A TEXTBOOK OF

MEDICAL JURISPRUDENCE AND TOXICOLOGY

BY

RAI BAHADUR JAISING P. MODI, L.R.C.P. & S. (EDIN.), L.R.F.P.S. (GLASGOW).

Member of the Medico-Legal Society (England) ; Honorary Member of the Medico-Legal Society (Bihar) ; Examiner in Medical Jurisprudence in the Universities of Bombay, Lucknow and Patna.

Late Honorary Lecturer of Medical Jurisprudence and Toxicology, Sheth Gordhandas Sundardas Medical College, Bombay ; Formerly Reader in Forensic Medicine in the University of Lucknow ; Professor of Materia Medica and Medical Jurisprudence, King George's Medical College, Lucknow ; Physician, King George's Hospital, Lucknow ; Medico-Legal Officer for the City and the District of Agra and for the City and the District of Lucknow ; Examiner in Medical Jurisprudence in the Osmania University, in the College of Physicians and Surgeons, Bombay and in the State Medical Faculty of the United Provinces ; Lecturer of Medical Jurisprudence, Hygiene, Chemistry and Physics, Medical School, Agra.

Author of "Elements of Hygiene and Public Health."

SIXTH EDITION, REVISED AND ENLARGED THROUGHOUT WITH THREE PLATES (TWO COLOURED) AND ONE HUNDRED AND FIFTY-EIGHT ILLUSTRATIONS IN THE TEXT.

BUTTERWORTH & CO. (INDIA) LTD.,
INCORPORATED IN ENGLAND

PHOENIX BUILDING, GRAHAM ROAD, BALLARD ESTATE, BOMBAY,

AND AT

AVENUE HOUSE, CHOWRINGHEE SQUARE, CALCUTTA.

1940

First Edition 1920
Second Edition 1922
Third Edition 1924
Second Impression 1928
Third Impression 1930
Fourth Edition 1932
Fifth Edition 1936
Sixth Edition 1940

PRINTED AT THE INDIA PRINTING WORKS, 9, BAKEHOUSE LANE, FORT, BOMBAY.

PREFACE TO THE SIXTH EDITION

The appreciation of this work has continued to be such as to necessitate the publication of a new edition within the space of three years. In the preparation of this edition, as before, alterations and additions have been made throughout and new cases have been introduced with a view to illustrate important and interesting medico-legal points. New sections on Pyrogallic Acid (Pyrogallol), Paraldehyde, Trional, Tetronal, Sulphanilamide and War Gases have been inserted, and nineteen new illustrations and two more plates have also been added.

It is my pleasant duty to acknowledge gratefully my indebtedness to Rai Bahadur Dr. K. N. Bagchi, B.Sc., M.B. (Cal.), D.T.M. (Cal. and L'Pool), F.I.C. (London), Chemical Examiner to the Government of Bengal, for kindly revising and rewriting the Chapter on Examination of Blood and Seminal Stains, etc., and for supplying me with a micro-photograph of *ganja* hairs, which is reproduced as Fig. 151. I also express my gratitude to Major S. D. S. Greval, B.Sc., M.D., Ch.B., D.P.H., I.M.S., Imperial Serologist with the Government of India, for his valuable suggestions and his permission to allow Rai Bahadur Dr. K. N. Bagchi to use table No. III published on page 1048 of *the Indian Journal of Medical Research,* April, 1939, and to make use of his notes from the annual reports of his department and to Dr. Gaya Parshad, M.D., of the Prince of Wales Medical College, Patna, for his valuable assistance in connection with the section of Blood Grouping Test.

I wish to offer my sincere thanks to the author, the editor and the publishers of the *Indian Journal of Medical Research* for their kind permission to publish Plate II from their Journal of April, 1939, and particularly to the publishers for a loan of the block for the same plate. I also desire to tender my cordial thanks to Dr. H. S. Mehta, M.B., M.S., F.C.P.S., Professor of Medical Jurisprudence, Grant Medical College, Bombay, for the photographs which are reproduced as Figs. 22, 23, 37, 41, 110, 116, 126, 131 and 132, to Dr. P. V. Gharpure, M.D. (Bombay), D.T.M. & H. (Eng.), Professor of Pathology, Grant Medical College, Bombay, for permission to take photographs of the specimens from the Pathological Museum which are reproduced as Figs. 127, 128 and 158, to Dr. G. B. Sahay, B.Sc., M.B., D.T.M., Lecturer of Forensic Medicine, Prince of Wales Medical College, Patna, for many helpful suggestions and the photographs which are reproduced as Figs. 32 and 52, to the Superintendent of Police, Ahmedabad District, for a photograph which is reproduced as Fig. 88 and to Mr. Harihar Lal Merh of the School of Arts and Crafts, Lucknow, for his kindness in drawing the sketches of Figs. 152 and 153.

I regret very much the inconvenience caused to my readers, especially medical students, owing to the delay in the publication of this edition on account of war exigencies. I am thankful to my publishers in helping me to obtain the requisite printing paper. Lastly, I should like to express my warm appreciation of the splendid co-operation, helpfulness and unfailing courtesy of Mr. V. V. Bambardekar, Manager, India Printing Works.

BOMBAY,
February, 1940.

J. P. MODI.

PREFACE TO THE FIRST EDITION

In accordance with the wishes of the Principal of the Agra Medical School and the Examiner in Medical Jurisprudence, this book has been written chiefly as a text-book for students reading in medical schools and colleges; but in the hope that it may also prove useful to medical and legal practitioners I have tried to incorporate my practical experience as a medical jurist for about fifteen years and as a lecturer in this subject in the Agra Medical School for eleven years and since then in the Lucknow Medical College. I have also given in the form of appendices copies of Government orders in relation to medico-legal work, and certain sections of the Indian Evidence Act, Criminal Procedure Code, Indian Penal Code, Lunacy Act, Poisons Act, etc., which have a direct bearing on legal medicine.

The students of medical schools and colleges while reading for their examinations may conveniently omit the text printed in smaller type which, not being included in their course, is meant only for practitioners.

I must admit my responsibility for the opinions expressed in the text, though in the preparation of this book I have freely consulted various text-books and periodicals, to the authors of which I acknowledge my grateful thanks.

I have also to express sincere thanks to Dr. E. H. Hankin, M.A., Sc.D., Chemical Examiner and Bacteriologist to the Government of United Provinces, for his kindness in revising certain parts of the manuscript and for much valuable assistance and suggestions, especially in the section on Toxicology and to Lieutenant-Colonel E. J. O'Meara, O.B.E., F.R.C.S., I.M.S., Principal, Agra Medical School, who has rendered every assistance to facilitate the completion of the book.

In conclusion I further desire to acknowledge my great indebtedness to Mr. H. M. Rogers of Messrs. Butterworth's for assisting me in reading the proofs.

LUCKNOW,
1920.

J. P. MODI.

MEDICAL JURISPRUDENCE AND TOXICOLOGY

SECTION I

MEDICAL JURISPRUDENCE

CHAPTER I

LEGAL PROCEDURE IN CRIMINAL COURTS

Definition.—Medical Jurisprudence, Forensic Medicine, Legal Medicine and Judicial Medicine are all synonymous terms used to denote that branch of state medicine which treats of the application of the principles and knowledge of medicine to the purposes of law, both civil and criminal. It does not include sanitation, hygiene or public health; both this and medical jurisprudence being distinct branches of state medicine. Medical jurisprudence proper, as distinguished from hygiene, embraces all questions which affect the civil or social rights of individuals and injuries to the person and bring the medical man into contact with the law; while Toxicology deals with the diagnosis, symptoms and treatment of poisons, and the methods of detecting them.

In his professional career the medical man will have frequently to give evidence as a medical jurist in a Court of law to prove the innocence or guilt of his fellow subjects, or to authenticate or disprove a criminal charge of assault, rape or murder brought against an individual. He must remember that as a medical jurist, his responsibility is very great, for very often he will find that his is the only reliable evidence on which depends the liberty or life of a fellow-being. He has, therefore, to acquire the habit of making a careful note of all the facts observed by him, and to learn to draw conclusions correctly and logically after considering in detail the pros and cons of the case, instead of forming hasty judgments.

It is very essential that a medical jurist must have a fair knowledge of all the branches of medical and ancillary sciences taught to a medical student in the course of his studies, inasmuch as he is often required to invoke the aid of these subjects in the elucidation of various problems of medico-legal interest in the Courts of law. He must also be well acquainted with the Government orders, statutes and acts affecting his privileges and obligations in medical practice, and some of the sections of Indian Evidence Act, Criminal Procedure Code and Indian Penal Code relating to the various offences, in the investigation of which his assistance is generally requisitioned.

It has been repeatedly remarked by judges that members of the medical profession are not very careful in drawing up medico-legal reports and consequently cut a very poor figure as expert witnesses, but the experience of medico-legal work in India leads one to believe that this carelessness complained of by the judges is not due to any wilful negligence on the part of medical men, but to want of sufficient data supplied by the Police, and also to their want of practical knowledge of legal procedure in criminal Courts owing to lack of opportunities afforded to students to be present in Courts, when any cases of medico-legal interest are being tried. Again, in Medical Schools and Colleges great stress is laid on the theoretical teaching of this subject, but its practical side is altogether neglected. Medical Jurisprudence is a practical subject, and the class lectures should be illustrated with practical examples, as far as possible, while the students ought to get ample opportunities to examine cases of injury and poisoning, and to witness medico-legal post-mortem examinations.

To obviate this difficulty it is necessary to give first a brief account of the procedure adopted in a legal inquiry and of the criminal Courts of India, before the subject proper is treated.

LEGAL PROCEDURE AT AN INQUEST

Coroner's Inquest.—In the Presidency towns of Calcutta and Bombay, the Coroner with the help of a jury holds inquests or inquiries in cases of sudden, unnatural or suspicious deaths, or in cases of deaths occurring in a jail within the jurisdiction of his Court. The Coroner is authorised to order the post-mortem examination of a body to be made by any medical man, usually the Police Surgeon, whom he summons to his Court to give evidence at the inquest. At such an inquest or inquiry he summons witnesses, takes their evidence on oath, receives evidence on behalf of the accused and then with the help of the jury finds a verdict as to the cause of death. If he finds a verdict of foul play against the accused person, he issues his warrant for the apprehension of such accused person and sends him forthwith to the Magistrate empowered to commit him for trial. Where there is enough evidence of foul play, but the perpetrator of the crime is not identified, the Coroner's jury returns an open verdict against some person or persons unknown, and the matter is held in abeyance, until further inquiry throws more light on the perpetration of the crime.

Police Inquest.—In Mofussil towns, an officer[1] usually of the rank of a Sub-Inspector of Police in charge of a police-station, on receiving information of the accidental or unnatural death of any person, informs immediately the nearest Magistrate of the same, and proceeds to the place where the body of the deceased person is lying and there, in the presence of two or more respectable inhabitants of the neighbourhood, makes an investigation and draws up a report of the apparent cause of death as judged from the appearance and surroundings of the body, describing such wounds, fractures, bruises and other marks of injuries as may be found on the body, and stating in what manner or by what weapon or instrument

1. Vide *Appendix VI, Sec.* 174, *Cr. P. C. In the Presidencies of Madras and Bombay investigations may be made by the head of the village. In the United Provinces of Agra and Oudh head constables specially selected by the Superintendent of Police are empowered by the Local Government to hold inquiries.*

(if any) such marks appear to have been inflicted. The report is then signed by the investigating police officer and by the persons present at the inquest. In a case of suspected foul play or doubt regarding the cause of death, the police officer forwards the dead body for post-mortem examination to the Civil Surgeon of the district or other qualified medical man authorised to hold such examination, furnishing him with the descriptive roll and as full particulars as possible to enable him to find out the probable cause of death, if the state of the weather and the distance admit of its being so forwarded without risk of such putrefaction on the road as would render such examination useless. In order to shirk responsibility the investigating officer is apt to send all dead bodies irrespective of the cause and manner of death to the Civil Surgeon for post-mortem examination. The Civil Surgeon, immediately after holding the post-mortem examination, has to give a statement as to the cause of death in police form No. 289 to the constable accompanying the dead body for communication to the investigating officer, and to send the full report in police form No. 33 (*Vide* Appendix IV) later on to the Superintendent of Police, who forwards it to the Sub-Divisional Officer or Magistrate concerned.

In cases of rape and other cognizable offences the individual is sent by the Sub-Inspector of Police to the Civil Surgeon for medical examination along with his statement recorded in the vernacular. In cases of assault or other non-cognizable crime the injured person may go direct to the Civil Surgeon with the permission of the police officer, if he thinks it necessary, or he may file an affidavit in the Court of a Magistrate who will send him to the Civil Surgeon with a forwarding letter, which the latter officer has to fill up (*Vide* Appendix IV).

DIFFICULTIES IN THE DETECTION OF CRIME

The Civil Surgeon or the Medical Officer, who is always ready to assist the course of justice, finds it, at times, very difficult to arrive at correct conclusions in medico-legal cases for the following reasons :—

(1) The investigating police officer, on hearing of an incident, may not proceed at once to the place of occurrence, being already engaged in investigating another case or for some other reason ; consequently valuable time is lost in obtaining a clue to the crime. As an instance of the dilatory habits of the police I may cite below one of many similar cases.

A Hindu female, about 45 years old, of P. S. Mandiaon, Lucknow District, died in the King George's Hospital at 4 p.m. on the 20th September, 1921. The police were informed immediately of her death, and yet the necessary papers for post-mortem examination were handed over to me at 9 a.m. on the 22nd September, 1921. The result of this unnecessary delay was that the cause of death could not be ascertained owing to decomposition of the body.

(2) The police officer, even if he reaches the place in time, may not touch the dead body and scrutinise it for any marks of violence or identification on account of caste prejudices or some such scruples, but may depend on the illiterate villagers present at the inquest, who may have some motive in concealing the real facts. To illustrate these remarks I cite the following four cases :—

1. In February, 1917, the body of a Mahomedan woman was taken out of a well in Akbar's palace at Fatehpur Sikri, and was sent to the Agra Medical School

Mortuary for post-mortem examination with a police report that she was young, had thirty-two teeth, and her hair was dark; while at the autopsy it was found that the woman was more than 60 years old, had no teeth, all the alveoli had been absorbed, and the plait of the hair of the head that was lying loose owing to decomposition was mostly of a silvery white colour.

2. In a case of double murder which occurred in Chowk, Lucknow, on or about the 10th August, 1923, the age of one of the victims was put down to be 54 years by the police officer holding the inquest, but on inspection on the 12th August, the age was ascertained to be only 14 years, and the age of the other victim who happened to be the mother of the girl (first victim) was forty years.

3. On the 26th August, 1923, a body was removed from a well situated within the jurisdiction of Police Station, Chowk, Lucknow. It was sent to the Medical College Mortuary for post-mortem examination with a report that it was the body of an unkonwn *woman*. At the autopsy it was found to be the body of a tall and well-built *male*. The body was in an advanced state of decomposition but the penis and scrotum were easily recognisable.

4. On the 10th July, 1924, a headless body was found floating in *Nahair* (canal) Ghyasudin Haidar within the jurisdiction of Police Station, Hazaratganj, Lucknow. The body was taken out and forwarded to me for examination with a report that the body was that of a *woman*. On examination I found that the body was that of a Hindu male, as the penis which was distended owing to decomposition was not circumcised.

(3) The report supplied by the police officer is very often quite meagre, as, for want of powers of observation and habits of accuracy, he rushes through an inquest, and omits to note many points, which would, otherwise, help to prove the manner of death, or, for want of the most elementary knowledge of medical jurisprudence, though the subject is taught in police training schools, he mistakes the marks of post-mortem staining for those of violence and describes injuries where there are none or omits to mention them when they are present, and thus unwittingly misleads the medical officer, especially if the body happens to be highly decomposed.

The following cases from my note book would be quite sufficient to substantiate the above remarks:—

1. In October, 1919, the body of a Hindu girl, aged 10 years, was forwarded from Police Station, Kakori, with the station officer's report that the deceased was found with *a wound* at the back of the neck. On examination *eleven incised wounds* were found on the right mandible, chin, and the right side and back of the neck cutting into the third, fourth and fifth cervical vertebræ and the spinal cord. There was also an incised wound along the front of the left thumb.

2. In October, 1921, the body of a Hindu boy, about 12 years old, was brought from Police Station, Goshaingunj, Lucknow District, with a report that the deceased was said to have been beaten with a *lathi* which resulted in his death and that there were *four* marks of injuries on his body. The post-mortem examination was held twenty hours after death when only *one* bruise, ¾" x ½", was found on the lower part of the right shoulder-blade, and death was due to asthenia from chronic malaria.

3. On the 1st August, 1922, a post-mortem examination was held on the body of Budhu, aged 40 years, brought from Police Station, Malihabad, Lucknow District, with a report that the deceased died from five injuries inflicted on the body, *viz.*, one on the right temple, one on the left shoulder, and three on the right side of the back. No external injury could, however, be detected except an abrasion, ½" x ¼" above the left cheek bone. On opening the abdomen the spleen which was enlarged was found ruptured.

4. The body of a Mahomedan male, 48 years old, was forwarded for post-mortem examination on the 18th March, 1923, with a report that the deceased had been killed by dacoits on the night of the 17th March and that there were several bruises on the face, neck and other parts of the body. On examination, no injury was found anywhere on the body except a slight laceration across the left upper eyelid and a small abrasion across the left side of the neck. The stomach and intestines, on the contrary, revealed the signs of irritant poisoning.

5. On the 26th February, 1927, the body of one Kashi Parshad *alias* Kashidin, aged 22 years, was forwarded to the King George's Medical College Mortuary for post-mortem examination with a police report that "after turning the body on all sides the deceased was found to have been shot in the head, and that there was an abrasion on the right arm." On examination I found no gun-shot wound on the head, but detected two contused wounds on the head, and nine contusions, varying from two and a half to four inches by one inch, and five abrasions, varying from one-fourth to half an inch by one-fourth inch, on several parts of the body. There was also an extensive fracture of the vault of the skull and a laceration of the brain.

6. On the 14th March, 1928, the body of one Raja Ram was sent to me for post-mortem examination from Police Station, Alam Bag, Lucknow, with a report that "the left jaw was cut, the left testicle was pierced with some sharp pointed thing and there were bruises round the loin and all over the chest and legs." On examination none of these injuries were found on any of the parts mentioned above. But death was found to be due to strangulation caused by a cord twisted twice round the neck.

(4) The police officer is not to blame in all cases, as he sometimes finds it very difficult to furnish the medical officer with really trustworthy information for his guidance inasmuch as, owing to the unwillingness of the relatives and neighbours to appear before a Magistrate, and give evidence on oath, or, owing to a false notion about the honour of the parties concerned, no one comes forward to volunteer a statement, even if he was present when the crime was committed.

(5) A lot of crime goes undetected owing to the prevailing custom of cremation or burial of bodies soon after death, and that too without any medical certificate. Besides, owing to tanks, lakes, canals, rivers, wells and jungles situated on the outskirts of villages, there is great facility for concealing dead bodies, which are likely to be eaten by dogs, jackals and birds of prey to an extent which will render them difficult of recognition. In October, 1918, I saw the body of a Brahmin male, whose ears had been so nicely gnawed through by rats that they appeared to have been cut away by a knife, unless examined very carefully.

(6) Owing to the climatic conditions in India decomposition of bodies takes place much more rapidly than in Western countries, and this is a frequent occurrence in the hot and rainy seasons owing to the fact that a body has to be carried for long distances in a *dooly* either in a bullock cart or on the heads of *Chamars* before it can be taken to the *sadr* station for autopsy; for, in most districts the Civil Surgeon is the only officer authorised to hold medico-legal post-mortem examinations. As a precaution against decomposition, the police in the United Provinces of Agra and Oudh[1] were instructed to protect the body either by wood-charcoal and ferrous sulphate (*kasis*), phenyle and mustard oil or carbolic dust, but this process does not, in any way, retard putrefaction. On the contrary, it helps to disfigure the external wounds so much that in some

1. Vide *Appendix I, Police Regulations, Sec.* 121.

cases it may be difficult to differentiate their varieties. Hence on my representation to the Inspector-General of Civil Hospitals, U. P., these instructions have been cancelled, and the police are now required to forward the body in a shell in the state in which it was found.[1]

A medical officer must never hesitate to hold a post-mortem examination of a body on the ground of advanced decomposition, although it is, at times, very trying and disgusting to do so. It is very essential to make as thorough an examination as practicable in order to find some clue to the cause and manner of death, especially in a case where there is suspicion of foul play.

On account of districts being spread over a large area, it is impossible to avoid such difficulties. Hence it appears to be desirable for members of the Provincial Subordinate Medical Service in charge of branch dispensaries to be authorised to hold post-mortem examinations, and I do not see any reason why these officers should be debarred from holding autopsies, seeing that they have to go through a four years' course in a recognised medical school and have to pass three stiff examinations before they are qualified to practise in medicine and surgery.

(7) To fabricate a false charge against an enemy it is usual for one party to kill a relation, probably a child or old person, and then to accuse the opposite party of murder. Even on the occurrence of a natural death in the family the relatives make a false report to implicate their enemies, and attribute the death to some previous quarrel or fight that had taken place between the two parties. Sometimes, someone disappears from the scene and after a time a decomposed body found lying on the outskirts of a village or dug up from a grave is claimed as the body of the absconding person, and a false charge of murder is laid at the door of an unwary enemy who, though innocent, not infrequently makes a confession of guilt, possibly to avoid police torture, or when, for other reasons, he finds it difficult to escape the net of conspiracy spread around him.

Illustrative cases.—In the District of Hardoi, a lad, named Chitowri, was missing. About a dozen persons claiming to be eye-witnesses swore before the police that they had seen the boy being strangled by his brother-in-law and other accomplices and thrown into a river. The principal complainant, Ramlal, Chitowri's brother, fainted at the police station while he was describing his brother's alleged murder. The police searched the river and instituted a murder case, and the Magistrate issued warrants for the arrest of the accused. The bottom was knocked out of the case, however, on the accidental discovery by the police of the "dead" lad very much alive in a friend's house, several miles from the village. Ramlal and others were prosecuted for making false complaints to mislead the police.—*The Times of India, July* 15, 1937.

2. A priest suddenly disappeared from his village, and police inquiries led to the arrest of two men, one of whom was the priest's nephew and the other a teacher in the village school. They were charged with murder in the Court of the Dewan of Dharamjaigarh State in Bilaspur District, Central Provinces. The police produced charred bones, believed to be those of the missing priest, who, they alleged, had been taken to the jungle and murdered by the accused with an axe. Prosecution witnesses were called to support the police story and the accused were committed to the Sessions. While they were awaiting judgment, however, the missing priest wrote to the Dewan informing him that he was returning from a pilgrimage he had

1. Vide *G. O.* 4451|*V—VIII—9 dated the 23rd August,* 1924, P. D., *to the Inspector-General of Police, U. P.*

undertaken. Subsequently the priest himself appeared before the Court, and the accused were acquitted. The police sub-inspector who investigated the case and the school teacher who deposed that the axe had actually been taken from him for the murder had been arrested and prosecuted.—*Times of India, Nov.* 23, 1935.

3. One Harbans, son of Tarif Jat of village Dabathua in Meerut district, was sentenced to death under section 302, I.P.C., for having caused the deaths of his two daughters, aged seven years and two years respectively with a *gandasa* for the purpose of implicating his father and two brothers in the crime.

The prosecution story was that Tarif had partitioned his lands amongst his sons, whereafter Harbans accused and his brother Des Raj began to live jointly. On January 19, 1937, a dispute arose between Harbans accused and his father with regard to the payment of canal dues. There was mutual abuse between the two and Harbans knocked down his father who was rescued by his other sons. The accused received a few *lathi* blows from the rescuers. Upon this Harbans became furious and left the place saying that he would get them hanged. Reaching his house he chained the outer door from inside and catching hold of his elder daughter struck her with a *gandasa* which caused her immediate death. Harbans then snatched the younger daughter from the lap of her mother and killed her also with the *gandasa.* Shortly after this the accused went to the police station and reported that his father and his brothers had killed his two daughters. Just as the writing of this report was completed the *chaukidar* of the village arrived and reported that it was generally rumoured that Harbans had himself killed his daughters with a *gandasa* and had come to report. As Tarif could not dare go to the police station lest Harbans should assault him he went straight to the Superintendent of Police and reported the whole incident. The station officer of Police Station Sardhana after making investigation challaned Harbans under section 302, I. P. C.—*The Leader, Sept.* 5, 1937.

4. In a murder trial of accused Pitai *alias* Pitambar and Chhidu for causing the murder of one Mt. Pemo of village Ladhera, District Badaun, the following facts were narrated :—

Mt. Pemo, whose husband died about ten or eleven years ago, used to live in her house with her son, Udho, and his wife, Mt. Reoti. The accused Chhidu was in her service, and used to work at her house. She was suspected of carrying on an illicit intrigue with Chhidu by her husband's brother's son, Govind, Girdhari and others. A few days before her death Girdhari, Hetu, Gangi and others went into her house, and threatened to kill her if she did not sever her connections with Chhidu. At about midday on the 14th April, 1927, she went to the *thana* (Police Station) and made a report against them at the instance of Pitai accused under Section 506, I. P. C. Pitai, accused, whose relations with Girdhari, Hetu and others were highly strained, entered into a conspiracy with the accused Chhidu and formed a plan of making a mark of injury with some sharp-edged instrument on the neck of Mt. Pemo with the object of implicating them in a criminal case. In pursuance of this plan they went into the house of Mt. Pemo at about midnight of the 16th April, 1927, and obtained her consent to their making a mark or injury on her neck with a *gandasa.* Pitai put his *gandasa* on her neck and pressed it with his hands, while Chhidu accused stood close at hand. The *gandasa* went deep into the neck and cut her windpipe and gullet, which resulted in her instantaneous death. Both the accused then left the scene and went back to their houses. Early on the next morning Pitai went to her son, Udho, who was on his threshing floor and told him that his mother was killed by Girdhari, Govind, Hetu and Chunilal and asked him to go to the *thana* and make a report. He was prevailed upon by him to go straight to the *thana* with the village Chowkidar, without even seeing his mother. Being an immature boy of 15 years he fell an easy victim to his persuasions and departed for the *thana* direct from the threshing floor and made a report against the five persons mentioned above. The Sub-Inspector of Police in charge of the *thana* investigated the case and came to the conclusion that Mt. Pemo was murdered by both the accused, and sent them up for trial as a result of investigations. Both were convicted of murder under Section 302, I. P. C., and sentenced to death.—*K. E.* v. *Pitai and Chhidu, Allahabad High Court, Criminal Appeal, No.* 671 *of* 1927.

5. During a quarrel over a young widow one Lachman Ahir and his father, Umedi were beaten with *lathis* and admitted into the hospital at Gunnaur in the District of Badaun. The father and the son were provided with only one bed, there

being no more beds available in the hospital. In order to implicate his enemies and make them responsible for his father's death Lachman got up at night and murdered his father by strangulation.—*Leader, April* 18, 1930.

6. In a village of Daheli Kalan in Etah District one Ram Lal Lodh kept a Brahmin woman as his mistress. This displeased the Brahmins of the village very much. So from time to time there used to be quarrels between the Brahmins and Ram Lal and his brothers, Dina and Nek Ram. On the 28th February, 1929, Dina was drawing water from a well to irrigate his field. His mother, Mt. Bishmia, was directing water in the field. Nek Ram, Ram Lal and Musammat Chameli, the Brahmin woman, were also at the well. In the evening Lala Ram, Sham Lal and some other Brahmins went to the well with *lathis* (clubs) in their hands. Dina thinking that the Brahmins would kill him called his mother and killed her by inflicting wounds on her neck with a *gandasa* (chopper). He then began to cry that Jwala, Lala Ram and other Brahmins had killed her. Dina was, however, sent up for trial on a charge under Section 302, I. P. C. for having committed the murder of his mother, and was found guilty and sentenced to be hanged by the neck till dead.—*King-Emperor* v. *Dina, Allahabad High Court, Cr. Appeal, No.* 527 *of* 1929.

7. One Fauz Khan and his uncle, Roshan Khan owned a field in Dasauli village, which had been under mortgage for nearly twenty or twenty-two years and the mortgage was not redeemed. Roshan Khan was in pecuniary embarrassments in other ways and had many debts to pay. His nephew, Fauz Khan, asked him not to execute any fresh documents to consolidate his debts and advised him upon a new way of paying off all old debts. He asked his uncle to accompany him to their mortgaged field and receive from him two or three severe *lathi* blows so that he could bring a false charge of assault against his creditors to whom he was heavily indebted. Thus, by the threat of a criminal prosecution he could coerce them to hand back the securities relating to his loans and also make them pay him a sum of money by way of compensation. Strangely enough, Roshan Khan fell in with the suggestion and accompanied his nephew to their jointly mortgaged field. The latter then made a determined assault upon his uncle and caused him a number of injuries which soon resulted in death. The plot was, however, found out during the police investigation and Fauz Khan was sentenced to death under Section 302, I. P. C.—*Leader, Dec.* 17, 1930, *p.* 6.

8. One Karim Bux killed by throttling his daughter, Must. Subratan, aged 10 years, on the night between the 16th and 17th March, 1931, placed the corpse near the house of Mangat and brought a false charge of murder against Mangat and Sujan, who were his enemies. He also inflicted two parallel abrasions within the vulvar orifice to the left of the hymen so as to lead to a suspicion of rape. Karim Bux was subsequently convicted of the offence of the murder of his daughter under section 302, I.P.C., and sentenced to death.—*King-Emperor* v. *Karim* or *Karim Bux, Allahabad High Court, Criminal Appeal, No.* 69 *of* 1931.

9. One Imrati, 50 years old, resident of Police Station Mandiaon, District Lucknow, died on the 5th January, 1932, and a report was made at the police station that the deceased was beaten with shoes, kicks and fists during a quarrel seven days before death, and had received three or four marks of external injury on his back, one injury on the head towards the back, and also internal injuries, from the effects of which death occurred. At the post-mortem examination held on the next day, I found no marks of injury on the back or the head or on any other part of the body, but found both the lungs to be pneumonic. Hence I gave my opinion that the death was due to pneumonia and not due to any injuries.

10. On April 4, 1935, the house of Mangal Chunilal in the village of Ratanpur was burgled. Cash and ornaments were stolen. The police patel sent information to the nearest police station. The police sub-inspector at Godhra was communicated with. As he was busy with the investigation of another case, he sent Narsing Chandrasing, one of his constables, for inquiry. The constable reached Ratanpur on the night of April 5. Next morning, he sent for five villagers from Ankadia on suspicion that they were concerned in the burglary. On their arrival they were questioned and, on their denying knowledge of the burglary, they were made to stand in a courtyard with their legs stretched apart. They were asked to bend down, and pebbles were placed on their necks and backs. Those who dropped the pebbles

were severely belaboured with sticks. Two of them were hung from rafters in a room, their legs being tied with ropes. Various other forms of torture were alleged to have been practised on them. The "stinging nettle" was used on Batha Natha, one of the victims. The torture extended over a period of thirty-six hours. They were ultimately set free. When the police had left the village, those who had received injuries went to Godhra for medical treatment. Batha was admitted to the Civil Hospital, as it was found that one of his palms had been crushed under the legs of a cot. The other victims made a petition to the District Superintendent of Police.

The Additional District Superintendent personally conducted the inquiry and arrested Narsing Chandrasing and six others on charges of torture, wrongful confinement and abetment. They were tried by the Sessions Judge, Broach and Panch Mahals, who sentenced Narsing constable to three years' rigorous imprisonment for causing grievous hurt to Batha with a view to extorting a confession, and to one year's rigorous imprisonment for wrongful confinement, the sentences to run concurrently. Virsing, assistant to the police patel was bound over in the sum of Rs. 200 for a year. The other five accused were found not guilty and were acquitted.

Narsing filed an appeal in the High Court of Bombay against his conviction and sentence, but their lordships upheld the conviction and sentence.—*Times of India, March* 5, 1936.

CRIMINAL COURTS AND THEIR POWERS

There are three kinds of courts for the trial of offenders in British India. These are the High Courts, the Courts of Session and the Courts presided over by Magistrates. There are three classes of Magistrates, the first, the second, and the third. There is also the class of the Presidency Magistrates, who are appointed for Presidency towns. First Class Magistrates commit their cases to the Courts of Session and Presidency Magistrates direct to the High Courts. From the class of the Magistrates of first class a Magistrate is appointed to the charge of a district and is called the District Magistrate. A Magistrate of the first or second class when placed in charge of a sub-division is known as the Sub-Divisional Magistrate.

The High Courts are the highest tribunals in the country, and are constituted by Parliamentary Statutes. They are established at Allahabad, Bombay, Calcutta, Lahore, Madras, Patna and Nagpur, while the Chief Court is the highest Court in Oudh, and the Judicial Commissioner's Courts in Sind and North West Frontier Province. These Courts may try any offence and pass any sentence authorised by law.

In these Courts cases are tried before a Judge and a common jury of nine persons. A common jury is composed of persons whose names appear in the general list of those liable to serve as jurors. Medical men are, as a rule, exempted from serving on a jury (*Vide* Appendix VI, Cr. P. C., Section 320). A special jury is composed of persons taken from a special list of jurors prescribed by the High Court. A special jury is empannelled in trials pertaining to offences punishable with death and in any other cases directed by a Judge of the High Court. The verdict of the jury is to be delivered through their foreman to be chosen by the jurors themselves, in the first instance. The unanimous verdict of the jury is to prevail in the High Court, but if the jury are not unanimous and the Judge disagrees with the verdict of the majority he may discharge the jury and order a new trial. The accused person has the right to challenge the jurors individually as they are called.

The Courts of Session are invested with jurisdiction over all kinds of offences. But they can only try cases which have been committed to them by a Magistrate. They may pass any sentence authorised by law, but a sentence of death passed by a Court of Session must be confirmed by the High Court before it can be carried out. An Assistant Sessions Judge may pass any sentence authorised by law, except a sentence of death or of transportation or imprisonment for a term exceeding seven years. The trials before these Courts are ordinarily conducted by the presiding Judge with the assistance of three or four assessors, but the Local Government may, with the previous sanction of the Governor-General-in-Council, by order in the Official Gazette, direct the trial of all offences or of any particular class of offences before any Court of Session in any district to be by jury.[1] In trials by jury before the Court of Session the jury shall be composed of not less than five or more than nine men. In cases where an accused person is charged with an offence that is punishable with death the number of the jury shall, as far as possible, be at the full strength and in no case less than seven.

The Sessions Judge is not bound to accept the opinion of the assessors. If he happens to differ from their opinion, he can pass a sentence without referring the fact to the High Court to which he is subordinate, but if he disagrees with the verdict of the jury, whether it be in favour of the prisoner or against him, he can only submit the record to the High Court which may, on submission being made, pass any order which it deems proper to pass.

The procedure at the trial of an European or Indian British subject is prescribed under Chapter XXXIII of the Code of Criminal Procedure which has been completely recast and remodelled by Act XII of 1923. The provisions of this chapter are only applicable to those cases where the person aggrieved and the person accused, or any of them, are respectively European and Indian British subjects, or where it is deemed expedient owing to the connection with the case of both an European and an Indian British subject that the ordinary mode of trial should be departed from. In such cases the accused is committed to the Court of Session to take his trial in respect of offences, punishable with imprisonment for a term exceeding six months. In petty cases the trial is to take place before a Bench of two Magistrates, one of whom shall be an European and the other an Indian. In the case of disagreement between the members of the special bench the file is to be laid before the Sessions Judge who may pass such order in the case as he considers proper.

The sentences authorised by law are—

(i) death,
(ii) transportation,
(iii) imprisonment (including solitary confinement),
(iv) fine, and
(v) whipping.

Of these, a Magistrate of the first class may pass a sentence of imprisonment not exceeding two years. He is also empowered to direct

1. Vide *Appendix VI, Cr. P. C., Sec.* 269.

that a certain portion of the sentence shall be served out in solitary confinement within the limits laid down by the Indian Penal Code. The power to inflict the punishment of whipping is also vested in a Magistrate of the first class. The term of imprisonment which a second class Magistrate may award is six months, but a Magistrate of the third class cannot pass a sentence of imprisonment exceeding one month. All classes of Magistrates are also authorised to pass a sentence of fine, but a Magistrate of the first class cannot pass a sentence of fine exceeding one thousand rupees, a Magistrate of the second class, one exceeding two hundred rupees, and a Magistrate of the third class, exceeding rupees fifty. Magistrates of the second and the third class are not empowered to pass a sentence of whipping. As regards solitary confinement a Magistrate of the third class is not, but a Magistrate of the second class is, authorised to order that a portion of the sentence of imprisonment should be of the description known as solitary confinement. Twice the amount of imprisonment which a Magistrate is authorised to award may be inflicted by him when passing a sentence for two or more offences at one trial. Of course, the Court of any Magistrate may pass any lawful sentence combining any of the sentences which it is authorised by law to pass.[1]

In the Punjab, Sind, the Central Provinces, Coorg, and Assam and in Oudh and some other parts of the country the Local Government may also confer on certain Magistrates of the first class the powers resembling those of an Assistant Sessions Judge. Such Magistrates can pass any sentence except that of death and of transportation or imprisonment in excess of seven years.

Subpoena.—A subpœna is a document compelling the attendance of a witness in a Court of law under a penalty. When it is served on a witness to give evidence and produce documents before a Court, he must do so punctually. Non-compliance in a civil case may render him liable to an action for damages, and in a criminal case, to fine or imprisonment, unless some reasonable excuse is forthcoming.

In civil cases it is customary to offer a fee, termed *conduct money,* on serving a subpœna. If this is not done, the medical man may ignore the subpœna, if he so desires. In a case where a medical man considers the fee offered at the time of the service of a subpœna less than what he is entitled to, he must ask to have his proper fee paid before being sworn to give evidence, and the presiding Judge will decide the fee to be paid in the circumstances. It is possible that the fee allowed by the Judge may be much less than what he expected. Hence, in order to avoid disappointment, the medical man will be well advised to make sure of his adequate remuneration before giving a report on a case which will eventually lead to litigation, unless it happens to be a case where he is bound in duty to give evidence. It should, however, be remembered that no unreasonable difficulty in the matter of payment of fees should be raised in cases tried in civil Courts under the Workmen's Compensation Act, 1923 as modified up to the first August, 1938.

