. It likewise often proves fatal, and may assume an epidemic type. It is, however, rarely seen, except in military hospitals, and seems to be connected with faulty hygienic arrangements.

4. *Surgical interference, including the Use of Anaesthetics.*—In wounds dangerous to life, the question of the propriety of a surgical operation becomes paramount; the patient will certainly die without the operation, and, on the other hand, he may die from shock, as the immediate result of the operation. The question of the legal responsibility of the death then becomes a serious one, and in the event of a trial, the counsel for the prisoner, who had originally inflicted the wound, will endeavor to show that the death was not really the result of the wound, but was rather owing to the surgical operation. Whatever plausibility there may be in such an argument, it would not likely avail with an intelligent court and jury, unless it could be proved that the original wound was not of a dangerous character; and, secondly, that the surgical interference was unwarrantable, and unskillfully employed. The same remarks will apply to the use of anaesthetics (ether and chloroform) in the performance of surgical operations. Their employment in such cases has now become so universal throughout the civilized world, that the occasional fatal results attending their administration should be regarded as exceptions to the universal rule of safety accompanying their employment, and as in no wise inculpating the attending surgeon; consequently, the fatal result that might happen to follow their use should not be considered as offering any extenuation for the prisoner, if the latter has inflicted a dangerous or fatal wound upon the deceased. The only medico-legal point at issue would be—was the administration of the anaesthetic a necessary and
proper part of the treatment, and was it skillfully administered?

It will be proper to devote a brief consideration to the subject of *Wounds in different regions of the body*, inasmuch as these present certain individual peculiarities, which give to them special medico-legal importance.

*Wounds of the head.*—Scalp wounds are not usually attended with danger, except sometimes, when followed by erysipelas, and when the blow has been so severe as to produce concussion of the brain. It must not be forgotten that fracture of the skull may exist without any wound of the scalp; also that fatal effusion of blood upon the brain may be produced by a blow on the head, without causing either a wound of the scalp, or a fracture of the skull.

*Concussion of the brain* may result either from a direct blow upon the head, or from a violent fall upon the feet or buttocks. Sometimes death ensues immediately from concussion, leaving behind it no perceptible lesion, though, doubtless, some molecular change has been caused in the nerve cells, not recognizable by the microscope. Such fatal concussion may occur without either fracture of the skull, or even a wound of the scalp. The symptoms of concussion are faintness, nausea and vomiting, pallor of face, feeble pulse, loss of consciousness, either partial or complete, with subsequent confusion of ideas, and tendency to sleep. Concussion may be confounded with intoxication, compression of the brain, opium poisoning, sunstroke, etc.

It is particularly important, for medico-legal reasons, to distinguish between concussion and intoxication. Doubtless, many cases of supposed drunkards, arrested in large cities at night by the police, and left unattended in the station house till morning, are, in reality, cases of concussion, or compression of the brain, which may prove fatal, simply for want of
timely relief. What adds greatly to the difficulty of the diagnosis is the fact that the two conditions are so frequently coincident in the same individual. It is the drunken man who is most apt to engage in a brawl which results in a broken head. Generally, the history of the case (if it can be obtained), and the odor of the breath, will afford us the best means of diagnosis. In intoxication, the temperature is usually below 96° F.—sometimes below 90°; the loss of power and of sensation are not unilateral, as in compression; the bladder is generally full of limpid urine, which will furnish evidence of the presence of alcohol on distillation. (Vid. Alcohol, post.) The pupils are sometimes contracted, and again dilated.

Fracture of the skull is the result either of a direct blow upon the head, or of a fall upon the head, striking a stone, or other hard body. The usual consequence of such a fracture is pressure on the brain by the depressed bone, or by the extravasated blood from a ruptured vessel, or vessels. Fracture of the base of the skull is the most dangerous of all. It is nearly always fatal; and unless carefully looked for in the autopsy, it may entirely escape notice.

Compression of the brain may result either from effusion of blood or serum upon, or within the brain, with or without fracture, or depression of the bone; also from suppuration, or tumors in the brain, from congestion of the cerebral vessels, and likewise from narcotic poisoning. The symptoms are essentially those of apoplexy, viz.: loss of consciousness, paralysis (usually hemiplegia), dilated pupils (except where the effusion is on the pons Varolii, when, according to Dr. Wilks, the pupils are contracted), stertorous breathing, a full, slow pulse, and coma. It is important to remember that the effusion of blood resulting from a blow may be very gradual, so that the person seemingly

ANOTHER IMPORTANT MEDICO-LEGAL POINT IS, THAT A FATAL EFFUSION OF BLOOD MAY TAKE PLACE SIMPLY FROM GREAT EXCITEMENT, IN A QUARREL, ESPECIALLY IF ACCOMPANIED BY INTOXICATION. IN A TRIAL FOR HOMICIDE THIS IS LIKELY TO BE URGED BY THE DEFENCE AS THE PROBABLE CAUSE OF DEATH IN THE DECEASED, WHERE THERE HAS BEEN AN ASSAULT OR PUGILISTIC ENCOUNTER, WHICH TERMINATES FATA LLY. IN A CASE OF THIS NATURE IT MIGHT BE EXTREMELY DIFFICULT TO DECIDE HOW FAR THE FATAL EFFUSION WAS DUE TO NATURAL CAUSES, SUCH ASATHEROMA OF THE CEREBRAL ARTERIES (WHICH, IN AN HABITUAL SPIRIT-DRINKER, MIGHT ALSO BE CONNECTED WITH A DISEASED LIVER AND KIDNEYS), OR HOW FAR IT WAS TO BE ATTRIBUTABLE TO THE EFFECTS OF VIOLENCE. WE ARE, HOWEVER, OF THE OPINION THAT, IF THE ASSAULT COULD BE CLEARLY PROVEN, EITHER IN CONNECTION WITH A DIRECT BLOW UPON THE HEAD, OR INDIRECTLY, BY A FALL UPON A STONE OR OTHER HARD BODY, THE MERE FACT OF THE PREEXISTING DISEASE OF THE ARTERIES, OR THE OTHER ORGANS, WOULD NOT EXCULPATE THE PRISONER, NOR ACQUIT HIM OF THE CHARGE OF HOMICIDE. IF, HOWEVER, THE AUTOPSY SHOWS THAT THE EFFUSED CLOT, OR SERUM, WERE OF OLDER DATE THAN THE ALLEGED INJURY, THIS WOULD CERTAINLY BE A STRONG ARGUMENT FOR HIS ACQUITTAL.

WOUNDS OF THE SUBSTANCE OF THE BRAIN ARE NOT ALWAYS FATAL.
As regards the *symptoms* of injury to the different portions of this organ, as indicating the localization of its various functions, the reader must be referred to the writers on this special department of science. It is well known that considerable portions of the cerebral substance have escaped through the skull, after fractures, not only without loss of life, but without any sensible impairment of the mental powers.

*Wounds of the Face* are not usually dangerous unless they involve the orbit; a penetrating wound of this part may readily reach the brain, with a fatal result. So also, a severe blow upon the nose may so injure the ethmoid bone as subsequently to involve the brain.

*Wounds of the Neck* are attended with much danger, owing to the presence of the large vessels and nerves. In cut throats, the great danger arises from the sudden and profuse hemorrhage. The section of the larynx and trachea is not necessarily fatal, the chief danger arising from suffocation from the flowing back of the blood. A division of the *esophagus* is almost necessarily fatal, chiefly because of its involving the section of the great vessels of the neck.

*Wounds of the Spine* are dangerous in proportion to the degree that the spinal marrow is involved. In concussion of the spine, death sometimes takes place instantly. If the spinal cord be wounded high up, above the region of the phrenic nerve, the function of respiration is immediately arrested, and death ensues. Wherever the injury occurs to the spinal cord, it is understood that there is a complete suspension of the functions of the parts below. In *fracture of the vertebrae*, the great danger arises from pressure on the spinal marrow. Sudden death has been produced by the spontaneous luxation of the second cervical vertebra, arising from the fracture of the odontoid process, through dis-
ease. Sir A. Cooper's case was of this character (Frac. and Disloc. p. 463). These fractures are justly considered as having an important medico-legal bearing.

Wounds of the Chest.—The great danger here lies in the hemorrhage from the heart, great vessels and lungs; hence, such wounds often prove rapidly fatal. The hemorrhage in wounds of the chest is nearly always internal. Wounds of the lungs, though they may not prove immediately fatal, frequently so terminate after a lapse of time; this is especially true of gunshot wounds, if the bullet or other foreign substance happens to be retained. Wounds of the Heart nearly always terminate fatally and rapidly, if the cavities be penetrated. Gunshot wounds of the heart do not necessarily cause immediate death; cases are recorded where the patient survived several months; after death the ball has been found in the substance of the organ. Even where the cavities of the heart have been perforated by a cutting instrument, there have been instances where the person survived for eleven days (Wharton and Stillé, Med. Jurisp. p. 745). Rupture of the heart may be the result of a violent blow upon the thorax, or it may follow any intense excitement or emotion, if this organ happens to be in a diseased condition, as in fatty degeneration, etc. In a medico-legal case, where death has resulted from this cause in a brawl, in which the deceased received a severe blow on the chest, if the pre-existing disease of the organ can be established, it would be a question how far the violence, and how far the disease was to be credited with the result. The case is very similar to the one where death follows a blow upon the head, terminating in compression of the brain, and where a diseased condition of the cerebral vessels existed.

Wounds of the Abdomen.—Even a superficial wound of the abdomen may prove fatal, by dividing the epigastric
artery. A severe blow upon the epigastric region has frequently produced immediate death, from shock upon the solar plexus of nerves. Blows upon any part of the abdomen may be followed by peritoncal inflammation, which often proves fatal. Penetrating wounds may terminate fatally from the same cause. Wounds of the stomach and intestines are exceedingly dangerous, and are often mortal, either from hemorrhage, or from inflammation, or both.

Wounds of the Liver are dangerous, according to their extent and depth. If the gall-bladder is involved, death is apt to ensue, in consequence of the peritonitis. The danger from wounds of the kidneys arises from the effusion of urine, and the consequent inflammation.

In relation to wounds of the bladder, it should be remembered that this organ may be ruptured spontaneously, from over-distention. It is sometimes ruptured by a blow, or kick of a horse, upon the lower part of the abdomen. In a trial for homicide, in a case of death produced by rupture of the bladder, the defence would probably try to set up the plea of spontaneous rupture of this organ. Frequently, there is no external injury to indicate the true nature of the case, the autopsy alone revealing it, and disclosing, also, extensive peritoneal inflammation, resulting from the escape of urine.

Wounds of the Genital Organs are, in the male, usually self-inflicted, and they are met with most generally among the insane. They comprise castration—more or less complete, and amputation of the penis—partial or entire. The danger to life is great in proportion to the hemorrhage, and injury to the organs. In certain other cases, where the injury has been inflicted by others, and when in a state of erection, the urethra has been found violently torn across, and
the corpora cavernosa and spongiosa divided. In females, the chief point of medico-legal interest is to discriminate between wounds of the genitals inflicted by another, and spontaneous hemorrhages from a ruptured vein in the labia. Here, of course, a rigid inspection of the injured parts will be required before arriving at a definite conclusion.
CHAPTER VII.

EXAMINATION OF BLOOD STAINS.


The identification of blood-stains not infrequently constitutes a most important link in the chain of evidence, in a trial for homicide. It is a very common practice for a murderer to attribute certain suspicious red stains discovered upon his garments, or implements, to the blood of some domestic animal or bird. Within a few years past the resources of science have afforded us material aid in distinguishing human blood-stains from those of the inferior animals, so that the legal physician may now feel much more confident in delivering his testimony in a trial for homicide, than he could have done in former years.

The appearance of blood-stains to the naked eye will vary in size, shape and color. Sometimes it may be a mere film or smear, but generally it presents the form of distinct spots of different sizes; and if the blood has spurted obliquely upon a surface, the spots will have assumed a comet-like shape, terminating in a bulbous tail. The color of the stain will depend (1) upon its freshness: if recent, it will have a bright red hue; if old, the color will be brownish, or brown-red. (2) Upon its thickness; being darker in proportion to the density of the stain. (3) Upon the material on which it has fallen; if the latter is porous, as soft wood, or linen or cotton fabrics, the tint will be rather dull, but if on polished and hard substances, such as metals or polished wood, the
spots have a darker and shining appearance, and on drying they are apt to crack from the centre, and may thus easily be removed. When dried upon linen or cotton, they usually have a stiffened feel, like a spot of dried albumen or gum. If the stains be upon a colored substance, they can best be distinguished by artificial light; indeed, they may be entirely invisible in bright daylight.

We possess three methods of identifying blood stains: (1) the chemical; (2) the microscopic; (3) the spectroscopic or optical. But previously to employing these methods, it will be always proper to examine the suspected spot with a good magnifier; the spot, if a blood stain, will frequently exhibit minute coagula or clots of a shiny hue, intermixed with the fibres of the material on which it is fixed.

I. The Chemical Tests.—Before noticing these, it will be proper to remark briefly on the solubility of the coloring matter of blood. Modern research has shown that the coloring matter of blood, when quite recent (haemoglobin or oxy-haemoglobin), is very soluble in cold water, but when old, so as to have changed to a brown color, it is converted into haematin, or deoxidized haemoglobin, which is insoluble in water. This is a fact of considerable medico-legal interest. For if a garment, or other article stained with blood, is immediately washed in cold water, the whole of the blood will probably be discharged, so as to leave no trace of it behind. But if (as is usually the case) the garment be kept for some time before the attempt is made to remove the stain by washing, the soluble haemoglobin will have become more or less connected with the insoluble haematin, and enough of the blood will remain upon the article to suffice for future identification. Hot water will not remove
a recent blood stain as effectually as cold water, on account of the action of the heat upon the hæmoglobin.

If the blood-spot be recent, the examiner should cut out a small piece of the fabric stained, and suspend it, by means of a thread, in a test-tube containing cold, distilled water. In a few minutes the coloring matter will be observed to separate from the material, and to descend to the bottom of the water, forming a bright-red solution. If the stain is a little older, more time will be required to effect the solution, which will have a browner hue; and if the stain is very old, there will be no solution whatever.

If the stain be upon a porous substance, such as wood, brick, etc., it should be cut or scraped out, reduced to powder, and then soaked in cold water for some hours, and afterwards filtered. If the spot be upon a hard metallic surface, as a knife, sword, etc., it should be carefully dried, when it will be apt to crack off; otherwise it may be scraped off with a knife, and the scraping soaked for some time in cold water, and afterwards filtered. If the solution should not be complete, a little dilute ammonia may be added, and if this should fail, Dr. Tidy recommends to use a trace of citric acid to effect the solution.

Having procured the clear red solution, the next step is to heat it in a test tube over a spirit lamp. Four results are thus obtained: (1) the red color disappears; (2) coagulation takes place; (3) a brownish-green precipitate is formed. If there is a sufficient quantity of this precipitate it may be collected, dried and heated with a weak ammonia solution, which will dissolve it. The solution will appear dark green by reflected, and red by transmitted light. (4) A weak solution of ammonia added to it, either produces no change of color, or it merely intensifies it. It never changes it to green or crimson, as it does with cochineal, and red fruit
colors. *Tincture of galls* gives a red precipitate to the original solution. A solution of *chlorine* causes no change in it.

The above tests will suffice to distinguish blood from all other red solutions, such as cochineal, kino, madder, logwood, and the various red fruit juices, none of which coagulate by heat, and *all* of which are changed in color by the addition of ammonia. The stain produced by lemon or orange juice on the blade of a knife (citrate of iron) after exposure to the air, may bear some resemblance to an old blood stain; but the test of tincture of galls, or of tannin, to the solution would immediately detect the difference. So, the stain from red paint (which contains iron), or from iron mould, is easily identified by their solubility in dilute muriatic acid, and by subsequently testing for iron.

**The Guaiacum Test.**—This constitutes a beautiful and satisfactory portion of the chemical test for blood. Dr. Day, of Australia, has experimented extensively upon this test, and Prof. Taylor has fully confirmed his results. It depends upon the following conditions: A freshly-prepared tincture of guaiacum, if dropped into water, precipitates the resin, which, if exposed to the air, gradually acquires a bluish color. If it be exposed to a jar of oxygen gas, the blueing process is more rapid; and if brought in contact with ozone, the blue color is instantly produced. Hence, the blueing must be owing to oxidation. But it is a remarkable fact, as discovered by Schönbein, that *autozone*, as found in the peroxide of hydrogen (in which the oxygen is in the positive state), has no effect at all in changing the guaiac resin to a blue color. Moreover, while the resin is blued by a variety of mineral and organic substances, the coloring matter of the blood has no effect upon it. The *guaiacum test* depends, then, upon the fact that while the blood has no
power to oxidize, or blue the resin, the presence of peroxide of hydrogen (antozone), which itself has no power to oxidize the guaiacum, causes the resin then to be oxidized by the blood, and the blue color appears. According to Prof. Taylor, an excellent way of showing the experiment is to add a few drops of the tincture (freshly prepared) to a small quantity of water; this will precipitate the resin. Divide the water suspending the resin into two portions; into one of them pour a little solution of red coloring matter of blood; to the other, add a few drops of ozonized ether (peroxide of hydrogen dissolved in ether); no change of color is observed in either portion. Now, to the first portion add a few drops of the ether, and to the second a few drops of red solution; in both cases the sapphire-blue color will soon be seen. In case the solution is turbid, from an excess of the resin, the addition of a few drops of alcohol will instantly clear it, and bring out the fine blue color distinctly. If the simple addition of the blood solution to the guaiac produces a blue color, we may be certain that some oxidizing substance is present besides blood, and which conceals its presence. The force of the experiment consists in the fact that blood, of itself, will not blue guaiacum, but in the presence of ozonized ether, the blue color is speedily produced.

Objections have been raised against this test, on the ground that other substances beside blood will produce a blue color in the presence of guaiac and peroxide of hydrogen, such as saliva, bile and red wine; but as regards the two former, their color should at once distinguish them from blood, while the latter substance requires some hours' exposure to produce the same result; whereas, in the case of blood the effect is immediate. This test is as available for old, as for fresh blood, for concentrated, or diluted blood;
hence, for a washed-out blood stain,—wherever, in fact, a particle of red coloring matter remains. If no blueing occurs in the presence of the guaiac and the peroxide, it will be safe to affirm that there is no blood present. In an old blood-stain, or where it is too small to afford a sufficient solution, or where there may be some doubt of its presence on a colored material, a very good plan is to moisten the spot first with a few drops of water, then with a sufficient quantity of the guaiac tincture, and afterwards with a few drops of the ozonic ether, and then press upon it a piece of white tissue or filtering paper; immediately there will appear upon the paper the characteristic blue stain. A number of such impressions may thus be taken from one spot, by simply adding a little more of the guaiac and the peroxide, and repeating the pressure upon the paper.

The chemical tests will not distinguish arterial from venous blood, nor human blood from that of the lower animals. The statement of M. Barruel, that if blood be shaken up with one-third its volume of pure sulphuric acid, a peculiar odorous principle will be evolved, resembling the particular animal from which the blood was obtained, has been disproved by subsequent investigations.

II. The Microscopic Test.—This consists in the identification of the blood corpuscles—especially the red ones—by means of the microscope. To effect this, the stain (unless too old) should be cut out and placed on the glass, or on a watch crystal, and moistened with a few drops of pure cold water,* a glass rod being pressed against it, to effect the separation; then cover the specimen over with a thin glass,

*As water alone may cause the corpuscles to swell up, it should be mixed with one-seventh part of glycerine, or with a small portion of common salt, or sulphate of sodium.
and examine with a one-fourth inch power, and measure the corpuscles with a micrometer. If the stain has been washed, very possibly there will be no satisfactory result; but the identification of a single red corpuscle would be proof of the presence of blood. The white corpuscles may sometimes be detected where the red disks cannot be distinguished. They are much fewer in number and colorless. If very abundant in a specimen, they might be owing to pus rather than to blood, or to disease (leukæmia). If only a minute speck of dried blood, taken from a weapon or from a garment, is to be examined, it may be laid upon the glass, which has previously been breathed upon several times, and after again breathing upon it, it should be covered with the thin slide, and examined, as before. The condensed moisture of the breath serves the place of water in breaking up the dried clot, without destroying the corpuscles by too much dilution (Dr. A. Taylor, from Casper).

The human blood corpuscle is a round, bi-concave disk, without a nucleus. All mammalian corpuscles have the same form, with the exception of those of the camel tribe, which are oval. The corpuscles of birds, reptiles and fishes are oval, larger in size, and nucleated. It is well to remember that oval corpuscles may become globular by treatment with an excess of water. The outlines of dried blood corpuscles are irregular and jagged, and more or less stellate.

The average diameter of the human blood corpuscle is about the \( \frac{2}{3} \) of an inch (max. \( \frac{2}{3} \), min. \( \frac{1}{3} \), Gulliver). The average, according to Taylor, is \( \frac{3}{10} \) of an inch (max., \( \frac{3}{10} \), min., \( \frac{2}{10} \)). The corpuscles of most of the lower animals are smaller than those of man. The average diameter in the monkey is \( \frac{3}{7} \) of an inch; of the dog, \( \frac{3}{5} \); of the horse, \( \frac{3}{5} \); of the cow, \( \frac{2}{5} \); of the goat, \( \frac{2}{3} \); of the sheep, \( \frac{2}{3} \); of the cat, \( \frac{2}{3} \); of the pig, \( \frac{2}{3} \); of the mouse, \( \frac{2}{3} \);
of the hare. From the above enumeration it will be seen that the monkey, dog and hare, and, perhaps, the mouse, are the animals whose blood corpuscles most closely approximate in size to those of man, while those of the domestic animals whose blood would be most likely to be confounded with human blood, viz., the horse, cow, pig, sheep and goat, are notably smaller than the human. The corpuscles of birds, reptiles and fishes, as already mentioned, are oval in shape, and considerably larger than those of mammals. The long diameter of the corpuscle of the common eel is \( \frac{7}{16} \) of an inch; the short diameter, \( \frac{3}{16} \); of the shark, long diameter, \( \frac{7}{16} \), short diameter, \( \frac{5}{16} \); of the turtle, long diameter, \( \frac{3}{16} \), short diameter, \( \frac{5}{16} \); of the Congo lizard, long diameter, \( \frac{1}{8} \), short diameter, \( \frac{1}{16} \); of the proteus, long diameter, \( \frac{1}{16} \), short diameter, \( \frac{7}{16} \); of the frog, long diameter, \( \frac{1}{16} \), short diameter, \( \frac{1}{8} \); of the pigeon, long diameter, \( \frac{1}{16} \), short diameter, \( \frac{1}{8} \); of the common fowl, long diameter, \( \frac{1}{16} \), short diameter, \( \frac{1}{8} \); of the duck, long diameter, \( \frac{1}{8} \), short diameter, \( \frac{3}{16} \).

The important medico-legal question in connection with blood-stains is, whether it is possible to distinguish human blood from that of one of the lower animals. Of course, there can be no difficulty in recognizing this difference in the case of birds, reptiles and fishes; the shape and size of the corpuscles, in each of these orders, will at once make it manifest. But as regards the blood of the common domestic animals, such as the cow, horse, pig and goat, the difficulty is vastly increased. In a case of homicide, where a blood-stained garment might constitute an important link in the chain of evidence, and where the accused might very naturally assert that the suspicious stains were accidentally caused by the blood of one of these animals, it becomes of unspeakable importance to be able to decide this point.
Until within a few years it has been considered impossible to give a positive answer to this question. To Prof. J. G. Richardson, of Philadelphia, is, we think, justly due the merit of having demonstrated the possibility of distinguishing between human blood and that of the horse, cow, sheep, pig and goat—those animals whose blood would be most likely to be confounded with that of man. He has conclusively shown that, by employing very high microscopic powers, such as the $\frac{3}{5}$ of an inch objective, magnifying with a micrometer eye-piece over 3000 diameters, the human corpuscle appears about $\frac{6}{10}$ of an inch in diameter, whilst those of the ox and sheep are about $\frac{5}{6}$ of an inch in diameter, indicating a very obvious difference in their respective sizes. The use of the ordinary powers (500 or 600 diameters) fails entirely to recognize the difference.

It is certainly to be regretted, for medico-legal reasons, that the size of the blood corpuscles of the monkey, dog and rabbit approximates so closely to that of the human corpuscle, that it is not possible, by the microscope, nor by any means yet discovered, to recognize the distinction between them; but it is equally no slight triumph for microscopic science to have accomplished as much as it has done. In several homicide cases, Prof. Richardson has been able to give most positive and valuable testimony as to the identity of human blood-stains, by his microscopic researches. His method of procedure is to scrape off a minute particle of the suspected blood-clot from the stained article with the point of a cataract needle, letting it fall upon a clean microscopic slide. A thin cover is then laid upon the fragment, and pressed down firmly, so as to crush the particle to powder, and the whole transferred to the stage of the microscope. Pure water should be introduced at the margin of the cover, and allowed to flow very slowly towards the specimen; when this is reached, a move-
ment is observed, after which an aggregation of compressed corpuscles, very faint and colorless, but yet very distinct, comes into view. These are rendered more obvious by introducing at the margin of the cover a minute portion of iodine, or red aniline solution.* The author has verified, by personal experiment, the correctness of Prof. Richardson's deductions. On one occasion, specimens of dried human blood, together with that of the ox, horse and sheep, were submitted, unmarked, to Prof. R., for microscopic examination, and the correct result was returned in every instance.

It may be proper to allude to certain bodies that might be mistaken, under the microscope, for blood corpuscles, such as starch-granules, the sporules of fungi, and the disks of coniferous woods. But as these generally possess certain special marks by which they can be recognized, they need never be confounded with blood disks, by a practiced microscopist.

Blood Crystals constitute another test for blood. They can be obtained from all kinds of red blood, being, in fact, due to the crystallization of the haemoglobin. To procure them, according to Lehmann, evaporate a drop of blood to dryness on a piece of glass, add a drop of distilled water, and cover the whole with a slip of thin glass. After a time, when the water has nearly evaporated, microscopic crystals, of various sizes and forms, are visible. Those of man are prismatic, or rhomboidal; those of the inferior animals are either similar in shape, or else tetrahedral or hexagonal. In order to procure crystals from the blood stains upon a fabric, such as linen or muslin, or from a dried coagulum, the process of Kunze and Neumann seems to be the best. This consists in soaking the stained portion for several hours in a small quantity of cold water, until the coloring matter is

dissolved; the reddish solution is then evaporated to dryness; the dry residue boiled with an excess of glacial acetic acid, until a red solution is procured; this is then slowly evaporated on a glass slide until the crystals form, when they are examined by a power of 500 diameters. The blood crystals appear in groups, frequently crossing one another, intermixed with cubic crystals of chloride of sodium. But the similarity of human blood crystals to those of the lower animals is too great to permit of the forming of a positive diagnosis as to their real origin. Consequently, they cannot be regarded as affording much practical assistance in the identification of blood stains.

III. The Optical or Spectroscopic Test.—The application of the spectroscope to the identification of blood depends upon the fact that various colored solutions possess the power of absorbing different portions of the spectrum, and of producing in the latter certain dark lines, just as certain vapors and gases affect the spectrum. Blood, in this respect, produces a very decided effect, causing absorption bands (dark lines), which are very characteristic of its presence, even in minute quantities.

It will be remembered that in perfectly fresh arterial blood, the coloring matter exists as haemoglobin. (1) When this is examined by the spectroscope, it will be seen to produce two absorption bands in the spectrum, in the yellower half of the green space, the lower one being twice as broad as the upper; also the blue end is darkened. When haemoglobin is acted upon by acids and alkalies, or kept for a long time, especially in a damp place, it acquires a brown color, becomes deoxidized, and is finally changed into haematin. (2) The spectrum of deoxidized haemoglobin, or of venous
blood, shows a single broad absorption band, visible in the green; the blue end is also darkened. (3) After a short exposure to the air \((\text{methæmoglobin})\), it gives a spectrum with the blue end darkened, the \textit{two} bands of oxidized \text{haemoglobin} much weakened, with a \textit{third} band visible in the red. (4) The spectrum of deoxidized \text{haematin}, or of blood after prolonged exposure to air, shows the blue end darkened, and \textit{two} well-defined bands in the green, but stronger than in (1), and with disappearance of the band in the red.

The form of apparatus best adapted for spectroscopic examination of blood is that suggested by Mr. Sorby, to whose researches we are chiefly indebted for our knowledge in this branch of investigation. It is a combination of the microscope and spectroscope. The blood solution is prepared in the same manner as that described above for the chemical tests. The details of the examination by the spectroscope would exceed the limits of this work; the reader is referred to the larger treatises for a fuller description.

The important question in this connection is—Do other substances give similar spectra to those of blood? According to Mr. Sorby, nothing gives a spectrum \textit{precisely} similar to that produced by \text{oxy-haemoglobin}, although certain other bodies produce absorption lines \textit{somewhat} resembling the former, but easily distinguishable by a practiced observer. Thus, the coloring matter of the petals of \textit{Cineraria} give two absorption bands; but they are easily distinguished by the action of ammonia. Cochineal, madder, and other red dyes, dissolved in alum, although affording bands somewhat resembling those produced by blood, may be distinguished from the latter by the use of ammonia and potassic sulphite.
We must, therefore, admit that the spectroscope, *in the hands of a skilled operator*, affords the most certain and delicate test known for the presence of blood. It cannot, however, discriminate between human blood and that of any of the lower animals; in this respect, therefore, it is inferior to the microscope as a test.
CHAPTER VIII.
BURNS AND SCALDS.

DEFINITION.—CLASSIFICATION.—SYMPTOMS.—CAUSE OF DEATH.—
POST-MORTEM APPEARANCES.—DISTINCTION BETWEEN BURNS MADE
BEFORE AND AFTER DEATH.—ACCIDENTAL, SUICIDAL AND HOMICIDAL BURNS.—SPONTANEOUS COMBUSTION.

A burn is an injury to the body, caused by heat applied either in the form of a heated solid substance, or by flame, or by radiant heat.

A scald is an injury produced by a liquid, heated above a certain point, applied to the body.

Burns and scalds are not, strictly speaking, wounds; though legally they are comprised under the term bodily injuries. The effects of corrosive liquids, such as sulphuric and other mineral acids, and the strong alkalies, closely resemble burns, and they are so regarded in law. Boiling liquids taken internally may produce internal scalds.

The intensity of a burn is dependent upon the degree of heat applied; it varies from a slight redness, to a complete charring of the tissues. Metals heated to redness produce very severe burns, even to the destruction of the flesh; but if in a state of fusion, the injury is yet more serious, in consequence of the partial adhesion of the molten mass to the skin. Boiling oils produce as decided effects as hot solids or molten metals. Boiling water causes scalds, more or less severe, attended with vesications containing serum; but it never chars, or destroys the tissue.

According to Dupuytren, burns may be classified as follows:—
(1) Superficial inflammation of the skin, without vesication.

(2) Vesication, or blisters, containing serum, sometimes clear and sometimes opaque and bloody. If the cuticle be removed, the true skin is very red and granulated, and secretes pus.

(3) Destruction of the external surface of the true skin, forming an eschar, which may be soft and yellow if made by a liquid, or hard and brown, or black, if resulting from a solid. The surrounding skin is red and blistered. This form of burns leaves ugly cicatrices, which are white and shining.

(4) Disorganization of the whole skin: these differ from the last only in the deeper destruction of the parts, and in the thickness of the sloughs. The resulting scars are puckered, and depressed below the level of the skin.

(5) The destruction here extends through the skin, and includes the cellular tissue and a portion of the muscles. The general character is the same as in (4).

(6) Complete carbonization of the burnt part, as when a portion of the body is roasted by the fire.

The important medico-legal question to determine is—was the burn upon the body made before, or after death? It is evident that an assassin might murder his victim, and then set fire to the house, hoping thus to escape detection. If a body be found completely charred, it will be impossible to determine whether it was living or dead when acted upon by the heat.

As regards vesications, which result from moderately heated solids, or from scalding liquids, if they contain serum, their presence, as a rule, indicates that the burn was inflicted during life. The experiments of Christison, Taylor and Tidy go to show that, although the application of heat to a body within a few minutes after death may sometimes
produce a blister, this does not contain serum, but only air; serous exudation must be regarded as vital. There may be an exception to the above rule in the case of dropsical subjects, in whom it is stated, on good authority, that serous blisters may be produced after death, by the application of heat. But, on the other hand, the absence of vesication should not be regarded as a proof that the burn was not inflicted during life, since vesication is not always a necessary result of a burn; besides, it is quite possible that only the more serious results may be visible. It is recommended, in all doubtful cases, to examine the cuticle minutely, with a lens, for minute apertures through which the serum may have escaped.

Another sign of burning during life is the presence of a red line around the burn, which gradually merges into the color of the surrounding skin. This red border remains after death, and cannot be produced, according to Christison and Taylor, by the application of heat to the dead body. Dr. Tidy's conclusions (Legal Med., vol. I, p. 482), based upon a series of his own experiments, are that "where there are serous blisters on a dead body, the serum being thick and rich in albumen, and the blisters surrounded by a deeply injected red line, the true skin, after the removal of the cuticle, also presenting a reddened appearance, the evidence is strong that the burn was produced during the life of the person; while it is conclusive that it was caused during the life of the part. But if the blister contained air, the true skin, after the removal of the cuticle, appearing dry and unglazed, of a dull white color or grayish; or, if the blister contains a little thin, non-albuminous serum, there being in neither case any red surrounding line, nor any injected condition of the cutis vera, the evidence is strong that the burn was inflicted after death."
The Danger of burns depends more on their extent than their depth. The reason of this is that extensive burns involve a greater number of sensory nerves, and a greater extent of surface is prevented from performing the function of excretion and heat regulation. Thus, a large superficial scald, especially in young children, is very apt to prove fatal, the symptoms being stupor, somnolence, pallor of face, and feeble pulse, with slow and stertorous breathing—very similar to those of narcotic poisoning, for which, indeed, they have sometimes been mistaken. It has been ascertained that if one-half to two-thirds of the entire skin be involved, the burn will certainly prove fatal; but practically, one involving one-third of the body, if severe, would be very likely to cause death. But here, many circumstances will have to be considered, such as the age, constitution, the part affected, and the character of the burn. Burns are more dangerous in the young; more so on the trunk of the body than on the limbs; and more so if in separate patches than if continuous, provided they are of equal extent. Gunpowder burns are considered more dangerous than those produced by steam.

The Causes of death in burns are various: as (1) bodily injury; as in the case of conflagrations of buildings, where instantaneous death may result from the fall of timbers, walls, etc., or from leaping out of a window, or from a roof. (2) Suffocation, either from the smoke, or from the want of air. (3) Shock; this is probably the most frequent cause of death after extensive burns. (4) Coma, convulsions or tetanus. (5) Bronchitis, pneumonia and other thoracic symptoms. (6) Enteritis and peritonitis. (7) Exhaustion. (8) Gangrene, pyæmia, etc.

The Post-mortem appearances.—These are often by no means well marked, the most constant lesions being a
capillary injection of the mucous membrane of the alimentary canal and bronchi, and serous effusion into the ventricles of the brain. In cases where the death has occurred from injury, or from suffocation, the usual lesions would, of course, be discovered after death; but if the body has been completely charred or roasted, it will probably be impossible to distinguish anything to enable us to form an opinion as to whether the death had preceded the burning or not. The means of identifying the charred remains of a burnt body have already been pointed out (ante, p. 88).

Wounds upon the burned.—From the fact that murder is frequently committed, and the body subsequently burned by a criminal, with a view of destroying the traces of his crime, it is important for the legal physician always to examine the body for wounds. There are certain mechanical effects produced upon the body by fire, which might possibly be mistaken for wounds made before death, such as fissure in the thorax or abdomen, or in the neighborhood of the large joints. These fissures are generally irregular in form, and, as the blood vessels, by their elasticity, are apt to escape being torn, these may be seen intact, stretching across the fissure. This appearance is always indicative that the opening was caused by heat, and was not a real wound. A case is mentioned in which two old people were found burned in their house; the fact of their having been previously stunned, if not killed, by blows on the head, was ascertained by the existence of fractures of the skull, under which coagulated blood was found upon the dura mater. Where the heat has been excessive, the bones of the deceased may be found more or less cracked or split, and sometimes even crumbled to pieces. Ordinary incised, punctured or contused wounds, made before death, could not be identified in a body completely charred by fire.
As to the question of the burning being accidental, suicidal or homicidal, it may be assumed that death by burning is nearly always accidental; and such cases are, unfortunately, of frequent occurrence. Death, in such instances, mostly occurs at some distance from the fire. The fact that a dead body is found near the fire does not, however, preclude the idea of accident, since an intoxicated or a diseased person may have caught fire, and been unable to remove. In all such cases it is important to examine the body for marks of violence, with the precautions given above. Furthermore, a case might present itself where severe wounds were found on a burnt body, and the question might arise whether the wounds, or the fire had been the cause of death? No general rules can be given for guidance in such cases; each one must be determined by the attending circumstances.

The question of Spontaneous Combustion of the human body presents itself here for a brief notice. It has occasioned considerable discussion in the scientific world for many years past, but although some remarkable instances are related of apparent spontaneous combustion of the human body, originating while alive, we believe that, on close investigation, it will be found that some source of fire had invariably been present, from which the combustion took its origin—such as a lighted pipe or candle,—and that the body was that of an habitual spirit drinker, and nearly always that of a very fat woman—conditions highly favorable for the process of combustion, when once originated.

From the known composition of the human body—nearly 75 per cent. being water—it would seem to be chemically and physiologically impossible even to burn up a dead body, except on the application of an extraordinary degree of heat, such as is required by the process of cremation. Certainly, the weight of authority is against the belief in spontaneous
combustion of the human body; no person of position or authority has ever witnessed such a phenomenon, and we must therefore express our disbelief in it, and assign the wonderful accounts which from time to time have appeared in the newspapers and books to the region of romance and fiction.

It is, of course, an admitted fact that various organic and mineral substances undergo spontaneous combustion, through the agency of absorbed oxygen, especially when exposed to the action of the air, in a state of fine powder or extended surface. Conflagrations of large buildings have frequently thus originated, involving important legal questions as to incendiarism.
CHAPTER IX.

VIOLENT DEATH FROM DIFFERENT FORMS OF APNEA (ASPHYXIA).

This includes death from Suffocation, Strangulation, Hanging and Drowning, in all of which life is destroyed chiefly, if not exclusively, by apneea or asphyxia. All these modes of violent death possess certain points in common, while, at the same time, each of them is distinguished by individual peculiarities, which render a separate consideration desirable. Their common properties will be first briefly considered.

In all cases of apneea, it will be remembered that death begins in the lungs, and that this is brought about simply by excluding the air (oxygen) from these organs. This is accomplished by numerous and diverse means: as by mechanical pressure upon the throat or thorax, as in throttling; by a ligature around the throat, as in hanging and strangling; by the flow of water into the windpipe, as in drowning; by foreign bodies getting into the larynx and trachea, as in choking; by being shut up in a box (entombed alive), or buried under ruins, or a sand bank, or snow drift; or by some disease of the throat, as oedema of the glottis, membranous croup, etc.;—all of which produce death simply by arresting the function of respiration.

Likewise, there are exhibited certain signs or phenomena, both before and after death, which indicate death by apneea. These are lividity of the lips, fingers, and other extremities, and generally of the whole face, together with a swollen appearance of the countenance; convulsive move-
ments of the arms and legs, at first partly voluntary, but soon becoming spasmodic and involuntary, as seen in the struggles to breathe; the veins become turgid; the pulse, at first full and rapid, soon becomes feeble; there is often frothing at the mouth, which may, at times, be tinged with blood; there is frequently turgescence of the genital organs, with involuntary discharge of semen, urine and faeces. Abortive attempts at respiration are made for awhile, but finally these cease, and the heart at last ceases to pulsate.

Consciousness is lost very early, although in the earliest stage there is a remarkable activity of the senses; the memory is surprisingly acute, so that the events of a lifetime seem to be crowded into a moment. But this stage only lasts for a very brief space of time; such is the testimony of persons who have been rescued from drowning, or who have been cut down from hanging, and of those who have experimented upon themselves by partial strangulation.

This kind of death is rapid, not requiring more than three to five minutes, though there are some apparent exceptions in the case of *drowning*. These will be referred to hereafter.

The *post-mortem appearances* in all these varieties of death by apnoea are, in the main, very similar. These are lividity of the lips, fingers and other parts of the body, as seen before death; in *drowning*, the face is apt to be pale; sometimes, likewise, in *hanging*. The venous system is generally full of blood. The right side of the heart, together with the lungs, is usually gorged with dark blood; the mucous membrane of the bronchial tubes deeply congested. In young persons, the blood vessels of the lungs will often be found empty, and the lungs emphysematous, from the violent efforts made to respire. Minute extravasations of blood (ecchymoses) are found in the mucous and serous
membranes, as the pleura, pericardium, endocardium, peritoneum, etc. (Tardieu). The veins and sinuses of the brain are usually tinged with blood, and the brain itself filled with bloody points. The solid viscera, as the liver, spleen and kidneys, will generally be congested. The blood itself is mostly fluid and dark colored, except in suffocation from carbonic acid, when its color is bright red.

SECTION I.

DEATH BY SUFFOCATION.

ACCIDENTAL SUFFOCATION.—SUICIDAL AND HOMICIDAL SUFFOCATION.—POST-MORTEM SIGNS.

Suffocation, properly speaking, includes every variety of death resulting from an impediment to respiration. But, as Strangulation, Hanging and Drowning are considered separately, the term is here restricted to the other modes of death by apnoea.

Cases of accidental suffocation are numerous. Infants have thus perished by being too closely wrapped up, or by being overlaid by their mothers, who are often intoxicated. Young children, feeble persons, epileptics and drunkards, have been suffocated by falling into ashes, soft mud, feathers and similar articles. Mechanical pressure on the thorax, as occurs in vast crowds of people, has destroyed life by suffocation. The accidental slipping into the larynx of small bodies, such as peas, grains of corn, marbles, etc., from the mouth, particularly in children; the lodgment of a piece of meat in the air passages (choking), from over-haste in eating; the detachment of a bronchial gland which became impacted in the larynx; the escape of a lumbricus from the stomach, and its entering the larynx; the passing of vomited matters into the windpipe; various disorders of the throat,
as oedema and spasm of the glottis, croup, diphtheria, abscess, etc.—all these are examples of accidental suffocation.

Suicidal suffocation is extremely rare, though a few remarkable cases are mentioned by authors, in one of which—that of a young woman—death was caused by a ball of hay which she had thrust down the throat into the pharynx, behind the larynx, and which was just visible when the mouth was widely opened (Year Book of Med. and Surg., 1864, p. 458).

Homicidal suffocation is usually practiced upon infants, the aged, or those who are otherwise helpless. Suffocation is undoubtedly a very common mode of destroying newborn children; it is very easily effected, and leaves behind it no characteristic traces; death, in such cases, being usually attributed to convulsions. The notorious Edinburgh murderers, Burke and Hare, destroyed their victims by suffocation by forcibly closing their mouth and nostrils, and at the same time bearing their whole weight upon their breast. A curious Scotch case is mentioned, where an intemperate woman, between sixty and seventy years of age, was found dead with a wound upon the scalp, emphysema in the chest, and seven ribs fractured. The face was pale and composed, the eyes closed, and the tongue slightly protruding. On examination, the cork of a quart bottle was found in her larynx, the sealed end being uppermost. The epiglottis, trachea and larynx were considerably injected. It was attempted, on the trial, to show that the deceased had drawn out the cork with her teeth, but that it was suddenly forced into her windpipe, while she was dead drunk. But this was negatived by the fact that the sealed end of the cork was uppermost, and also by the marks of the corkscrew! It was thence concluded that the cork had been forcibly
pushed into her windpipe, while she was unable to resist, through intoxication. Another case of homicidal suffocation is related (Woodman and Tidy's *Foren. Med.*, p. 851), of a Russian sentry on guard, being found dead in his watchbox, with a large piece of meat in the lower part of the pharynx, pressing upon, and partly in the glottis. His death was therefore supposed to be accidental. Some years after, his superior officer, in dying, confessed that he had first suffocated the man, and then placed the piece of meat in his throat, in order to divert suspicion from himself.

*Post-mortem appearances.*—Lividity and swelling of the face and lips, though often, in accidental cases, the face is placid; the eyes are congested; minute ecchymoses on the neck and chest; mucous froth, sometimes bloody, about the mouth and nose; the lungs and right side of the heart may be gorged with dark blood, although in some cases, especially of young children, the lungs may be empty of blood and emphysematous. Tardieu lays great stress upon the presence of *minute punctiform ecchymoses*, especially on the lungs of new-born infants, who have been suffocated. These spots are also found on the pleura, lining membrane of the heart, membranes of the brain, peritoneum, and mucous lining of the windpipe. Other authorities, among whom is Dr. Ogston, deny that these extravasations are peculiar to cases of suffocation, as they are found in other modes of death. The blood is dark and very fluid. The kidneys are deeply congested.

As these post-mortem signs are also found in other forms of death by apnoea, they cannot be considered as *characteristic* of death by suffocation. Consequently, the examiner should be cautious in expressing his opinion as to the cause of death. If a dead body be discovered in sand, earth, ashes, or similar substances, the question whether it was placed
there before or after death must be decided by a careful examination. If the substances be found in the air passages, and especially in the oesophagus and stomach, it may be concluded that the person was alive at the time.

SECTION II.

DEATH BY STRANGULATION.

MODES OF STRANGULATION.—EXTERNAL SIGNS.—INTERNAL LESIONS.—ACCIDENTAL, SUICIDAL AND HOMICIDAL STRANGULATION.—MARK OF THE CORD.

Strangulation is produced either by pressure upon the neck by means of an encircling cord, or by direct pressure made by the hand on the windpipe, as in throttling. The means by which the constriction is produced are various: sometimes a rope is used, sometimes a strap, a handkerchief, a ribbon, or a strip torn from a sheet or the clothing. In Spain, the usual mode of execution of criminals is by the garrotte—a steel collar tightened by a screw; in Turkey, it is by the bow string. Death results, in most cases, from the combined effect of the deprivation of atmospheric air, producing apnoea, and from congestion of the brain, due to the pressure upon the jugulars, preventing a return of blood from the brain. It differs from hanging chiefly in the obliquity of the cord around the neck in the latter, while in strangulation, the cord is wound horizontally around the neck. It is important, medico-legally, to distinguish between death from strangulation and death from hanging, as the former is nearly always the result of homicide, while the latter is usually to be traced to suicide. The first question that presents itself here is—was the death caused by strangulation? The appearances of one strangled are usually very distinctly marked: these are livid and swollen
face; staring eyes, with dilated pupils, and protruding tongue, which may be bitten; livid extremities; flattened larynx; blood may issue from the nose, mouth, or even ears; the face, neck, chest and eyes are studded with ecchymoses; the genital organs frequently turgid; and there may be an escape of urine and faeces, as in hanging. Internally, the right heart and venous system are sometimes gorged with blood; but this is less frequent than in other forms of death from apnoea; this is also true of the congestion of the liver and kidneys. Tardieu states that the lungs are seldom very full of blood, but he places great reliance upon the emphysematous appearance of these organs, arising from a rupture of the pulmonary vesicles. The sub-pleural ecchymoses, which he regards as characteristic of suffocation, he says are rare in strangulation. There are also extravasations of blood in the lungs, but none in the brain, whereby it is distinguished from apoplexy, which it resembles in a few of its symptoms.

Among the external signs, the marks of the cord, and of the fingers on the neck deserve special attention. These are more evident and reliable here than in hanging, because in homicidal strangulation very considerable force is generally employed by the murderer, in order to accomplish his object. If the hand has been used, as in throttling, the marks of the fingers will be found upon the front of the throat, sometimes of two or more fingers and the thumb, so that the particular hand employed may even be determined. If a cord has been used, the mark will be horizontal, not oblique, as in hanging; sometimes there may be two or three parallel marks, where the cord has been wound around the neck several times. The mark of the cord is apt to be less deep than in hanging, and subcutaneous extravasation is not always found; but the parts beneath may show con-
sizable infiltration of blood. Fractures of the hyoid bone, and of the ossified thyroid cartilages are reported as having occurred. The interior of the larynx and trachea is congested, of a uniform red or violet color, and is coated over with a frothy, bloody mucus, which extends also into the smaller air tubes. This internal discoloration of the windpipe should not be mistaken for the early signs of putrefaction of this organ (Vid. ante, p. 47).

The mark of the cord around the neck may unquestionably be produced on the dead body, if the attempt is made within a few hours after death, and while the body is still warm, but not (according to Casper) after six hours. Therefore, this one particular sign should never be relied on to the exclusion of the other characteristic evidences of death by strangulation, such as the livid, swollen countenance, the protruded tongue, the staring eyeballs, etc., none of which are produced by strangulation after death. Hence, although a murderer may place a cord around the neck of his dead victim, with a view to make the case simulate a suicide, there will be little difficulty in detecting the ruse.

Was the strangling accidental, suicidal or homicidal? Cases of accidental strangulation not unfrequently occur. Prof. Taylor records two: one, of a girl carrying fish in a basket, which was strapped around the upper part of her chest in front. She was found dead, sitting on a stone wall. The basket had probably slipped off while she was resting, and had thus raised the strap, which firmly and fatally compressed the trachea. The other case was that of a boy, whose silk necktie, knotted and tightly twisted around his neck, was caught in the band of an engine, and his neck drawn down against one of the revolving shafts. He was rescued after his neck had been compressed at least one minute. He became black in the face, and blood escaped
from the mouth and ears. For several minutes after the removal of the ligature, he was insensible, but ultimately recovered. Another instance is related by Dr. Gordon Smith, of a lad who used to carry a heavy weight suspended from his neck by a string. One day he was found quite dead, sitting in a chair. He had probably gone to sleep, the weight had slipped, and drawn the cord tight around his neck.

Suicidal strangulation is comparatively rare, except among the insane, with whom it is by no means uncommon. The facility of effecting their purpose by such simple means as a garter, a ribbon, a handkerchief, or a strip torn from a garment, may readily account for such occurrences, and still further, when it is remembered how very rapidly and insidiously unconsciousness steals over the senses under a pressure of the windpipe, thereby taking away from the individual the will and the power to escape. A case mentioned by Dr. Taylor (Med. Jurisp.) will illustrate this. An insane gentleman, with suicidal tendencies, was placed in a private asylum, with especial directions to watch him closely, to prevent his taking his life. Two attendants were placed over him. On retiring to his bed, these attendants remained at his bedside; but on his requesting them to retire to a little distance, they complied, still keeping a close watch upon him. Two hours afterward, the physician, on visiting the patient, was informed by the attendants that he had been sleeping quietly for some time. On approaching the bed, to their horror and surprise, they found the gentleman dead! He had strangled himself simply by tearing off a strip from the bottom of his shirt, rolled it into a cord, and tied it around his neck. Other cases of strangulation are recorded of determined suicides, where the cord was found coiled around the neck several times; in one instance, the
ligature had been tightened by a stick thrust in and twisted like a tourniquet; and in still another, a sabre had been used for the same purpose.

_Homicidal_ strangulation, as already mentioned, is the most frequent variety of this form of violent death. It is usually recognized by the marks upon the neck and elsewhere, indicating a greater amount of violence employed. Thus, the impression of the ligature on the neck will be deeper and more ecchymosed than occurs in a suicide; it may also be accompanied by the marks of the fingers on the throat, which latter are never found either in a suicidal or accidental case. Besides these, there will frequently be seen contusions or injuries of other parts of the body, and other evidences of a struggle.

It should not be forgotten that the marks of homicidal strangulation may often be discovered many weeks, or even years, after burial. One is mentioned by Wharton and Stille (_Med. Jurisp._, vol. II, p. 830), where, after thirty-eight days' interment, the evidence of strangulation was obtained chiefly from the striking contrast of the integuments of the neck with those of the rest of the body. There was a white, shriveled space over the larynx, half an inch broad; also a groove around the neck, of a blackish-brown color and parchment-like appearance; this condensed skin was difficult to cut, and its section was perfectly dry and yellowish-white. Another remarkable case occurred in Paris, where the body had been buried several years, and was reduced almost to a perfect skeleton. Several of the cervical vertebrae, together with the right clavicle, were found held together by a blackish mass, in the composition of which no tissue could be recognized. This mass was surrounded by several twists of a cord two lines in diameter. The cord was much decayed, showing no knots, and its direction was
SECTION III.

DEATH BY HANGING.

CAUSE OF DEATH IN HANGING.—POST-MORTEM APPEARANCES.—CORD-MARK.—GENERALLY SUICIDAL.

Hanging is that mode of death caused by suspension of the body by the neck, the weight of the body acting as the constricting force. Physiologically, it is the same as strangulation, and, like the latter, the cause of death is partly apncea and partly cerebral congestion, and more frequently a combination of the two. The following table exhibits the relative frequency of each form of death:

<table>
<thead>
<tr>
<th></th>
<th>Remer.</th>
<th>Casper.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Apoplexy</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Asphyxia (Apncea)</td>
<td>68</td>
<td>62</td>
</tr>
<tr>
<td>Mixed</td>
<td>83</td>
<td>85</td>
</tr>
</tbody>
</table>

If the cord encircles the neck below the thyroid cartilage, the death is more rapid, and is to be ascribed to apncea; but if higher up, as in executions, where it is apt to slip under the chin, some little space on either side may escape constriction, so as to admit a slight amount of air into the lungs; in this case the death will be slower, and be due rather to cerebral congestion. In the great majority of cases, however, as shown by the above table, the cause of death is of a mixed nature. In some cases of public executions, where the fall was very considerable, and where a violent rotary swing was given to the body of the criminal at the moment of the drop, the odontoid process of the second cervical vertebra has been found either fractured or dislocated, causing immediate death, owing to pressure on the spinal cord. But death, in hanging, from fracture of the
vertebræ, is far less frequent than is popularly imagined. Orfila states that in the bodies of fifty persons who had been hanged he met with a fracture of the os hyoides in only one case, while he had never met with a fracture or luxation of the vertebrae.

There is reason to believe that death by hanging is nearly painless. The convulsive movements of the limbs, as is well-known, are no indications of suffering. Unconsciousness very speedily supervenes, especially if the trachea is compressed, and death occurs in a very few minutes. Persons who have been cut down after a few minutes' suspension are very rarely resuscitated. And even after an apparent partial recovery, death often follows from secondary effects, especially from congestion of the brain.

The insidious manner in which the loss of consciousness steals upon the brain in hanging deserves especial notice, because it satisfactorily explains the facility with which death takes place, even when the suspension of the body is not complete, but when there has been simply a pressure of the ligature against the windpipe, the person meanwhile resting on the knees or toes, or being in a semi-recumbent posture.

Post-mortem appearances.—In the main, they resemble those attending death from strangulation. Externally, swelling and lividity of face, congestion of the eyelids, dilated pupils, eyes red and protruding, tongue swollen, livid, often protruded or compressed between the teeth, lower jaw retracted, often a bloody froth escaping from the mouth and nostrils. In addition, there are often petechial effusions on the neck, shoulders, arms and hands. In many cases, however, the countenance is calm, the face pale, the eyes and tongue natural. Sometimes there is turgescence of the genital organs, with an involuntary escape of the urine,
fæces and semen; but these signs are by no means peculiar to death by hanging. The position of the head varies according to the part of the neck where the knot was placed. As the latter is usually behind the neck, the head is generally flexed forward. If the knot were in front, the head would be found extended backward (Tardieu). The hands are generally closed, often tightly; the legs extended, and often livid. The neck is nearly always stretched, owing to the weight of the body, and it presents very decided marks of the cord, varying however somewhat, according to the nature of the latter and its mode of application. Thus, the mark may be deep or superficial, single or double, according to the strain made upon it, and its thickness, roughness, or duplication. The skin under this mark becomes very dense and tough, and of a yellowish-brown color, and has been aptly compared to old parchment. This appearance is more marked some hours after death, if the cord has been removed; and the cellular tissue underneath is also condensed, and has a silvery appearance. Besides the above, there is often a livid mark (ecchymosis), where great violence has been used, as in executions; but the latter is quite distinct from the true mark of the cord, with which it has been confounded. The livid line is much less frequently met with than was formerly supposed.

The groove or furrow in the neck, in the great majority of cases, will be found between the chin and larynx; its direction is oblique (which distinguishes it from strangulation); it may also be double (arising from a double fold of the cord), and irregular or interrupted. In general, the narrower the ligature, and the longer the suspension, the deeper the furrow. A broad leather thong, pressing only by its borders, might produce a double mark.

Internally, the appearances usually accompanying as-
phyxia are met with, such as engorgement of the lungs, right side of the heart, and venous system with dark fluid blood. The lining membrane of the larynx and trachea is deeply congested, as in strangulation, and is sometimes coated with a bloody froth. The vessels of the brain are generally congested, but extravasation of blood into the brain, or upon its membranes is extremely rare. The brain itself, when cut into, presents numerous bloody points. The kidneys are usually congested; the stomach frequently presents evidences of such deep congestion as to suggest the idea of an irritant poison. The same is true also of the intestines. Dr. Yellowly has found coagulated blood on the mucous membrane of the stomach, in two out of every five cases of death by hanging.

Among the occasional lesions may be mentioned fracture of the hyoid bone and thyroid cartilage, and rupture of the internal and middle coats of the common carotid artery. According to Dr. Dyer (New York Med. Jour., 1866), a transverse fracture of the crystalline lens is a frequent result of death by hanging. He discovered it in three cases out of four, one of a man, and three of dogs. Some years ago these experiments on dogs and cats were repeated by one of the author’s students, at the University of Pennsylvania, but in every instance with a negative result.

An important medico-legal question to settle is—was the death caused by hanging? This cannot always be satisfactorily determined by mere medical evidence, since there are no positive or characteristic signs of this kind of death. The mere suspension of the body is no proof, since a murderer might easily suspend the body of his victim, in order to divert suspicion from the true cause of death. The mark of the cord can be imitated by suspending a dead body by the neck immediately after death, and, according to Casper,
even up to seventy-two hours after, especially if the body be forcibly pulled downward. The livid or ecchymosed line is less likely to be found, under these circumstances, than the brownish, parchment-like furrow. Hence, it follows that the mark of the cord cannot be regarded as evidence of death by hanging; and the other usual signs, such as turgescence and lividity of the face, congested eyes, swollen tongue, etc., are all met with in strangling, and other forms of death, while these very signs may be absent in certain cases of hanging. Dr. R. F. Hutchinson states that an invariable sign of death from hanging is the flow of saliva out of the mouth, down the chin, and straight down the chest. The appearance is unmistakable and invariable, and could not occur in a body hung up after death, the secretion of saliva being a living act (Chevers, quoted by Husband).

To determine the question whether the hanging was suicidal, homicidal or accidental, regard must be had to the attending circumstances; remembering always that hanging is a particularly frequent method of suicide. Out of 368 cases of suicide occurring in Berlin, 189 resulted from hanging. Hence, the presumption is always in favor of suicide; besides the difficulties that would attend an attempt at murder by this means. If, however, the body exhibit evidences of great violence externally, denoting a struggle, or marks of the fingers about the throat, or of internal laceration, these would be more consistent with homicide. The position of the body will throw very little light upon the question, since it is fully demonstrated that complete suspension is not necessary to produce death. In numerous instances the body has been found, after death, resting upon the knees, the toes, or the buttocks, or semi-recumbent, and in one case entirely supported by the bedstead, while the neck rested in a loop of leather.
Even if the hands and feet are found tied, the inference is not warranted that the act was homicidal, since determined suicides have been known to perform this very act previous to hanging themselves. Nevertheless, if a person be found with his hands and feet tied, and suspended from a position which obviously he could not have reached himself, the presumption of homicide would certainly be justified.

The age of the deceased might be supposed to assist in solving the question. If a very young person were discovered dead from hanging, it would naturally be attributed to homicide; yet numerous instances have occurred in this country, within the last few years, of suicidal hanging of children not over twelve or fourteen years of age.

Cases of accidental hanging are of occasional occurrence, especially among children, who have, while swinging, or otherwise playing, accidentally become entangled in a noose or loop of cord, which was then drawn tightly enough around the neck to strangle them.

SECTION IV.

DEATH BY DROWNING.

MODE OF DEATH IN DROWNING.—TIME REQUIRED.—SIGNS OF DEATH, EXTERNAL AND INTERNAL.—ACCIDENTAL, SUICIDAL AND HOMICIDAL DROWNING.

Drowning is that special form of death by suffocation, in which the breathing is arrested by water, or some other liquid, and even more effectually than by a ligature drawn around the neck. It is not necessary that the whole body should be submerged in order to cause death by drowning. This may be accomplished by merely immersing the face, so as to keep the nose and mouth under the liquid, as is wit-
DROWNING.

nessed in the case of drunkards, epileptics and very young children falling with their faces into very shallow pools, and perishing from inability to extricate themselves. In drowning, in addition to the usual cause of death by asphyxia—the deprivation of air—there is superadded the physical impediment of the introduction of water into the minute air tubes and vesicles of the lungs by aspiration, in the violent efforts of the person to breathe. This is demonstrated by the experiments made by the Committee of the Medico-Chirurgical Society of London. Two dogs of the same size were submerged at the same moment, but one had his windpipe plugged, so as to prevent the ingress of both air and water, while the other had not. After two minutes they were taken out together; the one with the windpipe plugged recovered at once on removing the plug; the other died. In three experiments, dogs with their windpipes plugged were kept under water four minutes, and recovered perfectly on being taken out (Report on Suspended Animation, Med. Chir. Trans., 1862, p. 449). On inspecting the bodies of the animals, the difference was at once manifest; in those that were simply deprived of air by plugging the windpipe, the lungs were congested; but in those that had been submerged in their ordinary condition, i.e. actually drowned, the lungs, besides being congested, exhibited in their bronchial tubes and air vesicles a bloody, frothy mucus, which completely filled the air vesicles and small tubes, forming a mechanical impediment to the ingress or egress of air. The lungs were sodden with water, heavy, soft and doughy to the feel, and pitted on pressure of the finger. In the lungs of animals that recovered after a short submersion, very little, if any, of this mucous froth was found; its amount was always proportionate to the time of submersion. There is no doubt that this froth is produced by the violent
efforts to breathe which are made within a minute after submersion.

Hence, the probability of recovery after drowning is mainly dependent upon the quantity of this mucous froth existing in the air tubes and vesicles of the lungs, and also of the water that has penetrated into the substance of the lungs. If the quantity is large, the result is almost certainly fatal; if it is small, there is always good hope of recovery. Asphyxia occurs in a minute, to a minute and a half after submersion. If the submersion has been complete for four minutes, the case may be considered hopeless, unless syncope had occurred at the moment of entering the water. This, by partially suspending the attempts at respiration, would undoubtedly tend to prolong life for some minutes longer. Cases are reported of resuscitation after being fifteen minutes under water; but these are exceptional. It should be remembered that the heart may continue to beat some minutes after respiration has ceased; but, in the present instance, the pulsation of the heart is no criterion of the power of recovery, on account of the physical impediment in the lungs just alluded to.

Dr. Taylor gives the following excellent résumé of the circumstances attending on a case of drowning: "When a person falls into the water and retains his consciousness, violent attempts are made to breathe; at each time that he rises to the surface a portion of air is received into the lungs, but, owing to the mouth being on a level with the liquid, water also enters and passes into the throat. A quantity of water thus usually enters the mouth, which the drowning person is irresistibly compelled to swallow. In his efforts to breathe while his head is below the water, a portion of this liquid is drawn into the air-tubes and cells of the lungs. The struggle for life may continue for a
longer or shorter period, according to the age, sex and strength of the person, but the result is that the blood in the lungs is imperfectly aerated, the person becomes exhausted, and insensibility follows. The mouth then sinks altogether below the level of the water; air can no longer enter into the lungs; a portion of that which they contained is expelled, and rises in bubbles to the surface; an indescribable feeling of delirium, with a ringing sensation in the ears, supervenes; the person loses all consciousness, and sinks asphyxiated. In the state of asphyxia, while the dark-colored blood is circulating, convulsive movements of the body take place, and the contents of the stomach are sometimes ejected by vomiting. There does not appear to be any sensation of pain, and, as in the other cases of asphyxia, if the person recovers, there is a total unconsciousness of any suffering” (Med. Jurisp., Am. ed., p. 416).

Even after resuscitation from drowning, death frequently takes place within a few hours or days, from secondary causes, as exhaustion, obstruction to respiration from the condition of the lungs, convulsions and spasm of the glottis.

**Signs of death by Drowning.—1. External.** These vary, according to the length of time the body has been in the water, and the interval after it was taken out. Supposing the immersion not to have been over two or three hours, and the inspection to be made immediately, the face will be found to be pale, the expression placid, the eyes half open, the eyelids livid, and the pupils dilated, the mouth half closed or open, the tongue swollen and congested, often indented by the teeth, and perhaps lacerated; the lips and nostrils covered with a mucous froth, which issues from them. The skin is cold and pale, and generally contracted so as to present the appearance of "goose skin" (cutis anse-
This, being a vital act, is a pretty sure sign that the body was living when immersed in the water. It is not dependent on cold, as was at one time supposed. In males, the *retraction of the penis* is considered by Casper and Kanzler as a very positive sign of drowning.

Besides the above, there are sometimes seen marks of abrasion on the body, especially on the hands, together with sand, gravel or mud under the nails, weeds, pieces of wood or other matters locked in the hands, all of which would seem to indicate that the person had been alive when first immersed in the water, although the *abrasions* might very possibly have resulted from the body rubbing against some rough substances after death. After several days' immersion, the palms of the hands and soles of the feet become white, thickened and sodden, the result of imbibition.

If putrefaction has commenced before the body is removed from the water, the face will have assumed a reddish, or bluish-red coloration.

2. *Internal.*—Along with the usual evidences of death from asphyxia (in an early examination), the following signs will be observed: the lungs are distended, overlapping the heart, and are in a flabby condition; this latter is owing to the water taken in by aspiration, during the struggles for breath, which penetrates even the air vesicles, and renders them sodden and doughy. When cut into, the lungs exude a bloody, mucous froth. *The presence of this froth in the smaller tubes and air cells, together with the sodden condition of the lungs,* is regarded as one of the most positive signs of death by drowning. Nevertheless, its absence should not be accepted as a proof against drowning, since it has not been found in the bodies of persons who have sunk at once in the water, and never risen to the surface to breathe. Dr. Ogston states that in 48.7 per cent. of cases, no water
was found in the lungs, and he accounts for its absence by its transudation from the lungs into the pleural cavities, where it was found in quantities varying from one to thirty-four ounces. In a case examined by the author a year ago, of the body of a woman taken out of the river Delaware, there was an absence of this characteristic froth in the minute bronchial tubes, and also of the peculiar flabby condition of the lungs. The absence of these same peculiarities in the lungs of the deceased, in the late celebrated Jennie Cramer case, at New Haven, Ct., created a doubt in the minds of many that it was not a case of suicide by drowning, but that the girl was murdered before the body was thrown into the water. The fact of the discovery of a considerable amount of arsenic in the body of the deceased was, of course, sufficient to account for the death. We believe that these peculiar conditions of the lungs of the drowned have not yet been sufficiently determined. It is quite possible that some cases of bodies taken out of the water, and reported by coroners' juries as "found drowned," may in reality have met their death by other means, prior to their immersion. It is important to remember that the presence of this mucous froth in the air-passages is not seen after putrefaction, or after long exposure of the body to the air: this may account for the fact of its occasional non-observance in the bodies of the drowned.

Another important indication of death by drowning is the presence of water in the stomach, which had been swallowed in the act of drowning, especially if this corresponds with the water in which the body was found. The value of this is enhanced, if, along with the water, there be discovered in the stomach fragments of weeds, sand, mud or other articles, corresponding with the like substances existing in the pond or river where the drowning occurred. The quantity of
water in the stomach varies considerably; it was found to be greater in an animal that was allowed to come to the surface frequently, than in one kept completely submerged, because in the latter the power of swallowing was sooner lost, in consequence of the early occurrence of asphyxia.

The absence of water from the stomach is not to be considered as disproving a case of drowning, inasmuch as it is not present (because not swallowed) in cases where either syncope or apoplexy had occurred at the moment of immersion.

The mere discovery of water in the stomach is not of itself a positive indication of death by drowning, since it may have been swallowed before immersion; but with this allowance, and with the restrictions above mentioned, it does constitute a very important sign, inasmuch as it has been ascertained by experiment that water will not penetrate into the stomach after death, unless putrefaction has advanced to a great extent; consequently, its presence indicates pretty certainly that it had been swallowed in the act of drowning. Orfila's experiments prove that water may penetrate into the larger bronchial tubes after death, but not into the air vesicles of the lungs; besides, in such cases, there is no accompaniment of mucous froth in the air tubes.

The condition of the heart affords no positive indication of death by drowning. In the majority of cases the right cavities are full, and the left ones empty, as in asphyxia generally; but very often the two sides are equally full.

The brain exhibits no characteristic post-mortem sign. There may be some general fullness of the vessels, but never extravasation of blood, unless a sudden apoplexy had supervened, as when a person plunges suddenly into cold water after eating heartily, or by striking the head against a hard body, in the act of diving. The blood is usu-
SIGNs OF DROWNING.

ally dark and fluid. The mucous lining of the stomach and bowels is usually congested, and if the body had been long in the water, of a deep violet color; this might lead to the suspicion of irritant poisoning. Occasionally, in cases of drowning after a full meal, vomiting occurs, and the contents of the stomach are found in the windpipe and lungs; this is a conclusive evidence that the person must have been alive at the time.

The time at which the bodies of the drowned will float varies with the temperature of the air, the water, the age, sex and corpulence of the person, etc. As the human body is slightly heavier than water, it must remain submerged until it becomes lighter, through the development of the gases of putrefaction. Hence, in summer, the body may rise within twenty-four hours. In salt water, it will float sooner than in fresh; very fat bodies float sooner than lean ones; the bodies of women sooner than those of men.

To determine the time that has elapsed since the act of drowning, when the body is discovered in the water, is not always possible. After putrefaction has set in, it is altogether mere guesswork. The most certain criteria to guide the examiner are the presence of the mucous froth in the air tubes and cells, and the presence of water in the lung tissue, both of which indications disappear after long exposure to the air, and after putrefaction. Hence, the importance of an early inspection.

If marks of violence be found on the bodies of the drowned, of course, suspicion will be aroused of foul play, unless these marks can be satisfactorily attributed to some post-mortem cause. A murderer may destroy his victim, and then throw the body into the river, pond, or well, with the intention to elude suspicion of the real cause of death. A close examination of the body for wounds, and other
injuries, together with the absence of the known signs of drowning, will generally enable the examiner to form a correct conclusion. This is a very common method of disposing of the bodies of new-born infants, in cases of infanticide.

The question of accident, homicide or suicide, in the case of drowning, must claim the attention of the legal physician. *Homicidal* drowning is rare, except in the case of infants. It is denoted by the marks of violence on the body, which cannot be explained by any post-mortem influence. It should be remembered that determined suicides frequently inflict dangerous wounds upon themselves, and then terminate their lives by drowning. Such cases might possibly be mistaken for homicide. The presence of the usual signs of drowning would at least show that the body was alive at the time of immersion. *Suicidal* and *accidental* drowning cannot always be distinguished from each other; inferences may, however, be drawn from the circumstances attending the cases, as the existence of a motive to suicide, or a tendency thereto; the proximity of a precipice, or other dangerous place, to the water in which the deceased was found would naturally suggest *accident*, especially in the case of a child. The tying of the hands and feet of a person found dead in the water is no proof of homicide, since many instances are recorded of suicides binding themselves in this same manner, and also of attaching heavy weights to their bodies before throwing themselves into the water.

*The restoration of the drowned* depends chiefly on exciting artificial respiration. The clothes should be immediately removed, and the body quickly wiped dry and wrapped in a blanket; clear the mouth and nostrils of mucus and water; draw forward the tongue; place the body with the face downward, the forehead resting on one arm, for a few moments,
to allow the fluids to run out of the mouth; apply ammonia cautiously to the nose. If respiration is not restored, place the body on the back, with the head raised, and adopt Sylvester's process of artificial respiration, by carrying the arms gently outward and upward above the head, for a few seconds: this movement expands the chest. Then lower the arms, and bring them to the sides of the chest: by this action, expiration is effected. These alternate movements should be made each about every two seconds. All rough handling, such as the absurd, vulgar plan of rolling on a barrel, should be avoided. As soon as any signs of respiration are manifested, warmth should be applied to the skin by a warm bath, or stimulating friction. When able to swallow, the patient may take a little warm spirit and water, and then be put to bed and allowed to sleep. This treatment has been rewarded with success after being persisted in for some hours.
CHAPTER X.
DEATH BY LIGHTNING.

MEDICO-LEGAL RELATIONS OF DEATH FROM LIGHTNING.—MODE OF DEATH.—POST-MORTEM SIGNS.

Death caused by Lightning is often accompanied by results which resemble very strongly the effects of homicidal violence. The subject should, therefore, claim the attention of the legal physician, inasmuch as he may be called upon to determine the cause of death in an unknown case, when the body has been discovered in a remote and solitary situation, and bearing upon it marks of severe external injury.

The destructive effects of lightning exactly resemble those of a powerful electric battery, thus demonstrating the identity of the two forces. In a thunder-storm the electric condition or polarity of the cloud is nearly always positive, while that of the earth immediately beneath it is negative. When these polarities become intensified by mutual induction, the disruptive discharge ensues through the air, or any other body that may happen to intervene—the human body, for example.

The only rational explanation of the fact that more men than women are killed by lightning is, that the former, from the nature of their employments, are mostly out of doors, and are, therefore, more exposed to the danger. Experience proves that persons in the open fields, especially under trees, are much more liable to be struck by lightning than those within doors.

The fatal effects of lightning are usually instantaneous,
death being caused by shock. At times, however, it produces lesions of the brain and spinal cord, such as epilepsy, paralysis, effusion of blood, tetanus, etc., which may subsequently prove fatal. Generally speaking, if death does not follow immediately, or soon after, there may be hopes of recovery.

The visible effects produced by a fatal lightning-stroke are remarkably varied. Sometimes, a deep, punctured or lacerated wound will indicate where the fatal blow was struck, upon the head, neck, or other part of the body; the hair may be singed, or burnt off; the clothing may be burned, or completely stripped off; the boot may be split open. Again, the course of the electric current may be marked by a deep, or superficial burn, extending from the point of entrance, down and around the body, to the ground. If there should happen to be any metallic substances in contact with the body, such as chains, coins, a watch, etc., as these are good conductors of the electric current, it will be certain to include them in the circuit, and they will be frequently found to have been melted.

In other cases of death by lightning, no external wound or burn may be visible. Sometimes there may be severe external injuries, while the clothes entirely escape. Again, the clothing may be completely torn off the body, while the latter exhibits no injury whatever.

The capricious action of the discharge is shown by the fact that out of a party of three or four sitting under a tree, one or two only may be killed, and the others escape. Again, it has occurred that persons under a low tree have been struck, although high trees, and a lightning rod, and an iron bridge were near (Tidy). Again, the same discharge may produce in one person wounds, and burns in another. The diversity of its action on the clothes may
probably be explained by the circumstance of a portion of the clothing being _wet_, and a portion _dry_: the former, being a good conductor, might escape the disruption which would be exhibited by the dry portion, which is a bad conductor.

_Post-mortem appearances._—In case of instantaneous death, the body may be found in the exact attitude in which it was struck. Some remarkable instances of this are recorded in the books. In such cases, the rigor-mortis occurs immediately after death. Hunter supposed that there was an absence of the usual rigidity after death, but in this he was in error. Coagulation of the blood also occurs, although it is delayed. The face is often bloated and discolored; and putrefaction is usually very rapid. Wounds of various characters are observed—contused, lacerated and punctured; also burns, vesications and ecchymoses; these latter sometimes exhibit a remarkable arborescent appearance. Occasionally, fractures of the skull and of other bones are noticed. The blood is dark and fluid.

The brain and its membranes generally suffer most severely, the head being usually the part first struck. Congestion of the brain, effusion of blood under the skull and into the ventricles, and even complete disorganization of the brain substance, have all been observed. The lungs are sometimes found congested and injured, and the air tubes full of mucus. The stomach, intestines, liver and spleen, are also usually much congested. The heart does not exhibit any special alteration.

The medico-legal interest, in cases of death from lightning, is centered in the question of being able to identify such cases, and to distinguish them from those of homicidal violence. A close observation of all the circumstances of
the case—such as the occurrence of a thunder storm about the time of the death, the peculiar appearance of the wounds and burns, especially if the two co-exist on the same body, the half-melted appearance of metallic articles, such as buttons and coins, on the person of the deceased, etc.—will tend to throw much light upon it.
CHAPTER XI.

DEATH FROM HEAT AND COLD.

DIVERSE EFFECTS OF HEAT UPON THE BODY.—POST-MORTEM APPEARANCES.—EFFECTS OF COLD.—POST-MORTEM SIGNS.

The effects of extreme heat on the human system are familiarly witnessed in tropical and semi-tropical climates, during the heated term, in the mortality arising from what is popularly denominated sunstroke. In such cases, the dangerous and fatal results are attributable directly to solar heat. But effects equally serious are known to be produced by exposure to artificial heat, if too long continued, as is witnessed in the employés in engine-rooms, factories, etc., where a very high temperature is habitually maintained. There would seem to be, according to the observations of Dr. H. C. Wood (Phila. Med. Times, 1876), three distinct conditions of the human body occasioned by excessive heat, in the first (which is rare), we have acute meningitis or phrenitis (coup-de-soleil); in the second, we have heat-exhaustion with collapse, accompanied by a rapid, feeble pulse, a cool, moist skin, and a tendency to syncope; in the third, we have true thermic fever—that condition which results especially from exposure to artificial heat. But something more than mere heat is required to produce thermic fever. It does not occur in a perfectly pure and dry atmosphere, because the profuse perspiration which is immediately developed by its rapid evaporation keeps the temperature of the body down nearly to the normal standard. If, however, the air is already saturated with
moisture, this will prevent the evaporation from the body, and its temperature will rise to a dangerous height.

The symptoms vary in intensity, from a mere headache with drowsiness, to complete insensibility, coma, and paralysis. In many instances, death appears to be caused by paralysis of the heart.

The post-mortem appearances are by no means constant. In some cases (true coup-de-soleil) we find decided congestion of the brain and its membranes, with serum in the ventricles, together with congestion of the lungs and of the abdominal viscera generally; and the heart, as in ordinary death from asphyxia. In other cases, there is anaemia of the substance of the brain, along with distention of the larger vessels with dark, fluid blood, but the minute vessels empty.

Cases of insolation do not often claim the attention of the legal physician, yet as they might occur remote from witnesses, and with a fatal termination, it is proper that the medical examiner should understand their nature, together with the ordinary accompaniments.

The effects of cold upon the animal body are immediately depressing, but if it be of short duration, and the system is healthy, reaction takes place and stimulation follows. The healthy human body has the power to maintain its normal temperature of about 98.6° F. independently of the external temperature. It has been ascertained by actual experiment that a warm-blooded animal will not survive if its temperature is reduced down 16 to 20 degrees below the normal. There is no authentic account of the recovery of a warm-blooded animal, much less a human being, after the whole body was frozen, although fishes and other of the lower animals are said to have been resuscitated from a frozen state.
Death from cold is hastened by whatever exhausts the system, as fatigue, both bodily and mental, loss of rest, want of proper food and nourishment, mental depression, and particularly intoxication. A damp cold (such as wet clothing) is more dangerous than a dry one. The fatal effects of exposure to cold are witnessed, even in comparatively temperate climates during the winter, in the cases of the destitute, and especially where this condition is associated with intemperance.

Cases of death from cold do not often require the attention of the medical jurist. There are, however, certain conditions under which they may occur, which demand a brief consideration.

A not infrequent form of infanticide is the exposure of a new-born child to the extreme cold air. Death will soon ensue under such circumstances, since the infant's power of resistance to cold is extremely limited. In such a case it will be the physician's duty to examine the body of the child, and consider the circumstances of the case, such as the place where it was found, the temperature of the air, the possibility of its being accidental, etc. As regards the body, he should notice if the pallor is extreme; if frozen stiff, he should distinguish this rigidity from rigor mortis; the arterial color of the blood; the accumulation of blood on both sides of the heart and in the larger vessels. There may also be marks of violence upon the body.

Occasional instances of the exposure of young children to cold with homicidal intent, are recorded. Such a case is related (Ann. d’Hyg., 9, 1831, p. 207), of two inhuman parents causing the death of a daughter, aged eleven years, by compelling her to get out of bed on a very cold night, and place herself in a vessel of ice-cold water.

In the treatment of the insane, the barbarous and im-
proper use of the cold shower bath, for reducing intractable patients to submission, was formerly much more in vogue than at present. It need hardly be said that such treatment is extremely hazardous, and it has been followed by fatal results. Dr. Taylor records an instance of a lunatic, aged sixty-five, who was subjected to the cold shower, at 45° F., and who afterwards took a dose of tartar emetic; he died in fifteen minutes subsequently. Cases of this character would very properly come under the notice of the legal physician, and the authors of such treatment would be justly liable to indictment for manslaughter.

Post-mortem appearances.—These cannot be considered as very characteristic; hence, the examiner should be cautious in deciding, in any given case, as to whether exposure to cold was the primary cause of death. All the circumstances of the case here require special consideration, such as the season of the year, the temperature of the air, the place of exposure, etc. Rigor mortis generally sets in slowly, and lasts a long time. According to Dr. Ogston (Brit. and For. Med.-Chir. Review, 1855), the four following appearances, in the absence of any other obvious cause, would justify the conclusion that the death had resulted from cold, although the signs were not so well marked in children as in adults:—

(1) An arterial hue of the blood, except when viewed in mass within the heart; some exceptions are, however, noted.

(2) An unusual accumulation of blood on both sides of the heart.

(3) Pallor of the general surface of the body, and congestion of the viscera most largely supplied with blood. In some cases the congestion of the brain and liver was only moderate.
(4) Irregular and diffused dusky-red patches on limited portions of the exterior of the body, even in non-dependent parts (distinguishing them from suggillations).

As putrefaction does not occur at a freezing temperature, the discovery of a decomposing corpse in the ice or snow would afford a very strong, though not absolutely conclusive, evidence that the death was not the result of exposure to cold, but rather that the body had been frozen after death.
CHAPTER XII.

DEATH BY STARVATION.

ACCIDENTAL, HOMICIDAL AND SUICIDAL STARVATION.—PRETENDED CASES OF VOLUNTARY STARVATION.—SYMPTOMS AND POST-MORTEM SIGNS.—MEDICO-LEGAL RELATIONS.

Cases of death by Starvation are of sufficiently frequent occurrence to merit the notice of the medical jurist. *Acute starvation* implies the sudden and complete deprivation of all food. *Chronic starvation* is the result of a continued deficient supply of food, both in quantity and quality. *Homicidal* death from acute starvation is very rare, but cases of accidental death from this cause are sufficiently numerous, as in the instances of miners buried in the earth, shipwrecked mariners, and others cut off from food. Occasionally, prisoners and lunatics will undertake to commit suicide by voluntary abstinence from all food; in the great majority of cases, however, their courage fails them after some days' experience, and they give up the attempt.

The many notorious cases of voluntary fasting which have claimed the notice of the public during the past years, have proved, on close investigation, to be deceptions, food and drink having been supplied surreptitiously to the individuals concerned. Among these instances may be mentioned the case of Ann Moore, of Tetbury, England, who was alleged to have abstained from all food from 1807 to 1813. Another case was that of the *Welsh Fasting Girl*, aged thirteen years, who is stated to have absolutely fasted for two years. Both these cases were shown to be impostures. The notorious Dr. Tanner, of our own country, undertook, for a considera-
tion, to perform the feat of a *forty days' absolute fast*, in New York, in August, 1880, and, *to all appearance*, he accomplished it! It is stated that during all this time, he absolutely partook of nothing, save some ounces of pure water, each day; and that his loss of weight at the end of forty days was thirty-six pounds. The fluctuations in his pulse, temperature and respiration were unimportant. This case was not under very strict medical supervision, and there is doubt about its perfect genuineness; this would seem to be confirmed by the fact of his voracious appetite on the completion of the fast, unattended by any bad effects, which is contrary to the general experience of others who have been deprived of food for a length of time.

*Chronic starvation*, as the result of disease, is a frequent cause of death, as is witnessed in stricture of the cesophagus, cancer and other disorders of the stomach and bowels, disease of the pancreas, marasmus, etc. It is likewise the cause of disease and death in young children fed upon unhealthy milk (either from the nurse or cow), where this fluid is deficient in some of its proper constituents, thereby causing defective nutrition. Such cases are abundantly illustrated in the miserable victims of *baby-farming*. It is also witnessed on a large scale in districts of country where famine has prevailed, as in certain parts of India, and in the Irish famine of 1847.

The *symptoms* of chronic starvation are generally well-marked. The sense of hunger is not very urgent; emaciation, especially in the last stage, is extreme; the eyes are hollowed, the pupils dilated; the skin is harsh and dry, and hangs loosely over prominent bones, and, in chronic cases, becomes covered with a brownish, dirty-looking coating, and exhales an offensive odor, like that of putrefaction. The bowels are either very constipated, or the faeces are
scanty, dry and dark colored. There is great muscular debility, palpitation, with tinnitus aurium; pains in the stomach, with a dry, parched mouth; the intellect sometimes clouded, but again clear to the end, with despondency of mind. The pulse is at first somewhat quickened, afterward it is slower; the temperature is usually below that of health.

Post-mortem appearances.—Great emaciation of the body, with an almost entire loss of fat. The skin shriveled, and emitting a disagreeable odor. The muscles soft, pale and wasted. The brain sometimes congested, and at others pale and soft, with effusion of serum on the surface, and in the ventricles; the lungs healthy, or anæmic; the heart more or less contracted, and void of blood; stomach and intestines contracted, thin and transparent, the latter usually empty; the bladder contracted and empty.

As regards the medico-legal relations of starvation, although it is rarely the cause of homicidal death, it should be remembered that the law does not require the absolute deprivation of food to be proved, but only the necessary quantity and quality to be withheld, provided this has been done with an evil intention.
CHAPTER XIII.
DEATH FROM POISONING.
(TOXICOLOGY.)

Poisoning is the most frequent of all the causes of violent death (the casualties of war excepted), as is shown by the statistics of different countries. The facility with which poisons may be procured, the ease with which they can be administered, and the close resemblance that many of them bear to disease in their symptoms and post-mortem lesions, will account for the fact of their extensive employment, both for homicidal and suicidal purposes.

The science of Toxicology, which treats of the nature, symptoms, effects, doses and modes of detection of poisons, is very properly included in a treatise on Medical Jurisprudence; and since, as already remarked, so large a proportion of violent deaths is to be ascribed to poisoning, it is important that the medico-legal student should be properly instructed in this branch of the subject.

SECTION I.
DEFINITION OF A POISON.—EFFECTS.—PROOFS OF ABSORPTION.—SUBSEQUENT DISPOSITION OF THE POISON.—ELIMINATION.—HOW DO POISONS CAUSE DEATH?—CIRCUMSTANCES MODIFYING THEIR ACTION.—ANTAGONISM OF POISONS.

A Poison is a substance which, when introduced into the body by swallowing, or by any other method, occasions disease or death; and this as an ordinary result, in a state of health, and not by a mechanical action. It must be as an ordinary result: a substance, for example, which affects one person injuriously, through idiosyncrasy, is not a poison.
Again, it must be *in the healthy system*: many diseases render the system extremely susceptible to impressions by external agents; *e. g.*, in gastritis, the blandest substance—even water—may excite vomiting. Again, the substance must not act *mechanically*: thus, powdered glass, fragments of iron, etc., may produce death when swallowed, yet these cannot be regarded as poisons.

According to the above definition, it matters not by what avenue a poison gains access into the body, its ultimate effects are the same. The stomach, of course, is the most usual means; but the rectum, the skin, the lungs, and the cellular tissue by hypodermic injection, and even the nose, ear, and vagina are also channels of entrance. Inhalation of poisonous vapors through the lungs, and the subcutaneous introduction by the hypodermic syringe, affect the system far more rapidly than by swallowing.

The mere *size* of the dose constitutes no distinction, legally, between a poisonous and a non-poisonous substance; thus, half a grain of strychnia, or half an ounce of oxalic acid, may be the quantity which proves fatal.

*The Effects of Poisons* are local and remote. The *local* effects are the direct impressions produced on the part of the body with which the poison comes into contact, *e. g.*, the corrosion of the stomach and bowels by the immediate contact of the mineral acids and alkalis. Often a poison may act both *locally*, by its causing inflammation of the stomach, and also *remotely*, on the brain and nervous system. Arsenic frequently acts in this twofold manner.

*The remote* effects of a poison are those results which are produced on parts of the system remote from that to which it was first applied. These remote effects constitute, in fact, the usual *symptoms* of poisoning—one very important factor in the diagnosis of the case.
Mode of Action of Poisons.—In order that a poison should produce its peculiar effects on the system, it is necessary (except in the case of *corrosives*) that it should get into the circulation, so as to be conveyed to distant parts of the body; and for this purpose it must first be absorbed. Although other modes of transfer of the poisonous impression to remote parts of the system have been, at various times, recognized—such as nervous communication, and contiguity of structure—the present accepted doctrine is that of *absorption into the circulation*.

The proofs of absorption are abundantly afforded, (1) by the detection of poisons in the blood; (2) in the secretions, especially the urine; and (3) in the different viscera of the body, as the liver, kidneys, lungs, spleen, brain, etc. An essential part of the duty of the toxicologist is not merely to discover the poison in the stomach of the deceased (since that might possibly have been introduced after death), but to detect it, *in the absorbed state*, in the viscera.

The *rapidity* of absorption is materially influenced (1) by the solubility of the poison; (2) by the *nature of the surface to which it is applied*, it being in direct ratio to the vascularity of the part. It is for this reason that the most rapid absorption is from the air-cells of the lungs, when the substance is inhaled in the form of vapor. For this same reason, also, when it is injected directly into the blood vessels, the effect is almost instantaneous. Certain animal poisons, such as the virus of glanders, syphilis, smallpox, etc., *when swallowed*, appear to undergo a change, through digestion, which renders them innocuous. The absorption of poisons from the stomach is modified by the full or empty condition of that organ—being most rapid when it is empty. The *sound skin* may sometimes become the avenue for the introduction of poisons, as witnessed in the absorption
of arsenic, tartar emetic, corrosive sublimate and opium, when applied to that surface. By removing the cuticle, the absorption is much more rapid, as seen in the endermic method. (3) Fullness of the blood vessels. The rapidity of absorption is inversely to the quantity of the circulating fluid; hence, depletion by bleeding or purging will favor absorption.

Subsequent disposition of the Poison.—After absorption into the blood, as it passes through the different organs, a portion of the poison is immediately separated by these, and is at once eliminated by the various secretions, as the bile, urine, saliva, pancreatic fluid and sweat. Another portion is temporarily deposited in the organs and tissues, and usually in the following order, as to quantity: the liver, spleen, kidneys, heart, lungs, brain, pancreas, muscles and bones. To this order there may be occasional exceptions, as some recent experiments seem to prove that lead and certain other mineral poisons show an especial affinity for the spinal marrow and brain. Only a minute quantity of the poison is circulating in the capillaries at any one time; yet there is good reason to believe that it is exclusively this portion which is really noxious; while still remaining in the stomach, or retained in the organs, it is harmless. Hence, it is a common mistake to attribute death to the actual quantity of the poison found in the stomach of the deceased; this is only the surplus, or complement of what was necessary to kill. Although that portion of the poison which is retained in the organs (absorbed) is, for the time being, innocuous, yet, as it is liable to be reabsorbed into the circulation, it may again prove active.

While we have no positive proof that all poisons are deposited in the organs, we know that this is true of the mineral, and of some of the vegetable poisons. The gaseous
poisons appear to be eliminated by the lungs immediately, without this deposition. This was proven by Bernard's experiments with sulphuretted hydrogen on dogs (*Leçons*, p. 59).

The time required for an absorbed poison to be removed from the circulation, either by elimination or by deposition in the organs or tissues, varies for different substances, and also, probably, for different conditions of the system. Certain medicinal substances are known to appear in the urine a few minutes after being swallowed, e.g. iodide of potassium and turpentine. In relation to mineral poisons, there is reason to believe that they are rapidly separated from the blood. Experiments have shown that arsenic may be diffused throughout the body of an animal in an hour and a half after being swallowed. It has also been found in the urine of a horse within one hour after administration. Prof. Taylor found arsenic in the human liver *four* hours after being swallowed. Doubtless, it reaches this organ much sooner, although no opportunity has as yet been afforded of proving the fact, since death rarely occurs sooner than the above period. Taylor believes that the liver acquires its maximum of saturation by arsenic in fifteen hours after being swallowed. He gives a table of the estimated average amount of this poison that will be found in this organ at different periods: In five to seven hours after taking, the quantity is 0.8 grain; in nine hours, 1.2 grains; in fifteen hours, 2.0 grains; in seventeen to twenty hours, 1.3 grains; in fourteen days, 0.17 grains. It is generally admitted that arsenic is entirely eliminated from the human system in about *fifteen days*; but cases have been reported where the poison was detected in the urine as late as the twenty-fifth day. As a rule, the analyst need hardly expect to find any traces of arsenic in the body of a person who has survived fifteen days.
Other mineral poisons require a longer time for their elimination from the human body. According to M. L. Orfila, arsenic and corrosive sublimate require thirty days; antimony, four months; silver, five months; lead and copper, over eight months. (Tardieu *sur l’Empoison.*, p. 19.)

The *mode of death* by poisons has been a subject of much discussion. It must be admitted that we are not in possession of the full explanation of this subject. We know that the various poisons circulate through the blood, and thus come in contact with the different organs, and then produce their specific effects—one, as opium, on the brain, causing narcotism; another, as prussic acid, on the heart, producing asthenia; a third, as strychnia, acting on the spinal cord, causing tetanus, etc.; but why they possess this elective affinity for different organs, we are unable to explain. Neither do we understand why different poisons exhibit a similar election in their modes of elimination from the system, *e.g.* iodide of potassium passing out, by preference, through the urine; mercury, by the saliva; arsenic, by the glands of the stomach, etc.

It has been supposed by some that the poison produces some *chemical* alteration in the blood, thereby rendering it unfit for life. This, however, cannot be proved, although it is true that some poisons, when introduced into the circulation, do undergo a chemical change, as chloroform into formic acid, and the salts of the vegetable acids passing out through the kidneys as *carbonates*; so, also, a combination of emulsin and amygdalin, when injected into the blood, results in the production of prussic acid. This, however, fails to explain the true *modus operandi* of poisons.

*Modifying circumstances connected with poisons.*—Some of these relate to the poison itself, and others are connected with the system. Among the former, the *dose*, and *mode of*
administration require notice. As a rule, the larger the dose the more speedy the action. An exception to this is seen in the case of some irritants, such as arsenic, where a large dose may be rejected by vomiting, and might thus prove innocuous, whilst a smaller one would be retained. The effect of some poisons is much modified by the dose; thus, a large dose of oxalic acid kills almost immediately by shock, while a smaller one will act upon the heart and nervous centres, and prove fatal later.

The effect of combination of poisons is sometimes to increase, sometimes to diminish, their activity, and again, to antagonize, or neutralize their action. According to Christison, the effects of arsenic are decidedly modified by intoxication, which seems in some way to arrest or suspend its action. This is also probably true of other irritant poisons. The same authority mentions a case where a very large dose of corrosive sublimate and laudanum was taken, and there was a remarkable postponement of all the usual symptoms.

The question of the Antagonism of Poisons—that is, whether, by a combination of poisons, their action upon the human system will become so modified as to conceal the symptoms, and prevent their discovery after death by the usual chemical tests—is one of considerable medico-legal importance. This doctrine was for the first time, we believe, in this country, urged with some apparent plausibility at the celebrated trial of Dr. Paul Schöpppe, at Carlisle, Pa., in 1869. After the failure by the prosecution to establish the allegation of poisoning by prussic acid, it set up the claim that the death was produced by a mixture of this poison and morphia, and ascribed the absence of all the usual symptoms of prussic acid poisoning, and the failure to
detect either it (except by the merest trace, which was shown might result from the faulty method of the analysis) or the morphia, to the alleged antagonism of the two substances! In the year 1870, the author made a number of experiments upon dogs, with a view of determining this question. A few of the results will be briefly detailed here.

(1) Morphia and Prussic Acid.—If both poisons were given in full lethal doses, the symptoms of both toxic agents were exhibited. The morphia never counteracts the fatal effects of the prussic acid, if the latter be taken in full poisonous doses.

(2) Morphia and Atropia.—The mutual antagonizing influence of these two alkaloids is now fully recognized in the human subject; but it is less manifest in dogs.

(3) Strychnia and Prussic Acid.—These powerful poisons evince no real antagonism. When both were taken in full doses, the usual symptoms of each were exhibited alternately—ordinary convulsions and tetanic spasms.

(4) Strychnia and Morphia.—These alkaloids show no disposition to antagonism, when given in full doses. The narcotism of the morphia (taken first) was speedily followed by the tetanus of the strychnia (taken afterwards).

(5) Atropia and Eserine.—The investigations of Dr. Frazer with these substances, on dogs (Trans. Roy. Soc. Edin., Vol. XXVI), demonstrate a real antagonism, which was confirmed by the author's experiments.

(6) Atropia and Strychnia.—There would seem to be a true antagonism between these two alkaloids, sufficient to justify a resort to the use of atropia in a case of strychnia-poisoning.

There also appears good reason for admitting the antagonism between Aconite and Digitalis—sufficiently so to warrant a trial of digitalis in a case of poisoning by

The conditions of the system that modify the action of poisons are, habit, idiosyncrasy, and disease. Habit usually diminishes the power of poisons, as shown especially in the case of the narcotics opium and alcohol. It is also alleged to be true in the case of arsenic, as seen in the arsenic-eaters of Styria, and other mountainous countries.

The effect of disease in modifying the action of poisons is witnessed in the tolerance by the system of opium in tetanus and mania-a-potu; and of its increased susceptibility to this drug in apoplexy and inflammation of the brain. In paralysis, the susceptibility to the action of strychnia is diminished.

The influence of sleep is usually to diminish, or retard the action of poisons. This is true of arsenic and the irritants generally. The narcotism produced by opium seems to produce a similar effect, and also to mask their symptoms.

SECTION II.

EVIDENCES OF POISONING.

1. EVIDENCES FROM SYMPTOMS.—2. FROM POST-MORTEM LESIONS.—
3. CHEMICAL ANALYSIS.—POST-MORTEM IMBIBITION OF POISONS.—
RULES IN PERFORMING A TOXICOLOGICAL ANALYSIS.—4. PHYSIO-
LOGICAL EXPERIMENTS.—5. CIRCUMSTANTIAL EVIDENCE.—MEDICO-
LEGAL CONCLUSIONS.—CLASSIFICATION.

A knowledge of the evidences of poisoning constitutes the chief business of the toxicologist. It is by this means that he reaches a definite conclusion in the cases submitted to his investigation. These evidences comprise: (1) those derived from the Symptoms; (2) those obtained from the Post-mortem appearances; (3) those afforded by Chemical
SYMPTOMS OF POISONING.

analysis; (4) those derived from Experiments on animals; (5) the Moral or Circumstantial evidences.

I. Evidences afforded by Symptoms.—These constitute a very important factor in the diagnosis of poisoning, but alone, they can never be sufficient to establish the charge, for the reason that there are no characteristic symptoms of any poison; if this were possible, there would be no need of ever making a chemical examination, since the symptoms alone would be sufficient to decide the case.

The first of these symptoms to notice is their sudden occurrence in a perfectly healthy person, soon after taking food of drink. Most poisons produce their effects very soon after their administration—some of them almost immediately. But if given in very small quantities, and at intervals, as in slow poisoning, the symptoms may come on gradually, and be readily mistaken for disease. The physician should be extremely cautious about mentioning his suspicions of poison in a case of this character, before he has analyzed the suspected food and drink, and especially the urine of the patient.

The suspicion is strengthened, if several persons, after partaking of the same food, are suddenly seized with the same severe symptoms. But even here it might happen that some disease, like cholera, may have simultaneously attacked several persons, after partaking of a meal. Taylor mentions an instance of this character, occurring in London, where three, out of four members of a family, under suspicious circumstances, were suddenly seized with violent symptoms, which proved to be malignant cholera, which was prevailing at that time.

A third feature connected with the symptoms is their rapid course toward a fatal termination. This, however, is
not of much practical value, since the most active poisons
do not always prove fatal immediately, while, on the other
hand, many diseases run their course very rapidly.

From what has just been said about symptoms, it will be
readily understood that the practical difficulty consists in
distinguishing between these, and the symptoms of disease.
We shall, therefore, briefly consider those diseases whose
symptoms most resemble the signs of poisoning.

The disorders which most simulate irritant poisons are
cholera morbus, malignant cholera, gastro-enteritis, perito-
nitis, ulceration of the stomach, ilius, and hernia. Those
which most resemble narcotic poisoning are apoplexy,
epilepsy, inflammation of the brain, tetanus and cardiac
diseases.

*Cholera morbus* most resembles arsenic poisoning, and it
is frequently mistaken for the latter. Two cases of this
character fell under the author's notice a short time ago,
where death occurred in about eight hours, both of which
were mistaken for cholera morbus by the attending phy-
sician, but both however yielded, by analysis, the most
positive evidence of arsenic poison.

*Malignant cholera* most resembles the action of tartar
emetic in its symptoms, such as the excessive nausea and
vomiting, the rice-water dejections, the cramps, the extreme
weakness, etc. *Gastro-enteritis, peritonitis, ulceration of the
stomach, ilius, and hernia*, all present symptoms which
strongly resemble many of those witnessed from irritant
poisons.

Many of the features of *apoplexy* bear a striking resem-
blance to the symptoms of opium poisoning; whilst *epilepsy*
somewhat resembles poisoning from *prussic acid*, and the
effects of *strychnia* bear a strong likeness to those of
tetanus.
A knowledge of the above facts should put the practitioner upon his guard against too hastily deciding on a case of poisoning from the symptoms alone; and, on the other hand, he should not be misled in attributing to a supposed disease what is really the result of a poison.

II. Evidences obtained from Post-mortem Examination.—
The rules governing an autopsy in a case of poisoning are the same as those which regulate other judicial post-mortem examinations. One important rule should always be observed, namely, that the examination should be thorough and exhaustive, "so as to overlook no lesion whatsoever, and no cause of either accidental or natural death." The rules already given for conducting a post-mortem investigation (Vid. ante, p. 56) need not be repeated here. It should not be forgotten that a careless, superficial autopsy of a body, where the symptoms had strongly resembled those of poisoning, might possibly lead to the conviction of an innocent person.

The importance of receiving the stomach and other viscera into a perfectly clean jar may be inferred from the fact, that the showing that this vessel was not clean, at the trial, would be sufficient to destroy all the chemical testimony. This is well illustrated by a case communicated to the author by the late Prof. R. Bridges, which occurred to himself. The poison suspected was arsenic, but the stomach, etc., were carelessly thrown into an old tin can that had formerly contained zinc-paint, before being sent to the analyst. He discovered zinc in the viscera, for which he was at a loss to account, until the above fact was ascertained.

In the examination of the stomach, it is recommended to open this organ along the lesser curvature, and after care-
fully collecting and measuring the contents, to spread it out upon a clean pane of glass, with the mucous surface outward; it should then be carefully inspected, with the aid of a magnifier, and any abnormal appearance noted, together with any foreign substance, such as crystals of arsenic, fragments of phosphorus, suspicious powders, pieces of vegetable matter, etc. These should afterward be examined with the microscope.

The evidences furnished by the post-mortem, like those derived from the symptoms, can never be absolutely conclusive, but only strongly suggestive—and for a similar reason, viz., because many diseases exhibit precisely the same post-mortem lesions. Sometimes the external inspection of the body may throw some light on the case, as when certain stains of the mineral acids are discovered about the mouth, cheeks, tongue and fauces, and also on the dress of the person. Occasionally, the odor of prussic acid, opium, alcohol, nicotine and phosphorus may be perceived on the corpse. On opening the body, the odor of the above substances, if present, is usually more decided; and in phosphorus-poisoning the white fumes, which are luminous in the dark, as well as the alliaceous odor, are often very perceptible. Again, the remains of certain poisons may be, at times, discovered in the stomach and bowels, such as cantharides, Scheele's green, nux vomica, arsenious acid and orpiment; also vegetable leaves and fibres, which latter may be recognized by their botanical features. The aid of the microscope may also be required.

As regards the true pathological lesions resulting from poisoning, it may be remarked that, as a rule, the irritant poisons leave behind them decided marks of congestion and inflammation of the mucous membrane of the stomach and bowels, together, at times, with ulceration, perforation and
gangrene; while the neurotics leave their impress upon the brain and spinal cord, in the form of congestion, inflammation and effusion in these organs, and sometimes congestion of the lungs. The negative evidence, in the absence of all marks of irritation of the stomach and bowels, against irritant poisoning, although strong, is not positive, because, in exceptional cases, death from these powerful irritants may occur without leaving behind any pathological lesion.

Among the most common of the post-mortem signs produced by irritant poisons is redness; this, however, is a constant symptom attendant on many disorders, and according to Dr. Yellowly, it often occurs simply as a post-mortem change. Ulceration is occasionally the result of irritant poisoning. The author has seen it twice in acute arsenical poisoning. It is however much more frequently the sequence of disease; and as this latter is apt to be insidious, and generally unsuspected until a sudden fatal termination, it might readily be mistaken for a case of poisoning. Softening of the mucous lining of the stomach and bowels may result from both poisoning and disease; it cannot, therefore, be accepted as a proof of the former. Perforation may occur from the action of a corrosive, as the mineral acids and alkalies, and also from disease; but, in the latter case, the aperture is small, while in the former it is large and ragged and its edges are soft and friable; moreover, the poison escapes into the abdomen, and can there readily be detected.

III. Evidences from Chemical Analysis.—The actual discovery of the poison by means of chemical analysis is usually regarded as the most satisfactory and positive evidence of poisoning; and it is a prevalent notion that the case cannot be made out without the production of the poison as the corpus delicti. This is, however, an error. The law requires
the *satisfactory proof of death* by poisoning. The question is—can satisfactory proof be afforded without the chemical detection of the poison? The reply to this inquiry is, that it undoubtedly can, in certain cases. Many convictions have occurred in trials for poisoning, without this particular line of proof. If it were always deemed absolutely essential, doubtless many criminals would escape. We believe this position to be a safe and logical one:—if all the other factors of evidence are perfect—the symptoms, the post-mortem appearances, the effects on living animals, and the moral evidences,—then the chemical analysis is not necessary to substantiate the charge. It is well understood that for some poisons there is no known chemical test, especially for those derived from the vegetable and animal kingdom; besides, circumstances may interfere to prevent a proper chemical examination. If, however, the other branches of evidence fail, and if at the same time, the chemical proofs are unsatisfactory, then the accused must be acquitted.

On the other hand, supposing the analysis reveals the presence of poison in the stomach, this does not necessarily prove that the death resulted from poisoning. Indeed, in the absence of the usual symptoms, the pathological lesions, and the moral proofs, it might plausibly justify the suspicion that the poison had been secretly introduced into the body after death, for sinister purposes.