1. See *Appendix VI for different sections of Cr. P. Code.*

In criminal cases no fee is tendered at the time of serving a subpœna ; the independent medical practitioner may demand a fee at the time of giving professional evidence before taking the oath, but he should not insist on its payment if the presiding officer of the Court is not willing to sanction the sum demanded by him. He must give evidence, or he may find himself in the inconvenient position of being charged with contempt of court. In the case[1] of *K. E.* v. *Ram Narain Sharma* it was held that " in a warrant case ordinarily it is the Government that may pay the expenses of the witnesses both for the crown and the defence and, therefore, it is the duty of the Magistrate to fix the fees of the witnesses. He cannot leave to the parties to negotiate with the witnesses and fix the fees, even in the case of experts. If an expert witness on payment of a reasonable fee fixed by the Magistrate declines to give evidence the Magistrate can compel him to do so." The Government have not laid down a definite scale of fees payable to medical practitioners for attending to give professional evidence in criminal prosecutions, although in framing the rules under Section 544, Criminal Procedure Code, for the payment of the expenses to the witnesses attending before any criminal Court, they have laid down that " witnesses following any profession, such as medicine or law, shall receive a special allowance according to circumstances and custom."[2] It[3] is customary to pay the usual fee of sixteen rupees to a Civil Surgeon and ten rupees to a member of the Provincial Medical Service in charge of a dispensary for giving evidence in a Magistrate's Court as expert witnesses in summons cases under Section 244 (3) of the Criminal Procedure Code[4] and for the defence in warrant cases under Section 257 (2) of the Criminal Procedure Code.[4] When summoned to give evidence in warrant cases medical officers in Government service are not entitled to their fees as experts, but are usually paid two rupees as travelling expenses if they are employed in the town where the Court is held.

When summoned on the same day to attend at two Courts, civil and criminal, the medical witness should attend at the criminal Court, and inform the civil Court of his inability to do so on account of his presence in the criminal Court, which has the preference over it. If summoned to two Courts, both civil or criminal, the witness should first attend at the higher Court. If, however, both Courts happen to be of the same status, he should go to the Court from where he received the summons first, and inform the other Court of the fact, or should attend there after he has done with the first Court.

Oath.—On being called into the witness-box the witness has to take the oath before he gives his evidence. It may be noted here that the medical witness, if he happens to be a gazetted officer, has not to stand in the witness-box, but is usually offered a chair on the dais by the side of the presiding officer. As a rule he is shown special consideration, as the nature of his duties is such that he is not kept in attendance in the

1. *Criminal Law Jour., Nov.*, 1932, *p.* 761 ; *also* vide 38 *Crim. Law Jour.*, 1937, *p.* 133 (*Lahore High Court Crim. Rev. Pet. No.* 521 *of* 1936, *K. E.* v. *Purshotam Das*).

2. Vide *Appendix II, Section* 107.

3. Vide *Appendix II, Sec.* 107, 4 (c).

4. Vide *Appendix VI.*

Court longer than necessary, and his evidence is often interposed out of its turn, so that he is released at the earliest moment.

A Christian in taking the oath has to kiss the "book", but this is not right from a hygienic point of view and he would be well advised to insist on taking it after the Scotch form, raising his right hand above his head and saying in a firm and loud tone:—"I swear by Almighty God, as I shall answer to God at the great Day of Judgment, that I will tell the truth, the whole truth, and nothing but the truth." A non-Christian in taking the oath has to repeat, while standing, "the evidence which I shall give to the Court shall be the truth, the whole truth, and nothing but the truth. So help me God." If a witness wishes to give his evidence on solemn affirmation, he has to say "I solemnly affirm that the evidence which I shall give to the Court shall be the truth, the whole truth, and nothing but the truth."

In whatever form the oath is taken it renders the witness liable to be prosecuted for perjury under Section 193 of the Indian Penal Code,[1] if he fails to state what he knows or believes to be true. His evidence is then recorded in the following manner:—

(1) Examination-in-chief, (2) Cross-examination, (3) Re-examination, (4) Questions put by the Judge, juror, or assessor.

(1) **Examination-in-chief.**—This is the first examination of a witness by the party who calls him. In Government prosecution cases the prosecuting inspector, as a rule, first examines the witness to elicit the principal facts concerning the case. If the witness is summoned by a private party, he is at first examined by the pleader of that party. In this part of the examination leading questions,[2] *i.e.*, questions which suggest their answers, are not allowed except in those cases in which the Judge is satisfied that a witness is hostile, and tries to conceal the true facts. "Did you see X striking Y with a stick on a certain afternoon" is a leading question, as that suggests the answer "yes". It cannot, therefore, be put to the witness. The proper forms of the question in a case of an assault are:—"When did this incident occur? Where were you at the time? What did you notice? and so on." In that case the witness will narrate the whole incident of X striking Y as he saw it.

(2) **Cross-examination.**—This is held by the counsel for the accused who tries to elicit facts, or demonstrate the possibility of theories, not necessarily inconsistent with the evidence the witness has given, but helpful to his own case. In this examination leading questions are permissible, and the witness should be very cautious in answering them. He should not attempt to answer the questions, unless he clearly and completely understands them, as the cross-examiner often tries every possible means to weaken his evidence, thereby showing to the Court that the evidence in question is conflicting and worth nothing.

In some instances cross-examination acts as a double-edged sword, which cuts both ways, *i.e.*, it may damage the defence as much as, nay, sometimes more than, the prosecution, specially if the counsel is not

1. Vide *Appendix VII.*
2. Vide *Appendix V, Sec.* 141, *Indian Evidence Act.*

familiar with medical science, and the witness happens to be well up in his subject, and at the same time honest and straight-forward.

There is no time limit to the cross-examination. It may last for hours or even days, although the presiding officer can always disallow irrelevant questions and cut short the cross-examination. On one occasion I was cross-examined for six days (the examination lasting two hours every afternoon) in a civil case for the recovery of professional fees against a barrister who raised an issue of malpractice. In the end the case was compromised and the barrister had to pay full fees including expenses. On another occasion I was cross-examined for six hours in a case of murder. At last when the defence pleader could not shake me in my statement, he appealingly asked if there was anything in favour of his clients. I replied that I would have informed the Magistrate long time ago if there was anything in their favour, as I was there to assist the administration of justice.

(3) **Re-examination.**—The prosecuting inspector or the counsel, who conducts the examination-in-chief, has the right of re-examining the witness to explain away any discrepancies, that may have occurred during cross-examination; but the witness should not introduce any new subject without the consent of the Judge or the opposing counsel, lest he should be liable to cross-examination on the new point thus introduced.

(4) **Questions put by the Judge, Juror, or Assessor.**—The Judge, juror or assessor may question the witness at any stage to clear up doubtful points.

MEDICAL EVIDENCE

Medical evidence given before a Court of law is of two forms, *viz.*, (1) documentary and (2) oral or parole.

(1) **Documentary Evidence.**—This includes

(a) Medical Certificates.

(b) Medico-legal Reports.

(c) Dying Declarations.

(a) **Medical Certificates.**—These are the simplest forms of documentary evidence, and generally refer to ill-health, unsoundness of mind, death, etc. These certificates should not be given lightly or carelessly, but with a due sense of responsibility for the opinion expressed in them. They are not accepted in a Court of law unless they are granted by a medical practitioner registered under the Provincial Medical Council Act.

In giving a certificate of ill-health a medical man should mention the exact nature of the illness, and preferably should take, at the bottom of the certificate, the thumb-mark impression or signature of the individual to whom it refers.

A medical man should remember that, on the occurrence of the death of a person whom he has been attending during his last illness, he is legally bound to give a certificate stating, " to the best of his knowledge and belief," the cause of death, for which he is not allowed to charge

a fee. The granting of such a certificate is not to be delayed, even if the fee for attending the patient during his lifetime is not paid. The medical man may, subsequently, sue the legal heirs of the deceased for his dues if he so desires. However, he must decline to give a certificate, if he is not sure of the cause of death, or if he has the least suspicion of foul play. In such a case the proper course for him is to report at once to the police authorities, before the body is removed for cremation or burial.

Civil Surgeons and superior medical officers are, sometimes, called upon to countersign death certificates, but they should not do so without inspecting the body. From the non-observance of such a precaution it has, sometimes, happened that a medical officer has been placed in an awkward position in a Court of law.

(b) **Medico-legal Reports.**—These are the documents prepared by the medical officer in obedience to a demand by an authorised police officer or a Magistrate, and are referred to chiefly in criminal cases relating to assault, rape, murder, poisoning, etc. These reports consist of two parts, *viz.*, the facts observed on examination, and the opinion or the inference drawn from the facts.

In order that they may be admitted as exhibits in evidence, these reports should be written up by the medical officer at the time the examination is made or immediately afterwards. They form the chief documents in judicial inquiries, and are likely to pass from the lower to the higher Courts, as well as to be placed in the hands of pleaders ; hence the utmost care should be used in preparing them. No exaggerated terms, superlatives, or epithets expressing one's feelings should be used. For instance, one should never say that " extensive damage to the skull and brain was the result of a particularly brutal, murderous assault," or " the deceased was evidently subjected to a particularly murderous attack in which throttling was also indulged in."

After noting the facts, the opinion should be expressed briefly and to the point. The medical officer must remember that he should always base his opinion on the facts observed by himself. He should not be biased by the statements of others. In drawing conclusions in medico-legal reports he should not depend upon information derived from any other source. However, if his opinion tallies with the information supplied, he should say so in his report.

An injury case should be kept under observation, and the fact notified to the police, if it is not possible to form an opinion immediately after examining it ; a hasty opinion should not be formed, even if pressed by the police.[1]

Articles of clothing, weapons, etc., sent for medical examination should be described with full particulars to facilitate their identification later on in Court. They should be labelled with the differentiating numbers or marks, and returned to the Superintendent of Police or Magistrate in a sealed cover, one's private seal being used ; the signature of the person, usually the police constable, receiving them should be taken. Those articles, which are likely to be sent to the Chemical

1. Vide *Appendix I.*

Examiner, should be kept under lock and key in the custody of the medical officer.

(c) **Dying Declarations.**—A dying declaration is a statement, verbal or written, made by a person since deceased, relating to the cause of his or her death or any of the circumstances of the transaction resulting in death. The medical officer in charge of a hospital or dispensary should at once send for a stipendiary or honorary Magistrate to record the dying declaration of a person, who is likely to die from the effects of criminal violence or other criminal cause.[1] If, in his opinion, there is no time to call a Magistrate, the medical officer should himself record the declaration. It should be recorded in full detail in the vernacular in the identical words of the declarant, in the form of question and answer, and in the presence of respectable witnesses. The accused or his pleader, if present, should be allowed to put questions to the declarant. The declaration should then be read over to the declarant, who should affix his or her signature or mark to it. When concluded, it should be signed by the medical officer recording it, who should also obtain signatures of respectable witnesses. If the declarant becomes unconscious, while the statement is being recorded, the medical officer writing it must record as much information as he has obtained and sign it, and obtain the signatures of the witnesses. If the statement is written by the declarant himself, it should be signed and attested by respectable witnesses. The declaration should then be forwarded in a sealed envelope direct to the District Magistrate or Sub-Divisional Officer concerned. If it can be avoided, the police officer who is engaged in the investigation of the case should not be allowed to be present, when the dying declaration is recorded. No undue influence should also be brought to bear on the declarant, who should be permitted to give his statement without any outside prompting or assistance.

It should be noted that the Calcutta High Court has ruled that in a case where a dying person is unable to speak and can only make signs in answer to questions put to him, the questions and signs put together might properly be regarded as a "verbal statement" made by a person as to the cause of his death within the meaning of Section 32 of the Indian Evidence Act, and are, therefore, admissible in evidence.[2] But statements of witnesses as to what interpretations they put upon the signs made by the declarant are not admissible.[3]

Under the Evidence Act of India, a dying declaration is admissible in Court as evidence whether the person who made it was or was not, at the time when it was made, under expectation of death, but it is essential that the declarant must be in a sound state of mind at the time of making the declaration.[4] It is, therefore, the duty of a medical attendant to certify that his patient is in a fit mental condition to make a statement before it is recorded. A dying declaration is admissible in all criminal and civil cases, where the cause of death is under enquiry.

---

1. *For full details of the procedure* vide *Appendix I, Dying declarations.*

2. *Criminal Reference, No. 49 of* 1921; *Criminal Law Journal, May,* 1924, *Vol. XXV, p.* 529.

3. 38, *Criminal Law Jour.*, 1937, *p.* 281 (*Chandara Sekera alias Alisandiri, Privy Council Appeal from Supreme Court of the Island of Ceylon, Nov.* 12, 1936).

4. Vide *Appendix V, Sec.* 32 (1), *Indian Evidence Act.*

Under English law, a dying declaration is admissible as evidence if the declarant, at the time when the declaration was made, was in full possession of his senses, and believed that he was about to die and that his recovery was impossible, the legal assumption being that an individual would speak nothing but the truth during the last moments of life. The admissibility of a dying declaration is confined to criminal charges of murder or manslaughter only.

(2) **Oral Evidence.**—Oral evidence must in all cases whatever be direct, that is to say, if it refers to a fact which could be seen, heard or perceived by any other sense, or in any other manner, it must be the evidence of a witness who says he saw, heard, or perceived it by that sense or in that manner; if it refers to an opinion or to the grounds on which that opinion is held, it must be the evidence of the person who holds that opinion on those grounds.[1] It is more important than documentary evidence, since a medical man has to prove on oath or affirmation documentary evidence supplied by him to the Court, that it is true and correct and is in his own handwriting; the following are the exceptions:—

1. Dying declaration.
2. Printed opinions of experts.
3. Deposition of a medical witness taken in a lower Court.
4. Chemical Examiner's report.
5. Evidence given by a witness in a previous judicial proceeding.

1. **Dying Declaration.**—This is accepted in Court as legal evidence after the death of the person who made it. Should the person chance to live, his statement ceases to have any legal force as a dying declaration, but it may be relied on under Section 157 of the Indian Evidence Act[2] to corroborate the statement of the complainant when examined in the case.[3]

It should be remembered that a dying declaration does not become invalid, if the declarant dies some days after making the declaration. In the case of *K. E.* v. *Thakura Singh and another*, where one Gurcharan who was severely beaten at about 5 or 5-30 p.m. and had no fewer than eight incised wounds causing a fracture of the skull bone and protrusion of the brain matter was able to make his dying declaration at 9 p.m. on the same day and died after six days, it was held that the fact that the declarant had lingered for some days after making the declaration does not render a dying declaration inadmissible in evidence.[4]

2. **Printed Opinions of Experts.**—Experts' opinions printed in any treatise commonly offered for sale, and the grounds on which such opinions are held, may be proved in Court by the production of such treatise if the author is dead, or cannot be found, or has become incapable of giving evidence, or cannot be called as a witness without an amount of delay or expense which the Court regards as unreasonable.[5]

1. Vide *Appendix V, Section* 60, *Indian Evidence Act.*
2. Vide *Appendix V.*
3. *E.* v. *Rama Sattu.* 4 *Bom., L. R.* 434; *Sarkar, The Law of Evidence in India, Ed. IV, p.* 235.
4. *Criminal Law Journal, Jan.,* 1929, *p.* 65.
5. *Appendix V, Section* 60, *Indian Evidence Act.*

3. **Deposition of a Medical Witness taken in a Lower Court.**—Evidence[1] given by a medical witness in a lower Court is accepted in a higher Court, provided it is recorded and attested by a Magistrate in the presence of the accused, and a certificate signed at the bottom of the deposition in the following form :—"The foregoing deposition was taken in the presence of the accused, who had an opportunity of cross-examining the witness. The deposition was explained to the accused, and was attested by me in the presence of the accused." His evidence without this certificate is not accepted in a higher Court ; hence the medical witness should himself see that the above certificate is written by the Magistrate at the foot of his deposition, specially in those cases which are likely to be sent up for trial before the Sessions Court, if he wants to avoid the trouble of being summoned there. He is, however, liable to be summoned if the Judge wishes to clear some point regarding the subject-matter of his deposition. It must also be noted that after he has given evidence in the Presidency Magistrate's Court, Bombay, in those cases which are triable before the Criminal Sessions of the High Court, the medical witness is required to sign an undertaking to appear before the High Court when summoned.

4. **Chemical Examiner's Report.**—Section 510 of the Code of Criminal Procedure[2] provides that a report signed by any Chemical Examiner or Assistant Chemical Examiner to Government upon any matter or thing duly submitted to him for examination or analysis and report may be admitted in evidence without requiring the officer concerned to be examined in Court to prove the report. In connection with the rule of evidence embodied in this section a bench of the Oudh Chief Court, consisting of Chief Justice Sir Wazir Hasan and Mr. Justice B. N. Srivastava, made the following observations in the case of *K. E.* v. *Mst. Gaya Kunwar* charged under Section 302 of the Indian Penal Code with murdering her husband, Lalta Singh, by administering arsenic to him[3] :—

"We regret to note that what the law intended to be done as a matter of discretion, has been used almost as a general rule according to the practice obtaining in this province. It is to be expected that whenever a Magistrate or a Court of Sessions finds that the report of the Chemical Examiner is inadequate, they should not admit it in evidence unless the officer concerned submits a full and satisfactory report or he has been examined in support of it."

5. **Evidence given by a Witness in a Previous Judicial Proceeding.**—Evidence given by a witness in a previous judicial proceeding is admissible as evidence in a subsequent judicial proceeding, or in a later stage of the same judicial proceeding, when the witness is dead or cannot be found or is incapable of giving evidence, or is kept out of the way by the adverse party, or if his presence cannot be obtained without an amount of delay or expense which, under the circumstances of the case, the Court considers unreasonable, provided that the adverse party in the first proceeding was afforded an opportunity to cross-examine him.[4]

1. *Appendix VI, Section* 509, *Criminal Procedure Code.*
2. Vide *Appendix VI.*
3. *Leader, Nov.* 18, 1933, *p.* 6 ; *also* vide *K. E.* v. *Happu* (*A.I.R.* 1933, *All.*, 837), 36, *Criminal Law Journal*, 1935, *p.* 17.
4. Vide *Appendix V, Section* 33, *Indian Evidence Act.*

KINDS OF WITNESSES

Witnesses are of two kinds : common and expert.

A common witness is one who testifies to the facts observed by himself.

An expert witness[1] is one who, on account of his professional training, is capable of deducing opinions and inferences from the facts observed by himself or noticed by others. Thus, it is apparent that a medical witness is both common and expert. He is a common witness when he gives evidence as regards the variety, size and position of injuries, and is an expert witness when he mentions the nature of these injuries as to whether they were caused during life or after death, whether they were accidental, suicidal or homicidal and so on.

RULES FOR GIVING EVIDENCE

The medical practitioner, when summoned to Court as an expert witness, must remember that he is there to tell the truth, the whole truth and nothing but the truth, and should, therefore, give his evidence irrespective of whether it is likely to lead to conviction or acquittal of the accused.

He should speak slowly, distinctly and audibly to enable the Judge and Counsel to hear him and to take notes of his evidence.

He should use plain and simple language avoiding all technicalities, as the bench and the bar are not expected to be familiar with medical terms. For instance, he should use " bruise ", " bone of the arm ", " shoulder blade ", " collar bone ", " gullet ", " windpipe ", " lining membrane of the stomach ", " bleeding ", " covering of the heart ", etc., for " contusion ", " humerus ", " scapula ", " clavicle ", " œsophagus ", " trachea ", " gastric mucous membrane ", " hæmorrhage ", " pericardium ", etc. It is no use showing his erudition by using these terms ; however, if he cannot help using any medical term he should try to explain it in ordinary language as far as that is possible.

He should avoid long discussions, especially theoretical arguments. His answers should be brief and precise, and in the form of " yes " or " no ". However, by so doing, if he finds that his meaning is not understood, he can explain his answer after obtaining permission from the Judge.

If he does not know or remember any particular point, he should not be ashamed to say so, and must not hazard a guess in a doubtful case.

He should remember that the lawyer has practically unlimited license and latitude in putting questions to the witness in cross-examination, and consequently he should never lose his temper, but should appear cool and dignified, even though questions of an irritable nature be put to him. I may, however, mention that as a medical jurist of twenty-eight years' experience I have had no complaint against lawyers. They have great regard for me, and have shown the greatest amount of courtesy to me at the time of my deposition in Court.

1. Vide *Appendix V, Section* 45, *Indian Evidence Act.*

He may refresh his memory from his own report already forwarded to the Court, but should not do so from his private notes, unless they agreed word for word with the original, were made at the time of, or immediately after, the occurrence of the event, and were written by him or certified to be correct if written by his assistant; besides, he should be prepared to have them put in as exhibits if desired by the Judge or counsel to do so.

He should not quote the opinion of other medical men or quote from text-books concerning the case. He is supposed to express an opinion from his own knowledge and experience.

When counsel quotes a passage from a text-book, and asks the witness whether he agrees with it, he should, before replying, take the book, note the date of its publication, read the paragraph and context, and then state whether he agrees or not; for, counsel usually reads only that portion which is favourable to his case, and the meaning may be completely altered if the whole passage is read. In spite of this precaution he should stick to his opinion if it is still his opinion, and if he finds that it differs from one expressed in the book. To avoid being surprised by such quotations, however, it is advisable to study all the available literature on the subject before giving evidence in Court.

Volunteering of a Statement.—It is said that a witness is not supposed to volunteer a statement in Court, unless called upon to do so. This may be true in the case of a lay witness, but it cannot be so in the case of a medical witness. Even though a medical witness is called by one side to give evidence in Court, he must not forget his duty towards the opposite party of honesty and fair dealing. He must also remember that the Judge regards him not as a medical advocate put forward by one side to establish the case but as an officer of justice to help the Court to elicit the truth. It is, therefore, the duty of a medical witness to state fairly all the medical facts bearing on the case without any reservation. Hence my advice to him is to volunteer statements and suggest questions to Court, especially when he finds that there is danger of justice being miscarried owing to the Court having failed to elicit any important point. Many years ago I had a talk with a Judge of the Judicial Commissioner's Court (now Chief Court) of Oudh about the volunteering of statements by a medical witness in Court, and he agreed with me that I should never hesitate in making such statements. Since then I have, as a rule, followed this practice, which has been appreciated so much by the Magistrates and especially lawyers that the latter very often put only one question during cross-examination, *viz.* "Doctor, please tell us if there is any point in favour of our client (accused)." By following this practice my evidence in a lower Court becomes so complete that on a very rare occasion I am summoned to the Sessions Court.

Professional Secrets.—Under section 126 of the Indian Evidence Act[1] a lawyer can claim privilege and will not at any time be permitted to disclose in Court any communications made to him in the course and for the purpose of his employment as such by his client except with his express consent, but a medical witness cannot claim such privilege as

1. Vide *Appendix V.*

regards professional secrets communicated to him by his patients during their treatment. Nevertheless, he should, on no account, volunteer these secrets, but should divulge them under protest to show his sense of moral duty, when pressed by the Court to do so. Non-compliance with the order of the Court may render him liable to contempt of Court. In certain American and Continental Courts medical men, like priests in the confessional, are privileged not to divulge communications which have been made to them in their professional character by any of their patients.

It should be borne in mind that under English law a medical witness, like any other witness in Court, is absolutely privileged, and no action lies against him in respect of his statement in the witness-box.[1] He is also not compelled to answer questions which have a tendency to expose him (or the wife or husband of the witness) to any criminal charge, for no one is bound to criminate himself and to place himself to peril. Under section 132 of the Indian Evidence Act a witness is not excused from answering any question upon the ground that the answer to such question will criminate, or may tend directly or indirectly to criminate himself, but no such answer which he shall be compelled to give shall subject him to any arrest or prosecution, or be proved against him in any criminal proceeding, except a criminal proceeding for giving false evidence for such answer.[2]

During the trial of the Duchess of Kingston for bigamy held in 1778 before the House of Lords, Mr. Cæsar Hawkins, sergeant-surgeon to the King, was asked if he knew of any marriage between the accused and the Earl of Bristol who was believed to be her husband when she went through the ceremony of marriage with the Duke of Kingston. On his replying that he did not know how far anything that had come before him in a confidential trust in his profession could be disclosed consistent with his professional honour, Lord Mansfield who presided at the trial made the following remarks :—

"If a surgeon was voluntarily to reveal secrets, to be sure he would be guilty of breach of honour and of a great indiscretion ; but to give that information in a Court of justice, which, by the law of the land, he is bound to do, will never be imputed to him as any indiscretion whatever."

In a divorce case[3] before Mr. Justice Horridge the question of professional secrecy arose when the husband's lawyer called a physician who had treated his wife. The physician asked to be relieved from giving evidence on the plea that the Ministry of Health had passed a regulation that "all information obtained in regard to any person treated shall be regarded as confidential." But the Judge said that the Ministry of Health had no power affecting the jurisdiction of the Court ; physicians were subject to the orders of the Court and must disclose what they knew. The physician said he was placed in a difficult position by this ruling. The Judge replied, "I cannot see that you are bound to observe the regulations not to disclose voluntary information you obtained ; but so far as giving information which you are bound to give in assisting the administration of justice it is your duty to give it." The physician then gave the evidence.

In a matrimonial suit[4] where the petitioner claimed a dissolution of the marriage on account of the cruelty and adultery of her husband, an interesting point arose when the three doctors who had treated the husband for two well-known venereal

1. *Dawkins* v. *Rokeby, L. R. 7 H. L. 744, etc.; Sarkar, The Law of Evidence in India, Ed. IV, p. 879.*
2. Vide *Appendix V.*
3. *Jour. Amer. Med. Assoc., July* 23, 1921, *p.* 298 ; *Brit. Med. Jour.,* 1921, *Vol. II, p.* 17.
4. *Leader, Oct.* 27, 1932.

diseases claimed privilege urging that the relationship of doctor and patient was confidential. Mr. Justice Young said that the law on this point was clear. Section 126 of the Evidence Act gave protection to a barrister, attorney, pleader or vakil with regard to communications made to him in the course of his employment as such by a client. There was no protection afforded by the Evidence Act to a doctor as such. When a doctor was called to give evidence he was in the same position as any other person not exempted by the Act. It was his duty to assist the Court in every way possible and to disclose to the Court all the information in his possession relevent to the matter in issue. His lordship, therefore, had to disallow the plea of the doctors that they were entitled to withhold their evidence in this case.

A doctor's protest against the disclosure of a patient's confidence was upheld in a case heard at the Mayor's and City of London Court.[1] Mr. A., a tuberculous patient, under periodical supervision at a London tuberculosis dispensary, was employed as a telephone operator by a City firm and underwent a sanatorium treatment in 1929. Four years later, Mr. B., his deputy telephone operator, developed pulmonary tuberculosis and sued the firm for compensation under the Workmen's Compensation Act, claiming that his disease was the result of using the telephone instrument used by Mr. A. The tuberculosis officer at the dispensary attended by Mr. A. was summoned to go to the Court and to produce the records of the dispensary showing his patient's condition and treatment there. The officer made a protest both on the general ground that the evidence required of him was obtained by him in confidence as a medical practitioner, and on the special ground of a statutory obligation to secrecy. Article 10 of the Public Health (Tuberculosis) Regulations of 1930 directs that "every notification and every document relating to a person notified under these regulations shall be regarded by the medical officer of health and by every person who has access thereto as confidential." Under section 1 (3) of the Public Health Act, 1896, as the witness reminded the Court, refusal to obey the regulations would expose him to a penalty of £100. The Judge upheld the protest, and the tuberculosis officer was absolved from giving evidence.

1. *Lancet*, Oct. 13, 1934, *p.* 835.

CHAPTER II

PERSONAL IDENTITY

Definition.—By identity is meant the determination of the individuality of a person.

The question of the identification of a living person is raised in criminal Courts in connection with absconding soldiers and criminals, or persons accused of assault, rape, murder, etc. It is also frequently raised in civil Courts owing to fraudulent personation practised by people to secure unlawful possession of property or to obtain the prolongation of a lapsed pension. Several cases of mistaken identity have been recorded (see cases at the end of the chapter).

The examination of a person for the purpose of identification should not be undertaken without obtaining his free consent, and at the same time it should be explained to him that the facts noted might go in evidence against him. It should be remembered that the consent given before the police is of no account, and that the law does not oblige any one to submit to examination against his will and thus furnish evidence against himself.

The identification of a dead body is required in cases of fires, explosions, railway accidents, foul play, etc.

In India, the identification of a dead body, sometimes, becomes very difficult owing to its rapid decomposition in the hot season, or through damage caused by wild animals when exposed on the outskirts of villages. However, it is very essential that a dead body should be thoroughly identified and the proof of *corpus delicti* established before a sentence is passed in murder trials, as unclaimed, decomposed bodies or portions of a dead body or even bones are, sometimes, brought forward to support false charges, and in a country like India it is not difficult to obtain such bodies, since villagers are in the habit of cremating bodies very partially, or throwing them into shallow streams, rivulets or canals, or burying them in shallow graves whence carrion feeders may dig them out.

Ram Adhar was convicted of an offence of murder and sentenced to transportation for life by Mr. Asghar Hasan, Additional Sessions Judge of Gonda, with the following remarks :—

"As to the question of sentence the body not having been found in an identifiable condition the mere possibility, though not even the remotest improbability, remains of Ram Narain turning up alive. It would be imprudent on this ground to pass an irrevocable sentence."

During the trial evidence was led in that the accused killed the deceased with an axe. Bones of a dead body were recovered from a tank and a *dhoti* (loin cloth) found nearby was identified to be that of the deceased. In an appeal in the Chief Court of Oudh their lordships held that the identification of the bones by means of an ordinary *dhoti* was far from certain and discarded all the evidence of the eye witnesses and the motive for the murder. As to the portion of the Sessions Judge's judgment that the accused might possibly return alive and that he shrank from passing the death sentence, their lordships said that it was necessary to prove first that a certain person was murdered and, secondly, that the accused person committed the murder.

When first of these essential ingredients was missing, their lordships were of opinion that no conviction could result. In the result their lordships allowed the appeal, set aside the conviction and sentence and directed the acquittal of the accused.—*Leader, Feb.* 2, 1929, *p.* 5.

Cases have, however, occurred where the death sentence was passed even when the body was not forthcoming or was not identified. Sir Samuel Stuart, Kt., Chief Judge, and Mr. Justice Raza of the Chief Court of Oudh state in their judgment that where an offence of murder is proved, the mere fact that the body of the murdered man is not found is not a sufficient reason for not awarding capital sentence.[1]

1. In the case of *K. E.* v. *Nazir,* resident of Kosi Kalan, District Muttra, the body of the victim, Chanda, was not forthcoming, and yet the Sessions Judge relying on the strong evidence against the accused found him guilty of an offence under Section 302, I.P.C., and sentenced him to death. It was alleged that the accused after shooting Chanda in the back carried the body to the neighbouring canal, where it was dismembered with a sword and thrown into the running stream. Some of the articles recovered from the house of the accused were found to be stained with human blood by the Imperial Serologist, who also found such stains on a piece of mud and a piece of bone and flesh found on the canal bank.—*Allahabad High Court, Cr. Appeal, No.* 610 *of* 1923.

2. One Behari had been convicted and sentenced to death by the Sessions Judge of Etah on a charge of having murdered his cousin, Lankush. The prosecution story disclosed that the deceased, at about sunset on the evening of the 3rd of September, 1923, went out of his house, wearing a pair of wooden slippers and an *angaucha* on his head and was carrying a *lota* in one hand and a *lathi* in the other. As Lankush did not return for a long time, his wife and other relations went in search of him but returned disappointed, and the deceased was missed the whole night. The next morning one Musammat Nasiban informed the *Mukhia* that she had heard at night the cry of a man as if he was being murdered and a search was instituted at the spot and some blood marks were discovered, which were being obliterated by Behari accused's mother. The matter was reported to the police and a suspicion at once fell on the accused who bore a long-standing enmity against the deceased, and who handed over a *gandasa* stained with blood and the *lota* belonging to the deceased. The deceased's body was never discovered and it was believed that after murder the body was thrown into the Ganges. The accused also made a confession in which he admitted having killed the deceased. The confession was subsequently retracted and the accused pleaded not guilty. In the appeal preferred by the accused before the High Court their lordships confirmed the sentence.—*Leader, December* 22, 1923.

It will thus be seen that identification may be required of a living person, of a dead body, of fragmentary remains, or of bones only.

The following points are usually noted for the purposes of identification :—

1. Race.
2. Sex.
3. Age.
4. Complexion and features.
5. Hair.
6. Anthropometry.
7. Foot prints.
8. Deformities.
9. Scars.

1. *King-Emperor* v. *Ramnath alias Nattha, Criminal Appeal, No.* 702 *of* 1925; *Criminal Law Journal, April,* 1926, *p.* 460.

10. Tattoo-marks.
11. Occupation marks.
12. Handwriting.
13. Clothes and ornaments.
14. Speech and voice.
15. Gait.
16. Tricks of manner and habit.
17. Mental power, memory and education.
18. Amount of illumination required for identification.

1. RACE

The question of the determination of race or community arises in the identification of unknown or unclaimed dead bodies found in railway carriages, or lying in streets, roads, and fields in the vicinity of villages, or recovered from wells, tanks, canals and rivers. This question also arises in seaport towns, where there is always a conglomeration of races and communities.

The two important communities of Hindus and Mahomedans in India can be recognised by noting the following chief points :—

Hindu Males.	Mahomedan Males.
1. Not circumcised.	1. Circumcised. *N.B.*—Jews are also circumcised.
2. Sacred thread worn over left shoulder in high castes, *dwija* or twice-born.	2. No such sacred thread.
3. Necklace of wooden beads (*Tulsi* or *Rudraksh*) round the neck.	3. No such necklace.
4. Marks on the forehead painted red, yellow (saffron coloured) or white (sandal wood), indicating different religious sects.	4. No such marks; but callosities on the centre of forehead, patella, tuberosity of left tibia and tip of left lateral (external) malleolus owing to special attitudes adopted during prayers.
5. Tuft of hair usually grown longer on middle of back of head below the crown.	5. No such tuft of hair. Head clean shaved, especially among *Bohras*. *N.B.*—I saw a Mahomedan male having a tuft of hair on his head. On inquiry he said that he kept it in imitation of his Hindu friends living near his house in his village (Vide Fig. 1).
6. *Angarakha* or *Mirzai* when worn leaves an opening about 5" or 6" × 1" along the right side of chest showing a brown sun-burnt mark, as nothing else is worn next to skin, especially among villagers.	6. Similar sun-burnt mark on left side of chest on account of the *Angarakha* or *Mirzai* opening on that side.
7. Ear lobules usually pierced.	7. Ear lobules not pierced, but left lobule may be pierced in a few cases.
8. Palms and fingers not stained with *henna*.	8. Palm of left hand and tip of little finger sometimes stained with *henna*.

Hindu Females.	Mahomedan Females.
1. Tattoo-marks between eyebrows, below crease of elbow, on dorsum of hand, and on chest, especially among low castes.	1. No tattoo-marks except among proselytised women.
2. Nose-ring aperture in left nostril; in a few cases in septum as well.	2. Nose-ring aperture in septum only.
3. A few openings along helix for ear-rings.	3. Several openings along helix for ear-rings.
4. Vermilion painted in hair parting on head and red mark on centre of forehead in women having husbands alive.	4. No such paint.
5. Iron-wristlet worn on left wrist in Bengal and ivory *churis* in Bombay and several glass bangles in U. P.	5. No iron-wristlet or *churis* worn but very few glass bangles.
N.B.—These are only worn by women whose husbands are alive.	
6. Head shaved among high class (Brahmin) widows.	6. No shaving of head.
7. Toes wide apart as usually no shoes are worn, but silver ornaments called *Bichhawas* are carried on the toes.	7. Shoe marks probably with corns on toes.
8. Trousers not worn except by Panjabi women.	8. Trousers worn.

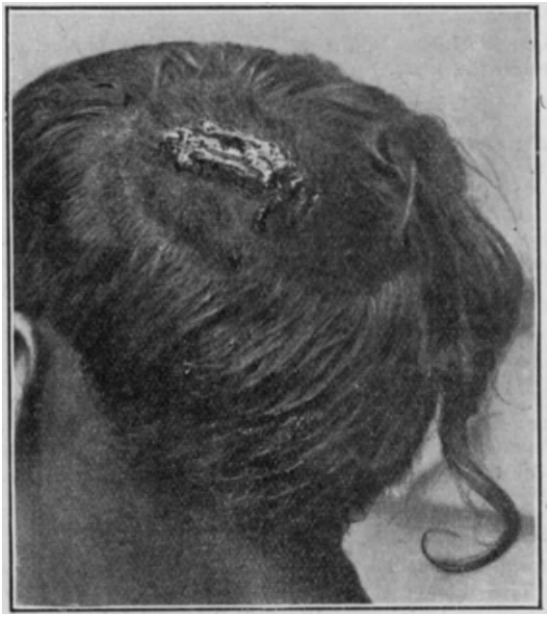


Fig. 1.—A Mahomedan male having a tuft of hair.

Parsi males wear a sacred thread (*Kashti*) round the waist and a *sadra* (muslin *kurta*) on the body. Parsi women in addition tie a *Mathabanu* (white piece of cloth) on the head.

Indian Christian males usually wear pants and short coats and their women put on skirts and cover their head with a *Chadar*.