The detection of the *absorbed* poison, in the organs, as the liver, spleen, kidney, etc., is justly regarded as a more satisfactory proof of poisoning than the mere discovery of it in the stomach. Indeed, it is by some considered as *positive* and incontrovertible evidence. We do not fully assent to this, although admitting the statement as correct in the majority of cases. It should not be forgotten that, if a poison in a liquid state be introduced into the stomach or
rectum of a dead body, by means of a tube, in a short time the liquid will penetrate through the walls of the viscus, by osmosis, and will come in contact with the adjacent organs—the liver, lungs, pancreas, kidney, spleen, etc.—and will penetrate into these organs, so as to contaminate them more or less. Now, if, after several weeks or months' interment, a suspicion be aroused that the death had been caused by poison, and the body then be opened, very decided evidences will be afforded by the organs of what might very naturally be taken for absorbed poison.

Cases of post-mortem imbibition of poisons are extremely rare; indeed, many authors deny their existence; but there is good reason to believe that they have occurred, and that they may be repeated. The author is familiar with the facts of one such case, the particulars of which were communicated to him; and in order to establish the possibility of its occurrence, together with the circumstances most favorable for its production, he had a series of experiments made, under his supervision, by Dr. Geo. McCracken, of the University of Pennsylvania, on the bodies of dogs and cats, with solutions of arsenious acid, corrosive sublimate and tartar emetic, confining his experiments, for the time, to mineral poisons. These solutions were severally injected into the stomachs of the animals, and their bodies were buried for periods, respectively, of three, five, six and seven weeks, when they were disinterred, opened, and the different viscera subjected to chemical analysis, with the following results:—after three weeks' burial, in the case of all the poisonous solutions, the characteristic colored spots of the respective sulphides were seen on the spleen, under surface of the liver, and that portion of the peritoneum posterior to the stomach,—yellow in the case of arsenic; red in the case of antimony; and black in the case of mercury. Each
of the metals was likewise discovered by chemical analysis in the liver, spleen and left kidney; the greatest amount being found in the spleen; next, in the portion of the liver joining the stomach; then in the left kidney; and next in the portion of liver farthest from the stomach; and none in the right kidney. After six and seven weeks' interment the colored sulphide deposits were much more decided, being noticed on the upper, as well as the lower surface of the liver, together with the spleen, intestines, omentum and both kidneys; and, in the case of arsenic, even extending as low down as the fundus of the bladder. By chemical analysis, also, the poisons were detected in all the above-mentioned organs.

The inference from the above facts would naturally lead to the necessity of excluding the idea of the post-mortem introduction of the poison, in every toxicological investigation connected with a trial for murder by poisoning. It is evident that, given a sufficient motive for the deed, it would not be a very difficult matter secretly to introduce a poisonous liquid into the stomach of a dead person, and after the lapse of a few weeks or months to circulate the rumor of the death having been produced by poison. This would probably lead to the disinterment of the body; and the chemical examination would reveal the existence of the poison, not only in the stomach, but also in the liver and other viscera. The conclusion, then, would naturally be that the individual had died from poison, because it had been discovered in the organs; and this alleged discovery might lead to the conviction of an innocent person. Under such circumstances, we deem it of the greatest importance to examine the brain, because a poison injected into the stomach could not possibly find its way, by osmosis, through the bony cranium, into the brain. The finding of the poison, therefore, after
death, in the brain or spinal marrow, may be regarded as one of the strongest evidences of its ante-mortem administration. But we do not maintain that the poison must be found in the brain, in order to substantiate the charge.

Chemical analysis sometimes fails to discover the poison after death, and for this failure several good reasons can be assigned: (1) It may all have disappeared before death, by vomiting and purging, and by elimination through the secretions. Arsenious acid, however, is very apt to adhere to the mucous lining of the stomach, in spite of long and violent vomiting. (2) It may be undiscoverable by chemical analysis, from its very nature; there is no known reagent that will detect the poison of glanders or rabies, and also many of the vegetable poisons. The mineral poisons may usually be easily identified. (3) Loss by absorption and elimination. This is apt to be the case where the dose of the poison was only just sufficient to cause death, and death was not very rapid. (4) The decomposition of the poison in the blood, or during its elimination. This is much more apt to occur with organic, than with inorganic substances. (5) Its possible decomposition in the dead body. This does not occur with the mineral poisons; although the chemical composition of these may undergo change after death, as, e.g., arsenious acid into the yellow sulphide, yet the metal remains indestructible.

In performing a toxicological analysis, certain rules should be observed, which will greatly facilitate the process. First of all, the examiner should, if possible, inform himself of the character of the symptoms, and (if the case was fatal) of the post-mortem, as these will usually indicate to what particular class of poisons he should direct his researches. Secondly, his analysis should be conducted with scrupulous care and accuracy. In searching for the more complex
organic poisons, it is a good plan to reduce the liquid, by evaporation, to a very small bulk, since a minute quantity of a poison diffused through a large amount of water may fail to respond to the proper tests. It is best, also, to operate on one-half of the material, reserving the other portion in case of accident, or for further experiments. The suspected substance ought to respond to all the recognized tests, the characteristic ones being first applied; and, in metallic poisoning, we deem it essential for the analyst to produce the metal, along with the other results. This can always be accomplished without much difficulty, e.g. in the case of arsenic, mercury, antimony, copper, lead, etc. This remark, of course, does not apply to the metals of the alkalies or earths. Too much reliance should not be placed on the mere color of precipitates, as this is often fallacious, from being disguised by admixture with foreign matters, or uncertain, from its resemblance to other substances. As instances, we may cite the impure sulphides of arsenic and antimony, the two liquid tests for arsenic, and the resemblance between the action of the persalt of iron upon opium, and upon the saliva. Finally, the analyst should be careful to test the purity of all his reagents, remembering that many of the so-called chemically pure reagents often contain impurities which may seriously damage his examination.

IV. Evidences from Experiments on Living Animals.—In cases where the poison cannot be identified by the symptoms, post-mortem lesions and chemical tests, the suspected material may be introduced into a living animal (a dog, cat, rabbit, guinea pig, or mouse), and its effects noted. In the case of strychnia, the frog would be appropriate as a corroborative test. Birds are not so well adapted for experi-
CIRCUMSTANTIAL EVIDENCE OF POISONING.

The character of the information thus derived is confined to the mere fact of poisoning, together with some of its physiological and pathological actions. By this means the presence of digitalis was identified in a celebrated French case, and aconite in the case of Dr. Lamson, in England.

The material to be employed in such cases is usually the matters vomited, or that found in the stomach and bowels of the deceased; but the examiner should avoid a too hasty conclusion, inasmuch as disease might cause the secretions of the alimentary canal to become infected, and thus to act upon the animal poisonously, although no poison had really been taken by the deceased; and, on the other hand, although poison may originally have been present in the stomach, it might have all been expelled by vomiting, or undergone decomposition, so that the contents of the stomach would no longer produce a poisonous impression on the animal.

Another fact to be noticed in this connection is, that a poison may be introduced into the human system through the body of an animal, without the latter being affected by it. A case is recorded where a family exhibited all the evidences of belladonna-poisoning after partaking of a rabbit pie; the defence, which was successfully set up, was, that the animal had previously eaten of the belladonna plant, by which its flesh had become poisonous. It is well known that the cow and goat will feed upon the stramonium with impunity, and that their milk will act poisonously upon those who partake of it.

V. Evidences derived from Circumstances.—Although the medical expert is not generally concerned with this sort of testimony, yet, in poison cases, the medical and moral evidence are often so closely connected that the expert may
throw considerable light upon it. These "circumstances" are the following: (1) The suspicious conduct of the accused before the event, such as dabbling in certain poisons not in the line of his calling. This was a very strong point against the Count Bocarmé who poisoned his brother-in-law with nicotine. (2) The purchase and possession of poison by the accused. Of course, this may be satisfactorily accounted for. (3) The proof of administration in the food or drink of the deceased. (4) A sufficiently strong motive for the act. (5) Suspicious conduct of the accused during the illness, and after the death of the deceased—such as preventing his obtaining medical advice; assuming the exclusive care of the person, as to the giving of his food or administering the medicines; carefully removing and disposing of all vomited matters, together with the excreta; and expressing the opinion of the probability of a speedy and fatal termination of the case; and, after the death, opposing an autopsy, hastening the burial, and giving a false account of the illness.

The above series of "evidences" of poisoning constitutes a chain of proof which is perfectly conclusive in any individual case; but we are not always able to exhibit every link of this chain; nor is this always necessary in order to substantiate the allegation of poisoning, since "satisfactory proof" of poison having been the cause of death may be made out in the absence of one, or even two, of the above "evidences."

Certain medico-legal questions will naturally present themselves in every case of poisoning that comes up for trial: (1) Is the death or sickness to be ascribed to poison? This question is fundamental, as it compels the expert to exhibit his proofs of the poisoning. (2) What is the nature of the alleged poison? It is rarely in the power of the toxicologist
to exhibit the identical poison that caused the death, as the corpus delicti. In most cases, all that is possible to do is to demonstrate all the known chemical and (occasionally) physiological tests. In the case of the mineral poisons, it is deemed sufficient to exhibit the metal, and the results of the recognized chemical reactions. In some cases of mineral poisons, however, it is possible to extract the identical substance that was administered, if it were crystalline—such as arsenious acid, corrosive sublimate—and tartar emetic, by the process of dialysis. (3) Was the substance administered capable of causing death? This question is likely to arise only in non-fatal cases. If it can be shown that the substance, although criminally administered, was not poisonous (although supposed to be), conviction would not follow; neither, if the substance were poisonous only in large doses, as oxalic acid, and a very small quantity—only a few grains, had been given. (4) Was the poison taken in sufficient quantity to produce death? The discovery of a large amount of poison in the body is a pretty sure evidence of the cause of death; but the finding of only a minute quantity, or its total absence from the body, is not positive proof that death was not caused by poison (Vid. ante, p. 192). (5) When was the poison taken? This question can generally be answered by referring to the time of the first appearance of the symptoms, together with their duration; but it is affected by various conditions (Vid. ante, p. 187). (6) May the poison have entirely disappeared from the body, without leaving any trace? The answer must be affirmative, if the person has survived long enough to allow of its complete elimination (ante, p. 181). (7) Might the poison found in the body be ascribed to any other source than to poisoning? Not, if it is found in considerable quantities, and in the absorbed state, in the organs. But, if in minute quantity, it might have been
introduced medically, or accidentally. (8) Can poisoning be pretended? Undoubtedly, just as various diseases are feigned for some special motive; but the imposture can be discovered by close watching. The idea of being poisoned is a very common delusion of the insane.

The above medico-legal questions have been chiefly taken from the treatise of Tardieu (Sur l'Empoisonnement).

Classification of Poisons.—Of the numerous classifications of poisons which have been proposed at various times, two only require notice here. One of these is founded on the natural source or kingdom from which the poison is derived, and is expressed by the two classes of Inorganic and Organic poisons; or by those of Mineral, Vegetable and Animal poisons. The other classification, which may be termed the physiological, has reference to the effects of poisons upon the healthy animal system. The classification adopted in the present treatise is founded upon the latter arrangement, as being the most philosophical. It is based upon the one proposed by Dr. Taylor, with some few modifications.

All poisons are divided into two Classes, I. Irritants; II. Neurotics.

I. Irritants.—These include such poisons as produce an irritant action upon the mucous coat of the alimentary canal, the effects being an acrid, burning taste on swallowing, nausea, vomiting, purging, pain in the abdomen, cramps in the stomach; the matters vomited and purged being at times mixed with blood. The post-mortem lesions are more or less inflammation of the gastro-intestinal mucous membrane; sometimes ulceration, perforation and gangrene.

The Irritants may be subdivided into two orders: (1) Simple irritants; and (2) Irritants possessing remote
CLASSIFICATION OF POISONS.

specific properties. They may further be separated into three sections, depending on the source from which they are procured, viz. (a) Mineral, (b) vegetable, (c) animal; and the mineral are finally subdivided into non-metallic and metallic. Some of the irritants are properly named corrosives, on account of their destructive chemical action on the tissues. If diluted, the corrosives act as simple irritants.

II. Neurotics.—These are so named on account of their specific action on the great nervous centres, the brain and spinal marrow. The symptoms are altogether distinct from those of the former class, being directed especially to the brain and spinal cord. These are drowsiness, giddiness, headache, delirium, stupor, coma, and sometimes convulsions and paralysis. They are naturally subdivided into three Orders: (1) Cerebral, (2) Spinal, (3) Cerebro-spinal. The first of these Orders comprise (a) Narcotics, (b) Anaesthetics. The second Order includes those which act directly upon the spinal cord, such as strychnia; they are sometimes termed Tetanics. The third Order comprises those which influence both the brain and spinal marrow, producing delirium, coma, convulsions and paralysis. These latter may be grouped under the three heads of Deliriants, Depressants and Asthenics. The above arrangement is to a great extent an arbitrary one, and must, of course, be somewhat imperfect, as the boundary line between the different classes and orders of poisons cannot always be accurately drawn. The following tabular arrangement exhibits the classification at a glance:

| Class I. | Order 1. Irritants proper. { Mineral. } Non-metallic. |
| Class II. | Order 1. Cerebral. { Narcotics. } |
| NEUROTICS. | " 2. " producing remote specific effects. { Veg'table. } Metallic. |
| " 2. Spinal or Tetanics. |
| " 3. Cerebro-Spinal. { Deliriants. } |
| " " Depressants. |
| " " Asthenics. |
CHAPTER XIV.

CLASS I.—IRRITANT POISONS.

POISONING BY THE MINERAL ACIDS.

CERTAIN COMMON SYMPTOMS.—POST-MORTEM APPEARANCES.—TREATMENT.—CHEMICAL ANALYSIS.—TOXICOLOGICAL EXAMINATION FOR THE DIFFERENT ACIDS.

The mineral acids—Sulphuric, Nitric and Muriatic—possess certain general characters, and produce certain common effects upon the system, which may properly be considered together. This action is entirely local. They are seldom used for homicidal purposes except in the case of young children; they are occasionally employed by suicides, but more frequently are the cause of accidental death.

Their symptoms are exhibited immediately on being swallowed, and consist of a burning in the mouth and gullet, with intense pain in the stomach, attended with constant eructations and vomiting of a brownish or blackish matter, often mixed with blood, together with mucus and shreds of detached mucous membrane. The ejected matters are intensely acid, and if they happen to fall upon a marble slab they produce effervescence; they also change the color and destroy the texture of the cloth, or other material, on which they may fall. Swallowing is very painful, and sometimes impossible. Thirst is intense; the bowels are constipated and the urine diminished. The pulse is small and weak and the skin cold and clammy. Respiration becomes difficult, and the countenance expressive of great anxiety. There may also be cough and difficulty of speaking. Death may
occur from suffocation, when the force of the acid is spent upon the glottis and upper portion of the windpipe. The mouth is excoriated, and the lips are stained and shriveled. When the acid has been poured far back down the throat, in the case of infants, the mouth and lips may entirely escape injury, the corrosive action being confined to the glottis and adjacent parts. The mental faculties usually remain clear, the patient dying convulsed or suffocated.

The result is generally fatal, although the period of death may vary from a few hours, to weeks or months.

_Post-mortem appearances._—Stains of a brownish or yellowish hue are apt to be found on the lips and cheeks; also, on portions of the dress of the deceased. The lining of the mouth and tongue is shriveled and eroded, stained yellowish in the case of nitric acid, and sometimes of a whitish color. At times the mucous membrane of the windpipe appears to have suffered most from the corrosive action of the poison, and cases are reported of sulphuric acid poisoning, where all other parts of the body had entirely escaped. The lining membrane of the oesophagus is usually softened, detached in long shreds, and deeply congested; the stomach contracted, often perforated, sometimes blackened, containing a dark grumous liquid; at other times it presents a yellowish appearance. The intestines are likely to be inflamed, unless the death has been very rapid. If the contents of the stomach have escaped into the cavity of the abdomen, through perforation, the peritoneum will be found intensely inflamed, with more or less of dark, effused blood.

_Treatment._—No remedies are likely to prove efficient when the undiluted acid has been swallowed. The proper treatment consists in administering a solution of the bicarbonate of potassium or sodium; or, in the absence of these, of chalk or magnesia, stirred up in water, together
with copious diluents, such as barley water, flaxseed tea, oil, etc. The stomach pump should not be employed, on account of the risk of perforating the softened oesophagus.

**Sulphuric Acid.**—This acid is commercially named *Oil of Vitriol.* In its concentrated state, it is a heavy, oily liquid, of a light-brownish color; sp. gr. 1.845; intensely sour, and has a very acid reaction.

The *diluted* acid is colorless, very acid, non-corrosive; it chars paper which has been dipped into it and dried by the aid of heat.

Sulphuric acid is more frequently the cause of death than the other mineral acids. Several cases of homicide are reported where it was introduced into the rectum and vagina. The *fatal dose* for an adult is a fluid drachm; for an infant, half this quantity. The danger depends more on the degree of concentration than upon the absolute quantity swallowed. Death usually occurs within twenty-four hours, and in cases where its action is spent upon the rima glottidis, producing suffocation, the fatal result may be almost immediate. According to Casper, the bodies of those poisoned by sulphuric acid resist putrefaction for a long period.

There is good reason for believing that this acid is absorbed into the circulation and eliminated by the secretions. It has been detected in the urine during life.

*Chemical analysis.*—The concentrated acid is recognized by its oily appearance; it chars organic bodies; it evolves considerable heat when mixed with an equal bulk of water; it gives off sulphurous acid fumes when boiled with copper, mercury, wood chips or charcoal. The *diluted acid* is easily detected by its producing a white precipitate, with the nitrate or chloride of barium, insoluble in nitric acid. To confirm this result, the precipitated sulphate of barium should be
dried and mixed with some reducing agent (charcoal, cyanide, or ferrocyanide of potassium), and heated to redness; the sulphate of barium is by this means converted into the *sulphide*; and when this is moistened with diluted hydrochloric acid, the smell of sulphuretted hydrogen is at once perceived, proving the presence of sulphur in the original acid.

It may be objected to this test that several other acids besides sulphuric throw down white precipitates with nitrate of barium, as, *e. g.*., carbonic, phosphoric, oxalic, boric acids, etc. The answer to this is, that either nitric or hydrochloric acids will immediately dissolve all the last-named precipitates, while the *sulphate* remains untouched by them.

Another objection is that any neutral sulphate will produce the same precipitate with nitrate of barium as the free acid. This is true; but the two may easily be distinguished by evaporating a drop of the suspected solution to dryness on a piece of glass; if it consisted of free acid, no residue would remain; but if it contained a sulphate, a saline residue would be left. But the case may present where the solution contains both the free acid and some soluble sulphate; here, finely-powdered carbonate of barium should be added, first warming the liquid; this will precipitate the *free* sulphuric acid only; hence, the resulting sulphate of barium will represent all the free acid present.

Another delicate test is *veratria*. A small portion of this alkaloid is introduced into the diluted acid and carefully evaporated to dryness; a beautiful crimson-purple color is developed. Moreover, as this test produces no effect upon a sulphate, it serves to distinguish the latter from the free acid.

*Toxicological examination.*—The organic matters, if thick and viscid, should be boiled with the addition of distilled
water, and the solution filtered, and a measured portion, acidified by nitric acid, next precipitated by nitrate of barium, and the precipitate washed and dried. In medico-legal cases, this precipitate may be reduced by heat and ferro-cyanide of potassium, and further tested, as mentioned above.

It might happen that the solution contained a soluble sulphate, along with some other acid—citric, acetic, etc. The mode of distinguishing this from a solution containing free sulphuric acid is as follows: a given volume of the solution is acidulated with nitric acid and precipitated with nitrate of barium, and the precipitate washed, dried and weighed. An equal volume of the original solution is evaporated to dryness, in order to dissipate any free sulphuric acid, and is then dissolved in pure water, filtered, and precipitated as before, and the dried deposit weighed. If the weight of each of these precipitates is equal, there was no free sulphuric acid present; but if the weight of the former precipitate exceeds that of the latter one, then the excess of weight will indicate exactly the amount of the free acid present in the original solution.

Detection of stains on clothing.—The color of the stains made by sulphuric acid on dark cloth is red or brownish-red, and they retain their moisture for a long time; on other colored substances they produce a bright red, and sometimes a yellowish stain. To recognize the acid, a few of these spots should be cut out of the garment and boiled with a little distilled water, and tested with the nitrate of barium. A portion of the cloth should also be tested at the same time, in order to show the absence of any sulphate.

Quantitative analysis.—Sulphuric acid is estimated as a sulphate; the precipitated sulphate, after careful washing in hot water, with a little hydrochloric acid, is collected on a filter, dried and weighed; 100 parts of the sulphate are equal to 42.02 parts of monohydrated sulphuric acid.
Nitric Acid (Aqua Fortis), as found in commerce, is a powerful corrosive acid, of a yellow or orange color, the color being due to a mixture with peroxide of nitrogen. Sp. gr. 1.35 to 1.45. It is apt to be contaminated also with sulphuric acid, chlorine and iron. It is seldom used as a poison. Orfila relates a case where a man poured this acid into the ear of his drunken wife, which caused her death by inflammation of the brain and destruction of the bones, seven weeks afterwards. This, however, can scarcely be regarded as an instance of poisoning.

Symptoms.—Similar to those caused by sulphuric acid, except that the lips, tongue and inside of the mouth are stained yellow. Spots upon the cheeks, neck and other parts of the body, and of the dress, are also yellow. The teeth are white, but yellowish at their junction with the gums. The purging is sometimes accompanied with blood. The vapors of this acid may cause death by bronchial congestion; care should therefore be taken to avoid their inhalation in the manufacturing of gun-cotton, etc.

Fatal dose.—Two drachms of the concentrated acid have proved fatal to an adult; though larger doses have been taken with impunity. Life is usually destroyed within twenty-four hours, but frequently it is protracted, and in a case related by Tartra, death did not occur for seven months.

Treatment.—This is essentially the same as that recommended for sulphuric acid.

Post-mortem appearances.—The lips, tongue and inside of the mouth present a yellow, or yellowish-brown appearance; the mucous membrane of the oesophagus is colored yellow, softened, and peels off in pieces; the larynx and glottis may have suffered, as in the case of sulphuric acid. The stomach may be distended, presenting a greenish color, due to the action of the acid on the bile; it may be found in a pulpy
state, perforated, and adherent to the adjacent viscera, and even partially destroyed. The contents have usually a yellow color; the lining membrane is deeply congested, and the vessels filled with dark blood; sometimes the open mouths of the vessels can be seen. The upper portions of the intestines may exhibit the same appearances as the stomach. The large intestine is apt to escape. In chronic poisoning there is great emaciation, and, after death, contraction of the pylorus, with softening of the mucous membrane, has been found.

Chemical analysis.—The concentrated acid is recognized by—(1) giving off colorless, or orange-colored fumes when exposed to the air; (2) by leaving no residue when heated in a watch glass; (3) by giving off dense orange-red fumes, in the cold, when poured on fragments of copper, or on mercury, zinc and tin (dilute acid requires to be boiled on these metals to produce the same result); (4) by its negative action on gold leaf; but if about twice its volume of strong hydrochloric acid be added, a solution of the gold immediately takes place.

The dilute acid is tested as follows: (1) The addition of nitrate of barium and nitrate of silver causes no precipitates, showing the absence of sulphuric and hydrochloric acids. (2) Boil with fragments of copper; the red fumes will indicate the nitric acid action. (3) Neutralize with carbonate of potassium, and moisten a piece of filtering paper in the resulting solution (nitrate of potassium); when the paper is dried, it will scintillate on burning, like touch-paper. (4) Evaporate the above solution until crystallization; examine the crystals with a magnifier (six-sided striated prisms of nitre). If the solution be neutralized with carbonate of sodium, the crystals will present the rhombic form of nitrate of sodium. (5) Put a fragment of these crystals into a small
test-tube, along with a little copper filings and a few drops of sulphuric acid and water; slightly heat, when there will be an escape of orange-red fumes, and the production of a blue liquid (nitrate of copper). (6) Proceed as in (5), but instead of copper filings, add a fragment of morphia, when an orange-colored solution will result, the color becoming fainter on boiling. (7) As in (5), substituting for the copper a crystal of brucia, which will yield a blood-red color, disappearing on the addition of chloride of tin. (8) The iron test. Proceed as in (5), except to add an excess of sulphuric acid and allow it to cool; then pour in gently a freshly-prepared solution of sulphate of iron; a fine purple color is immediately formed at the line of junction, which speedily extends throughout the liquid, if cool; if heated, the color disappears with the evolution of the orange-red fumes. (9) A weak solution of sulphate of indigo; when heated in contact with a crystal of nitrate of potassium and sulphuric acid, the color disappears. (10) As in (5), using a crystal of narcotina instead of copper; a reddish-brown color is produced, changing by gentle heat to a blood-red. (11) The gold test, as in (5), substituting for the copper a piece of gold leaf and some pure hydrochloric acid; the gold will dissolve, and the solution may be tested by protochloride of tin, which imparts to it a purple color. (12) As in (5), substituting for the copper a fragment of pyrogallic acid and a few grains of chloride of sodium; an intense purple hue is imparted to the solution.

Toxicological examination.—First, test the organic matters (as contents of stomach, etc.), with litmus paper; the acid may have been neutralized by the alkaline antidotes. If viscid, add a sufficient quantity of distilled water, and boil gently for about a quarter of an hour; filter; if found to be acid, neutralize with carbonate of potassium and crystallize
by evaporation; drench the crystals with strong alcohol, to remove impurities, and test them by the methods above described. If the matters examined are not acid, they should be boiled with strong liquor potassae, to break them up; then add sulphuric acid; filter, crystallize and test as above.

The urine should always be examined for nitric acid, by distillation with sulphuric acid, and neutralizing the distillate with carbonate of potassium.

The tissues may be examined as follows: Make a mixture of equal parts of sulphuric acid and water, and put twenty or thirty drops into four test-tubes.

To (1) add a little brucia; no change should result. Add now a little piece of the tissue to be examined, when the solution, if nitric acid be present, will turn a blood-red color.

To (2) add a trace of sulphate of indigo; no action should result. Add now a piece of the tissue, when, if nitric acid is present, the color will disappear.

To (3) add a few grains of copper filings and a piece of the tissue, and boil. The evolved reddish vapors, if nitric acid be present, will give a blue color to a piece of white paper moistened with a solution of iodide of potassium and starch.

To (4) add a solution of sulphate of iron; on adding a piece of the tissue, the solution will become of an olive-brown color, if nitric acid is present. (Woodman and Tidy, For. Med. p. 251.)

Suspected stains are examined by soaking the fabric or other substance in warm distilled water. If acid, the solution should be neutralized with carbonate of potassium, and the resulting crystals treated as above directed. The color of these stains, as before mentioned, is yellow; they can be
distinguished from stains of iodine and bile by the application of a weak solution of potassa, which intensifies the nitric stain, whilst it discharges the iodine (or bromine) stain, and does not affect the bile. Free nitric acid in solution may be estimated by saturating it with recently ignited carbonate of sodium; every 53 grains of which will neutralize 54 grains of anhydrous acid $\text{N}_2\text{O}_5$.

**HYDROCHLORIC ACID.**—This acid, known commercially as *Muriatic Acid*, and *Spirit of Salt*, as usually found, has a light yellow color, fumes when exposed to the air, is powerfully acid, and has a sp. gr. of about 1.15. When pure it is colorless; its yellow hue is due to chlorine or chloride of iron. The commercial acid frequently contains nitric acid, arsenic and antimony, as impurities. Instances of poisoning by it are comparatively rare, except as the result of accident.

**Symptoms.**—These are, generally, similar to those occasioned by the other mineral acids. A grayish or white appearance of the tongue and interior of the mouth, with the formation of a false membrane, is usually observed.

**Fatal dose.**—Half an ounce for an adult; a drachm has destroyed a child. The *fatal period* varies from a few hours to many weeks.

The *treatment* is the same as that above described for the other mineral acids.

**Post-mortem appearances.**—These, on the whole, resemble the lesions found in cases of death from the other two acids, although they more closely assimilate the appearances produced by sulphuric acid, in the blackened or charred ridges often noticed in the interior of the stomach. The oesophagus presents a denuded appearance, from the detachment of its
mucous membrane. The glottis and larynx may also be deeply injected and corroded, in cases where the poison has spent its effects on these organs.

Chemical analysis.—The strong acid is distinguished from other acids—(1) by its yellow color; (2) by its giving off dense white fumes in contact with ammonia—best shown by holding a glass rod wetted with the acids over an open bottle of aqua ammoniæ; (3) by its negative action on copper or mercury, even when heated; (4) by its evolving chlorine when heated with peroxide of manganese.

The dilute acid is tested by a solution of nitrate of silver, which is characteristic. A very dilute acid gives with this solution a copious white, curdy precipitate of chloride of silver, which soon darkens on exposure to light, and is insoluble in boiling nitric acid, but very soluble in ammonia. When dried and heated, it fuses into a yellow liquid, which on cooling becomes a soft, horny mass. As any soluble chloride, e.g. common salt, will yield the same precipitate with nitrate of silver, a drop of the original solution should be evaporated on a glass slide; if it was free acid it will all disappear; if a chloride, a residue will be left.

Nitrate of silver also precipitates several other acids and substances, but they all, with the exception of that from hydrocyanic acid, dissolve in nitric acid. The cyanide of silver is also soluble in boiling nitric acid; and is further distinguished by its crystalline appearance, and by its giving off cyanogen gas, when heated in a glass tube.

Toxicological examination.—As this acid is volatile, the distilling process may be applied. The organic matters (such as the contents of the stomach, together with the viscera, properly cut up) should be put into a glass retort with a small quantity of pure water, and distilled nearly to dryness. The first portion of the distillate may be rejected;
but the remainder will usually yield the characteristic test with nitrate of silver.

The above method, however, is liable to two fallacies: First, there is always apt to be present in the gastric juice of the human stomach a variable quantity of normal hydrochloric acid; and secondly, chloride of sodium is frequently found there, being introduced with the food. Consequently, it would not be safe, in a medico-legal case, to rest the proof of poisoning by hydrochloric acid on the chemical analysis exclusively. If the symptoms and the post-mortem lesions did not indicate death from a corrosive acid, the mere detection of this acid after death by the chemical examination would be of no value.

In order to determine whether hydrochloric acid is present in the free state, or merely as a chloride, in any organic mixture, or whether in both conditions, the solid matters should be divided into small fragments and sufficient distilled water added, and the whole gently boiled for half an hour, then cooled and filtered. Equal measures of the liquid are then taken; one is precipitated with nitrate of silver, after adding a little nitric acid, and the precipitate is washed, dried and weighed. Evaporate the second portion of the original liquid to dryness in a water bath, so as to dispel all the free acid; dissolve the residue in pure water and precipitate with nitrate of silver, as before; filter, dry and weigh this precipitate. The weight of the first precipitate of chloride of silver will give all the hydrochloric acid present—both free and in combination; the weight of the second precipitate indicates only the combined acid; the difference in the two weights will indicate the amount of free acid present in the original solution.

The stains of hydrochloric acid on dark cloth are at first of a bright-red color, changing, after some days, to a red-
dish brown. They may be examined after the method already described. The experiment should, however, be verified by testing another portion of the garment not having a stain.

This acid is sometimes used to erase ink marks in cases of forgery. The paper thus acted on should be treated in the same manner as mentioned for the cloth.

Hydrochloric acid is estimated as chloride of silver; every 100 grains of the latter are equivalent to 77.80 parts of acid of sp. gr. 1.15.
CHAPTER XV.

POISONING BY THE ALKALIES AND THEIR SALTS.

SECTION I.—POTASSA.—SODA.—AMMONIA.

SIMILARITY OF EFFECTS.—SYMPTOMS.—FATAL DOSE.—TREATMENT.—
POST-MORTEM APPEARANCES.—CHEMICAL ANALYSIS.

The three alkalies, Potassa, Soda and Ammonia, on account of their similar qualities, may properly be considered together in their toxicological relations. Their effects upon the animal system are very analogous, and these again strongly resemble the impressions produced by the mineral acids—being powerfully caustic in their concentrated state. They attack the tissues with which they come in contact by virtue of their chemical affinities, causing their disorganization and complete destruction. They are very rarely used for homicidal poisoning, but they occasionally prove fatal as the result of accident.

Pure caustic potash and soda are found almost exclusively in laboratories. Commercial potash and soda occur under the names of pot and pearl ashes, and soda ashes, or soap lees. These are all impure carbonates, containing a variable proportion of the respective alkalies. They are much employed in the arts in the manufacture of glass and soap. They are highly caustic in their effects on the system.

Ammonia, or volatile alkali, in its pure state, is a gas; the Aqua Ammoniae of the shops is a concentrated solution of this gas in water, and is a highly caustic substance. The two former alkalies, together with their salts, are fixed when heated; the latter is volatilized by heat, by which circumstance they are readily distinguished from each other.
Symptoms.—In the concentrated state they occasion an acrid, nauseous taste, followed by a burning sensation in the throat and stomach; violent abdominal pain, increased by pressure; vomiting of mucous matters, tinged with blood; purging of a similar character, with tenesmus; difficulty of swallowing, hoarseness and coughing. The pulse is quick and feeble, the countenance anxious; the body is covered with a cold, clammy sweat; the respiration is rapid, with great muscular prostration. Death may ensue in a few hours, or it may be postponed for months, or even years. In the latter cases, the fatal result is owing to secondary causes, such as stricture of the oesophagus and of the pylorus, occasioning starvation.

The effects of swallowing a strong solution of ammonia are similar to those above described, except that they are often more rapid, and are apt to be directed to the organs of respiration by the vapor given off.

The incautious application of the vapor of ammonia to the nose, in cases of syncope, is sometimes followed by fatal results, for this same reason.

Fatal dose.—As in the case of the mineral acids, the fatal effects of the alkalies depend rather upon their degree of concentration than upon the actual amount swallowed. Half an ounce of caustic potash is the usual fatal dose; but an ounce and a half of the solution of the shops, containing about forty grains, has caused death. Strong aqua ammonice has proved fatal in the dose of two drachms; but instances of recovery are more frequent from this than from the fixed alkalies. Recovery has occurred after swallowing over an ounce of liquid ammonia.

Treatment.—The stomach pump should never be used. Dilute vinegar or lemon juice should be freely swallowed, together with oil, or other demulcents. Opium may be given
to quiet pain, and stimulants to counteract the depression. The inhalation of acetic acid might prove beneficial in poisoning by vapor of ammonia.

Post-mortem appearances.—The lining membrane of the mouth, throat, œsophagus and stomach exhibit evidences of corrosion in their softened and abraded condition; at times the mucous coat of the stomach is blackened from the effused blood, and may be completely destroyed. The larynx and bronchi may be inflamed and softened, especially in the case of ammonia poisoning. In chronic cases, the mucous membrane of the œsophagus may be much thickened, and its calibre, as also that of the pylorus of the stomach, extremely contracted.

Chemical properties.—All the alkalies neutralize acids, turn turmeric paper brown, and restore the blue to reddened litmus. They are not precipitated by sulphuretted hydrogen, ammonium sulphide, or ammonium carbonate. Corrosive sublimate causes a yellowish precipitate with the fixed alkalies, and a white one with ammonia.

Potassa is distinguished from soda as follows:

(1) Bichloride of platinum causes with the former, if not too dilute, a yellow double chloride of potassium and platinum, which under the microscope is seen to be in octahedral crystals. But this reagent will also precipitate ammonia; hence, in an analysis, the absence of the latter alkali must be insured. Ammonia can readily be detected by heating a little of the suspected liquid in a test-tube with hydrate of lime; the vapor may be recognized by its odor, and by its action on turmeric paper.

(2) Tartaric acid throws down from a strong solution of potassa, or its salts, a white crystalline precipitate (cream of tartar). This action may be facilitated by adding a little
alcohol, and by stirring with a glass rod. Here, also, the absence of ammonia must first be proved.

(3) Neutralize each alkali with nitric acid, and evaporate the solutions. The resulting crystals are readily distinguishable. The potassic nitrate (nitre) is in six-sided, striated crystals; the sodic nitrate is in cubes.

(4) Heated on a platinum wire, in the blowpipe flame, potassa gives a violet color; soda, a yellow one.

(5) Antimoniate of potassa (freshly prepared) yields with soda and its salts, a white crystalline antimoniate of soda. This reagent gives no precipitate with potassa or ammonia; but is affected by several of the metallic salts.

(6) The spectrum process is an exceedingly delicate test for either soda or potassa, giving with the former a well-defined yellow band, and with the latter two lines, one in the yellow, and one in the blue space.

(7) Polarized light affords a most delicate test for soda. A drop or two of the solution, to which a drop of hydrochloric acid has been added, is put upon a glass slide, and a drop of the solution of bichloride of platinum is then added, avoiding an excess. The mixture is next to be carefully evaporated, until it begins to crystallize; it is then placed under the microscope, furnished with a polarizer. On turning the analyzer until the field becomes perfectly dark, the crystals exhibit a beautiful play of colors; whilst, if no soda is present, no effect is produced. According to Prof. Andrews, \( \frac{1}{100000} \) of a grain may be thus detected (Chem. Gaz. X, p. 378).

Toxicological examination.—The mixture will usually have a soapy feel and frothy appearance. The absence of ammonia should first be established (ante. p. 217). Evaporate to dryness, and heat to redness in a capsule, in order
to char the organic matters. The ash is then to be digested in distilled water and filtered; the alkali will be found in the solution as a carbonate, when it may be tested as above described.

If required to separate the caustic alkali from the carbonate that may be present in the original mixture, the latter must first be evaporated to dryness, and the residue treated with absolute alcohol, which will dissolve out the free alkali, but not the carbonate. After filtration, evaporate to dryness and incinerate; dissolve the residue in water, and test as above directed.

Quantitative determination.—Potash is estimated as a double chloride with platinum (ante, p. 217). The precipitate should be washed with strong alcohol, dried and weighed. Every 100 parts of the double chloride represent 22.5 parts of caustic potassa, or 28.25 parts of anhydrous carbonate.

Chemical analysis of Ammonia and its salts.—Solution of ammonia (aqua, or liquor ammoniæ) is a colorless liquid, having a very pungent odor, and an acrid, alkaline taste. When heated it evolves gaseous ammonia. It leaves no residue when evaporated to dryness. It gives to a solution of a copper salt a characteristic purple color.

The salts of ammonia are colorless, and volatilize when heated; and if, at the same time, they are mixed with lime or potassa, they yield the characteristic ammoniacal odor. The other tests for the salts of ammonia are—(1) bichloride of platinum; (2) tartaric acid; (3) carbazotic (or picric) acid; (4) Nessler's test—this consists of adding an excess of iodide of potassium to corrosive sublimate until the mercuric iodide formed is redissolved, an excess of free potassa being added; it gives a brown discoloration with the smallest trace of ammoniæ.
Toxicological examination.—If the mixture be in a state of decomposition, it will be useless to attempt the analysis, inasmuch as ammonia is a result of the putrefactive process. Unless it be found in very small quantities, the characteristic odor will determine its presence. Distill over about a fourth of the organic liquid, conducting the vapors into a well-cooled receiver, containing a little water, and test the solution as above directed. If no ammonia be given off, then examine the contents of the retort for an ammoniacal salt, by first treating with strong alcohol; filter the solution, and re-distill, along with hydrate of lime or potassa; this will yield the free ammonia.

Ammonia is determined, quantitatively, like potassa: every 100 parts of the double chloride represent 7.62 parts of pure ammonia.

SECTION II.

POISONING BY THE ALKALINE AND EARTHY SALTS.

NITRATE OF POTASSIUM.—BITARTRATE OF POTASSIUM.—SULPHATE OF POTASSIUM.—ALUM.—CHLORINATED POTASSÆ AND SODA.—SALTS OF BARIUM.

Nitrate of Potassium (Nitre; Saltpetre).—This salt is much used in the arts, especially for the manufacture of gunpowder; it is likewise employed in medicine, in small doses. It occurs in six-sided, striated crystals; taste saline and cooling; very soluble in water; deflagrates when thrown upon hot coals; gives off nitrous fumes when acted upon by sulphuric acid.

Symptoms.—In large doses, and in the concentrated state, it acts as a powerful irritant to the alimentary mucous membrane, causing violent burning pain in the gullet and stomach; vomiting, sometimes of blood; bloody stools; cold, clammy skin; weak, frequent pulse; collapse and
death. Besides these evidences of a local irritant action, it occasions certain nervous symptoms, such as convulsions, tremors, partial paralysis, loss of speech and delirium. Occasionally, a very large dose has produced comparatively slight local symptoms, while the impression upon the nervous centres has been more decided.

*Fatal dose.*—Death from this salt is generally the result of accident, it having been swallowed by mistake for other salts. An ounce to an ounce and a half taken in a concentrated state, has frequently proved fatal in a few hours. The largest dose recorded to have been taken is mentioned by Wharton & Stillé (Med. Jurisp. 1884, ii, p. 110). A German, by mistake, swallowed *three and a half ounces* of this salt. He complained of but slight pain or sense of heat in the stomach, and was purged three times within three or four hours. About five hours after taking the nitre, he suddenly fell out of his chair and expired. There was no autopsy. In this case, the excessive dose of the poison seemed to have destroyed life by shock. The rigor mortis was very imperfect, and the countenance and lips retained their life-like appearance to a remarkable degree for three days after death.

*Post-mortem appearances.*—The lining membrane of the stomach is usually highly inflamed, and detached in places; perforation has been observed in one instance. The intestines are often similarly affected. The contents of the stomach are sometimes tinged with blood.

*Treatment.*—Free vomiting should be promoted by the use of bland mucilaginous drinks; opium and stimulants, to relieve pain and depression; together with external applications, as fomentations, etc. There is no chemical antidote.
Toxicological examination.—See Nitric Acid, p. 208.—According to Orfila and Wöhler, nitre has been detected in the urine, liver, spleen and kidneys of those poisoned with it.

Bitartrate of Potassium (Cream of Tartar).—This salt is very much used in medicine, and is not generally considered to be poisonous; but in large and concentrated doses—two ounces—it has proved fatal, causing symptoms strongly resembling those produced by nitre.

Alum (Sulphate of Aluminum and Potassium).—This salt, in large doses, is very irritant to the stomach and bowels, producing vomiting and purging, although in small quantities its action is that of an astringent. It has proved fatal to animals, but no death in the human subject has been recorded.

Sulphate of Potassium, like the foregoing salts, is highly irritating in large doses. It has occasioned death in the quantity of ten drachms and upwards. It is used sometimes as an abortive, and with fatal results. Arsenic has at times been found as an impurity in this salt, derived, doubtless, from an impure sulphuric acid used in its manufacture. Arsenic, in this way, might find its way into Dover's powder, etc. It would be proper to test for arsenic in any sample of this salt which causes irritation.

Large doses of the ordinary purgative salts, under certain circumstances, may prove powerfully irritating, and even fatal. Instances of death following the administration of overdoses of Epsom salt and common salt are reported by Christison and Taylor.
Chlorinated Soda and Potash.—Known in commerce as Bleaching Salts, and much used in France for cleansing clothes, have occasionally produced fatal effects when swallowed. Tardieu has reported such a case (Sur l’Empoisonnement), where a child died some weeks after taking this substance in divided doses. It was identified by the discovery of an abnormal quantity of chloride of sodium in the urine and kidneys; also, by the formation of chloride of lead on the leaden mouth-piece of the bottle which contained the poison, and likewise by characteristic stains of a reddish-white color on the child’s cap.

Salts of Barium.—All the compounds of barium (with the exception of the insoluble sulphate) are poisonous. They occasion symptoms very analogous to those caused by nitrate of potassium, such as pain in the stomach, vomiting and purging, with decided nervous symptoms, and palpitation of the heart. The post-mortem lesions are inflammation of the mucous coat of the stomach and bowels, great congestion of the brain and lungs, and the heart full of dark blood.

The proper treatment is to promote the evacuation of the poison by the use of emetics and mucilaginous drinks, and the free administration of sulphate of sodium, or magnesium, and the subsequent use of antiphlogistic remedies.
CHAPTER XVI.

ORDER II.—IRRITANTS POSSESSING REMOTE SPECIFIC PROPERTIES.

This subdivision of the Irritants includes such poisons embraced under Class I as, besides producing inflammation of the gastro-intestinal mucous membrane, cause other symptoms, which indicate an impression on the great nervous centres. Several of the alkaline and earthy salts already described are of this character, and might very properly have been considered under this head, but they were more conveniently discussed in connection with the Alkalies.

SECTION I.

POISONING BY PHOSPHORUS.

SYMPTOMS.—FATAL DOSE.—TREATMENT.—MORBID APPEARANCES.—DIAGNOSIS.—CHEMICAL ANALYSIS.—AMORPHOUS PHOSPHORUS.

Phosphorus, in combination, is largely diffused as a constituent of the animal body. Its presence is essential to the performance of its normal functions, and this is especially true in reference to the great nerve centres, of which it constitutes a comparatively large proportional part. It is eliminated from the system in the urine, in the form of phosphates.

In its free state, phosphorus is a powerful irritant poison. It is less employed for poisoning in this country than in Europe, and is seldom used for homicidal purposes. Fatal results have frequently occurred by its accidental and suicidal employment—chiefly the former—from the swallow-
ing of phosphorus-paste (used for destroying vermin), and the tops of lucifer matches.

Symptoms.—These do not usually appear for some hours after the poison has been taken. There is first perceived a disagreeable, garlicky taste, and an alliaceous odor may often be noticed from the breath. This is followed by a burning pain in the throat and stomach, with intense thirst and nausea, distention of the abdomen, with vomiting and purging; pupils dilated; cold perspiration, and great anxiety, with small, frequent and irregular pulse. The matters first vomited generally exhale an alliaceous odor, and are luminous in the dark; their color is dark green, or like coffee grounds. The discharges from the bowels have also been observed to be phosphorescent. Sometimes convulsions precede death; again, the patient may die quietly, or in a coma. If the case is protracted for some days, jaundice is apt to occur, and likewise hemorrhage from the stomach, bowels, nose and other parts of the body. Paralysis is an occasional result. The urine is highly albuminous.

Chronic poisoning, accompanied with all the above symptoms, though in a less violent degree, may result from the inhalation of the vapors of phosphorus in the manufacture of lucifer matches. This form of poisoning comes on very insidiously, and is very apt to be fatal. It generally manifests itself first in the jaws, causing caries of the teeth and necrosis of the bone. Some authorities state that this form of necrosis cannot attack those who have perfectly sound teeth, but only those whose teeth are carious (St. Barth. Hosp. Reports, vol. XII). And it is further stated that in some match factories it is required that the workmen should have sound teeth.

Fatal dose.—Less than a grain has proved fatal. A child died after sucking two matches; another older child died from
the effects of swallowing the tops of eight matches. Dr. Taylor mentions the case of a lunatic who died from swallowing one-eighth of a grain (On Poisons, p. 315).

Phosphorus is occasionally employed in medicine, in doses of \( \frac{1}{60} \) to \( \frac{1}{10} \) of a grain; but even in these small doses its effects are uncertain, and it may sometimes act with unexpected severity.

Fatal period.—It is not to be classed among the rapidly fatal poisons. It usually causes death in from one to four or five days; in exceptional cases, earlier. Casper quotes the case of a young lady who died in twelve hours after swallowing three grains of phosphorus in the form of an electuary (Foren. Med., II, p. 100). Dr. Habershon reports a case which proved fatal in half an hour. Cases of chronic poisoning may last for months, or even for years.

Treatment.—There is no chemical antidote known. Free emesis should be encouraged by the use of albuminous and mucilaginous drinks holding hydrate of magnesia in suspension. The use of oil is objectionable, as this is a solvent for phosphorus, and would consequently tend to diffuse the poison. Oil of turpentine is highly recommended by Dr. Percy (Prize Essay, 1872) as a reliable antidote, if given early, and before the poison is absorbed. The old oil, or that which has become oxygenated, is to be employed, not the fresh hydrocarbon. He also recommends the introduction of oxygenated water into the stomach, through a tube, and the inhalation of free oxygen into the lungs.

Animal charcoal has also been recommended, from its power of absorbing free phosphorus, and likewise nitrate of silver, from its power to form an insoluble compound of silver and phosphorus (Brit. and For. Med.-Chir. Rev., 1870).

It has been supposed by some that the poisonous effects of phosphorus are owing to its conversion into phosphorous
acid, at the expense of the oxygen of the blood; but there is good evidence to show that it is absorbed and eliminated uncombined, as the urine has been observed to be luminous. It is more probable that it acts as a blood poison. The blood corpuscles undergo a speedy disintegration, which is doubtless the cause of the ecchymoses seen upon the different organs.

Post-mortem appearances.—According to Tardieu (Sur l' Empoisonnement, p. 437), who has given special attention to this subject, the lesions produced by phosphorus vary according to the form in which it is taken. It is when in the pure state, or simply dissolved in oil, that it most frequently occasions lesions in the oesophagus and alimentary canal. Fragments of phosphorus may be discovered adhering to the mucous membrane, even of the large intestines, and at these spots the bowel is liable to perforation during the examination. In the oesophagus, stomach and intestines, ecchymotic or gangrenous spots are scattered about. The mesenteric glands are engorged, and often soft and friable.

In other cases, as in poisoning by phosphorus-paste, there may be no special morbid appearance, but even in the absence of redness or ulceration, there will be ecchymoses, more or less abundant, over the mesentery and visceral peritoneum. The pleural and pericardial sacs contain bloody serum. Irregular bloody spots are scattered over the pleura, pericardium, and even the endocardium. The heart is soft, distended, or contains fluid blood. The blood itself is very dark, fluid, and syrupy; it appears to be completely devitalized; the corpuscles are disintegrated and transparent, by loss of their coloring matter. In certain cases, the mucous coat of the stomach and duodenum is so softened as to break under pressure of the knife; ulcerations, also, some-
times occur in the stomach. It is stated that the intestines, and even the flesh, of animals poisoned by phosphorus have the odor of garlic, and appear luminous in the dark.

This luminosity of the viscera has been observed in the human subject.

The exterior of the body often exhibits an icterode appearance. Sometimes, the red or blue coloring matter of the lucifer matches that have caused death may be found adhering to the inside of the alimentary canal, a considerable time after death. The general appearance of the gastrointestinal mucous membrane is hemorrhagic rather than inflammatory, ecchymoses being scattered throughout. The contents of the intestines are liquid and bloody. The bladder contains bloody urine, and often presents submucous ecchymoses.

A peculiar pathological alteration, revealed by the microscope as the result of phosphorus-poisoning, is *fatty degeneration of the liver*, and other organs of the body. But these peculiarities are not absolutely characteristic of this poisoning, since they occur in poisoning by other agents, as ammonia, arsenic, alcohol, antimony, etc., and also as the result of disease; nevertheless, they possess especial importance from their association with other notable symptoms of phosphorus-poisoning, such as the jaundice, muscular pains and weakness, diseased condition of the blood, and albuminous urine.

The contents of the stomach in some instances evolve the odor and white fumes of phosphorus; and in a case mentioned by Casper, two days after death luminous vapors issued from the vagina, and a whitish vapor having a phosphorous odor issued from the anus. In this instance, no smell, or vapor of phosphorus could be detected on opening
PHOSPHORUS—CHEMICAL ANALYSIS.

the stomach; nor was there any part of its lining membrane either softened or corroded.

Diagnosis.—Generally, in acute cases, there will be no difficulty in recognizing the evidences of phosphorus-poisoning, both from the symptoms and post-mortem lesions, as above detailed. Chronic cases, accompanied by jaundice, might, however, be mistaken for yellow atrophy of the liver. The following are diagnostic points: The sensation of heat in the throat, eructations and vomiting of matters having a garlicky odor and a luminous appearance, would indicate phosphorus poison. The icterode appearance is not so intense in the poisoning as in the disease, nor is it accompanied with the injection of the eyes, or with the fever, which mark the latter. The fatty change of the viscera may be seen two days after the ingestion of the poison (Tardieu). According to other authorities, the liver in phosphorus-poisoning is enlarged, of a dull appearance, doughy, uniformly yellow, with the acini well marked; in acute atrophy, the liver is diminished in size, greasy on the surface, of a dirty yellow color, and the acini nearly obliterated. In the former, also, the hepatic cells are either filled with oil globules, or entirely replaced by them; in the latter, the cells are filled with a fine granular detritus and thin structure, replaced by a newly-formed connective tissue (Husband).

Chemical analysis.—Phosphorus is a white, waxy solid; sp. gr. 1.83; fuses at 110° F.; at a higher temperature it takes fire, burning with a brilliant white light, becoming converted into the white fumes of anhydrous phosphoric acid. It evolves white fumes of phosphorous acid at ordinary temperatures, when exposed to the air, which also appear luminous in the dark. The smell and taste of phosphorus resemble those of garlic, by which means it may be easily
recognized when mixed with food and drinks. The fuming of phosphorus in the air, as also its luminosity, is completely prevented by the presence of alcohol, ether, chloroform, oil of turpentine and ammonia, even in minute quantities. Although insoluble in water, phosphorus imparts to it poisonous properties, from the production of phosphorous acid. It is tolerably soluble in fixed and volatile oils, by the aid of heat; also in ether, chloroform and naphtha; its best solvent is bisulphide of carbon. Nitric acid converts it into phosphoric acid. It is not affected by either sulphuric or hydrochloric acids. It is best preserved under water, to protect it from oxidation.

In its free state, phosphorus is easily detected by its sensible properties, as already described. A fragment put into the materials for generating hydrogen will evolve phosphoretted hydrogen, easily recognized by its luminosity in the dark, and from being sometimes spontaneously inflammable; this gas, when ignited at a jet, burns with a greenish-blue flame. When it is passed through a solution of nitrate of silver, the latter is blackened by the production of metallic silver, and phosphoric acid is formed in the solution, and may be detected by the appropriate reagents.