Race can also be determined from certain differences in the skeleton given below in a tabulated form :—

Race.	Caucasian.	Mongolian.	Negro.
	1. Skull.—rounded.	1. Square.	1. Narrow and elongated.
	2. Forehead.—raised	2. Inclined.	2. Small and compressed.
	3. Face.—small proportionately.	3. Large and flattened, malar bones being prominent.	3. Malar bones and jaws projecting; teeth set obliquely.
	4. Upper extremities.—normal.	4. Small.	4. Long in proportion to body; forearm large in proportion to arm; hands small.
	5. Lower extremities.—normal.	5. Small.	5. Leg large in proportion to thigh; feet wide and flat, heel-bones projecting backwards.

The skull of an Indian is Caucasian with a few negroid characters. This fact must not be taken as evidence of any racial affinity between Indians and the inhabitants of Africa.

The Cephalic Index.—The important test for determining race is the *cephalic index or index of breadth,* which is obtained by multiplying the maximum breadth of the skull measured transversely by 100 and dividing the result by the greatest length measured from before backwards. Skulls having the cephalic index between 70 and 74.5, as observed among the Aborigines and pure Aryans, are called *Dolichocephalic* or long-headed; skulls denoting 75 to 79.9 cephalic index are called *mesati-cephalic* and are characteristic of the Europeans and Chinese, while skulls with 80 to 84.9 cephalic index are termed *brachy-cephalic* or short-headed, as observed in the Mongolian race.

Variations in the Lower End of Femur.—From investigations carried out on two hundred femora from Indian bodies Siddiqi has come to the following conclusions[1] :—

1. In the femora of Indians who, as a rule, adopt a squatting posture, the intercondyloid line is in the majority of cases crossed by a distinct groove for the post-cruciate ligament.

2. The depth of intercondyloid fossa is greater in the femora of Indians (squatters) than it is in those of Europeans (non-squatters), and

1. *The Journal of Anatomy, April,* 1934, *p.* 331.

that its cause is due to the pressure by the post-cruciate ligament when the joint is fully flexed as in squatting.

3. The ratio between the depth of the intercondyloid fossa and the height of the articular surface is such that in non-squatters (Europeans) it tends to rise above and in squatters (Indians) to fall below 3.3.

2. SEX

The determination of sex becomes necessary in cases relating to heirship, disposal of property, marriage, education, impotence, rape and allied subjects.

It is easy to determine the sex in normal cases from external inspection only, but it becomes difficult in malformed individuals called hermaphrodites, concealed sex, advanced decomposed bodies, and in the skeleton.

In some cases it may be impossible to affirm the sex during childhood owing to the non-descent of the testicles or such other reason. If so, a positive answer must be delayed until the child reaches puberty, when characters peculiar to each sex usually arise. The distinguishing characters essential to each sex are tabulated below :—

Male.	Female.
1. A testicle secreting semen; the prostate, vesiculæ seminalis, penis, etc., being mere appendages.	1. A functioning ovary with periodic discharges of blood; the uterus, Fallopian tubes and vagina being appendages only. In the absence of an ovary, the presence of uterus or the opening of a cul-de-sac below the mouth of the urethra and in front of the rectum.
Additional confirmatory signs	
2. Build, generally larger.	2. Build, generally smaller.
3. Shoulders, broader than hips.	3. Hips, broader than shoulders.
4. Pomum Adami, developed and prominent.	4. Pomum Adami, not developed.
5. Breasts, not developed, though may be so very rarely.	5. Breasts, developed.
6. Lineæ Albicantes, not to be found except in very stout males or in the case of previously distended abdomen by disease.	6. Lineæ Albicantes, found on abdomen, breasts and buttocks, as well as on thighs indicating previous pregnancy.
7. Pubic hair, thick and extending upwards to the navel.	7. Pubic hair, horizontal and covering mons veneris only.
8. Hair over the chest, more or less present.	8. Hair over the chest, absent.

Hermaphrodites.—These are individuals in whom the essential parts of the generative organs of both sexes are included. This abnormality occurs from a faulty development of the sexual organs in the embryo and is known as *hermaphroditism.* It is classified as *spurious* or *false* and *true.*

Spurious or False Hermaphroditism.—In this class of hermaphroditism there is a malformation of the external genital organs only. Thus, in *Androgyni* (womanly men) the penis may be very small and perhaps hypospadic or epispadic, the scrotum may be cleft in the middle, and the testicles may be undescended. There may also be a development of the breasts with feminine contour.

Upendranath Mandal[1] describes the case of a spurious hermaphrodite, who was brought up as a female child up to the age of 9 years when she was married to a boy, 14 years old. Upto the age of 15 years menstruation had not started and the breasts had not also developed. Hence the female inmates of the house grew suspicious and found the presence of a rudimentary penis and two small testicles. She was then treated as a male, her long hairs of the head were cropped, and he was married at the age of 18 years to a girl of 12 years. The doctor examined him when he was 21 years old and found that he had a small penis, about ¾" long, a urethra opening on its under surface and a cleft scrotum with a small testicle on each side. Both the testicles could be pushed upwards through the inguinal canal up to the internal abdominal ring. Semen and urine came through the slit-like urethral opening which was about an inch in length from above downwards. The slit-like urethral opening and the ununited scrotum simulated the external female genital organs, especially when both the testicles were pushed upwards towards the inguinal canal. There was no moustache and no beard except two to four hairs in the centre of the chin.

In *Androgynæ* (manly women) the clitoris may be thickened and elongated, and the labia majora may be united. The uterus or ovaries may be prolapsed and the body may show a hirsute appearance in some cases.

True Hermaphroditism.—True hermaphroditism is that in which one or more portions of the generative apparatus of both sexes are represented in the same individual. In old times there was a false belief that this condition owing to the union of the sexes in the same individual led to self-production ; but such a condition does not exist. Hermaphrodites are usually impotent and sterile.

True hermaphroditism has been classified into three distinct types, *viz.*, lateral, transverse, and vertical or double.

Lateral.—In this type a testicle is developed on one side, and an ovary on the other.

Transverse.—In this type male organs are developed externally and female organs internally, or the reverse.

John W. Heekes[2] reports the case of a true hermaphrodite, who was a woman, aged about 32 years, well developed, of average height and with normal breasts. She had externally the external genitals quite normal, the vagina being about three inches long, but internally she had no cervix or uterus. She had never menstruated. She was operated for appendicitis and was found internally to contain two white structures which were found to be normal testicles, and the tubules were vestigial Fallopian tubes without cilia.

Vertical or Double.—In this type various combinations of the generative organs of both sexes are seen, *e.g.*—

1. Ovaries with combined male and female passages.
2. Testicles with combined male and female passages.
3. Ovaries and testicles co-existing on one or both sides.

1. *Indian Journal of Medicine, Aug.*, 1931, *p.* 189.
2. *Brit. Med. Jour., July* 31, 1937, *p.* 243.

In the case of a doubtful sex a very thorough examination should be made bimanually as well as by rectal palpation and an opinion should be given from the anatomical condition and the habit of the individual predominating most. In this connection it may be mentioned that Frank and Goldberger[1] have devised a test which permits of the determination of sex owing to the presence in the circulating blood of the female sex hormone which can produce puberty, if injected into immature animals, such as mice. In the case of a small child, however, an opinion may be deferred until puberty, when the sex may be determined from emission of semen or menstruation.

Concealed Sex.—Criminals may try to conceal their sex by dress or by some other means to avoid detection, or some persons, *e.g.*, eunuchs, dancers, etc., may do so from moral obliquity. These cases do not present any difficulty if they are stripped naked. But the most remarkable case of concealed sex is that of Colonel James Barry,[2] Army Surgeon and Inspector-General of Hospitals, who successfully practised deception on the public until death at the age of eighty, when it was found that *he* was a female. A case[3] is also recorded in which Mrs. Smith, wife of an officer of the Australian Forces, masqueraded at Andover in Sussex as a man without being found out for six years, and but for the institution of bankruptcy proceedings which led to the discovery of her sex it is possible that she might have kept up the deception until her death. During this period she adopted the name of Colonel Victor Barker, lived with another woman as her "husband," opened an antique shop, played for the Andover Cricket Club, sang as a tenor in the church choir, strode the streets in khaki shorts with a shirt open at the neck and dropped in at the "Star and Garter" for *chota pegs.*

Individuals are mentioned possessing features representing no sex (sexlessness), but such a condition is very rare indeed. Tardieu has reported the case of a person who was married as a woman at the age of twenty-five. This person had never menstruated and had neither breasts, vagina, uterus nor ovaries. There was also an absence of the penis and testicles.[4]

Decomposed Bodies.—In the absence of all other evidence the presence of the uterus, which, if unimpregnated, resists putrefaction for a considerable time, will decide the sex. In the case of mutilated remains the determination of the sex is only likely to be accurate from hairiness on the head, face, chest or pubes, development of the breasts and lineæ albicantes, if any of these parts are available.

In the case of a head and two upper extremities, taken out of a well in Police Station, Goshaingunj, Lucknow District, in December, 1921, the sex was determined to be male from the presence of dark hair about 4" to 5" long on the crown of the head, about 1" long on the temples, and very short dark hair on the chin and face even though these parts had been badly decomposed. In another case the male sex was determined from the growth of dark hair on the skin of the chest and on the abdominal skin below the navel, when a decomposed trunk without any organs and a few bones had been sent for examination from Police Station, Mandiaon, District Lucknow. These were afterwards identified to be the remains of one Ahir, a male, by the *dhoti* left round the trunk.

1. *Journ. Amer. Med. Assoc., Jan.* 14, 1928, *p.* 106.
2. *Taylor's Principles and Practice of Med. Juris., Ed. IX, Vol. I, p.* 135.
3. *Illustrated Weekly of India, March* 1, 1929.
4. *Taylor's Principles and Practice of Med. Juris., Ed. IX, Vol. I, p.* 141.

Skeleton.—It should be borne in mind that it is not possible to determine the sex from a skeleton with a full amount of certainty in individuals who have not reached puberty, seeing that the sexual characteristics of the bones do not begin to manifest themselves until this period is attained.

The bones of the adult female are usually smaller and lighter than those of the male, and have less marked ridges and processes for muscular attachments.

The adult female skull is, as a rule, lighter and smaller, its cranial capacity being about ten per cent. less than that of the male. The glabella, zygomatic and superciliary arches, mastoid processes, and the occipital protuberance are less prominent. The facial bones are more delicate and smaller, especially the maxillæ, mandible and the teeth contained in them.

The female thorax is shorter and wider than the male. The sternum is shorter and its upper margin on a level with the lower part of the body of the third thoracic (dorsal) vertebra, while in the male it is on a level with the lower part of the body of the second. The ribs are thinner and have a greater curvature, and the costal arches are larger.

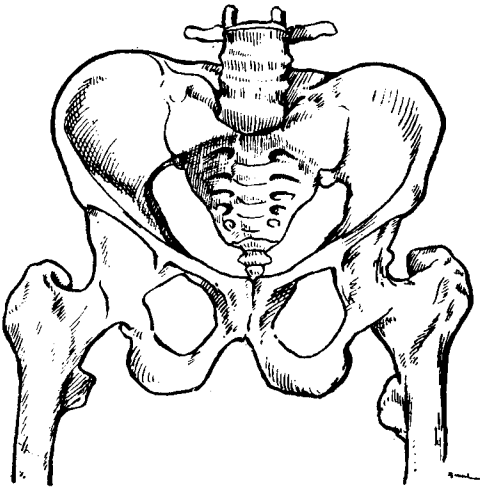


Fig. 2.—The Male Pelvis.

The pelvis affords the best marked and most reliable characteristics for distinguishing the sex. The female pelvic cavity is shallower and wider, the sacrum is shorter and wider, and its upper part is less curved ; the obturator foramina are triangular in shape and smaller in size than in the male. The inferior aperture is larger and the coccyx more mobile. The pre-auricular sulci are more commonly present and better marked. The ilia are less

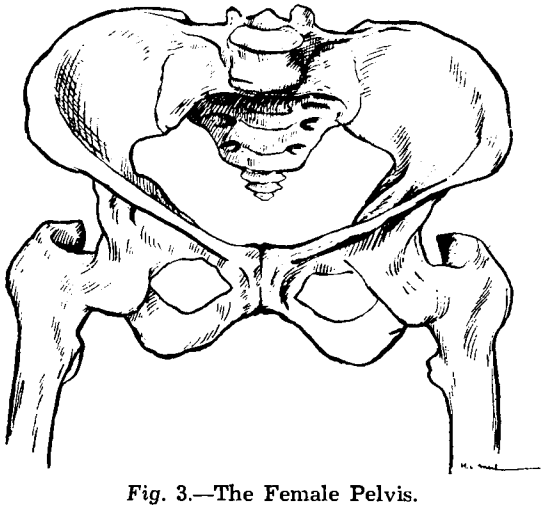


Fig. 3.—The Female Pelvis.

sloped, the anterior iliac spines are more widely separated and the great sciatic notches are much wider, forming almost a right angle. The superior aperture of the lesser pelvis is larger, more nearly circular, and its obliquity is greater. The ischial tuberosities and the acetabula are wider apart, and the former are more everted. The pubic symphysis is less deep, and the pubic arch is wider and more rounded than in the male, where it forms an angle rather than an arch.

The neck of the femur forms almost a right angle with its shaft in the female, and an obtuse angle in the male.

3. AGE

The principal means which enable one to form a fairly accurate opinion about the age of an individual are teeth, height and weight, ossification of bones and minor signs.

Teeth.—The estimation of age from the teeth with some amount of certainty is only possible upto 22 to 25 years of age; beyond that it is merely guess work.

There are two sets of teeth called *temporary* and *permanent.*

The temporary teeth are also called deciduous or milkteeth and are twenty in number: four incisors, two canines, and four molars in each jaw. They appear in infancy, are shed in the course of a few years and are replaced by the permanent teeth, which are twirty-two in number, consisting of four incisors, two canines, four premolars or bicuspids, and six molars in each jaw.

The following table shows the average periods of eruption of the temporary and permanent teeth.

—	Temporary.	Permanent.
Central Incisors	....	7th to 8th year.
Lower	6th to 8th month.	
Upper	8th to 10th month.	
Lateral Incisors	....	8th to 9th year.
Lower	10th to 12th month.	
Upper	7th to 9th month.	
Canines	17th to 18th month.	11th to 12th year.
Anterior Premolars or First Bicuspids	Absent.	9th to 10th year.
		10th to 12th year.
Posterior Premolars or Second Bicuspids	Absent.	6th to 7th year.
First Molars	12th to 14th month.	12th to 14th year.[1]
Second Molars	22nd to 24th month.	
Third Molars or Wisdom Teeth	Absent.	17th to 25th year.

General Characteristics of Teeth.—In some rare cases the temporary teeth may either appear abnormally early or be present at birth, a

1. *In one case I found that a boy, aged 15, had no second molar teeth. Dr. Sahay of Patna reported to me that he examined a police recruit, aged 21, who had only twenty-four teeth. None of his second molars had erupted. He was well-built and had no abnormality or deformity.*

condition probably due to hereditary syphilis. P. S. Khosla[1] reports the case of a Hindu male child in whom he found the lower central incisor coming out of gums, when he saw him the next day after his birth. S. N. Chatterji[2] also reports the case of a male child who was born with two well-marked incisor teeth in the lower jaw.

In certain diseases affecting the bony skeleton, *e.g.*, rickets, the eruption of the temporary teeth may be delayed for a considerable time. Herbert Gregory[3] reports the case of a man who had never had any teeth till the age of 54. In that year the upper right canine appeared, but disappeared a year later. It should also be remembered that some of the temporary teeth or all of them may be retained till advanced adult age. Professor Glaister[4] has seen them in a man over sixty years in whose upper jaw especially there was a double row of teeth.

The permanent teeth erupt in the lower jaw but are not always regular in their appearance. In a few cases they may appear earlier than the average period. The time of eruption of the third molar teeth or wisdom teeth is more uncertain. After the eruption of the second molar teeth the body of the jaw grows posteriorly and the ramus is elongated to make room for the appearance of the third molar teeth. Hence, during the examination of a minor for determining his age a note should always be made as to whether there was a space in the jaw behind the second molar teeth, if the third molars were absent. These teeth are usually cut between 17 and 25 years of age, but I have seen the left lower third molar tooth in a Mahomedan boy and a Mahomedan girl, aged 14 years, respectively, and in a Hindu boy of 15 years. I have also found the lower third molar teeth in a Hindu boy of 16 years. Lall and Townsend[5] found one third molar tooth between the ages of 15 and 16 years, two third molar teeth between the ages of 16 and 17 years and three third molar teeth between 17 and 18 years of age in a majority of 125 girls examined in Lucknow. On the other hand, in some rare cases the third molar teeth may not appear till adult age is advanced. I have seen a man of 40 and another of 48 who had not cut their third molar teeth. K. Venkat Rao[6] reports the case of a man, who, at the age of 54 years, cut his third molar tooth in the right upper jaw, while it was still absent in the left upper jaw. Both the third molar teeth were present in the lower jaw.

The notched and stunted upper central incisors of inherited syphilis, known as Hutchinson's teeth, are always permanent. In old age the teeth have either fallen out or the crowns are worn away to the sockets. Sometimes a smooth rounded surface is to be seen in place of alveolar cavities. Cases have been recorded in which a third eruption of the teeth occurred in advanced age.[7] For instance, a Mahomedan male who died in Bombay at the age of 117 years, was reported to have cut a third set of teeth on completing his hundredth year. Although not of the normal

1. *Indian Med. Gaz., Feb.*, 1927, *p.* 68.
2. *Indian Medical Gazette, January*, 1932, *p.* 57.
3. *Brit. Med. Jour.*, 1902, *Vol. I, p.* 1660.
4. *Med. Juris. and Tox., Ed. VI, p.* 84.
5. *Ind. Med. Gaz., Oct.*, 1939, *p.* 614.
6. *Indian Medical Gazette, March*, 1932, *p.* 177.
7. *Collis Barry, Leg. Med. and Tox., Vol. II, p.* 34.

size the new teeth functioned well and were responsible for giving him the look of a much younger man.[1]

The crowns of the temporary teeth are of a white china-like colour and are marked with a ridge or thick edge at their junction with the fangs; while the crowns of the permanent teeth are ivory white and have no ridge. The anterior temporary teeth are vertical, and the permanent teeth are usually inclined a little forward.[2]

Height and Weight.—A full-term child at birth is, on an average, 19 to 20 inches in length and 6 to 7 pounds in weight. It is generally 24 inches in length at the age of the sixth month and 27 inches at the end of the first year. At the end of the fourth year it is, on an average, double its length at birth. If the health and nutrition are maintained, the child gains in weight nearly one pound a month during the first year, so that it is generally double its birth-weight at the end of the fifth month, and treble its birth-weight at the end of the first year. The following table[3] shows the average height and weight according to ages:—

Males.			Females.		
Age.	Height in inches.	Weight in pounds.	Age.	Height in inches.	Weight in pounds.
1	33.50	18.8	1	27.5	..
2	33.70	32.5	2	32.33	25.3
3	36.82	34.0	3	36.23	31.6
4	38.46	37.3	4	38.26	36.1
5	41.03	39.9	5	40.55	39.2
6	44.00	44.4	6	42.88	41.7
7	45.97	49.7	7	44.45	47.5
8	47.05	54.9	8	46.60	52.1
9	49.70	60.4	9	48.73	55.5
10	51.84	67.5	10	51.05	62.0
11	53.50	72.0	11	53.10	68.1
12	54.99	76.7	12	55.66	76.4
13	56.91	82.6	13	57.77	87.2
14	59.33	92.0	14	59.80	96.7
15	62.24	102.7	15	60.93	106.3
16	64.31	119.0	16	61.75	113.1
17	66.24	130.9	17	62.52	115.5
18	66.96	137.4	18	62.44	121.1
19	67.29	139.6	19	62.75	123.8
20	67.52	143.3	20	62.98	123.4
21	67.63	145.2	21	63.03	121.8
22	67.68	146.9	22	62.87	123.4
23	67.48	147.8	23	63.01	124.1
24	67.73	148.0	24	62.70	120.8
25-30	67.80	152.3	25-30	62.02	120.0
30-35	68.00	159.8	30-35	61.15	120.8

The height and weight according to age vary so much in individuals that they are of no use in estimating the age in medico-legal cases. They

1. *Times of India, Aug.* 17, 1936, *p.* 10.
2. *Powell, Ind. Med. Gaz., June,* 1902, *p.* 233.
3. *Report of The Brit. Anthropological Committee,* 1883; *Glaister, Med. Juris. and Tox., Ed. V, p.* 270.

may be of service in ascertaining the normal nutrition of individuals. Mention must also be made of the fact that no data are available in India for determining the age from the height and weight.

Ossification of Bones.—This is an important additional sign for determining the age until ossification is completed, for skiagraphy has now made it possible to determine even in living persons the extent of ossification, and the union of epiphyses in bones. Owing to the variations in climatic, dietetic, hereditary and other factors affecting the people of the different provinces of India it cannot be reasonably expected to formulate a uniform standard for the determination of age of the union of epiphyses for the whole of India. However, from investigations carried out in certain provinces it has been concluded that the age at which the union of epiphyses takes place in Indians is about 2 to 3 years in advance of the age incidence in Europeans and that the epiphysial union occurs in females somewhat earlier than in males.

In ascertaining the age of young persons radiograms of both sides of the body should be taken, and an opinion should be given according to the following table, but it must be remembered that too much reliance should not be placed on this table as it merely indicates an average and is likely to vary in individual cases even of the same province owing to the eccentricities of development. (For *Table see* pp. 36 to 39.)

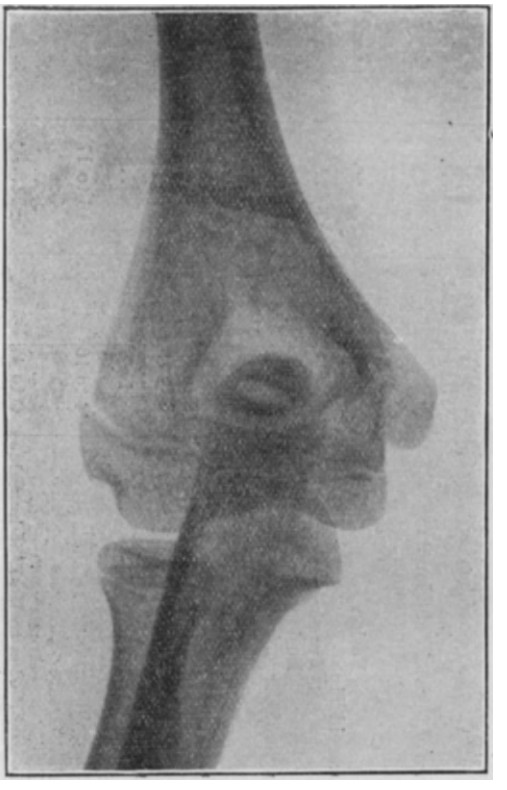


Fig. 4.—X-Ray Photograph of the elbow of a girl, aged 15 years: Lower end of humerus and upper ends of radius and ulna not united with shafts.

Table showing the Age in years of the Appearance and Fusion of some of the Epiphyses as observed by different Authors.

	Galstaun (Bengalees)		Basu & Basu (Bengalee Hindu Females)	Hepworth (Punjabis)	Lall & Townsend (Females of United Provinces)	Lall & Nat (Males of United Provinces)	Pillai (Madrasis)	Flecker (Australians)		Davies & Parsons (Englanders)
	Females	Males						Females	Males	
Clavicle (Sternal End)										
Appearance	14 to 16	15 to 19	..	..	..	..	..	21	21	17
Fusion	20	22	..	..	..	..	..	22	22	25
Base of Coracoid of Scapula										
Appearance	2½ Months	2½ Months	..	..	..	..	..	..	..	1
Fusion	2½	2½	..	..	..	..	..	..	..	..
Coracoid Tip										
Appearance	10 to 11	10 to 11	..	..	..	..	13	..	..	1
Fusion	16	16	..	..	..	..	14	..	..	16
Angle of Coracoid										
Appearance	8 to 10	10 to 14	..	..	..	..	..	..	..	..
Fusion	16	17 to 18	..	..	..	..	..	..	..	..
Acromion										
Appearance	12 to 14	14 to 17	..	..	..	..	13 to 14	14	15	..
Fusion	13 to 16	14 to 19	..	..	..	..	18	17	17	..
Humerus : Head										
Appearance	1	1	..	..	..	..	..	Before Birth	Before Birth	At Birth
Fusion to shaft	14 to 16	14 to 18	16 to 17	17 to 18	..	..	14 to 17	17	19	19 to 21
Greater Tubercle										
Appearance	..	..	..	..	..	..	..	..	..	..
Fusion to Head of humerus	2 to 4	2 to 4	..	..	..	..	..	4	5	..
Fusion to lesser Tubercle	5 to 7	5 to 7	..	..	..	..	..	..	..	5
Humerus : Trochlea										
Appearance	10	11	..	..	..	..	..	9	10	10
Fusiou to Capitulum	9 to 13	11 to 15	12 to 13	..	..	..	..	..	13	..
Lateral Epicondyle										
Appearance	10	12	..	..	..	..	..	11	12	12
Fusion to Capitulum	10 to 12	11 to 16	12 to 13	14 to 15	..	..	13 to 14	13	13	16

—	Galstaun (Bengalees) Females	Galstaun (Bengalees) Males	Basu & Basu (Bengalee Hindu Females)	Hepworth (Punjabis)	Lall & Townsend (Females of United Provinces)	Lall & Nat (Males of United Provinces)	Pillai (Madrasis)	Flecker (Australians) Females	Flecker (Australians) Males	Davies & Parsons (Englanders)
Medial Epicondyle										
Appearance	5	7	..	..	..	..	..	..	6	5
Fusion to shaft (Distal humeral epiphysis)	14	16	13 to 14	14½	14 to 15	15 to 17	14 to 17	15	16	20
Head of Radius										
Appearance	6	8	..	..	..	..	..	4	5	4
Fusion	14	16	13 to 14	14 to 15	16	17	14 to 17	14	16	15–16
Distal End of Radius										
Appearance	1	1	..	..	..	..	..	1	..	10 to 12 Months
Fusion	16½	18	16 to 17	16 to 17	19	19	14 to 18	18	19	19–20
Olecranon										
Appearance	9 to 12	11 to 13	..	..	..	..	..	8	10	..
Fusion	15	17	13 to 14	..	15	16 or earlier	14 to 16	14	16	17
Distal End of Ulna										
Appearance	8 to 10	10 to 11	..	..	..	..	..	..	6	7 to 8
Fusion	17	18	16 to 17	..	19	19	14 to 18	17	19	20
Crest of Ilium										
Appearance	14	17	..	..	..	..	..	14	..	18
Fusion	17 to 19	19 to 20	..	..	..	..	14 to 18	15 to 16	18	23
Ischium and Pubis										
Fusion	8½	8½	..	..	..	..	..	7	9	8
Acetabulum										
Disappearance of Triradiate Cartilage	14	15 to 16	..	..	..	..	11 to 14	13	15	16
Ischial Tuberosity										
Appearance	14 to 16	16 to 18	..	..	..	..	..	16	19	..
Fusion	20	20	..	..	..	..	..	..	20	..
Head of Femur										
Appearance	1	1	..	..	..	..	..	1	1	½ to 1
Fusion	14 to 15	16 to 17	13 to 14	15½ to 17	..	..	14 to 15	14	17	19–20
Great Trochanter										
Appearance	..	..	..	..	..	..	..	5	5	4
Fusion	14	17	14	16 to 17	..	..	14 to 17	14	17	18 to 19
Lesser Trochanter										
Appearance	..	..	..	..	..	..	..	14	..	12 to 14
Fusion	15 to 17	15 to 17	13	..	..	..	14 to 17	..	..	18

—	Galstaun (Bengalees) Females	Galstaun (Bengalees) Males	Basu & Basu (Bengalee Hindu Females)	Hepworth (Punjabis)	Lall & Townsend (Females of United Provinces)	Lall & Nat (Males of United Provinces)	Pillai (Madrasis)	Flecker (Australians) Females	Flecker (Australians) Males	Davies & Parsons (Englanders)
Distal End of Femur—Appearance	Before Birth	Before Birth	..	..	..	..	..	Before Birth	Before Birth	Before Birth
Fusion	14 to 17	14 to 17	16	16½ to 17½	..	..	14 to 17	17	19	19
Proximal End of Tibia—Appearance	Shortly before or after Birth	Shortly before or after Birth	..	..	..	..	..	Before Birth	Before Birth	Before Birth
Fusion	14 to 15	14 to 17	15 to 16	16½ to 17½	..	..	14 to 17	..	18	19 to 20
Distal End of Tibia										
Appearance	..	..	..	..	..	..	..	1	..	½ to 1
Fusion	14.1 to 14.4	16	14 to 15	16 to 17½	..	..	14 to 17	14	17	17 to 18
Proximal End of Fibula										
Appearance	..	..	..	..	..	..	..	3	5	5 to 6
Fusion	14 to 16	14 to 16	16 to 17	16½ to 17½	..	..	14 to 17	17	19	18 to 19
Distal End of Fibula										
Appearance	..	..	..	..	..	..	..	1	2	2
Fusion	13 to 15	14 to 16	15	17 to 18	..	..	14 to 17	14	17	18
Patella										
Appearance	4	3 to 7	..	..	..	..	..	3	5	3
Fusion	..	..	..	..	..	..	..	..	..	..
Carpal Bones										
Capitate (Os magnum) Appearance	1/2	1/2	..	..	..	..	..	1/2	1/2	3 to 5 Months
Hamate (Unciform) Appearance	8 to 14 Months	8 to 14 Months	..	..	..	..	..	6 to 10 Months	6 to 10 Months	4 to 6 Months
Triquestrum (Cuneiform) Appearance	2 to 3	3 to 4	..	..	..	..	..	1½	3	2 to 4
Lunate (Semilunar) Appearance	5	5	..	..	..	..	..	..	4	3 to 4
Multangulum Majus (Trapezium)—Appearance	5 to 6	7	..	..	..	..	..	5	5	6 to 7
Multangulum Minus (Trapezoid)—Appearance	5 to 6	4 to 7	..	..	..	..	..	5	6	7 to 8
Navicular (Scaphoid)—Appearance	6	7 to 11	..	..	..	..	..	5	5	6 to 8

—	Galstaun[1] (Bengalees) Females	Galstaun[1] (Bengalees) Males	Basu & Basu[2] (Bengalee Hindu Females)	Hepworth[3] (Punjabis)	Lall & Townsend[4] (Females of United Provindes)	Lall & Nat[5] (Males of United Provinces)	Pillai[6] (Madrasis)	Flecker[7] (Australians) Females	Flecker[7] (Australians) Males	Davies & Parsons[8] (Englanders)
Pisiform—Appearance	9 to 12	12 to 17	..	..	..	..	10 to 12	9	11	12
First Metacarpal bone										
Appearance	3	4	..	..	..	..	..	2	2	3
Fusion	14 to 16	16 to 18	..	..	..	..	14 to 17	16	18	18
Second, third, fourth, and fifth Metacarpal Bones										
Appearance	2 to 3	3 to 4	..	..	..	..	..	2	2	3
Fusion	14 to 15	16 to 18	..	..	..	..	14 to 17	16	18	18
Phalanges of The Hand										
Proximal Row—Appearance	1½	2 to 4	..	..	..	..	..	2	2	1 to 2½
Fusion	14 to 15	17 to 18	..	..	..	..	14 to 17	16	17 to 18	18
Middle Row—Appearance	2 to 3	3	..	..	..	..	..	..	..	22 to 30 Mths.
Fusion	14 to 16	16 to 18	..	..	..	..	14 to 17	16	17	18
Terminal Row—Appearance	3	3 to 5	..	..	..	..	..	2	2	2 to 3
Fusion	15	17 to 18	..	..	..	..	14 to 17	15	16 to 17	18
Tarsal Bones										
Calcaneus (Os Calcis) and Talus (Astragalus)—Appearance	At Birth	At Birth	..	..	..	..	..	Before Birth	Before Birth	Before Birth
Cuboid—Appearance	At Birth	At Birth	..	..	..	..	..	Before Birth	1	1
Internal Cuneiform—Appearance	1 to 3	1 to 4	..	..	..	..	..	2	2	End of 2nd yr.
Middle Cuneiform—Appearance	1 to 3	2 to 4	..	..	..	..	..	2	2	End of 3rd yr.
External Cuneiform—Appearance	1 to 3	1 to 4	..	..	..	..	..	2	3	1½
Navicular—Appearance	1 to 3	2	..	..	..	..	..	2	3	3
Metatarsal Bones—Appearance	3	4 to 5	..	..	..	..	..	2 to 4	2 to 5	3 to 5
Fusion	14 to 15	16 to 18	..	..	..	..	14 to 17	14 to 15	17	18
Tarsal Phalanges										
Proximal Row—Appearance	1 to 3	2 to 4	..	..	..	..	..	2 1/3	3 1/6	3 to 4
Fusion	14 to 15	16 to 18	..	..	..	..	14 to 17	14	17	17 to 19
Middle Row—Appearance	3 to 4	3 to 4	..	..	..	..	..	3	4 to 5	3 to 4
Fusion	14 to 15	16 to 18	..	..	..	..	14 to 17	13	15	17 to 19
Terminal Row—Appearance	4 to 6	4 to 6	..	..	..	..	..	2 1/3	1 3/4	4 to 6
Fusion	13 to 14	15 to 17	..	..	..	..	14 to 17	15	17	17 to 19

1. *Ind. Jour. Med. Res., July,* 1937, *p.* 267. 2. *Jour. Ind. Med. Ass., Aug.,* 1938, *p.* 571. 3. *Ind. Med. Gaz., March,* 1928, *p.* 128.
4. *Ind. Med. Gaz., Oct.,* 1939, *p.* 614. 5. *Ind. Jour. Med. Res., April,* 1934, *p.* 683. 6. *Ind. Jour. Med. Res., April,* 1936, *p.* 1015.
7. *Jour. Anat., Oct.,* 1932, *p.* 118. 8. *Jour. Anat., Oct.,* 1927, *p.* 58.

In old age the long bones become lighter and more brittle owing to the increase in the inorganic constituents. The skull bones tend to become thinner and lighter from absorption of the diploe, and are, therefore, liable to fracture more easily through violence. In a few cases, however, the skull bones become thicker and heavier owing to the hypertrophy of the inner table. The sutures of the vault of the skull are ossified and are completely obliterated, first on the inner surface, then on the outer surface, although the parietal sutures may remain separate throughout life.

The laryngeal and costal cartilages become ossified, and the greater cornua of the hyoid bone are firmly joined to the body by bony union; the lesser cornua which are usually connected to the body by fibrous tissue throughout life, may occasionally become ankylosed in advanced life.

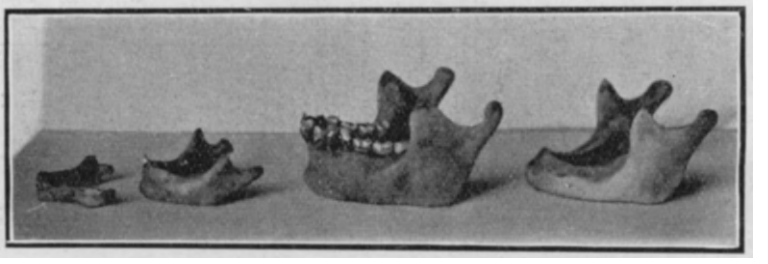


Fig. 5.—The lower jaw showing angles at various ages.

In infancy the mandible (lower jaw) has a short oblique ramus, which forms an obtuse angle with the body. The mental foramen opens near the lower margin, the condyloid process is nearly in line with the body, and the coronoid process projects above the condyle. In adult life the ramus joins the body almost at a right angle, and the mental foramen opens midway between the upper and lower borders of the body. The condyle is elongated and projects above the coronoid process. In old age the ramus forms an obtuse angle with the body which is reduced in size owing to the loss of the teeth and the absorption of the alveolar processes. The mental foramen is closer to the alveolar (upper) border.

Minor Signs.—The growth of hair appears first on the pubes and then in the axillæ (armpits). In the case of girls it commences with the appearance of down on the pubes at the age of about 13 years, and a few sparse dark hairs appear at about 14 years. The growth becomes thicker in the course of a year or two, when hair commences to grow in the axillæ. In the case of boys down appears on the pubes at about 14 years, and a few dark hairs appear at about 15, when downy hair begins to grow in the axillæ. A thick growth of dark hairs is well marked on the pubes, scrotum and in the axillæ at about 16 or 17 years. Hair begins to appear on the chin and upper lip between 16 and 18 years.

The development of the breasts in girls commences from thirteen to fourteen years, but it is liable to be affected by loose habits and social environments.

Boys develop a deep voice between 16 and 18 years when Pomum Adami becomes more prominent.

Hair tends to become grey usually after forty years of age and silvery white in advanced old age. Grey hair is sometimes seen among young people. In a few cases it is a hereditary peculiarity. Cases have occurred in which the hair of the head has suddenly changed to grey from extreme terror, grief, shock or some unaccountable reason. A case is recorded in which hair turned suddenly white in a single night, owing to fright produced by a dream.[1] In another case hair turned snow white a day or two after an automobile accident.[2] A girl, 17 years old, lost all her hair in five days owing to fright.[3] Circumscribed patches of grey hair on the head may also be due to trophic changes produced by neuralgia or other disease affecting the fifth nerve.

Atheromatous arteries, and an opaque zone in the cornea, known as *arcus senilis,* are rarely seen before forty. Wrinkles on the face begin to appear after this age; but no reliance can be placed on these signs inasmuch as arcus senilis has been seen as early as twenty-eight and wrinkles may not appear until a very late age, as they depend more or less on the nutrition of the body.

A horoscope can form a very important piece of evidence in deciding the question of age, but every one knows how easy it is to produce a fictitious one. Birth registers maintained in Municipalities will not be of much assistance in determining the age of a particular individual, as the names of children are not given in the registers.

Medico-Legal Aspect of Age.—The following are the cases in which a medical man is called upon to give his opinion as regards age:—

1. Criminal responsibility.
2. Marriage contract.
3. Kidnapping.
4. Rape.
5. Attainment of Majority.
6. Eligibility for employment.
7. Judicial Punishment.
8. Infanticide.
9. Criminal abortion.