Toxicological examination.—If the materials evolve whitish fumes, which are luminous in the dark and have an allaceous odor, there can be no doubt of the presence of phosphorus. If the mixture be ammoniacal, from putrefaction, sulphuric acid must first be added, to neutralize the ammonia, since this would prevent the display of luminosity. Sometimes the particles of phosphorus may be separated mechanically from the inside of the stomach and bowels, which, when found, should be carefully washed and set aside. Or, the mass may be spread out on a metallic plate, and gently heated over a spirit lamp, when the minute frag-
merits of phosphorus will take fire and burn with a brilliant light. The suspected particles may be heated under water, when they will melt and run together into a globule, which will solidify on cooling, and may easily be identified.

_Bisulphide of carbon_ may be used to dissolve out the phosphorus from many organic mixtures, as when phosphorus paste has been employed. On allowing the solution to evaporate spontaneously, the phosphorus will remain in minute globules, which can readily be examined. If, however, the poison is in solution, or in too minute a quantity for the above tests, it must be examined by the following processes:

*Method of Mitscherlich.*—The suspected liquid, acidified by sulphuric acid, is to be distilled in the dark, and the vapors conducted through a long glass tube, kept cold, the end of which passes into a receiver. On gently heating the retort or flask, the vapors, as they pass through the cold tube, condense and display a distinct luminosity. The phosphorus thus distilled collects with the aqueous vapor in the receiver, to which it imparts the usual garlicky odor. A portion of it may likewise collect in the receiver in the form of globules. This test is exceedingly delicate and satisfactory. Dr. Taylor states that the head of one lucifer match produced a luminosity which continued for half an hour, in the condensing tube. We have ourselves verified this, by experimenting with a granule containing the \( \frac{1}{60} \) of a grain of phosphorus, with satisfactory results.

The presence of _solid_ phosphorus in the distillate would render further experiments unnecessary; but in the absence of any granules, the distillate, after filtration, should be acidified by nitric acid, which will convert any phosphorus into phosphoric acid; the liquid should then be concentrated by evaporation, and tested (see _post._) If no luminosity has
been observed, the presence of a small amount of the oxides of phosphorus in the distillate is not sufficient to warrant the supposition of poison, since these might have been derived from the food or tissues, and carried over mechanically. It should be remembered that it is only free phosphorus that gives out the luminosity by the above process; the distillation of the brain, or any other tissue that contains this substance in combination, never produces it.

Method of Lipowitz.—This consists in boiling the suspected liquid, slightly acidulated with sulphuric acid, with fragments of sulphur, in an apparatus similar to that employed in the method of Mitscherlich, the experiment being conducted in the dark. The sulphur abstracts the phosphorus from even complex mixtures, and combines with it. The boiling is continued for about half an hour, after which the pieces of sulphur are withdrawn and washed in water. They will now emit the peculiar odor of phosphorus, and appear luminous in the dark. On gently heating them with nitric acid, a mixture of phosphoric and sulphuric acids will result. By evaporating this solution to near dryness, to get rid of the sulphuric acid, then diluting and filtering, the phosphoric acid may be recognized by the usual tests. In prosecuting this test, unless the amount of phosphorus is very minute, the luminosity of the vapor may also be observed in the cool tube.

The hydrogen method.—This process resembles the method of Marsh employed in arsenic testing. The suspected material, properly prepared, is put into the jar containing the materials for generating hydrogen; the resulting gas is then passed over hydrate of lime, for the purpose of removing any sulphuretted hydrogen; it is then ignited at the end of the delivery tube, producing a green flame. The
phosphoretted hydrogen is luminous in the dark, and affords a black precipitate with nitrate of silver.

Phosphorus has been detected in the free state as late as fourteen days, and three weeks after death; but it is very apt soon to become oxidized in the body, in which case it can only be identified as phosphoric acid.

The method of testing for phosphorus, or phosphoric acid, in a case of suspected poisoning, is very unsatisfactory, since this acid exists in combination with the tissues and secretions, as well as in many articles of food.

The mode of procedure in such a case is to treat the mixture with a little pure nitric acid, and concentrate by evaporation. It is then treated with a slight excess of pure carbonate of sodium and evaporated to dryness, and fused in a porcelain crucible. The resulting phosphate of sodium may then be tested as follows:

(1) Nitrate of silver throws down a yellow tribasic phosphate of silver, soluble in ammonia, and in nitric and acetic acids. Hydrochloric acid converts it into the white chloride. Nitrate of silver also gives a yellow precipitate with arsenious acid, which behaves in the same manner as the phosphate; they are distinguished by drying, and heating in a reduction tube; the arsenic yields a ring of sublimed octahedral crystals (vid. post.)

(2) Ammonio-sulphate of magnesia. — This compound gives with a phosphate a characteristic crystalline precipitate—the ammonia-phosphate of magnesia; the minutest quantity can be identified by the microscope.

(3) Molybdate of ammonia.—This reagent produces a yellow, pulverulent precipitate—the phospho-molybdate of ammonia; it is insoluble in the strong acids, but soluble in alkaline phosphates, alkalies and alkaline carbonates.

Red, Amorphous, or Allotropic Phosphorus.—This singular
variety of phosphorus is procured by exposing ordinary phosphorus to a heat of 450° F., in an atmosphere deprived of oxygen (as in carbonic acid gas) for a number of hours, when it will have changed into a hard, brick-red mass, totally unlike the ordinary substance in its chemical, physical and physiological properties, although retaining its original chemical composition. The difference between the two is shown by reference to the following table, taken from Dr. Percy's essay above referred to:—

<table>
<thead>
<tr>
<th>COMMON PHOSPHORUS</th>
<th>RED PHOSPHORUS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Poisonous.</td>
<td>Innocuous.</td>
</tr>
<tr>
<td>Evolves a strong odor.</td>
<td>Nearly odorless.</td>
</tr>
<tr>
<td>Phosphorescent.</td>
<td>Not phosphorescent.</td>
</tr>
<tr>
<td>Melts at 108° F.</td>
<td>Melts at about 500° F.</td>
</tr>
<tr>
<td>Transparent.</td>
<td>Opaque.</td>
</tr>
<tr>
<td>Almost colorless.</td>
<td>Varies in color from a reddish-black to crimson.</td>
</tr>
<tr>
<td>Freely soluble in various liquids.</td>
<td>Nearly insoluble with liquids.</td>
</tr>
<tr>
<td>Distinctly crystalline.</td>
<td>Amorphous.</td>
</tr>
<tr>
<td>Soft and waxy.</td>
<td>Hard as red brick.</td>
</tr>
<tr>
<td>Flexible.</td>
<td>Brittle as glass.</td>
</tr>
<tr>
<td>Oxidizes in the air.</td>
<td>Unalterable in the air.</td>
</tr>
<tr>
<td>Unites readily with other elements. Is acted on by other elements with difficulty.</td>
<td>Produces no effect.</td>
</tr>
</tbody>
</table>

Nitric acid acts on it with great energy. Produces no effect.

SECTION II.

POISONING BY IODINE, BROMINE AND CHLORINE.

Iodine occurs in shiny, dark iron-gray scales; it has a peculiar odor and disagreeable taste; when heated it gives off beautiful violet-colored fumes, which are irritating to the nostrils and throat. Sparingly soluble in water, very soluble in alcohol and ether, and also in the aqueous
POISONING BY IODINE.

solution of iodide of potassium. It is used medicinally in the form of tincture, compound tincture and ointment.

Symptoms.—Like phosphorus, iodine produces a local irritant effect, and a remote influence; the latter, the result of using it in small quantities. In large doses it occasions burning heat in the throat; severe pain in the abdomen; vomiting and purging, the vomited matters having the peculiar odor of iodine, sometimes mixed with blood; the color of the matters is yellowish, except when they consist of farinaceous articles, in which case it is blue. The bowels may also exhibit the presence of iodine. Other symptoms are giddiness, headache, thirst, anxiety, convulsions and fainting.

In chronic poisoning (iodism) the symptoms are nausea, vomiting, purging, pain of stomach, tremors, palpitation, salivation, cramps, general emaciation, and a tendency to absorption of certain glands, especially the testes of males, and the mammary of females.

There is a remarkable diversity in the effects of iodine upon the human system, some persons bearing very large doses with little or no suffering, while others are seriously affected by the smallest quantities. Overdoses have occasionally proved fatal, leaving morbid appearances very similar to those produced by the irritant alkaline salts.

Chemical analysis.—In its free state, iodine may readily be distinguished by its solid form, color, odor, volatility, and its action on boiled starch—quickly turning it blue. In combination, as iodide of potassium, the iodine must first be set free (by chlorine, or nitric acid) and then the starch-test applied.

From organic mixtures the iodine may be separated by bisulphide of carbon, which dissolves it, forming a pink solution; remove the watery liquid, and evaporate the bisulphide solution to dryness, when the iodine will be left.
If this process fails, on account of the conversion of the iodine into hydriodic acid, or into an iodide, it will be advisable to transmit sulphuretted hydrogen through the mass, properly diluted; this will convert any free iodine to hydriodic acid; drive off the excess of gas by heat; add potash in excess; filter and evaporate to dryness. Char the residue at a low red heat, to get rid of the organic matter; pulverize and dissolve in water. Concentrate the solution, and add strong nitric acid and boiled starch, which will develop the iodine, if any be present.

**Iodide of Potassium.**—Although much used in medicine, in doses up to sixty grains it occasionally produces violent effects upon the system, such as headache, griping abdominal pains, thirst, inflammation of the nostrils and eyes, and frequent pulse, together with salivation, and a pustular eruption. As found in the shops, the salt is apt to be considerably adulterated with the carbonate.

**Bromine.**—This is a dark red, volatile liquid, excessively pungent to the eyes and respiratory organs, having an acrid odor and taste. It is highly corrosive, destroying animal tissues very rapidly. It has occasionally proved poisonous fatally. A case is reported by Dr. Sayre, of New York, of a daguerreotyper, who swallowed an ounce of bromine for the purpose of self-destruction. The immediate symptoms were spasmodic action of the muscles of the larynx and pharynx, with great difficulty of breathing, followed by intense burning pain in the stomach, with great anxiety, restlessness and trembling of the hands. The pulse was rapid, tense and cored, and respiration greatly hurried. Collapse soon followed, and death took place in seven and a half hours after swallowing the poison.

The post-mortem examination revealed vivid injection of
the external coat of the stomach and of the abdominal
viscera generally, which were stained of a deep yellow
color. Portions of the stomach were softened. Its con-
tents resembled port-wine dregs, and exhaled the odor of
bromine. Its whole interior was covered with a thick black
layer resembling coarse tanned leather. The mucous mem-
brane was very thin and very deeply injected (Whar. and

Chemical analysis.—Bromine may be separated from
organic matters by means of bisulphide of carbon, or by
ether, after the method described for iodine (p. 235). The
bromides may be decomposed by chlorine, or by a strong
acid. Bromine is characterized by imparting a deep yellow
color to boiled starch.

Chlorine is a powerfully irritating gas, of a greenish-
yellow color. If inhaled into the lungs it may destroy life,
like gaseous ammonia. Chlorine is readily recognized by
its smell and color, and especially by its powerful bleaching
properties.
CHAPTER XVII.

POISONING BY ARSENIC.

METALLIC ARSENIC. — ARSENIous ACID. — PROPERTIES. — SYMPTOMS. — CHRONIC POISONING. — FATAL DOSE. — TREATMENT. — POST-MORTEM SIGNS. — CHEMICAL ANALYSIS. — TOXICOLOGICAL EXAMINATION. — OTHER PREPARATIONS OF ARSENIC.

The term arsenic, as employed in toxicology, always signifies (unless specially qualified) arsenuous acid, or white oxide of arsenic. The metal is brittle, of a steel-gray color, very volatile when heated, its vapor having a strong, garlicky odor, by which it is easily recognized. It is very rarely used as a poison; it is sold, however, under the name of fly powder, which is a mixture of the metal and arsenuous acid.

Arsenic is one of the most important of all the poisons. The facility of procuring it, and its ease of administration contribute greatly to its extensive use, both as a homicidal and suicidal agent. Arsenic exists in nature in the form of the metal, and in combination with other metals, particularly iron, copper, zinc, nickel and cobalt; also with sulphur, as native orpiment and realgar.

In the arts, arsenic enters into numerous compounds, as in the manufacture of enamel and glass, composition candles, vermin killers, etc. It is used by shipbuilders to protect timber from worms; by farmers to preserve their grain for seed and for washing sheep; by grooms to improve the coats of their horses; and, if we may credit the accounts of travelers, by the inhabitants of Styria and other mountainous countries, to increase their physical powers of endurance and to improve the complexion of the females.
Properties of Arsenious Acid, $\text{AsO}_3$.—It occurs in commerce either as a heavy, white powder, or in masses, which are at first translucent, but afterwards become opaque. It is nearly tasteless, or at most has a faint sourish taste, not \textit{acrid}. It is only slightly soluble in cold water; boiling water dissolves about the eighteenth to the fortieth part of its weight, or from six to twelve grains to the ounce. Cold water takes up only about \textit{half a grain} to the ounce. Its solubility is much increased by the addition of an alkali, but diminished by the presence of organic matter. It is easily held in suspension by soups, coffee, tea, milk, etc.

When arsenious acid is heated at a temperature near 400° F., it sublimes in the form of a white vapor, which is inodorous, and is deposited on a cool surface, either as an amorphous powder, or in octahedral crystals. If thrown upon red-hot charcoal it is decomposed, and the vapor will have an alliaceous odor, because it results from the reduced metal.

\textit{Symptoms}.—The rapidity and virulence of the symptoms depend somewhat on the form of the poison (\textit{i. e.}, whether in solution or otherwise), and also on the fullness or emptiness of the stomach. As a rule, the symptoms do not occur for half an hour, or an hour. There is first a sense of faintness, attended with a feeling of heat and constriction of the throat, together with thirst, nausea and burning pain in the stomach, increased by pressure. Vomiting and retching soon follow; the matters ejected are sometimes streaked with blood; they may be variously colored. Purging, accompanied with tenesmus, comes on, and along with the vomiting, may be incessant, though affording no relief to the sufferer. Cramps in the legs are apt to be present, along with great depression, cold sweat, intense thirst, and a feeble, frequent pulse. The whole train of symptoms
strongly resembles a severe case of cholera morbus, for which it has frequently been mistaken. As a rule, the symptoms are continuous, although there are, occasionally, remissions, and even intermissions. Coma, paralysis and convulsions may supervene before death. The urine is often partially suppressed. If the patient recovers from the immediate attack, he may suffer for a long time after from indigestion, partial paralysis, or from epilepsy.

Certain anomalies may occur. The pain may be absent or slight. Vomiting and purging and thirst may not be present. Some cases especially resemble cholera morbus, from the intensity of the gastro-enteric irritability; whilst others indicate severe nervous disturbance, by the intense headache, giddiness, restlessness, violent cramps, delirium, convulsions and coma. Again, there may be immediate collapse, little or no pain, vomiting or purging, but a cold, clammy skin, extreme prostration, very frequent and feeble pulse, slight coma, with perhaps convulsions and death within a few hours after swallowing the poison. In yet other instances, the symptoms resemble those of narcotics, the person falling into a profound sleep, deepening into coma, and dying in a few hours without rallying. In the latter cases, the autopsy has revealed no trace of inflammation of the stomach.

All the above varieties of symptoms occur quite independently of the size of the dose, or mode of administration, and they cannot be satisfactorily explained except by referring them to constitutional peculiarities.

The symptoms of chronic poisoning usually result from small doses of arsenic frequently repeated, or from exposure to the vapors of arsenical products, as in the case of workmen, or from the accidental inhalation of arsenical dust from wall papers. The eyes become inflamed and watery,
there is great gastric distress, with frequent sickness and vomiting, diarrhœa, headache and giddiness, a jaundiced skin, an eczematous eruption, local paralysis, general emaciation, falling out of the hair, salivation and excoriation of the tongue, with hemorrhage; and death may occur from exhaustion. The symptoms in such a case are frequently very obscure and misleading, and perhaps chance alone may reveal the real source of the disorder.

The time when the symptoms appear varies considerably. As before stated, these do not generally manifest themselves for a half hour or an hour after swallowing the poison, but there are numerous exceptions. Cases are recorded where they appeared in the act of swallowing; others, in which they were exhibited in eight, ten and fifteen minutes after. On the other hand, numerous instances are recorded where the time was protracted for many hours. The longest interval is mentioned in the U. S. Dispensatory (1865), where the symptoms were delayed for sixteen hours, after a dose of a drachm of the poison. This discrepancy may, in part, be accounted for by the state of the stomach, it being empty or full at the time of administration; also by the form of the dose, whether solid or liquid; also by the time of day, whether before going to sleep, or otherwise. The simultaneous use of opium or alcohol would, no doubt, exercise a modifying influence over this poison. (See ante, p. 186.)

The external application of arsenic, either to the sound skin, or still more to abraded or ulcerated surfaces, is often followed by fatal results. Proofs of this are exhibited in the effects of the applications of cancer-curers to ulcerated breasts; also in the use of arsenical solutions to the sore heads of children. In some of these instances, absorbed arsenic has been detected, after death, in the viscera of the
body. Arsenic has also proved fatal when injected into the rectum and vagina.

In all the above cases of the external application of this poison, its usual constitutional effects were produced, such as burning and constriction of the throat, thirst, vomiting and purging, great depression, and the various nervous disturbances above described.

Fatal dose.—Two grains may be considered the minimum fatal dose for an adult, but smaller quantities have produced alarming symptoms. On the other hand, recoveries have often occurred after very large doses—one to two ounces—have been swallowed.

Fatal period.—The great majority of deaths occur within twenty-four hours, and of these the most within eight or ten hours. The shortest period is recorded by Dr. Taylor, of a youth, aged seventeen years, who died in twenty minutes from the effects of a large dose accidentally swallowed; the symptoms were of a tetanic character. Other cases are reported where death occurred in two or three hours. On the other hand, life may be prolonged for weeks, months or even years, the patient suffering greatly during the whole interval.

Arsenic is not a cumulative poison; it is temporarily deposited in the liver and other organs of the body after absorption, but it is rapidly eliminated from the system by the urine, bile and other secretions. Should the person survive for two or three weeks, no trace of the poison may be found after his death, in consequence of its total elimination during the interim. The exact period at which arsenic is completely eliminated from the human system is not fixed, but the analyst need hardly expect to discover it after sixteen days, although it has been detected in the urine of a man twenty-one days after swallowing a large dose, and who subsequently recovered.
The rapidity with which it is absorbed and deposited in the tissues is very great. Dr. Taylor (On Poisons, p. 46) found it in the liver four hours after it had been swallowed. He believes that the liver acquires its maximum of absorbed arsenic (about two grains) in fifteen hours, after which the quantity gradually diminishes.

The question of the elimination of arsenic from the human system may have an important medico-legal bearing, as where a person who has been taking small doses of arsenic, medicinally, for a length of time, should suddenly die from gastro-enteritis, and a post-mortem examination should reveal the presence of absorbed arsenic in the organs. Here both the symptoms and the chemical analysis would strongly confirm the suspicion of arsenical poisoning, unless the fact of the medicinal administration of the drug could be satisfactorily established.

Arsenic is known to be deposited in all the tissues of the body, including the bones; but not in the hair.

Post-mortem appearances.—The most decided evidences of the irritant character of the poison are exhibited by the stomach, the mucous lining of which is usually highly inflamed, sometimes presenting a uniform, deep red color, at others, showing patches of diffused dark redness. Arsenic seems to have a specific effect on the stomach, no matter by what avenue it is introduced into the system. Occasionally, the lining membrane is thickened and corrugated; again, it is softened, and readily separated. When the poison has been taken in substance, it is not unusual to find patches several inches in extent, consisting of tough, yellowish-white bases of arsenious acid, mixed with lymph and mucus, firmly adherent to the membrane, and forming so many foci of intense inflammation. White spots of arsenic are often found between the rugæ, and when a long interval has
occurred before the examination, yellow stains may be found, as the result of a decomposition into the yellow sulphide.

Ulceration of the stomach is rare; but we have witnessed it in two instances, one quite recently, where death occurred in eight hours after swallowing the poison; in the other case the examination was not made until four months after death; here the ulcer was a quarter of an inch in diameter, and was surrounded by a deep zone of dark, effused blood, and had penetrated down to the peritoneal coat of the stomach. Perforation is still more rare.

The upper portion of the small intestine is very apt to be involved in the inflammation; also the cæcum and rectum. Other organs, as the lungs, brain and bladder, are occasionally found congested; but these offer no diagnostic points. The most remarkable fact connected with the post-mortem appearances is the occasional absence of all signs of inflammation, even in cases where there had been violent inflammatory symptoms before death.

An important circumstance is the antiseptic power of arsenic, which accounts for the remarkable preservation of the body for many months after death, whereby the detection of the poison is possible for a long period after burial—in one case, fourteen years after death. Of course, after such an interval, most of the body would be decomposed, but still enough remains for the purpose of identification. In such cases there is generally noticed an absence of the usual cadaveric odor, and also the presence of numerous yellow patches over the abdominal viscera, due to the production of the yellow sulphide, by the action of sulphuretted hydrogen on the arsenious acid.

It should, however, be stated that arsenic does not uniformly exert this preservative power on a dead body; in fact,
in some cases it would seem that putrefaction has advanced with increased rapidity. These instances, however, are exceptional; but they should put the expert on his guard against too positive an assertion as to the presence of arsenic when a body exhibits an unusual degree of preservation, since this may be due to other causes (vid. ante p. 53).

**Treatment.**—If vomiting is not active, a quick emetic (sulphate of zinc and ipecac) should be administered, or a draught of mustard water; the stomach pump may be employed, if on hand; warm diluent drinks are useful; after this, the free use of hydrated sesquioxide of iron. This can be prepared extemporaneously by diluting the tincture of the chloride and adding ammonia in excess, and washing the precipitated iron. This antidote must be taken in large doses, frequently repeated. Afterwards, a dose of castor oil should be given.

There are numerous attestations to the value of this antidote; it acts by converting the poison into the insoluble arsenate of iron. The freshly precipitated hydrate of magnesia is also recommended as an antidote.

**Chemical Analysis.**—I. *In the solid state.* (1) a small quantity of the white powder placed on platinum foil and heated is entirely dissipated in white inodorous fumes; (2) slowly heated in a narrow glass tube, it sublimes, forming a white ring of octahedral crystals on the cool portion of the tube, visible by a good magnifier. Calomel and corrosive sublimate will each form white rings under similar conditions; the arsenical deposit is distinguished from these (a) by the octahedral crystals; (b) by the action of liquor potassae, which dissolves it without color, while it gives a black color to calomel and a yellowish-red to corrosive sublimate; (3) moistened with sulphide of ammonium, and evaporating, produces the yellow sulphide; (4) put into a reduction tube along with some reducing agent, as charcoal,
black flux, or dried ferrocyanide of potassium, and heated by the flame of a spirit lamp, it is reduced, and the metal is volatilized in the form of a brilliant steel-gray ring or mirror.

In order to effect the sublimation successfully, certain precautions are necessary. The reduction tube should be small—about the eighth of an inch in diameter, and three inches long; it should be perfectly clean and free from moisture. The reducing agent should be perfectly dry, and thoroughly mixed with the arsenious acid, in the proportion of three or four to one of the latter. After it is introduced into the tube, this should be wiped out with a wad of cotton or a roll of filtering paper. The tube should first be gently warmed just above the contents, and then fully heated in the flame. This precaution insures a better formed metallic ring.

For the sublimation simply, the above process is to be pursued, with the omission of the reducing agent.

The obtaining the metallic ring or mirror, by the reducing process, may be regarded as positive proof of the presence of arsenic; but in a medico-legal case this should be confirmed by further proofs: (1) the arsenic mirror is wholly soluble in hypochlorite of sodium; (2) it is soluble in hot nitric acid, and the solution, on evaporation, leaves a brick-red deposit when touched with nitrate of silver solution, due to the formation of arsenate of silver; (3) if the closed end of the tube be broken off, and heat applied to the sublimate, it will readily volatilize, and, combining with the oxygen of the air, will condense on the upper portion of the tube, in a white ring of arsenious acid; (4) this latter may be dissolved in a few drops of warm water, and subjected to the liquid tests.

II. The liquid tests.—These are the ammonio-sulphate of copper and the ammonio-nitrate of silver. They should be
prepared only when required for use. The former is made by the cautious addition of aqua ammoniae to a somewhat dilute solution of sulphate of copper, until the precipitated oxide is barely redissolved. When this reagent is added to a solution of arsenious acid, it throws down a light green arsenite of copper (Scheele's green). This precipitate is soluble in ammonia, and in free acids. If the arsenic is in very minute quantities, the characteristic color does not appear immediately.

The silver test is prepared by adding aqua ammoniae to a strong solution of nitrate of silver until the precipitated oxide is barely redissolved. When this is added to the arsenical solution, a canary-yellow precipitate occurs (King's yellow), arsenite of silver, which, like the former, is freely soluble in ammonia and the acids.

These liquid tests are available only in perfectly pure solutions of arsenious acid; they are inadmissible in the presence of organic matter, e. g., the contents of a stomach, since various organic substances will produce similar colors with both copper and silver. They may, however, be satisfactorily confirmed (1) by heating either of the dried precipitates (arsenite of copper or silver), either alone, or with a reducing agent, in a reduction-tube; the former experiment will yield a sublimate of octahedral crystals, the latter the metallic mirror. (2) If the blue ammoniacal solution of the arsenic is poured over a crystal of nitrate of silver, a film of yellow arsenite of silver is immediately formed around it.

III. The sulphuretted hydrogen test.—This consists in passing washed sulphuretted hydrogen gas through the solution of arsenious acid, slightly acidified by hydrochloric acid; a clear, yellow precipitate falls—tarsulphide of arsenic (orpiment), which is soluble in the alkalies, and insoluble in
acids. In very dilute solutions, the precipitate does not separate until the excess of the gas is driven off by heat.

Fallacies.—Cadmium, tin and selenium yield somewhat similar precipitates with sulphuretted hydrogen. Practically, the only one that need be considered is cadmium, which, however, is easily distinguished from arsenic, as follows: (1) the arsenic sulphide is soluble in ammonia and insoluble in the acids; with cadmium sulphide it is precisely the reverse; (2) when dried and sublimed with a reducing agent, the arsenic sulphide yields a metallic ring; the cadmium, a brown oxide.

In a poison case, the sulphide of arsenic should always be proven by (1) obtaining the metallic ring by subliming it with a reducing agent; (2) by boiling the sulphide in hydrochloric acid, along with a piece of bright copper foil; a steel-gray deposit shows the presence of arsenic.

IV. Marsh's Test.—The principle here involved is, that when arsenic comes in contact with nascent hydrogen, it combines with it to form arseniureted hydrogen, a gas which possesses peculiar properties, by means of which the arsenic may be recognized with great certainty. Practically, the simplest and best mode of performing the experiment is to add to the materials for generating hydrogen (zinc, water and sulphuric acid), in a wide-mouth flask, the suspected arsenical solution. The cork fitted to the mouth of the flask should have two perforations, through one of which a perpendicular glass tube passes down below the surface of the liquid contents; through the other aperture a tube bent at right angles is inserted, out of which the generated gas issues. A drying tube (containing fragments of fused chloride of calcium, or of pumice stone moistened with sulphuric acid) is fastened by one end to the exit tube, and by the other extremity to a horizontal tube of hard German
glass, about a foot long, which may be turned up at the farthest end, and made to terminate in a small point, for burning the gas in a jet, as it escapes.

In performing this experiment certain precautions are necessary. In the first place, the absolute purity of the zinc and sulphuric acid must be secured, since both of them are liable to be contaminated with arsenic. Secondly, caution should be exercised to have the atmospheric air completely expelled from the apparatus before lighting the jet, otherwise the mixture of hydrogen and air will produce a violent explosion. The evolution of the hydrogen should be rather slow and gradual. After waiting the proper time, the jet may be lighted; it will burn, if pure, with a scarcely perceptible flame. The purity of the materials may now be tested by applying the flame of a large spirit lamp, or a Bunsen burner, to the horizontal glass tube until it is red hot; if no stain or deposit occurs just beyond the heated spot, the absence of arsenic is certain; or, if no deposit forms on a piece of white porcelain held over the burning jet, the same conclusion may be held.

A small quantity of the suspected solution is now to be introduced through the upright tube; its decomposition immediately commences, freeing the arseniureted hydrogen, which yields the following characteristic results:

(1) The ignited jet.—As soon as the arsenic combines with the hydrogen, an immediate change occurs in the appearance of the flame, which increases in size and acquires a faint, bluish color, and unless the arsenic be in minute quantity, it evolves white fumes, and gives out an allaceous odor. If these fumes are received into a short, wide glass tube, they will condense into a white powder, sometimes crystalline, and may be identified as arsénious acid.

If the jet be made to impinge on a piece of glass or
white porcelain, held horizontally, and just within the flame, a deposit of pure metallic arsenic, of a brilliant steel-gray or brownish-gray color occurs, which may be multiplied to any extent by changing the position of the porcelain. In order to procure the finest deposits, the flame should be steady and not too large. Although these spots may vary somewhat in color, they are always brilliant and never sooty.

These deposits may be identified (1) by their immediate solubility in *hypochlorite of sodium*: stains of antimony, which they most resemble, are not thus affected. (2) When touched with a drop of *sulphide of ammonium*, they do not immediately disappear; antimony stains are instantly dissolved. (3) Both metals dissolve in hot nitric acid, and on evaporation yield white residues; if now touched with a drop of strong solution of nitrate of silver, the arsenic spot assumes a brick-red color, while the antimonial stain remains unaffected.

(2) Decomposition of the gas by heat.—On placing the flame of a large spirit lamp, or a Bunsen burner, immediately below the horizontal tube (which should previously be contracted after heating it in several places), when it becomes nearly red hot, a deposit of metallic arsenic begins to form just in advance of the flame, which should be held a little behind one of the contracted spaces; the deposit continues to increase until it may completely occupy the whole of the narrow space, and even advance beyond it. This constitutes the *arsenical mirror*. It may have the steel-gray, brilliant appearance already described, or even a coppery hue, and it is highly characteristic of the presence of arsenic. Several such mirrors may thus be obtained, by moving the flame to different parts of the horizontal tube, provided there is a sufficient amount of the poison to operate upon. The tube
may afterwards be filed, so as to separate the mirrors, which may be retained for exhibition in court, as positive proof of the detection of the poison.

This mode of experimenting yields even more delicate results than the jet; but unless the quantity of arsenic is extremely small, it will always be possible to obtain both results by Marsh’s process.

One fallacy only might interfere with this experiment—the presence of antimony, which in contact with hydrogen yields a gas very similar to arseniureted hydrogen, and like the latter, is decomposed by heat, yielding a metallic deposit. They may be distinguished as follows: the antimony mirror is deposited just over the heated spot, and not in advance of it; it has usually a darker appearance than the arsenical mirror; the latter is more easily volatilized than the former, and condenses higher up in the tube, in octahedral crystals. The two deposits may also be tested by the different reagents mentioned above (vid. p. 246); also, by dry sulphuretted hydrogen, which produces with the arsenical gas a yellow deposit, and with the antimonial gas an orange-red.

(3) Decomposition by nitrate of silver.—If the arseniureted hydrogen gas be passed through a solution of nitrate of silver, it immediately blackens it, from the precipitation of metallic silver, arsenious acid remaining in the solution. The filtered clean solution will contain, also, free nitric acid, and any excess of nitrate of silver. On neutralizing with ammonia, a yellow precipitate will fall—arsenite of silver (vid. p. 247). The analyst should not rely on the mere production of the black color, since other gases beside arseniureted hydrogen might cause this, but he should continue the experiment as above described.

V. Reinsch’s Test.—This consists in producing a deposit of metallic arsenic on bright copper foil. The suspected
solution, acidulated with about one-sixth of its bulk of pure hydrochloric acid, is first brought to the boiling point, and a piece of bright copper foil is introduced, and the boiling continued. The presence of even a very minute quantity of arsenic is soon indicated by the tarnishing of the copper, which ultimately assumes a dark steel-gray, or even black, color. If the quantity of arsenic be large, the deposit is immediate, and very dark; it may even break off in scales; if the amount of the poison is very small, the stain upon the copper will be fainter, and merely of a violet, or bluish tint. Moreover, the deposit on the copper is affected by the degree of dilution; hence, if the quantity of the water be large, it may require boiling for half an hour before a visible deposit occurs.

This reaction is very delicate and extremely satisfactory. One great advantage that it possesses over the other tests is, that it may be practiced in complex organic fluids; hence, this test is usually employed in toxicological research, as the trial test.

Certain precautions are, however, required in employing it. First, the purity of the hydrochloric acid must be insured; this is easily accomplished by first boiling some of the acid diluted with water, and then introducing a slip of the copper. If no stain appears upon the latter after fifteen minutes, we may be certain of the absence of arsenic or antimony from the acid. Secondly, the copper must be both bright and pure. Its brightness is effected by rubbing it with emory paper, and it may be regarded as pure if, when boiled in the acid arsenical liquid, it is not dissolved, and does not impart a green color to the liquid. But, if deemed necessary to further test its purity, the process of Mr. Abel may be adopted: Add to pure hydrochloric acid, diluted with six parts of water, one or two drops of a weak solution
of perchloride of iron; boil the acid liquid, and introduce into it the copper, well polished; if it contains arsenic it soon becomes tarnished; if pure it remains bright. (Taylor, Med. Jurisp., 1883, p. 268.)

In applying this test, it is best to use small pieces of copper successively, removing each fragment as it becomes coated. By this means the whole of the arsenic may be removed from the solution. We have ascertained, by actual experiment, that one grain of arsenious acid dissolved in the acid solution, and treated by Reinsch's process, will impart a distinct, dark, steel-gray coating to three hundred square inches of copper surface. This method will, therefore, serve for an approximative quantitative estimate of the poison.

Another caution to be observed is not to remove the copper too soon from the liquid, in case no deposit occurs; in doubtful cases, the boiling should be continued for half an hour. But, on the other hand, if the copper be kept in for an hour or longer, it may acquire a dark film, independently of any arsenic.

Fallacies.—Other metals beside arsenic will impart a dark coating to copper by Reinsch's process, such as antimony, mercury, silver, bismuth, tin, gold, platinum and palladium, and likewise organic matter, especially if it contain sulphur. Hence, in the application of this test, the mere production of a dark deposit on the surface of the copper is not sufficient to establish the presence of arsenic, but further corroborative proof is required. This is afforded by washing a fragment or two of the coated copper, and then thoroughly drying them between the folds of filtering paper, (avoiding touching with soiled fingers), and rolling them up into small coils, and then introducing one or more of them into a small, clean reduction-tube and applying the heat of
a spirit lamp. The arsenic will volatilize and condense in the cool part of the tube, in a white ring of octahedral crystals. The only other metals which could volatilize under such circumstances are antimony and mercury; but the sublimate from antimony is either amorphous, or else in fine, acicular crystals, while the mercurial deposit consists of fine, spherical globules of the metal, easily recognized by a magnifier.

The attention of the toxicologist should especially be directed to the fact, that if copper be boiled for some time in an acid solution of complex organic matters, it will become coated with a decided dark stain, and will, moreover, yield, when heated in a reduction-tube, an amorphous sublimate, which may even sometimes show acicular crystals, consisting apparently of a compound of copper. We have repeatedly verified this by our own observations. Hence, it follows that, for the complete corroboration of Reinsch's test for arsenic, in a medico-legal case, we can admit nothing short of the production of the octahedral crystals, and their subsequent identification.

It must also be remembered that the presence of certain substances in the arsenical solution may prevent the deposit of this metal upon the copper, viz., a chlorate, binoxide of manganese, or other bodies that decompose hydrochloric acid and evolve chlorine; likewise strong nitric acid. Consequently, Reinsch's test is not applicable to the clear solution obtained by boiling the viscera in hydrochloric acid and chlorate of potassium (vid. post.)

VI. Bloxam's method.—The principle here involved is the same as that in Marsh's process—the action of arsenic on nascent hydrogen; only, electrolysis is employed to decompose the water instead of zinc. It is a delicate and satisfactory method, but it should be remembered that the
arsenic must always be present in the form of arsenious acid.

There are some other reagents of inferior importance for the recognition of arsenic, as lime water, iodide of potassium, bichromate of potassium, etc., but these require no further notice.

Toxicological examination.—The analyst should always first search for particles of solid arsenious acid in the stomach and the vomited matter, and carefully remove these for examination. Organic mixtures should be diluted, if necessary, with distilled water, and acidified with about one-sixth part of hydrochloric acid and boiled gently for about fifteen minutes; when cooled the mixture should be strained and concentrated by evaporation over a water bath. A portion may now be subjected to a trial test by Reinsch's process; if no deposit takes place after boiling for half an hour, it is safe to conclude that no arsenic is present. But if deemed advisable, another portion of the filtrate may be subjected to Marsh's process, and to the action of sulphur-etched hydrogen.

The stomach and contents.—This organ should first be carefully examined as to its pathological condition (vid. ante, p. 61), and also for the presence of solid particles of the poison. It should then be cut up into small fragments, with scissors known to be perfectly clean, and together with its contents, placed in a clean, porcelain evaporating dish, distilled water added in sufficient quantity, together with about one-sixth the bulk of pure hydrochloric acid, and the whole boiled gently for about an hour, when most of the solid portions will have become disintegrated. After cooling, the mixture is thrown upon a muslin strainer, and the solid matters washed several times with pure warm water and squeezed. The strainer and contents should be pre-
served for subsequent examination, if required. The filtrate should be concentrated by evaporation over a water bath, and then filtered through paper.

Reinsch's process may now be applied as a trial test to a portion of the liquid. If no result is afforded after a sufficient boiling, other portions may be tried by Marsh's process, and by sulphuretted hydrogen; and if these give negative results, the absence of arsenic may be regarded as established.

But if the presence of the poison is revealed by the trial test, a given portion of the liquid may be completely exhausted by Reinsch's process (vid. ante, p. 251), and the balance treated with sulphuretted hydrogen for several hours, until all the arsenic is precipitated. This process is facilitated by gently warming the liquid. The resulting precipitate will have a dirty yellowish color—not the bright yellow where the arsenic is pure,—and will contain both organic matter and reduced sulphur, in greater or less amounts.

The mere production of such a precipitate is not sufficient, of itself, to establish the presence of arsenic, since it is known that in an acid, complex, organic solution, associated with coloring matter, sulphuretted hydrogen will throw down a precipitate very much resembling either an impure arsenical or antimonial sulphide, but consisting only of organic matter and free sulphur; hence, a further examination is required to verify this suspected sulphide.

The precipitate then should be washed carefully on a filter, and digested with pure aqua ammoniae, which will dissolve out all the sulphide of arsenic, together with some organic matter. The solution is filtered and carefully evaporated to dryness. If much arsenic is present it will have a decided yellow color. When perfectly dried it should be verified by the methods described above (p. 248).
If, however, only a minute quantity of arsenic be present in the dried residue, which will have a brown color, it must be purified as follows: it is placed in a porcelain capsule, and a little concentrated nitric acid is added, and the mixture evaporated to dryness over a water bath, the acid being repeated until the moist residue has a yellow color. It is next moistened with a few drops of solution of caustic soda, together with a little pure carbonate and nitrate of sodium well stirred, and cautiously evaporated to dryness. The heat is now gradually increased until the mass becomes colorless, when the organic matter may be considered completely destroyed. The cooled mass consists of a mixture of the arsenate of sodium with nitrate and nitrite of sodium. It should be dissolved in warm water, and after filtration, should be acidulated with pure sulphuric acid and evaporated till dense white fumes appear. By this treatment the residue is reduced to a mixture of the arsenate and sulphate of sodium. A portion of this solution may now be tested in a Marsh's apparatus; another given portion, by sulphuretted hydrogen, for quantitative determination, the arsenic acid being first reduced to arsenious acid by sulphurous acid, or sulphite of sodium.

Separation of absorbed arsenic from the tissues.—It is always desirable, if not indispensable, in a poison case, to prove the presence of absorbed arsenic in the different viscera, as the liver, kidneys, spleen, etc., inasmuch as its detection in the organs is positive proof that the poison had been actually taken during life, provided always that post-mortem imbibition can be excluded (vid. ante, p. 193). Besides, it may happen that, if the quantity swallowed has been only just sufficient to have caused death, the whole of it may have disappeared from the stomach by absorption, and can only be discovered in the organs. The brain should
always be examined; in the only case where this organ was submitted to an examination by us, arsenic was readily discovered by the usual tests.

Several methods are described for this sort of research, all having reference to one common end—the destruction of organic matters. In several instances, we have succeeded perfectly in detecting arsenic in the organs by simply boiling the finely-divided tissue in water and hydrochloric acid, and applying Reinsch's test.

Method of Fresenius and Babo.—The solid matters (as about one-fourth of the liver) should be finely divided, pressed in a mortar, and pure water added to bring it to the consistence of thin gruel. The whole should then be digested in a porcelain dish over a water bath, with pure hydrochloric acid about equal in weight to the dry material. Small quantities of powdered chlorate of potassium are from time to time added to the hot liquid, when effervescence will occur, with escape of chlorine gas. In a short time the solid matters will disappear, and the liquid will acquire a clear, yellow color. The heat should be continued until all odor of chlorine has disappeared. When the liquid has cooled, it should be properly strained. Any arsenic present would exist in the form of arsenic acid.

A portion of this liquid may be tested in a Marsh's apparatus (vid. p. 248). But for the other tests it is necessary that the arsenic acid should be reduced to the lower oxide—arsenious acid. This is effected by adding sulphite of sodium to the solution, and heating it until all odor of sulphurous acid has disappeared. It is now allowed to stand for several hours, and any deposit removed by filtration. The resulting solution may be examined by sulphuretted hydrogen, but not by Reinsch's process, for the reason above given (vid. p. 254).
Method of Danger and Flandin—The organs, properly divided, are introduced into a glass retort, together with strong sulphuric acid, and heated on a sand-bath, until the whole is thoroughly carbonized, and dried. After cooling, the mass is removed and powdered. The powder is moistened in a porcelain capsule, with one-tenth of its weight of pure nitric acid, and heated on a water-bath for half an hour. This converts the arsenic into arsenic acid. Warm distilled water is now added, and the matters filtered through paper. The filtrate is colorless, if pure; if colored, it must be evaporated to dryness, treated again with nitric acid and water, and filtered the second time. The acid liquid must next be evaporated to dryness, to get rid of the nitrous vapors. It should now be mixed with a sufficient quantity of water, when it will be fit for testing, as above described.

The distillation process.—The tissue should first be thoroughly dried over a water-bath, and then mixed with about its own weight of pure hydrochloric acid, and distilled in a retort over a sand-bath, almost to dryness, the distillate being received into a small quantity of water properly refrigerated. By this process the arsenic is separated as a terchloride. It possesses the advantage of immediately separating the arsenic, in a tolerably pure state, from the tissues. The distillate may be subjected to all the usual tests.

The urine can be examined by Reinsch's test, by first concentrating by evaporation; or it may be evaporated to dryness, and then treated with hydrochloric acid and chlorate of potassium, and examined in the usual way.

Arsenic is not a normal constituent of the human body. Neither is it ever found in the soil of cemeteries in a soluble state; consequently, there need be no apprehension of a
dead body ever imbibing this poison, after burial, from the surrounding earth.

Arsenic is estimated quantitatively, as a *sulphide*; 100 grains of pure dried sulphide represent 80.48 of arsenious acid.

**OTHER PREPARATIONS OF ARSENIC.**

*Arsenite of Potassium—Fowler’s Solution.*—This preparation, much used in medicine, is made by boiling arsenious acid with carbonate of potassium and tincture of lavender. It contains *four* grains of arsenic to the fluid ounce.

*Arsenic Acid.*—A powerful poison, but not employed as such. It is tested as arsenious acid; with sulphuretted hydrogen it yields a yellow precipitate after a considerable time. Its most delicate test is *nitrate of silver*, which yields a brownish-red precipitate—*arsenate of silver*.

The *N. Y. Medico-legal Journal*, March, 1884, contains an interesting account, by Prof. B. Silliman, of the death of a boy, aged between three and four years, from *arsenate of sodium*, a poisonous preparation sold in New York, under the name of *pest poison*, for destroying potato bugs. The most singular circumstance connected with the case is the entire absence of all the usual symptoms of arsenic poisoning, such as pain, vomiting and purging, etc.; but, on the contrary, those of a powerful narcotic, like *belladonna*, or *stramonium*. There were profound stupor, dilatation of the pupils, a rapid pulse, and hurried respiration. After partial recovery, a relapse took place, the child dying, apparently, from asphyxia, about nine hours after swallowing the poison.

*Arsenite of Copper—Scheele’s Green.*—A fine green powder, containing one part of arsenious acid to two of oxide of copper. By sublimation in a reduction-tube, it yields crys-
tals of arsenious acid. It is soluble in ammonia and in nitric acid.

_Aceto-arsenite of Copper—Schweinfurt, or Brunswick Green—Vienna, or Emerald Green—Paris Green._—A pigment very much used for staining wall paper, bon-bon paper, toys, etc. Also, to give a fine green color to articles of dress, artificial flowers, and millinery. It is composed of six parts of arsenious acid, two of oxide of copper, and one of acetic acid. It is readily identified by heating it in a test tube, when it gives off fumes of acetic acid, deposits crystals of arsenious acid, and leaves a residue of oxide of copper.

Paper, and other articles colored with this pigment, may be easily tested by dipping them into a weak solution of ammonia, when they will be speedily bleached, and the solution will become blue. If now a crystal of nitrate of silver be placed in the latter, a film of yellow is immediately formed around it—arsenite of silver. A drop of aqua ammonia applied to paper colored by this pigment immediately turns it blue.

Chronic arsenical poisoning is a frequent result of living in rooms whose walls are covered with this green paper; the fine powder or dust detached from the walls is inhaled into the lungs, and produces the symptoms above described.

_Sulphides._—There are two native sulphides, the yellow (tersulphide) or orpiment, and the red (pentasulphide) or realgar. The yellow sulphide is sometimes taken as a poison. They are both soluble in ammonia, and when mixed with a reducing agent and sublimed, they yield metallic mirrors (vid. ante, p. 248).
CHAPTER XVIII.

POISONING BY ANTIMONY—(TARTAR EMETIC).

PROPERTIES OF TARTAR EMETIC—SYMPTOMS—FATAL DOSE—POST-MORTEM APPEARANCES—SLOW POISONING—CHEMICAL ANALYSIS—TOXICOLOGICAL EXAMINATION.

The only preparation of antimony of medico-legal importance is tartar emetic. Occasionally, the chloride is a cause of poisoning.

Tartar Emetic (tartarized antimony; stibiated tartar, tartrate of antimony and potassium). This is a double salt, consisting of tartaric acid in combination with toioxide of antimony and protoxide of potassium. When pure, it occurs in large, colorless, octahedral crystals; also as a white powder. The commercial salt sometimes contains traces of arsenic. Heated in a reduction-tube, it readily blackens, from the decomposition of the organic acid, and is reduced to a mixture of carbon and metallic antimony. Heated on charcoal, before the blowpipe, it is also reduced, yielding globules of the metal, along with a white incrustation of the oxide.

It is soluble in three parts of boiling, and fifteen of cold water; its solution soon undergoes decomposition. It is insoluble in alcohol. A hot solution on evaporation yields tetrahedral crystals.

The taste is nauseous, metallic and acrid, or, according to some, slightly sweetish and styptic.

Symptoms.—A harsh, metallic taste is perceived on swallowing, soon followed by nausea, retching, violent and incessant vomiting, great thirst, constriction of the throat,
burning pain in the stomach and abdomen, profuse purging of a watery character; sometimes blood is found in the discharges both from the stomach and bowels; severe cramps in the extremities, a very feeble, rapid pulse, profuse perspiration, extreme prostration, with a disposition to syncope. The urine is generally increased in quantity, but is voided with pain; at times there may be delirium and convulsions preceding death. In exceptional cases, there is an absence of vomiting and purging, the symptoms being those of extreme collapse, with a cold, clammy sweat, feeble respiration, irregular pulse, delirium, unconsciousness and tetanic convulsions. (Husemann, Toxicol. p. 853.)

An occasional symptom, if the patient survives three or four days, is a pustular eruption over the body, similar to that produced by the external application of tartar emetic. In some instances it appears to exert a slightly corrosive impression, causing aphthous ulceration of the tongue and inside of the mouth.

While acting as an irritant to the gastro-enteric mucous membrane, it undoubtedly exerts a depressant effect upon the heart.

_Fatal dose._—This has not been precisely determined. A good deal depends on the idiosyncrasy. In some cases, two or three grains have produced alarming and even fatal effects, whilst, in others, enormous doses, up to an ounce, have failed to destroy life. Large doses, by exciting speedy vomiting, generally relieve themselves. Probably, twenty to forty grains may be regarded as the usual minimum fatal dose for an adult.

_Fatal period._—From an hour, up to several days. In an exceptional case related by Deutsch, a woman took by mistake a scruple of tartar emetic, and died one year afterwards, from the irritant effects on the alimentary canal.
Post-mortem appearances.—The irritant effects of this poison are displayed upon the lining membrane of the stomach and bowels, which is deeply reddened, softened and covered with a blackish, thick and viscid secretion, sometimes streaked with blood. The throat, oesophagus, stomach and bowels also exhibit aphthous-looking spots, or excoriations, and occasionally true pustules may be seen scattered throughout the intestinal tract.

The liver is generally enlarged and softened, and seems to have undergone a fatty degeneration. It is stated that the natives of Brunswick feed their geese upon the oxide of antimony, for the purpose of fattening them by increasing the size of their livers.

The lungs are often deeply congested, sometimes exhibiting a true apoplexy. The mucous lining of the windpipe and bronchi is uniformly reddened. The brain is generally congested, both in its membranes and substance, the latter presenting, when cut, numerous bloody points. The ventricles occasionally contain an excess of serum, and there may also be some sub-meningeal serous effusion. The heart exhibits nothing abnormal.

Treatment.—Vomiting should be assisted by warm mucilaginous drinks, or the stomach-pump may be employed. The proper antidote is tannin, in the form of some astringent vegetable infusion, such as green tea, or galls. Afterward, opium and stimulants will be necessary.

Chronic poisoning.—This method of poisoning is believed to be more frequent than formerly. The symptoms are a distressing nausea, with occasional vomiting, diarrhoea, with pasty stools, loss of appetite, emaciation, slimy tongue, feeble action of the heart, difficult breathing, a pale and anxious countenance, faintings, with increased perspiration and urination.
External application.—When applied to the skin, tartar emetic occasions deep pustulation; it is also readily absorbed, especially from abraded surfaces, and produces all its constitutional effects the same as if swallowed, such as nausea, vomiting, debility, etc. Fatal effects have thus resulted, and the poison has been detected, after death, in the stomach, liver, kidneys, and other organs.

Chemical analysis.—1. As a solid.—Touched with a drop of sulphide of ammonium, or a solution of sulphuretted hydrogen, it immediately acquires an orange-red color; this is characteristic of all the salts of antimony in their pure state. Heated in a reduction-tube, it blackens (vid. p. 262).

2. As a liquid.—(a) A drop of a strong solution, evaporated on glass, will exhibit the tetrahedral crystals; a weak solution gives a mass of confused crystals. (b) Either of the mineral acids dropped into it produces a white precipitate, soluble in an excess of the acid; this precipitate is also soluble in tartaric acid. (c) No precipitate by ferrocyanide of potassium. (d) Acidulated with hydrochloric acid, and boiled on bright copper foil, the latter acquires a violet-colored deposit of metallic antimony (Reinsch's test). (e) The above solution imparts a black stain to a strip of pure tin foil in the cold, whereby it is distinguished from arsenic. (f) Sulphuretted hydrogen, or sulphide of ammonium, throws down from a pure solution a characteristic orange-red precipitate of sulphide of antimony. This precipitate is soluble in caustic alkalies, but scarcely so in ammonia; insoluble in dilute hydrochloric acid, but if boiled in the concentrated acid, it is decomposed with the escape of sulphuretted hydrogen, and the formation of the terchloride of antimony. The resulting solution, if not too acid, when dropped into water immediately throws down a copious, white, flaky precipitate (the oxychloride, or powder
of algaroth), which is quite characteristic. This may be identified as antimonial (1) by its solubility in tartaric acid; (2) by touching it with sulphide of ammonium, which imparts to it an orange-red color. The white precipitate obtained by dropping the nitrate of bismuth into water is not soluble in tartaric acid, and is blackened by sulphide of ammonium.

(g) The Galvanic test.—This is made by placing a few drops of the solution, acidified by hydrochloric acid, upon a platinum capsule, and touching the latter, through the liquid, with a strip of bright zinc; metallic antimony is deposited on the platinum at the point of contact, as a brownish or black film. The liquid should then be poured off, and the platinum washed in distilled water. A small quantity of sulphide of ammonium poured upon the stain speedily dissolves it (if antimony) by the aid of heat, and on evaporation, an orange-red sulphide remains. A modification of this test may be advantageously applied for the detection of antimony in the organs (vid. post.)

(h) Marsh's test.—This is employed in the same manner as for arsenic (vid. ante, p. 248). If a solution of tartar emetic, or any of the soluble antimonial salts, be subjected to Marsh's test, antimonetted hydrogen is generated in precisely the same manner as is arseniureted hydrogen, under the same conditions.

(1) If the gas is inflamed at the jet, it burns with a bluish flame, evolving white fumes of teroxide of antimony, and if these fumes are received into a short, wide test-tube, held just above the flame, the white deposit of the teroxide may be collected, which may be identified by sulphide of ammonium. If a piece of cold white porcelain be held horizontally just within the flame, the metal is deposited (as in the case of arsenic) in the form of a black, or nearly black,
spot, which is usually surrounded by a grayish ring. These deposits may be multiplied by simply changing the position of the porcelain.