Criminal Responsibility.—A child under the age of seven years (eight years according to the Children and Young Persons Act, 1933, of England) is presumed by Indian law to be incapable of committing an offence, and is therefore, exempt from punishment,[4] but this presumption is only confined to the offences prescribed under the Penal Code of India and does not extend to local or special Acts. For instance, a child even under seven years is liable to punishment for certain offences committed under the Indian Railways Act, 1890 (Act IX of 1890).[5]

1. *Lancet,* 1873, *p.* 675.
2. *The Medico-Legal Jour., Vol.* 49, *No.* 2, 1932, *p.* 50.
3. *Brit. Med. Jour., Vol. II,* 1879, *p.* 346.
4. *Appendix VII, Section* 82, *Indian Penal Code.*
5. *Appendix VII, Section* 130, *Act IX of* 1890.

In a case[1] where one Inderjit, a lad of about six years of age, was sent up to the Sessions at Budaun for trial on a charge of pelting stones at the engine of the 10 down mixed train under section 127-130 of the Indian Railways Act, the learned District Judge held the accused guilty of pelting stones at the engine. But taking the lad's tender age into consideration, the Judge ordered him to be let off with admonition on his father's executing a bond in a sum of Rs. 100 binding himself in such penalty to prevent the minor accused from being again guilty of any of the acts referred to in sections 127 and 130 of the Railways Act.

A child above seven years of age and under twelve in India and more than eight and under fourteen years of age in England is presumed to be capable of committing an offence, if he has attained "sufficient maturity of understanding to judge of the nature and consequences of his conduct on that occasion."[2] In this connection it may be noted that according to Indian law the maturity of understanding the nature and consequences of his conduct at the time of committing an offence is to be presumed in a child between seven and twelve years unless the negative is proved by the defence. Whereas by English law the incapacity to commit a crime by a child between eight and fourteen years is to be presumed until the contrary is proved.[3]

In a case in which two Pasi boys of ten and twelve years respectively and one Pasi girl of ten years were put on their trial on a charge of murder of a Brahmin girl of nine years by strangulation, the Sessions Judge of Sitapur held that the accused were undoubtedly old enough to understand the nature and consequences of the act committed by them, but in view of their tender age and the fact that they were compelled by hunger to resort to such a crime, he thought fit to exempt them from the extreme penalty of law. He accordingly passed a sentence of transportation, but ordered them to be sent to the Reformatory School.[4]

A child under twelve years of age cannot give valid consent to suffer any harm which may occur from an act done in good faith and for its benefit; while a person under eighteen years cannot give consent, whether express or implied, to suffer any harm which may occur from an act not intended to cause death or grievous hurt.[5]

Marriage Contract.—In India there was no limit of age for contracting marriage till the Child Marriage Restraint Act (Act XIX of 1929) came into force on the first day of April, 1930. By this Act a girl under fourteen years of age and a boy under eighteen years of age cannot contract marriage, while according to the law of England (Age of Marriage Act, 1929) a boy or girl cannot contract marriage until he or she has attained the age of sixteen years.

Kidnapping.—To constitute the offence of kidnapping or abducting a minor from lawful guardianship the age of a boy should be under fourteen years and that of a girl under sixteen years.[6] To constitute the offence of procuring a minor girl for illicit intercourse or of selling or buying a minor girl for purposes of prostitution her age should be under eighteen years.[7] Again an accused person can be indicted for importing into

1. *The Leader, May* 28, 1933.
2. *Appendix VII, Section* 83, *Indian Penal Code.*
3. *Gour, Penal Law of India, Vol. I, Ed. II, p.* 478.
4. *The Leader, April* 30, 1927.
5. *Appendix VII, Sections* 87, 89 *and* 90, *Indian Penal Code.*
6. *Appendix VII, Sections* 361 *to* 366, *Indian Penal Code.*
7. *Appendix VII, Sections* 366-*A,* 372 *and* 373, *Indian Penal Code.*

British India from a foreign country a girl for purposes of illicit intercourse, if she happens to be under twenty-one years of age.[1]

Rape.—Sexual intercourse by a man with a girl under thirteen years of age even if she be his own wife or with any other girl under fourteen years of age even with her consent constitutes rape.[2] According to the law of England a boy under fourteen is incapable of committing rape, but there is no such limit in India.

Attainment of Majority.—Under English law a person attains majority at twenty-one, when he enjoys the full privileges of an adult. Legally he is supposed to have attained majority a day previous to his twenty-first birthday. In India majority is reached at eighteen except in the case of ruling chiefs, or where the property is under a Court of Wards, when English law is applicable. A minor is incapable of selling his property, making a valid will or serving on a jury. A witness can give evidence in Court at any age, provided the Judge is satisfied that he understands the difference between truth and falsehood, and the necessity for speaking the truth.

Eligibility for Employment.—Twenty-five is ordinarily the limit for entering into Government service. Under the Factories Act,[3] 1934, (India Act No. XXV, 1934) as modified up to the 15th August, 1937, an "adult" is defined as a person who has completed his seventeenth year, an "adolescent" is defined as a person who has completed his fifteenth year but has not completed his seventeenth year and a "child" is defined as a person who has not completed his fifteenth year. No child who has not completed his twelfth year shall be allowed to work in any factory. No child who has completed his twelfth year and no adolescent shall be allowed to work in any factory unless a certificate of fitness granted to him by a certifying surgeon is in the custody of the manager of the factory, and he carries, while he is at work, a token giving a reference to such certificate. An adolescent shall be allowed to work as an adult in a factory if a certificate has been granted to him that he is fit to work as such. While at work in the factory he is required to carry a token giving reference to such certificate. But an adolescent who has not been granted such certificate of fitness to work in a factory shall be regarded as a child. No child shall be allowed to work for more than five hours in any day, and the hours of his work shall be so arranged that they shall not spread over more than seven-and-a-half hours in any day. No child shall also be allowed to work in a factory except between 6 a.m. and 7 p.m. provided that the Provincial Government may, by notification in the Official Gazette, in respect of any class or classes of factories and for the whole year or any part of it, vary these limits to any span of thirteen hours between 5 a.m. and 7-30 p.m.

Under the Indian Mines Act, 1923 as modified up to the first October, 1938, no child shall be employed in a mine, or be allowed to be present in any part of a mine which is below ground. No person who has not completed his seventeenth year shall be allowed to be present in any part of

1. *Appendix VII, Section* 366-B, *Indian Penal Code.*
2. *Appendix VII, Section* 375, *Indian Penal Code.*
3. Vide *sections* 2, 50, 51, 52, 53 *and* 54 *of the Factories Act,* 1934 (*India Act No. XXV,* 1934) *as modified up to the* 15*th August,* 1937.

a mine which is below ground, unless a certificate of fitness granted to him by a qualified medical practitioner is in the custody of the manager of the mine, and he carries while at work a token giving a reference to such certificate.[1]

Under Section 22 of the United Provinces Excise Act (Act IV of 1910) a licensed vendor is not permitted to sell any spirit or intoxicating drug to persons apparently under the age of sixteen years, while under Section 23 a licensed vendor is not allowed to employ children under the age of fourteen years in the premises in which foreign liquor or country spirit is consumed by the public.

Judicial Punishment.—Males above forty-five are exempt from whipping. A youthful offender is a person who has been convicted of an offence punishable with transportation or imprisonment and who, at the time of such conviction, was under the age of fifteen years[2] (sixteen years in the Presidency of Bombay[3]). Such an offender may be sent to a reformatory school, but must not be detained there beyond the age of eighteen years. He may also be ordered by the Court to be committed to the care of his parent or guardian or to be placed under the supervision of a trustworthy person. Under the several Children's Acts[4] in India no child under the age of fourteen years shall be sentenced to death or transportation, and no young person who is fourteen years of age or upwards but under the age of sixteen years shall be committed to prison unless the Court certifies that he is of so unruly or of so depraved a character that he is not a fit person to be sent to a reformatory school and that none of the other methods in which the case may legally be dealt with are suitable.

Under the Children and Young Persons' Act, 1933, of England, a person under the age of eighteen years cannot be sentenced to death. There is no such statutory provision in Indian law, although it is in the discretion of the Court to regard youth as an extenuating circumstance justifying the imposition of a lesser sentence of transportation instead of death. The Calcutta High Court sentenced a girl of sixteen years to transportation for life who was charged with deliberately killing her husband by administering arsenic.[5] The Nagpur High Court sentenced a boy, aged 13½ years, to transportation for life for having killed a man by shooting him with a rifle, but ordered him to be detained in a reformatory school for a period of four years.[6] On the other hand, cases are recorded where the tender age of the accused is not taken into consideration for awarding the lesser penalty of transportation for life, especially in cases of a ruthless and brutal murder. The Amritsar Sessions Judge sentenced to death one Didar Sing, 16 years old, for cutting off the head of his relative with a sickle.[7]

---

1. Vide *sections* 26 *and* 26-*A of the Indian Mines Act,* 1923, *as modified up to the* 1*st October,* 1938.
2. *Section* 4, *The Bombay Reformatory Schools Act,* 1897.
3. *Bombay Act No. XIII of* 1924.
4. *Section* 22 *of Madras Act IV of* 1920; *Section* 21 *of Bengal Act II of* 1922; *Section* 22 *of Bombay Act No. XIII of* 1924.
5. *Jasha Bewa* (1907), 11 *C. W. No.* 904; 6 *Crim. Law Jour., p.* 154; *Ratanlal and Thakore, The Law of Crimes, Ed. XIV, p.* 742.
6. *Daljit Singh* v. *K. E., Cr. Law Jour., Jan.,* 1938, *p.* 92.
7. *Times of India, Dec.* 11, 1934.

Infanticide.—In a charge of infanticide, where a newly born infant alleged to have been killed shows the signs of immaturity, it is necessary to determine whether the infant had attained the age of viability, which is certain after the 210th day of intra-uterine life and may, in exceptional cases, be after the 180th day. An infant born earlier than this period is not, in ordinary circumstances, capable of maintaining a separate existence after birth. Hence the charge of infanticide will fall through, if the infant is proved to be under the age of six months of intra-uterine life.

Criminal Abortion.—In criminal abortion it is necessary to find out whether a woman has passed the child-bearing period, lest it might be a false charge. It is also necessary to find out the age of the fœtus from the characteristics of its development.

4. COMPLEXION AND FEATURES

The complexion may be fair, wheat coloured, dark, brown or sallow. The colour may change from residence in a tropical country. The features of an individual may resemble those of his supposed parents or relatives, or his photograph, but this is not always the case. The features may change considerably from disease or dissipation or even from worries of a long duration. Again, there are some persons who can cleverly alter their features by changing the expression of their face, so as to evade detection. Peterson, Haines and Webster[1] quote the case of Tidy in which Charles Peace, a burglar, who was executed for the murder of William Dyson in 1879, had such a remarkable power of changing his features and altering his expression that he was accustomed to face the detectives who not only knew him well but were actually seeking to arrest him at the time he was talking to them, and was, moreover, able to deceive his wife and son as to his identity.

Photographs of the front and profile views of the face may serve as a means of identification, and are specially useful in cases of disputed paternity. While examining photographs the chief point to note is the character of the angle which the eye forms with a line drawn through the middle of the forehead or nose; but the medical man should never risk an opinion on this point, as he should remember that he is not an expert in photography where a photographer or an artist is better qualified to give an opinion on such a point.

The details of the features as regards the eyes, nose, ears, lips, chin and teeth should be carefully noted. The irises of the Indians are generally dark brown, but are grey in a few cases, especially among the Punjabis. In some individuals the colour of one iris may differ from that of the other. Coloboma or hiatus may be found, if an operation has been performed on the iris. The bridge of the nose may be narrow, flat or broad, and the nostrils may be distended or the reverse. The ears may be small or large in size. Their lobules may be free or adherent to the face. The lips may be thin or thick, and the upper lip may hang over the lower lip, or may look shorter owing to the upper incisor teeth projecting outwards. The chin may be rounded, square, protruding or double from excessive fat.

1. *Legal Medicine and Toxicology, Vol. I, Ed. II, p.* 165.

Kumar Ramendra Narayan Roy, the second son of Raja Rajendra Narayan Roy Bahadur of Bhowal estates in Dacca went to Darjeeling in 1909, where he died of biliary colic. Twelve years later in 1921 a Sadhu came to Dacca and declared that he was Kumar Ramendra Narayan Roy and claimed one-third share of the Bhowal Raj Estate. He further declared that in 1909 he went to Darjeeling on a rest cure and while there he was the victim of a murder conspiracy. He alleged that arsenic was administered to him with the intention of killing him, and that owing to its administration he relapsed into coma and was taken for dead. His body was accordingly removed to the cremation ground at night, but a heavy storm came up and the funeral party left his body on the cremation ground without attempting to light the funeral pyre. While still in an unconscious condition he was found on the funeral pyre by some *Naga Sanyasis* who revived him, and carried him with them. He stayed with them as a pupil of their religious doctrines until 1921.

The Kumar being baffled in all his attempts to regain his share of properties brought a suit in the Court of the Subordinate Judge of Dacca in 1930 which was eventually transferred to the file of Mr. Pannalal Bose, the Subordinate Judge, who during the course of the trial was promoted to be an Additional District and Sessions Judge of Dacca. The suit was keenly contested by Shrimati Bibhabati Devi, who was married to the second son of the Raja Bahadur, and others. Their contention was that the plaintiff was an impostor, named Sundardas Naga, a disciple of a Hindu holy man from the Punjab, and that the second son of the Raja Bahadur actually did die at Darjeeling in 1909 and that his body was duly cremated.

During the hearing of this remarkable case, popularly called, Bhowal Sanyasi Case, which lasted for more than two years, about 1,069 witnesses on the plaintiff's side and 470 on the defence side were examined, and photos and documents numbering over 2,000 were exhibited before the Court. The judgment was delivered in favour of the plaintiff who was declared to be Kumar Ramendra Narayan Roy and was declared to be entitled to the status and title of the second Kumar of Bhowal and to one-third of the property.

The following marks and features which are exceptional serve as identifying marks :—

Kumar	Plaintiff
Complexion.—Pink and white.	Pink and white.
Hair.—Brownish.	Brownish.
Hair-form.—Wavy.	Wavy.
Moustache.—Lighter than hair.	Lighter than hair.
Eyes.—Brownish.	Brownish.
Lips.—Twist on the right lower lip.	Twist on the right lower lip.
Ears.—A sharp angle at the rim.	A sharp angle at the rim.
Lobes of ears.—Not adherent to the cheeks and pierced.	Not adherent to the cheeks and pierced.
Adam's Apple.—Prominent.	Prominent.
The left upper first molar tooth.—Broken.	Broken.
Hands.—Small.	Small.
Index and middle fingers of the left hand.—Less unequal than those of the right.	Less unequal than those of the right.
Point of flesh or something in the right lower eyelid.—Present.	Present.
Feet.—Scaly. Size, 6 for shoes	Scaly. Size, 6 for shoes.
Irregular scar on the top of the left outer ankle.—Present.	Present.
Syphilis.—Present.	Present.
Syphilitic ulcers.—Present.	Marks of such ulcers.

In addition to the above, there were other marks of resemblance ; *viz.*, photographs, the boil-mark on the head, the boil-mark on the back, the operation mark near the groin, the tiger-claw mark on the right arm, a minute mole on the dorsum of the penis, the gait, voice and expression.[1]

1. *Mitra and Chakravarty, The Bhowal Case,* 1936.

The teeth afford a useful means of identification especially in the case of bodies which have been destroyed by injury, fire or decomposition. They are more resistant to destructive agents than any other structure, and are well protected. The teeth may be artificial or natural or a few may be missing, carious or worn out or may have been set irregularly or coloured.

A case occurred at the Cumberland Assizes held at Carlisle in which two colliers were charged with breaking into the Co-operative Society's Store and stealing some valuable articles. On examination of the premises it was found that some one had bitten a piece off a cheese and had left the marks of his teeth. On suspicion two men were arrested and one of them unwittingly permitted a cast of his teeth to be made, which was found to fit exactly into the marks on the cheese. A dentist stated in his evidence as an expert that no two sets of teeth were identical. This accused was very anxious that his mouth should be examined to see if his teeth would fit the impression on the cheese. When this was done, the very damning evidence was ascertained that since his arrest he had knocked out a stump.[1]

In December, 1918, a Mahomedan prostitute was sent to me for examination by a Bench Magistrate. She had superficial lacerations in the form of a circle on her right cheek as a result of teeth-bite, but in the upper half of the circle there was a blank space which coincided with the missing right upper central incisor tooth of the accused.

In connection with a burglary in a jeweller's shop in Lucknow the police arrested a goldsmith who was found in possession of several diamond crystals. When the crystals were identified by the proprietor of the jeweller's shop the goldsmith confessed that he got the crystals from a Sindhi merchant who had one gold tooth which gleamed whenever the man smiled but was not visible otherwise. Later, in a restaurant in Aminabad the proprietor of the jeweller's shop came across a man who answered to the general description of the suspect given. The difficulty was about the gold tooth, for when the man spoke it was not visible. The proprietor was wondering whether he should send for the police when some one in the restaurant cracked a joke in Sindhi. The suspect laughed at the joke revealing the gold tooth. His doubts being at rest, the proprietor of the jeweller's shop immediately sent for the police and had the man arrested.[2]

The record which a dentist keeps of the condition of his patient's teeth or the cast which he takes of his patient's jaw for fitting artificial teeth may, sometimes, be very valuable for purposes of identification.

A diplomatic official in Chile complained of threatening letters, and one night the embassy was burned down and his charred body was found in the ruins and buried with great public lamentation and eulogy. The Director of the Chilean Dental School had his doubts and examined the corpse an hour before burial. The next day he announced that the deceased was not a missing official; within a few days the latter was arrested on the frontier with the whole negotiable wealth of the embassy. The body was that of the night porter, who had never been to the dentist; although the murderer had been to particular pains to mutilate the teeth, they were easily distinguishable from his own described in his dentist's records.[3]

Owing to decomposition, more often in summer, the features of a dead body become bloated beyond recognition. It has been suggested that the altered features due to decomposition may be restored to natural features by bathing them with chlorine, salt and hydrochloric acid, as well as by injecting them with chlorine, zinc chloride and ferric chloride; but this does not seem to be possible.

1. *Brit. Med. Jour., Feb.* 10, 1906, *p.* 343.
2. *Pioneer, Dec.* 2, 1933.
3. *Alexander Klein, Schmeiz, Montas f. Zahnnh.,* 1929, *XXXIX; Lancet, May* 17, 1930, *p.* 1130.

5. HAIR

This forms an important means in establishing identity, as it resists putrefaction. The hair of the Indians is generally dark and fine. That of the Chinese and Japanese is dark and coarse, while that of the Negroes is curly and woolly. It has been observed by Tidy that the hair grows even after death, but this growth is only apparent probably from the shrinking of the skin which takes place after death.

Change in Colour.—To disguise identity hair may be artificially coloured with henna, darkened by dyes and cosmetics containing metallic salts of lead, bismuth or silver, or rendered lighter by using chlorine, hydrogen peroxide, dilute nitric acid or nitrohydrochloric acid. In India some old people colour their hair red or black for the purpose of concealing their age and of looking young. It must be remembered that a change may occur in the colour of the hair of men working in certain trades. For instance, the hair of ebony-turners and copper-smelters may acquire a greenish hue, while that of indigo-workers becomes blue and cobalt miners exhibit a bluish tint.

A Mahomedan midwife, named Sharifan, disappeared suddenly from her house in Ganda Nalla, Delhi. On suspicion the police raided the house of one Shahab-ud-Din in Hamilton Road after several weeks, and unearthed the dead body of a woman after getting a large portion of the house dug. The body was in an advanced state of decomposition, but it was identified as that of the missing midwife from the dyed hair which was intact. A rope was found round the neck, which led to the suspicion that death was in all probability brought about by strangulation. The owner of the house was arrested, against whom a case under section 302, I.P.C., had been registered.[1]

Detection of Colour.—The colouring of the hair can be detected by examining the scalp, which will, as a rule, be found dyed, and the colour of the hair will not be uniform, the roots being different in tint from the rest. Such hair is rough, brittle and lustreless. The colouring can also be ascertained by comparing the hair of the head with that of other parts of the body, such as pubes and armpits, which is usually not dyed, as it is not likely to be open to the gaze of the public. In doubtful cases the hair should be shaved or cropped closely and the colour of the growth of the new hair should be observed, while the person is kept in custody for a few days.

Chemical Examination.—To find out the mineral used for dyeing, some hair should be steeped or boiled in dilute hydrochloric or nitric acid to dissolve out the metal and the appropriate tests should then be applied to the solution thus obtained.

6. ANTHROPOMETRY

This is a system chiefly used for the identification of habitual criminals. There are two methods by which this is carried out. One is called the *Bertillon System* or *Bertillonage* and the other is called the *Galton System.*

Bertillon System.—This system is called Bertillonage from the name of its inventor, M. Alphonse Bertillon. It is applicable only to the adult, since it is based on the principle that after twenty-one years of age no

1. *Hindusthan Times, Dec.* 11, 1932.

change occurs in the dimensions of the skeleton during the rest of the life and that the ratio in the size of the different parts to one another varies considerably in different individuals.

In consists in taking the measurements of certain parts of the body and then classifying the individual. The measurements that are usually taken are the height of the person while standing, the length of the head, the width of the head, the length of the right ear, the width of the right ear, the span of outstretched arms, the height of the trunk while sitting, the length of the left foot, the length of the left middle finger, the length of the left little finger and the length of the left forearm and hand (cubit). These measurements are entered upon cards which are kept in a specially arranged cabinet, so that they can be easily picked out when required. The colour of the iris and certain peculiarities, such as scars, etc., are noted on these cards, and photographs of the full face and the right profile are also kept along with them. This system is useful for the identification of criminals, but it necessitates the employment of special instruments and a large number of men, so that there is always a possibility of errors creeping into the records of the actual measurements.

Galton System.—This system consists in taking the impressions of the bulbs of the fingers and thumbs with printer's ink on an unglazed white paper and then examining them with a magnifying lens. It is based on the principle that the individual peculiarities of the patterns formed by the arrangement and distribution of the papillary ridges on the finger tips are absolutely constant and persist throughout life, from infancy to old age, and even after death up to the time when the skin perishes through putrefaction, and that the patterns of no two hands resemble each other.

The following case[1] well illustrates the fact that it is possibile for any two persons to bear striking points of resemblance on the body, but it is never possible for them to have identical finger impressions :—

In 1917, Professor Canella of Milan, while serving the Army in Macedonia, was reported missing and was never heard of again. In 1924, a man suffering from loss of memory was admitted into a Piedmont asylum and he remained there for two years. Afterwards the wife and the daughter of the professor unhesitatingly identified him as the professor, as he bore remarkable external resemblance. He was at once taken to Milan, where all the friends of the professor at once recognised him. By degrees he appeared to recover his memory, and then asserted that he was indeed the lost professor, and purported to recall many incidents which had happened in the latter's career. All seemed well, but suddenly there fell a bolt from the blue. A woman appeared on the scene and identified him as her husband, Bruneira, who had absconded three years ago after a career of crime. The family and acquaintances of Bruneira, one and all, likewise identified him. He was examined and found to possess certain marks on the body, which, however, curiously enough were alleged by both parties of relatives to be life long marks of Canella and Bruneira respectively. The Italian Police produced the finger prints of Bruneira which were alleged to be identical with those of the man whose identity was in dispute.

The ridges on the fingers and hands are studded with microscopic pores, which are the mouths of the ducts of the sweat glands situated below the epidermis. These pores may be used for personal identification, as they are permanent and immutable during life and vary in size, shape,

1. *Times, March* 15, 1927 ; *Criminal Law Jour., Vol.* 28, 1927, *p.* 62.

position, extent and number over a given length of the ridges in each individual. This method of identification by examining the pores is known as poroscopy, and is of the greatest value when a small fragment of a finger impression or an impression of a part of a palm is available for comparison.

Before taking the impressions the fingers should be thoroughly washed and rubbed clean and dry, as the slightest perspiration will cause blotches and blur the print. It should be remembered that the finger prints of lepers should, on no account, be taken, while those of persons suffering from infectious or contagious diseases should not be taken until they have completely recovered.

Fingers smeared with blood, grease, dirt or slight perspiration may leave their impressions on weapons, clothing, glass panes, utensils, furniture, etc.; hence considerable care should be taken in handling such articles during the investigation of a crime, and any articles found to possess such prints should be preserved for further examination.

Finger impressions are either rolled or plain. A rolled impression is obtained by first inking the bulb surface of the finger or thumb between the nail boundaries and then placing the inked finger or thumb on the paper so that the plane of the nail is at right angles to the plane of the paper. The finger or thumb is then pressed lightly on the paper and turned over so that the bulb surface which originally faced to the left, faces to the right and vice versa, the plane of the nail being again at right angles to the paper. A plain impression is obtained by lightly pressing the inked bulb surface of the finger or thumb upon the paper without any turning movement.

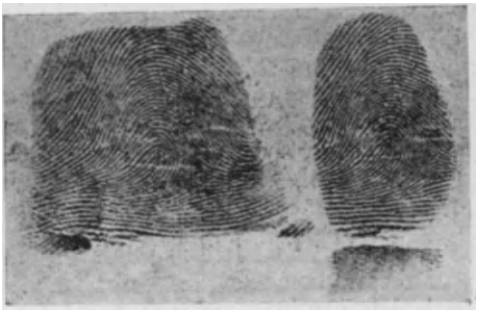
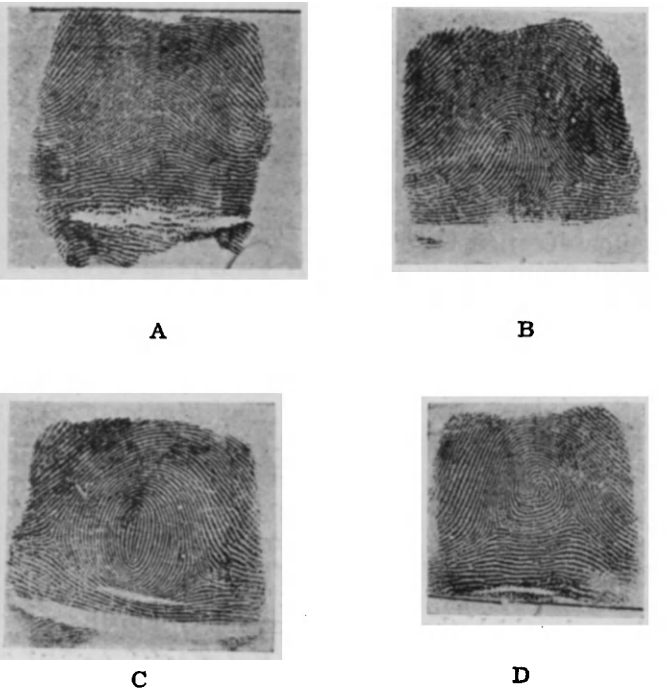


Fig. 6.—Rolled and plain impressions of the left thumb.

In a plain impression the whole contour of the pattern does not appear, whereas in a rolled impression the whole pattern is delineated. It is, therefore, easier to determine the type of pattern from a rolled impression, and its greater surface enables the expert to select a larger number of points for comparison.

All impressions are classified as arches, loops, whorls and composites. In arches the ridges run from one side to the other without marking any backward turn. The ridges may converge together and by an upward thrust in the middle look like a tent, when the arches are known as "tented." In loop, whorl and composite types there are fixed points, which are known as the *delta* or *outer terminus* and the *point of the core* or *inner terminus*. These serve a useful purpose in the classification of finger impressions. The *delta* may be formed by the bifurcation of a single ridge or by the abrupt divergence of two ridges running side by side. The *core* of the loop may consist either of an even or uneven number of ridges, termed rods, not joined together, or of two ridges joined together at their summit, termed *staple*. Some of the ridges exhibit a backward turn without any twist. The ridges round about the *core* frequently deviate in course from the general course of the other ridges and leave a space which is described as a *pocket* and the loop is then known as a *pocket* loop. Again loops are described as *twinned*, when a well-defined loop rests upon or surrounds another of the same variety. In circular or elliptical whorls, the centre of the first ring is the *point of the core*. In spiral whorls the point from

which the spiral begins to revolve is *the point of the core*. In composite types, arches, loops and whorls are grouped together in the same impression. They also include a small number of irregular patterns which cannot be classified under any



A B C D

Fig. 7.—Patterns of finger-impressions.

A. Arch. B. Loop.
C. Whorl. D. Composite.

(*By permission of the Superintendent, Finger Print Bureau, U. P., Allahabad*).

known variety of loops. For want of a better designation they are known as *accidentals*. The finger impression printed on a paper is a reversal of the pattern on the finger. For instance, if the pattern on the finger is a loop with slope from right to left, it will appear in the print as a loop with slope from left to right. A loop is called radial when the downward slope of the ridges about the core is from the direction of the little finger towards that of the thumb. A loop is ulnar, when the downward slope is from the direction of the thumb towards the little finger. About 5 per cent of impressions are arches, 60 per cent loops and 35 per cent whorls and composites. For the purposes of primary classification for criminal work arches are included under loops and composites under whorls. All the ten fingers are taken in the following pairs :—

$$\frac{\text{Right index}}{\text{Right thumb}}, \frac{\text{right ring finger}}{\text{right middle finger}}, \frac{\text{left thumb}}{\text{right little finger}},$$

$$\frac{\text{left middle}}{\text{left index}}, \frac{\text{left little}}{\text{left ring}}.$$

When a whorl occurs in the first pair it counts 16 ; in the second pair 8 ; in the third 4 ; in the fourth 2 ; and in the fifth 1. No value is fixed for loops. Obtain a new numerator and a denominator by adding together all the numerators and all the denominators. Add 1 to the numerator and also to the denominator thus obtained.

The fraction now obtained is the classification number and indicates that the slip is to be placed in the pigeon-hole bearing that number. Primary classification numbers are $\frac{1 \text{ to } 32}{1 \text{ to } 32}$ or 1024 pigeon holes. The formula for the classification of these digits may be represented as $\frac{W}{L}, \frac{L}{W}, \frac{L}{L}, \frac{W}{W}, \frac{W^1}{L}$.

The system was first used by Sir William Herschel, I.C.S., but the credit is given to Sir Francis Galton for having systematised it for the identification of criminals. The system is so very perfect that it has now been adopted all over the civilized world. It may be noted that this science of finger prints was known in ancient Assyria, and was used for purposes of identification in 700 A.D. The Chinese police were using it in the thirteenth century.[2] In 1899 an Act was passed by the India Council that the evidence given by experts to decipher finger prints was relevant in any case.[3] Persons have often been convicted of a criminal charge from the only evidence of their finger impressions left on furniture or some articles, as in the Muttra murder case of 1901, where the murderer's thumb-impression was left on a brass *lota* which he had used in washing his hands after killing his victim. One Elahi Bux[4] was arrested for committing dacoity in a village of Saicha, Raipura, from the impressions of his right thumb and index finger which were completely separated with a *dao* by the inmate of the house and handed over to the police. In a case[5] where burglary was committed in the house of a doctor in the small hours of May 5, 1929, the burglars were arrested and convicted from the finger impressions left by them on the broken glass panes of the sky-lights through which they effected entrance into the room.

The London Police have devised a system[6] by which finger prints can be sent by cable or wireless telegraphy to all parts of the world. In this system the various arches, whorls and loops are described by index letters and figures. At the receiving bureau the finger prints can be reconstructed in five minutes. In one case the finger prints thus sent to New York enabled a counterfeiter, who had escaped while on bail, to be identified, and in another case a man detained by the London Police was believed to be wanted by the Chicago Police and his finger prints were taken and sent by wireless to the United States.

Fugitives know full well that as long as their finger prints are on the files of the Criminal Investigation Department they cannot hope to avoid detection by merely adopting aliases and changing their bases of operation. Hence they, sometimes, attempt to mutilate the patterns by inflicting injuries, such as wounds or burns, on the bulbs of their fingers, but they forget that the resultant scars do not necessarily obliterate the patterns, as there will still exist definite delineations, unless the true skin is completely destroyed.

1. *For detailed description see Finger Print Manual, U. P.*, 1925 *Reprint.*
2. *R. Scheffer, Zeutzalblatt Gynakologie, Leipzig,* 50, *Oct.* 2, 1926, *p.* 2559; *Journal Amer. Med. Assoc., Jan.* 15, 1927, 214.
3. Vide *Appendix V, Section* 45, *Indian Evidence Act.*
4. *Leader, Aug.* 22, 1930, *p.* 6.
5. *Lahore High Court, Criminal Appeal, No.* 1168 *of* 1929, *King-Emperor* v. *Sardara; Criminal Law Jour., Sep.*, 1930, *p.* 877.
6. *Jour. Amer. Med. Assoc., June* 9, 1928, *p.* 1883; *Lancet, Oct.* 10, 1931, *p.* 812.

In the case of criminals and emigrants, and in the case of persons in subordinate Government service—both civil and military—while preparing their service books and pension papers, impressions of all the ten fingers are taken, but for the purpose of identification while giving a medical certificate and for other civil purposes the left thumb impression only is taken.

The police are required to take the finger prints of an unidentified corpse, or of a person whose identity has not been established by ordinary enquiries and who has died in an accident, or under suspicious circumstances, or in the commission of a crime. Ordinarily there is not much difficulty in taking impressions from the fingers of a corpse, but it is, sometimes, difficult to obtain decipherable prints in a decomposed body, where the skin of the fingers is contracted and wrinkled. In such cases the police should request the medical officer holding the post-mortem examination to remove the skin from the bulbs of the fingers. The medical officer should pack each piece in a separate envelope marking on the outside the finger to which it belongs.[1] For the United Provinces of Agra and Oudh, these envelopes should then be sent to the Finger Print Bureau at Allahabad for opinion.

Faint and invisible finger prints can be rendered quite clear and distinct by dusting them with some fine, impalpable powder. They can then be examined with a lense or enlarged permanently as a photograph.[2] If the finger prints are on paper or a light-coloured surface, graphite (plumbago) or lamp black is used. If the prints are on glass or a dark-coloured surface, grey powder, magnesium carbonate, white lead, red lead or ferric oxide will develop them.

Finger prints on paper may also be developed by exposing it to the vapours of iodine or osmium tetroxide and by brushing the surface with some coloured solution. The prints developed with the aid of iodine vapours are fugitive, and should, therefore, be photographed at once. Mitchell[3] suggests the application of the osmium tetroxide vapour by exposing the prints to the vapour of a boiling one per cent solution of this reagent in water. The coloured solution may be writing ink of any colour or some dye dissolved in water or alcohol. A solution of osmium pyrogallate prepared by mixing 2 c.c. osmic acid and 0.05 gramme pyrogallic acid in 2 c.c. water gives satisfactory results.[4]

Major Henry Smith, I.M.S., has discovered that it is possible to forge thumb-impressions by covering the original thumb-impression with a piece of damped paper and pressing, by which method the reverse of the original is transferred to the damped paper. Another piece of damped paper is then put over the reverse and pressed, and a true copy of the original is thus obtained.[5]

Tamassia's Identification of Veins.—Tamassia has, for the purpose of identification, made use of the fact that the peculiar distribution and

1. *Appendix I, Police Regulations, Section* 118-*A.*
2. *J. G. Garson, Trans. of the Medico-Legal Soc., Vol. II,* 1904-1905, *p.* 115.
3. *The Analyst, XLV,* 1920, *pp.* 122-29; *Lucas, Forens. Chem. and Scientific Investigations. Ed. III, p.* 184.
4. *Ibid., p.* 185.
5. *Ind. Med. Gaz., June,* 1902, *p.* 255.

arrangement of the veins on the dorsum of the hand remain constant. The veins are rendered conspicuous by pressing the arm with a ligature and the dorsum of the hand is photographed.

7. FOOT PRINTS

The impression of a foot or a boot left on the ground in the vicinity of the place of occurrence of a crime has often led to the arrest of the criminal. To identify the footprint a fresh footmark of the suspected person should be obtained and compared with the original. During the examination a careful note should be made if there are any peculiarities in the foot, such as flat foot, scars resulting from wounds, or callosities, as these are likely to be found in the footprint if it is well marked. In the case of a bootmark the peculiar arrangement of the nails, or holes in the sole may be useful in comparing with the original. It is often said that a footprint made by an individual while he is walking is smaller than the one

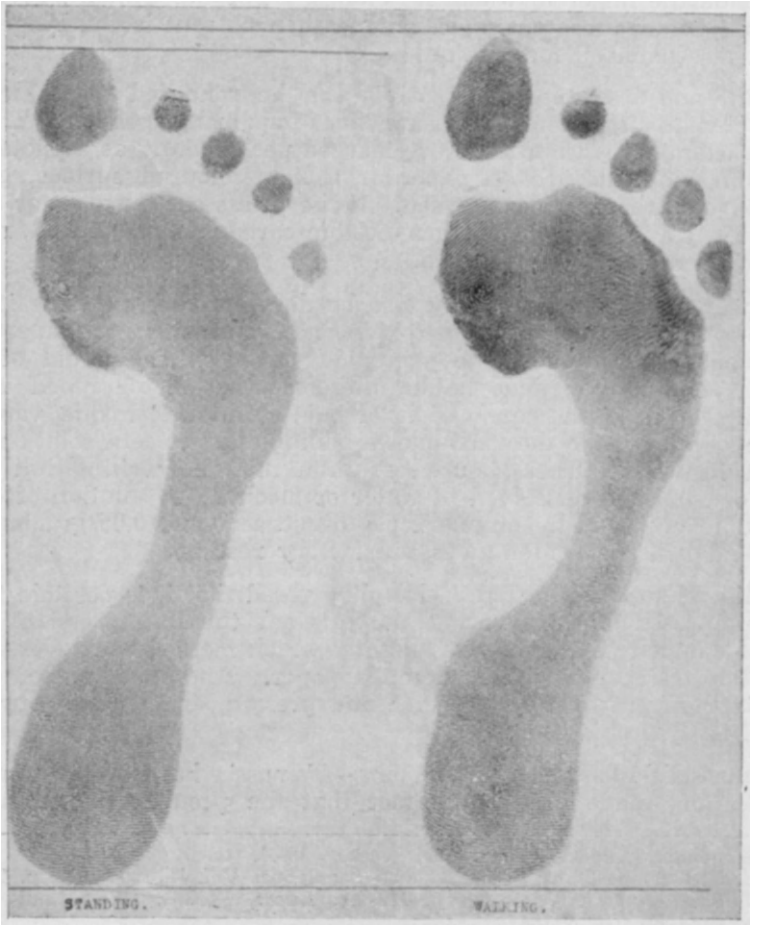


Fig. 8.—Foot print: Standing and Walking.

made by him while he is standing, but I have found from experiments that a footprint produced in walking is generally larger than the one produced in a standing position. It is usually assumed that the impression left on the material composed of loose particles, such as sand, is smaller than the foot or boot producing it, while the impression on mud, clay or some material not composed of freely movable particles, is larger.