The only fallacy to which this test is liable is from arsenic, which, as has been shown, behaves in a precisely similar manner. But they can readily be distinguished from one another by a little attention. The antimonial deposit is, as a rule, blacker and less brilliant than the arsenical; but if the spots of antimony are extremely small—as when the quantity examined is minute—this distinction is not so observable. Again, the antimony stains are more slowly dissipated by heat than the arsenical; the former immediately dissolve in a drop of sulphide of ammonium, leaving, on evaporation, an orange-red deposit; the latter (arsenical) are slowly affected by it, and leave, on evaporation, a yellow residue. Furthermore, the arsenic deposit is immediately soluble in a solution of hypochlorite of sodium, which has little, or no effect upon the antimonial stain. Nitric acid will also serve to distinguish them: both are dissolved by it, but on evaporation to dryness, the arsenical residue gives to a solution of nitrate of silver a brick-red color (arsenate of silver), but the antimonial residue is not affected by it.

(2) If heat be applied to the horizontal tube in Marsh's apparatus during the passage of the antimonetted hydrogen, decomposition takes place, as in the case of arsenic, but the deposition of the antimonial mirror occurs immediately over and around the heated portion, and not in advance of it. If the quantity operated upon is very small, the deposit may take place wholly within the point of heat. These metallic deposits exhibit the same chemical reactions as those produced on porcelain by the ignited gas.

(3) If the antimonetted hydrogen be passed into a solu-
tion of nitrate of silver, the latter (as in the case of arsenic) becomes black; the whole of the antimony is precipitated as *antimonide of silver* (with arsenic, the precipitate consists of metallic silver, the arsenious acid being kept in solution). This black precipitate should be collected on a filter, washed and boiled with *tartaric acid*, which dissolves out the antimony, and leaves the silver. On filtering the solution, and treating it with sulphuretted hydrogen, the characteristic orange-red sulphide is precipitated.

**Toxicological examination.**—In certain cases it might be desirable to separate the tartar emetic, as such, from the stomach; this may sometimes be accomplished by *dialysis*. The exhibition of the poison in the exact state in which it had been swallowed, would always strongly impress a jury. But for all practical purposes, it is deemed sufficient if the analyst can detect the *antimony*. The process of dialysis may also be employed to separate tartar emetic from food and vomited matters, but not, of course, for detecting absorbed antimony in the tissues.

The stomach, properly divided, and contents, should be acidulated with *tartaric acid* and gently heated, with sufficient distilled water, over a water bath, for about half an hour. When cold, the matters should be strained through muslin; the solid portions are to be washed and pressed, and the whole of the liquid carefully evaporated to about one-half. Trial tests may now be made on a portion of this liquid (a) by inserting a piece of pure tin foil in the cold; it will soon blacken if antimony is present; (b) acidulate with hydrochloric acid and boil; then place a piece of bright copper foil (Reinsch's test), it will speedily acquire a violet stain. (c) The remainder of the liquid, slightly warmed, should be treated with sulphuretted hydrogen gas for several hours; a dirty, orange-red or brown precipitate
will be thrown down, consisting of tersulphide of antimony, organic matter and reduced sulphur (*vid. ante*, p. 256).

The importance of identifying this precipitate cannot be over-estimated by the toxicologist. The mere production of a reddish-brown deposit under these circumstances is not sufficient to establish the presence of the alleged poison; what has been said upon this point under the head of Arsenic applies with equal force to antimony. Neither will it suffice to proceed only one step farther, and dissolve the suspected sulphide in boiling hydrochloric acid, and throw the resulting solution into water, and obtain a white precipitate therein, since all these results may ensue, as our experience can testify, from a similar treatment of the colored sulphur-organic deposits above alluded to, where no antimony has been present; for these also are, to a great extent, soluble in hot hydrochloric acid, and the resulting solution, if thrown into water, will occasion a white precipitate. We do not wish to be understood as saying that these sulphur-organic precipitates possess all the characters of the true sulphide of antimony—especially such as would result from a pure solution,—but they do resemble in many respects the precipitate from an antimonial solution *mixed with organic matters*. It is for this reason that we insist, in a poison case, on a very searching corroboration of this particular test. The analyst should proceed still another step, and subject the white precipitate to the action of tartaric acid, and of sulphide of ammonium (*vid. p. 266*).

It may properly be remarked here that in every medico-legal case of poisoning with antimony (as indeed with other metals), the actual obtaining of the metal should be rigorously insisted on, as the only absolute and unequivocal proof; and this, too, in quantities sufficient to admit of its positive identification by all the recognized tests. Nor should
this be considered as a mere arbitrary or capricious rule. The highest toxicological authorities, such as Orfila, Tardieu and Taylor, sanction it. Besides, the extraction of the metal is not difficult, e. g., by tin foil, by galvanism, by Marsh’s and Reinsch’s processes, and by the blowpipe.

In the organs and tissues.—Most of the absorbed poison will be found in the liver and kidneys. A given portion of these organs, properly divided, should be boiled in water acidulated with about one-sixth of hydrochloric acid. After proper concentration, trial tests may be made with a strip of tin foil in the cold, and copper foil in the boiling solution. If any indications of antimony are given, Reinsch’s process may be carried out by subjecting a number of pieces of copper to the boiling acid liquid. These should be thoroughly washed, and dried between the folds of bibulous paper; then rolled up, and introduced into glass reduction-tubes, and heated by the flame of a spirit lamp; a white sublimate will be deposited on the cool portion of the tube, as in the case of arsenic; but it is either amorphous, or else composed of very fine acicular crystals (Miller’s Inorganic Chem. p. 602).

The true nature of this antimonial deposit is best shown, according to Watson, by boiling the coated copper in a dilute solution of caustic potassa, the metal being occasionally withdrawn from the liquid and exposed to the air to favor the oxidation of the antimony, when, after a time, the deposit will be wholly converted into antimonate of potassium, which will be in solution. The copper strip should now be removed; acidulate with hydrochloric acid, and pass sulphuretted hydrogen through the liquid, when the pentasulphide of antimony will be thrown down, of an orange-red color. The whole of the antimony may be thus removed by employing successive slips of copper, and subjecting them to the above treatment.
The galvanic test may also be applied with great certainty, to detect the presence of antimony in the tissues. Prof. Taylor's plan is an excellent one: Coil a portion of pure zinc foil around a piece of clean platinum foil, and suspend them in the acid solution of the tissues, sufficiently dilute to prevent too violent an action on the zinc. The liquid should be warmed. Sooner or later, according to the quantity of antimony present, the platinum will be coated with an adhering black powder of metallic antimony. Wash the platinum foil, and digest it in strong nitric acid, which will dissolve off the antimony; remove the platinum, and evaporate to dryness. Re-dissolve the residue in hydrochloric acid; dilute the solution, and treat with sulphuretted hydrogen, which will precipitate the pure sulphide; or, the deposit on the platinum may be dissolved off by sulphide of ammonium (vid. p. 266).

The absorbed antimony may also be extracted by means of chlorate of potassium and hydrochloric acid (vid. p. 258). But, in this case, in the subsequent application of sulphuretted hydrogen, there is no occasion to employ sulphurous acid to effect a reduction to a lower oxide, as in the case of arsenic.

It has been ascertained that antimony may be eliminated through the glands of the stomach, even when introduced into the system by some other avenue, e. g., by antimonetted hydrogen through the lungs.

The urine should always be examined in cases of suspected antimonial poisoning. This secretion is very soon affected by the salts of antimony, and it may contain traces of them for some time after their discontinuance. The urine should be evaporated nearly to dryness, when it may be examined either by Reinsch's test, by tin foil, by the galvanic test, by Marsh's process, by chlorate of potassium
and hydrochloric acid, with sulphuretted hydrogen, and by carbonizing with sulphuric acid (vid. post., p. 259).

*Chloride of Antimony (Butter of Antimony).*—A strong, corrosive poison, and one that has proved fatal in a number of instances. Its symptoms and post-mortem lesions resemble those of the corrosive acids rather than those produced by tartar emetic. When thrown into water, the *oxychloride* is generated, and falls as a copious, white, flaky precipitate. This is soluble in tartaric acid, and is instantly colored orange-red when touched with sulphide of ammonium. The clear liquid contains hydrochloric acid, as shown by nitrate of silver, which precipitates *chloride of silver*.

Antimony is estimated quantitatively as a *tersulphide*. Every 100 grains of pure, dry tersulphide are equivalent to 85.75 of the teroxide, or 202.85 parts of crystallized tartar emetic.
CHAPTER XIX.

POISONING BY MERCURY—(CORROSIVE SUBLIMATE).

CORROSIVE SUBLIMATE.—PROPERTIES.—SYMPTOMS.—POST-MORTEM APPEARANCES.—FATAL DOSE.—ANTIDOTES.—CHEMICAL ANALYSIS.—TOXICOLOGICAL EXAMINATION.—SALIVATION.

Mercury is not poisonous in the metallic state. Liquid mercury was formerly administered to relieve constipation. The vapor is poisonous when inhaled, and as this is given off from the metal, even at ordinary temperatures, it happens that artisans who work in mercurial ores, looking-glass platers, water gilders, barometer makers, etc., are very liable to become poisoned by the fumes. The symptoms of this sort of poisoning may come on gradually, or suddenly; they may or may not be accompanied with salivation. They are chiefly marked by the production of tremors of the limbs and paralysis, indicating the action of the metal on the nerve centres. The general condition thus induced is named mercurial tremors, and shaking palsy. The upper extremities are usually first affected, and then, by degrees, all the muscles of the body. There is an unsteadiness in the arms and legs, so that the patient cannot grasp an object, nor walk firmly on the ground. In bad cases, he can neither speak, nor chew his food. If the disorder be not checked, it proceeds to a fatal termination, attended with a loss of memory, insomnia and delirium. Another curious symptom, not generally recognized, but usually present, is a brittle state of the teeth, causing them to chip (Guy's Foren. Med.)

The proper prophylactic treatment in this affection consists in cleanliness and good ventilation, together with the
free internal use of albumen, in the form of white of eggs.

All the mercurial compounds are more or less poisonous, but the most important one, from a medico-legal point of view, is corrosive sublimate.

**Corrosive Sublimate**—(*Mercuric Chloride—Corrosive Chloride of Mercury*).—Occurs either in heavy crystalline masses of prismatic crystals, or as a white powder. It has a powerful metallic, styptic, nauseous taste, and is soluble in about sixteen parts of cold, and three of boiling water. Alcohol and ether, also, freely dissolve it, and the latter has the power of abstracting it from its aqueous solution.

**Symptoms.**—These usually come on immediately after taking the poison. A strong metallic, styptic taste is perceived, with a sense of heat, and choking in the throat. A fierce, burning pain is felt, extending from the mouth to the stomach; nausea, retching and vomiting of stringy mucus, often tinged with blood; pain in the abdomen, which usually is swollen and tender to the touch; severe purging, sometimes of bloody matters, accompanied with tenesmus, as in dysentery. The pulse is feeble, quick and irregular; countenance flushed and swollen, though sometimes it is pale and anxious; the tongue is white and shriveled; skin cold and clammy; respiration difficult; intense thirst; urine scanty or suppressed; cramps of the extremities; stupor, fainting, convulsions and death. *Salivation* is apt to appear on the second or third day, but it is not an invariable symptom in acute cases.

In some exceptional instances there has been an absence of abdominal pain, as also of vomiting and purging.

Poisoning from corrosive sublimate differs from arsenical poisoning: (1) the former poison has a very distinct acrid taste, whilst the latter is almost tasteless. (2) The symp-
toms of the former come on almost immediately after it is swallowed; those produced by the latter are generally postponed for half an hour to an hour. \(3\) The discharges from corrosive sublimate poisoning are more frequently bloody than those from arsenic.

The external application of corrosive sublimate has often been attended with fatal consequences, and both the symptoms and post-mortem lesions, in such cases, resemble those produced by swallowing the poison, such as vomiting, purging, suppression of urine, salivation, etc., injection of the stomach and kidneys, with ecchymoses throughout the intestines and bladder. Cases of this character, resulting fatally, are reported, where a solution of corrosive sublimate was applied to the scalps of children, for the cure of porrigo and ringworm.

Fatal dose.—The minimum fatal dose for an adult may be considered to be three grains, although, as in the case of other mineral poisons, very large quantities have been taken with impunity, having been speedily vomited, or promptly neutralized by proper antidotes.

Fatal period.—Dr. Taylor reports the shortest period on record, where death occurred in half an hour from an unknown amount of the poison. In the majority of cases life is prolonged for several days—from one to five. In a summary of cases given by Prof. Guy (Foren. Med. 1868, p. 475), about half the number died in less than twelve hours, and the remaining half in a period varying from three to eleven days. More than one-half the cases terminate fatally.

Treatment.—Promote vomiting by the free use of warm diluent drinks. The proper antidote is albumen, as found in eggs. This decomposes the mercurial salt, forming an insoluble albuminate; a large excess of albumen will re-
dissolve the precipitate. The white of one egg is supposed to be capable of neutralizing four grains of corrosive sublimate. In the absence of eggs, gluten or wheat flour, in the form of paste, may be freely exhibited. Milk may also be freely used. 

Post-mortem appearances.—These are generally confined, as in the case of arsenic, to the mucous membrane of the stomach and bowels, but the corrosive action of the mercurial is more marked. The stomach, together with the mouth, throat and oesophagus, is often softened, of a white or grayish color, and corroded. The slate-gray color is ascribed to the reduction of metallic mercury upon the lining membrane. The intestines, especially the cæcum, often exhibit similar appearances. Perforation of the stomach is rare. The kidneys and bladder are usually highly inflamed, the former especially congested about the Malpighian bodies, and the epithelial cells deformed, granular, and partially destroyed. The bladder is empty and contracted.

According to Dr. Byasson (Woodman and Tidy, Med. Jurisp. p. 204), corrosive sublimate takes two hours to reach the urine, and four hours to reach the saliva. He never found it in the perspiration. He considers it to be completely eliminated in twenty-four hours after it has been taken.

In chronic or slow mercurial poisoning, the symptoms generally presented are loss of appetite, metallic taste in the mouth, fetid breath, soreness of the gums, increase of salivary secretion, pain in the stomach and abdomen, with diarrhœa, quick pulse, hot skin, weakness and emaciation. A bluish line has been noticed at the edge of the gums, as in lead poisoning.

Salivation, although often absent in acute mercurial poi-
NON-MERCURIAL SALIVATION.

soning, is nearly always observed in the chronic form. But as this symptom accompanies the use of many other drugs, it cannot, of itself, be regarded as a proof of the administration of mercury. In a doubtful case, however, the matter may always be decided by a chemical examination of the saliva for mercury (vid. infra). Doubtless, other mineral poisons are eliminated by this secretion, and their presence might be detected in it, with proper attention.

The relationship between salivation and mercurial poisoning is a subject of considerable medico-legal importance, since charges of malpraxis have often been made against physicians in cases of profuse and fatal salivation, accompanied by necrosis and gangrene, where, in some instances, no mercury whatever had been administered, and in others, where the dose has been exceedingly small. It is well known that there is no fixed, definite period when the salivation comes on; rarely before two days, often later. A case reported by Dr. Wood (Ed. Med. and Surg. Jour., vol. LI, p. 141), in which a teaspoonful of corrosive sublimate had been swallowed, salivation was profuse in the course of a few hours. It has been suggested that this very early flow of saliva was probably due rather to the local irritant action of the poison, than to the result of absorption.

An important fact, not to be lost sight of, in this relation is that salivation may be produced by various other agents besides mercurials, such as iodide of potassium, iodine, the preparations of copper, lead, bismuth, arsenic, antimony, digitalis, croton oil, cantharides, colchicum and other drugs. A case was recently mentioned to the author where a patient was profusely salivated by a single dose of five grains of iodide of potassium. It is true that in the majority of the instances of non-mercurial salivation, there is an absence of the usual mercurial fetor of the breath and the coppery
taste, but it would appear, from some recorded cases, that these symptoms have been equally noticed in the salivation produced by arsenic and bismuth.

Another point of consideration for the legal physician is the great difference in the susceptibility of persons to the mercurial impression. Thus, it is almost impossible to salivate a very young, healthy, child. Certain morbid conditions of the system, however, seem to predispose to its action, as anaemia and albuminuria. We have known a dose of compound cathartic pills (containing only three grains of calomel) to produce very severe ptialism. Dr. Christieon states that three five-grain doses of blue pill, one every night, proved fatal; and that two grains of calomel have caused ulceration of the throat, exfoliation of the jaw, and death.

Mercurial salivation may be intermittent, ceasing for a time, and reappearing without the further exhibition of the medicine during the interval.

Furthermore, salivation may arise spontaneously, from mechanical irritation of the mouth, or as the result of exhausting diseases, especially among the children of poverty and squalor, who are surrounded by bad hygienic influences. Among the last-named subjects, the two diseases of cancrum oris and of gangrene of the mouth are of frequent occurrence. The symptoms of these conditions strongly resemble a very severe case of mercurial ptialism, so that the diagnosis may be difficult. If, in such a case, the physician should have happened to have administered, at the beginning of the sickness, even a small dose of calomel, it might easily become a serious question to determine whether the death actually resulted from the mercury acting as a poison, or from the disease; and it would be no difficult matter to get up an action against the medical man
for alleged malpraxis. Dr. Taylor cites a case in point (On Poisons, p. 406). A charge was made against a medical practitioner for having caused the death of a child, aged four years, by administering an overdose of some mercurial preparation, for the treatment of whooping cough. On the fourth day the child complained of soreness of the mouth; the teeth became loose and fell out; the tongue and cheek were much swollen, and the child died, in the course of a few days, from gangrene of the left cheek. The answer to the charge was, that not a particle of mercury had been given—a fact clearly proved from the prescription-book of the medical attendant. This was clearly an instance in which gangrene from spontaneous causes had been mistaken for mercurial poisoning. As before observed, the chemical analysis of the saliva would settle any question of this kind.

Chemical analysis.—(1.) As a solid.—(a) A fragment heated on platinum foil is entirely dissipated in white, acrid fumes, which condense on a cool surface in white, radiating crystals. (b) Touched with a drop of liquor potassae, it turns a yellowish color; calomel, under similar circumstances, becomes black. (c) A solution of iodide of potassium imparts a bright scarlet color; this is a very delicate test. A drop of this latter solution placed upon a piece of bright copper, in contact with the smallest fragment of corrosive sublimate, will produce a bright, silvery stain upon the copper, especially if it be rubbed with the finger; this stain is immediately removed by heating it. (d) Sulphide of ammonium at first turns it yellowish, but subsequently black. (e) Heated in a reduction-tube with dried carbonate of soda, it sublimes, forming a white ring on the cool part of the tube, which, under the microscope, is seen to consist of minute globules of metallic mercury. The white residue in the tube is
shown to contain chlorine, by dissolving it in water and applying nitrate of silver.

(2.) As a liquid.—(a) A drop evaporated on a glass slide will yield large, needle-shaped or prismatic crystals. (b) Solution of potassa gives a yellow oxide of mercury; this, when dried and heated in a reduction-tube, will yield a sublimate of mercury globules, with the evolution of oxygen gas. (c) Ammonia produces a white precipitate. (d) Iodide of potassium first causes a yellow and immediately afterwards a bright scarlet precipitate, soluble in an excess of the reagent. When this iodide of mercury is dried and heated, it volatilizes, and condenses in a yellow deposit, which gradually changes to scarlet. (e) Protochloride of tin first throws down a white precipitate (calomel), and, if in excess, a dark gray precipitate (metallic mercury), which runs into globules on being boiled. (f) Sulphuretted hydrogen and sulphide of ammonium first cause a whitish precipitate, soon changing to red, and ultimately to black. (g) The copper test. A piece of bright copper put into a cold solution of corrosive sublimate, acidulated with hydrochloric acid, speedily acquires a silvery white coating of metallic mercury. When the copper slip is dried and heated in a reduction-tube, a sublimate of metallic globules is obtained, easily identified by the microscope. This test is extremely delicate, and will detect the $\frac{1}{10}$ of a grain, if the copper surface is very small and is heated in a very small tube. According to Wormley (Micro-Chem. of Poisons, p. 339), a far smaller quantity—even the $\frac{1}{1000}$ of a grain—may be identified by employing a very small, thin, glass tube, the one-tenth of an inch in diameter, and drawing it out, by heating, into a thin capillary neck. The small fragment of coated copper is introduced through the wider portion of the tube to the point of contraction, and the wider end is
now carefully fused shut by the mouth blowpipe, so as to give it the appearance of a small thermometer tube, the bulb containing the coated copper. The tube is now heated at the bulb, and the capillary end closed. On examination under the microscope, a well-defined ring of mercurial globules will be visible on the capillary tube, just above the bulb.

In case the mercurial sublimate in the reduction-tube should be dim and unsatisfactory, Tardieu (Sur l' Empois., p. 580) recommends to introduce a minute crystal of iodine into the tube, pushing it down as far as the sublimate with a platinum wire. The open end of the tube is then stopped with wax, and it is kept in a horizontal position for about twelve hours, at a temperature of 30° or 40° C., when the deposit will assume a bright scarlet color, due to the production of iodide of mercury. After removing the iodine, the tube may be gently and progressively heated from below by the flame of a spirit lamp, when the scarlet color will change to yellow, and on cooling the latter color will give place to scarlet again.

(h) The galvanic test.—This consists in winding a strip of gold around a strip of zinc (or iron) and placing the coil in the acidulated solution. Prof. Guy recommends a simpler and equally certain method—to moisten a narrow slip of zinc with water, and to take up as much gold leaf as will adhere to it, and introduce this into the acid solution. The gold will, in a short time, become coated over with a silver-colored coating of mercury. It is then to be carefully washed and dried, and heated in a reduction-tube, when the usual mercurial sublimate will be obtained. The gold test is generally regarded as the most delicate of all.

Toxicological examination.—In a case where the poison was administered in the solid form, fragments of it may be
found in the stomach yet undissolved; these should be collected and identified. But as corrosive sublimate is easily decomposed by albumen, gluten and other substances, much of it may be changed into insoluble compounds. If the quantity in solution is considerable, it may be extracted by simply agitating it with twice its volume of ether, and after it has settled, removing the ether by means of a pipette, and allowing it to evaporate spontaneously, when the salt will crystallize in white, silky prisms. These may be purified, if necessary, by dissolving in water or alcohol, and again crystallized. This method has the advantage of recovering the poison in the exact state in which it was swallowed, with the reservation, however, that ether would act upon any mercurial salt in the presence of an alkaline chloride—e.g., chloride of sodium—in a similar manner.

The stomach and its contents should be prepared in the usual manner, already described, and heated with distilled water and hydrochloric acid. After proper filtration and concentration, a trial test may be made with a strip of copper, allowing this to remain in the solution, if necessary, for several hours. The gold test may be applied in a similar manner. By either of these processes the metal can be recovered in a satisfactory manner. The other tests above mentioned may also be applied as corroborative proofs.

The solid matters remaining, after straining off the liquid, will probably contain much of the poison combined with organic substances. These should be boiled in distilled water with hydrochloric acid, until disintegrated, then filtered and concentrated, and tested as above. Another method is to dry the solid matters thoroughly, and digest them in warm nitro-muriatic acid, by which the insoluble mass is converted into soluble corrosive sublimate. The acid liquid is then evaporated to dryness, the residue dis-
solved in water and filtered, and the usual tests applied; or the corrosive sublimate is dissolved out by ether.

In the tissues.—The liver, or other organs, should first be crushed in a mortar, with sufficient alcohol to render filtration easy. Acidulate the mass with hydrochloric acid, and gently warm for some time; then filter through paper, and apply the copper or galvanic test, and sulphuretted hydrogen to the filtrate. All the solid portions are now to be mixed with water and four parts of hydrochloric acid, and boiled for some time; when cool, they are to be filtered and the filtrate examined as above.

Should arsenic happen to be present in the tissues along with corrosive sublimate, on the application of Reinsch's test both metals will be precipitated on the copper, and both will sublime from the latter when it is heated in the reduction-tube; but in the cold, mercury alone will be deposited on the copper.

To detect mercury in the saliva, acidulate about two drachms of this fluid with one-fourth of hydrochloric acid, and introduce into the mixture a small piece of bright copper foil, and the whole kept warm for several hours. The silvery deposit on the metal will indicate the presence of mercury, which will be confirmed by heating it, when washed and dried, in a reduction-tube, and procuring the characteristic mercurial globules by sublimation.

In examining the urine, evaporate about twelve or fourteen ounces down to one ounce; acidulate this with hydrochloric acid; filter and boil the filtrate, and introduce a fragment of bright copper, and confirm as directed above.

It should be remembered that death may ensue from corrosive sublimate, and no mercury be found in the tissues, as where the person has survived for a number of days. Also, as in the case of other poisons, there may be a failure to
detect it in the stomach after death, even when large doses had been swallowed.

On the other hand, the detection of minute quantities of mercury in the organs is not always evidence of poisoning, inasmuch as the person may have lately taken blue pill or calomel as a medicine; hence, caution should be exercised in reference to this point.

Corrosive sublimate is usually estimated quantitatively, as a sulphide, by carefully washing and drying the precipitate obtained by sulphuretted hydrogen. Every 100 grains of dried sulphide are equivalent to 116.81 grains of anhydrous corrosive sublimate. Sometimes the protochloride of tin is used to precipitate metallic mercury from a given quantity of the mercurial solution. The globules should first be purified by boiling them in a solution of potassa, and afterwards in hydrochloric acid. Every 100 grains of metallic mercury represent 135.5 grains of corrosive sublimate.

Other compounds of mercury may occasionally prove poisonous, as the red and white precipitates, red oxide, calomel, the sulphides, nitrates and sulphates.
CHAPTER XX.

POISONING BY LEAD.

FREQUENCY OF CHRONIC POISONING.—ACETATE OF LEAD.—SYMPTOMS.—TREATMENT.—POST-MORTEM APPEARANCES.—PAINTERS' COLIC.—LEAD PALSY.—TOXICOLOGICAL EXAMINATION.

In the metallic state, lead is not considered poisonous; but, as it is easily oxidized by the fluids of the stomach, it would soon be converted into a deleterious compound. All its salts are poisonous, with perhaps the single exception of the sulphate, which is very insoluble.

Acute poisoning by lead is very rare, except as the result of accident. On the other hand, chronic, or slow lead-poisoning is of frequent occurrence, since there is no metal more constantly and insidiously introduced into the human system than lead, under its varied forms. In the arts, the workmen in this metal inhale the fumes and powders in smelting the ores, and manufacturing white lead. Painters, plumbers, pewterers, and glazers of pottery are all exposed to similar danger. Even sleeping in a freshly-painted room has been known to cause an attack of colic and paralysis, from breathing the emanations containing carbonate of lead. Dr. Taylor (On Poisons, p. 434) alludes to himself as having suffered from this latter cause.

The frequent handling of pewter vessels, and especially of new type, has produced lead palsy. The use of glazed pottery is another source of contamination, arising from the action of acids, such as vinegar, and of oils and fats, also of alkalies, on the glaze, which consists of litharge. Even
milk has become poisoned in this way. Cider and beer, drawn through leaden pipes, may become contaminated in the same manner. Wine may become affected by contact with the shot left in the bottles through carelessness. New rum is apt to contain lead, derived from the leaden worm of the still, while old rum is free from this adulteration. This is ascribed, with great probability, to the fact that old rum, being kept in oak casks, is deprived of its lead by the tannin of the cask.

Certain medicinal substances often contain lead, derived from the mode of their manufacture; thus, carbonate of ammonia, sublimed in leaden vessels; borax, and other salts, crystallized in leaden pans; tartaric acid, from the lead attached to the strings used in its crystallization. Solutions of soda and potash, when kept in flint-glass bottles, soon become impregnated with lead; and the sulphuric acid of common use almost invariably contains lead, derived from the leaden chambers.

Many articles in domestic use are not unfrequently contaminated by lead, as flour (from the plugs of lead imbedded in the millstones), sugar, snuff, tobacco, chocolate, and bonbons—the latter articles from the impure tin-foil wrappers.

The external application of the preparations of lead is often the cause of slow poisoning, as in handling the metal, already alluded to; the use of hair dyes and cosmetics; and even from the glazed lining of hats. The direct application of white lead to the scalded surface, as a dressing, has been known to produce symptoms of lead colic.

Probably the most frequent source of chronic lead poisoning is through drinking water, which has, in some way or other, been in previous contact with metallic lead. The conditions under which this occurs should be thoroughly understood by the legal physician. Pure distilled water, which
in nature is represented by rain and melted snow, in contact with lead and exposed to the air, speedily acts upon the metal, producing a mixture of the hydrated oxide and carbonate of lead, which are very poisonous. Consequently, rain water, which is frequently preserved in cisterns for drinking purposes, should never be collected from a leaden roof, nor be conducted through leaden pipes, nor in any way come in contact with this metal. On the other hand, river and spring water, which always contain more or less of saline ingredients, exert no deleterious influence upon lead, in consequence of the preservative action of the carbonates, sulphates and phosphates existing in the water; these form an insoluble film or coating upon the surface of the metal, thus preserving it from any further action of the water. The presence of the chlorides, nitrates and nitrites would, on the contrary, increase its corrosive action on the lead. Free carbonic acid in the water very much increases the danger of contamination, in the absence of any protecting salt, in consequence of its solvent power over carbonate of lead.

Another cause of contamination may arise from a galvanic action between lead and other metals soldered together, and especially when in contact with carbonic-acid water. Danger also arises from a leaden cover being over a pump or cistern. The vapor of the water (which is equivalent to distilled water), impinging on the metal surface, dissolves off the poisonous oxide and carbonate of lead, which may in time fall into and contaminate the water.

The only compounds of lead, of medico-legal interest, are the acetate and carbonate.

**Acetate of Lead**—*(Sugar of Lead)*.—Occurs in commerce in masses of white, or light-brownish crystals, somewhat resembling loaf sugar in appearance. It has an acetous odor, and a sweetish, astringent taste. Soluble in water; less
so in alcohol. Its aqueous solution becomes milky on exposure to the air, owing to the carbonic acid of the latter.

It is not a very active poison; it is much used in medical practice, but its continued employment has occasionally resulted in bringing on symptoms of lead poisoning.

In doses of an ounce or two, it acts as a powerful irritant, causing burning pain in the throat and stomach, and thirst, vomiting, twisting, colicky pains, with tenderness in the abdomen, obstinate constipation, retraction of the abdominal walls, anxious countenance, cold sweats and convulsions. The urine is diminished in quantity. In protracted cases, there is often paralysis of one or more of the extremities. Its influence on the nerve centres is marked by the giddiness, stupor, convulsions and coma. In some cases, there is purging of bloody matters, though usually the fecal discharges are hard, dry and black. The peculiar blue line upon the edge of the gums, characteristic of chronic lead poisoning, may sometimes be observed in acute cases.

The fatal quantity is uncertain; an ounce has been swallowed with impunity, though a less quantity has occasioned alarming symptoms. The fatal period is equally uncertain, varying from a few hours to several days.

The proper antidotes are the soluble alkaline and earthy sulphates, especially the sulphate of magnesia, which forms the insoluble sulphate of lead. At first, however, vomiting should be promoted by sulphate of zinc; afterwards, opium and castor oil may be required. The urine should be frequently examined for the presence of the metal.

Post-mortem appearances.—Often no well-marked lesions are discoverable. Again, more or less inflammation of the alimentary tract has been observed. Sometimes the inner coat of the stomach and bowels is covered with a thick, whitish layer of mucus mixed with the salt of lead, beneath
which the membrane is reddened, or even abraded. The intestines are generally found contracted. As regards chronic cases, there is nothing very definite to record, except the contraction of the large intestines, and the flabby and whitish appearance of the muscles specially affected.

**Chronic poisoning.**—This may result from the continued internal use of any of the salts of lead; but it is more frequently produced among artisans working in white lead and litharge, or by the accidental introduction of the metal into the system through drinking-water, or articles of food. The earlier symptoms are grouped under the names of *Lead Colic*, *Painters' Colic*, or *Colica Pictonum*; the later symptoms are named *Lead Palsy*.

**Lead colic.**—The earlier symptoms are obstinate constipation and indigestion, with great depression. Then there is a feeling of twisting, grinding pain about the umbilicus, which may be rather relieved by pressure. The abdomen is hard and retracted; sometimes there are scanty, hard evacuations, with much suffering. The urine is scanty, and voided with difficulty. The countenance is dull and anxious; skin cold and clammy; pulse about natural; respiration quick and catching; loss of appetite, with dryness of mouth and throat; the breath is fetid, and often a metallic taste in the mouth. A characteristic sign of saturnine poisoning is the *blue line* at the margin of the gums, where they join the teeth, especially noticeable on the upper incisors. This is due to the deposition of the metal in the form of a sulphide in the capillaries of the gums, as can be shown by the microscope. Some other metals, as silver and mercury, occasionally produce a somewhat similar blue line, and it is wanting in some exceptional cases of chronic lead-poisoning. When once established, this symptom is very persistent.
Lead palsy.—Lead colic, if allowed to continue unchecked, is very apt to terminate in paralysis, especially after repeated attacks of the former. Again, it may come on without any previous attack of colic. It usually affects the upper extremities. At first there is a dull, numb feeling in the skin of the fingers and forearms, trembling of the arms and legs, unsteadiness of gait, loss of power in the hands and arms, which gradually waste away. The extensors are more affected than the flexors, so that, when the arm is raised, the hand drops by its own weight, whence the common name of “wrist drop” for this disease. If unchecked, brain symptoms present themselves, such as giddiness, torpor and apoplexy; sometimes there are epileptic fits, oedema, albuminuria and convulsions, ending in coma and death.

Lead has been found, after death, in the brain, and especially in the gray matter of the spinal cord, also in the bones, liver and kidneys. Doubtless, many cases of obscure spinal, cerebral and cardiac disease are really owing to chronic lead poisoning—the metal having been introduced unsuspectedly into the system.

Chemical analysis.—I. In the solid state.—Heated in a test-tube, it evolves an acetous odor, and fuses into a white mass; if the heat is continued, it again fuses and slowly chars, and is converted into a reddish-brown mixture of the oxides of lead. Heated on charcoal, before the blowpipe, it is converted into globules of metallic lead, with a surrounding incrustation of yellow oxide. A fragment dropped into a solution of the iodide of potassium instantly turns yellow; touched with sulphide of ammonium it immediately is blackened.

II. In the liquid state.—(1) A drop, evaporated on glass, yields opaque needles, which turn yellow when touched
with a drop of iodide of potassium solution, or solution of bichromate of potassium; or black, by sulphide of ammonium. (2) Dilute sulphuric acid causes a white precipitate, soluble in hot hydrochloric acid, and in large excess of potassa and soda. (3) Iodide of potassium gives a bright yellow precipitate, soluble in boiling water, which deposits it in brilliant yellow scales, on cooling. The iodide of lead is also soluble in potassa and strong hydrochloric acid. (4) Bichromate of potassium gives a bright yellow precipitate. (5) Sulphuretted hydrogen is the most delicate test, revealing, according to Taylor, a quarter of a grain of this salt in a gallon of water. The black sulphide is confirmed by heating it on charcoal, before the blowpipe; or by dissolving it in nitric acid, by the aid of heat, evaporating to dryness, dissolving in water, and applying the usual tests. (6) The galvanic test.—A drop or two of the solution, slightly acidified with acetic acid, is put into a platinum capsule, and a strip of zinc is made to touch the platinum through the liquid; crystals of metallic lead are deposited on the zinc; or a fragment of zinc may be placed in the lead solution in a watch glass, when very soon metallic lead will be deposited upon the zinc in an arborescent form. A salt of tin, under similar circumstances, would yield an arborescent deposit of tin. Hence, the metallic deposit must be further tested by dissolving it in nitric acid, and applying the usual tests.

Other tests of minor importance are potassa and ammonia, oxalate of ammonium, alkaline carbonates, and red and yellow prussiate of potassium.

Toxicological examination.—As acetate of lead is easily decomposed by many organic substances, such as albumen, casein, mucus, etc., the poison may exist both in the soluble and insoluble condition. As a trial test, a good plan is to
wet a piece of bibulous paper in the suspected solution and expose it to a jet of sulphuretted hydrogen gas, which will blacken it, if it contains any lead. If the presence of this metal be indicated, the mixture should be acidulated with pure nitric acid, and boiled for some time; when cold, it should be filtered, and the solids on the filter thoroughly washed, and reserved for future examination. Concentrate the filtrate by evaporation, and treat with sulphuretted hydrogen; allow the precipitated sulphide of lead to collect, pour off the supernatant water, boil in dilute nitric acid, add sufficient distilled water, filter, and apply the usual tests (p. 291).

The solids on the filter should be dried, and incinerated in a porcelain capsule; dissolve the ash by heat in dilute nitric acid, filter, and treat with sulphuretted hydrogen, and prove the precipitated sulphide.

If an alkaline sulphate has been given as an antidote, a white precipitate of sulphate of lead may be found in the stomach. This should be collected and boiled with pure potassa (proven itself to contain no lead), and the solution tested with sulphuretted hydrogen.

The solid organs (liver, spleen, etc.) may be examined either by boiling with nitric acid and water, evaporating to dryness, incinerating in a porcelain crucible and again dissolving by heat and dilute nitric acid, or by directly incinerating them in a porcelain crucible and dissolving out the lead with strong nitric acid, evaporating to dryness, diluting with water, and precipitating with sulphuretted hydrogen.

As lead remains in the system for a considerable time, in case of the detection of only a minute quantity in the body after death, inquiry should always be instituted in reference to the possibility of its accidental introduction into the sys-
tem through the occupation, mode of living, etc., of the individual.

The examination of the urine should never be neglected, seeing that lead is eliminated from the system chiefly through this secretion. From fifteen to twenty ounces of urine, acidulated with nitric acid, should be evaporated to dryness, and incinerated as above directed, and the ash treated as already described.

For the detection of lead in sweetmeats, etc., suspected of being colored with this metal, slightly moisten them with water and put them on a plate, placing in the centre a little capsule containing about a drachm of sulphide of ammonium, and cover the whole with a tumbler. If lead be present, the sweetmeats will, after a short time, be blackened by the sulphuretted hydrogen gas evolved (Woodman and Tidy).

Lead is quantitatively determined as a sulphide. Every 100 parts of pure, dried sulphide represent 93.31 parts of the oxide, or 158.37 parts of crystals of the acetate.
CHAPTER XXI.

POISONING BY COPPER.

ACCIDENTAL POISONING.—SALTS OF COPPER.—FATAL DOSE.—TREATMENT.—MORBID APPEARANCES.—CHEMICAL ANALYSIS.—TOXICOLOGICAL EXAMINATION.

Copper, like many of the other metals, is not poisonous in the metallic state; but if taken into the stomach, it is soon corroded, and forms highly poisonous compounds. Copper coins, sometimes swallowed by children, may thus produce dangerous results. The inhalation of copper alloy in fine powder, by artisans who work in what is termed gold printing, causes serious results, such as constriction and heat of throat, vomiting, loss of appetite, and severe itching of the parts of the body covered with hair, which, on examination, are found to be of a deep green color (Falconer, on Copper Poisoning).

Cases of accidental copper-poisoning can frequently be traced to want of cleanliness in cooking, or to keeping food in copper vessels, particularly such articles as contain a vegetable acid, as vinegar, an alkaline chloride, as common salt, or any kind of oil or fat. So long as copper utensils are kept perfectly clean and bright, no risk is incurred in using them; but if cleanliness is neglected, a deposit of the green carbonate collects, which is very poisonous, and will contaminate any food with which it may come in contact. There is no risk in boiling articles of food or preserves in clean copper vessels, although it is unsafe to keep these articles, cold, in the same vessels; in the latter case, the atmospheric air acts upon the metal at the point of contact.
of the contained substance. Dr. Falconer gives an instructive example of this. A servant left some sour-krout, for only a couple of hours, in a copper pan which had lost its tinning. Her mistress and daughter, who ate of the cabbage, died after twelve hours' sickness. Wildberg found the cabbage so strongly impregnated with copper that it was detected by metallic iron.

The inference from the above facts would be that it is extremely unsafe to employ copper vessels either for cooking, or still more so for preserving articles of food. Even tinned copper vessels are not safe, because the tinning consists of an alloy of tin and lead, and the latter metal might, in its turn, prove a source of danger.

The alloy termed Dutch metal, used for ornamenting cakes and confectionery as a substitute for gold leaf, may also prove a source of poisoning, as likewise the blue and green papers used as wrappers for bon-bons, although the chief source of danger from the latter arises from the arsenic contained in their composition.

The fine green color on many samples of pickles and preserved fruits is owing to the adulteration of a salt of copper, as verdigris or blue vitriol. This may be easily detected by placing the suspected article in ammonia, when, if copper be present, it will turn blue. A simpler method is to plunge a bright needle into the article; if copper be present, it will speedily receive a reddish coating of the metal.

All the cupreous salts are poisonous; those of most medico-legal importance are the sulphate (blue vitriol, blue stone), and the sub-acetate (verdigris). The arsenite and aceto-arsenite have already been described under the head of Arsenic. What is commonly called verdigris is the carbonate of copper, which is produced by the action of moist air on the metal, or on brass.
The salts of copper are rarely used for homicidal poisoning, as they can be so readily recognized, both by their color and taste. Occasionally, they have been taken suicidally, and more rarely by accident. The sulphate has been employed as an abortive.

**Symptoms.**—The sulphate may be taken as a type of all the salts. In large doses it produces speedy and powerful vomiting, which would probably expel the whole of the poison, and thus prevent a fatal result. There are pain in the stomach and bowels, great thirst, purging, headache, prostration, small, frequent pulse, with increased flow of saliva. The matters vomited are bluish or greenish; those from the bowels are greenish, and tinged with blood. Sometimes there are severe cramps and convulsions. There is also suppression of urine, and in fatal cases, paralysis and tetanus have preceded death. Jaundice is also an occasional result. Dr. Tidy speaks of it as “the specially diagnostic symptom of copper poisoning.” It is not met with in poisoning by either arsenic or mercury.

The symptoms of slow poisoning (which is generally the result of the accidental introduction of the metal in articles of food) are an acrid, styptic, coppery taste in the mouth, dry and parched tongue, coppery eructations, continual spitting, nausea and vomiting, colicky pains in the bowels, diarrhoea of bloody stools, with tenesmus, great thirst, heat of skin, small and tense pulse, scanty urine, headache, vertigo, faintness, cramps of the legs and convulsions; occasionally, jaundice and a blue line on the margin of the gums.

**Fatal dose.**—Not positively determined. Half an ounce of verdigris has proved fatal, and an ounce of the sulphate; but larger quantities have been swallowed without producing death. The usual emetic dose is five to fifteen grains. The usual fatal period is from four to twelve hours.
Treatment.—Free vomiting should be assisted by the use of warm diluent drinks. The best antidote is albumen in the form of white of eggs, as for corrosive sublimate. Milk is also very useful.

Morbid appearances.—These indicate the action of a powerful irritant to the mucous membrane of the alimentary canal, from the throat downward. The lining membrane of the stomach is inflamed, softened, and sometimes ulcerated. It usually exhibits a bluish or greenish appearance, due to the color of the salt taken. The same is true, also, of the intestines. As a somewhat similar appearance may result from the presence of bile, it is proper to distinguish between them by the addition of ammonia, which will impart a deep blue color if copper is present, but will cause no change in the green color, if due to bile. Perforations have been found in the small intestines; the rectum is occasionally ulcerated, and the lungs congested.

Chemical analysis.—All the salts of copper possess either a blue or green color. Only a few other metallic salts are thus colored, as some of the cobalt salts, blue, and some of those of nickel, chronium and uranium, green. When heated in the blowpipe flame, they impart to it a beautiful green color; and when heated on charcoal, with dry carbonate of soda, before the blowpipe, globules of metallic copper are obtained.

The sulphate, or blue vitriol, occurs in large, handsome crystals, efflorescent, soluble in water, having a nauseous, styptic, metallic taste. The verdigris of commerce occurs in masses of a light green, or bluish-green color. It is a mixture of the sub-acetate and other acetates of copper. It is soluble in water and in dilute acids.

(1) Ammonia gives to the solution a bluish-white amorphous precipitate—hydrated oxide of copper, which is re-
dissolved by an excess of the precipitant, yielding a beautiful, clear, dark purple-blue solution; this color is immediately removed by an acid. The salts of cobalt, nickel and chromium give somewhat similar results.

(2) *Ferrocyanide of potassium* gives a reddish-brown precipitate, insoluble in excess of the reagent, but soluble in ammonia. If the copper solution be very dilute, no precipitate may take place, but only the distinct brownish-red discoloration. We have found this test rather more delicate than that of ammonia.

(3) *Sulphuretted hydrogen*, or *sulphide of ammonium* yields a brownish-black precipitate of the sulphide. This should be corroborated by boiling in nitric acid, evaporating to dryness, dissolving in water, and applying the usual tests.

(4) *The iron test.*—This simple and satisfactory test consists in immersing a piece of bright iron or steel in a slightly acidulated solution of copper, when, sooner or later, according to the strength of the solution, it will acquire a red coating of metallic copper. If the solution be very dilute, it should be concentrated by heat, and a very small iron surface should be exposed; a fine sewing needle may be suspended in it for some hours. After it has received the copper coating, it may be removed and put into a porcelain capsule, with a little ammonia, which, in a short time, will assume a blue color.

(5) *The galvanic test* consists in placing the copper solution, slightly acidulated, in a platinum dish, and touching the latter, through the liquid, with a piece of zinc. The metallic copper will be deposited on the platinum, in the form of a reddish incrustation. The latter may be dissolved off the platinum by dilute nitric acid, evaporating to dryness, moistening it with water, and testing it as above directed.
The blowpipe, as already mentioned.

Toxicological examination.—A portion of the copper may be found in a soluble, and some in an insoluble state. The liquid part has usually a bluish or greenish color. This should be filtered, concentrated by heat, and a trial test, by means of a bright sewing needle, applied. Any reddish deposit on the latter should be proved, as above mentioned. Sometimes the needle may acquire a reddish coating simply from the oxide of iron: ammonia will serve to distinguish them. If a large amount of copper should be indicated, sulphuretted hydrogen should be passed through it until all the metal is precipitated as a sulphide. This is to be treated after the manner before described. If the amount of copper be extremely small, the galvanic test is the most suitable. The filtered liquid, acidified with sulphuric acid, is placed in a platinum capsule, and fragments of pure zinc are put into it; wherever there are points of contact between the two metals, there will be a reddish deposit on the platinum. This should be washed, and the copper dissolved off with a drop or two of dilute nitric acid. The nitric solution is to be evaporated to dryness, diluted with water, and tested as before described.

If neither of the above tests reveal the presence of copper, it cannot be present in the liquid matters; but the solids may possibly contain it. These should be boiled with dilute hydrochloric acid and water, filtered, concentrated by heat, and tested.

Traces of arsenic are sometimes found in the sulphate; when the latter has been taken as a vomit, traces of this substance have been discovered in the matters vomited, and in the stomach (Taylor).

In searching for the salts of copper in the stomach, this organ should be carefully examined for blue or green particles. After treating the stomach and its contents in the
usual manner, with the addition of water and hydrochloric acid, and filtering and concentrating by heat, the iron and galvanic tests may be used as trial tests. Sulphuretted hydrogen gas should then be passed through the liquid until all the sulphide of copper is precipitated. This should be boiled in dilute nitric acid, evaporated to dryness; if much organic matter is present, it should be moistened with strong nitric acid and heated until all the organic matter is destroyed. The dry residue is now dissolved in dilute nitric acid, and again evaporated to dryness, dissolved in water, and the usual tests applied.

In the organs.—These should be finely divided and thoroughly dried, and then incinerated in a porcelain crucible, and the resulting ash treated with pure hydrochloric acid by heat, and then evaporated to dryness; dissolve in water, and apply the usual tests. Copper remains longer than arsenic in the tissues and organs; as long as sixty days in the liver and lungs, according to M. L. Orfila.

In the urine.—Evaporate six to eight ounces to dryness; treat the residue with the nitric acid and chlorate of potassium, with the aid of heat, to complete incineration. Dissolve the resulting ash in hot dilute nitric acid, and evaporate to dryness. Dissolve the residue in warm water, and test as above.

Copper is not a normal constituent of the human body. It exists in minute proportions in certain vegetables, which, doubtless, obtain it from the soil.

The discovery of faint traces of copper in the body after death is no proof of copper-poisoning, since it may have been introduced into the system accidentally, either through the food, or otherwise.

Copper is determined quantitatively as the black oxide, every 100 parts of which are equivalent to 314.21 parts of crystallized sulphate.
POISONING BY ZINC.

CHAPTER XXII.

POISONING BY ZINC, BISMUTH, TIN, IRON AND CHROMIUM.

SECTION I.—POISONING BY ZINC.

Cases of zinc-poisoning are comparatively rare. In the metallic state, zinc is probably inert; but if swallowed, it would soon be acted upon in the stomach, and converted into a salt, where it might occasion serious results. The sulphate and chloride are the preparations most likely to produce poisonous effects.

The zinc of commerce (spelter) is apt to contain arsenic and other impurities.

Sulphate of zinc—White vitriol.—A white, crystalline, soluble salt. It has a metallic, astringent taste; effloresces on exposure to the air. It acts as a prompt, active emetic, without causing much depression of the system; hence, is indicated in cases of narcotic poisoning. It is used in small doses as a nerve tonic, and the system soon acquires a tolerance of the medicine. As much as two scruples three times a day, have been given for a period of three weeks, in a case of epilepsy.

Poisonous dose.—Half an ounce, to an ounce.

Symptoms.—A strong, metallic taste, attended with a burning sensation, and constriction of the throat, nausea, violent vomiting, intense pain of stomach and bowels, purging, small and frequent pulse, great anxiety, cold sweats, extreme prostration, dilated pupils, coma and death. Experiments on animals show it to be a powerful heart depressant.
Fatal period.—A case is reported by Wormley, of a woman who swallowed by mistake for Epsom salt, an ounce and a half of this salt, and death ensued in thirteen hours and a half.

Post-mortem appearances.—Decided evidences of inflammation in the mucous membrane of the alimentary canal, such as redness, softening, ecchymoses, and sometimes ulceration; a yellowish, pultaceous matter covering the inner surface of the stomach and bowels; congestion of the brain and membranes, also of the lungs, with bloody effusion into the pleura, and a distended, flabby heart.

Chloride of zinc.—In strong solution, this is known as "Sir William Burnett's Disinfecting Fluid." It is much used as a deodorizer. It contains about two hundred grains of the salt to the ounce of water. It is a powerful corrosive, and has frequently caused death when taken by mistake or suicidally.

The symptoms are, in general, the same as those produced by the sulphate, only much more intense in their character, and resembling somewhat those of the mineral acids. They come on immediately after swallowing; the matters vomited and purged are frequently tinged with blood, and mixed with shreds of mucous membrane; froth may issue from the mouth, and a white appearance of the inside of this cavity be noticed. There may also be loss of voice.

Fatal period.—Dr. Taylor records the most rapidly fatal case—four hours. On the other hand, the case may become chronic, lasting for years, and ending in stricture of the oesophagus and exhaustion.

Post-mortem lesions.—In addition to the usual inflammatory signs, those of a corrosive will be noticed, such as erosion or destruction of the coats of the stomach. Sometimes these are hard and leathery, thickened and corrugated.
The pylorus has been found constricted, and appearing as if cauterized. Constriction of the oesophagus has also been noticed, together with a softened condition of its mucous membrane. The brain and lungs are congested; the heart not affected, but usually distended. The blood dark and uncoagulated.

Treatment.—Assist the evacuation of the poison by the free use of mucilaginous drinks. The best antidote is albumen, as in corrosive sublimate and copper-poisoning. Opium should be given, to combat the irritation.

Chemical analysis.—In the solid state, the sulphate may be distinguished from Epsom salt and oxalic acid (which it much resembles in appearance), by exposing a small fragment mixed with carbonate of soda on a piece of charcoal, to the flame of the blowpipe; it quickly fuses, and the vapor forms an incrustation on the charcoal, which is first yellow, and becomes white on cooling. Heated with cobalt under the blowpipe, the fused bead of zinc has a green color.

In solution. (1) The alkalies precipitate the white hydrated oxide, which is soluble in excess of the precipitant. (2) The alkaline carbonates throw down the white carbonate, insoluble in excess of the precipitant, but soluble in excess of carbonate of ammonia. (3) Ferrocyanide of potassium gives a white precipitate. (4) Sulphuretted hydrogen throws down the white sulphide, soluble in hydrochloric acid. This should always be verified by dissolving it in hot hydrochloric acid, filtering, diluting and subjecting it to the other tests. (5) The galvanic test.—Place the solution on a platinum capsule, and touch it with a piece of magnesium, when the metal will be deposited on the platinum.

Toxicological examination.—In a case of suspected poisoning, it should always be remembered that sulphate of zinc may have been administered as an emetic; hence,
although discovered in the body after death, it may not have been the actual cause of death. If found, other poisons should also be sought for. The organic matters, along with a little acetic acid, should be gently heated for some time, in order to dissolve out the zinc that may have combined with albumen, fibrin, etc. After cooling, the solution should be filtered and concentrated, and then heated with sulphuretted hydrogen. The precipitated sulphide is collected on a filter, washed and dissolved in strong nitric acid. The nitrate is evaporated to dryness, dissolved in water, and subjected to the usual tests.

As the preparations of zinc usually contain iron, the presence of the latter metal will more or less modify the reactions of the former. The iron may be separated by an excess of ammonia, which will precipitate the oxide of iron, whilst it retains the zinc oxide in solution.

The tissues (liver, kidney, spleen, etc.) may be examined either by boiling with nitric acid, evaporating to dryness and adding small quantities of nitric acid, and heating until all the organic matter is destroyed; or else by incinerating the perfectly dried viscera in a porcelain crucible, and treating the resulting ash with nitric acid; evaporating to dryness, and dissolving the residue in water; acidulate with hydrochloric acid; again evaporate to dryness; dilute with water, and apply the usual tests.