Casts of foot prints may be taken by smearing the prints with oil and pouring in liquid plaster of Paris, or by dusting them with powdered stearic acid or with solid paraffin, which may be melted by holding a hot iron over the prints. This process may be repeated until the cast obtained is sufficiently thick to be removed.

In some maternity hospitals the system of taking the impressions of foot prints of newly-born infants has lately been introduced to avoid the confusion of their being mixed or to prevent their deliberate substitution or changeling. These form a permanent record for future identification.

8. DEFORMITIES

Deformities form an excellent means of identification; hence they should be very carefully noted in the description of the person of a living individual or in the external examination of a dead body. They may be congenital or acquired. Congenital deformities, such as cleft-palate, hare-lip, supernumerary fingers or toes, supplementary mammæ, web-fingers or toes, birth-marks (nævi) and moles, are hereditary in many cases, and known to occur through successive generations in the same family.

Dr. Young of Parkhead, Glasgow, has recorded a very interesting case in which he could trace the hereditary digital abnormality through four generations; it had been transmitted almost invariably from the paternal side.[1] A genealogical tree is given of a family with supernumerary digits in which the deformity skips two generations to reappear in one member of the third and in five of the fourth.[2]

Acquired deformities such as malunited and united fractures, are the results of previous injuries.

Two cases are recorded by Chevers.[3] In one the identity of a murdered person was established by a peculiarity in the jaw bone. In the other an impostor pretended that he was identical with a Bengali gentleman whose death and consignment to the Ganges, some years before, were plainly proved. He allowed the latter fact, though he denied the former, and declared that he had revived after he was thrown into the river and had since lived as a *fakir*. Medical evidence was of great importance in this case as it helped to expose the imposture by proving that the body of the deceased had been eaten away by an incurable disease.

The body of Livingstone, the great African traveller, was recognised by the ununited oblique fracture of the humerus exactly in the region of the attachment of the deltoid to the bone.[4]

9. SCARS

A scar or a cicatrix is a fibrous tissue covered by epithelium formed as a result of the healing process of a wound or injury in which there has

1. *Brit. Med. Jour., Sep.* 10, 1898, *p.* 715.
2. *Ibid., May* 28, 1904, *p.* 1254.
3. *Med. Juris., pp.* 48 *and* 100.
4. *Lancet, April,* 1874.

been a breach of continuity of substance. It has no hair follicles or sweat-glands, but it is slightly vascular owing to the presence of a few capillaries.

Character of Scars.—A scar generally assumes the shape of the wound causing it. A scar resulting from an incised wound which has healed by first intention, is usually linear and straight. If it has healed by the formation of granulation tissue, the resulting scar will then be wider and thicker in the centre than at the periphery. But a scar following an incised wound in the axilla or upon the genitals may be irregular on account of the loose folds of the skin. Such a scar may also be smaller than the original wound.

Broad and irregular scars are caused by lacerated wounds. Large, irregular scars accompanied very often by keloid patches are the results of extensive burns and scalds. Scars resulting from bullet wounds are generally irregular in shape and larger than the bullet, and are usually surrounded by the grains of unburnt gun-powder, if the weapon was discharged within a few inches of the body. While they are depressed, disc-shaped, adherent in the centre and smaller than the bullet, if discharged from a distance. The scar of the wound of entrance is, as a rule, smaller than that of the wound of exit. Doubtful cases should always be examined by X-Rays in order to determine the presence or absence of shots embedded in the underlying tissues or of injuries to the underlying bones.

In January, 1924, the District Magistrate of Gonda referred to me a case in which one Bisheshwar Singh was suspected of having taken part in a dacoity that took place three years ago, and was consequently injured in the thighs by the discharge of fire-arms. On examination I found several rounded, nodular and depressed scars on the front of the thighs resulting probably from gun-shot wounds. Skiagrams that were taken in the X-Ray Department of the King George's Hospital, Lucknow, showed several shots embedded in the soft tissues of the thighs. The man was prosecuted and sentenced to a term of imprisonment for the offence of committing dacoity.

Scars from wounds produced by stabbing instruments are triangular in shape, smaller in size than the blade of the weapon, and are less depressed than the scars of gunshot wounds. Scars resulting from leech-bites are triradiate. Scars due to syphilitic and tuberculous ulcers are irregular and thick in parts, while those due to vaccination and small-pox are pitted.

Appearance of Scars.—A scar appears in four or five days when a wound heals under a scab as in the case of a superficial cut on a finger or a shave-cut on the chin or cheek. In the case of a clean aseptic wound which is caused by a surgical operation, and heals by first intention, the scar usually appears in a fortnight, while in a suppurating wound it appears from two weeks to three months, or more.

Disappearance of Scars.—Scars resulting from wounds and skin diseases which involve the whole thickness of the skin are always permanent, but superficial linear scars involving only the epidermis or cuticle layer of the skin may disappear in the course of a few years. It is not possible to remove a scar successfully but its size and shape can be altered by an operative procedure. A faint scar may be made more visible by rubbing or slapping the part, when the surrounding skin will be red, and the scar will appear whitish in colour. If necessary, it should

be examined by the aid of a lens. While describing a scar for the purpose of identification, its shape, size and situation should be mentioned.

Age of Scars.—It is difficult to tell the exact age of a scar ; hence the medical witness must be very cautious in answering this question which may connect an accused person with the perpetration of a crime in which he is alleged to have been wounded. When first formed, a scar is red, tender and covered by a scab, subsequently it becomes brown and lastly, on account of the obliteration of the capillaries due to their being affected by the growth of connective tissue, it acquires a white and glistening appearance which remains permanent for the rest of life. These changes are generally produced in three to four months, but the variations in the time are so great that it is not safe to fix any time-limit within which these changes are produced.

Growth of Scars.—Scars produced in childhood grow in size with the natural development of the individual, especially if situated on the chest and limbs.

Case.—At the trial of Crippen charged with having murdered his wife, Belle Elmore, Mr. Pepper and Drs. Spilsbury and Willcox were able to establish the identification of the mutilated remains found buried in a hole dug in the floor of the cellar occupied by Crippen to be those of his wife by the discovery of an old scar on a piece of skin measuring seven inches by six inches which came from the lower and front part of the abdominal wall. At its lower margin there was a row of short, dark hairs. The scar was situated in the middle line commencing just above the pubes and extending vertically upwards for four inches or a little over. It was bigger at the bottom, being seven-eighths of an inch wider than at the top where it tapered to something like one-fourth of an inch while it measured half-an-inch in middle. It was in a place corresponding with an operation performed for removal of the ovaries or uterus. Mr. Pepper also said in his evidence that a scar in that position in the male as the result of an operation performed for removing stones or tumours from the bladder would be "less likely to be so wide, because, as a rule, there is not so much distension." It was proved beyond dispute that Belle Elmore had undergone an abdominal operation for ovariotomy. It was further brought out in evidence that a scar viewed under a microscope might show a sebacious gland or a hair follicle, if, in stitching up a wound a piece of epidermis was turned in and involved in the wound.—*Bri. Med. Jour., Oct.* 29, 1910, *p.* 1372.

10. TATTOO-MARKS

The practice of tattooing is prevalent all over the world, though more common among the lower order of society. Designs of all sorts varying from initials to gods of worship and even those indicating emblems of moral depravity are not only found tattooed on the arm, forearm and chest but on the other parts of the body. While describing tattoo-marks, their design and situation should be carefully noted. It is possible to find the same design at the same situation in more than one individual, if the operator happens to be the same person. Complications, such as septic inflammation, erysipelas, abscess, gangrene and even syphilis, leprosy and tuberculosis, are known to have followed this operation.

Disappearance of Tattoo-marks.—From experiments[1] Hutin, Casper and Tardieu have proved that tattoo-marks may disappear during life without leaving any trace on the body after a period of at least ten years provided the pigment used is vermilion or ultra-marine and if it has not penetrated deep into the skin. Even in these cases the pigment may be

1. *Casper, Forens. Med., Vol. I., Eng. Trans., pp.* 106-108.

seen deposited in the neighbouring lymphatic glands, if examined after death. But the marks are indelible if some such pigment, as Indian ink, soot, gunpowder or powdered charcoal, has been used and has penetrated

Fig. 9.—Designs of Tatto-marks.

deep into the fibro-elastic tissue of the skin. These marks are so very permanent, that they may be recognised even in decomposed bodies after the skin has peeled off. The letters "P.L." tattooed on the left forearm were evident in a badly decomposed body examined a fortnight after death.[1] A faded tattoo-mark may, sometimes, be rendered visible by friction and then examined with a magnifying lens in strong light.

Artificial Removal of Tattoo-marks.—These may be removed artificially (1) by the surgical method, (2) by electrolysis, and (3) by the application of caustic substances.

(1) *Surgical Method.*—The earliest surgical method employed for the removal of tattoo-marks was the production of a burn by the application of a red hot iron to the design. When the dead tissue sloughed off, it took the tattoo-mark with it but usually left a bad scar in its place. The use of carbon dioxide snow produces similar results. The simplest surgical method used at present is the excision of the tattoo-marks with or without skin grafting. This is quite a successful method but is always followed by some scarring.

Lacassagne and J. Roussel[2] recommended the scarification of the tattooed spots, and then sprinkling over with finely powdered potassium

1. *K. E.* v. *Mangali, All. H. Crt., Criminal Appeal No.* 149 *of* 1921.
2. *Bull. Soc. Franc. Dermat.* 37, *No.* 4, 1930; *Ars. Medici, Nov.* 30, 1930, *p.* 124.

permanganate. After a few days the scab falls off, and a smooth, colourless almost unscarred skin appears.

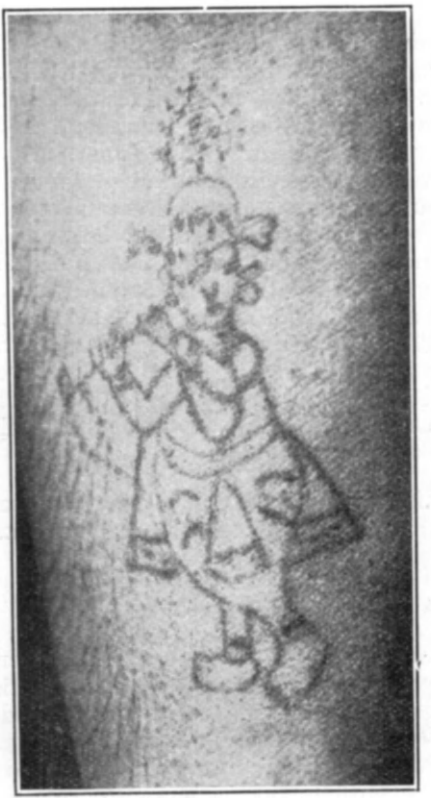


Fig. 10.—Tattoo-marks over the forearm.

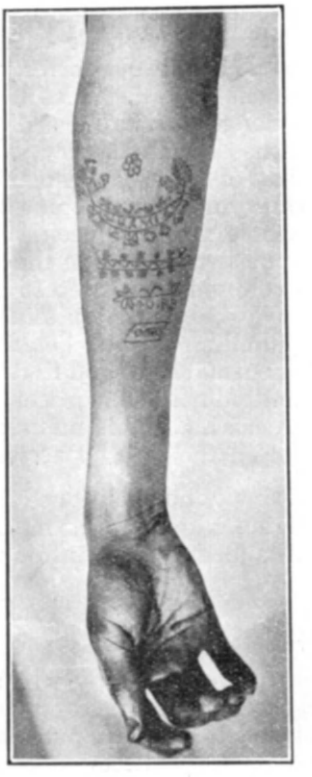


Fig. 11.—Tattoo-marks over the forearm.

(2) *Electrolysis.*—Miller[1] suggests electrolysis for the removal of tatoo-marks by means of a needle attached to the negative pole of a battery in order to get the softening action of the alkali formed there. After the pigment is laid bare by the needle, it is scraped and picked away, as the softening action of the alkali continues on the tissues in which the pigment is deposited. Another method is to insert the needle into the tattoo-mark a sufficient number of times, using a current of 5 to 2 milliamperes. This forms a superficial eschar, which drops off in the course of a week or so, taking the pigment with it, and leaving a white superficial scar.

(3) *Application of Caustic Substances.*—Caustic substances applied to the mark remove the pigment by producing an inflammatory reaction and a superficial eschar. For instance, a mixture of papain in glycerine often removes a tattoo-mark. Tardieu[2] reports the case of a criminal who

1. *Electrolysis for the Removal of Tatto-marks, Medical Council, Philadelphia* 13, 374, 1908 ; *Jour. Amer. Med., Assoc., Jan.* 14, 1928, *p.* 94.
2. *Peterson, Haines and Webster, Leg. Med. and Toxic., Ed. II, Vol. I, p.* 160.

successfully removed a tattoo-mark made of Indian ink in six days by first macerating the skin in a paste of lard and acetic acid, then thoroughly rubbing it with a solution of caustic potash or soda, and lastly with dilute hydrochloric acid. Brault[1] recommends the tattooing of a solution of zinc chloride to 40 parts of water by means of a needle into the design. After a few days a crust forms, which removes the pigment, when it falls off. These caustic substances have to be used with great care as they are not only dangerous but are often followed by disfiguring scars and keloids. Shie[2] has obtained very satisfactory results from Variot's method which consists of tattooing into the design a 50 per cent solution of tannic acid in water and then rubbing it vigorously with a stick of silver nitrate until the whole surface becomes black from the formation of silver tannate in the superficial layers of the skin. The field of the operation is then washed with cold water. After 15 or 16 days a black, dry slough comes off spontaneously resembling a thin piece of a leather and leaving a thin new layer of epithelium formed beneath it. This gradually assumes the appearance of the normal skin, and in favourable cases no scarring persists. If, however, the hair follicles have been destroyed with the tattoo-mark, there will be some scarring. This method is also suitable for the removal of blemishes caused on the face by accidental tattooing.

It may be mentioned that confluent small-pox has been known to obliterate tattoo-marks in children,[3] and chronic eczema may also cause the disappearance of tattoo-marks.

11. OCCUPATION MARKS

These are helpful in identifying unknown dead bodies, as certain trades leave marks by which persons engaged in them may be identified. For example, horny and rough hands are observed among individuals employed in hard, manual labour. *Kahars* or dooly-bearers have usually horny, callous marks on their shoulders. An Indian weighman, who has to weigh corn by lifting up a balance with heavy scales, gets a callosity, usually on the hypothenar eminence of his right palm. A depression in the lower part of the sternum is found among shoe-makers due to the constant pressure of the last against the bone. Tailors have marks of needle punctures on their left index finger, and a bursa on the lateral malleolus from the attitude of sitting adopted while sewing. Photographers, dyers and chemists generally have their fingers stained with dyes or chemicals. The occupation of a person may, sometimes, be revealed from the microscopic examination of waxy deposits from his ears and the dust and debris from under his nails, if these will show the presence of particulate matter of an organic or inorganic nature which is usually found floating in the atmosphere of factories.

12. HANDWRITING

The medical jurist is hardly called upon to give his opinion as regards the identification of handwriting, since there are experts in this line. However, according to Lord Brompton, better known as Sir Henry

1. *Jour. Amer. Med. Assoc., Aug.* 4, 1923, *p.* 409.
2. *Ibid., Jan.* 14, 1928, *p.* 94; *Brit. Med. Jour., Feb.* 25, 1928, *p.* 318.
3. *Brit. Med. Jour.,* 1871, *Vol. II, p.* 532.

Hawkins, these handwriting experts are not at all infallible, and their evidence is usually conflicting and very often fallacious. A learned Judge of the Lahore High Court has also held that in a charge of forgery, the opinion of a handwriting expert should not ordinarily be accepted as conclusive to prove the facts deposed to by him and a conviction for forgery cannot be sustained merely on the evidence of an expert.[1] Sometimes the medical man may have to examine a person to see if he is able to write when a plea of mental incapacity or some paralytic affection is raised. He should, therefore, remember that mental and nervous diseases, especially those attended with tremors, as also rheumatic diseases of the joints of the hand, alter the character of the handwriting by producing more or less irregularity in the formation of letters.

13. CLOTHES AND ORNAMENTS

These do not form any essential piece of evidence in the identification of a living person as the individual can change them at will, but they are very valuable in establishing the identification of a dead body. It is, therefore, necessary to preserve them along with any articles, such as a watch, visiting card, diary, etc., found on a dead body or lying in its vicinity for the purpose of future identification. The clothes should be examined carefully for the presence of the name of the owner or tailor or the mark of a *dhobi* (washerman) on any of them. In the Kakori conspiracy case one of the accused was identified by means of a bed-sheet found in his possession, as it had the marks of the *dhobi* employed in a hotel in Lucknow where he was alleged to have stayed for some time. The clothes should also be examined for the presence of cuts or rents or for the presence of blood, seminal or other stains. If dust of organic or mineral matter is found clinging to the clothes or in the pockets, it should be collected and submitted to microscopic examination, as it may give some indication of the business of the person.

14. SPEECH AND VOICE

There are certain peculiarities of speech, *e.g.*, stammering, stuttering, lisping, and nasal twang. These peculiarities become more evident when an individual is talking excitedly, as in a quarrel. Speech is also affected in nervous diseases, such as general paralysis of the insane and disseminated sclerosis. Defective speech depending on some organic defect of the mouth, such as cleft-palate, may be cured by a surgical plastic operation, while functional stammering can be cured without any operation.

To recognise a person from his voice is an every day occurrence, though it is too risky to be relied upon in criminal cases. In the case of *King-Emperor* v. *Bhaktu*[2] it was held that the identification of the accused in a pitch dark night by the modulation of his voice could not be relied upon for his conviction. It is possible for a person to alter his voice at will. The best example of this is the ventriloquist. The absence of the teeth, the use of false teeth, and the presence of diphtheria may alter the voice. With the progress of science it may be presumed that the

1. *King-Emperor* v. *Prabhu Dayal, Crim. Law Jour., Sept.*, 1932, *p.* 593.
2. *Lahore High Court, Crim. Law Jour., January*, 1928, *p.* 759 ; *Rangoon High Court, Crim. Law Jour., January*, 1938, *p.* 34.

registering of the voice by a gramophone disc will be used in the near future for the purpose of identification in Court.

15. GAIT

An individual can be recognised even from a distance by watching his gait, but such evidence is far from conclusive, inasmuch as the gait may be altered by an accident or disease, especially of a nervous nature, such as locomotor ataxy, hemiplegia, spastic paraplegia, etc. In civil suits the medical man may, sometimes, be requested to express his opinion if a particular individual is really lame or malingering, if he has filed a suit against his employer for the recovery of damages for an accident caused to him during his legitimate work.

16. TRICKS OF MANNER AND HABIT

These are not infrequently found to be hereditary; as an example left-handedness may be cited.

17. MENTAL POWER, MEMORY AND EDUCATION

The consideration of these points for the identification of an individual is of great importance, especially in cases of imposture, as in the well-known Tichborne case.

18. AMOUNT OF ILLUMINATION REQUIRED FOR IDENTIFICATION

In questions regarding the amount of light sufficient for recognition of the features for subsequent identification of the individual the following points should be borne in mind:—

1. A flash of lightning produces sufficient illumination for the identification of an individual.

A lady, on her passage home from India, was awakened one dark night by some one moving about in her cabin. A sudden flash of lightning enabled her to see a man bending over one of her trunks, and his features appeared so distinct that she was able next day to recognise him. The stolen articles were found upon him and he acknowledged the theft.[1]

2. According to Tidy the best known person cannot be recognised in the clearest moonlight beyond a distance of seventeen yards. Colonel Barry, I.M.S., is of opinion that at distances greater than 12 yards the stature or outline of the figure alone is available as a means of identification.[2] To define the features even at a shorter distance is practically impossible by moonlight.

3. No definite statement can be made about artificial light. The best thing is to make actual experiments with the class of light used before an opinion is given.

4. In the absence of any other light the identification of a person is possible with the flash of light produced by a firearm if the person is

1. *Montgomery, Cyclopoedia of Practical Med.; Peterson, Haines and Webster, Leg. Med. and Toxic., Ed. II, Vol. I, p.* 166.
2. *Legal Medicine, Vol. I, p.* 572.

standing in close proximity of five to twenty paces on one side of the line of fire and if the powder is at the same time smokeless, though it is not possible to mark the different characters of the features beyond three paces. In such cases an experiment should be tried with the weapon and powder used before an opinion is given.

CASES OF MISTAKEN IDENTITY

I. **The Burdwan Case.**—Pratap Chandra, the only son of the Raja of Burdwan, died in 1820-21 during his father's lifetime. In 1835, a pretender in the garb of a *Sanyasi* called himself Pratap Chandra and claimed the Burdwan estate. He said that he had not died as was alleged, but had been living in secret retirement and had now returned. His story was believed by certain people who created a breach of the peace for which he was sent to jail. On being released he was given monetary help by some of his old adherents and he went with a large following to take forcible possession of the estate, causing a disturbance, in the quelling of which three persons were shot by the military police. He was charged with having fraudulently assumed the name and title of the deceased Raja. The death of the real Pratap Chandra was testified to by the doctors who had treated him, by persons who were present at cremation and by the priests who performed the *Shradh* ceremonies. Mr. H. T. Princep, Secretary to the Government of India, and several others who had met Pratap Chandra declared the claimant to be an impostor. On the contrary, General Allard, Major Marshall, Dr. Scott, Civil Surgeon of Burdwan, Dr. Holliday and others acknowledged him to be the real Pratap Chandra. Several witnesses deposed that he was Kristo Lal, son of a priest, a former resident of Burdwan. The features of the prisoner, especially in the shape of the nose and the colour of the eyes, were similar to those of Pratap Chandra. Certain other marks of identification were also common to both; *viz.*, a slight mark behind the right ear produced by the glazed string of a kite, a scar between the shoulders caused by the bite of a horse, a mark on the knee and a scald mark of the size of an eight-anna piece on the left hand. The case having been proved against the prisoner, the High Court sentenced him to a fine of Rupees one thousand.—*Waddell, Lyon's Med. Jur., Ed. VIII, p.* 62.

II. **The Tichborne Case.**—In 1874, Arthur Orton, a butcher's son, came forward claiming to be Sir Roger Tichborne who was supposed to have died at sea in 1854, and thus claimed to be the heir to the Tichborne estates in Hampshire. The following were the chief points that were discussed at the trial in London:—

1. It was illustrated from the photographs produced that the eyes of Roger Tichborne were blue and directed upward from the middle line while those of the claimant were of dark slate colour and directed downward from the middle line. The eyebrows of Roger Tichborne were wide apart and well defined, while those of the claimant were much nearer together, and of ill-defined outline. The ear of the claimant was longer by one-third, the greater length being largely due to the detached pendulous lobe, which did not exist in Roger Tichborne. The nose of the claimant was a narrow one with broad face, while that of Roger Tichborne was a broad one with inflated nostrils in a thin face. The central groove which joins the nose to the upper lip was narrow in the claimant and wide in Roger Tichborne.

2. Roger Tichborne was able to speak French fluently, but the claimant did not know the language at all. He could not recollect his alleged mother's name, nor his birth place nor the companions of his childhood and youth.

3. Roger Tichborne had tattoo-marks near the left wrist and on the left fore-arm, but the claimant had none.

4. Roger Tichborne had a scar on the left shoulder caused by an issue kept standing for three years. The claimant had a scar in this situation, but it was the result of several insertions of vaccine matter at points equidistant. He had also another scar above the left wrist.—*Guy and Ferrier, Forens. Med., Ed. VI, pp.* 14, 15, 16, 17.

III. **Adolf Beck's Case.**—In 1877, one John Smith was convicted of theft at the old Bailey, London, and sentenced to five years' imprisonment. The following marks of identification were noted on his admission to the jail:—

1. Circumcised, hence a Jew.

2. A marked scar on the point of the jaw on the right side.

In 1896, a man who called himself Adolf Beck was charged with similar offences. He was identified by five women as the man who had defrauded them and was identified by a police officer as John Smith convicted in 1877. On comparing the documents found in Beck's possession at the time of his arrest with written documents given by the offender to the defrauded women, a handwriting expert said that he was "perfectly satisfied that they were all in the self-same handwriting" but disguised. Beck asserted that he was not John Smith and pleaded an *alibi* by declaring that he had been in Lima from 1875 to 1882. But the Judge did not believe in the *alibi* and sentenced him to seven years' imprisonment. After he had served two years of his term of imprisonment in Portland the authorities discovered that Beck was not circumcised, and hence he could not be Smith as the latter had been circumcised. Owing to his good conduct in jail he was released in July, 1901, two years before the expiry of his term of sentence, but, in 1904, was rearrested by an Inspector of Police for similar fraudulent thefts. When his case was pending in Court, a man was apprehended for defrauding two women of their jewellery. A Chief Inspector of the Metropolitan Detective Department identified this prisoner as the real John Smith by the scar on the point of his jaw which had not been found on Beck. John Smith had also a conspicuous wart on one eye, while Beck had a scar on the left arm from a cut by a sabre. On this discovery being communicated to the authorities Beck was at once released and was granted a free pardon for previous convictions of crimes of which he was quite innocent.—*Glaister, Med. Juris. and Toxic., Ed. V, p.* 104.

CHAPTER III

POST-MORTEM EXAMINATION (AUTOPSY)

The Object.—The object of the post-mortem examination of a body is to establish its identity when not known, and to ascertain the time since death and the cause of death; but in addition, the question of live birth and viability has to be determined in the case of the body of a newly-born infant.

Rules.—The medico-legal post-mortem examination should never be undertaken unless there is a written order from the Superintendent of Police or the District Magistrate. Before commencing the examination, the medical officer should carefully read the police report on the appearance and situation of the body when it was first discovered, and the cause of death as far as could have been ascertained. This precaution is necessary, especially in the case of a decomposed body, so as to enable him to examine particularly the organ or the part of the body most suspected for the evidence of death.

The examination should be conducted in daylight, and not in artificial light. It should also be as thorough and complete as circumstances permit. The three great cavities and the organs contained in them should all be carefully examined even though the apparent cause of death has been found in one of them, just to avoid unnecessary and, sometimes, unpleasant cross questions in Court, inasmuch as evidence of factors contributory to the cause of death may be found in more than one organ.

Ordinarily a dead body is sent to the morgue but in exceptional cases the medical officer may be taken to the place where the dead body is lying. In that case he should note the place and nature of the soil where he found the dead body, and also its position especially as regards the hands and feet and the state of the clothes, if any. He should also note, in cases of death from violence, the position of the body in reference to surrounding objects, such as sharp stones and the like, contact with which, it might be alleged, had produced the injury, and also whether any blood-stains were visible on such objects or anywhere near the corpse, and whether any weapons were lying near it. The ground in the vicinity should be carefully searched for the presence of footprints and evidence of any struggle. In the case of suspected death from poisoning, he should note whether any appearance as of vomited matter, etc., was present in the neighbourhood of the body.

All the details observed by the medical officer should be carefully entered on the spot by himself in the post-mortem report or in a note-book, which can be used as evidence in a legal inquiry. He should not mind the report getting soiled; this will enhance its value, inasmuch as it goes to prove that it was written at the time when " facts were still fresh in the mind." If there is an assistant, the best plan is to dictate to him as the examination proceeds step by step, and then read, verify and attest

the report. It is not safe to trust to memory and to write the report later on after completing the examination. The notes and the report to be sent to Court must tally with each other. There should be no discrepancy. Nothing should be erased, and all alterations should be initialled.

The medical officer holding a post-mortem examination should be familiar with the normal and pathological appearances of the viscera. He should note the time of the arrival of the body at the morgue, the date and hour of the post-mortem examination and the name of the place where it was held. The necessary papers authorising the medical officer to hold an autopsy are frequently brought by the police long after the body has arrived. This dilatory method on the part of the police has occasionally led to the decomposition of the body in the post-mortem room even when it has arrived in a good condition. It is, therefore, safer to note the exact time of delivery of these papers. There should be no unnecessary delay in holding the post-mortem examination. It should be made as soon as the papers are brought, and the excuse of attending upon a midwifery case or any other similar reason should not prevent him from performing this most important, though too frequently unpleasant, duty.

No outsider should be allowed to be present at the autopsy.

Instruments.—The following instruments should be at hand before commencing the examination :—

1. Scalpel. 2. Large Section knife. 3. Dissecting forceps. 4. Scissors. 5. Saw. 6 Costotome. 7. Enterotome. 8. Blunt probe. 9. Blow pipe. 10. A pair of iron hooks. 11. Curved needles. 12. Strong twine. 13. A measuring tape. 14. Measuring and graduated glass containers. 15. China plates. 16. Basins to contain water. 17. Sponges. 18. A pair of thick India-rubber gloves with gauntlets or photographic gloves. 19. Machine for weighing organs. 20. At least two wide mouthed, white glass bottles (with glass stoppers) of about one litre capacity, to contain portions of viscera.

EXTERNAL EXAMINATION

The following steps should be followed for the external examination :—

1. The body should be identified by the police constable and the *chaukidar*, who brought it to the mortuary. It should also be identified by a relative or friend of the deceased present at the spot. These persons will be required to give evidence in Court of having identified the body in the presence of the medical officer holding the post-mortem examination, in case a person is tried for having caused the death of the deceased.

2. In the case of an unknown body, a general appearance of the body describing the race, sex, age, stature, features, scars, tattoo-marks, etc., should be noted for the purpose of identification. The body should be photographed and the finger prints taken. The police should arrange for such a body to be photographed at once, before it gets decomposed. The

photograph is worthless after the features have become bloated and distorted from putrefaction, but I have seen bodies photographed even after advanced putrefaction. At the request of the police the head may be preserved, for future identification, in methylated spirit and a little formalin in a large closely fitting glass jar or any other receptable.

3. If there are clothes on the body, they should be carefully examined for stains of mud, tears, etc., indicating a struggle, before they are removed. Stains of blood, semen, vomit or fæcal matter should be described and preserved for chemical analysis. Cuts or rents caused by a cutting instrument, burns caused by fire or acids, or blackening caused by discharges from firearms should be carefully noted and compared with injuries on the body.

4. In the case of a cord or ligature round the neck, its exact position, manner and application of a knot, or knots and its material should be noted.

5. The age should be given from the presence of the teeth and other appearances. If, owing to rigor mortis, the jaw cannot be opened to count the teeth, the cheeks should be cut to expose them.

6. The time since death should be noted from the temperature of the body, post-mortem staining, rigor mortis, stage of putrefaction, and even from the degree of digestion of the stomach contents which, however, only yields evidence of doubtful value.

7. The condition of the body, whether stout, emaciated, or decomposed, should be mentioned. The eyes should be examined and the opacity of the cornea and lens should be noted particularly in vehicular accidents. The state of the pupils should also be noted as to whether they were contracted or dilated.

8. The natural orifices, *viz.*, nose, mouth, ears, anus, urethra and vagina, should be examined for the presence of injuries, foreign bodies or discharges, such as blood, pus, etc. The mouth and nostrils should be particularly examined for the presence of froth, and the position of the tongue should be noted in connection with the front teeth.

9. The hands should be examined for any article, such as hair, fragments of clothing or a weapon grasped by them or the presence of mud or blood on them or under the nails.

10. The direction of blood smears and the signs of spouting of blood should be noted, if any.

11. The situation of post-mortem staining, if present, should be noted.

12. After washing the body a careful search for the presence of injuries or marks of violence should be made all over the body from head to foot. on the front as well as on the back. In the case of a female

body the hair of the head should be removed to examine the scalp. If any injuries are found on the body, they should be photographed or marked carefully on sketches, before they are described in detail in the post-mortem report. Such a procedure is very helpful in enabling the Magistrate and counsel of both sides to understand the exact nature, extent and situation of the injuries on the body.

Contusions and abrasions, if any, should be described as regards their length, breadth and their exact positions. Contusions should be incised to find out if they were inflicted before or after death and to differentiate them from suggilation.

Wounds. if present, should be described as regards their nature, size, direction and position. The conditions of their edges should also be mentioned. The exact size ought to be noted with a measuring tape and some fixed bony points should be taken to describe their exact position. The means by which they were inflicted should also be noted.

In the case of gunshot wounds the course and direction of the bullet should be ascertained by dissection rather than by the use of a probe, and the injured nerves and blood-vessels, if any are found, should be noted. If there is only one opening, a search should be made for the bullet, which must be preserved. It should be remembered that a bullet takes a very tortuous and erratic course in its passage through the body. A note should also be made, if the skin in the vicinity of the wound is blackened and if the hair is scorched.

Ligature marks or finger marks, if present on the neck, should be noted.

In the case of burns, their position, extent and degree should be mentioned, as also the manner of their causation as to whether they were caused by fire, scalding fluids, corrosives or explosives.

Penetrating or deep wounds should not be investigated by means of a probe, until the body is opened.

13. All the bones should be carefully examined for the presence of fractures, and the joints for dislocations. If any fracture is present, the soft parts overlying the fractured piece should be dissected and examined for laceration or ecchymosis. Lastly, all the external injuries should be compared with those noted in the descriptive roll supplied by the police and any discrepancy should be mentioned in the report.

14. In the case of the body of a newly-born infant it is necessary to examine the lower epiphysis of the femur for the centre of ossification to prove its maturity. For this purpose, the knee joint should be opened by making an incision across its front and the lower end of the femur should be pushed through the wound. The epiphysial cartilage should now be sliced off in thin sections until a central pink spot is seen. The slicing of the cartilage should be continued till the greatest diameter of the ossified point is reached.

INTERNAL EXAMINATION

If there be a fatal wound leading to one of the cavities, that cavity should be opened first, or the head should be opened first, and then the thorax and the abdomen. Professor Harvey Little John[1] recommends the examination of the head first in cases of alleged infanticide so that the examiner may have an opportunity of inspecting the contents of the skull before the blood can possibly drain away during the examination of the thorax and trunk, and also because he will be better able to interpret appearances in the lungs when they are examined. Every organ contained in the cavities must always be examined, but the spinal cord need not ordinarily be examined unless there is suspicion of some injury to the vertebral column or the alleged cause of death is due to some spinal poison or some such disease as tetanus. In that case it should be examined last of all.

Fig. 12.—Section through the lower epiphysis of the femur showing the centre of ossification.

HEAD

A transverse incision across the vertex should be made from ear to ear and, after reflecting the flaps anteriorly up to the orbits and posteriorly up to the occipital protuberance, the inner surface of the scalp should be examined for extravasation of blood and the skull bones should be examined for evidence of any fracture or separation of sutures after the periosteum is denuded and the temporal muscles have been dissected off. In a doubtful case the skull should be tapped with a hammer; it would elicit a ringing note if there is no fracture. To remove the skull cap a circular cut is then made with a saw round the cranium keeping close to the reflected flaps. Its inner surface should then be examined for fracture of the inner plate, or effusion of blood, which may be found on the dura mater. The longitudinal venous sinuses should be examined for evidence of laceration or thrombosis. The dura mater should be removed by cutting longitudinally along both the sides of its middle line noting any effusion of blood or serum, or the presence of pus. It would not be out

1. *Trans. Medico-Legal Soci., Vol. XVI, p.* 88.

of place to note here the distinction between meningitis and mere effusion of blood in the meninges. In the former the surface of the brain looks greasy and dull, but not so in the latter. The brain should now be removed by raising the anterior lobes with the fingers of the left hand and cutting through the various nerves at its base and the medulla as low down as possible. The brain should be placed in a large clean receptacle or dish and examined minutely on its upper as well as under surface (base) for the evidence of any injury, effusion of blood, inflammatory products, embolism of arteries, morbid growths or any disease of its tissue. The ventricles should also be examined.

The dura mater should lastly be stripped from the base of the skull to facilitate its examination for the presence of fractures.

THORAX

Before examining the thorax, both the cavities, the thorax and the abdomen, should be opened by making a longitudinal incision from above the middle of the sternum to the pubic bone, keeping wide away from any wounds existing in its line. In infant bodies the incision should be carried a little to the left of the umbilicus. The integument, fascia and muscles should now be reflected and examined for extravasation of blood in their inner surface. The abdominal cavity should be examined before the chest cavity is opened. The colour and appearance of the abdominal viscera, as also the position of the diaphragm with respect to the ribs (especially in full term newly-born infant bodies) should be noted. It should also be noted if there is any collection of blood, serum, pus, or fæcal matter in the cavity.

After this preliminary, the examination of the thorax should be proceeded with. The ribs and sternum should first be examined for evidence of fracture and then the cavity of the thorax should be opened by dividing the ribs at their cartilages and the sternum at the sterno-clavicular junctions with the costotome and lifting up the sternum separating it from the underlying parts without injuring them. The pleural cavities should be examined for the presence of adhesions, foreign bodies or fluid of a bloody or purulent nature. The pericardium should be opened and examined for any adhesions between its two surfaces or if there is any abnormal quantity of fluid present in its cavity. Normally about a drachm of bloody serum is found in the pericardial sac. The condition of the chambers of the heart should be examined by opening them *in situ*. The lungs and heart should be removed from the cavity and laid on the table. The lungs should be cut open for evidence of disease, congestion, injury, Tardieu's spots, etc., and the bronchi should be examined for the presence of pent up expectoration, pus, or any foreign body. The heart should be opened and its chambers examined for the presence of valvular disease, and the condition of the endorcardium should be noted. The aorta should be examined for aneurysm or calcareous degeneration.