Since chloride of zinc is often employed for embalming the dead, the discovery of zinc in the body after death might possibly be ascribed to this cause.

Quantitative estimate.—Zinc is usually determined as an oxide. The boiling solution is precipitated with carbonate of soda. The precipitate is collected on a filter, washed and dried, and then ignited. The protoxide is then weighed. Every 100 grains represent 354.13 grains of pure crystallized sulphate, or 167.77 grains of anhydrous chloride of zinc.
SECTION II.

POISONING BY BISMUTH.

Subnitrate of Bismuth—Magistery of Bismuth—Pearl White.—This substance is considerably employed, both medicinally and as a cosmetic. As a medicine it is given in doses of five to thirty grains. Several fatal cases have been reported as resulting from large doses; the symptoms being those of a violent irritant poison. Many authorities ascribe these results to the adulteration of the bismuth with arsenic. Dr. Taylor states that this adulteration is very common, and that he detected arsenic in comparatively large quantities in three out of five specimens obtained from respectable London druggists. The arsenic may readily be discovered by dissolving the subnitrate in pure hydrochloric acid, slightly diluted, and employing a Marsh's apparatus. This same adulteration has occasionally been found in the subnitrate of our own shops; and it should be looked to by physicians as being the probable cause of the irritation which occasionally follows the use of this medicine.

This impurity may essentially modify a medico-legal opinion as to the presence of arsenic in a body, where bismuth had been previously administered medicinally. A case of this nature (State of Virginia vs. Mrs. E. E. Lloyd, 1872) occurred, in which the defence contended that the trace of arsenic alleged to have been discovered in the liver of the deceased was to be ascribed to the subnitrate of bismuth which had been taken before death. This bismuth was found to be contaminated with arsenic. The prisoner was acquitted.

By the process recommended by the present U. S. Pharmacopoeia, the bismuth is entirely freed from arsenic.

Subnitrate of bismuth is in the form of a white powder,
insoluble in water, but soluble in nitric acid. The solution, thrown into water, yields a copious white precipitate, which blackens by sulphide of ammonium, and is not soluble in tartaric acid.

A delicate test for a bismuthic salt is a piece of paper wetted with a solution of sulphocyanide of potassium and dried; a beautiful yellow spot appearing at the point of contact. It is stated that the urine will reveal the presence of bismuth, a few hours after taking the subnitrate, by means of this test.

**SECTION III.**

**POISONING BY TIN, IRON AND CHROMIUM.**

*Salts of Tin.*—The only preparations of tin requiring notice are the chlorides. The effects of these salts upon the system are those of the metallic irritants; but the instances of poisoning by them are rare.

The protochloride is precipitated by sulphuretted hydrogen of a dark chocolate color. Corrosive sublimate throws down the gray metallic mercury. Chloride of gold gives a fine purple precipitate—the purple of Cassius. A fragment of zinc precipitates metallic tin, in an arborescent form.

*Bichloride of tin* is precipitated yellow by sulphuretted hydrogen. This sulphide is distinguished from the yellow sulphide of arsenic by being insoluble in ammonia, and from sulphide of cadmium by being insoluble in hydrochloric acid. Corrosive sublimate and chloride of gold yield no precipitate with it.

The preparations of *Silver, Gold* and *Platinum* (with its allied metals) are all highly irritant and corrosive; but they so rarely are the occasion of poisoning in the human subject, that they need no further discussion here.
Preparations of Iron.—The sulphate (green vitriol) is highly irritant in large doses, having proved fatal in several instances. Its action resembles that of sulphate of copper, though less violent.

The chloride, in the form of tincture (muriated tincture of iron), is much used in medicine. In large doses it acts as a violent, corrosive poison. Christison records the case of a man who swallowed, by mistake, an ounce and a half of the liquid, and who died in about five weeks. It is occasionally used as an abortive.

Preparations of Chromium.—The salts of chrome most used in the arts are the chromate and bichromate of potassium, and the chromate of lead. The two former are violent irritants in large doses; sometimes acting, also, as corrosives to the lining membrane of the alimentary canal.

Bichromate of potassium may be distinguished—(1) by its deep orange-red color; (2) by solution of acetate of lead, which precipitates the yellow bichromate of lead; (3) by nitrate of silver, which throws down a deep red precipitate; (4) sulphuretted hydrogen gives a dingy-green sulphide; (5) sulphuric acid added to a solution, along with alcohol, instantly throws down the green oxide, with effervescence; yielding, at the same time, the odor of aldehyde.
CHAPTER XXIII.

VEGETABLE AND ANIMAL IRRITANTS.

SECTION I.—POISONING BY OXALIC ACID.

SOURCES OF THE ACID IN NATURE.—SYMPTOMS.—FATAL DOSE.—TREATMENT.—MORBID APPEARANCES.—CHEMICAL ANALYSIS.—TOXICOLOGICAL EXAMINATION.—BINOXALATE OF POTASSIUM.

Oxalic Acid, in combination with lime and potash, exists naturally in certain plants, as the rhubarb, wood-sorrel, dock, etc. It is extensively used in the arts, under the name of acid of sugar. It is rarely employed for homicidal poisoning, since it would be easily detected by its excessively sour taste; but it is sometimes resorted to for suicidal purposes, and it has been frequently the cause of accidental death, from its being mistaken for sulphate of magnesia (Epsom salt), which it much resembles in appearance.

Symptoms.—These depend very much on the size of the dose and the degree of concentration. In the quantity of half an ounce to an ounce, it acts as a prompt, violent, corrosive poison. In smaller doses and more diluted, its irritant effects may be much lessened, or altogether lost; but its remote, specific operation on the heart and nerve centres is very observable in the acute pain in the back, extending down the limbs, attended with tingling and numbness, and with tetanic spasms, together with occasional narcotism. On the heart, it acts as a decided depressant.

When swallowed in a very large dose and dissolved in a small quantity of water, the effects are immediate. An intensely sour taste is followed by a burning sensation in the gullet, extending down to the stomach; violent pain in
the abdomen, increased by pressure; constriction of the
throat; vomiting of a greenish-brown or black matter,
sometimes mixed with blood. If the patient lives, purging
of a similar character sets in. The remaining symptoms
are those of collapse, such as extreme debility, a cold,
clammy skin, feeble, rapid pulse and hurried respiration.
There are also soreness of the mouth, swelling of the
tongue, intense thirst, restlessness and distressing cough;
also, frequently, cramps and numbness of the legs and arms,
loss of voice, acute pain in the back and head, delirium and
convulsions—symptoms that indicate the action of the poi-
son on the nervous system. As in the case of other violent
poisons, the above-mentioned symptoms are liable to many
exceptions and anomalies; thus, vomiting and pain may
both be absent.

There is every reason to believe that the poison is ab-
sorbed into the blood, though hitherto experiments have
failed to detect it in that fluid. Christison mentions a case
where leeches, that had been applied to the epigastrium of a
patient who had been poisoned by this acid, fell off dead,
showing evidently that it had gotten into the circulation.
The urine contains crystals of oxalate of lime in abundance,
also albumen and tube-casts; and according to some
writers, deposits of similar crystals within the renal tubules.

Fatal dose.—Half an ounce to an ounce is regarded as a
fatal dose for an adult. Dr. Taylor quotes a case where
one drachm of the solid crystals proved fatal to a boy six-
teen years old, in eight hours. There are, however, cases
on record where much larger doses have been swallowed
without causing death.

Fatal period.—In a large, concentrated dose, oxalic acid
is one of the most energetic poisons known. Christison
calls it "the most rapid and unerring of all the common
poisons.” Dr. Ogilvie reports the most rapidly fatal case known, where death occurred in three minutes after swallowing it (Lancet, Aug. 1845). In other cases, death has followed in ten minutes; the majority of cases prove fatal within one hour. Again, instances have occurred where the patient has survived for many hours, and even days. Dr. Beck alludes to the case of a woman who died from the secondary effects of the poison, after several months of suffering.

**Treatment.**—This should be prompt, in order to be of any service. The best antidotes are chalk and magnesia, mixed up with milk. The alkalies and their carbonates are inadmissible, on account of their forming soluble poisonous compounds with the acid. The scrapings of a whitewashed wall should be resorted to in the absence of chalk and magnesia. Lime-water and oil are useful. Opium is indicated to relieve the severity of the symptoms.

**Post-mortem appearances.**—The lining membrane of the mouth, throat and gullet will usually be found white, shriveled and easy of removal; it may be covered over with the brown matters discharged from the stomach. The mucous membrane of the oesophagus may be entirely separated, displaying a surface of a brown color, and raised in longitudinal folds. The stomach, which is frequently contracted, contains an intensely acid, brown, gelatinous fluid; the mucous membrane, if death has been rapid, may appear soft and pale, often without marks of decided inflammation; but if death has been delayed, it is usually black in some places, and in others intensely congested and in rugae, with portions peeling off, revealing a gangrenous condition of the subjacent tissue. Such cases strongly resemble those of sulphuric acid poisoning. Perforation of the stomach is rare.
The intestines are usually highly congested, if death has been at all delayed. The lungs generally, and the brain occasionally, have been found congested. The heart is sometimes quite empty, and at others, distended with dark blood. The blood throughout the body is fluid. A few cases have occurred where all morbid appearances have been absent.

**Chemical analysis.**—(1) As a solid. When pure, it occurs in colorless four-sided crystals, of an intensely sour taste (by which it is immediately distinguished from sulphate of magnesium), being soluble in water, especially hot; soluble, also, in alcohol, but insoluble in ether, and nearly so in chloroform. It is completely volatilized by heat, leaving no residue; this is not the case with the sulphates of magnesium and zinc, for which it has been mistaken.

(2) As a liquid.—It reddens litmus paper; a drop evaporated to dryness leaves long, slender prisms. (a) *Nitrate of silver* gives a copious white precipitate of oxalate of silver, distinguished from the chloride and cyanide by being soluble in cold nitric acid. If the precipitate is thoroughly dried and heated on platinum foil, it is completely dissipated in a white vapor, in a succession of puffs or slight detonations. (b) *Sulphate of calcium* (or any lime salt) gives a white precipitate of oxalate of calcium, which is distinguished from the carbonate and phosphate by being insoluble in acetic acid, but being soluble in nitric, and rather less so in hydrochloric acid. Sulphate of calcium will also precipitate solutions of barium, strontium and lead; but the sulphates of these bases are insoluble in nitric acid. (c) Chloride of barium, nitrate of strontium and acetate of lead all precipitate the white crystalline oxalates of these bases, which are soluble in nitric and hydrochloric acids; but these tests are of inferior value to the former ones. (d) Sulphate
of copper yields a faint bluish-white oxalate of copper, which is almost insoluble in nitric acid.

Toxicological examination.—If the contents of the stomach are highly acid, the poison may probably be separated by dialysis, or they may be digested with distilled water, at a moderate heat, for some hours, and then filtered, the filtrate concentrated, and tested with sulphate of copper. If this test affords evidence of the presence of oxalic acid, the liquid may be evaporated to crystallization, and the crystals thus obtained re-dissolved in pure alcohol, and the solution again crystallized.

But it is usually recommended to treat the first filtrate with an excess of solution of acetate of lead; wash the precipitated oxalate of lead on the filter; then diffuse it thoroughly in pure water and pass through it a current of washed sulphuretted hydrogen, until all the lead and organic matter is thrown down. Heat a little while, to expel the excess of sulphuretted hydrogen, then filter, and crystallize the filtrate by evaporation. Purify the crystals, if necessary, by re-solution, and apply the usual tests.

But it may happen that all the oxalic acid in the stomach has been neutralized by the antidotes that were administered—lime or magnesia, in which case the contents of the stomach would not be acid. Here the inspected solids should be collected, and thoroughly washed with warm water, and the liquid decanted off; if this liquid is acid, it should be reserved for examination; if not acid, it may be thrown away. The solids should then be diffused in pure water, and boiled for some time with pure carbonate of potassium, which decomposes the oxalates, forming soluble oxalate of potassium and insoluble carbonate of lime and magnesia. These are separated by filtration, and the solution concentrated by gentle heat, until the crystals are formed.
In a case of alleged oxalic acid poisoning, it might be objected that the presence of a minute quantity of oxalic acid found in the stomach after death might be due to rhubarb or sorrel that had been eaten by the deceased. The answer to this is obvious: if there is an entire absence of all the characteristic symptoms of this active poison, the discovery of a small quantity of the acid is certainly no evidence of poisoning; but on the other hand, if the peculiar symptoms of this poison, and morbid lesions are present, then the obtaining of only a small amount of the acid should not negative the charge of poisoning.

In a case of suspected poisoning by oxalic acid, the urine should always be examined for an increase of the octahedral crystals of oxalate of lime. This fluid should be collected in a conical glass, and the sediment which collects after some time should be examined with the microscope. But all the lime-oxalates may not be thus precipitated; a portion may be held in solution by the acid phosphate of sodium which exists in the urine.

It should, however, be remembered that these same crystals may be found in the urine of persons who have partaken of food containing oxalic acid, such as rhubarb and sorrel.

To detect free oxalic acid or a soluble oxalate in the urine, add a little acetic acid and concentrate to about one-fourth its bulk, then add acetate of lead in excess, filter; decompose the oxalate of lead with sulphuretted hydrogen, and treat the filtered solution as above directed.

Stains of this acid on cloth, parchment, paper, etc., may be discovered by boiling them in water, and applying the usual tests. The color of these stains on black cloth is orange, and brownish-red. This acid is sometimes employed to remove writing-ink, in cases of forgery; but usually there
are left on the paper traces of iron, existing in the ink, which can readily be recognized by applying a solution of the ferrocyanide of potassium, which will turn it blue.

Oxalic acid is usually estimated quantitatively as oxalate of lead; every 100 parts of the dried pure oxalate are equivalent to 42.5 of the crystallized acid.

Oxalic acid is not a normal constituent of the human body; although supposed by Liebig and others to be one of the ultimate results of the oxidation of uric acid in the economy, yet no one has been able to demonstrate its existence in the blood. In disease it is frequently found in the tissue in the form of oxalate of lime, in which form also it constitutes the variety of calculus named the mulberry calculus.

Binoxalate of Potassium—(Salt of Sorrel—Essential Salt of Lemons).—This salt is much used in the arts for bleaching, etc. It is almost as active a poison as oxalic acid. The symptoms, dose and lesions are very similar to those of the acid. It is distinguished from the latter, (1) by heating a fragment on platinum foil; an ash is left (carbonate of potassium), while the acid is entirely dissipated; (2) it crystallizes in feathery forms; (3) it is distinguished from cream of tartar by lime water, which precipitates both, but the tartrate of calcium is immediately re-dissolved by tartaric acid, while the oxalate is insoluble. This salt is a natural ingredient in the sorrel.

Tartaric and acetic acids act as powerful irritant poisons in the concentrated state and in large doses. They have both produced fatal results when taken in doses of an ounce. Their proper antidotes are the alkaline carbonates, and chalk or magnesia.
Carbolic Acid—(Phenic Acid—Phenol)—is one of the products of the distillation of coal tar. When pure it is in the form of white acicular crystals, which, when exposed for some time to the light, acquire a reddish tinge. They have a strong affinity for water, and liquefy when exposed to the air. It is not, however, very soluble in water; much more so in alcohol, ether and glycerine. It has a burning taste, and a peculiar, strong, creasote odor. It is sometimes named coal-tar creasote. It is powerfully antiseptic, quickly destroying the germs of the lower forms of organic life. Applied in its undiluted state to the skin, it acts as a corrosive and anaesthetic, corrugating and hardening it. Taken internally, undiluted, its effects are those of an energetic, corrosive and neurotic poison.

Symptoms.—Intense burning pain in the mouth, throat and stomach; the pupils are contracted; the conjunctiva insensible to the touch; marked odor of the acid exhaled from the breath; the skin cold and clammy; the temperature rapidly falls; the pulse becomes weaker and weaker, but fluctuates in its beats; respiration is labored and ultimately stertorous; vomiting of a frothy mucus sometimes occurs; the mouth is white, and hardened, from the local effects of the acid. Coma usually precedes death, which may also sometimes be accompanied by convulsions. Death may occur within an hour from swallowing the poison; in one case, within ten minutes. The urine, as shown by Stevenson, is often of an olive-green color.

Many of the above symptoms have been produced by the external application of the acid, especially to denuded surfaces.
Fatal dose.—Dangerous symptoms have occurred from doses of six or seven drops. The deaths recorded have resulted from doses of one to two ounces; but much less quantity would certainly prove fatal.

Treatment.—Assist the evacuation of the poison by emetics of mustard water, and sulphate of zinc, albumen, oil and demulcients. Sulphate of soda is regarded by some as a true antidote. Dr. Husemann considers the saccharate of lime an antidote. Oil is the best outward application to the skin. Stimulants must be freely given, to combat the collapse.

Post-mortem lesions.—The mouth and oesophagus are usually white, soft and corroded, but sometimes hardened and corrugated. The brain is generally normal, but occasionally congested, and the fluid in the ventricles exhal- ing the strong odor of the acid. The lungs are usually gorged with blood. The left ventricle of the heart is generally contracted, while the right is flaccid. The blood is uniformly dark colored and fluid. The odor of car- bolic acid is detected in the stomach, sometimes in the intestines, and even in the other viscera. The mucous lin- ing of the stomach has been found white, hardened and wrinkled; but, again, highly congested and corroded. The bladder is usually empty, any urine passed being of a dark, or olive-green color.

Chemical analysis.—The odor is probably the best test. It has a slight acid reaction, and forms salts with bases. It imparts a transient, greasy stain to paper. It coagulates albumen. It gives a deep violet color to perchloride of iron, and a bluish tint to ammonia and hypochlorite of lime; if this be acidulated, it turns red. Bromine water causes a whitish-yellow flocculent precipitate; if this be treated with sodium-amalgam, carbolic acid is set free (Landolt). Heated
POISONING BY CROTON OIL.

with cyanide of potassium, it gives a red tint. A splinter of wood, moistened with the acid, and afterward dipped into nitric or hydrochloric acid, turns of a greenish-blue tint when dry (Woodman and Tidy).

Toxicological examination.—Generally, the characteristic odor of the acid will be perceived in the body after death. The organic matters should be distilled along with dilute sulphuric acid.

Carbolic acid can generally be detected in the urine, both by the odor and by chemical reagents. The urine may either be distilled without sulphuric acid (as it is said this acid may develop carbolic acid from some of the normal constituents of the urine); or by agitating it with an excess of ether, and subsequently removing the ethereal layer by means of a pipette, and evaporating in a shallow dish; a minute oily residue is left, having the character of carbolic acid.

SECTION III.
POISONING BY CROTON OIL, ELATERIUM, CASTOR-OIL BEANS, COLCHICUM, AND SAVIN.

Croton oil is a fixed oil, extracted by pressure from the seeds of Croton tiglium. It is used in medicine as a powerful purgative, in doses of one or two drops. In over-doses, it acts as a violent irritant to the gastro-intestinal mucous membrane, causes excessive vomiting and purging, followed by collapse, as in cholera. Its poisonous properties seem to be dependent on a peculiar fatty acid named crotonic, which exists in the oil in variable quantities. When deprived of this acid the oil is harmless.

Croton oil is of a light yellow color, has an unpleasant odor, and a hot, acrid, burning taste. It is very soluble in ether. Nitric acid, with the aid of heat, imparts to it a dark brown color.
Death has resulted in one case from taking, by mistake, an embrocation containing thirty minims of the oil. In another case, two and a half drachms proved fatal in four hours; whilst in a third instance, half an ounce of croton oil was taken by mistake, and after exciting violent vomiting and purging, with symptoms of collapse, the patient recovered, after fourteen days.

**Analysis.**—Separate the oil from the contents of the stomach by means of ether, and evaporate the ethereal solution spontaneously, and test the resulting oil with nitric acid and heat.

**Elaterium.**—This substance is procured from the juice of *Ecbalium officinarum*, or squirting or wild cucumber. It is used in medicine as a powerful drastic purgative. Its effects are very similar to those of croton oil. One grain of good elaterium has produced very violent effects. It owes its activity to a neutral resinous principle, *elaterin*. Nitric acid has no action upon it. Sulphuric acid turns it of a reddish-brown color.

**Castor Oil Beans** are derived from the *Ricinus communis*, and yield by pressure the castor oil of commerce. The seeds contain an irritant, poisonous principle, which causes them to act violently when swallowed. They have occasionally proved fatal. In one case *three* seeds destroyed life in an adult male in forty-six hours, and *twenty* seeds proved fatal to a young lady in five days, after violent symptoms, strongly resembling those of malignant cholera.

**Colchicum.**—The *Colchicum autumnale*, or meadow saffron, contains a powerful alkaloidal principle, *colchicina*, which strongly resembles veratria in many of its properties. It
abounds chiefly in the bulb of the plant, but is also found in the seeds.

The effects of a large dose upon the system are those of a violent irritant, such as burning pain in the throat and stomach, great thirst, vomiting and soreness, purging, cramps, cold, collapsed skin, feeble pulse, dilated pupils, suppression of urine, and rapid exhaustion. Sometimes there are delirium and convulsions.

The strength of the preparations of colchicum varies greatly, depending upon the time of gathering the plant, and also its place of growth.

Death has resulted in seven hours, and again has been delayed for several days. Generally, it occurs within twenty-four hours. Less than half an ounce of the wine of the root, forty-eight grains of the dried bulb, and a tablespoonful of the seeds have severally proved fatal. A frightful accident occurred in Montreal, Canada, in 1873, to a company of eight or nine persons. They had freely partaken of what they supposed to be ordinary wine, but which was really wine of colchicum. In the course of a few hours they became alarmingly ill, with nausea, vomiting, excruciating pains, purging, cramps and prostration. Five of the cases terminated fatally within thirty-six hours.

Morbid appearances.—These consist of inflammation of the stomach and bowels. In some instances, no morbid appearances exist. In one case, the pia mater was much congested, when there was an absence of inflammation from the stomach. The lungs are usually deeply congested.

Colchicina occurs in fine, white crystals. It is soluble in water, has a feeble, alkaline reaction, and a bitter, acrid taste. Its best test is nitric acid, which gives it a violet color, changing to blue and brown. It differs from veratria in its negative action with sulphuric acid. It may be recovered
from organic mixtures by a modification of Staas' process (vid. post.) Less than half a grain of colchicina has proved fatal. There is no known antidote to it.

Savin.—The tops of the Juniperus sabina contain a volatile yellow oil (oil of savin), which may be procured by distillation. Both powder and oil are employed in medicine, and both possess powerfully irritant properties. They are seldom or never used as poisons, but they frequently produce fatal results when used as abortives. Although they do not possess specific ebolic properties, uterine contractions may follow their powerful irritant effects upon the pelvic viscera; but death is a more frequent result, without the expulsion of the foetus.

In cases of poisoning by the powder, this may often be discovered in the stomach and bowels, by microscopic inspection. The oil may be separated by distillation from the contents of the stomach, and then agitating the distillate with ether, in which it is soluble, and allowing the solution to evaporate. It is recognized by its peculiar terebinthinate odor.

SECTION IV.

POISONING BY THE HELLEBORES.

VERATRIA—YELLOW JESSAMINE—POISONOUS MUSHROOMS.

Black Hellebore—(Helleborus niger)—formerly named Melampodium, is sometimes employed in medicine. It is a powerful irritant to the stomach and bowels, and has proved fatal in overdoses, occasioning violent vomiting, purging, abdominal pain, cold sweats, collapse and convulsions.

Green Hellebore (Veratrum viride—American Hellebore—Indian Poke).—This species possesses very active properties, and has occasioned fatal results. The tincture is used in medicine as a powerful cardiac depressant. Two active
alkaloidal principles exist in this drug—*veratroidia* and *viridia*—which, though resembling veratria in some points, are distinct from it.

*White Hellebore (Veratrum album).*—This is the most poisonous of all the hellebores. The powder produces violent sneezing. Taken internally, it causes a sense of burning heat and constriction of the throat, great anxiety, nausea, vomiting and purging, pain of the abdomen, trembling of the limbs, great prostration, cold sweats, very feeble pulse, giddiness, convulsions, insensibility and death. Death has occurred in three and six hours after taking it.

The active principle is *Veratria*, which also exists in the *Veratrum sabadilla*.

*Veratria.*—As found in the shops, this alkaloid is in the form of a white powder. It may be crystallized with difficulty. It has an acrid, bitter taste, followed by a sense of dryness in the throat. It is a violent irritant to the nostrils, causing excessive sneezing. It is insoluble in water, soluble in alcohol, ether, chloroform, benzole and amylic alcohol. Heated on porcelain, it melts and blackens, evolving a pungent vapor.

*Effects.*—Dr. Taylor (*On Poisons*, p. 510) mentions the case of a lady on whom the ½ of a grain occasioned most alarming symptoms, such as insensibility, cold sweats, failing pulse and collapse. It acts as a local irritant to the stomach and bowels, and at the same time as a general depressant.

*Chemical analysis.*—The characteristic test is *sulphuric acid*. A drop applied to the pure alkaloid imparts a yellow color to it, followed by a reddish tint, which gradually passes to a deep crimson. This change is brought about immediately by heat. Even a very dilute acid causes this reaction, by evaporating to dryness. (See Sulphuric Acid, 15
According to Wormley, the \( \frac{1}{10000} \) of a grain of veratria may be thus detected.

Other substances give a red color to sulphuric acid—such as solanine, narcorine, salicine, piperine, etc., but these are immediately colored by cold sulphuric acid, whereas veratria requires the lapse of some time before this change is effected.

Trapp's test consists in warming the colorless solution of veratria in concentrated hydrochloric acid, when a persistent dark red color results.

In organic mixtures, veratria may be separated by a modification of Stass' process, and the ultimate chloroform extract tested by sulphuric acid. Dr. Wormley states that by this test he was enabled to recognize the presence of veratria in an ounce of blood of a cat which had been killed, in less than one minute, by two grains of veratria.

Yellow Jessamine (Gelsemium sempervirens).—The root of this plant is considerably employed in medicine, especially in the Southern States of our country, in the treatment of neuralgia, and analogous complaints. It has frequently produced fatal results, the symptoms somewhat resembling those produced by veratria. Wormley found it to contain a powerful alkaloidal principle—gelsemia, one-eighth of a grain of which killed a rabbit in an hour and a half, together with another organic principle—gelseminic acid. The above authority succeeded in detecting both of these principles in the stomach of a woman who was poisoned by three teaspoonfuls of the extract, several months after death (Am. Jour. Med. Sci., April, 1870).

Poisonous Mushrooms.—As is well known, certain varieties of fungi are edible, while others possess noxious, and
even fatal properties. It is not always possible to distinguish between these, inasmuch as climate, season, and idiosyncracy may occasion the difference. The poisonous principle of certain fungi appears to be volatile, since boiling renders them innocuous.

**Symptoms.**—The effects of poisonous mushrooms on man are those of the narcotic irritants, causing violent vomiting, purging, abdominal pain, thirst, anxiety, cold sweats, together with giddiness, dimness of vision, trembling, dilated pupils, delirium, illusions, stupor, coma, convulsions and death.

It is stated that the very same fungi have acted on some members of a family as vomitants only, and on others as narcotics.

Generally, the symptoms show themselves within one hour—especially the narcotic symptoms. Orfila (*Toxicol. II*, p. 433) relates the following interesting case of poisoning of a family of six persons by the *Amanita citrina*. The wife, servant and one of the children had vomiting, followed by deep stupor, but they recovered. The husband had violent cholera; he recovered also. The two other children became profoundly lethargic and comatose; emetics had no effect, and death ensued. The individuals who recovered were not completely well until three weeks after the fatal repast.

**Morbid appearances.**—These are imperfectly described; they indicate a great tendency to rapid putrefaction, lividity of the body, fluidity of the blood, absence of cadaveric rigidity, numerous ecchymoses in the serous membranes and parenchymatous organs, signs of violent and even gangrenous inflammation of the stomach, and congestion of the vessels of the brain, with decomposition of the tissues.
The chief medico-legal interest connected with this subject is the fact that the symptoms occasioned by eating poisonous fungi might easily be attributed to poisoning—homicidal, or otherwise. A microscopic examination of the contents of the stomach and bowels will usually reveal the botanical character of the fragments of the fungi, if the poisoning has been due to them. (See Orfila and Christison on the subject of *Poisonous Fungi*.)
CHAPTER XXIV.

ANIMAL IRRITANTS.

POISONING BY CANTHARIDES.—POISONOUS ANIMAL FOOD.—SAUSAGE POISON.—TRICHINOSIS.—CHEESE POISON.—POISONOUS FISH.—PUTRESCENT FOOD.—POISONED FLESH.

Cantharides.—The Cantharis vesicatoria, or Spanish fly, is much used in medicine, both externally as a vesicant, and also internally. In large doses it acts as a powerful local irritant to the alimentary canal, and also to the urino-genital organs. It is often used as an abortive, and has not unfrequently produced fatal effects when employed for this purpose. It owes its active properties to a crystalline principle named cantharidin, which exists in the proportion of about one grain to half an ounce of the powder.

Symptoms.—A burning sensation in the mouth and throat, with constriction and difficulty of swallowing; violent pain in the abdomen, increased by pressure; nausea and vomiting of a bloody mucus and shreds of membrane, along with great thirst, and dryness of the fauces. Soon the characteristic impression on the genito-urinary organs displays itself, in a dull, heavy pain in the loins, an urgent and incessant desire to urinate, which is attended with great pain and the voiding of merely a few drops of bloody urine, accompanied with tenesmus. Priapism frequently occurs in males, and swelling and heat of the labia in women, together with abortion, at times, in pregnant females. Purging generally supervenes, the stools being bloody and mucous, and accompanied with tenesmus. Sometimes there is profuse salivation, and in fatal cases faintness, giddiness, and convulsions.
If the substance has been taken in the form of powder, the characteristic shining green particles may generally be recognized in the discharges from the stomach and bowels. If the tincture has been taken, the above symptoms come on more rapidly.

All the above symptoms have been produced by the external application of cantharides.

_Fatal dose._—Twenty-four grains of the powder, and an ounce of the tincture, have caused death.

_Treatment._—Speedy evacuation by emetics and cathartics (castor oil); opium and stimulants.

_Post-mortem appearances._—Intense inflammation of the mucous membrane of the alimentary canal, from the mouth downward; also of the ureters, kidneys and bladder. Congestion of the brain has been observed. The peculiar shining green particles can generally be distinguished in the stomach and bowels. But if the tincture has been swallowed it will be necessary to procure the extraction of _cantharidin_ from the organic matters.

_Chemical analysis._—The suspected materials should be dried, and digested in successive portions of ether, until exhausted; this will dissolve out the cantharidin. The ethereal solution is to be evaporated until nearly dry, and the residue should be spread on oiled silk, and a portion applied to the lips, or on the thin portion of the skin of the arm, when the resulting vesication would denote the presence of cantharides.

_Poisonous animal food._—It occasionally happens that various kinds of animal food, such as sausages, cheese, fish, mussels, etc., produce poisonous symptoms, either owing to some idiosyncrasy on the part of those who have partaken of them, or depending upon some noxious agent
connected with the food itself, either introduced from without, or spontaneously generated within.

Such cases are often attended with symptoms of a violent character, which naturally suggest poisoning, and they then become the subjects of medico-legal examination (*vid. post.*, *Plomaines*).

**Sausage poison.**—It was formerly supposed that, under certain peculiar circumstances, not well understood, sausages when cured and dried were capable of developing a poisonous principle, in the shape of a peculiar fatty acid, named by Bushner *batrolinic acid*. But physicians and physiologists of the present day are disposed to attribute the cause of the so-called sausage-poison to the presence of an ento- zoön, named *Trichina spiralis*, which especially infests the muscles of the hog, and which, when the pork is eaten uncooked, and unless it has been exposed for some time to a temperature above 212° F., very soon penetrates the muscular coat of the intestines, and thence spreads rapidly through the muscles generally, and which increase in numbers prodigiously. The sudden liberation of a multitude of these parasites from their cysts, in the intestines and muscles, produces the irritation of the bowels, and the subsequent loss of muscular power that are so characteristic of *trichinosis*.

It may readily happen that the symptoms thus occasioned might be attributed to slow poisoning by one of the mineral irritants. A careful microscopic examination of the suspected food, or of a fragment of a muscle of either a living or dead subject, will reveal the true source of the disorder.

The symptoms of *Cheese poisoning* are very similar to those of ordinary irritant poisoning. The cause of the
development of poisonous properties in cheese is by no means well understood. By some it is ascribed to the production of an acrid, oily matter derived from an improper fermentation of the milk, analogous to caseic and sebacic acid. Instances of cheese poisoning are more common in Germany than in this country.

Poisonous fish.—In certain individuals, probably through idiosyncrasy, many kinds of fish act poisonously, i.e., they excite severe gastro-intestinal symptoms, resembling cholera morbus. It is quite possible that some peculiar organic change in the food itself may have taken place. Mussels, which are quite extensively used in Europe as food, occasionally produce most violent and alarming symptoms, which cannot be ascribed to any rational cause.

These symptoms are not of a uniform character. Sometimes they are those of a simple irritant, such as nausea, vomiting, purging, pain in the abdomen, cramps, small and frequent pulse. The fatal cases disclose, on post-mortem examination, evident signs of inflammation. In other instances, the gastro-enteric disturbance has been slight, whilst the nervous symptoms are well marked, such as delirium, insensibility and coma, with dyspnœa and convulsions. Again, the most conspicuous symptoms have been a peculiar eruption resembling urticaria, along with severe asthma. The symptoms usually do not appear until the lapse of twenty-four hours, but there are cases where they come on very much earlier. In fatal cases, the autopsy usually reveals nothing that will satisfactorily account for the result.

No rational, adequate cause of this singular poisonous action of the shell fish has yet been discovered. We must, therefore, ascribe it to some peculiar animal principle generated under unknown conditions.
Putrescent or decayed meat, if eaten by man, will produce not only gastro-enteric symptoms similar to those described above, but also those of a typhoid character, or septicæmia, or true blood poisoning. The game that has been kept long enough to delight the taste of the epicure has produced a severe cholera in persons not accustomed to its use.

Putrid animal matter injected into the blood vessels proves quickly fatal. Dissecting wounds thus may produce alarming symptoms, which may terminate in death.

Poisoned meat.—The flesh of an animal or bird which has become poisoned by arsenic, strychnine, or some other deleterious substance, may become the cause of poisoning to man. Thus, the common pheasant of this country (Tetrao umbellus) which has fed upon the leaves and buds of the kalmia (laurel), has proved poisonous to persons who have eaten the birds. It is well known that the milk of cows and goats that have fed upon the Datura stramonium may prove poisonous to those partaking of it. In one case of alleged poisoning by belladonna, the defence was that the family had eaten a rabbit pie, and that the animal had fed upon the leaves of the belladonna plant, so that, without being affected injuriously itself, it had conveyed the poison to those who had partaken of it.
CHAPTER XXV.

CLASS II.—NEUROTIC POISONS.

This second division of Poisons embraces those whose effects are displayed chiefly on the great nervous centres—the brain and spinal marrow. Their symptoms are drowsiness, headache, giddiness, stupor, delirium, convulsions and paralysis. They produce little or no irritation, or inflammation on the mucous membrane of the alimentary canal. Their morbid effects are not very distinctly marked. These consist of more or less fullness of the cerebral vessels, rarely effusion of serum; more rarely still, effusion of blood in the brain. Hence, it is quite impossible to diagnose a case of neurotic poisoning by these lesions exclusively.

ORDER I.—CEREBRAL NEUROTICS.—(1) NARCOTICS.

SECTION I.—POISONING BY OPIUM.

NATURE OF OPIUM.—POISONOUS SYMPTOMS.—FATAL PERIOD.—FATAL DOSE.—TREATMENT.—MORPHIA.—PROPERTIES.—TESTS.—MECONIC ACID.—TESTS.—TOXICOLOGICAL EXAMINATION IN OPIUM POISONING.

Opium and its preparations constitute a very large proportion of poisoning cases, both in this country and Great Britain.

Opium is the dried juice of the capsules of the poppy (Papaver somniferum). It has a very complex composition, containing numerous active principles, the chief of which are morphia, meconic acid, narcotina, codeia, narceine, thebaine and papaverine. In a medico-legal view, the only important ones are morphia and meconic acid, since, in an analysis for
the detection of opium in a case of suspected poisoning, the investigation is narrowed down to the discovery and identification of these two substances.

It should be remembered that different specimens of opium differ considerably in their contained amount of morphia, this varying from two to thirteen per cent. The average may be taken at eight per cent. in Smyrna opium. The tincture (laudanum) of the shops is far from being of a uniform strength, owing to this variation in the amount of the active principle in the opium, and also to fraudulent dilution. Average laudanum should contain about five grains of opium to the fluid drachm, which is equivalent to one grain to twenty-five drops. The black drop (Acetum Optii) is about double the strength of laudanum. Wine of opium (Sydenham's laudanum) is about the strength of laudanum.

**Symptoms.**—These vary according to the size of the dose. A large, but not fatal dose occasions, at first, general excitement of the system, as evinced by increased fullness and frequency of the pulse, flushed face, brilliancy of the eyes, and increased activity of the brain. This is soon followed by calm repose, which in turn gives place to profound sleep. In proportion as the amount of opium is increased, the first period of excitement is shortened, the more characteristic soporific effects manifesting themselves sooner. In such a case there will be giddiness and drowsiness, rapidly passing into profound sleep or stupor, from which it will be difficult to arouse the patient; this stupor gradually ends in coma. The pupils are contracted. At first the pulse is full and slow; subsequently it becomes weak. The respiration is generally slow and stertorous; the skin warm, and the face flushed. As the case advances, the countenance becomes pale, the lips livid, the skin cold and clammy, the respiration very slow—we have noticed it reduced down to five or six
in a minute; the muscles are relaxed; convulsions sometimes occur just before death, but these are more common in children than in adults. Sometimes there is vomiting, which is to be regarded as a hopeful sign; and occasionally also there may be purging. At times, the skin is bathed in a profuse perspiration.

Certain variations in the above symptoms should be noticed. The pupils are usually strongly contracted; towards the termination of the case they may sometimes be dilated. Occasionally one pupil may be contracted, and the other dilated. The contracted state of the pupils is usually regarded as a diagnostic sign of opium-poisoning, but Dr. Wilks has shown that this same condition of the eyes occurs in apoplexy of the pons varolii, and that two cases of this latter disease were mistaken for opium-poisoning (Med. Times and Gaz., 1863, I, p. 214). The same contraction of the pupil occurs also in uræmic poisoning, in the course of Bright's disease.

First appearance of symptoms.—This will depend on the size of the dose, the form of administration, and the condition of the stomach at the time. As a rule, the symptoms usually commence within an hour after swallowing the poison. But, if taken in the liquid form and in full quantity, they may manifest themselves in a few minutes. We have often seen full narcosis produced in five to ten minutes by the subcutaneous injection of a quarter of a grain of morphia. On the other hand, cases are reported where the symptoms were delayed, even after swallowing very large doses, for many hours. Sometimes a partial remission of the symptoms occurs, and the patient gives hopes of recovery; but they return again only to terminate in death. There seems good reason to believe that alcohol tends to postpone the development of the usual symptoms of opium.
Fatal period.—The average duration of a fatal case is from seven to twelve hours. Cases are reported where the symptoms appeared in thirty-five minutes, and death in three-quarters of an hour; whilst, on the other hand, death has been, in some instances, delayed for twenty-four to forty-eight hours.

Fatal dose.—Four or five grains may be regarded as the minimum fatal dose for an adult. Children are particularly susceptible to the action of this drug; in very young infants, fatal effects have resulted from taking two or three drops of laudanum. An infant may be narcotized by the milk of a nurse who has taken opium.

On the other hand, recoveries constantly take place from very large doses—even up to several ounces. It is notorious that the human system soon acquires a remarkable tolerance for this narcotic by habit. De Quincey thus brought himself to the daily use of nine ounces of laudanum, which is equivalent to about three hundred and sixty grains of solid opium.

Occasional instances of idiosyncrasy occur, in which the susceptibility to the narcotic influence of opium is greatly augmented; and also, on the other hand, where there seems to be a natural tolerance for the drug. As regards the opium habit, we think there can be no doubt of its ultimate deleterious effects upon the human system.

The external application of opium, especially to an abraded surface, may prove highly dangerous, and even fatal, especially in the case of infants. Christison relates an instance where a laudanum poultice, applied over the abdomen of an infant to relieve pain, produced fatal narcotism in some hours; and where, at the autopsy, a strong odor of opium was exhaled from the body, showing how completely the poison had been absorbed.
Toxicology.

Post-mortem appearances.—These are neither certain nor characteristic. There is usually some fullness of the vessels of the brain; occasionally, extravasation of serum into the ventricles; very rarely of blood. Sometimes there is congestion of the lungs and other vascular organs. The blood is apt to be fluid. The stomach and bowels may be perfectly natural in appearance. The odor of opium may be observed in opening the body. It is hence impossible to diagnosticate a case of opium-poisoning from the post-mortem appearances exclusively.

Treatment.—Remove the poison from the stomach as speedily as possible, by the stomach-pump, or by a prompt emetic, as sulphate of zinc, or mustard water. The next indication is to overcome the increasing lethargy, by rousing the patient, dashing cold water over the face and chest, and making him walk about between two attendants. He should swallow some strong coffee. Atropia should now be carefully administered hypodermically, every half hour, watching its effects upon the pupils. Electro-magnetism should be employed, if the other remedies fail.

As regards the antagonism of morphia and atropia, our own experiments confirm those of Dr. John Harley, on the lower animals (dogs and cats), viz., that in these animals there is no real antagonism between these drugs. In the human subject, however, we think the accumulated testimony of physicians as regards their mutual antidotal operation cannot be disregarded; our own experience in their employment also confirms this opinion (vid. ante, p. 185).

Morphia.—When pure, morphia is in the form of colorless rhombic crystals, very bitter to the taste, very slightly soluble in water, soluble in alcohol, especially when hot, almost insoluble in chloroform and pure ether, very soluble
in acetic ether. It is slightly alkaline, forming salts with acids. Its solutions, in common with the other alkaloids, are precipitated by tannic acid. Its salts are soluble in water and diluted alcohol, but insoluble in chloroform, ether, amyl alcohol and pure acetic ether.

The symptoms produced by morphia resemble those of opium, except that they ordinarily manifest themselves rather earlier, and possibly tend to produce convulsions rather more frequently than opium. Occasionally, these convulsions were of a tetanic character, suggesting the presence of strychnia.

**Fatal dose.**—One grain has, on several occasions, occasioned death. We have known a case where three-quarters of a grain administered hypodermically proved fatal to a gentleman within twenty-four hours. On the other hand (as in the case of opium), enormous doses have been swallowed with impunity. Dr. Norris reports a case (**Am. Jour. Med. Sci**, 1862, p. 395), where a druggist took, with suicidal intent, seventy-five grains of sulphate of morphia. No marked symptoms appeared for an hour and a half. He then became unconscious, but under active treatment, including extract of belladonna, he entirely recovered on the second day after the occurrence.

The external application of morphia to an abraded surface has been attended with fatal effects.

There are no characteristic post-mortem lesions produced by morphia. The general appearances are similar to those caused by opium.

**Chemical analysis.**—There is no chemical test for opium, as such; it is identified by its sensible properties, and by its physiological action on animals. The only mode of identifying it chemically is by detecting the presence of its morphia and meconic acid.
Detection of morphia.—(1) In the solid state.—Strong nitric acid dissolves it with effervescence, evolving red fumes, and gives an orange-red solution, slowly fading to yellow. Nitric acid also produces a deep-red color with brucia, which, on the addition of protochloride of tin, changes to a bright purple; whereas no change is produced in the case of morphia. (2) Strong sulphuric acid dissolves it without change of color; if now a crystal of bichromate of potassium be added, it acquires a green color. (3) Neutral perchloride or persulphate of iron imparts a deep blue color to it, changing to green if added in excess. For this experiment no free acid must be present. (4) Iodic acid added to a fragment of morphia, along with freshly-made starch, produces the characteristic blue color from the liberated iodine.

2. In the liquid state.—(1) Nitric acid, in excess, gives an orange-red color, which becomes light yellow on boiling. (2) Neutral perchloride or persulphate of iron acts as on solid morphia. (3) Iodic acid in bisulphide of carbon added to a solution of morphia causes a precipitate of a pink or red color, consisting of iodine dissolved in the bisulphide. (Taylor). (4) Sulpho-molybdic acid, made by dissolving five or six grains of molybdate of sodium or ammonium in two drachms of strong sulphuric acid. A drop of this reagent in contact with a fragment of morphia dissolves it, producing a beautiful violet color, changing soon to green and ultimately to a sapphire-blue. (5) If morphine be mixed with a little cane-sugar, and treated with concentrated sulphuric acid, a wine-red color is produced. (Weppen).

The other tests mentioned in the books, such as terechlo-ride of gold, iodine in iodide of potassium, bromine in hydrobromic acid, etc., are of less importance.

Meconic Acid.—As this acid is peculiar to opium, its de-
tection affords positive proof of the presence of that substance. In its pure state, it occurs in the form of colorless crystals, tolerably soluble in water, more so in alcohol.

Tests.—(1) Perchloride or persulphate of iron imparts to either the solid or liquid form a blood-red color, which is not removed by a solution of corrosive sublimate. The only fallacy likely to occur in a medico-legal case is from the presence of some sulpho-cyanide in the material examined, as the saliva, which yields a similar color with the iron salt; but the red color in the latter case is instantly discharged by corrosive sublimate.

Strong acetic acid, or its salts, likewise give a red color with the ferric salts; and this color, moreover, is not removed by corrosive sublimate. But if the acetate be previously boiled with dilute sulphuric acid, it gives no color with the iron salt. (2) Acetate of lead yields a yellowish-white precipitate of meconate of lead. (3) Chloride of barium yields a white crystalline deposit of a peculiar form. (4) Nitrate of silver gives a yellowish-white precipitate, which becomes red on adding a persalt of iron.

Toxicological examination.—Sometimes on opening the stomach the strong odor of opium is readily detected, and also in the matters vomited. The discovery of this poison in the stomach is often unsuccessful, owing, probably, to its decomposition, and absorption in the body. This is especially true in the case of infants, in whom a very few drops suffice to destroy life. The highest authorities unite in declaring that the analyst will fail to discover this poison in the stomach after death, in the majority of the cases. It is much more likely to be found in the vomit.

The stomach should be cut up in small fragments, adding water with a little alcohol, and acidulating with pure acetic acid, and the whole exposed to a gentle heat, for about one
hour. After cooling, it should be strained through muslin, and the solid residue washed with strong alcohol and pressed, and the washings added to the first liquid. The liquid should then be evaporated over a water-bath to a small volume, and when cooled filtered through paper. To the clear filtrate, acetate of lead is to be added in excess, to precipitate the meconate of lead. The morphia remains in the solution as an acetate. These are to be separated from one another by filtration, and the solid matter washed with water.

(a) The solid portion (meconate of lead) is to be diffused through water, and treated with sulphuretted hydrogen gas, which precipitates the black sulphide of lead, and leaves the filtrated meconic acid in solution; this latter is concentrated by a gentle heat. A trial test on a small portion of this liquid, by the iron-salt, may be made; if a deep red color is imparted, meconic acid may be suspected; to the remainder of the liquid, the other tests may now be applied. If present in sufficient quantity, meconic acid will crystallize out, on evaporation of the liquid. If the quantity, however, be minute, the liquid should be carefully concentrated to a small volume, and the characteristic tests employed.

(b) The liquid portion, containing the morphia in the form of acetate, together with the excess of lead acetate, is to be treated with sulphuretted hydrogen in order to remove the lead; then filtered, and the filtrate concentrated by gentle heat to dryness. The residue is then treated with a few drops of warm distilled water, and a portion of it examined for morphia by the nitric acid and iodic acid tests. The remaining liquid should then be made alkaline by pure potassium carbonate (diluting, if necessary, with water); it is next to be shaken up with an excess of absolute ether, which will take up the impurities, leaving the morphia
unaffected. The ethereal solution is removed by means of a pipette, and reserved for future examination. The remaining alkaline solution is now to be thoroughly shaken with two or three times its volume of, either a mixture of two parts of absolute ether and one of alcohol, or of two or three volumes of hot *amylic alcohol*, or of a similar bulk of *acetic ether*. By either of these processes, the morphia is taken up by the solvent, which floats upon the top of the mixture, and which may be removed by a pipette and allowed to evaporate spontaneously on watch glasses. Ordinarily, the morphia thus recovered is amorphous, and may require resolution in hot alcohol, and to be crystallized therefrom by evaporation.

In cases of poisoning by morphia alone (or one of its salts) the above process may be employed, omitting the acetate of lead, inasmuch as no meconic acid is present.

*Detection in the tissues and blood.*—There is generally a failure to detect this poison in the organs and tissues, or in the blood. Yet, on the other hand, cases are reported where it has been discovered in the body several months after death. We can account for this discrepancy only on the supposition either that opium (morphia) undergoes some decomposition in the living organism, which interferes with its detection by chemical analysis, or else that life has been prolonged sufficiently long to allow of its diffusion throughout the body so thoroughly, as to render it impossible to detect it in any one particular organ; this would be especially the case if the dose had been comparatively small, as where an infant had died from a few drops of laudanum. There is some doubt about the detection of these principles in the *urine*, inasmuch as the results alleged to have been produced by certain reagents, and supposed to indicate the presence of morphia, or meconic acid,
have since been proved to be due to substances existing normally in the urine.

The toxicologist should be cautioned against a too hasty conclusion as to the presence of opium, or its alkaloid, in a medico-legal case, based upon the color alone. Orfila tells us (*Toxicol.*, II, p. 232) that Mm. Ruspini and Cogrossi found that a decoction of a calf’s intestines, although no morphia was present, acted upon iodic acid and starch like that alkaloid. In another case, morphia was pronounced to be present in the urine, by reason of the action of the extract of this secretion on iodic acid; the effect was found to be really due to uric acid and urate of ammonia.

Inasmuch as the symptoms of opium-poisoning strongly resemble those of apoplexy, it could readily happen that a case of the latter disease, attended with suspicious surroundings, might be mistaken for the former, and the contents of the stomach might even probably reveal a red color, when treated with nitric acid. But, if no morphia (nor meconic acid) was actually separated, we think the examiner would not be authorized to pronounce upon the presence of this poison simply from the one single reaction above mentioned. Prof. Taylor cites an instructive illustration of this hasty conclusion, in which a certain distinguished (?) chemist made oath of the discovery of “distinct traces of morphia” in the stomach; whereas, in reality, no morphia had been taken at all (as was proved by an analysis of the medicine taken by the deceased); but the death was due entirely to natural causes.
The poisonous effects of Alcohol are of a twofold character—acute and chronic. The former are witnessed in those cases where a large quantity of spirits is taken at a single draught, as in a silly bravado, or for a wager, and also accidentally by young children. The latter are illustrated in the common dram drinkers, and by a train of symptoms with which we are, unfortunately, but too familiar. The former only will be discussed here.

**Symptoms of acute poisoning:**—These come on usually in a few minutes after the ingestion of the poison, if the amount is large. They are, first, giddiness, confusion of ideas, unsteadiness of gait, incoherent talking, followed by stupor and coma. The features have a vacant, ghastly expression, or they may be suffused and bloated; the lips are livid; the pupils usually dilated and fixed; the conjunctivæ are red; an alcoholic exhalation from the breath is perceived; convulsive movements of the limbs; respiration, at first stertorous, becomes more and more difficult; a bloody froth may appear on the lips; involuntary evacuations occur, and death may ensue in half an hour, or even earlier, after the fatal drink (Tardieu). In other instances, the person may apparently recover from the first effects, and then suddenly become insensible, and die in convulsions. If free vomiting occurs, followed by a prolonged sleep, recovery is apt to take place. The sensibility of the pupil to light may also be regarded as a favorable symptom.

The rapidity with which the symptoms show themselves will depend upon the previous habit of the individual, and
the strength and quantity of the spirit taken. The very large quantities seem to destroy life by shock.

Acute alcoholism may be mistaken for opium-poisoning and concussion of the brain. Usually, the odor of the breath is sufficient to reveal the case; also, the dilated pupil; but this condition of the eye is not invariably present. In concussion, there are often marks of injury in the head; the face is pale and cold; there is also an absence of the alcoholic odor.

*Post-mortem appearances.*—There is generally a remarkable absence of putrefaction in the body. The stomach exhibits marks of intense congestion, in the deep-red color of its lining membrane, either diffused or in patches. More or less congestion of the brain and its membranes, with serous effusion under the arachnoid, and in the ventricles. Sometimes, there is a true apoplectic extravasation of blood. The lungs are almost invariably congested. Usually, a strong alcoholic odor is perceived from the different tissues of the body; but the organs for which the poisonous fluid displays the greatest affinity are the brain and liver.

Alcohol is very rapidly absorbed into, and eliminated from the system; so that, if the person has survived several hours, all traces of it may have been removed from the body.

*Treatment.*—Immediate evacuation by means of the stomach pump, or by an active emetic; affusion of cold water over the head; a free supply of fresh air; if there be asphyxia galvanism may be tried, also ammonia and the liberal use of coffee as a drink.