To examine the larynx, trachea and œsophagus an incision should be made from the chin to the upper part of the sternum after throwing the head well backward and placing a block of wood beneath the neck. After reflecting the soft parts on each side of the middle of the larynx, both the trachea and the œsophagus should be removed and examined by

cutting them open from their posterior surface. The interior of the œsophagus should be examined for evidence of congestion, inflammation or ulceration of its mucous membrane, and the presence of a foreign body, tumour or stricture. The larynx and trachea should be examined for the presence of froth or a foreign body in their interior, and their mucous membrane should be examined for congestion or inflammation.

ABDOMEN

The peritoneum should be first examined for evidences of adhesions, congestion, inflammation, or exudation of lymph or pus. The abdominal and pelvic cavities should then be examined for the presence of a serous, bloody or purulent fluid. Now the abdominal organs should be removed and examined separately as below :—

Stomach.—Under ordinary circumstances the stomach is examined by making a cut while *in situ* for the contents as regards their quantity and quality, and the degree of their digestibility. But in suspected poisoning the stomach should be removed after tying a double ligature at both ends. It should then be opened in a thoroughly clean plate ; after emptying the contents its mucous surface should be carefully examined noting its appearance, and any suspicious particles found adherent thereto should be picked off with a pair of forceps and placed in a separate small phial for chemical analysis. The contents of the stomach should also be examined as regards their smell, colour and character and for the presence of any foreign particles or lumps ; these, if present, should be felt between the thumb and index finger as to their roughness or smoothness.

For the purposes of chemical analysis the following articles should be preserved separately in clean, wide mouthed, white glass bottles with glass stoppers.

1. The stomach and its contents.

2. A portion of the liver, not less than 16 ounces in weight, or the whole liver if it weighs less than 16 ounces, one kidney and the spleen.

If the spleen is very large, a portion only need be preserved.

If there is suspicion of a vegetable poison having been used, the following articles, in addition, should be preserved.

3. The upper part of the small intestine with its contents.

4. Any urine found in the bladder.

5. The heart and a portion of the brain, if strychnia or nux vomica is suspected.

According to the revised rules of the U. P. Government the stomach and its contents are to be preserved in one bottle and pieces of the liver, spleen, kidney and of the upper part of the small intestine, in another bottle but it is advisable to preserve the stomach and its contents together with a piece of the upper part of the small intestine in one bottle and pieces of the liver, spleen and kidney in another bottle. In the case of infants one bottle is quite sufficient. These viscera are to be preserved in rectified spirit except in cases of suspected poisoning by alcohol, phosphorus,

paraldehyde or carbolic acid and other drugs of the phenol group, when a saturated solution of common salt is to be employed.[1] It should be remembered that the quantity of the spirit or the saturated solution of salt should be equal to that of the viscera in bulk. The viscera and spirit together should not fill the bottle, but only reach to two-thirds of its height, in order to diminish the risk of the bottle bursting in case any gas is given off.[2] A sample of the preservative used—either rectified spirit or saturated solution of common salt—should always be forwarded in a separate phial to the Chemical Examiner.

In addition to these viscera, blood and lung tissues should be preserved without adding any preservative in cases of suspected poisoning by carbon monoxide or hydrocyanic acid and should be sent for chemical examination as soon as possible.

Unless the viscera are forwarded to the Chemical Examiner, they are to be preserved for a period of six months, and are then to be destroyed after obtaining the District Magistrate's assent.

Intestines.—The intestines should be removed after ligaturing at both ends and should be cut longitudinally to examine the inner surface for the presence of congestion, inflammation, erosions, ulcers, perforation or any other lesion. In cases of suspected poisoning the contents should be preserved and sent for chemical analysis wherever possible, as they may, sometimes give valuable clues as to the nature of the poison.[3]

Liver.—The surface of the liver should be examined as regards its smoothness or roughness. If there is any injury to the liver, its nature and dimensions should be noted as well as the size and weight of the liver. Normally the liver measures 12" × 7" × 3½". The organ should be cut open by deep incisions in several places, and the colour, consistence and blood supply of its tissue should be carefully marked; at the same time the presence of an abscess, new growth or amyloid degeneration should be observed.

The gall bladder should be opened and the presence or absence of bile stones and the character and quantity of the bile should be noted.

Spleen.—The size, colour and consistence of the organ should be noted as well as the condition of its capsule. In the case of a rupture of the spleen, its size and position should be described, as well as the size and weight of the spleen. The normal spleen in the adult measures 5" × 3" × 1".

Kidneys.—The size, colour and weight of the kidneys should be noted. Normally the size of a kidney is 4" × 2" × 1". Its capsule should be examined as to whether it is adherent or strips off easily. The kidney should be cut open and the internal cut surface should be examined for the presence of Bright's disease or amyloid degeneration; the pelvis should be examined for calculi and evidence of inflammation.

---

1. *The U. P. Med. Manual*, 1934, *p.* 224; *Bombay Civil Medical Code*, 1926, *p.* 152; *Directions for forwarding cases to the Chemical Examiner, Bengal, for Medico-Legal Examination*, 1937, *p.* 4.
2. *The U. P. Medical Manual*, 1934, *p.* 223.
3. *The U. P. Medical Manual*, 1934, *p.* 225.

Bladder.—The bladder should be examined for congestion, hæmorrhage, inflammation and ulceration of its mucous membrane. It may be opened *in situ* and its contents noted, but in a suspected case of poisoning the urine should be removed and preserved for chemical analysis in a clean glass phial with an equal quantity of rectified spirit or with fine grains of thymol if rectified spirit is contraindicated. The urine may, sometimes, give a valuable clue as to the nature of the poison.

Uterus.—In female bodies the uterus should always be examined for its size and shape. The normal size of the organ is 3"× 2" × 1" and weight, from one to one-and-a-half ounces; but the size and weight vary considerably during pregnancy or when there is any tumour. The condition of its mucous membrane and the thickening of its wall should be examined after the uterus is opened longitudinally. During menstruation the mucous membrane is thickened, softer and of a darker colour, and covered with blood and detritus. In old age it becomes atrophied, and paler and denser in texture. If the uterus contains a fœtus, the age of its intra-uterine life should be noted. The ovaries and Fallopian tubes should also be examined. The ovaries should be chiefly examined for corpora lutea. The vaginal canal should be opened and examined for the presence of a foreign body or marks of injury. The colour of its mucous membrane and the condition of the hymen should also be noted.

In fatal cases of suspected criminal abortion the uterus and its appendages together with the upper part of the vagina should be preserved for chemical analysis, if thought necessary. Sticks or other foreign bodies found in the genital tract should be preserved in a separate bottle after removal and drying when practicable.

Spine and Spinal Cord.—The spinal canal need not be examined unless there is any indication of disease or injury. If necessary, the body should be turned over on the face with a block beneath the thorax and an incision made along the entire length of the vertebral column extending from the occiput to the lower end of the sacrum. After reflecting the integuments, dissecting away the muscles and noting extravasation of blood in the soft tissues, the laminæ should be sawn through vertically on each side and the detached portions removed, when the dura mater would be exposed. After noting its appearance, the dura mater should be opened and an examination made for the presence of hæmorrhage, inflammation, suppuration or tumour. The cord should now be removed, laid on the table, cut transversely in several places, and examined for evidences of hæmorrhages, softening and inflammatory lesions.

The vertebral column should be examined for the presence of fractures or dislocations after the cord has been removed.

As soon as the post-mortem examination is finished, the body should be thoroughly washed, the organs should be replaced into the cavities, and the dissected flaps should be brought in apposition and well sutured with strong twine. The body should then be covered with a cloth before it is returned to the relatives or friends so as to avoid hurting their feelings. In the absence of the relatives or friends the body should be returned to the police constable accompanying it, who should cremate or bury it according to the religious customs of the deceased, but should never throw it into a running stream or river as is often done.

Table showing the weights of the chief viscera of healthy Indians of the United Provinces of Agra and Oudh, varying from 10 to 70 years of age, who died from violence.

Organs.	Males.			Females.		
	Weight in ounces.			Weight in ounces.		
	Min.	Max.	Average.	Min.	Max.	Average.
Brain	35	57	47.34	30	48	38.29
Right Lung	7	30	18.15	6	20	13.5
Left Lung	5	30	16.58	5	18	11.75
Heart	3.5	13	10	4	8	6.5
Stomach	3	9	5.6	3.5	8	5.75
Liver	26	64	43.78	30	50	38.25
Spleen	2.5	11	6.03	2	9.5	5.14
Right Kidney	2.5	6	3.64	2	4	3.08
Left Kidney	1.5	6	3.63	2	4	2.95

THE CAUSE OF DEATH

After completing the post-mortem examination, the medical officer should form an opinion as to the cause and manner of death, based on the appearances observed by him and should immediately give in the vernacular the abstract of his opinion in the police form No. 289 (Appendix IV) to the police constable accompanying the body for communication to the investigating officer. If he has based his opinion on the post-mortem appearances, as well as on the statement of the police, he should mention the fact in his report. The report should be as complete as possible, but concise and clear ; it should be forwarded to the Superintendent of Police as soon as possible, but not later than two days. Post-mortem reports drawn up by Civil Assistant Surgeons (members of the Provincial Medical Service in charge of dispensaries) have to be countersigned by Civil Surgeons, but this appears to be unnecessary and meaningless, as responsibility still rests with Civil Assistant Surgeons.

Some medical officers labour under a mistaken belief that they should never be definite in their opinion as to the actual cause of death, and should, therefore, qualify their opinion by using the word, " probably ", in their post-mortem report. This dictum is, sometimes, carried so far that instead of helping the Judge to come to a definite conclusion their opinion unnecessarily creates a bad impression on his mind. For instance, a Civil Surgeon mentioned in a case where a man was murdered by the discharge of a gun in the abdomen that in his opinion death was *probably* due to shock and internal hæmorrhage resulting *probably* from the wound in the abdomen which was *probably* caused by the discharge of a fire-arm. In cross-examination he had to admit that there was no possibility of any

other cause of death in the case, and he used the word, "probably", so often in his report, as it was customary to do so among medical officers. In connection with the use of the word, "probably", by medical officers in their post-mortem reports the Sessions Judge of Agra made the following interesting observations in the course of his judgment in the case of K. E. *v.* Gulkandi charged under section 302 of the Indian Penal Code:—

"I have already drawn the attention of the District Magistrate to the deplorable manner in which medical evidence is often recorded in Magistrates' Courts. Medical officers appear to derive some inward satisfaction from the use of the word, "probably", in giving the cause of death and Magistrates blindly record such statements. In this case I had to ask the Magistrate to recall for proper examination the medical officer whose evidence was for the purpose of this case vitiated by the use of the word, "probably". A medical officer should be asked, what, in his opinion, was the cause of death and every effort should be made to root out the vagueness against which I am perpetually fighting in Court, in this, as in other matters."

In a case where a jugular vein and an external carotid artery were cut in an incised wound, 4" by ½" by 1", the medical officer holding the post-mortem examination gave an opinion that the deceased appeared to have died from the wound of the neck, thus implying that the actual cause of death might be something else.

My advice to medical officers is that they must never hesitate to give a definite opinion whenever they can reasonably do so. But in those cases, where they are unable to find any cause of death, all the organs being healthy, and there being no injury sufficient to account for death, they must mention in their post-mortem report that they cannot come to any definite conclusion, and in doing so they must explain their position by reasoning out all the facts. In such cases it is advisable as a precautionary measure to preserve the necessary viscera for chemical analysis and pieces of the brain, lungs, liver, spleen, etc., for microscopic and bacteriologic examinations.

EXAMINATION OF DECOMPOSED BODIES

The examination should be complete and should be held on the same lines as in ordinary autopsies. To save hand work on decomposed bodies and thus to lessen the chances of septic poisoning a pair of hooks made of ¼" iron or steel 9" long and with 3" bent in to form a handle is very convenient for hooking up the abdominal and other incisions so as to keep the parts open and also for opening the pericardium and hooking up the heart, lungs and other organs.[1]

In cases of external fatal injuries it is not difficult to find out the cause of death. In October, 1930, the body of a Hindu male, 40 years old, was brought in a very advanced state of decomposition with a police report that "death was caused by the deceased being thrown into a well with the hands and legs tied together." On examination I found that the lower limbs were flexed at the hip joints and passed over the trunk near the neck where the hands, feet and neck were tied together by several

1. *Circular No. 52 of the I. G. C. H., U. P.*, 1910; *The U. P. Medical Manual*, 1934, *p.* 213.

turns of a loin cloth (*dhoti*). The soft tissues from over the trunk had given way exposing the thoracic and abdominal cavities. The buttocks and soft tissues of the upper and lower limbs had undergone saponification. The windpipe and gullet were cut through below the thyroid cartilage. There were two cuts obliquely across the front of the left

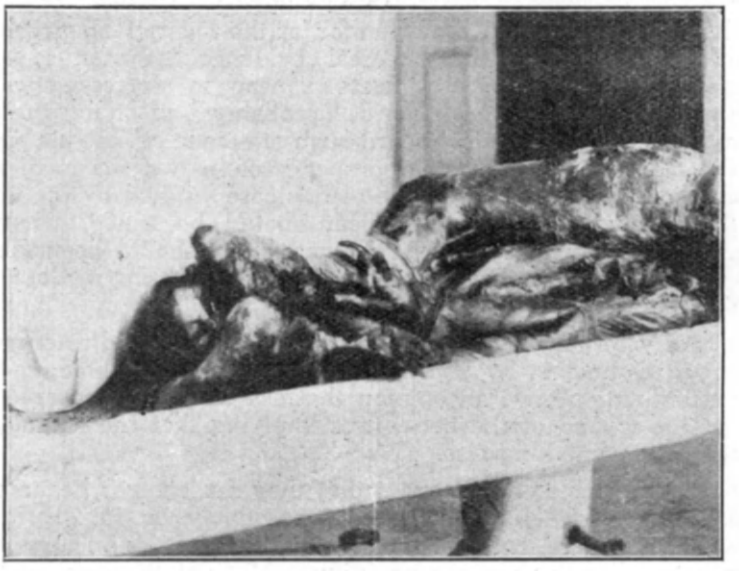


Fig. 13.—Decomposed body of a Hindu male with hands, feet and neck tied together.

seventh rib and three vertical cuts through the upper part of the sternum near its junction with the cartilage of the left first rib. The thoracic and abdominal organs were mostly absent. I gave my opinion that the deceased was first killed by a wound on the neck and stab wounds on the chest, and then tied with a *dhoti* and thrown into a well (Fig. 13).

In cases of strangulation and hanging the cord mark would be apparent, even if the skin had peeled off, as the skin on and round about the mark persists for some time. In a case of hanging I found a ligature mark in the neck on the sixth day after death when the body had been putrefied to a large extent.

The presence of mud in the right bronchus at the post-mortem examination held on the fifth day after death when the body was advanced in putrefaction led me to form a diagnosis of drowning.

In a fracture of the skull bones disorganised clotted blood may be found on their inner plates, or on the surface of the dura mater and on the brain in spite of its soft and pulpy nature if decomposition has not far advanced; but the mere effusion of blood on the brain would not be enough to warrant a statement that the fracture was caused before or after death. In doubtful cases a guarded opinion should be given that the injuries found on the body, if inflicted during life, were sufficient to cause death and that they might have been caused by such and such a weapon.

The necessary viscera should always be preserved for chemical analysis in those cases where the cause of death cannot be found owing to advanced decomposition.

EXAMINATION OF MUTILATED BODIES OR FRAGMENTS

Mutilation of a dead body is not always the act of a criminal, who wants to destroy all traces of identity and thus to get greater facilities for its disposal. In India, animals, such as jackals, dogs, vultures, etc., mutilate a body in a very short time if exposed in the open after death. Besides, it is not an uncommon sight to notice the dead bodies of lunatics, *fakirs* and pilgrims, lying on the road side or on remote spots in the vicinity of villages, and attacked by birds of prey, dogs and other animals. If the village *chaukidar* happens to find such a mutilated body, he hurriedly runs to the police station to make a report about his wonderful discovery, and the remnants of the body are forwarded to the Civil Surgeon for post-mortem examination.

In such cases the medical examiner should first ascertain if the parts sent are human or not. This is only difficult when a piece of muscle without the skin or a viscus is sent. In such a case a definite opinion can be given by resorting to the precipitin test which is equally applicable to blood as well as muscle or any other soft tissue. Having determined that they are human he should try to elucidate the following points :—

1. All separate parts should be fitted together, and it should be determined whether they belonged to one and the same body.

2. The nature and character of the parts should be described, as also the colour of the skin, if any.

3. The manner of separation as to whether they had been hacked, sawn through, cut cleanly, lacerated, or gnawed through by animals.

4. The sex can be determined, if the head or trunk is available, from the presence or absence of the hair and general conformation.

5. The probable age may be ascertained from the skull, teeth, colour of the hair, trunk, size and degree of development of fragments, and ossification of the bones.

6. Identification can be determined by tattoo-marks, scars, colour of the hair, deformities, recent and old fractures, or by the discovery of certain articles of clothing known to have belonged to a missing person in association with the mutilated bodies or fragments of a skeleton.

7. The probable time since death may be ascertained from the condition of the parts.

8. The cause of death can be ascertained, if there is evidence of a fatal injury to some large blood vessel or some vital organ. For instance, a penetrating wound on the left side of the chest cutting the left ventricle of the heart was noticed on the mutilated body of a Hindu male packed in a steel trunk and found lying in a first class compartment of No. 6 down train of R. M. Railway at Agra Fort Station on the 7th August, 1909. The head, upper half of the lip, penis and extremities had been severed from the trunk.

In September, 1922, a body found in a well in a very advanced state of decomposition was sent for examination from Police Station, Hasanganj, Lucknow. All the internal organs had disappeared except a small portion of the small intestine and the uterus. The lower jaw and the hands were missing. The skull was denuded free of soft tissues, but had a depressed fissured fracture at the junction of the parietal bones with the frontal. There was a necklace of glass beads round the neck, the soft parts of which were destroyed in front by maggots, which were crawling all over the body. The body appeared to be that of a Hindu female who had been killed by fracturing the skull bone with a blunt weapon and then thrown into the well.

The Ruxton Case.—On the 29th September, 1935, several mutilated and dismembered human remains, consisting chiefly of two heads, thorax, pelvis, segments of the upper and lower limbs, three breasts, portions of female external genitals, and the uterus and its appendages, were found lying in the bed of Gardenholme Linn, below the bridge on the Moffat-Edinburgh road. With a view to efface all evidence of sex and identity the ears, eyes, nose and lips had been removed from both the heads. The skin of the faces had also been removed and the teeth had been extracted. The terminal joints of the fingers had been removed from the hands, so that no identification could be possible from finger-prints or some peculiarity of the nails or finger-tips. All the remains were assembled and found to represent two female bodies, apparently well developed and well nourished. From investigations carried out by several specialists it was proved beyond doubt that these bodies were those of Mrs. Isabella Ruxton, the wife of Dr. Ruxton, aged about 35 years, and Miss Mary Rogerson, the nurse-maid of Dr. Ruxton, aged about 20 years, who had both disappeared from the house of Dr. Ruxton in Lancaster on the 15th September, 1935, and were never again seen alive. Photographs were taken of the skulls and superimposed on those of the heads of Mrs. Ruxton and Miss Rogerson, and were found to tally in every respect. Casts made of the reconstructed left feet of both the bodies fitted perfectly shoes belonging to Mrs. Ruxton and Miss Rogerson.

The police searched the house of Dr. Ruxton and found numerous stains of human blood in the bath room, on the bannister, stair rails, stair carpets, pads, surgical towel, and a suit of clothes belonging to him. The police subsequently arrested Dr. Ruxton, who was charged with having wilfully murdered Mrs. Isabella Ruxton and Miss Mary Rogerson. He was found guilty of murder and sentenced to death.[1]

EXAMINATION OF BONES

When a skeleton or isolated bones are sent for medical examination, the usual questions that a police officer puts to a medical officer are: (1) whether the bones are human or not; (2) if human, whether they are male or female; (3) whether they belong to one or more individuals; (4) the stature of the individual to whom the bones belonged; (5) the age of the individual to whom the bones belonged; (6) the time of death; (7) whether the bones have been cut, sawn, gnawed by animals or burnt; (8) the probable cause of death.

The above questions may be answered by observing the following points:—

(1) Owing to prevailing ignorance the police as well as the public not infrequently mistake the bones of animals, especially dogs, pigs and goats for those of human beings. Thus, a village *chaukidar* in the district of Lucknow mistook a few bones of a bird lying near a tree in a field for those of a newly born infant, suspected a case of criminal abortion and sent them for medical examination. In a suspected case of murder in the District of Meerut during the month of September, 1921, several bones

1. *For full details see John Glaister and James Copper Brash, Medico-Legal Aspects of the Ruxton Case,* 1937.

were picked up by the police and forwarded to me for expert opinion. Among these the bones of the upper extremity were human while the remaining including the jaw and skull were animal bones. The knowledge of human as well as comparative anatomy is, therefore, necessary to find

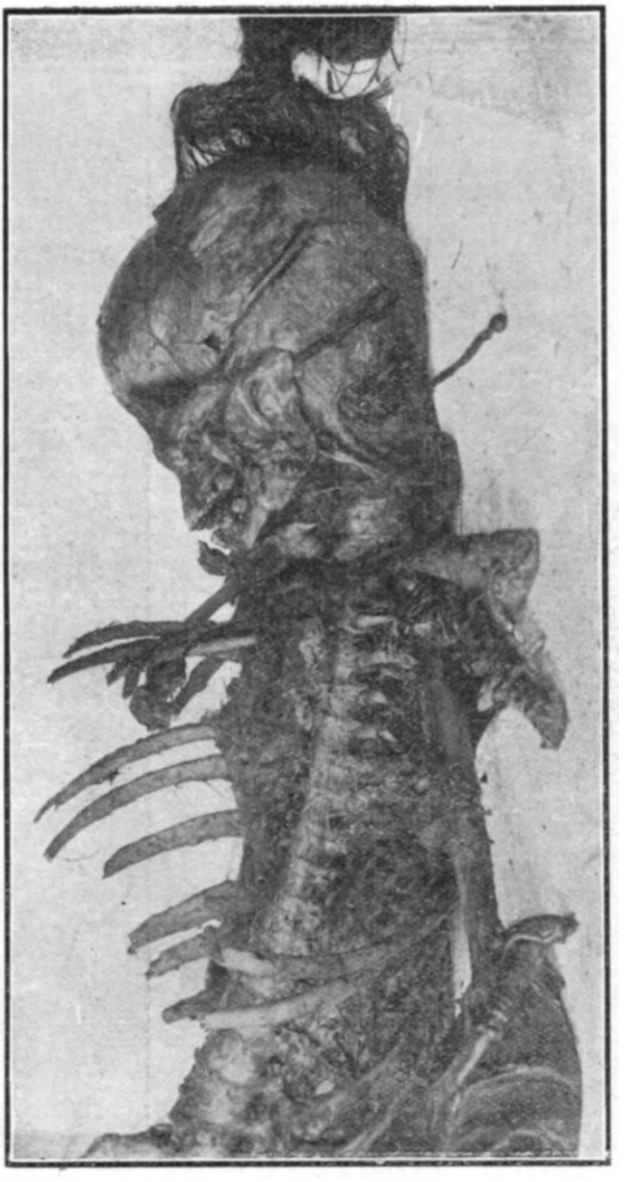


Fig. 14.—The body of a female showing incised wounds on the head. It was decomposed and mutilated by animals after death. Maggots were also crawling all over the body. Post-mortem examination was held 4 or 5 days after death.

out whether the particular bones are human or not. The answer is quite easy when the bones are entire or when the skeleton is sent, but great caution should be used in giving a definite opinion, when a fragment of bone is available without any characteristic features, such as tuberosities,

etc. Such fragments should be examined under the microscope or forwarded to an expert for his opinion.

(2) The sex may be determined from the distinguishing marks of the male and female bones. The determination is more accurate if the adult pelvis is forthcoming.

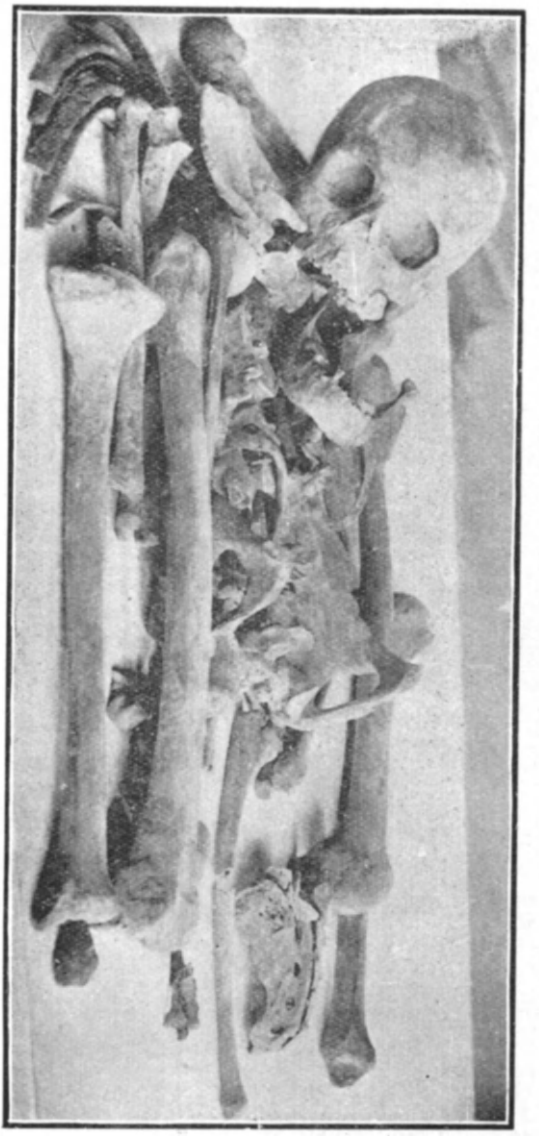


Fig. 15.—Bones as sent for examination.

Fig. 16.—Skeleton reconstructed from the bones.

(3) The bones sent for examination should be assorted according to the side to which they belong and then it should be noted if there were bones of one kind more than necessary as required for one individual, or if there were bones of the same kind more than necessary on the same side.

(4) To estimate the height of an individual an inch or an inch-and-a-half for the soft parts should be added to the length of the entire skeleton, if it is available. As a general rule the stature of an individual is approximately the length measured from the tip of the middle finger to the tip of its opposite fellow, when the arms are extended fully in a horizontal position, but this is not always the case. If only one arm is sent for examination, the height can be fairly ascertained by multiplying its length by two and adding twelve inches for the clavicles, and one-and-a-half inches representing the width of the sternum. The length of the forearm measured from the tip of the olecranon process to the tip of the middle finger is also stated to be equal to five-nineteenths of the height of the body. The symphysis pubis forms the exact centre of the body usually from the 20th or 25th year until old age.

It is possible to estimate the stature of a normal individual within an error of one to two centimeters from the long bones, especially the femur, humerus, tibia or radius, by using the following formulæ compiled by Karl Pearson[1] :—

I. *Formula for calculating the stature in centimeters when the long bones are in a humid state with the cartilages attached.*

Male.	Female
$S = 81.231 + 1.880 \times F.$	$S = 73.163 + 1.945 \times F.$
$S = 70.714 + 2.894 \times H.$	$S = 72.046 + 2.754 \times H.$
$S = 78.807 + 2.376 \times T.$	$S = 75.369 + 2.352 \times T.$
$S = 86.465 + 3.271 \times R.$	$S = 82.189 + 3.343 \times R.$

II. *Formula for calculating the stature in centimeters when the long bones are in a dry state and from which all the soft tissues have disappeared.*

Male.	Female
$S = 81.306 + 1.880 \times F.$	$S = 72.844 + 1.945 \times F.$
$S = 70.641 + 2.894 \times H.$	$S = 71.475 + 2.754 \times H.$
$S = 78.664 + 2.376 \times T.$	$S = 74.774 + 2.352 \times T.$
$S = 89.925 + 3.271 \times R.$	$S = 81.224 + 3.343 \times R.$

Note.—S is equal to stature. F is equal to the length of the femur measured from the top of the head to the bottom of the internal condylar surface. H is equal to the maximum length of the humerus. T is equal to the length of the tibia measured from the upper articular surface to the tip of the malleolus excluding the spine. R is equal to the maximum length of the radius.

In order to obtain the stature of a living individual 1.25 cm. should be deducted from the total length in the case of the male and 2 cm. in the case of the female.

From his observations on 142 dead bodies including males and females of adult Hindus, chiefly of Bengal, Bihar and Orissa, Professor N. Pan[2] of the Anatomical Department, Calcutta Medical College, gives the following table of the length of long bones and their proportion to body height :—

1. *Philosophical Transactions of the Royal Society, A., Vol. CXCII, pp.* 169-244.
2. *Journal of Anatomy, Vol. LVIII, p.* 374.

Sex.	Body. Average Length.	Femur L.	Femur P.	Tibia. L.	Tibia. P.	Fibula. L.	Fibula. P.	Humerus. L.	Humerus. P.	Radius. L.	Radius. P.	Ulna. L.	Ulna. P.
Male	63.8	16.7		14.2		14.3		12		9.4		10.5	
Female	59	15.5	26.2	13.2	22.3	13.3	22.4	11.1	18.8	8.8	15.1	9.7	16.4
Both	62	16.2		13.8		13.9		11.6		9		10.2	

N.B.—L is equal to length, and P is equal to proportion to body length calculated to a scale of 100.

From the examination of 100 male bodies in the case of the bones of the upper extremities and of 80 male bodies in the case of the lower extremities Captain Nat, I.M.S., Professor of Anatomy, Lucknow University, has compiled the following table for estimating the stature of a body from a long bone within an error of two inches :—

1. Stature = length of humerus × 5.3.
2. Stature = length of radius × 6.9.
3. Stature = length of ulna × 6.3.
4. Stature = length of femur × 3.7.
5. Stature = length of tibia × 4.48.
6. Stature = length of fibula × 4.48.

It should be noted that the figures given above are primarily for application to the population of the United Provinces of Agra and Oudh, and the length of the bones was measured between the two extremities, where the articular cartilage was absent.

(5) The age may be determined with a certain amount of accuracy from the presence of the teeth in the mandible and maxillæ, as also from the formation of the centres of ossification and the junction of epiphyses with shafts or of bones with one another. For this purpose it is better to tabulate the reports as under, so as to avoid any mistake.

Kind of bone.	Centre of ossification.	Junction of epiphyses with shaft	Union of bones with one another.	Age.	Remarks.

Lastly, the approximate age should be given considering all these points.

The weight of the bones may help one to give an opinion as regards age though not with any certainty ; however, when the bones of an alleged adult are forwarded by the police and the medical man finds them to be those of a boy or child, it is much safer to weigh them to avoid future complications, as some cases have happened in which medical men were put to some inconvenience owing to their not having done so.

The specific gravity of a bone, which forms the densest part in the human body, is two. The average weight[1] of an Indian male skeleton, especially that of a Punjabi, is ten pounds and six ounces, which is about the same as that of a European male skeleton ; while that of an Indian

1. *Major H. Charles's paper on the identification of European and Oriental skeletons published at page* 511 *of the Transactions of the First Medical Congress,* 1894.

female (Punjabi) weighs six pounds and two ounces, which is less than that of a European female skeleton, which weighs eight pounds and thirteen ounces. Children attain half the adult weight at about 12 in the case of boys and under 11 in the case of girls.

(6) It is extremely difficult to tell the precise time of death from examining the bones, but a guess may be made by noting the existence of fractures, odour and the condition of the soft parts and ligaments attached to them. In the case of a fracture the time may be judged with a certain degree of accuracy by examining the callus after dissecting it longitudinally. The odour emitted by the bones of recent deaths is quite characteristic and offensive. It should be remembered that dogs, jackals, and ants denude the bones free of the soft tissues and even the ligaments in a very short time, but their peculiar odour will be still evident and will be different from that of the bones cleaned by decomposition in the earth.

After all the soft tissues have disappeared, bones begin to decompose from three to ten years, which is the usual period taken up by bodies when laid in coffins; but this period is much shorter in India, where most of the bodies are buried without any such protection.

The changes due to decomposition occurring in the bones are accompanied by the loss of organic matter and weight. Such bones become dark or dark brown in colour, and may be fragile. It is extremely difficult to assign the time when these changes occur, but it depends on the nature of the soil, the manner of burial (with or without coffin), and the age of the individual (more rapidly in young persons).

(7) Bones, particularly the ends of the long bones, should be examined very minutely and carefully to find out if they have been cut by sharp cutting instruments or sawn or gnawed through by animals and the medulla eaten away. Sometimes inexperienced police officers mistake the gnawing of bones by animals for cuts by sharp instruments, and then try to suggest all kinds of absurd theories to maintain their point.

Their nutrient canals should be examined for the presence of red arsenic or some other stain to ascertain if the bones came from a dissecting room as the pleader in a city where there is a medical school or college may raise the question as to whether they came from a dissecting room. To avoid such a possibility it is necessary for the authorities to see that all remaining parts are thoroughly incinerated after dissection is over.

(8) It is almost impossible to infer the cause of death from a bone or bones unless there is evidence of fractures which would, under normal conditions, prove fatal, *e.g.*, fractures of the skull bones or of the upper cervical vertebræ or a deep cut into any of these bones suggesting the use of a heavy cutting instrument, such as a *gandasa* or fracture of several ribs. Disease of the bones, such as caries or necrosis, should also be noted, if present.

The bones should not be returned to the police after medical examination, but should be retained and kept in one's own custody with a view to produce them in Court, if required.

Burnt Bones.—In some instances burnt bones and ashes are forwarded to the medical officer for inspection, when the police come to suspect

some foul play after the body is partially or completely burnt. If the body is not completely consumed, fragments of bones left would afford sufficient evidence to say that they were human or not. The combustion of a body is rarely so complete as to reduce it to ashes. Hence, by shifting the ashes through sieves fragments of bone can be collected and identified by a careful study.

A bone, when burnt in the open, is white in appearance, and black or ash grey when burnt in a closed fire. A burnt bone preserves its shape, but falls to powder when pressed between the fingers. It is said that it will be reduced to charcoal if treated with hydrochloric acid, but this is not necessarily true. If it is so much burnt that organic matter is destroyed no charcoal will be left on adding acid.

In cases of suspected poisoning with some mineral, all the available ashes and burnt bones should be preserved for chemical analysis. It is reported that the Chemical Examiner, United Provinces of Agra and Oudh, was able to detect arsenic and antimony in large pieces of bone mixed with ashes in some cases despite the volatility of these poisons.

I quote the following from my case-book :—

1. In August, 1918, a sealed box from Police Station, Itaunja, District Lucknow, was brought for examination. The box contained a skull, a pelvis with two femurs attached, two tibiæ (the extremities of which had been gnawed through by animals), three right and three left ribs and a piece of a rib (the ends of which had been torn away by animals) and ten dark hairs, each about ten to eleven inches long. From the examination of these bones, especially of the pelvis, and the hairs it was ascertained that the bones were those of a female, about thirty to thirty-five years of age. These were afterwards identified to be those of a female by an *orhni* (head dress), torn *saluka* (bodice) and a brass ear-ring found near the spot where the bones were discovered.

2. In March, 1922, an incomplete skeleton found in the Gomti river was certified to be that of a middle-aged male of about 5 feet 10 inches in height, the length of the femur being 19 inches. It was afterwards identified to be that of a male Ahir by the *dhoti* found round the pelvis to which soft parts were still attached.

3. In the case of a headless skeleton forwarded to me for post-mortem examination on the 2nd August, 1926, I could ascertain from a cut across the centre of the body of the third cervical vertebra and a similar cut across the upper part of the body of the fifth cervical vertebra that death resulted from the injuries inflicted on the neck with a heavy cutting weapon.

CHAPTER IV

EXHUMATION

It becomes necessary to exhume bodies from graves, when a suspicion of poisoning or some foul play arises sometime after death or, it may be, only for the purpose of identification. In India such a procedure is very rare owing to the custom of cremating the dead bodies among the Hindus, who constitute the larger proportion of the population.

Rules for Exhumation.—Under the written order of the District Magistrate the body should be exhumed in the early morning by the medical officer in the presence of a police officer. Before ordering the digging of the grave he should examine the plan of the graveyard to fix the exact situation of the grave, if any plan is available. After proceeding to the place the name plate, if any, should be identified and the undertaker should be asked to identify the stone if it is a *pucca* (masonry built) grave. The grave should now be dug up and the coffin, if used, should be identified by the undertaker who made it. Further, in cases of suspected mineral poisoning, about a pound of the earth in actual contact with the coffin or with the body (if the coffin is decayed or is not used) should be collected and preserved in a dry, clean glass bottle for chemical analysis.

The coffin or the body should then be raised from the grave and the latter should be identified by as many persons as possible, chiefly relatives, friends or servants who might have been present at the time of preparing and dressing the body for burial.

Examination.—If the interment has been recent, the post-mortem examination should be conducted in the usual manner either in the open near the graveyard but screened off from public gaze, or at the mortuary. But in the case of bodies which have lain underground for a sufficiently long time to undergo putrefaction, an attempt should be made to determine the sex, stature and marks of identification. Hair found on the body should be preserved in a dry, clean glass bottle for subsequent identification and chemical analysis. All the cavities should be examined and as many viscera as can be obtained should be preserved separately in dry, clean, wide-mouthed glass bottles or jars without exposing them unnecessarily to the air, and a sufficient quantity of preservative should be added. The viscera should not be brought in contact with any metal. These bottles or jars should then be closed with well-fitting glass stoppers covered with skin, preferably chamois leather, and delivered sealed to the Chemical Examiner on the same day if he lives in the same town, or they should be forwarded to him by a passenger train with the least possible delay. In the case of suspected metallic poisoning, long bones, such as the femur, should be preserved and sent to the Chemical Examiner. Search should also be made for recent or old injuries, such as fractures.