*Chemical analysis.*—If the case has not been too protracted, the alcohol may be recovered from the stomach and its contents by distillation in a capacious retort, on a water-bath, with a good condensing apparatus. If the materials
are acid, they should first be neutralized by carbonate of potassium or sodium. The distillate should be mixed with chloride of calcium, and re-distilled. The second distillate is to be shaken with an excess of carbonate of potassium (which absorbs the water) and set aside. The stratum of alcohol which rises to the top may then be separated by means of a pipette, and submitted to the following tests: (1) Its taste is hot and pungent; its odor is characteristic. It burns with a pale blue flame, leaving no carbonaceous residue, but yielding carbonic acid and water; if burnt under the mouth of a test-tube moistened with lime or lead water, the carbonic acid will produce a white film upon the sides of the tube. (3) It dissolves camphor. (4) On adding a solution of bichromate of potassium and sulphuric acid, the peculiar odor of aldehyde is developed, along with the green color of chrome oxide. In performing this latter test, Prof. Taylor recommends conducting the vapor from the retort in which the distillation is going on into a glass tube containing a few fibres of asbestos moistened with a mixture of a strong solution of the bichromate and sulphuric acid, when the merest trace of alcohol vapor will be sufficient to impart the green coloration.

Both ether and pyroxylic spirit (wood spirit) will produce this last effect, and likewise yield most of the results of alcohol. Ether may be distinguished by its odor, and by the yellow color of its flame; also by its smoky deposit on porcelain. Pyroxylic spirit may be recognized by its peculiar odor, and by its smoky flame on burning.

In the tissues.—The proof of the absorption of alcohol is afforded in its detection in the blood, urine, and different tissues of the body. If there is a failure to discover it in the stomach, it should be looked for in the brain and liver. Buchheim has devised an exceedingly delicate process for detecting it in small quantities in the blood and tissues,
based upon the conversion of the vapor of alcohol into aldehyde and acetic acid when passed over platinum-black. As much as possible of the material, neutralized first by carbonate of potassium, should be distilled from a capacious retort, on a water-bath. The neck of the retort should be slightly inclined, and be wide enough to hold a platinum tray about two inches long and half an inch wide, containing the platinum-black. Hanging over each end of the tray is placed a slip of moistened litmus paper, and touching the platinum-black. The tray is now pushed toward the body of the retort. As soon as there is any escape of alcoholic vapor by the distillation, it will be manifested by the reddening of the litmus paper at the farthest end of the tray, in consequence of the production of acetic acid, while the paper nearest the body of the retort will remain blue. If no reddening of the paper occurs, no alcohol can be present; if the reddening rapidly occurs, the tray should be removed, and the vapor should be condensed in the usual way.

As both ether and wood spirit produce a similar effect on platinum-black, this process offers no advantage over the chromic process above described, except when putrefaction has taken place, in which case the sulphuretted hydrogen evolved might reduce the chromic acid, but it would not affect the platinum-black (Taylor On Poisons, p. 643). But we may remark it is hardly supposable that the analyst would undertake to separate alcohol from a putrefied body.

A new test for alcohol is given by Lieben (Phar. Jour. 1869). A few grains of iodine and a few drops of solution of caustic soda are introduced into a test-tube, along with the suspected fluid. It is then heated without boiling, when iodoform is precipitated. It is stated that one part of alcohol in two thousand of the mixture can thus be detected; also, that it may thus be discovered in the urine after drinking, by first distilling it.
This subdivision of *Cerebral Neurotics* comprises those substances that display their power chiefly by producing insensibility to pain, and unconsciousness. The Anaesthetics here noticed are Ether and Chloroform. Under this head also it will be convenient to speak of *Chloral Hydrate*, although its action differs somewhat from that of the others.

**SECTION I.**

**POISONING BY ETHER, CHLOROFORM, AND CHLORAL HYDRATE.**

**Ether.**—Generally known as *Sulphuric ether*, because procured by the distillation of alcohol and sulphuric acid. It is a limpid, colorless liquid, of a peculiar odor, and hot, pungent taste; highly volatile and inflammable; sp. gr. 0.735; boils at 95° F.; burns with a bright yellow flame, depositing carbon on a cold porcelain surface. Sparingly soluble in water; very soluble in alcohol.

**Symptoms.**—In large doses, it produces much the same effects as alcohol. There is, usually, a short period of delirious excitement, followed by coma and other symptoms of narcotism, similar to those caused by alcohol.

**Post-mortem appearances.**—On account of its less solubility in water, ether is a more powerful local irritant than alcohol. The mucous lining of the stomach and duodenum of a dog poisoned with ether were found to be violently inflamed; the lungs deeply congested, and the heart full of black blood (Orfila, *Toxicol.*, II, p. 531).
The inhalation of ether, as is well known, produces rapid anaesthesia, on account of its prompt and speedy action on the brain. Its immediate effect, when inhaled, is the production of a transient excitation; this is soon followed, if the dose be sufficient, by stupor and insensibility. This last condition may be prolonged for a considerable time by continuing the inhalation. Occasionally, the excitement is of a violent character, along with a stubborn resistance to the anaesthetic influence; and again, there may be nausea and vomiting. These exceptional symptoms must be ascribed to the constitutional peculiarities of the patients.

Although, in a few instances, the inhalation of ether has been attended with fatal consequences, we are of the opinion that it is a much safer anaesthetic than chloroform.

Chemical analysis.—Ether is recognized by its odor and taste, by its mode of combustion and volatility, and by its action on sulphuric acid and bichromate of potassium—the same as in the case of alcohol.

From organic mixtures, as, e.g., the contents of the stomach, it is to be separated by the same process as that described for alcohol (vid. p. 343).

Chloroform.—A colorless, limpid liquid, very volatile, giving off a dense vapor; sp. gr. 1.497; boiling point, 142° F. It has an agreeable characteristic odor, and a smart, pungent taste. It is nearly insoluble in water, in which it sinks in globules. It is not inflammable, like ether and alcohol. It is a powerful solvent of many organic substances, the alkaloids among others. At a red heat, its vapor is decomposed into chlorine and hydrochloric acid.

Symptoms.—A large dose produces local irritation to the stomach, with, at first, a general stimulation of the whole system, soon followed by decided narcotism, as shown by
insensibility, stupor, convulsions, dilated pupils, flushed face, full and oppressed pulse, and frothing at the mouth. Cases are reported where the pupils were contracted.

Dr. Taylor reports a case where a boy, aged four years, died in about three hours, after swallowing one drachm of chloroform. It has often caused death in quantities of half an ounce and upward.

When taken by inhalation, its impression is more speedy than that by ether. There is, moreover, an absence of the previous excitement attendant on the latter, the patient almost immediately passing into insensibility. It appears to act as a depressant from the first, and if not properly diluted with atmospheric air, it may rapidly produce death. In one case, the fatal result took place in one minute after breathing only thirty drops in the state of vapor; and, in another instance, only fifteen drops proved fatal in a very short time. It is, undoubtedly, a far more dangerous anaesthetic agent than ether, and instances of its fatal effects are being constantly reported in the medical journals. The immediate cause of death from chloroform vapor appears to be, in the majority of cases, syncope, or the cessation of the heart's action; in others, asphyxia.

Post-mortem appearances.—In death from liquid chloroform, the characteristic odor may usually be recognized, together with slow putrefaction of the body, and persistent rigor mortis. There is also much irritation of the stomach, sometimes accompanied with softening, and in one case with ulceration.

In death from inhalation, there is very often no lesion discoverable. At times, there will be found considerable congestion of the lungs and bronchial tubes, and likewise of the vessels of the brain, together with a dark and fluid condition of the blood.
Treatment.—In poisoning by liquid chloroform, the stomach should be immediately evacuated by the stomach-pump, or by a prompt emetic, and stimulants afterward administered. If inhalation has caused the danger, the chloroform should be immediately withdrawn, and fresh air freely admitted; cold affusion should be applied to the face and chest; the tongue should at once be drawn out of the mouth, to facilitate respiration; artificial respiration, and the direct galvanic current should also be practiced.

Chemical analysis.—The odor will usually be present in organic mixtures, such as the contents of the stomach. These should be distilled on a water-bath, and the distillate re-distilled along with chloride of calcium, and the product subjected to the proper tests, as odor, taste, solubility, etc.

Toxicological examination.—The contents of the stomach, or the organs properly divided along with distilled water, should be put into a large flask, the neck of which is fitted with a cork perforated to contain a hard glass tube, bent at right angles, and from twelve to fifteen inches long. The flask is gradually heated on a water-bath, and at the same time the middle of the horizontal tube is heated red-hot by a Bunsen flame. At a red heat, chloroform is decomposed into chlorine and hydrochloric acid. A slip of moistened litmus paper placed at the mouth of the tube is first reddened, and then bleached; starch paper wetted with iodide of potassium is rendered blue; and if the end of the tube be made to dip into a solution of nitrate of silver, the white chloride of silver will be precipitated. The absence of any free hydrochloric acid in the original material should be first insured, by the addition of carbonate of sodium.

It is important to remember that if hydrate of chloral had been taken by the patient just previous to death, and the alkali be added to the mixture for examination, the chloral...
POISONING BY HYDRATE OF CHLORAL.

would be decomposed into chloroform, and produce all the above reactions.

There are certain important medico-legal questions connected with the administration of chloroform as an anaesthetic, with which the legal physician should be familiar, such as whether persons asleep may be chloroformed without their being awakened, and thus robbed or otherwise maltreated. It has been ascertained by direct experiment that this effect can be produced if the sleep is profound, but not if it is slight or partial.

Hydrate of chloral.—A solid, crystalline body, resulting from the action of chlorine on alcohol. It has a peculiar, disagreeable, pungent taste and smell; is tolerably soluble in water; not inflammable. Potassa added to its boiling aqueous solution instantly converts it into chloroform and formic acid. It decomposes a salt of copper, like grape sugar.

Symptoms.—Chloral has been introduced into medical practice within comparatively few years, as a hypnotic. Its indiscriminate use has led to many fatal results. Care should always be exercised not to repeat the dose too frequently, as there appears to be a tendency to accumulation, and a sudden and dangerous action of the drug. In moderate doses, it acts on the brain as a hypnotic; in large doses, it produces a powerful depressant action on the ganglia at the base of the brain, and the spinal cord, causing feeble action of the heart and lungs.

A full dose usually occasions deep sleep, followed, if the quantity be very large, by fatal coma. The pulse is usually very slow and feeble; the face pale; respiration slow, the heart being ultimately arrested in diastole.

Much discrepancy of opinion exists as regards the fatal
dose of chloral hydrate. Numerous instances are reported where ordinary doses of thirty grains have occasioned alarming, and even fatal effects; while, on the other hand, enormous quantities—over an ounce—have been swallowed with comparative impunity. As a rule, thirty grains may be considered as a safe maximum dose, and not to be repeated oftener than every six or eight hours.

The opinion of Liebreich, of Berlin, is that chloral, while circulating in the blood, undergoes decomposition into chloroform and formic acid, through the agency of the alkalies of the blood.

Chemical analysis.—The principle involved is the conversion of the chloral into chloroform, through the agency of an alkali, as explained above. The solid matters, properly divided, should be diluted with distilled water and rendered alkaline by caustic potassa, and heated in a flask, and the experiment conducted after the manner described under the head of Chloroform.
CHAPTER XXVII.
ORDER II.—SPINAL NEUROTICS OR TETANICS.—POISONING BY NUX VOMICA.—STRYCHNIA.

Poisonous dose of nux vomica.—Effects of strychnia.—Fatal dose.—Treatment.—Post-mortem lesions.—Diagnosis.—Chemical analysis.—Interferences.—Physiological test.—Toxicological examination.

Nux Vomica is by far the most important poison included under this order of Neurotics. It is the seeds of the *Strychnos nux vomica*, a tree growing in India. Several seeds are enclosed in a yellow fruit. These seeds are circular disks, an inch or less in diameter, concavo-convex, of a light brown color, covered over with short, whitish, silky hairs, extremely tough and difficult to pulverize; excessively bitter to the taste. They contain two powerfully poisonous alkaloids—strychnia and brucia, in combination with *strychnic* or *igasuric* acid. The amount of contained strychnia is estimated at one-half to one per cent. of the seed.

The smallest fatal dose of nux vomica is thirty grains (about the weight of one seed), and three grains of the alcoholic extract. The symptoms, treatment, etc., are precisely similar to those described under the head of *Strychnia*.

Strychnia.—Exists in several species of *Strychnos* besides the *S. nux vomica*; it is the poisonous principle of the *S. Ignatia*, or *St. Ignatius bean*; it is also found in *False Augustura* bark.

Strychnia is a very frequent cause of poisoning, whether accidental, homicidal, or suicidal. The celebrated *Palmer*
case, which occurred in England, in 1856, brought it prominently before toxicologists.

**Symptoms.**—These vary somewhat in the time of their appearances, according to the form of their administration. The first effect is a feeling of restlessness and general uneasiness, with a sense of impending suffocation, and want of air. Very soon, twitching of the muscles and jerking of the limbs and head come on. These are followed suddenly by a violent tetanic convulsion, which pervades the whole body; the legs are stretched out stiffly, and widely separated; the feet arched, and usually turned in; the arms are flexed, and tightly drawn across the chest; the head is bent back rigidly, and the whole body flexed backwards so as to rest upon the head and heels (opisthotonos). As the muscles of the chest and abdomen are spasmodically contracted, the respiratory movements become arrested; the face is livid and congested, especially around the lips; the eyes prominent and staring; pupils widely dilated; the muscles about the mouth contracted so as to produce the expression denominated *risus sardonicus*; the pulse is very rapid and feeble. Sometimes, there is foaming at the mouth, and the froth may even be tinged with blood. The intellect remains perfectly clear, while the patient is experiencing the most intense suffering, gasping for breath, and seeking in vain for relief in asking to be turned over, or moved, or held. The jaws are not always fixed during a paroxysm; the patient may hence be able to speak; and as there is often great thirst, he may ask for water, but the effort to swallow is apt to intensify the spasm, as in hydrophobia, and cause him to bite upon the vessel.

The paroxysm may last from half a minute to several minutes, when a complete relaxation occurs; the patient now feels exhausted, and is bathed in perspiration; the
pupils may now become contracted. In a short time—varying from a few minutes to half an hour—the fit returns. It is usually preceded by an apprehension of the impending danger, the special senses being exceedingly acute. The spasm may be brought on by the slightest cause, as the opening of a door, a sudden noise, a current of air, or an attempt to move. In some instances, the violence of the spasm is so great as to jerk the patient out of bed. Should the case prove fatal, the paroxysms increase in frequency and violence, until at last death ensues, either from asphyxia, the patient dying in a paroxysm, or from pure exhaustion, during an interval.

Although the intelligence continues unimpaired during the progress of the disease, it may happen that it becomes clouded just before the fatal termination, in consequence of the asphyxia causing a deficiency of aeration of the blood, and the consequent accumulation of carbonic acid. As a rule, when the paroxysms are once established, they progress either to a fatal termination, or toward a cure, within two hours of the seizure, though there may be some exceptions to this rule.

The time of the first manifestation of the symptoms varies from a few minutes, to some hours; the average is fifteen minutes, to half an hour. Dr. G. H. Barker reports (Am. Jour. Med. Sci., October, 1864) the case of a young, healthy woman, who took six grains of strychnia, in whom violent symptoms were manifested in three minutes, and death took place, in a convolution, in half an hour. In Dr. Warner's case, who took, it is supposed, less than half a grain, the symptoms appeared in five minutes, and death occurred in about eighteen minutes. In a case mentioned in the Ann. d' Hygiène, 1861, I, p. 133, convulsions came on in five minutes. On the other hand, this interval may be pro-
tracted for several hours. Dr. Anderson reports (Am. Jour. Med. Sci., April, 1848) the case of a gentleman who took, by mistake, three and a half grains of strychnia, and experienced no particular symptoms for two hours and a half, when he suddenly fell backward; but, on being raised, he was able to walk home, and finally recovered. Undoubtedly, the form in which the poison is administered has much to do with the rapid development of the symptoms. This is shown in a case cited by Dr. Taylor (Prin. and Prac. of Med. Jurisp., 1873, p. 405), of a boy aged twelve years, who swallowed a pill containing three grains of strychnia, in whom no symptoms were manifested for three hours; they then set in with their usual violence, and death took place in ten minutes. This pill had been prepared with mucilage eight months before, and was consequently hard and difficult to dissolve. In the Palmer case, Cook took two pills containing strychnia. No symptoms were observed for an hour and a quarter, after which death occurred in twenty minutes.

It must, however, be admitted that there are cases where the unusual delay cannot be thus accounted for, but where it must be referred to some individual peculiarity of the patient. Dr. Wormley (Micro. Chem. of Poisons, p. 40) mentions a case where the remarkable postponement of the symptoms for twelve hours appeared to be owing to the effects of a large dose of opium that had been taken simultaneously. Three grains of strychnia, a drachm of opium, and an indefinite quantity of quinine were taken at the same time (vid. ante., p. 184). Other equally remarkable instances might be adduced, showing the same apparent antagonism between strychnia and opium. Nevertheless, in some experiments of the author, made on animals with strychnia and morphia combined, the latter poison, so far
from antagonizing the former, appeared rather to intensify it (vid. ante., p. 185).

The subcutaneous injection of strychnia, as also its external application to a healthy mucous surface, produces a still more speedy manifestation of its peculiar symptoms. Some clinical experiments of Dr. Chisholm, of Baltimore, made on amaurotic patients, would seem to show that the human system acquires a tolerance of strychnia (Am. Jour. Med. Sci., April, 1872).

Fatal dose.—There is great difference as to the susceptibility to the action of strychnia. The average medicinal dose is about the one-sixteenth of a grain, though it is customary to commence with a smaller quantity. The above dose has proved fatal to a child between two and three years old. Dr. G. B. Wood mentions the case of a lady who was thrown into alarming spasms by one-twelfth of a grain (Thcrap. I, p. 834). The author has seen the case of a gentleman who had decided spasms after taking about one-twentieth of a grain.

The smallest fatal dose for an adult recorded is half a grain, which proved fatal to Dr. Warner. Dr. Ogston reports a case where three-quarters of a grain destroyed a man in three-quarters of an hour. A fatal dose for an adult may be stated to be half a grain, to one grain.

On the other hand, numerous instances are recorded of recoveries after enormous doses of this poison—ten, twelve, and even forty grains. In all these cases early vomiting was produced, which, doubtless, removed the strychnia before it was absorbed to a fatal extent. Besides, it is quite possible that the poison was not of full strength.

Fatal period.—This, like the fatal dose, is liable to considerable variation. Dr. Warner's case terminated fatally in eighteen minutes. Dr. Taylor mentions two cases in which
death occurred in ten and fifteen minutes respectively; in another case, in five minutes; in two others, in thirty minutes each.

On the other hand, life has been prolonged, even after large doses, for several hours. In Cook's case, death occurred in an hour and a quarter after swallowing the pill. In the case of a woman examined by the author, death did not occur for six hours after swallowing about six grains of strychnia (Am. Jour. Med. Sci., Oct., 1861, p. 409). Sir R. Christison reports a case in which a man died in fifteen minutes after swallowing a dose of nux vomica.

Treatment.—Prompt and free emesis is of the greatest importance. Copious draughts of warm mustard water, or a mixture of ipecac and sulphate of zinc should be given. The stomach pump may be used if the spasm of the jaws will permit. Chloroform, by inhalation, appears to have been attended with the happiest results. The patient should be constantly kept under its influence, carefully watching its effects. We would strongly advise its early administration. Bromide of potassium has also been given with the best results—sixty to eighty grains every hour, or half hour. Hydrate of chloral has also proved an efficient remedy in several cases, and nitrite of amy! has been recommended, from its known physiological effects. Atropia has also proved efficacious as an antidote in a case where chloroform failed, and where the paroxysms were very severe (Ed. Med. Jour., Sept., 1873).

As regards the remedial effects of tobacco, tincture of iron, tincture of iodine and aconite, we deem them of no value. We have experimented with them all on dogs that were poisoned with strychnia, but in no case did any of them exhibit antidotal powers.

Post-mortem appearances.—These are by no means char-
acteristic, nor are they always similar. Probably, the lesions most commonly observed are congestion of the brain and membranes, and of the spinal cord, with engorgement of the lungs, and a dark and fluid condition of the blood. The heart is sometimes contracted and empty, and at others full of blood. The rigor mortis is usually prolonged; in one case, we found it existing six weeks after death. There is also frequently noticed a livid appearance about the mouth and tongue, and also of the fingers and toes. It should be remembered that certain disorders of the brain and spinal cord, attended with tetanic convulsions, will leave precisely similar lesions to those just referred to as following death by strychnia.

Diagnosis.—The importance of a clear diagnosis, in a medico-legal case of strychnia-poisoning, cannot be too strongly urged, inasmuch as there may be, in such a case, a complete absence of all chemical proof. In the celebrated Palmer case, this question was most thoroughly sifted on both sides. Indeed, this very case affords an apt illustration of just the sort of difficulties that present themselves in forming a correct appreciation of the symptoms. In the Palmer case, the defence brought forward an immense array of diseases, which, as remarked by Tardieu, "have but a faint resemblance to, and often a complete diversity from, the characteristic phenomena of strychnia-poisoning." The only disease whose symptoms can possibly be confounded with those occasioned by strychnia, is tetanus, in its varieties of idiopathic, traumatic and hysterical, and possibly some forms of epilepsy.

If the expert were obliged to decide solely from the convolution—apart from its mode of invasion and seizure, its duration and termination, the condition of the intervals between the paroxysms, in fine, apart from the whole history
of the attack—he might probably be unable to discriminate between a case of strychnia-poisoning and one of tetanus; but where a careful examination of all these attending circumstances has been instituted, there can be no possible difficulty in reaching a satisfactory conclusion. The distinctive characters are the following: (1) In \textit{traumatic} tetanus, the history of the case, as being connected with some injury, such as a lacerated, contused, or punctured wound, involving tendons, nerves and fasciae, will always throw sufficient light on the case to admit of an easy diagnosis, although it must not be forgotten that the most trifling injury, such as the insertion of a splinter of wood beneath the fascia, and which may have entirely escaped recollection, may, after the lapse of several days, give rise to this frightful disorder; and such a case might be mistaken for \textit{idiopathic} tetanus. But as regards the latter form of the disorder, besides its extreme rarity in temperate climates, its mode of invasion (as likewise that of \textit{traumatic} tetanus), the duration of the attack and the character of the symptoms, are entirely different from those of strychnia-poisoning. In the former there are always manifested certain prodromes, such as chills, faintness, insomnia, headache, vertigo, and painful tension about the diaphragm, which may last for several days. These, of course, are entirely wanting in poisoning by strychnia, and they never can be mistaken for the general uneasiness which precedes \textit{for only a few minutes} the sudden outburst of convulsions, in the case of the poison. (2) The first symptoms in tetanus are a painful stiffness of the neck and jaws, with a difficulty of moving the head; after this, there is a gradual spreading of the rigidity over the muscles of the other parts of the body, usually the trunk first, then the limbs. In some instances the contractions reach their greatest intensity in the course of a few hours;
in others, several days may elapse. To contrast this with a case of strychnia-poisoning: in the latter, instead of the *gradual* invasion of the rigid spasms, commencing in the neck or jaws, there is a *sudden* tetanic seizure of all the muscles of the body simultaneously, producing the violent jerking of the body, and the arching of it backwards. Again, while the muscles of the neck and jaws are never the first to be affected by strychnia, but are often the last, the reverse is the case in the disease—the *trismus* being the first indication of its approach.  

(3) A third distinction is founded on the *progress* of the two cases: whilst the violent paroxysm produced by strychnia lasts only from half a minute to one or two minutes, and is succeeded by a complete relaxation, in tetanus, on the contrary, the rigidity is generally permanent, or if there be any remissions, these never exhibit the character of the complete intermissions characterizing the action of strychnia.  

(4) The *termination* of the cases is widely different; idiopathic tetanus never terminates fatally in two or three hours, but usually several days elapse; whilst in the case of the poison, death often occurs within half an hour, to two hours. Some cases of *traumatic* tetanus are reported, which proved fatal within twelve hours; and one remarkable case, quoted by Watson (*Lectures, art. Tetanus*), of a negro who lacerated his thumb by the accidental fracture of a china dish; he was seized with convulsions almost instantly, and died with tetanic symptoms in a quarter of an hour.

As regards the *hysterical* form of tetanus, although its very existence has been denied by some, especially in the male, it is admitted by numerous competent authorities; and, inasmuch as among other forms it may assume that of tetanic spasms, and might occasion doubt, under peculiar circumstances, the examiner should ascertain the previous
history of the case, which will serve to clear up the diagnosis.

In relation to epilepsy, there ought to be no difficulty in the diagnosis; the mode of seizure, the unconsciousness, and the peculiar clonic movements, are wholly different from the characteristic tetanic spasm of strychnia-poisoning. Again, the deep stupor which terminates an epileptic attack contrasts widely with the complete relaxation and perfect intelligence that follow the strychnia spasm.

Chemical analysis.—Strychnia occurs in the form of a white powder, and also in crystals, usually prismatic. It is almost insoluble in water—one part in seven or eight thousand. Absolute alcohol dissolves one part in about two hundred; amyllic alcohol, one in one hundred and twenty; pure ether, one in about fourteen hundred; commercial ether, one in about one thousand; chloroform, one part in eight. It is insoluble in the fixed alkalies, and very sparingly so in ammonia.

The salts of strychnia are very soluble in water and alcohol, but very slightly so in ether.

The taste is intensely and permanently bitter. This is one of its characteristic qualities. In fact, it is the bitterest substance known. As the result of numerous experiments, we have found distinct bitterness yielded by a solution of one grain of strychnia in several gallons of water. This bitter taste we regard as one of the strongest corroborative proofs of the presence of strychnia in a medico-legal case. Unless the ultimate extract obtained by the manipulation has a bitter taste, we need hardly expect to prove the presence of the poison by the usual chemical tests. But, of course, the mere presence of bitterness is not evidence of strychnia, since this quality pertains to numerous other substances, such as morphia, quinia, aloes, colocynth, quassia, picrotoxia, etc.
The strong mineral acids produce no coloration with strychnia, provided the latter is pure; if it contains brucia, it will impart a reddish color to nitric acid. Heated on porcelain, it melts slowly into a brown liquid, and is decomposed, giving off dense white fumes, and leaving carbon. It may be sublimed by heat, depositing crystals of pennate forms on a cold glass surface (Guy).

1. The Color test.—This is so named on account of the beautiful succession, or play of colors, that is developed by it. It consists in the application of a drop of pure sulphuric acid to a small fragment of strychnia, on a white porcelain surface, or on a watch glass over white paper. If the strychnia be perfectly pure, it will dissolve in the acid without any coloration. If now a fragment of bichromate of potassium, binoxide of manganese, binoxide of lead, ferrocyanide of potassium, or permanganate of potassium, be stirred in contact with the solution, by means of a pointed glass rod, this play of colors is instantly manifested. At first, it is of a rich, deep blue; this soon passes into violet and purple, which, in its turn fades into a pink, and finally into a red.

The relative duration of these shades of color depends on the quantity of strychnia operated on, and also on the relative amounts of acid and the other substance. Thus, if the amount of strychnia be extremely minute, the blue color may flash out but for a moment, leaving only the violet or purple, which quickly passes into the red.

The principle involved in the color test is the action of nascent oxygen (developed by the sulphuric acid on the various oxidizing substances above named) on the strychnia. For the success of the experiment, it is immaterial which one of these oxidizing bodies is employed, providing it is pure. Different authorities evince a preference for one or another, according to their individual tastes. As a rule, the
pure crystal of bichromate of potassium will yield satisfactory results.

It is very important for the medico-legal student to have clear and definite ideas about this color test for strychnia. It is not the mere production of a blue color that is of diagnostic value, for this might result from the application of permanganate of potassium to various organic bodies in the absence of strychnia; but it consists in the regular succession of colors—from blue to violet, pink, and red, the last continuing for some time, and ultimately changing to a dirty green. So far as is known at present, strychnia is the only substance that answers to the above requisition. There are others that react somewhat similarly, which will be noticed hereafter.

The exceeding delicacy of the color-test deserves special notice. If the strychnia be perfectly pure, and the manipulation be properly performed, so minute a quantity as the one-millionth of a grain can be detected, as we have repeatedly verified in our own experience, and as is corroborated by other experimenters. It depends altogether on the delicacy of the experiment. These minute quantities of strychnia are best obtained by first making a solution of the alkaloid in pure water, with the addition of acetic acid, of a definite strength. This may readily be reduced by the addition of more water. Fractional portions of the solution may be obtained by using a pipette drawn out to a capillary point, which will deposit minute droplets on a warmed, clean porcelain surface. The object here is to concentrate the quantity to be experimented upon into as small a space as possible. The drop should then be evaporated to dryness spontaneously. A small drop of pure concentrated sulphuric acid is then applied to the deposit, by means of a finely-pointed glass rod, and then a minute crystal of the
bichromate of potassium (or one of the other oxidizing bodies) is placed alongside of the acid solution, and then, by means of the rod, is drawn through the solution and gently stirred in it.

Interferences.—As above mentioned, the color-test, properly applied, will detect exceedingly minute portions of pure strychnia, but there are many organic substances whose presence will considerably modify and even completely disguise this test. This fact has been known to chemists since 1850, when it was first announced by Brieger (Chem. Gaz., VIII, p. 408). His results have been confirmed, and the list of interfering bodies has been extended by subsequent experimenters. According to Lyman (N.Y. Med. Gaz., Mar., 1871), permanganate of potassium is the only reagent that will develop the color-test with strychnia, when the latter is mixed with either morphia or quinia in excess. The most important of these interferences, considered medico-legally, is probably morphia, inasmuch as this substance might be likely to be given to allay the severity of the strychnia spasms, and would consequently be associated with the strychnia extracted from the body after death. A large number of experiments made by the author (Am. Jour. Med. Sci., Oct., 1861, and April, 1862) clearly confirm the fact of the interference of morphia with the usual color-test for strychnia, both in the pure state and when mixed with organic matters. One experiment only will here be mentioned: A small cat was poisoned "with one-twentieth of a grain of strychnia and one-tenth of a grain of morphia. The ultimate extract obtained from the stomach by Stas' process entirely failed to yield the color-test, although the bitterness of the extract, and the fact that its solution produced the characteristic tetanic convulsions in a number of frogs, distinctly proved its existence."
Admitting, then, the fact of these interferences, it is well to remember that, practically, they may be avoided, in a medico-legal investigation, by the employment of chloroform instead of ether, as the solvent to extract the strychnia from organic mixtures,—morphia and other interfering substances being insoluble in this menstruum.

Fallacies.—Exception has been taken to the color-test, on the ground that other substances besides strychnia will yield colors similar, if not identical, when similarly treated; but a careful attention will readily avoid all difficulty. The substances alluded to are curarine, veratrine, cod-liver oil, salicine, santonine, aniline, pyroxanthine, narceine, papaverine and solanine; but in relation to most of these, a radical ground of distinction is that they are colored by sulphuric acid alone, which is not the case with strychnia. A salt of aniline is not colored by the acid alone, but only in the presence of one of the above mentioned oxidizing bodies; but there is this difference between it and strychnia: the former is first colored green, then a very persistent blue, and finally black.

Curarine has many points of resemblance to strychnia; it is very bitter; it yields a succession of colors with sulphuric acid and bichromate of potassium; but it is colored by sulphuric acid alone; it is nearly insoluble in chloroform, and readily soluble in potash. Its physiological effects are the opposite of those of strychnia.

Cod-liver oil, when treated with sulphuric acid alone, affords a play of colors somewhat like those presented by strychnia.

2. The galvanic test of Dr. Letherby acts on the same principle, of presenting nascent oxygen to the strychnia; but in this instance it is evolved by galvanism. A drop of a dilute solution of strychnia is placed in a small depression made on platinum foil, or in a platinum capsule, allowed to
evaporate to dryness, and then moistened with a drop of sulphuric acid. The foil (or capsule) is connected with the positive pole of a single cell of Grove's battery, and the acid is touched with the platinum terminal from the negative pole. Instantly, the violet color will flash out on the metal, and on removing the pole from the acid, the tint will remain.

3. Potassa and ammonia precipitate the alkaloid from a somewhat concentrated solution, in the crystalline form. The best method is to expose a drop of the solution on a glass slide, to the vapors of ammonia, and place it under the microscope; the beautiful formation of the long stellate prismatic crystals can easily be distinguished; these can be identified by touching them with a drop of sulphuric acid and a fragment of bichromate of potassium, when the play of colors will take place.

4. Bichromate of potassium.—A solution of this salt throws down from a strychnia solution a bright yellow precipitate, which soon becomes crystalline. Placed under the microscope, these crystals appear in groups, mingled with octahedral plates. When dried, these should be verified by touching them with a drop of sulphuric acid. This is a satisfactory test.

5. Carbasotic (or picric) acid.—A solution of this acid precipitates strychnia from its solution in the form of abundant yellow crystals. The best mode of showing it is to add a drop of the solution to one of strychnia, on a glass slide, and view the reaction under the microscope. The precipitate which first forms soon assumes the appearance of tufts of yellow crystals, of a peculiar claw-like form. These, as in the former experiment, may be subjected to the color-test, by the same method.

Besides the above tests, there are others of inferior value—as corrosive sublimate, ferrocyanide of potassium, bichlorate
of platinum, iodated iodide of potassium, and sulpho-cyanide of potassium.

Tardieu considers chlorine gas to be a very delicate test for strychnia. When a small stream of this gas is slowly passed through a dilute solution of strychnia, each bubble of the gas becomes surrounded by a white film, and ultimately, quite a copious, white amorphous deposit takes place, which is soluble in ammonia. According to the above authority, no other alkaloid gives this reaction with chlorine.

The physiological or frog test.—The extreme susceptibility of the frog to the action of strychnia was first employed by Dr. Marshall Hall, as a test for this poison. It may be applied either by immersing a small frog in the strychnia solution, or else injecting it either into the throat of the animal, or preferably, under the skin. We have repeatedly resorted to this test, and uniformly with satisfactory results. One of these experiments demonstrates very clearly the extreme susceptibility of the frog to the influence of strychnia.

"The one five-hundredth of a grain of strychnia was put into the throat of a middling-sized frog; it was convulsed and died in about thirty minutes. The extract obtained from the stomach by Stas' process, although it afforded no perceptible color-test, had a bitterish taste, and produced tetanic spasms in several small active frogs."

Our experiments in this line further demonstrate the fact that while morphia, when present in excess, with strychnia in small quantities, has the power to disguise the color-test, it affords no obstacle to the employment of the frog-test. Two experiments only will be quoted under this head: "A frog weighing thirty-five grains was immersed in a solution containing one grain of strychnia and thirty-two grains of morphia in six pints of water; it was convulsed in twenty
minutes. Another animal, rather smaller, was convulsed in five minutes.” (In these experiments only a small portion of the solution was used—less than a fluid drachm—put into a conical glass, in which the hind-quarters only of the animal were immersed). “A cat was poisoned with one-twentieth of a grain of strychnia and one-tenth of a grain of morphia. The stomach, on examination by Stas’ process, failed to yield the color-test; but the watery solution of the extract produced most decided tetanic convulsions in eight frogs, generally resulting in death.”

Toxicological examination.—The stomach properly divided, together with its contents, and a sufficient quantity of distilled water, should be made distinctly acid with acetic acid. If the elaborate process of M. Stas is to be employed, the strongest alcohol must be used instead of water. In either case, the mass should be digested on a water-bath for several hours. A high temperature is objectionable, as it dissolves out the starchy matters. After cooling, it is strained through muslin, and the solid matters washed with dilute alcohol and pressed. The liquid should next be concentrated by evaporation, and filtered through paper. It should now be evaporated to dryness. The residue will contain any strychnia usually present in the form of acetate, mixed with organic matter. This residue should now be thoroughly mixed with a small quantity of distilled water containing a few drops of acetic acid, then filtered through paper, and the filtrate poured into a glass tube or flask, and an excess of solution of potassa or soda (or ammonia) added, which liberates the strychnia from its combination. Pure chloroform, slightly in excess of the mixture, is now added, and the whole briskly shaken together for some minutes. The chloroform dissolves out the alkaloid, and from its gravity settles to the bottom of the mixture, after the lapse of some time.
In order to separate the chloroform from the supernatant liquid, we have found that the easiest practical method is to transfer the whole mixture to a stoppered glass funnel, or what answers equally well, a glass syringe of proper size, after removing the piston, and having previously contracted the nozzle to a fine point by means of the flame. Before introducing the liquid, this small aperture should be plugged with a splinter of wood, and about half a drachm of pure chloroform first poured into the syringe, so as to about fill the narrow portion. The mixture is now to be carefully poured in, and a sufficient time allowed to elapse for the chloroform to separate and settle to the bottom.

By placing the thumb over the larger aperture of the syringe, and withdrawing the wooden plug, it will be very easy to control the flow of the contents. A few drops may be allowed to fall successively, as each one dries; into a warmed watch glass, or porcelain capsule, for a trial test, by means of sulphuric acid and bichromate of potassium (vid. p. 361). The whole of the chloroform is then permitted to flow out into one or more capsules, or watch glasses, great care being taken not to allow any of the other mixture to escape along with it. The remaining alkaline liquid may be shaken up with an additional portion of chloroform, and the separation again made as before. All the chloroform is now permitted to evaporate spontaneously to dryness. The contained strychnia, if of notable quantity, will be found in the deposit, in an amorphous—not crystalline—form, according to our experience.

A portion of this extract should now be examined by the taste, for bitterness; by the color-test (although this may not be very satisfactory, on account of the mixture with organic matter); and by the frog-test. The remaining portion of the extract is to be dissolved in a minute quantity of water,
acidulated with acetic acid, filtered, and subjected to the usual tests (vid. p. 361).

The main difficulty in conducting this experiment arises from the presence of organic matters in connection with the strychnia. If the chloroform extract has a yellow color (denoting its impurity), a few drops of strong sulphuric acid should be added to it and thoroughly stirred with a glass rod. This destroys and carbonizes all the organic matter, but merely converts the strychnia into a sulphate; add a few drops of water. After standing a short time, the dark liquid is filtered, solution of potassa is added in excess, then pure chloroform, as explained above. The second extract thus procured is generally sufficiently pure for all practical purposes.

It not unfrequently happens, when operating on complex organic mixtures by the chloroform process, that difficulty is experienced in getting the chloroform to separate from the alkaline solution; the whole mass forming a sort of emulsion. In such a case, the tube may be immersed in hot water for some time; and if this does not answer, nothing remains but to agitate the mixture several times successively with about half its volume of pure water, allowing it to rest each time, and separating the chloroform as before directed.

The method of dialysis has been recommended by some authorities, but we do not consider it as exhaustive and reliable as the one just described.

Detection in the tissues and blood.—Strychnia is absorbed into the circulation, and deposited in the various organs, just like the mineral poisons. The rapidity with which the absorption takes place is shown in a case mentioned by Taylor, where a man took five grains of the poison by mistake, and died in half an hour. Strychnia was discovered in the stomach in the quantity of one grain; it was also detected
in the liver and the tongue. This case shows that within half an hour, four-fifths of the poison had been removed from the stomach (or could not be detected there by chemical research), and had been diffused throughout the body. There are, however, other cases, where the circumstances were apparently just as favorable for the absorption and diffusion of the poison, but where there was a total failure to detect it in the organs, after death.

The process is the following: the organs are to be finely crushed and digested in strong alcohol, acidulated with sulphuric acid, in the proportion of eight drops to the fluid ounce of the mixture; this should be heated below $212^\circ$ F. for about an hour; when cool, it is to be filtered and concentrated, as before directed. The residue is then nearly neutralized by liquor potassae, care being taken to maintain an acid reaction, then filtered, and evaporated nearly to dryness. To the cooled residue a drachm or two of strong alcohol is added, and thoroughly stirred with it; this dissolves out the sulphate of strychnia, and leaves the sulphate of potassium and the organic matters. The alcoholic solution is now filtered, evaporated almost to dryness, the residue stirred with pure water, rendered alkaline by potassa, and finally agitated with chloroform, which deposits the alkaloid, if present, on evaporation.

Dr. Taylor recommends acetic, instead of sulphuric, acid; and ammonia instead of potassa, in these cases.

The method of Rodgers and Girdwood is somewhat similar, and they employ hydrochloric acid and ammonia as the reagents, along with chloroform.

Strychnia may be recovered from the blood by a similar process. In some experiments of Dr. Wormley, he detected the poison in the blood of dogs and cats, where death took place in three and six minutes respectively after its admin-
istration. This shows the extreme rapidity with which it is absorbed.

Detection in the urine.—The urine should be evaporated to a syrupy consistence, acidulated with acetic acid, mixed with an ounce of strong alcohol, filtered, and evaporated to near dryness. The residue is to be stirred with pure water, filtered, if necessary, liquor potassae added in excess, and agitated with chloroform.

Failure to detect.—It must be admitted that the most careful analysis sometimes fails to discover this poison in the body after death, and that, too, where the circumstances were apparently favorable to it. This failure may sometimes be ascribed to the smallness of the dose, and again, possibly, to some interference not well understood. Mere putrefaction of the body is no obstacle to its detection, since it has been recovered months after death, and where the body was in an advanced state of decomposition. Christison, Taylor and other well-known authorities have at times been foiled in their efforts. In a case that occurred to the author, some years ago, when a woman was poisoned (as was alleged) with six grains of strychnia, and where death was postponed for the unusually long period of six hours, there was a similar failure to detect the poison eight weeks after death, although the body was well preserved.

Brucia.—This alkaloid is generally found associated with strychnia. It occurs either in the form of a white powder, or in colorless prismatic crystals. It is more soluble in water and alcohol than strychnia. It is freely soluble in chloroform and alcohol. It has an intensely bitter taste. Concentrated sulphuric acid dissolves it, giving a faint rose coloration. Nitric acid gives a characteristic blood-red color.
Its poisonous properties are similar to those of strychnia, though less intense. As the symptoms of poisoning by brucia are similar to those caused by strychnia, the toxicologist should guard against being deceived in a medico-legal investigation, in the event of not discovering *strychnia* by the usual color test. In such a case, it would always be proper to search for *brucia*.

**Tests.**—The characteristic reagent is *nitric acid*, which instantly produces a blood-red color, with a speedy solution of the alkaloid. If heated, the color changes to yellow. If, after cooling, a drop of the solution of *protochloride of tin* be added, the color changes to a beautiful purple. The somewhat similar red color produced on *morphia* by nitric acid is *not* changed by protochloride of tin.

(2) *Sulphuric acid and nitrate of potassium.*—Touch the fragment of brucia with a drop of strong sulphuric acid, a faint rose color is produced; then add a small crystal of nitre, when the color changes to a deep orange-red.

(3) *Ammonia* produces, with a drop of brucia-solution, a beautiful crystallization, viewed by the microscope. Other tests of less importance are *sulpho-cyanide of potassium*, *bichloride of platinum* and *corrosive sublimate*.

The *frog-test* is equally applicable to brucia, allowing for its comparative inferiority in strength to strychnia. The toxicological examination for brucia is conducted in the same manner as described for strychnia. The ultimate extract is to be tested by nitric acid and protochloride of tin. Brucia has been detected in the blood of animals poisoned by it.
CHAPTER XXVIII.

ORDER III.—CEREBRO-SPINAL NEUROTICS.

(i) DELIRIANTS.

This subdivision of Cerebro-spinal Neurotics has received the name of Deliriants, because of the active delirium that constitutes one of their prominent symptoms. They also produce other effects in common, such as illusion of the senses, dilatation of the pupil, heat and dryness of the throat, a flushed face, and frequently a redness of the skin. They all belong to the same natural order of plants, Solanaceae. From their physiological property of dilating the pupil, they have received the name of Mydriatics. They comprise Belladonna, Stramonium, Hyoscyamus, and different species of Solanum.

SECTION I.

POISONING BY BELLADONNA—ATROPIA.

SYMPTOMS.—ATROPIA.—FATAL DOSE.—TREATMENT.—POST-MORTEM APPEARANCES.—CHEMICAL ANALYSIS.—TOXICOLOGICAL EXAMINATION.

Belladonna (Deadly Nightshade).—The leaves, berries and root of Atropa Belladonna are violently poisonous. The leaves and root are used in medicine. Children are frequently poisoned by eating the berries.

Symptoms.—A sense of heat and dryness in the mouth and throat, difficulty of swallowing, nausea, vomiting, giddiness, extreme dilatation of the pupil, loss of vision, flushed face, sparkling eyes, delirium of an excited, maniacal char-
acter, spectral illusions, convulsions, followed by stupor and coma. Irritation of the urinary organs frequently occurs, such as strangury, suppression of urine and haematuria. A scarlet eruption is often observed over the skin. Some of these effects have been produced by the external application of belladonna, in the form of a plaster or liniment. The symptoms of belladonna-poisoning usually show themselves from half an hour to two hours, occasionally sooner. They do not generally terminate fatally. Death, when it occurs, usually takes place within twenty-four hours.

In case of death from the leaves or seeds of belladonna, these can usually be distinguished in the alimentary canal, by their botanical characters.

Atropia.—This alkaloid is the active principle of belladonna, and is a very active poison, producing symptoms similar to those above described, only more speedily. The application of a weak solution to the eyes has occasioned symptoms of belladonna-poisoning. Used hypodermically, even in doses of one-fiftieth to one-tenth of a grain, it occasions, at times, violent symptoms. Employed in this manner in combination with morphia in excess, its activity appears to be modified. Death has resulted from the external use of a strong ointment of atropia.

Fatal dose.—One-half to three-quarters of a grain may be regarded as a minimum fatal dose for an adult. The criminal administration of this poison is very rare. Dr. Taylor records a case where a surgeon of a workhouse was fatally poisoned by a nurse, by administering it in milk. The diagnosis is not always easy, since the same symptoms are produced by hyoscyamus and stramonium. There appears to be a special tendency to its elimination from the system by the kidneys. Prof. Guy states, on the authority of Dr. John Harley, that the presence of atropia in the urine can
be readily proven within twenty minutes after the injection under the skin of one-forty-eighth to one-ninety-sixth of a grain, by the action of the urine on the eye. Twelve drops will largely dilate the pupil, and maintain it in that state for several hours (Foren. Med., p. 512).

Treatment.—The immediate evacuation of the stomach by an active emetic, or by the stomach-pump. There is no chemical antidote. The physiological antidote is *morphia*, which should be carefully and repeatedly administered (vid. ante p. 334).

Post-mortem appearances.—These are not characteristic. There may be congestion of the vessels of the brain, with some red patches of the stomach and oesophagus. When the poisoning has resulted from eating the ripe berries, the whole lining membrane of the alimentary canal may be dyed of a purple color, and portions of the berries and seeds may be discovered in the intestines, or in the stools. The blood is usually fluid, and dark-colored.

Chemical analysis.—Atropia, when pure, occurs in white crystalline tufts. Its taste is acrid and bitter; slightly soluble in cold water, very soluble in alcohol, ether and chloroform. It sublimes at 200° F. Its color is not changed by either of the mineral acids. It has alkaline properties, neutralizing acids, and forming salts.

The alkalies throw down a precipitate from a salt of atropia, which ultimately becomes crystalline. That produced by ammonia remains amorphous. It is also precipitated by chloride of gold, and by carbazotic acid. Wormley considers bromine in hydrobromic acid to be the characteristic test for atropia. The precipitate is at first amorphous, of a yellow color; but it soon becomes crystalline. It is insoluble in acetic acid, and but slightly so in either of the mineral acids. The one-ten-thousandth, to one-twenty-five
thousandth of a grain will give satisfactory results with this reagent (Micro-Chem. of Poisons, p. 631).

Toxicological examination.—We should first of all endeavor to discover any seeds, or remains of the leaves or berries of the plant. The vomit and stools should, if possible, be also examined for these. The stomach, with its contents, and other organs, properly comminuted, should be treated after a modification of Stas’ process, alcohol being used as the solvent, along with sulphuric acid. After heating, straining, evaporating, purifying by pure ether, removing the ether, and adding solution of potassa in excess, the ultimate extract is obtained by chloroform and tested, first, with the bromine-test, which, if successful, may be followed by the other tests.

The physiological test consists in applying a portion of the ultimate extract to the eyes either of a man, or one of the lower animals, as the rabbit. The minutest quantity will produce the characteristic dilatation of the pupil. It must, however, be remembered that other members of this class of vegetables will produce a similar result.

The rabbit evinces a remarkable tolerance for belladonna and its alkaloid. It will live exclusively on the former for many days, and tolerate enormous doses of the latter, either by the stomach or subcutaneously, without perceptible effects.

SECTION II.
POISONING BY STRAMONIUM, HYOSCYAMUS AND SOLANUM.

Stramonium (Thorn Apple, Jamestown Weed).—The Datura stramonium is a very common plant, abounding in this country, and also in Europe. It grows freely on waste grounds; other varieties occur in India. All parts of the plant are poisonous, especially the seeds and fruit. Its active alkaloid principle is named daturia.
Poisoning by Hyoscynamus.

Cases of poisoning by stramonium are usually accidental, and chiefly occur in children, from eating the seeds.

Symptoms.—Very similar to those produced by belladonna, such as dryness of throat, with difficulty of swallowing, dilated, insensible pupil, violent and incoherent delirium, nausea, vomiting, headache, vertigo, ringing in the ears, spectral illusions, followed by stupor and coma. Sometimes there are convulsions and paralysis, together with a scarlet efflorescence on the skin. The external application of the bruised leaves has occasioned symptoms of poisoning.

In India, the Datura is employed by the Thugs for the purpose of drugging their victims.

Post-mortem appearances.—Very similar to those resulting from belladonna. There is nothing characteristic. The seeds and remains of the leaves may be discovered in the alimentary canal, if these have been the cause of death.

Treatment.—The same as that recommended for belladonna poisoning.

Analysis.—The seeds are of a black or brown color, kidney-shaped, with a wrinkled surface. They are much larger than those of belladonna or hyoscyamus. According to Prof. Guy, it requires one hundred and twenty henbane seeds, and ninety of belladonna to weigh one grain, but only eight of stramonium. There is no known test to distinguish daturia from atropia; these two alkaloids are now generally regarded as identical.

The method of procuring daturia from the stomach and organs is the same as that above described for atropia (p. 332).

Hyoscyamus (Henbane).—The Hyoscyamus niger grows both in America and Europe. All parts are poisonous.

17*
The root is tapering, resembling that of the parsnip, for which it has often been mistaken. The medicinal preparations from the plant are extremely variable and uncertain, depending very much on the mode of growth, collection and preparation.

The symptoms, in general, resemble those of belladonna and stramonium.

Analysis.—It can only be identified in the matters vomited, or in the stomach and intestines after death, by the botanical characters of the seeds or fragments of the leaves discovered.

*Hyoscyamyia*, the active alkaloidal principle, occurs in white, silky crystals, inodorous when pure, but as usually found, possessing a disagreeable smell; taste acrid. It is difficult to isolate. There is no proper chemical test for it. It dilates the pupil, like atropia and daturia. It speedily passes into the urine, when swallowed.

*Solanum.—Three species of the genus Solanum are usually referred to in the books, as possessing poisonous properties: these are S. dulcamara, or Bittersweet, or Woody Nightshade; S. nigrum, or Garden Nightshade; and S. tuberosum, or common potato. These all contain an active alkaloid principle—*Solania.*

The S. dulcamara is a native of Great Britain, and is cultivated in our gardens as an ornament, for its purple flowers and bright red berries. The latter are frequently eaten by children, occasioning poisonous results. The dried stems are used medicinally.

The S. nigrum produces white flowers and black berries. The latter, like the fruit of the S. dulcamara, have frequently proved poisonous to children who have swallowed them. They are more powerful in their effects than the others.
POISONING BY SOLANIA.

_S. tuberosum_, or common potato.—The berries and young shoots have proved poisonous, the former fatally, in the case of a young girl reported in the Lancet, June, 1858. Christieon quotes from Dr. Kabler, of Prague, an instance where four persons of a family were seized with alarming symptoms, such as vomiting, coma and convulsions, after eating potatoes that had commenced to sprout and shrivel.

The general symptoms produced by _solania_ are very similar to those resulting from the other mydriatics. It is much less powerful than the other alkaloids of this class.

When pure, solania is in the form of delicate acicular crystals; nearly insoluble in water, soluble in alcohol, less so in ether, insoluble in chloroform. It is also soluble in amylic alcohol. Cold sulphuric acid first changes it to an orange-yellow, and then dissolves it, the solution becoming brown. Nitric acid dissolves it, the solution being at first colorless, and subsequently changing to a rose-red tint. The former acid is the best test for it. Other reagents do not give characteristic results.

Solania is separated from organic mixtures by a modification of Stas' process; alcohol and sulphuric acid being employed as the solvent, and warm alcohol to separate the final extract.
CHAPTER XXIX.

(2) DEPRESSANTS.

Under this subdivision of Cerebro-spinal Neurotics, are conveniently included several active poisons, which agree in the property of causing great depression of the muscular system, although in some other respects they may differ from one another. By thus grouping them together, it is not intended to imply that they all produce the same physiological effects.

SECTION I.

POISONING BY TOBACCO AND LOBELIA.

EFFECTS OF TOBACCO—POST-MORTEM LESIONS—NICOTINA—PROPERTIES—CHEMICAL REACTIONS—TOXICOLOGICAL EXAMINATION—LOBELIA.

TOBACCO.—The dried leaves of Nicotiana tabacum, a plant belonging to the natural order of Solanaceae. It owes its activity and poisonous properties to a volatile, liquid alkaloid, of an oily consistence, named nicotina, which somewhat resembles conia, and which exists in different proportions in different specimens of the leaves, varying from two to eight per cent.

Symptoms.—A large dose of tobacco (or even a small one to those unaccustomed to its use) produces very decided symptoms. Very soon after taking it, either by swallowing or by enema, it occasions nausea, giddiness, a sense of confusion of the head, vomiting, severe retching, great prostration, heat in the stomach, frequent and very feeble pulse, cold, clammy skin, trembling of the limbs, and sometimes severe purging. Respiration is difficult, and urination invol-
POISONING BY TOBACCO.