Disinfectants.—Disinfectants should not be sprinkled on the body but may be sprinkled on the ground in the neighbourhood of the body.

To avoid inhaling offensive gases, the medical officer should use for the mouth a gauze mask dipped in a solution of potassium permanganate and should wear thick India-rubber gloves with gauntlets or photographic gloves, which are now always kept in every public mortuary in the United Provinces of Agra and Oudh. He should also stand on the windward side of the body.

The Time of Exhumation.—In England no time limit is fixed for the disinterment of a body, but in Scotland twenty years is the limit fixed as no suspected person can be prosecuted for the perpetration of a crime after the lapse of that period. In France this period is reduced to ten years and it is raised to thirty years in Germany.

Report.—A verbatim report made by Major (now (Lieut.-Colonel) E. J. O'Meara, F.R.C.S., I.M.S., late Principal, Agra Medical School and Civil Surgeon, Agra, on the exhumation of the body of the late Mr. Fulham which was exhumed about fourteen months after death is given below with a view to illustrate how it should be made out in cases of exhumation :—

" On an order of the District Magistrate of Agra dated 6-12-1912, I proceeded to the Cantonment Cemetery of Agra at 8-45 a.m. on Sunday the 6th December, 1912, accompanied by Mr. Williamson, Superintendent of Police, and Dr. Modi, L.R.C.P. & S. (Edin.), Lecturer on Medical Jurisprudence, Agra Medical School.

The grave was identified by the Rev. Canon Menzies from the key to the Cantonment Cemetery plan as Book R. No. 129 non-masonry. A stone marked " No. 129, Mr. E. M. Fulham " stood at the head of the grave.

After 3¼ hours the coffin was raised ; it was then identified by Chiranji Lal of Messrs. Suntoke and Co., the undertakers, as having been made by that firm. There was no name plate. Samples of earth for examination by the Chemical Examiner were taken from above and below the coffin and in the direction of the flow of the subsoil water. The coffin was then placed in a shell on a hearse and sent under the charge of Police Sergeant Charlewood to the post-mortem room of the Thomason Hospital.

On arrival at the post-mortem room at about 2 p.m., the coffin was taken out of the shell and opened by the undertaker Chiranji Lal in the presence of—

Mr. H. Williamson, Superintendent of Police,

Mr. Emery, Merchant, Meerut,

Mr. Sarkies, Military Accounts Department, Meerut,

Dr. Modi,

Dr. Vyas,

Gur Bux, bearer to the late Mr. E. M. Fulham, and myself.

The coffin was much eaten by white ants and decayed but was intact with the exception of the lid which had given way down the centre of the coffin containing a quantity of earth. A sample of this earth was taken for despatch to the Chemical Examiner. On removal of this earth, the grave clothing, a white shirt, white drawers and black socks were identified by Gur Bux, the late Mr. Fulham's bearer, as having been the clothes in which the body had been dressed for burial.

1. Sex, identified as male from the scrotum ; there was no penis.

2. Stature, about 5 feet 5 to 6 inches, the remains in the coffin being 5'—3½".

3. Weight during life approximately 10 stone. It was impossible to distinguish race, age, scars, birth, tattoo or thumb marks. There were no peculiarities of nails, no injuries having permanent results or fractures that could be ascertained. Mr. Emery and Mr. Sarkies stated that Mr. Fulham had a withered left arm. This could not be definitely made out as the measurement of the lower third of the left arm

was only ¼" less than the right and there was only ¾" difference in the measurement of the upper third of the forearm.

The hair lying in the coffin and attached to the back of the head was identified by Mr. Emery, Mr. Sarkies and Gur Bux as being of the same colour as that of Mr. Fulham. The upper and lower jaws were preserved for further identification of the teeth, if necessary. The body was in a peculiar condition of decomposition, there was no skin or subcutaneous tissue left, a piece of white cloth adhered to the face, but all the soft parts and eyes were gone leaving the bones bare. The hair lay in a mass at the head of the coffin with some attached to the back of the head. There was a quantity of light coloured hair round the pubes. The muscles were very well preserved and of a dark red colour. The parieties were intact. On opening the chest the heart was found in a comparatively good state of preservation; the lungs had entirely disappeared. The diaphragm was extremely well preserved, and immediately below there was a mass of an organ in the position of the stomach. The liver was easily distinguishable. The mass of another organ was removed from the position of the spleen and another mass from the position of the left kidney. The intestines were well preserved, but it was impossible to separate the small from the large intestine. There was no sign of the urinary bladder.

The following were sent to the Chemical Examiner:—

(1) Earth taken from above the coffin.

(2) Earth taken from below the coffin.

(3) Earth taken from within the coffin.

(4) Hair from head.

(5) Hair from pubes.

(6) Heart.

(7) Stomach.

(8) Liver, spleen and left kidney.

(9) Intestines.

(10) Two femurs.

Articles upto 9 were packed in standard pattern boxes and article 10 was packed in a big glass jar.

All the regulations for the despatch of articles to the Chemical Examiner were complied with, with the exception that no preservative fluid was used[1] and the viscera were taken direct from the body and placed in tightly fitting stoppered bottles which were specially prepared. As an additional precaution glass stoppers were covered with Chamois leather. The post-mortem was finished at 3-45 p.m. and all the boxes were sealed by 4-25 p.m. The boxes were taken by Dr. Modi himself to the Chemical Examiner and were acknowledged to have been received by that officer at 5 p.m."

I have had occasion to hold post-mortem examinations of six more exhumed bodies. Of these I quote only three.

1. A Mahomedan woman, aged 22 years, resident of Police Station, Mandiaon, District Lucknow, committed suicide by jumping into a well on the 28th September, 1919. The deceased's father petitioned to the Magistrate that his daughter had been murdered; hence the body was exhumed and sent for examination on the 9th October, 1919. No marks of injury were detected on the body which was in a condition of saponification.

---

1. *Lieut.-Colonel O'Meara did not add preservative in this case* (1) *because the viscera were so fully decomposed that no further rapid decomposition could occur and* (2) *because he was able to send them direct to the Chemical Examiner's Office. In all cases in which these conditions do not exist preservative should be added.*

2. In January, 1920, the body of a Hindu male, about 20 years old, who died 5 days previously, was exhumed on suspicion having been raised against the deceased's wife that she had poisoned him. On examination the stomach was empty and was studded with blisters on its inner wall owing to decomposition. The necessary viscera were preserved and sent to the Chemical Examiner at Agra, who found "no trace of any poison".

3. At 5-30 p.m. on May 19, 1925, I held a post-mortem examination on the body of a male infant exhumed 20 days after death as the police suspected that the father thinking that the infant was suffering from tetanus burnt him to death under a superstition that the children yet to be born might not suffer from the disease. The skin was still intact, although the body had undergone putrefaction. There were no signs of burns or other injuries to the body.

CHAPTER V

EXAMINATION OF BLOOD AND SEMINAL STAINS, AND OF HAIR

[*By* RAI BAHADUR K. N. BAGCHI, B.SC., M.B. (CAL.), D.T.M. (CAL. & L'POOL), F.I.C. (LONDON), *Chemical Examiner to the Government of Bengal*]

BLOOD STAINS

Blood stains may be found on the person or on the garments of the suspected assailant or of the victim, as well as on weapons, tools, clubs, articles of furniture, leather goods, stones, plaster, earth, mud, grass, etc. In fact every conceivable article is collected and forwarded by the police for the detection of blood in the stains which may be of various kinds and shades of colour.

The examination of all kinds of stains in this country is left entirely with the Chemical Examiners attached to the Provincial Governments and the determination of the source of blood is chiefly the work of the Imperial Serologist at Calcutta, who is also the Chemical Examiner to the Government of India. According to the existing order of the Government of India, the Provincial Chemical Examiner should, in the first instance, examine the article to see if the suspected stains are due to blood or some thing else. If he is satisfied that they are due to blood, it is his duty to forward the cuttings where blood was actually detected or the entire article, if thought necessary, to the Imperial Serologist for serological tests. The police or the trying magistrate should, on no account, forward any exhibits having suspected blood stains direct to the Imperial Serologist. If an individual is arrested on suspicion and if it is necessary to examine his nails for the detection of blood, the medical officer should cut the nails carefully and collect them together with all the dirt under them and keep them properly packed and sealed in his custody till he receives intimation from the trying magistrate to forward the same to the Chemical Examiner.

All investigating police officers are instructed to dry thoroughly all articles of clothing, etc., having suspected blood stains before being sent to the medical officer for transmission to the Chemical Examiner. Exposure to the open air for a couple of hours will be sufficient in dry weather. Drying before a fire may be necessary in the rains, but, when doing so, great care should be taken that the articles of clothing are not scorched. Unless the clothing is dried thoroughly, putrefaction is likely to set in and render recognition of the origin of stains either difficult or impossible.[1] The investigating officers are also required to forward the entire garment or weapon to the Chemical Examiner along with a history giving all relevent information about the medico-legal aspects of the case and section of the Indian Penal Code under which the case has been registered. If the stains are on large and heavy articles, such as doors, cart-yokes, furniture, etc., or on walls, floors and other places which

1. *The U. P. Medical Manual,* 1934, *para* 797, *p.* 225.

cannot be sent entire, the stained portions should be cut out or sawn out as far as possible and sent to the Chemical Examiner.

The magistrate conducting the inquiry in a criminal case is authorised to make a reference to the Chemical Examiner for chemico-legal problems involved in the case, and the medical officer is required to prepare and despatch to the Chemical Examiner the articles having suspected blood stains which require chemico-legal investigations.[1] When such articles are brought by the police, it is the first duty of the medical officer to see that the articles tally with the description supplied by the police. If the description is not given, he should, before transmitting them to the Chemical Examiner, describe very minutely all the features of the articles, *e.g.,* the size, colour and consistence of the clothing, as well as the number, situation and pattern of the stains present. After completing the examination, he should label each article separately, and pack them in a sealed packet to be forwarded for chemical analysis on receiving intimation from the trying magistrate. This precaution is necessary to enable him to identify them subsequently in Court that those were the aritcles which he was asked to forward to the Chemical Examiner.

On receiving the parcel, the Chemical Examiner should note and record the nature of the packing and compare the seal impressions affixed to the parcel with the facsimile impressed on the forwarding letter. He should also obtain a certificate from the magistrate permitting him to cut out portions of the stains or to destroy the articles, if necessary, for purposes of examination. Without such a certificate, any examination by the Chemical Examiner is an irregular procedure involving probably legal complications. After the examination is over, the articles should be packed carefully and sealed in the presence of a gazetted officer and kept in the strong room until they are required for production during the trial of the case.[2] If not required by the Court, they are destroyed usually after six months.

The points that are usually required to be determined regarding stains on an article sent for examination in cases of alleged wounds, rape and unnatural offences are—

1. If the stains are due to blood or any other substance.
2. If they are due to human blood.

Examination of Blood Stains.—The examination of blood stains may be carried out by five methods—*Physical, Chemical, Microscopical, Spectroscopical* and *Biological.*

Physical Examination.—It is said that the physical examination is conducted with a view to determine the age of the stain and to ascertain whether the stain is of arterial or venous blood or of blood of menstruation, abortion, parturition or hæmatemesis, whether it is from an assailant or a victim, and whether it was shed before or after death. But it is

1. *In Bihar, Orissa, Assam and Bengal the police forward the articles direct to the Chemical Examiner either through the Superintendent of Police or the Sub-divisional Magistrate.*
2. *In the United and in the Central Provinces the Chemical Examiner is required to return clothes and other articles in blood stain cases to the District Magistrate after their examination is over* (vide *para* 800, *The U. P. Medical Manual,* 1934, p. 226).

not helpful in solving these problems. It gives an idea about the size, thickness and colour of the stain and perhaps the direction from which the blood came.

It is difficult for a medical man, even of much experience, to offer an opinion as to the age of blood stains. It is a fact that medical men in India do, sometimes, make far too definite statements in this matter. A young inexperienced doctor is apt to make such a statement under the impression that the Court would think him a fool if he did not give a definite opinion as regards the age of a stain, but he should remember that it is practically impossible to say more than that the stains are fresh or not fresh.

The appearances of stains as to whether they are fresh or not depend on the colour and the nature of the material. Recent stains on a white cloth are of a bright red colour which, on exposure to light and air, gradually changes to reddish-brown in about twenty-four hours, especially in hot weather, and subsequently changes to dull brown; this is fairly permanent but in the course of time it may become black. It is, therefore, evident that an expression of opinion as to the age of blood from consideration of its colour is well nigh impossible. Dry stains have a starchy feel on cloth composed of a thin fabric, such as cotton, silk or linen.

Stains of recently effused blood on a hard substance, such as stone, iron, steel or any other metal, have a dark shining appearance, while dry and old ones have often a cracked or fissured look. Recent stains are also more soluble in distilled water or normal saline than old ones in which hæmoglobin gradually changes to methæmoglobin and finally to insoluble hæmatin. Blood effused during life, when dry, can be peeled off in scales owing to the presence of fibrin in its coagulum, but it is liable to break up into a powder if shed after death.

The recently shed arterial blood is bright red in colour, and the venous blood is dark red, but this difference can hardly be distinguished in a dried stain. The arterial blood is seen in the form of jets or sprays, which have an appearance of elongated, pear-shaped marks which may be compared to 'signs of exclamation'. The jets may be projected to a distance of three to four feet, if effused from small arteries. The arterial blood is always shed during life, as blood-pressure in arteries falls to zero after death.

It is not possible to state from the appearance of stains whether they are of menstrual blood or from any other source. It may, however, be noted that stains due to menstrual blood and hæmatemesis are acid in reaction owing to the presence of vaginal and gastric secretions, but in blood stains the ordinary method of testing the reaction is hardly applicable. An examination under the microscope will determine the nature of the source. It is not correct to suppose that menstrual blood does not clot; the vaginal mucous secretion may only delay clotting.

Whether the blood stains belong to an assailant or a victim can be determined only from circumstantial evidence by examining an article of clothing. If the stains are on the inner side of a garment it is very probable that they belong to a victim, but, if on the outside, they may belong to an assailant, though not necessarily so, as the stains would be found on the outside of a garment worn by a man who received a blow

on the head, while standing. Again, an assailant may not show any stains of blood if he is so standing as to avoid splashing from the outflow of blood while inflicting an injury. Besides, he may have changed the clothes or may have washed them, but the stains are always visible, though faintly, if washing is done hurriedly or carelessly. They are more easily washed in cold than in warm water.

In this connection it may be mentioned that the accused person may, in defence, attribute the presence of a few blood stains on his garment to the stains left by the crushing of bugs, mosquitoes or other blood-sucking insects, which are not uncommon in India. Similarly, an investigating police officer may mistake the insect-stains caused by the splashing of a cow's tail on the wall of a cow-shed for human blood stains and may suspect murder where there is none. Owing to the similarity in their appearance it is not possible to distinguish them with the naked eye. It has been suggested that portions of the insects or their eggs are often found in insect-stains, if seen under the microscope; but this view has not been entirely confirmed by Sutherland who urges further observations on this point.[1] He failed to detect them in ten per cent of his known cases.

Chemical Examination.—The following chemical tests are applied for the detection of blood stains:—

1. Guaiacum Test (Van Deen's, Day's or Schonbein's Test).
2. Benzedine Test.
3. Kastle-Meyer Test (Phenolphthalein Test).
4. Leucomalachite Green Test.

1. **Guaiacum Test (Van Deen's, Day's, or Schonbein's Test).**—The usual procedure for the application of this test is to cut out a small piece of the stained fabric and to transfer it to a porcelain dish where it is soaked with a drop or two of fresh tincture of guaiacum. On the addition of a few drops of old turpentine, ozonic ether or hydrogen peroxide solution a beautiful blue colour appears immediately, if the stain is due to blood. If it is not desirable to cut out the stain, the best way of performing the test is to moisten a piece of white blotting or filter paper with distilled water and to press it with gentle rubbing on a small portion of the suspected blood stain. After a little while, the paper acquires a brownish stain which, if treated with fresh tincture of guaiacum and old turpentine, ozonic ether or hydrogen peroxide solution, assumes a blue colour.

It is a fairly delicate test revealing the presence of fresh blood in a solution of 1:5000, but it may not react to very old blood stains. The test also reacts with many other substances, such as saliva, pus, bile, milk, gluten, gum acacia, oxidising substances like nitric acid, chromic acid, potassium permanganate, peroxide of lead and manganese dioxide, chlorine and other halogens, ferric salts, cupric salts, potassium ferro- and ferric-cyanide, etc.[2] Owing to these limitations this test is of doubtful value in medico-legal work.

---

1. *Sutherland, Blood Stains,* 1907, *p.* 62.
2. *R. W. Webster, Leg. Med., and Toxicol.,* 1930, *p.* 167.

The guaiacum test was very popular among medical jurists about a quarter of a century ago, and in fact, it was the only reliable colour test known to them, but it has now been superseded by more reliable tests.

2. **Benzedine Test.**—This is a very delicate test, and will detect blood when present in a dilution of 1 in 300,000 parts. It is now largely used in medico-legal examination for the detection of blood. The reagents required for this test are :—

(i) *Benzedine Solution.*—It is prepared by taking 13 c. c. of chemically pure glacial acetic acid in a small conical flask and placing it on a water bath at 50° C. When warmed (in about 8 to 10 minutes), 1.5 gm. of chemically pure benzidine (Merck's guaranteed reagent for blood examination) are added and dissolved in glacial acetic acid. The flask is removed from the water bath and 57 c.c. of double distilled water (distilled in all-glass stills) are added.

(ii) *Hydrogen Peroxide* (3 *per cent*) *Solution.*—This is Merck's '10 volume' hydrogen peroxide. Instead of '10 volume' solution ordinary Merckozone or '12 volume' solution (equivalent to 3.6 per cent) may be used safely.

The solutions are usually kept in our laboratory for about a month after which they are discarded although they may be used for a longer period.

The best way of performing the test is to clip off a small fragment of the stained material or to tease out a fibre from the stained fabric and to place it on a porcelain tile. At first a drop of benzidine solution and then a drop of hydrogen peroxide solution are added, when an intense blue colour radiating out on the tile is produced immediately if blood is present. The test may also be obtained by gently pressing a piece of white blotting or filter paper moistened with a few drops of distilled water on the stain and by adding the reagents to the moistened paper.

The advantage of this test is that the same specimen with the blue colour streaming out may be transferred to a slide for microspectroscopy. For this purpose the material is treated at first with a drop of 10 per cent solution of potassium cyanide and then with a drop of ammonium sulphide; it is then covered with a cover slip and looked for cyanhæmochromogen bands. Coloured fabrics are, however, not suitable for direct spectroscopy.[1]

It must be borne in mind that a positive reaction may be obtained from certain other substances, such as sputum, pus, nasal secretion, plant juices, glutin and formalin,[2] but the reaction is decidedly weaker and differs in its sensitiveness and in its shade and depth of colour. They show a slow and faint colouration which should be ignored. A control test performed side by side with a known blood stain decides the issue. It never fails to detect blood even in very old, decomposed stains with all sorts of dirts. The negative result is undoubtedly valuable, but the positive results obtained so far in thousands of our cases never failed to satisfy the confirmatory tests for blood.

1. *Imperial Serologist's Annual Report, Calcutta,* 1937-38.
2. *Sydney Smith, Forens. Med., Ed. VI, p.* 204.

3. Kastle-Meyer Test (Phenolphthalein Test).—The principle of this test is based on the fact that if ordinary phenolphthalein of the laboratory is reduced by zinc dust in an alkaline solution, phenolphthalin is produced which if oxidised in the presence of an alkali, gives the characteristic red colour. The reagents required for carrying out this test are—

1. Hydrogen peroxide solution (20 volumes or 6 per cent strength).

2. A mixture containing 2 grammes of phenolphthalein, 20 grammes of potassium hydrate and distilled water in sufficient quantity to make up 100 c.c. of the solution. These three ingredients are boiled, and during the process 10 to 30 grammes of powdered zinc are added. Boiling is further continued until the solution becomes colourless. The solution thus prepared will remain effective for a long period if a small quantity of powdered zinc is left deposited at the bottom of the reagent bottle to ensure reduction.

If ten to twenty drops of the phenolphthalein reagent are added to a solution extracted from the stain with distilled water, a deep permanganate colour will be obtained instantaneously on the addition of a drop or two of hydrogen peroxide solution if blood be present.

This is an extremely delicate test. Kastle[1] detected blood in a dilution of 1 part in 80,000,000. Glaister[2] found that the reaction was instantaneous in dilutions of 1 part of blood in 800,000 of distilled water in medico-legal cases. It has, however, got its limitations. Traces of copper salts and certain other substances give an equally good positive reaction.[3] The negative result is, therefore, valuable and is conclusive as to the absence of blood.

4. Leucomalachite Green Test.—This test which was recommended by Adler in 1904 is quite useful, but it is not so sensitive as the benzidine test. It depends upon the fact that leucomalachite green is oxidised to malachite green with a bluish-green or peacock blue colour by hydrogen peroxide solution. The reaction occurs also with a solution of the blood pigment previously boiled. On the other hand, the reaction is negative when iron is removed from hæmoglobin forming hæmatoporphyrin.[4]

The reagent is prepared by dissolving 1 gramme of leucomalachite green in 48 c.c. of glacial acetic acid diluted with double distilled water and is then made up to 250 c.c. A drop of this reagent is placed on the stain and after a few seconds, a drop of hydrogen peroxide solution (3 to 3.6 per cent) is added, when the characteristic colour of malachite green appears if blood is present. It is not affected by those substances which interfere with the benzidine test.[5]

These four chemical tests are based on the fact that peroxidase present in hæmoglobin acts as a carrier of oxygen from the hydrogen peroxide to the active ingredients of the reagents (guaiacum resin, benzidine, phenolphthalein and leucomalachite green) and produces the characteristic

1. *Bulletin, 51, Hygiene Laboratory, U. S.*, 1909.
2. *Brit. Med. Jour., April* 10, 1926, *p.* 650.
3. *Sydney Smith, Forens. Med., Ed. V, p.* 206.
4. *Allen, Commercial Organic Analysis*, 1933, *p.* 33.
5. *Sydney Smith, Forens. Med., Ed. VI, p.* 204.

coloured compounds by oxidation. Oxidase and peroxidase are also present in all animal cells, but they are destroyed by boiling, while the peroxidase of hæmoglobin is not affected by such treatment.

Microscopic Examination.—This is useful not only for the detection of the red blood corpuscles but also for the recognition of pus cells, epithelial cells, bacteria, fæcal matter, etc., which are, sometimes, found mixed with blood in the suspected blood and other stains. The presence of squamous epithelial cells from the vagina or columnar cells of the uterus in blood stains may indicate the menstrual source of the blood. Similarly the epithelial cells of the respiratory tract with a large number of pus cells or food particles with sarcinæ and other bacteria will help the examiner in expressing an opinion as to the gastric source of the blood. The size, appearance and other histological features of the red blood corpuscles may also reveal the origin of the blood. Skill in micrometry is, therefore, essential for such work.

Several solvents have been recommended to dissolve out the blood stain for extraction of the red blood corpuscles for microscopic examination but the best for this purpose is Vibert's fluid, which is obtained by mixing two grammes of sodium chloride and half a gramme of mercuric chloride in a hundred cubic centimetres of distilled water. A small piece of the stain should be cut out and soaked in a watch glass with 2 or 3 drops of Vibert's fluid for half an hour. It should then be teased out with needles and examined under the high power. In the absence of Vibert's fluid, normal saline serves the purpose fairly well.

Some clots of blood or stains on a dyed cloth are not easily dissolved by these solvents. In such cases a dilute solution of ammonia will give much better results. Stains on leather or some kinds of wood containing tannic acid are not acted upon by any of these solvents, and a two per cent solution of hydrochloric acid is required for effecting the right amount of softening for proper microscopic examination.

In the case of stains on rusty weapons, stone, plaster, mud or earth, they should be scraped with a knife, and dissolved in a watch glass or test tube for examination. When investigating blood stains on a knife it very often happens that there are no stains on the blade or on the handle but only in the joint. It is, therefore, necessary in such a case to dismantle the parts of the knife for finding out the suspected stain. The blood stain is generally found inside the groove in the handle of the knife and not on its blade which is washed carefully by the assailant.

A drop of the blood stain solution thus obtained, placed on a slide and viewed under the microscope may reveal the presence of the red blood corpuscles which are circular, biconcave, non-nucleated discs in all mammalia except camels, in which the red blood corpuscles are oval and biconvex. In birds, fishes, amphibia and reptiles, they are oval, biconvex and nucleated. These corpuscles can only be detected and identified by one with considerable experience in microscopy and micrometry, and that too only when a stain is quite fresh, say, about twenty-four hours old, and when a small fragment of a clot is available. In old stains specially on a cloth the red blood corpuscles become shrunken, disintegrated and unrecognisable, especially during the hot weather in India.

It is impossible to decide by the microscopic examination of the stain if the blood is of human origin for which the serological test is to be sought for. But in fresh cases it is possible to state that the stain is of mammalian blood. In special cases some information may be obtained by the microscopic examination of the stain which may be of immense corroborative value ; for instance, in a case of murder in Calcutta, Bose found microfilaria in the stains on the assailant's shirt as well as in the victim's blood.[1]

Teichmann's Test or Haemin Crystal Test.—A small crystal of sodium chloride and two or three drops of glacial acetic acid are placed on a minute fragment of the stain on a glass slide. A cover slip is applied and the acid is evaporated by gently heating on a small flame. It is allowed to cool and examined under the high power of the microscope. Dark brown rhombic crystals of hæmin or hæmatin chloride, arranged singly or in clusters, are seen, if blood is present. Similar crystals may be obtained from indigo-dyed fabrics not stained with blood. Hence in a case of doubt a drop of hydrogen peroxide should be added to the crystals which, if of hæmatin, will give off bubbles of gas.

Fig. 17.—Microphotograph of Hæmin Crystals × 400. (*R. B. K. N. Bagchi*)

This test is of academic interest but not of much practical value. It is undoubtedly a delicate test for hæmoglobin of blood but is not always successful. If the stain is too old, is washed or is changed by chemical agents, the crystals are not formed. The addition of too much salt or presence of moisture in the acid or overheating of the slide also results in failure.

1. *Beng. Chem. Exam. Annual Rep.*, 1901.

Haemochromogen Crystal Test.—This is a delicate and confirmatory test for the presence of hæmoglobin. It consists in the addition of two or three drops of Takayama reagent to a small piece of the suspected material on a glass slide in the cold, and covering with a cover glass. Large rhomboidal crystals of a salmon-pink colour and, arranged in clusters, sheaves and other forms appear usually within one to six minutes under the low power of the microscope. Occasionally these crystals take longer to form, but slight warming of the slide, especially in cold weather, hastens the reaction. A negative result should not be recorded until after the lapse of half an hour. An important advantage of this test is its adaptability for the spectroscopic test. The same specimen may be examined with the microspectroscope for the spectrum of hæmochromogen.

Takayama reagent consists of sodium hydroxide (10 per cent) 3 c.c., pyridine 3 c.c., saturated solution of glucose 3 c.c., and distilled water 7 c.c. It should be freshly prepared if prompt action is required. It gives satisfactory results for about two months if kept in an amber coloured bottle. Greaves[1] has obtained crystals using the reagent, six months old.

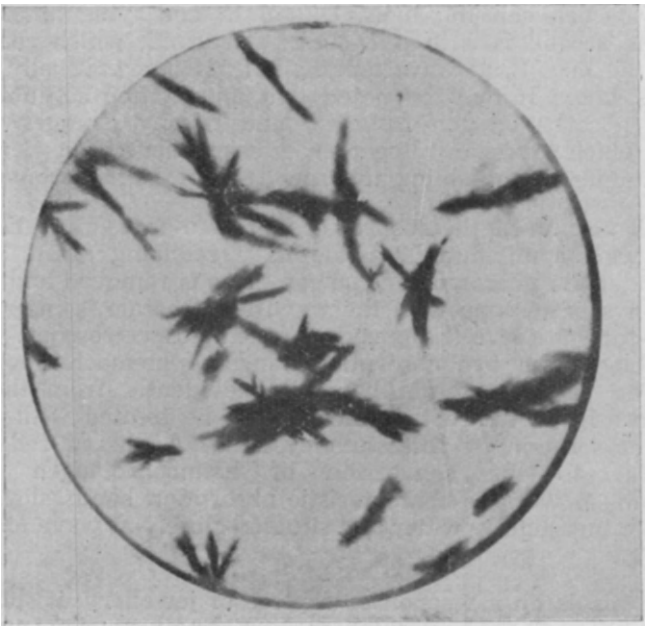


Fig. 18.—Microphotograph of Hæmochromogen Crystals × 500
(*Khan Bahadur Dr. N. J. Vazifdar*)

Spectroscopic Examination.—The spectroscopic examination is the most delicate and reliable test for determining the presence of blood in both recent and old stains, and is always employed by chemical examiners.

A recent blood stain gives a solution of oxyhæmoglobin which, when examined by means of a spectroscope, shows two dark absorption bands

1. *Brit. Med. Jour., May* 21, 1932, *p.* 932.

between the Frauenhofer lines, D and E, in the yellow-green in the solar spectrum. The first band is darker and more clearly defined, and lies at wave lengths 587-570, while the second band is lighter and less clearly defined, and lies at wave lengths 550-530. In an old stain oxyhæmoglobin is converted into methæmoglobin owing to exposure to air and light. Its spectrum consists of four absorption bands, one band in the red-orange between the lines, C and D, at wave length 634, two thinner and fainter bands between the lines, D and E, in the same position as those of oxyhæmoglobin, and a fourth band in the green between the lines, E and F, at wave length 500, but it is very seldom defined and seen (*See Plate I*).

In India, blood stains are liable to putrefy rapidly, if kept damp. If they are kept dry, they become insoluble and resist the action of the ordinary solvents. Either of these changes may render recognition of the stains difficult and sometimes impossible. Hence Hankin[1] has elaborated a method by which the stains may show the absorption bands of hæmochromogen or reduced hæmatin, even though the blood pigment is apparently insoluble. The spectrum of this compound is characterised by the presence of two absorption bands. The first is a very sharp and dark band midway between the lines, D and E, and lying at wave lengths 568-550; the second is a broader but paler band, which commences on the left of the line, E, at wave lengths 537-521 and gradually fades away beyond this line. It may be noted that an alkaline solution of hæmochromogen absorbs oxygen from air and readily changes to hæmatin (alkaline) which gives an altogether different spectrum. The following is the technique for obtaining the spectrum of hæmochromogen:—

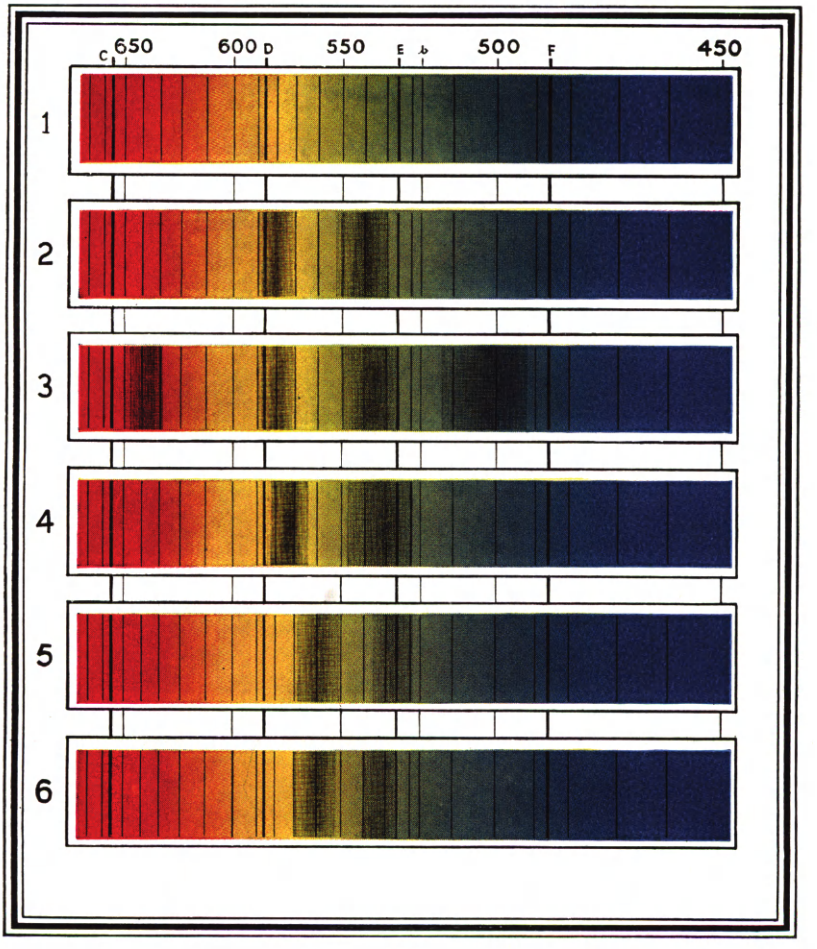
A small portion of the suspected stain is placed on a glass slide and moistened with ammonium sulphide as a reducing agent. It is then focussed under the microscope. The eye piece is removed and an ordinary direct vision spectroscope with the wave length scale is inserted into the microscope-tube to serve the purpose of a microspectroscope. If the stain is due to blood, the two absorption bands of hæmochromogen will be visible. The bands may not be visible evidently from the effects of putrefaction. In that case the stain should be treated with a drop of a ten per cent solution of potassium cyanide. A cherry red colour will develop due to the conversion of hæmochromogen into cyan-hæmochromogen with its characteristic absorption bands similar to those of the former but slightly wider and situated closely at wave length 570-550 and 540-527.

If the stain is on a weapon or a piece of jewellery, it should first be moistened with ammonium sulphide. A small portion may then be scraped off with a knife and treated as above. This is a most delicate and valuable test and should, therefore, be always employed.

The reduction into hæmochromogen may also be brought about by the Takayama reagent as described before or by an alkaline solution consisting of 4 grm. of sodium hydrosulphite, 10 c.c. of potassium hydroxide (10 per cent) and 2 c.c. of alcohol, which is said to be cleaner and much more efficient.[2]

1. *Brit. Med. Jour., Nov.* 10, 1906, *p.* 1261.
2. *Sydney Smith, Forensic Med., Ed. VI, p.* 210.

Plate I.



The Spectra of Blood.

1. The Solar Spectrum.
2. Oxyhæmoglobin.
3. Methæmoglobin.
4. Carboxyhæmoglobin.
5. Hæmochromogen.
6. Cyanhæmochromogen.

[*To face page 98*].

The spectrum of hæmochromogen or of cyanhæmochromogen is quite enough for purposes of identification and expression of a definite opinion about suspected blood stains. It is not necessary to examine any more spectra of acid or alkaline hæmatin, hæmatoporphyrin, etc. For practical purposes one chemical test, *viz.*, the benzidine test, and a confirmatory spectroscopic test for hæmochromogen or cyanhæmochromogen, are quite sufficient for a definite opinion, and these are the principal tests usually employed in the laboratories of the Provincial Chemical Examiners. For very old and scanty stains where there is not sufficient material for repeating the examinations, the Chemical Examiners are required, by the order of the Government of India, to forward the stains, as they are, to the Imperial Serologist, Calcutta, for identification of blood and for determination of its origin by the serological test.

Biological Examination.—This is undertaken for the purpose of determining whether the blood of a particular stain is derived from a human being, from a lower mammalian animal or from a bird.

Precipitin Test.—This test is based on the principle that a foreign protein or a protein-containing substance, when injected into an animal, produces antibodies in the blood serum of that animal, which will form a precipitate when mixed with a solution of that foreign protein. The protein thus introduced is called the *antigen* and the antibody capable of forming a precipitate is called *precipitin.* Relying on this principle, Uhlenhuth made several experiments and devised a method for recognising the different kinds of mammalian blood, found that the test was exceedingly delicate and suggested its applicability for the detection of human blood in medico-legal inquiries. Other workers have elaborated its technique to its perfection and have given it the name of the precipitin test. The method consists in injecting subcutaneously, intraperitoneally or intravenously a rabbit or a fowl with blood serum of an animal, a man for instance, at regular intervals. After a certain number of injections the serum obtained from the injected animal, when sufficiently diluted and added to a clear serum of human blood, produces at first a turbidity and then a flocculent precipitate but fails to do so with the serum of other species. Many workers prefer both intravenous and intraperitoneal injections of rabbits but the Imperial Serologist with the Government of India, who examines about 14,000 articles during a year and has perhaps the largest experience of this kind of work, prefers intravenous injections of blood serum in fowls except for antiavian serum for which he employs rabbits. He injects into the wing vein of a fowl 4 c.c. on the first day, 8 c.c. on the fourth day and kills the fowl on the twelfth day in order to collect the antiserum.

The following general remarks on the precipitin sera are quoted from the annual report of the Imperial Serologist for 1937-38 :—

" i. Some antisera produced against known sera are sometimes found unsuitable for the test. They do not yield sharp reactions in the required dilution and time, that is, they are not sensitive and are, therefore, discarded. It may be noted that every individual fowl does not yield a suitable serum ; some fowls may be entirely refractory and others may produce only weak sera.

ii. All antisera should be highly sensitive, reacting with solutions of animal sera in 1 in 1,000 dilution and reacting with a solution of human serum in 1 in 40,000 dilution.

iii. Some antisera react with sera which have not been used for their production, that is, they are not specific and should, therefore, be discarded. In order to prove their specificity the worker must observe carefully that they do not react with 1 in 1,000 dilution of sera not used in their production.

iv. The antisera which give the expected result with a certain dilution of a known serum (positive control) and do not give unexpected results with a certain dilution of known sera (negative controls) are only used."