Untary. In some cases, there is violent pain in the abdomen; in others, there is great sense of depression, and of impending death. The pupils are not always similarly affected. Taylor states that they are dilated. Percival speaks of it as differing from belladonna in contracting them; also by the absence of delirium, and of dryness of the throat. Wharton and Stillé (Med. Jurisp., 1873, II, p. 609) state that the pupils are but slightly affected.

The external application of tobacco, either to the sound skin, or to abraded surfaces, produces alarming, and even fatal effects. A wet leaf put around the throat in spasmodic croup often relieves the spasm, but it should be used with great caution on a young child. A decoction applied to the skin of a man for an eruptive disease, caused death in three hours (Am. Jour. Med. Sci., January, 1865).

Its fatal effects, when administered by the rectum, are well known. It was formerly much used in this manner, to aid the taxis in strangulated hernia; but it is always a dangerous remedy. Even tobacco smoke, diffused through water and swallowed, has caused the death of a young infant.

Tobacco smoking has been known to produce violent and even fatal effects, when carried to great excess, although there is considerable diversity of opinion as to whether nicotin is present in tobacco smoke, or not. Respectable authorities are found on both sides.

The rapidity of the effects of tobacco on the human system varies with the dose, and mode of administration. In one case, snuff swallowed in whiskey caused death in one hour. In another instance, quoted by Beck, an enema of tobacco, used to expel worms, produced violent convulsions and death in fifteen minutes. Christison gives another case, where a tobacco enema proved fatal in thirty-five
TOXICOLOGY.

minutes. The application of nicotina to the tongue of an animal caused death within two minutes.

Post-mortem appearances.—There is no characteristic lesion. A diffused redness over the mucous surface of the stomach and bowels, with an empty heart, and congestion of the vessels of the brain, liver and lungs, are about all that will be found. The blood is usually very dark and liquid. If the leaf or powder has been swallowed, these may be recognized by microscopic examination. In a case of suicidal death, examined by Dr. Taylor, there were general relaxation of the muscular system, staring eyes, bloated and livid features, the vessels of the brain and scalp, and also of the lungs, gorged with black blood, and the heart empty, except its left auricle. There was intense congestion of the mucous membrane of the stomach and of the liver. The blood was black and liquid, and in some parts had the consistence of treacle. No peculiar odor was perceptible (On Poisons, p. 661).

Nicotina.—This alkaloid, when pure, is a colorless, oily liquid, which, on exposure, becomes light yellowish, and thicker in consistence. It produces a greasy, volatile stain on paper, like conia. It is usually said to possess an acrid, unpleasant odor, but, if perfectly pure, the smell is ethereal and agreeable. It has a strong, alkaline reaction, and a density of 1.048. It is freely soluble in water, alcohol, ether, chloroform, turpentine, and the fixed oils. Ether and chloroform will extract it from its watery solution. Its taste is very pungent and acrid, even when much diluted, causing a peculiar sensation in the throat and air passages. It slowly distills at about 295° F., and boils at about 470° F. Heated on platinum, it burns with a bright flame, emitting a thick black smoke.

Nicotina is one of the most rapidly fatal poisons known,
even rivaling prussic acid. A single drop destroyed a rabbit in three and a half minutes. In Wormley's experiments, one drop, placed in the mouth of a full-grown cat, produced immediate prostration, continued convulsions, and death in seventy-eight seconds.

In the celebrated case of Count Bocarmé, who was executed in Belgium, in 1851, for poisoning his brother-in-law, Gustave Fougnes, nicotina was the agent used. An unknown quantity was forcibly put into the throat of the victim, the Countess assisting her husband as an accomplice in the murder. Death was believed to have taken place within five minutes. The poison was detected by M. Stas in the tongue, throat, stomach, liver and spleen of the deceased, and also from stains on the floor, near where the act was committed. From the excellent report of the examination of M. Stas, we may note the following particulars: The appearance of the tongue indicated the action of some highly acrid agent; it was swollen, blackened, softened and friable; the epithelium was easily detached. This was also the condition of the mucous lining of the mouth and pharynx; it was reddened, as if cauterized, and easily separated. The lining membrane of the stomach was intensely injected, exhibiting large patches, which were livid and black. The vessels were filled with a black coagulum, resembling blood that had been treated with sulphuric acid. The duodenum was also highly injected. There were no ulcerations or perforations of the stomach and bowels. The lungs were gorged with black blood, and exhibited the usual character of asphyxia. The heart was normal, its cavities contained black, liquid blood. No odor was observed in the body (Orfila, Toxicol., II, p. 498).

Chemical reactions.—If a drop be put into a watch glass, and this be covered with another glass, inverted, containing
a drop of either nitric or hydrochloric acid, the glass will become filled with white fumes, not so dense as from conia, nor do they give rise to the formation of crystals. The strong acids applied directly to it produce no characteristic effects.

Nicotina unites freely with acids, forming salts, which retain the peculiar taste of the alkaloid, but are destitute of odor. They are mostly soluble in water and alcohol, but not in ether or chloroform.

(1) *Bichloride of platinum* throws down a yellow precipitate, which becomes crystalline, seen under the microscope, which is soluble in hydrochloric acid. (No precipitate is caused by conia.)

(2) *Corrosive sublimate* gives a white crystalline precipitate, changing to yellow. These crystals assume a peculiar, beautiful appearance, in groups of various patterns. These are distinguished from the precipitates caused by this same reagent with ammonia and the other alkaloids, by the fact that the latter are amorphous, except that of strychnia, but which last is wholly unlike that produced by nicotina. This is a very delicate test.

(3) *Terchloride of gold* yields a yellow amorphous precipitate, but not characteristic. The same is true of *iodide of potassium*, and of *bromine in hydrobromic acid*.

(4) *Carbazotic acid* gives a yellow, amorphous precipitate, which ultimately assumes the form of a crystalline tuft, to be viewed by the microscope.

Toxicological examination.—The stomach and other organs, properly prepared, may be subjected to the process of Stas. In fact, it was the very process employed by its originator in the Bocarmé case above alluded to. Other good authorities have somewhat simplified his process. Water may be employed as the solvent, instead of alcohol; and either
acetic, sulphuric, or tartaric acid may be used. After proper concentration and filtration, it should be supersaturated with potash or soda, and shaken up with chloroform or ether, and these solutions, when properly separated, allowed to evaporate spontaneously on watch crystals, when the nicotina, if present, will be seen in the form of drops or oily streaks, having the peculiar odor of the alkaloid, which is rendered more distinct by heating. This should be dissolved in a few drops of water, and the appropriate tests applied. A drop or two may also be given to a small animal. Nicotina inserted under the skin of a frog produces peculiar muscular movements, slowing of the heart's action and of respiration.

**Lobelia.**—The *Lobelia inflata*, or Indian tobacco, is a native of this country, belonging to the natural order *Lobeliaceae*. It is extensively used both here and in Great Britain as the standard remedy of the *Thomsonian or Botanical Doctors*. According to Dr. Letheby, thirteen cases of poisoning by this substance had occurred in England within three or four years, and Dr. Beck states that "thousands of individuals in the United States have been murdered by the combined use of capsicum and lobelia, administered by the Thomsonian quacks" (*Med. Jurisp.*, II, p. 736). The leaves and seeds are the parts employed. They owe their activity to a fixed alkaloid named *lobelina*.

**Symptoms.**—In small doses lobelia acts as an expectorant; in large doses, as an emetic and depressant. In poisonous doses it produces distressing nausea and vomiting, sometimes purging, extreme relaxation, cold sweats, small, feeble pulse, great prostration, contracted pupils, stupor, occasionally convulsions, coma and death—symptoms strikingly like
those caused by tobacco. A drachm of the powdered leaves has proved fatal.

The post-mortem appearances are very similar to those caused by tobacco.

Lobelina, the active alkaloid principle, is a yellowish liquid, lighter than water, of a somewhat aromatic odor, and acrid, persistent taste; soluble in water, more so in alcohol and ether; has an alkaline reaction, forming soluble salts, with acids. Tannic acid precipitates it from its solutions. It resembles nicotina in most of its properties. On animals, lobelina seems to produce the narcotic, but not the emetic effects of the plant.

No case is recorded of death from lobelina. In the investigation of a case of death from lobelia, the diagnosis would be materially aided by the discovery of fragments of the leaves, or of the seeds. (For the report of two interesting trials for fatal poisoning by lobelia, under the "botanical treatment," see Wharton & Stillé's Med. Jurisp., 1873, II, pp. 586 and 963.)

SECTION II.

POISONING BY HEMLOCK—CONIA.

SYMPTOMS.—POST-MORTEM APPEARANCES.—CONIA.—CHEMICAL REACTIONS.—TOXICOLOGICAL EXAMINATION.—OTHER POISONOUS HEMLOCKS.

The Conium maculatum, or Spotted Hemlock of Great Britain and America, is believed to be the same plant as the Cicuta of the ancient Greeks, the one that furnished the celebrated State poison by which Socrates perished. It belongs to the natural order Umbelliferae, which also includes many other poisonous plants. All parts of this plant are poisonous; the leaves and root are employed in medicine, in the form of fresh juice and extract.
Poisoning by hemlock is generally the result of accident, the fresh leaves being used in soup in mistake for parsley, which it somewhat resembles. Its action on man appears to be very variable—at least the different accounts are very diverse.

Symptoms.—Headache, imperfect vision, dilated pupils, difficulty of swallowing, drowsiness, a tingling sensation along the muscles, gradually complete paralysis of the extremities; this extends finally to the muscles of respiration, and the patient dies, at last, from apneoa. If death be delayed for some time, there may be convulsions, coma, violent delirium, accompanied with salivation, and involuntary discharges from the bladder and bowels. Death usually takes place in one to three hours. One drop of conia is considered to be a poisonous dose. The treatment consists in a prompt evacuation of the stomach by emetics, or, the use of the stomach pump, followed by castor oil and stimulants.

Post-mortem appearances.—These are not at all characteristic; redness of the mucous membrane of the stomach and congestion of the lungs being usually observed. Fragments of the leaves and the seeds (if these have been swallowed) may often be recognized in the stomach and bowels, with the aid of the microscope. If the leaves be rubbed in a mortar with liquor potassae, they emit a peculiar mousy odor.

Conia.—This alkaloid exists most abundantly in the seeds. It is one of the most powerful and fatal poisons known. Christison states that a single drop, applied to the eye of a rabbit, killed it in nine minutes; and three drops, applied in the same manner, killed a strong cat in a minute and a half. In Wormley's experiments, a single drop placed upon the tongue of a large cat, caused the animal at first to stand still; in two minutes and a half it fell upon its side, voided urine, had violent convulsions of the limbs, with trembling
of the body, when it died in three and a half minutes from
the time of administration.

_Treatment._—Prompt emesis, to get rid of the poison, and
active stimulation. Strychnia has been suggested as a phy-
siological antidote, but it is too dangerous a substance to
deserve to be employed for this purpose.

_Chemical properties._—When pure it is a colorless, vola-
tile, oily liquid; the odor is peculiar, repulsive and suffoca-
tive, resembling that of a stale tobacco pipe. Diluted with
water, it emits an odor resembling mice. It gives a greasy
stain to paper, burns with a bright, smoky flame; taste dis-
agreeable and permanent. Exposed to the air, it becomes
yellowish and resinoid. It is partially soluble in water,
freely so in alcohol, ether and chloroform; the two latter
will separate it from its aqueous solutions.

_Tests._—A drop is placed in a watch glass, and covered
over with a precisely similar glass, holding a drop of pure
hydrochloric acid on its under surface; both glasses imme-
diately become filled with dense white fumes, and the drop
of conia is converted into a mass of beautiful, delicate crys-
talline needles, which do not deliquesce in the air. Sul-
phuric acid imparts to it a pale red color. Nitric acid
causes with it dense white fumes. Strong hydrochloric
acid imparts to it a faint tint, which gradually becomes much
deeper, and on evaporation, needle-shaped crystals appear.
Like the fixed alkaloids, it yields precipitates with tannic
acid, corrosive sublimate, terchloride of gold, bichloride of
platinum, iodide of potassium, etc. Its liquid, oily condi-
tion, together with its peculiar odor, will distinguish it from
all other bodies except nicotina; and the points of differ-
ence between the two are mentioned under the head of
_Nicotina_ (p. 383).

_Toxicological examination._—Search first for any remains
of leaves, or of seeds, in the stomach and intestines, and avoid mistaking the leaves of parsley for those of hemlock. Rub the leaves in a mortar, with potassa, to develop the peculiar mousy smell. Then distill, and examine the distillate before employing the more elaborate process of Stas. Water and acetic acid may be employed as the proper solvents; evaporate the filtered solution to a syrpyy consistence, mix with strong alcohol and a few drops of acetic acid, filter again and evaporate to near dryness; add a little distilled water, supersaturate with solution of potassa, and agitate with ether, repeating the process several times. Remove the ether, and allow it to evaporate spontaneously. Dilute the alkaloid, and subject it to the appropriate tests.

The toxicologist should guard against too strong a reliance upon the supposed odor of conia. Dr. Harley justly observes that potassa may often develop an odor from organic substances which might possibly be mistaken for that of conia, when the latter was not present. Nothing short of the isolation of this principle, in a search for the poison, should satisfy us.

The other hemlocks, viz., Cicuta virosa, or water hemlock, Enanthe crocata, or hemlock water-dropwort, and Aethusa cynapium, or Fool's parsley, or lesser hemlock, are all very poisonous; this is especially true of the Enanthe, which is one of the most poisonous of the umbelliferae.
SECTION III.

POISONING BY ACONITE AND CALABAR BEAN.

PROPERTIES OF THE PLANT.—EFFECTS.—POST-MORTEM APPEARANCES.—ACONITINE.—FATAL DOSE.—TREATMENT.—CHEMICAL ANALYSIS.—TOXICOLOGICAL EXAMINATION.—CALABAR BEAN.—ESERINE.

Aconite.—The Aconitum napellus (Monkshood, or Wolfsbane) is indigenous in Europe, but is cultivated in this country. It grows from two to four or five feet high, and has a spike of rich blue flowers. All parts of it are poisonous, the root most so, depending on the presence of the alkaloid aconitia or aconitine. The root is tapering, carrot-like, two or three inches long, having a number of curly fibres passing off from it. This root has frequently been mistaken for the root of the horse-radish, from which, however, it differs essentially in appearance; the latter being long and cylindrical and truncated, not tapering, of a light brown color externally, white internally, and of a sweetish, hot and pungent taste, totally distinct from that of aconite, which imparts to the lips, tongue and fauces a peculiar tingling, numbing sensation, which is very persistent.

There is considerable diversity in the activity of different specimens of aconite, depending, doubtless, on the time and modes of collecting and drying of the plant, and probably also on the place of growth. This may account for the discordant results obtained by different investigators.

Aconite root has been administered criminally in at least one recorded case, where the powdered root was mixed with pepper, and sprinkled over the greens used for dinner by the deceased (Dub. Jour., July, 1841).

Symptoms.—On animals, according to Dr. Fleming, there are weakness of the limbs and staggering, respiration slow and labored, paralysis, loss of sensation, increased difficulty
of breathing, and after a few spasmodic twitches, death by asphyxia. In a few instances there were decided convulsions, and even opisthotonos. The pupils were generally contracted. The heart continued beating after death. There was great congestion of the venous system, with distention of the right side of the heart.

On man.—There is first a dryness of the throat, accompanied with tingling and numbness of the lips, throat and tongue, followed by nausea and vomiting, with pain and tenderness of the epigastrium. The numbness and tingling now become more general, with diminution or loss of sensibility of the surface, vertigo, dimness of vision, tinnitus aurium, with occasional deafness, frothing at the mouth, sense of constriction of the throat, great muscular prostration, inability to walk, a slow, feeble pulse, difficulty of breathing, a cold, clammy skin, dilated pupils, features pale, perhaps a few convulsions, followed by death. The mind usually remains clear to the last. Delirium is rare. Death is apt to be sudden, either from shock, asphyxia, or syncope.

Post-mortem appearances.—There is nothing characteristic. There is usually general venous congestion of all the organs, especially the brain, lungs and liver. There may be redness of the lining membrane of the stomach. The blood is generally fluid, and dark in color. The heart may continue beating for a little while after death, indicating that this was caused by asphyxia. In other cases, the death may be ascribed to syncope.

The fatal dose is undetermined, in consequence of the diversity in the strength of the different preparations of the drug. The medicinal preparations are the tinctures of the leaves and root, and the alcoholic extract. The latter is apt to be inert. The tincture of the root is the strongest, and most reliable. Twenty-five drops of this preparation
have proved fatal. An excise officer in England died in a few hours after merely tasting Fleming's strong tincture. Pereira speaks of a case where two doses of six drops each, taken at an interval of two hours, produced most alarming symptoms in a young man; and Wormley alludes to an instance in which five drops of Thayer's fluid extract of the root produced most serious effects, which continued for two hours. Half a drachm to a drachm may be considered a fatal dose.

The symptoms may come on almost immediately, or be delayed for an hour or two. Death generally occurs within three or four hours; but it may be deferred, as in other poisons, for twenty-four hours.

Aconitia (Aconitine).—The active alkaloid principle, abounding most in the root, of which it constitutes about one-tenth, to one-fifth of one per cent. In its pure state, it is probably the most powerful poison known. Pereira states that one-fiftieth of a grain nearly proved fatal to an elderly lady. Much of the aconitia, as sold in the shops, is totally inert and worthless. The only reliable article is that of Morson, of London, and probably some of the German manufacture. One-tenth of a grain may be considered a fatal dose. This poison has lately been brought prominently into notice in the case of Dr. Lamson, who used it in destroying his brother-in-law, in England, about a year ago.

Treatment.—There is no chemical antidote. The stomach should be immediately emptied by the stomach-pump, or an active emetic. Animal charcoal is recommended by Headland, also tannin, or astringent infusions. Slight galvanic shocks are recommended to be passed through the heart, in order to arouse its action, also the employment of artificial respiration. Possibly, the inhalation of oxygen might be of some advantage.
As strychnia and aconitia appear to be mutually antagonistic, it might be well to employ the former, cautiously, in the treatment of poisoning by the latter. A case of a child is quoted, from Am. Jour. Med. Sci., January, 1862, in which the recovery was apparently due to two doses of tincture of nux vomica, administered twenty minutes apart.

It would also appear that digitalis possesses an antidotal power over aconitia. Dr. J. M. Fothergill discovered that digitalis administered to frogs that were under the influence of aconite relieved the heart from the depression produced by the latter poison, recalling its normal movements. A case is reported, in the Brit. Med. Journal, December, 1872, where recovery took place in a man who, when intoxicated, had swallowed an ounce of Fleming's tincture. The patient was apparently dying, when twenty minims of tincture of digitalis were injected subcutaneously, and after twenty minutes the patient had recovered sufficiently to swallow, when a fluid drachm of the tincture was given, along with brandy and ammonia, and was twice repeated within an hour. The above statement certainly warrants the employment of this remedy in a case of aconite-poisoning.

Chemical analysis.—Aconitia, when pure, is in colorless, transparent crystals. Taste, at first, acrid, soon followed by tingling, and numbness of the lips and tongue. Its solution, applied to the skin, occasions a feeling of heat and numbness. So active is this poison that, according to Stevenson, one two-thousandth of a grain of Morson's aconitine will destroy a mouse. This same quantity causes tingling and numbness of the lips and tongue, when applied to the tip of the latter organ; and one-hundredth of a grain, dissolved in spirit and rubbed into the skin, causes a loss of feeling, lasting for some time.

It has strong basic properties, forming salts with acids,
which are mostly soluble. It is very slightly soluble in water, quite soluble in alcohol and chloroform, but insoluble in ether. None of the mineral acids change it in the cold, but warm sulphuric acid imparts to it a brown tint. There is no characteristic chemical test for it. Its presence, in a medico-legal case, can only be satisfactorily established by the physiological test—the peculiar tingling, benumbing sensation imparted to the mouth and tongue when a minute fragment of the ultimate extract is tasted, or by a similar application to the skin, attended with similar results; together with its introduction into some small animal, hypodermically.

If the poisoning has occurred from swallowing the leaves or root of the plant, a careful microscopic inspection of the stomach and bowels, and of the matters vomited and purged, should be instituted, in order to identify their botanical characters.

Toxicological examination.—A modification of Stas' process should be employed, similar to that described for nicotine (p. 384). Chloroform is preferable as the ultimate solvent. The residue thus obtained should be dissolved in a few drops of pure water, slightly acidified with acetic acid, and submitted to the physiological tests above described. If these afford no satisfactory results, no mere chemical tests can be relied on; but if they give evidence of the presence of the poison, then the solution should be subjected to all the known reactions, such as carbasotic acid, bichloride of platinum, chloride of gold, and the bromine test.

Calabar Bean.—The Ordeal Bean of Calabar (Physostigma venosum) is a large leguminous seed, from an inch to an inch and a half long, of a brownish-black color. It is used by the natives of the West Coast of Africa as the
POISONING BY CALABAR BEAN—ANTIDOTE.

ordeal test for witchcraft—the suspected person being compelled to drink a decoction of the poisonous beans. It owes its activity to the alkaloid *physostigmina*, also named *eserine*, which resides in the cotyledons. These, when touched with nitric acid, assume an orange tint, and with perchloride of iron, a brown one. The alkaloid is a colorless, crystalline solid, bitter to the taste, very slightly soluble in water; soluble in alcohol, ether, chloroform and benzol.

*Bromine in bromide of potassium* produces with it a red color. It gives this color with less than \(\frac{1}{100}\) of a grain (Dragendorff). According to Dr. J. B. Edwards (*Med. Times and Gaz.*, 1864), it reacts with sulphuric acid and bi-chromate of potassium very much like strychnia—producing the play of colors; this, however, needs further confirmation.

The action of this poison upon the lower animals is that of a *spinal depressant*, causing, at first, tremors, and then paralysis, with muscular flaccidity; contraction of the pupils; respiration slow, irregular and stertorous; sometimes there are convulsions. The heart is found to beat for some time after death. Consciousness is preserved throughout.

The effects on man are similar to the above. They are the opposite to those produced by strychnia, which is a true spinal excitant. For this reason it has been employed as a remedy for *tetanus*, and also as an antidote for strychnia.

Its most characteristic physiological action is the property of contracting the pupil, which at once distinguishes it from belladonna, as also from conia and curarin, which it resembles in some particulars.

The true physiological *antidote* is *atropia*, used hypodermically, and repeated until expansion of the pupil is manifested. From the experiments of Dr. Fraser and others,
there can be no doubt of the mutual antagonism of atropia and eserine.

The most satisfactory test is the physiological one—its power to contract the pupil. A drop or two of the suspected fluid is put into the eye of a rabbit, or other small animal, and in the course of fifteen or twenty minutes the characteristic impression will be observed.

Dragendorff has succeeded in separating it from the tissues by a modification of Stas' process, employing benzol, instead of ether, as the ultimate solvent.

Six of the beans, when eaten, proved fatal to a boy, aged six years (Lancet, Aug. 27, 1864).
CHAPTER XXX.

(a) ASTHENICS.

This subdivision of Cerebro-spinants comprises those Neurotics which destroy life by asthenia, or failure of the heart's action. It is not intended to assert that they may not prove fatal in some cases, in another manner, as e.g., through shock, or asphyxia. But as the most strongly-marked symptoms are those of heart failure, this name answers sufficiently well for grouping together those neurotic poisons that especially display this property. The two most important members of this group are Hydrocyanic Acid and Digitalis. Cocculus Indicus is considered under the same head, for the sake of convenience.

SECTION I.

POISONING BY HYDROCYANIC ACID.

NATURAL OCCURRENCE IN VEGETABLES.—PURE AND OFFICINAL ACIDS.—SYMPTOMS.—FATAL PERIOD AND DOSE.—TREATMENT.—POST-MORTEM APPEARANCES.—TESTS.—TOXICOLOGICAL EXAMINATION.—CYANIDE OF POTASSIUM.—OIL OF BITTER ALMONDS.—CHERRY-LAUREL WATER.—NITRO-BENZOLE.

Hydrocyanic, or Prussic Acid, is one of the most energetic and rapidly fatal poisons known. It occurs as a natural product in the bitter almond, the kernels of the peach, apricot, plum and cherry, the pips of apples, and the flowers and leaves of the peach and cherry-laurel. From the latter, a very poisonous water (cherry-laurel water) is distilled. It also exists in the root of the mountain ash. Properly speaking, hydrocyanic acid does not pre-exist in
these vegetable substances, but is the product of the reaction of water upon two principles which they contain, **vis.**, amygdalin and emulsin, at a certain temperature.

Prussic acid, in its pure, anhydrous state, is a compound of cyanogen and hydrogen, HCy. It is a colorless, limpid liquid, extremely volatile, and having the odor of bitter almonds. It is one of the most active and rapidly fatal poisons known. A single drop placed upon the tongue of a large dog caused death in a few seconds. The anhydrous acid is rarely met with except in the laboratory of the chemist. It possesses no medico-legal interest. It is the **dilute** or **medicinal** acid that is so frequently the cause of death. This latter is merely a solution of the anhydrous acid in water.

It occurs in the shops under two different forms: (1) The **officinal** acid, of the average strength of two per cent.; and (2) **Scheele's acid**, of the average strength of five per cent. But the strength of both varieties varies considerably, and it is not uncommon to find some specimens totally inert. This may probably arise from the liability of the acid to undergo decomposition when exposed to the light. The dilute acid is colorless, and has the odor of bitter almonds, and a hot, pungent taste.

**Symptoms.**—These vary with the size of the dose. A large dose—half an ounce to an ounce of the diluted acid—may produce symptoms in the act of swallowing, or in a few seconds after. They are seldom delayed beyond one or two minutes. Tardieu describes them as "coming with lightning-like rapidity." There is an immediate loss of muscular power, with giddiness; the person staggers and falls to the ground; the respiration becomes hurried and gasping; the pulse imperceptible; the eyes glassy and protruding; the pupils dilated and insensible to light; the extremities cold;
and sometimes convulsions occur. Toward the last, the breathing is performed convulsively, in sobs. Sometimes the bladder and rectum are evacuated involuntarily. As regards the peculiar cry or shriek, such as is often heard in animals poisoned by prussic acid, the experience of all observers is against its existence in the human subject. The face is livid or pallid, the jaws spasmodically closed; there is frothing at the mouth, occasionally bloody; often the peculiar odor of the poison can be detected in the breath; death occurs sometimes in a violent convulsion; at others it is preceded by coma, with stertorous breathing. This latter symptom (stertorous breathing) is of considerable medico-legal importance, since it might easily lead to a mistaken diagnosis for apoplexy.

**Fatal period.**—Death generally occurs within ten or fifteen minutes after swallowing the poison. Rarely is it protracted for half an hour. One case is recorded where an hour supervened. Insensibility is not, however, always immediate; instances are recorded of persons, after swallowing very large and fatal doses of this poison, performing many voluntary acts, such as walking into another room, opening drawers, going down stairs, etc.

The symptoms attendant on a large, but not fatal, dose, are confusion of head, giddiness, a sense of weight upon the brain, great muscular debility, nausea, vomiting and possibly convulsions, oppressed breathing. Several days may elapse before complete recovery takes place.

The external application of this acid to the skin, especially if abraded, may occasion serious, and even fatal consequences. Christison reports a case where the liquid, applied to a wound in the hand, caused death in one hour.

**Fatal quantity.**—The minimum fatal dose for an adult
may be taken to be about fifty minims of the officinal acid, which is equivalent to nine-tenths of a grain of anhydrous acid. The largest dose from which there was a recovery was reported in the Lancet (January 14, 1854), in which one drachm of Scheele's acid, equivalent to 2.4 grains of anhydrous acid, was swallowed by mistake. Other instances are reported of recovery after taking doses equivalent to two grains, and under, of the anhydrous acid, in all of which prompt and vigorous measures were adopted.

The inhalation of the vapor is exceedingly dangerous, and has even proved fatal.

Treatment.—So rapid are the poisonous effects of hydrocyanic acid that there is scarcely any opportunity for the employment of remedies. The cold affusion, by dashing cold water over the face and chest, should be at once employed. This should be followed by the cautious inhalation of diluted ammonia and chlorine vapors, along with stimulants, applied both internally and externally. As a chemical antidote, a mixture of ferrous and ferric sulphates, followed by a solution of carbonate of potassium, has been proposed; this would produce, with hydrocyanic acid in the stomach, Prussian blue—an inert compound. The experiment has proved successful in animals.

Post-mortem appearances.—The face is pale, or livid; the eyes often glistening and staring, with the pupils dilated; the lips blue; jaws firmly set, with, at times, a bloody froth issuing from the mouth. The blood is of a dark blue color, and fluid. The cerebral vessels are congested. Tardieu alludes to effusions of blood and serum at the base of the brain, as an occasional occurrence, which might suggest the presence of apoplexy, which, however, is negatived by the absence of hemiplegia, and by the rapidity of the death. There is congestion of the lungs and liver; and the mucous
membrane of the stomach, especially about the cardiac extremity, is apt to be much reddened.

The exhalation of the peculiar odor is one of the most important post-mortem characters. This odor is sometimes perceived even before the body is opened, in recent cases, but it is particularly noticeable in opening the abdomen and thorax, and even the brain, but especially the stomach. But as the poison is very volatile, it may easily happen that the odor will have disappeared in a few hours or days, if the body has been much exposed. There is a singular variation in this respect in different cases. Moreover, the odor may be disguised by other more powerful smells, such as tobacco, mint, etc. The mere absence of odor is, therefore, no proof of the non-existence of the poison.

Chemical analysis.—There are four recognized tests for hydrocyanic acid, which may be briefly designated as the silver, iron, sulphur and copper tests. The first three are characteristic; and they may be applied to the acid either in its form of liquid, or vapor.

1. The Silver test.—A solution of hydrocyanic acid, or of a cyanide, gives with a solution of nitrate of silver a white crystalline precipitate, distinguishable from the white chloride, as follows: (1) By its crystalline characters (prisms or needles); the chloride is amorphous. (2) Its sparing solubility in ammonia; the chloride is very soluble. (3) The permanence of its color when exposed to the light; the chloride becomes dark-colored. (4) Its solubility in boiling nitric acid; the chloride is insoluble. (5) When perfectly dried, and heated in a small reduction-tube, the cyanide of silver is decomposed, evolving cyanogen gas, which burns with a characteristic roseate flame. (6) By adding to the cyanide of silver hydrochloric acid and persulphate of iron, Prussian blue will be formed.

18*
Another mode of identifying the cyanide of silver, recommended by Orfila and Tardieu, is, after thoroughly washing and drying it, to introduce it into a small glass tube, closed at one end, from five to seven inches long, and containing in its closed extremity a rather less quantity of pure iodine. On heating this end of the tube very gently, beautiful snow-white crystals of iodide of cyanogen are deposited upon the cool portion of the tube. These crystals may be preserved indefinitely in sealed tubes; and they may be used for developing Prussian blue, by dissolving them in a solution of potassa, and adding a mixture of a ferrous and ferric salt.

The silver test is particularly delicate when applied to the acid in a state of vapor. For this purpose the material containing the suspected poison is put into a beaker, or wide-mouthed flask, and a watch glass containing on its concave surface a drop or two of nitrate of silver solution is inverted over the mouth of the flask, which should be gently heated by immersion in warm water. The vapor of the acid immediately rises, and coming in contact with the silver salt, forms a white, opaque spot of cyanide of silver, which can easily be recognized by a lens, and by the other tests mentioned above. If, however, the material should be in a state of putrefaction, this vapor-test cannot be applied, since the black sulphide of silver resulting from the sulphuretted hydrogen of decomposition, would completely obscure the white cyanide.

The silver vapor-test is considered to be the most delicate of all the tests. It is stated that two of a grain of the acid may thus be distinctly recognized. Prof. Guy (For. Med., p. 575) mentions that a single apple pip, bruised and moistened with water, and placed in a watch glass, over which was inverted another glass moistened with the silver solution,
yielded twenty-two distinct reactions—each spot exhibiting, by the microscope, crystals of cyanide of silver.

2. The Iron test.—This consists in adding to the suspected solution a little liquor potassæ, and then a mixture of ferrous and ferric sulphates; a dirty greenish-blue precipitate is thrown down, which, on addition of a few drops of pure hydrochloric acid, becomes clear Prussian blue. If the amount of the poison be very minute, there is no immediate precipitate, although the solution has a blue (or at first, perhaps, a green) color; but in time, a blue precipitate will subside.

In a medico-legal case, where great precision is necessary, if the quantity is very small, it is recommended to throw the liquid upon a white paper filter, after adding the hydrochloric acid; the blue deposit on the paper, after washing with very dilute acid, will show very distinctly upon the white ground; the paper when dried may be preserved for exhibition, if needed.

In manipulating with this test, caution should be used not to employ an excess of the reagents, as this materially interferes with the success of the experiment.

The iron test may also be used as a vapor test. Moisten the watch glass with a drop of potassa solution, and after exposure to the suspected vapors, add a drop or two of the mixed iron salts, and develop the Prussian blue by a drop of dilute hydrochloric acid.

3. The Sulphur test (Liebig’s test).—If sulphide of ammonium be added to a solution of hydrocyanic acid, and gently heated to dryness, a white sulpho-cyanide of ammonium is formed; when this is touched with a drop of perchloride, or persulphate, of iron, there is instantly produced the blood-red sulpho-cyanide of iron, which is characteristic of the presence of prussic acid, in the absence of meconic acid (vid. Opium, p. 337).
The sulphur test is best applied as a vapor test. Moisten a watch glass with a drop or two of sulphide of ammonium, and invert it over the vessel containing the prussic acid, gently warming the latter, as above directed. The vapor will rise, and form the sulpho-cyanide of ammonium on the glass. When this is allowed to dry by evaporation, it appears as a white spot, and when it is touched with a drop of the iron salt it immediately assumes the characteristic blood-red color. If the evaporation should not have been complete, so as to thoroughly dry it, the application of the iron salt may produce a black stain (sulphide of iron), which will obscure the result.

The sulphur test, moreover, may be applied to confirm the silver test. For this purpose, the spot of cyanide of silver should be moistened with a drop of sulphide of ammonium, and, when thoroughly dried, touched with a drop of the persalt of iron. The characteristic blood-red color may be distinguished, in spite of the black sulphide with which it is associated.

4. The Copper test.—The liquid is first made slightly alkaline by liquor potassæ, and a dilute solution of sulphate of copper is added; a greenish-white precipitate is thrown down, which becomes nearly white on the addition of a little hydrochloric acid.

This test may be used, also, as a vapor test. The watch glass is moistened with a drop of the copper solution, made slightly alkaline, and, after exposure, a drop of dilute hydrochloric acid is added.

As regards the relative delicacy of the above tests, experiments show that for the liquid hydrocyanic acid, the iron and sulphur tests exceed the silver test; but, when in the form of vapor, the latter far surpasses all the others.

Toxicological examination.—The stomach, together with
its contents, and other viscera (having first been carefully examined for the peculiar odor) should be distilled in a glass retort, at a moderate temperature, care being first taken to ascertain if the material is acid or alkaline. Unless distinctly alkaline, no acid must be added, otherwise it will be impossible to determine whether the prussic acid found in the distillate was originally present in the free state, or whether it might not have resulted from the action of the acid used upon a cyanide, a ferrocyanide, or a sulpho-cyanide, that might have been present in the material. Therefore, in a medico-legal case of suspected poisoning by prussic acid, we deem it very unadvisable to employ any acid in the distilling process, since, by so doing, the examiner puts it out of his power to determine whether the poison was really present in the free state, or whether he may not have actually manufactured it out of the sulpho-cyanide of potassium, which is known to exist in human saliva, and which would be very likely to be present in the stomach of the deceased. Of course, if cyanide of potassium has been the poison employed, the contents of the stomach would give an alkaline reaction, in which case the addition of sulphuric, or some other acid, would be perfectly proper.

In the celebrated case of Dr. Paul Schöeppe, in Carlisle, Pa., in 1868 and 1872, this was made a capital point by the defence. The allegation at first had been that the deceased (a lady of fifty-four years) had been poisoned by prussic acid. It was afterward contended that the death was due to a mixture of prussic acid and morphia. The analyst employed the distillation process, along with sulphuric acid, and testified to his having only obtained faint traces of prussic acid by his process. It was very justly contended by the defence that these “traces” of the poison could readily be accounted for by the faulty process employed in
the analysis, being, in fact, the result of the action of the acid upon the sulpho-cyanide of potassium which is often found in the saliva. Moreover, there was an entire absence of the characteristic symptoms of the alleged poison before death. At the second trial, the evidence of the prosecution completely broke down, and the prisoner was acquitted.

The source of the poison found in the distillate, where an acid is employed, may be determined by treating a portion of the original material with a few drops of hydrochloric acid, stirring the mixture for a short time, and adding the perchloride of iron. If the liquid contains either a ferro-cyanide, or a sulpho-cyanide, the former will be indicated by the formation of Prussian blue, and the latter by the red sulpho-cyanide of iron; whereas, a simple cyanide, as cyanide of potassium, will not give any reaction under the circumstances.

As regards the question whether prussic acid can be generated spontaneously, by the distillation of putrescent animal matters, although Orfila appears to have inclined to this belief, it is not held by later authorities. Still, we are of the opinion that, in an important medico-legal case, involving the life of the accused, something more should be insisted on as proof of poisoning than the finding of "mere traces" of prussic acid, since these might possibly be the result of some spontaneous animal decomposition, brought about under conditions not yet perfectly understood. Especially should this be insisted on, where the symptoms preceding death did not agree with those characteristic of the alleged poison (vid., Ptomaines, post).

Period after death when the poison may be found.—On account of its volatility and ready decomposition, all traces of prussic acid may disappear very shortly after death. The most skillful analysts have failed to discover it in twenty-six
hours after death, in some cases; whilst in others it has been detected as late as twenty-three days after.

The mere fact of *putrefaction* is no obstacle to its detection, although in such a case it will not be discoverable, either by distillation, or by the vapor tests. It would have all been converted into sulpho-cyanide of ammonium by the sulphide of ammonium resulting from the putrefaction. In such a case, the material should be rendered slightly alkaline, and then acted on by alcohol, which dissolves the sulpho-cyanide; filter, and evaporate to dryness; dissolve the residue in water, and test by a persalt of iron.

*Quantitative determination.*—Free hydrocyanic acid is precipitated by nitrate of silver; the resulting cyanide is washed, dried, and weighed. Every 100 parts represent 20.15 parts of anhydrous acid.

**Cyanide of Potassium.**—This salt is very much employed in photography and electrotyping, and is a frequent source of poisoning to artisans engaged in the above employments. It is a powerful poison, causing death in doses under five grains.

It is a white, deliquescent salt, very soluble in water, less so in alcohol, the solution giving off the prussic odor; it has an alkaline reaction.

The symptoms, *post-mortem lesions* and treatment are similar to those described under Hydrocyanic Acid.

*Chemical analysis.*—1. It is decomposed by all acids, setting prussic acid free, which is readily recognized. 2. It gives, with nitrate of silver, the white cyanide of silver. 3. The potash is precipitated by tartaric acid, and bichloride of platinum. 4. The iron and copper tests may be used, as for prussic acid, but without the liquor potassæ.

In organic mixtures, the prussic acid may be obtained by
neutralizing with sulphuric acid, and distilling at a low temperature.

Oil of Bitter Almonds.—This does not pre-exist in the bitter almond, but results from the reaction of water upon its amygdalin and emulsin. It is obtained by distillation of bitter almonds, reduced to a pulp, along with water. It contains a variable proportion—seven to fourteen per cent.—of anhydrous prussic acid, together with hydrate of benzole, benzoin and benzoic acid. When entirely freed from prussic acid, the oil is innocuous.

Properties.—It has a light yellow color, pungent, prussic odor, and a bitter, aromatic, pungent taste. It is heavier than water, in which it is but slightly soluble; soluble in alcohol and ether. It is highly poisonous. The liquid sold as essence of bitter almonds is a solution of this oil in alcohol; it is a very dangerous substance for domestic use.

The symptoms, post-mortem lesions and treatment are the same as those described under the head of Hydrocyanic Acid. The fatal dose is about twenty drops.

Cherry-Laurel Water, obtained by distilling the leaves of the cherry-laurel (Prunus-lauro cerasus) contains a portion of an essential oil similar to the oil of bitter almonds. It owes its poisonous properties, like the latter, to the prussic acid contained. Cherry-laurel water has occasionally proved fatal. It is specially identified with the celebrated Donallen case, who poisoned his brother-in law, Sir Theodosius Broughton, in 1782.

The kernels of the peach, apricot and cherry have all proved poisonous, especially to children who have swallowed them. The symptoms are very similar to those produced by a moderate dose of prussic acid.
Nitro-Benzole, or Essence of Mirbane.—This substance is the product of the action of nitrous acid on benzole. It is a pale yellow liquid, with a strong odor of bitter almonds. It is used in perfumery and confectionery as a cheap substitute for the oil of bitter almonds. It is a powerful narcotic, producing effects resembling those of prussic acid, although much slower in its operation, requiring four or five hours before death occurs, which is usually preceded by coma, as in apoplexy. This poison operates more rapidly and powerfully when inhaled in the form of vapor.

In a fatal case it may be generally identified by its strong odor.

Chemical analysis.—It is distinguished from the oil of bitter almonds, which it so closely resembles in smell, by pouring a few drops of each upon a plate and adding a drop of strong sulphuric acid; the oil of bitter almonds acquires a rich crimson color, with a yellow border, while the nitro-benzole is not affected. It gives none of the reactions of hydrocyanic acid with the usual tests of this acid.

When associated with organic substances, as the stomach, etc., it may be separated by first adding sulphuric acid, and distilling.

SECTION II.

POISONING BY DIGITALIS.—DIGITALINE.

SYMPTOMS.—POST-MORTEM LESIONS.—FATAL DOSE.—DIGITALINE.—CHEMICAL ANALYSIS.—TOXICOLOGICAL EXAMINATION.—CASE OF DE LA POMERAI.

The purple Foxglove (Digitalis purpurea) is a native of Europe, but cultivated in our gardens. All parts of the plant contain the poisonous, active principle, digitaline, which, however, abounds most in the leaves of the second year's growth.
Symptoms.—Cases of digitalis poisoning are comparatively rare. Until recently, its action was generally regarded as a direct cardiac depressant, reducing both the force and frequency of the heart’s action. Modern therapeutists are disposed to consider it as a direct heart stimulant, asserting that, while the pulsations of the heart are diminished in frequency, they are increased in power.

The poisonous impressions in man are nausea and vomiting, purging, with severe abdominal pains, a sense of heat in the head, vertigo and disordered vision, dilated pupils, the pulse full and slow in the horizontal position, but rapid and feeble on sitting up. Prostration then comes on, with a tendency to syncope; the eyes very prominent and 'fixed, the sclerotic coat acquiring, according to Tardieu, a peculiar, characteristic blue color. Sometimes there is salivation and suppression of urine; delirium, stupor and convulsions are apt to occur just before death, which does not, as a rule, occur within twenty-four hours. Tardieu mentions a case who died in three-quarters of an hour after swallowing, by mistake, a very large dose.

Digitalis is a cumulative poison, showing a tendency to break out with great violence after taking a number of moderate doses. The most diagnostic sign of the action of digitalis is the peculiar enfeebled, intermittent pulse, which varies so notably between the supine and the erect position of the patient.

Post-mortem appearances.—Nothing very characteristic. Turgescence of the vessels at the base of the brain, together with redness of the lining membrane of the stomach.

Fatal dose.—Not accurately settled. As much as a drachm of the powder, and half a fluid ounce of the tincture, have been taken with impunity; but a far less quantity has produced decided effects on the heart’s action. The usual dose
is one to two grains of the powder, and ten drops of the tincture, to be repeated.

**Digitaline.**—A neutral principle, generally occurring as an amorphous powder, of a pale yellowish color; sometimes in the crystalline state. There seems to be much diversity of opinion concerning the percentage of digitaline in the leaves; some authorities giving it at about ten per cent., whilst Blaquart asserts that there are ten to twelve per cent. of the *crystallizable* variety.

There seems to be a true antagonism between digitaline and aconitine. It is stated that when the heart of the frog has almost ceased to beat, under the influence of digitalis, its movements are restored by aconitine; and a case is reported (*Brit. Med. Jour.*, Dec., 1872) of recovery after the ingestion of an ounce of Fleming’s tincture of aconite, apparently due to the hypodermic injection of twenty minims of tincture of digitalis, and the exhibition by the mouth of three doses of one drachm each, within an hour, together with brandy and ammonia (*H. C. Wood’s Therap.*, p. 125). As yet, no case of *digitalis-poisoning* in man has been recorded, in which the antidotal virtues of aconitine have been tested.

**Chemical analysis.**—Both the amorphous and crystalline varieties have a very bitter taste; very sparingly soluble in water, also in pure ether; but very soluble in ether containing alcohol. Chloroform is its best solvent. It has no alkaline reaction. Cold sulphuric acid imparts to it a brownish color, which gradually changes to a red. If warmed, the color passes to a brown. If to the cold brown solution an excess of water be added, the color changes to a green, depositing a green powder, and the liquid gradually assumes a yellowish tint (Tardieu). Strong nitric acid dissolves it with effervescence, giving off red fumes and
imparting an orange-red color, which gradually becomes fainter. Hydrochloric acid imparts to it a light-greenish tint. It is stated that if the brown sulphuric acid solution be exposed to bromine vapor, it assumes a violet hue, but Tardieu denies that this test is at all characteristic.

**Toxicological examination.**—In a suspected case the examiner should first carefully search for remnants of the powdered leaves in the matters vomited, and in the alimentary canal. If the tincture has been swallowed, the interior of the stomach might present a greenish color and emit a suggestive odor. If digitaline granules have been taken, a careful post-mortem inspection might possibly reveal the presence of some of them remaining in the stomach.

The viscera, properly comminuted, should first be heated on a water-bath, with strong alcohol, for a considerable time. After cooling and straining, and proper concentration by evaporation, part of the extract may be used as a trial test on a small animal. The rest of it should be further purified by another solution in alcohol, filtration and evaporation, and the physiological test again repeated.

All authorities unite in saying that, in a medico-legal case, it is impossible to determine the existence of the poison by any chemical tests, neither by the post-mortem lesions; our reliance must be solely on the physiological test—injecting hypodermically some of the ultimate extract into a small animal, as the frog. It seems well established, by numerous experiments, that death takes place by a sudden cessation of the heart's action, with a decided rigidity of the ventricles at the moment of death. In frogs, this stoppage occurs always in the state of strong systole of the ventricle.

By observing, then, the action of the suspected poison, introduced under the skin of the frog, the gradual irregu-
larity and slowness of the heart-beats, together with the manner of its final stop, and experimenting at the same time, with digitaline itself, upon another animal, we may be able to arrive at a satisfactory conclusion.

The most noted, if not the only, instance of homicidal poisoning by digitaline is that of de la Pomerais, a homoeopathic practitioner of France, who was tried and convicted for killing his former mistress, after having insured her life in various offices for his own benefit. After one of his visits to her she died, after suffering from violent vomiting and great depression of the heart's action and debility, in twenty-four hours. Her body was examined thirteen days after death, suspicion having been aroused against the prisoner. The examiners, Tardieu and Roussin, failing to discover any poison by chemical research, resorted to the above-mentioned physiological test, employing the extract obtained from the stomach and bowels and one procured from the scrapings of the floor on which the deceased had vomited, which responded also to all the known chemical reactions. A strong circumstantial evidence of the guilt of the accused was the finding in his possession an unusually large amount of digitaline—a substance that had only lately been discovered, besides a number of other deadly poisons. In short, he had the motive, the means and the opportunity for accomplishing his purpose. He was condemned and executed (Tardieu, Sur l' Empoison., p. 694).

Poisoning by Cocculus Indicus.—Cocculus Indicus (Levant nut) is the fruit of the Anamirta cocculus, a tree growing in the East Indies. The kernel of the berry is the only poisonous part. It has an intensely bitter taste, and contains a highly poisonous principle called Picrotoxine. It is chiefly employed as a fish poison, and also in Great
Britain for the malicious destruction of game. It is also popularly believed to be used for adulterating malt liquors, by imparting to them a bitter flavor, with a diminished amount of hops and malt. It is also used for the destruction of vermin.

The symptoms are somewhat singular, indicating an action on the cerebro-spinal centres. There is loss of voluntary power, but not of consciousness, the sufferer lying in a sort of nightmare. There may also be nausea, vomiting and severe abdominal pains. The late Dr. Fish, of Philadelphia, reported several cases of accidental poisoning by a decoction of this substance, of six persons in the Philadelphia Hospital. Two of these died in about half an hour. The remaining four were seized with violent symptoms within half an hour after swallowing the poison, and recovered after several hours. Their symptoms were faintness, confusion of mind, giddiness, dimness of vision, nausea, excessive thirst, severe abdominal pain, and in one case, insensibility. The pulse was much weakened and the respiration slow and labored.

The external application has been followed by violent and even fatal effects.

Picrotoxine, or picrotoxia is generally regarded as an alkaloid. It constitutes about one per cent. of the kernel. It crystallizes in colorless, silken, slender, six-sided prisms. Sparingly soluble in water, very soluble in alcohol, ether, chloroform and amylic alcohol. Cold sulphuric acid does not affect it; the hot acid imparts to it an orange-yellow color, which becomes pale on cooling. Strong nitric and hydrochloric acid do not affect it. It acts like grape sugar when boiled with sulphate of copper and potassa, and is said to belong to the glucosides.

It may be separated from organic liquids, such as beer,
or ale, by first acidulating with hydrochloric acid, and then shaking up with ether, which holds the poison in solution, and deposits it in crystals. By this same means it may be separated from the stomach, in case of poisoning.

There are several other vegetable poisons of minor importance; among them may be mentioned the bark and seeds of the Laburnum (Cytisus laburnum), a very common tree or shrub of Great Britain. It contains an active poisonous principle, cytisine, whose effects are those of an irritant narcotic. Death has frequently resulted from taking both the bark and seeds of this plant.

The leaves and berries of the Yew (Taxus baccata) act powerfully as an acrid, irritant narcotic, even in small quantities. So, likewise, do the Privet (Ligustrum vulgare); the Guelder Rose (Viburnum opulus); and the Holly (Ilex aquifolium).
CHAPTER XXXI.

THE ALKALOIDS OF PUTREFACTION—PTOMAINES.

Within the past few years the attention of toxicologists has been called to the existence of a certain class of bodies, to which Selmi has given the name of Ptomaines, resulting from the decay of organic substances—chiefly animal. These bodies strongly resemble the vegetable alkaloids in their chemical and physiological actions. Some of them are very poisonous, some are inert, and others again seem to act antagonistically to certain poisonous alkaloids.

There is, as yet, considerable uncertainty and vagueness about these bodies, so that we are not qualified to speak positively concerning them. It has long been known that putrescent meat will occasion severe, and sometimes fatal symptoms in persons who partake of it. These symptoms are of a narcotic-irritant character, and strongly resemble those produced by certain familiar poisons. Similar effects are also known to occasionally result from cheese, sausages and certain shell-fish, particularly from mussels (vid. ante, p. 328); and, in some instances, from canned vegetables. These anomalous symptoms have been attributed to various causes, but hitherto no very satisfactory explanation of them has ever been offered. If the existence of the cadaveric alkaloids, or ptomaines, should be positively demonstrated, the natural inference would then be that these were the true cause of the morbific symptoms above alluded to.

It has been further stated that decomposed maize contains a poisonous alkaloidal principle, which is capable of producing tetanic symptoms, and which are sometimes
attended with narcosis; and Ranke contends that the proper physiological action of the impure strychnia extracted from a putrefied body, may be masked by ptomaines. If this observation is correct, the fact may serve as a very plausible explanation of the occasional failure to discover strychnia and other alkaloids in a putrescent body. A convenient process for obtaining these bodies, according to H. Maas (Am. Jour. Med. Sci., July, 1884, from Gaz. Hebdom.), is to treat the putrefied mass with alcohol and acetic acid for several days; filter, and repeat the process. Reduce the alcoholic extract on a water-bath, and concentrate the aqueous extract to a syrup. This may be employed for experimentation on animals. By treating this acid solution with ether, or amylic alcohol, or chloroform (first adding soda), several different alkaloids have been separated, which exhibit different physiological effects on frogs—one acting like morphia, another like strychnia, and a third paralyzing the heart and decomposing the blood.

Some ptomaines strongly resemble, in their properties and effects, the alkaloid aconitine; others act very similarly to veratrum, morphia and codeia; and they might easily be confounded with them in a medico-legal investigation. Methods of discriminating between them have, however, been devised, in the use of potassium ferricyanide and silver bromide, as reagents, the former being reduced to the ferrocyanide in the presence of a ptomaine; while no such reduction occurs if an alkaloid is present. Selmi and others have succeeded in extracting poisonous bases from the urine of patients suffering from tetanus, progressive paralysis and miliary fever; one of these resembled nicotina in its general character, showing a special tendency to act upon the spinal marrow and heart; the other base resembled conia in odor.

Still later researches go to prove that animal fluids, such
as fresh blood and albumen, before undergoing putrefaction, give precisely similar reactions, with the reagents employed to those that are afforded by these same reagents with ptomaines extracted from a dead animal body.

It will be inferred from all that has been stated about the so-called ptomaines, that the whole matter may be regarded as being still sub judice; and in the language of Dr. Stevenson (from whose article in *Taylor's Prin. and Prac. of Med. Jurisp.*, 1883, much of the above is taken), "the existence of poisonous cadaveric alkaloids in human viscera, even when putrid and diseased, is, to say the least, very rare." Nevertheless, it cannot be doubted that their alleged existence will be constantly employed by counsel in defending a criminal charged with poisoning with one of the vegetable alkaloids, urging strongly before the jury the possibility that the alleged poisonous alkaloid was in reality one of these spontaneously generated ptomaines. Such a course is stated to have been actually taken at the late Lamson trial, which occurred in London, in 1883.