Application of the Precipitin Test.—The antisera which differentiate the blood of closely allied species of animals, *e.g.*, cow's blood from buffalo's or sheep's blood from goat's, are not prepared in the laboratory of the Imperial Serologist in Calcutta. But the blood of cow and buffalo (taken together) is differentiated from that of sheep and goat (taken together). This is carried out by means of two antisera, anti-buffalo and anti-sheep. The extract from a stain from any of these four animals will react with both the antisera but much more quickly with the antiserum corresponding to its group. The result so obtained is confirmed by testing further dilutions of the extract. A dilution will be found which will react with one of the two antisera only.[1]

As the precipitin test indicates the presence of the blood protein of an animal of a known species, its utility has been extended to protein materials other than blood stains. The origin of skin, flesh, bone or even secretions, such as saliva, milk and semen, is established by this test. Small fragments of bones and remnants of soft tissues which are scattered deliberately to conceal cases of murder are sometimes recovered by the police and sent for their identification as also for the determination of their source. A histological examination will indicate their nature and a serological examination will reveal their source provided the fragments of the bones and soft tissues are not absolutely dry and decomposed.

This test is also employed in detecting the fraudulent substitution of flesh of horse, cat, dog, etc., for beef, mutton and pork, and has been lately of considerable help to the food analysts in European countries for the detection of horse flesh, for instance, in a sausage.

Technique of the Test.—The first essential thing is to determine the presence of blood in a stain before proceeding with the serological test for ascertaining its source, otherwise, pus, semen, etc., if present, will respond to the test and will be interpreted as blood. After this preliminary precaution, it is necessary to see that all test tubes, pipettes and other glassware articles employed in performing the test are scrupulously clean. The next important item is to prepare an extract of the stained material by soaking it in a small quantity of 0.85 per cent saline solution. The addition of potassium cyanide or any other chemical for dissolving old stains is not desirable and is deprecated nowadays. The extract must

1. *The Imperial Serologist's Ann. Rep.*, 1937-38.

be perfectly clear and bright and may be filtered or centrifugalised if necessary. It should then be diluted with normal saline to the order of 1 in 1,000 dilution. The antiserum is not diluted and 2 drops of it are gently added to three-fourths of a cubic centimeter of the diluted stain extract in a small tapering test tube held in a slanting position. The antiserum slowly settles down at the bottom and at the junction of the two fluids a white ring with well-defined borders appears in the case of a positive reaction. The ring is situated mostly in the antiserum and not in the extract. In the case of a negative reaction no ring appears. A positive reaction should begin in 10 minutes and be read in 20 minutes. Several controls are put up to guard against all possible errors. The following controls are the most important:—

(*a*) The normal serum control of the extract, *i.e.*, the saline extract put up with the normal serum from the same species of the animal which has yielded the antiserum ; it should give a negative reaction.

(*b*) The positive control, and

(*c*) The negative controls, which have already been mentioned.

The results are all qualitative and expressed as positive or negative. " All doubtful reactions are read as negative for the purpose of medico-legal work. The negative results of old, faint and insoluble stains are reported as " disintegrated " with a view to avoid favouring the accused unduly by reporting that no human blood was found on the exhibit. Exhibits stained with blood of good quality but not giving the reaction of human blood, however, are reported as " not stained with human blood " or as " stained with the blood of a ruminant animal/bird " as the case may be."[1]

Limitations of the Test.—Very small stains do not give a satisfactory reaction and as such they are reported as " too small for identification of their source ". Blood stains which have been washed or mixed with mercuric chloride solution (1 in 1,000), potassium permanganate, copper sulphate, iron sulphate, calcium chloride, zinc chloride, sodium bisulphite, alcohol, formalin, acids and alkalis will not respond, partially or completely, to this test. Owing to some unknown reasons, the reaction in some cases may be such as " no opinion can be given as to origin " for medico-legal purposes. A well-equipped laboratory and long experience in this kind of work are essential for giving a decisive opinion. Hence one who is not conversant with the technique of the test and has not got sufficient experience in this branch of the serological work is not justified to undertake this work for giving an expert opinion.

Blood Grouping Test.—This is based on a normal phenomenon of agglutination of the red blood corpuscles on coming into contact with the blood serum derived from another individual of the same species. Investigations into this phenomenon led Landsteiner, Decastelo and Sturli, Ottenberg, Jansky and Moss to divide all human beings, without regard to race, sex or state of health, into four groups according to the behaviour of their sera and red blood corpuscles.[2]

1. *The Imperial Serologist's Ann. Rep.*, 1937-38.
2. *Jour. Amer. Med. Assoc., Aug.* 27, 1921, *p.* 682.

It is universally admitted that the red blood corpuscles contain two distinct agglutinable factors (agglutinogens), called iso-hæmagglutinogens A and B, and the blood serum contains two homologous factors (agglutinins), designated as iso-hæmagglutinins *a* and *b*. These iso-hæmagglutinogens may occur in a blood either singly or together, or they may not occur in a blood at all. The same is the case with the presence of iso-hæmagglutinins in a blood. These agglutinogens and agglutinins are permanent and persist in the blood throughout life without any change. The agglutinogens are almost always present at birth, but the agglutinins which are to characterise the individual throughout life are present in only about half of the newly born infants. These are believed to have been derived from the mother's blood by filtration through the placenta. The agglutinins which are present at birth diminish or disappear during the first ten days of life; after this the infants produce their own agglutinins according to their own blood groups.

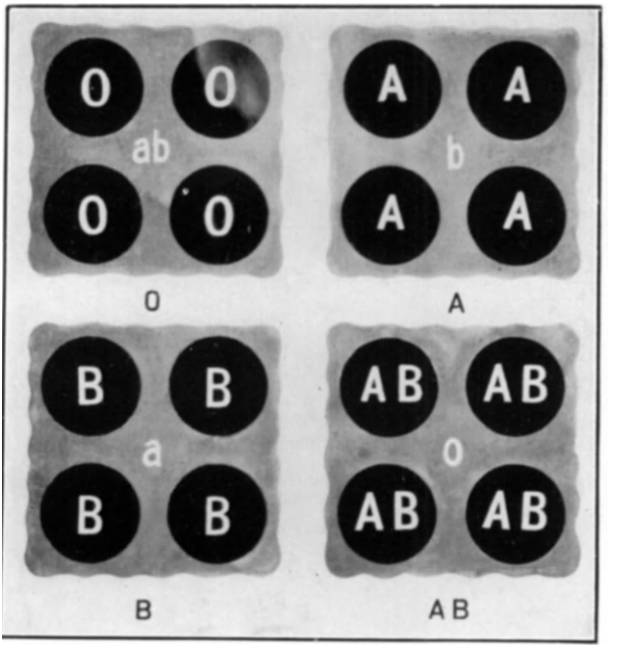
There is some difficulty about the classification of these groups. Moss has modified Jansky's classification by reversing groups I and IV, and retaining the position of groups II and III. To avoid this confusion a new international nomenclature based on the agglutinogen content of the red blood corpuscles has been adopted by the Health Committee of the League of Nations, and the four blood groups are now referred to as O, A, B, and AB, where O represents the absence of the iso-hæmagglutinogens and A and B, the presence of the same. Greval and his collaborators[1] have designed the diagram which illustrates well the distribution of the iso-hæmagglutinogens in the red blood corpuscles and the iso-hæmagglutinins in the serum and also explains the equivalents in the old and new nomenclatures of the blood groups[1] (*See Plate II*).

The two more hæmagglutinogens (not iso-hæmagglutinogens), known as M and N, which are quite unrelated to A and B, occur in the red blood corpuscles, and either M, N, or MN type is present in the red blood corpuscles of all human beings. The hæmagglutinins corresponding to the M and N hæmagglutinogens do not occur in the human sera; hence the presence of the M and N hæmagglutinogens can only be demonstrated by immunising a rabbit with the red blood corpuscles which contain the pure M or N factor and by using the serum thus obtained to test out the unknown corpuscles. It is possible that by the help of the M and N hæmagglutinogens the four classical blood groups may be further subdivided into twelve distinct types, *viz.*, OM, ON, OMN, AM, AN, AMN, BM, BN, BMN, ABM, ABN and ABMN. If sub-groups with A_1 and A_2 are also considered, there will be eighteen distinct types.

The following table[2] gives the percentage of the individuals in each group and also the percentage of the types occurring in the groups as obtained from the examination of the bloods of 300 Indians in Calcutta hospitals by Greval, Chandra and Woodhead:—

1. *Greval, Chandra and Woodhead, Ind. Jour. Med. Res., April,* 1939, *p.* 1042.
2. *Ind. Jour. Med. Res., April,* 1939, *p.* 1048.

Plate II



Blood Groups.

Jansky	Moss	New (International Nomenclature.)
I	IV	O
II	II	A
III	III	B
IV	I	AB

A and B are the iso-hæmagglutinogens.

a and *b* are the corresponding iso-hæmagglutinins.

The group is named after the iso-hæmagglutinogens.

The four groups indicate the only four possibilities, compatible with life, in which the iso-hæmagglutinogens and the iso-hæmagglutinins can co-exist in the same subject. In a 'defective' group an iso-hæmagglutinin which can exist compatibly with life is absent (*e.g.*, O, *a*; A, *o*).

Further division of A into A_1 and A_2 (and into A_1B and A_2B) increases the number of groups to six.

There are difficulties of technique in determining A_1 and A_2. They are, therefore, ignored by many workers in forensic medicine.

(By kind permission of Major S. D. S. Greval, I.M.S. and the Editor and Publishers of the Journal of the Indian Medical Research.)

[*To face page 102.*]

Groups.	Types.		
	M	N	MN
Per cent.	Per cent.	Per cent.	Per cent.
O 26.7	41.2	12.5	46.2
A 26.7	38.7	13.7	47.5
B 37.7	45.1	9.7	45.1
AB 9	48.1	0	51.8

Technique for Determining Blood Groups.—The technique for determining blood groups consists in the use of stock sera of group A and group B and a 5 per cent suspension in normal saline of the red blood corpuscles derived from the individual to be grouped. The stock sera should be obtained from a reliable institution and should be fresh and of high titre strength.

A 5 per cent suspension of the red blood corpuscles is prepared approximately by taking a large drop of blood obtained by pricking with a needle the finger or ear of the individual to be grouped and mixing it with 1 c.c. of normal saline solution in a test tube. A small quantity of 3 per cent sodium citrate solution should be added to the saline solution before preparing the solution, if it is thought necessary to keep the red blood corpuscles for more than a few hours before grouping. This suspension may be used directly or may be centrifugalised and the supernatant fluid pipetted off. The sediment is then suspended in normal saline solution to form a 2 to 5 per cent suspension. After the sera and the red blood corpuscles are ready, the following method is used for the application of the test:—

A drop of group A serum is placed on one end of a perfectly clean and dry glass slide, and a drop of group B serum on its other end. A drop of the red blood cell suspension is added to the serum on each end of the slide and stirred with a platinum loop. The slide is gently rocked to and fro to ensure a thorough mixing of the serum with the suspension and is then allowed to stand for half an hour. After the expiry of this period, irregular clumps of the red blood corpuscles will be noticed by the naked eye, with a hand lens or under the low power of the microscope, if hæmagglutination is present. These clumps cannot be disturbed on tapping the slide. On the other hand, in the case of pseudo-agglutination or rouleaux formation, which is a common phenomenon, the red blood corpuscles are arranged in regular piles; these can be easily disturbed on tapping the slide.

The group can be determined by observing the following rules:—

If agglutination occurs with group A serum alone, the blood belongs to group B. If it occurs with only group B serum, the blood belongs to group A. If agglutination occurs with both the sera, the blood belongs to group AB. If neither of the sera causes agglutination of the red blood corpuscles, the blood belongs to group O.

It is a useful thing to have also a stock of potent serum of group O. This serum agglutinates the red blood corpuscles of every group except

group O. Group O serum enables one to have a decision when there is a poorly marked reaction with either group A serum or with group B serum. If the red blood corpuscles from such an individual are not agglutinated by group O serum, the poor reaction with group A serum or with group B serum is negligible; whereas if agglutination with group O serum occurs, the poorly marked reaction with group A serum or group B serum must be read as positive.

It should be noted that dried serum retains its agglutinating property, and even though drying destroys the red blood corpuscles it does not destroy their agglutinogens. Moreover, it has now become evident that the agglutinogens are not only confined to the red blood corpuscles, but are also to be found in all tissues and even in secretions, such as saliva, urine, semen, and tears. The agglutinins, on the other hand, besides being present in the serum, are also present in body fluids, such as milk, lymph, exudates and transudates; they are not found in normal urine and cerebro-spinal fluid. Strictly speaking, the groups are not of the blood but of the whole tissue structure of the body. Thus, the group of a dead body may be determined by means of the serum agglutinins as long as any serum is available and, after that, by means of the tissue agglutinogens. These persist and remain identifiable until putrefaction is far advanced.

The medico-legal application of this test lies in the determination of cases of disputed paternity and in the grouping of blood stains in criminal cases.

Cases of Disputed Paternity.—In 1910, Von Dungern and Hirschfeld showed from experiments that the agglutinogens A and B are Mendelian dominants, and are transmitted from parent to offspring according to the well-established laws of inheritance. Bernstein has demonstrated that O is also a dominant.

Weiner[1] has drawn up the following table showing the possible and impossible children occurring in various blood groups of parents:—

Blood Groups of Parents.	Possible blood groups of Children.	Impossible blood groups of Children.
O × O	O	A, B, AB
O × A	O, A	B, AB
O × B	O, B	A, AB
A × A	O, A	B, AB
A × B	O, A, B, AB	None
B × B	O, B	A, AB
O × AB	A, B	O, AB
A × AB	A, B, AB	O
B × AB	A, B, AB	O
AB × AB	A, B, AB	O

Many thousands of families have been examined by the above-described formula and no real exceptions have been discovered. It is obvious from the above-mentioned table that a specific agglutinogen cannot appear in a child unless it was present in at least one of its parents.

1. *Amer. Jour. Med. Sc., Aug.,* 1933, *CLXXXVI, p.* 257.

For instance, if the iso-hæmagglutinogen A is present in a child, but not in its mother, it must have been present in its father. If two men are alleged to be the fathers of the child, and if one of them shows the iso-hæmagglutinogen A in his blood and the other does not, the one who has the factor A must be the father. If both men have the factor A, no definite opinion regarding the paternity can be given. Either of them can be considered to be the father of the child as far as the evidence from the blood groups is concerned.

The M and N agglutinogens are also inherited and transmitted as Mendelian dominants, and the following table gives the possible combinations of inheritance in the blood types :—

Types of Parents.	Types of Possible Children.	Types of Impossible Children.
M × N	M	N, MN
M × MN	M, MN	N
M × N	MN	M, N
MN × MN	M, N, MN	None
MN × N	N, MN	M
N × N	N	M, MN

The following two rules are deduced from the above table :—

(i) The agglutinogens M and N cannot appear in the blood of a child unless present in the blood of one or both parents.

(ii) A parent of type M cannot produce a type N child and a parent of type N cannot produce a type M child.

Blood group testing should always be performed first and would be quite sufficient in many instances. The advantage of testing the M and N types is that these have no relation to the primary blood groups. Thus, two individuals, for example, two possible fathers, may belong to the same primary blood group, and yet may have a different content of the agglutinogens M and N.

In cases of disputed paternity it cannot be said by reference to blood groups that a particular man is the father of a given child, but it can, sometimes, be said that he is not the father of the child. The importance of this means of establishing non-paternity is obvious and has its application in suits of maintenance of illegitimate children and in cases of alleged adultery and blackmailing. The test of blood grouping has not been adopted in the Courts of India and England. Towards the end of November, 1938, the Bastardy (Blood Tests) Bill was introduced as a private bill in the House of Lords so as to allow the use of blood grouping tests in evidence in paternity cases, but it is not passed into law as yet. The blood grouping tests have been accepted as evidence in the Courts of Austria, Italy, Russia and Germany. A case[1] is recorded where a woman at Wurtemberg applying for an affiliation order had sworn that a particular man was the father of her illegitimate child. Tests were made of the blood of the child, mother and the alleged father, and the qualities of the child's blood bore no resemblance to those of the blood of either the

1. *Lancet, Vol. II,* 1927, *p.* 1257.

man or the woman, but on the contrary, revealed entirely different characteristics. Apparently the Court accepted the results of these tests as proof that the paternity of the child must be attributed to some other man. It also accepted them as proof that the woman had committed perjury, and she was sentenced to six months' imprisonment.

A case[1] occurred in Dublin, where the blood-group test of relationship was accepted in Court as evidence for the first time. A District Justice made a maintenance order against an elderly man on the complaint of a young woman on behalf of her illegitimate child. The test was carried out in the presence of the solicitors of the two parties.

In certain cases of disputed maternity or of alleged substitution of one baby for another, it may be possible to identify the baby by knowing the blood group of each parent. For example, if the father and mother belonged to group O, and the two babies in question belonged to groups O and A respectively, the baby of group O must be the offspring of the two parents under consideration.[2]

Grouping of Blood Stains.—About 150 m.g. of the blood stained material or about 75 m.g. (about 1 1/6 grain) of dried blood and a control free from the stain should always be available for applying the grouping test to blood stains. When a comparison is to be made between the groups, both the blood stained materials should be adequate in quantity.[3] The determination of the group of a blood stain is more difficult than that of a fresh blood, and success depends to a certain extent upon the age of the blood stain, upon putrefaction and upon chemical changes.

It may be necessary to determine the group of a blood stain in a criminal case, where a stain detected on the person or clothing of an accused is found to be due to human blood, but the defence counsel may suggest that the blood stain was the accused's own blood as a result of an accident and not that of the victim. A preliminary agglutination test should be carried out, using the fresh red blood corpuscles drawn from the accused and the extract prepared by soaking the blood stain in an appropriate amount of normal saline solution. If there is agglutination of the blood, the stain cannot be that of the accused. If there is no agglutination or if two blood stains are to be compared, the stains should be grouped by testing saline extracts for the agglutinins *a* and *b*, using two sets of the fresh red blood corpuscles which are known to contain the iso-hæmagglutinogens A and B respectively. In addition an absorption test should be performed as suggested by Alieff.[4]

If the blood group of the stain be the same as that of the victim's, and different from that of the accused, this is an additional piece of evidence of the guilt of the accused. If, however, the accused and the victim are of the same blood group, the test has no medico-legal value. On the other hand, if the stain and the accused be of the same blood group, and the victim of a different group, the test will be of great help in proving the innocence of the accused.

---

1. *Lancet, Feb.* 27, 1932, *p.* 468.
2. *Snyder, Blood Grouping, Its relation to clinical and legal medicine,* 1929, *p.* 89.
3. *Instructions from the Imperial Serologist to the Chem. Examiner, Beng.* (vide *his letters Nos.* 3811|S., *dated* 28-8-36 *and* 3870|S., *dated* 7-12-37).
4. *Russkaya, Klinika, Moscow,* 1927, *VII, p.* 55; *Whitley and Britton, Disorders of the Blood, Ed. II, p.* 483.

Roche Lynch[1] cites a case in which an assailant murdered a young typist by cutting her throat and disappeared, and the razor which was assumed to have been used for cutting the throat was found subsequently on an omnibus eight or ten hours later. The blood stained clothing of the deceased girl and the blood stains on the razor were examined, and both were found to belong to Group AB; hence he concluded that the razor was used in the crime.

SUBSTANCES RESEMBLING BLOOD STAINS.

Certain substances produce beautiful dark or reddish-brown stains, especially on clothes, which resemble fresh and old blood stains very closely. The most important of them are rust or iron mould stains, red synthetic dye stains, stains caused by red paints of mineral origin and stains of vegetable origin produced by certain fruits, flowers, leaves, barks and roots.

Rust Stains.—Rust stains on knives and steel weapons often look like dried blood stains, but they seldom have a dark and glazed appearance and do not fall off in scales, when the other side of the blade is heated. Similarly, rust stains or iron mould stains on linen may present the appearance of old dried blood stains, but these stains do not stiffen the cloth. They are reddish-brown in colour and insoluble in water but are soluble in dilute hydrochloric acid. The usual tests for iron, *viz.*, potassium ferrocyanide and potassium sulphocyanide tests, may be employed after oxidising the stain with a drop of nitric acid if necessary. The addition of glacial acetic acid to the stain followed by a drop of tannic acid solution produces a blue or bluish-purple colouration if it is due to oxide of iron.

Synthetic Dye Stains.—These stains often resemble old blood stains but they may be easily recognised by treating them with strong acids and alkalies. Nitric acid, for example, changes them to a yellow colour and a strong solution of an alkali may restore the red colour in most cases. No such reaction takes place in the case of blood stains.

Mineral Stains.—These are mostly due to red paints containing oxides of iron. After dissolving with hydrochloric acid, the solution may be tested for iron. In certain circumstances, stains of red paint consisting of red lead or red sulphide of mercury (vermilion) are found in the garments of Hindu women or in Hindu temples. They can be easily identified by the application of chemical tests for lead and mercury.

Stains of Vegetable Origin.—Stains resembling blood may be produced on clothing from certain fruits, such as mulberry, currants, mangosteens, gooseberries and jambuls (Eugenia jambolana). They are changed to a greenish-yellow colour on the addition of ammonia and are bleached by chlorine water, which has practically no effect on blood. Knives which are used to cut acid fruits not unfrequently present stains having a strong resemblance to blood stains. These stains are due to the formation of citrate and malate of iron, are soluble in water, and give rise to Prussian blue if a drop of hydrochloric acid and potassium ferrocyanide solution be added. They do not show red blood corpuscles under the microscope, but present vegetable cells and detritus.

1. *The Medico-Legal and Criminological Review, April,* 1933, *p.* 112.

Reddish stains are also produced by henna, catechu, *pan* juice (with lime and catechu), tobacco, and by the barks, leaves and fruits of some trees, such as *babool* (Acacia Arabica) and *gab* (wild mangosteen or Diospyros Embryopteris). Most of them grow all over India and contain tannin, which will blacken the stain if a drop of ferric chloride solution is added to it. The addition of ammonia will change the colour to green, red or bluish-black, and dilute mineral acids will heighten the original colour, while chlorine water will bleach it. An acid decolourises a stain caused by *pan* juice, while an alkali restores its colour. The spectroscope does not show any absorption bands.

Certain red colouring matters, such as cochineal, lac dye, alkanet root, madder red, *munjeet* (*Sanskrit—Manjistha*) and petals of red hibiscus, give spectra which may be mistaken for those of blood, but the positions of the absorption bands in these spectra are not identical with those of hæmoglobin and its derivatives nor are they affected by reducing and other reagents in the same way as hæmoglobin changes to oxyhæmoglobin, hæmochromogen, etc. Moreover, the solutions of these colouring matters, when treated with alum, boric acid, dilute ammonia, sulphur dioxide solution or chlorine water, show well-marked alterations in the tone and depth of their colour, as also in the position of their absorption bands. Such changes never occur if the colouring matter is blood.

Other Stains.—Spots of grease, resin, tar and pitch, especially on dark fabrics, may resemble very old blood stains, but their solubility in alcohol, ether, chloroform, turpentine, or xylol differentiates them from blood stains. When a clean white filter paper is pressed on any of these spots with a hot iron, the paper absorbs the material and is stained.

Reddish-brown fæcal stains sometimes simulate old blood stains. Even the benzidine test may show a positive reaction owing to the presence of undigested fish or meat fibres. An examination under the microscope will, however, reveal the undigested food particles and decide the question.

SEMINAL STAINS

The question of detecting seminal stains arises in cases of alleged rape or unnatural offence. They are usually found on clothing but may be found on the person of either the victim or the accused. The matting of the pubic hair with semen is not an uncommon occurrence. Seminal stains may also be found on bed-clothes, on the seats of a motor car, on the floor, or on the grass where the offence was committed. They are sometimes found mixed with blood, mucus, pus or fæces, especially on the articles of clothing. In cases of rape on small girls, injuries to the genital organs sometimes cause considerable hæmorrhage, so that semen gets mixed up with a large proportion of blood which renders the identification of seminal stains difficult if not impossible.

Examination of Seminal Stains.—The examination of seminal stains may be carried out by the following methods:—

1. Physical.
2. Chemical.
3. Microscopical.
4. Biological.

Physical Examination.—Semen, when fresh, is a viscid, albuminous fluid of a faint greyish-yellow colour, possessing a characteristic odour and containing spermatozoa, epithelial cells, lecithin bodies, etc. When dry, semen gives a stiff, starchy feel to the cloth and produces slight deepening of the colour with the disappearance of its odour. In fabricated cases which are not uncommon in this country a solution of starch or white of egg is used in producing stiffening of the cloth which looks like a seminal stain in dirty and coloured garments. In fact, dry seminal stains have no reliable distinctive characteristics, when examined with the naked eye. Under certain conditions, stiffness may disappear if the garments are not properly dried in the open air before they are packed for despatch for medico-legal investigation. It is believed that in the presence of moisture certain bacteria act upon the protein constituents of semen, digest the dried protein and thus destroy its stiffness. The bacteria not only remove the albuminous matter but also disintegrate the spermatozoa beyond recognition. It is, therefore, necessary that the police and medical officers should thoroughly dry the garments having suspected stains before they are sent to the Chemical Examiner. They should also be careful not to fold or twist the cloth on the stained portion to prevent damage to spermatozoa.

Invisible and softened seminal stains on cloth can be rendered quite distinct by properly filtered ultra-violet rays which produce a bluish fluorescence on the stains, provided the cloth is clean and not dark coloured. More often than not, the victim's *sari* or underwear, coming as it does usually from the poorer classes, is so dirty that ultra-violet rays are not very helpful in searching for seminal stains. It may also be noted that a bluish fluorescence is not specific for seminal stains and may be seen in some other albuminous materials. The stiffening of cloth, if due to starch, pus, sputum, leucorrhoeal discharge, etc., may be proved by the presence of starch granules, pus cells, squamous and other epithelial cells and different kinds of bacteria under the microscope; these will also indicate the source of the stains.

Chemical Examination.—The chemical examination of seminal stains consists in the application of (1) Florence test and (2) Barberios' test.

Florence Test.—This is known after the name of Dr. Florence of Lyons, who first introduced it. It is based on the formation of characteristic crystals of choline periodide, when a solution of a seminal stain is treated with Florence's reagent containing iodine, 2.54 grm., potassium iodide, 1.65 grm., and distilled water, 30 c.c. It is not absolutely necessary to stick to this formula. In fact a slightly weaker mixture acts equally or better according to some workers.[1] A mixture consisting of 5 per cent of iodine and 8 per cent of potassium iodide in distilled water is used in our laboratory with satisfactory results. It keeps well for at least three months.

The following technique for Florence test has been found quite satisfactory and may be recommended for general application:—

The stained portion is snipped off with a pair of scissors and divided into small bits which are soaked in a watch glass with a small amount of water acidulated with hydrochloric acid (about 0.1 per cent solution or

1. *U. P. Chemical Examiner's Annual Report, 1924, p. 3.*

one drop of strong hydrochloric acid in 44 c.c. of distilled water) for about half an hour. A wet piece is transferred to a slide, is carefully teased with a pair of dissecting needles and is allowed to evaporate almost to dryness. A drop of Florence's reagent is added and a cover glass is placed over the specimen, which is then examined under the microscope.

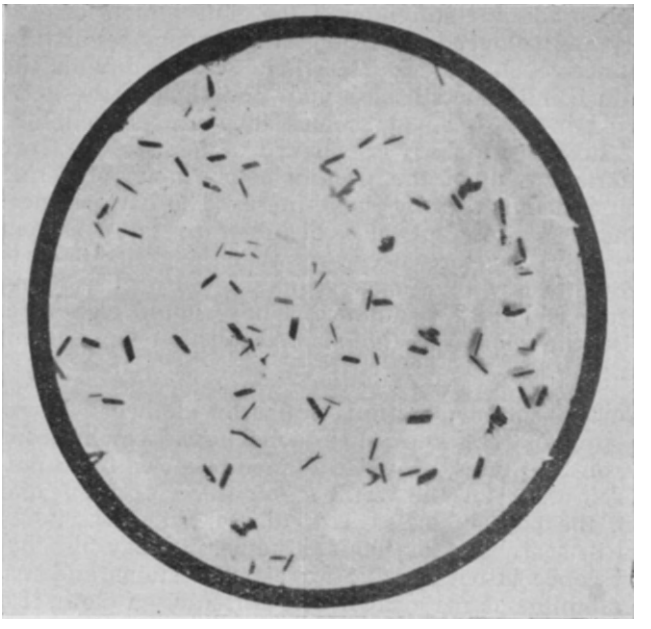


Fig. 19.—Microphotograph of Choline Periodide Crystals × 500
(Khan Bahadur Dr. N. J. Vazifdar)

If a stain be seminal, dark brown crystals in the form of monoclinic prisms or rhombic plates, often crossed or grouped in clusters, appear immediately at the contact of the two solutions and then throughout the specimen. The crystals resemble hæmin crystals in shape, size and colour. In dilute solutions the crystals are smaller and may appear as needles or think black rods instead of the usual rhombic plates. In scanty stains three, four or more pieces of wet specimens may be taken from the watch glass, teased on a glass slide and then squeezed between the thumb and index finger to obtain as much of the extract as possible. It is then evaporated and proceeded with as before.

It is claimed that this test is extremely delicate, even with minute traces. A negative reaction is conclusive proof that the stain is not seminal, but a positive reaction is merely a preliminary test like the phenolphthalein or benzidine test for blood and must be confirmed by the detection of spermatozoa in the remainders of the stains on the articles. After careful investigations on several thousands of specimens for a number of years we have come to the conclusion that the Florence test is not very delicate, and some chemical changes of an unknown nature frequently interfere with the test and give a negative reaction. Hence a negative reaction is not of much consequence. In such a case a thorough

search for spermatozoa is necessary. Entire spermatozoa were detected in several cases where the Florence test was absolutely negative. If the clothes having seminal stains are not dried carefully, choline which originates from the lecithin of semen and combines with iodine to form choline periodide is decomposed completely and a negative reaction is obtained. If the seminal stains are wet and mixed with blood, the Florence reaction is negative even after twenty-four hours owing to rapid decomposition, although entire spermatozoa are detected under the microscope.[1] On the other hand, a positive reaction given by an aqueous extract of a suspected stain on an article received in connection with a sexual offence indicates the presence of semen only and search of spermatozoa in such a case is useless. If the seminal stains are free from blood and other albuminous substances and are thoroughly dried and preserved, they are known to have given a positive reaction after several months. In one case a well-marked positive reaction was obtained after two and a half years, but a negative reaction was obtained from a specimen of six years' standing, although spermatozoa were well preserved in both the cases. It is said that choline periodide crystals may be obtained from watery extracts of various internal organs and certain other biological substances, but this is not true, inasmuch as several samples of pus, blood, sputum, fæces, nasal secretion, leucorrhœal discharge, etc., which contain choline and which are likely to be found in the garments of persons involved in sexual offences were examined and showed the absence of such crystals.[2]

It may be mentioned in this connection that the material richest in choline is the spermatic fluid with 0.514 per cent, the brain comes next with 0.325 per cent and the blood contains only 0.031 per cent.[3] However, the spermatic fluid alone responds to the Florence test, while the brain and the blood require very complicated processes for the extraction of choline.

While performing the Florence test it is essential to bear in mind the following points :—

i. The aqueous extract of the stains must be slightly acid or neutral. Alkali interferes with the formation of the typical crystals. Dilute hydrochloric acid (0.1 per cent) may be used to acidify the stains.

ii. Esters of choline do not give a positive reaction.[4] They are likely to be formed in the presence of other extraneous materials. Hence the extracts of such stains as give negative results should be hydrolysed with one per cent solution of sodium hydroxide, acidified with dilute hydrochloric acid, and then filtered.

iii. The crystals of choline periodide are not permanent. They gradually lose their form and become unrecognisable. The time usually required for taking a microphotograph of the slide is sometimes sufficient to cause the disintegration of a fine crop of the crystals.

iv. The presence of blood and other albuminous substances along with semen intereferes with the test and gives a negative result even in those cases where the garments were carefully dried.

1. *S. N. Chakravarti and S. N. Roy, Ind. Med. Gaz., July,* 1938, *p.* 412.
2. *K. N. Bagchi, Ind. Med. Gaz., Feb.,* 1937, *p.* 90.
3. *Fletcher, Best and Slandt, Biochem. Jour., Part II, Vol. XXIX,* 1935, *p.* 2278.
4. *Booth, Biochem. Jour., Part II, Vol. XXIX,* 1935, *p.* 2064.

Major (now Lt.-Col.) C. Newcomb, I.M.S., Chemical Examiner, Madras,[1] uses a modified form of this test and recommends the following procedure :—

"The suspected stain is cut out and wetted with sufficient distilled water to give one drop when the cloth is squeezed. The water is allowed to remain in contact with the stain for not less than five minutes. One drop of the watery extract is then squeezed on to a microscopic slide and a drop of a saturated solution of iodine in ten per cent potassium iodide solution put beside, but not touching it. A cover slip is then dropped on so that the two drops run together under it. The junction of the two drops is then examined under the low power of a microscope (magnification about 80 diameters), and if semen is present a crop of Florence's crystals is seen or soon appears. The high power of the microscope is then turned on (about 500 diameters) and the region of the crystals searched for spermatozoa, which, if they are present, will be stained a deeper or lighter brown according to the strength of the iodine at the point."

"If the stain contained much albumen as for instance when the semen is mixed with blood, the albumen interferes to some extent with the test by reacting in places with so much of the iodine as to leave too little over for the production of Florence's crystals. If but a small amount of albumen is present some crystals are generally formed but fewer, other things being equal, than when albumen is not present. When albumen is present in large concentration the thickness of the precipitate that the iodine forms with the albumen tends to prevent the two solutions mixing and to obscure any Florence's crystals that are formed. The difficulty can be got over by diluting the watery stain extract to give a sufficient bulk to work with conveniently, boiling, filtering and evaporating to dryness on a water bath. If the residue is now taken up in one or two drops of water this solution will give Florence's test. The spermatozoa are disintegrated by this procedure and must be looked for in a separate portion of the extract."

(2) **Barberios' Test.**—This is also a useful chemical test for the recognition of seminal stains and is believed to be specific for human semen. It is performed by soaking a piece, 1 cm. × 1 cm., of the cloth having seminal stains in a 2.5 per cent solution of trichloracetic acid in a tapered centrifuge tube for about an hour. The tube is then centrifuged and the clear supernatant fluid is added to an equal amount of a saturated aqueous solution of picric acid on a glass slide, when yellow crystals varying considerably in size and shape are seen under the microscope. These crystals may be described as (1) needle-shaped crystals arranged singly, in crosses or in stars, (2) lenticular crystals resembling Charcot-Leyden crystals with a refrangent line at their long axis, (3) crystals with irregular outlines and (4) crystals with feathery appearances.[2] The reaction is probably due to the prostatic secretion, as a positive reaction is given by semen in which there are no spermatozoa. Seminal stains as old as six years, are stated to have responded to this test, but we noticed that a stain of two and a half years' standing failed to give a positive reaction. In fact, we obtained negative results with this test even in some fresh stains where the Florence test was strongly positive.

Harrison[3] applied this test to cloth stained with human whole blood, oxalated plasma, serum, urine, fæces, sputum, pus, nasal discharge and cow's milk. Pus gave a slight amorphous precipitate, but no crystals. All other materials gave no precipitate and no crystals.

Considering its uncertainty, the time it takes, the pleomorphism of the crystals and the amorphous deposit it produces, this test has not been

1. *Madras Chem. Examiner's Annual Reports*, 1922, *p.* 5, *and* 1931, *p.* 6.
2. *Harrison, G. A., Lancet, Oct.* 29, 1932, *p.* 940.
3. *Ibid., p.* 940.

popular with the workers who are required to handle a large number of seminal stains.

Microscopical Examination.—The chief purpose of examining seminal stains under the microscope is to detect the presence of spermatozoa, which are usually associated with epithelia, fæcal matter, starch granules, pus cells, cloth fibres, etc. A medical jurist is rarely required to examine a fresh specimen of semen, although he may be called upon to find out living spermatozoa in the vagina, if a female is brought to him soon after an alleged rape. A drop of mucus is removed from the vagina by means of a glass rod, is placed directly on a slide, and is diluted with a drop of normal saline. It is then covered with a cover glass and examined under the high power of the microscope, when motile spermatozoa, if present, will be seen. Dried seminal stains cannot be examined so easily. They require suitable solvents for bringing out spermatozoa under the microscope. A solution conatining one drop of hydrochloric acid in 44 c.c. of water is considered the most suitable for obtaining the suspensions of spermatozoa from dried stains on fabrics. A ten per cent solution of glycerine in water or in normal saline has been suggested as a useful solvent, but it is regarded as unsuitable for making dry specimens on slides for staining. To suit the climatic conditions of Upper India Dr. Hankin,[1] late Chemical Examiner to the Governments of the United and the Central Provinces, elaborated a method for detecting spermatozoa in seminal stains. The method consists in boiling the stained fabric in a tannin solution before dissolving it in a solution of potassium cyanide so as to render the spermatozoa capable of removal. The fabric is then placed on a slide, teased with dissecting needles and stained with carbol fuchsin, when it is examined with a medium power lens. This method is too long and complicated to be of any use in a laboratory where a large number of seminal stains is examined every day.

A simpler method, which is equally effective and is largely used, consists in moistening a small strip of the stained fabric with a few drops of acidulated water in a watch glass for thirty to sixty minutes in the case of fresh stains and for three to four hours in the case of old stains, and keeping it covered to prevent drying. During this period the spermatozoa are softened completely and are easily detachable from the fabric. A piece is transferred to a slide with its stained surface downwards, and is gently dabbed on the slide with a pair of forceps. It is then teased with needles to disentangle completely the spermatozoa left in the meshes of the fabric. Two or three slides may be prepared from this specimen. One of them is covered with a cover slip and examined for entire spermatozoa under the high power of the microscope and the other slides are allowed to dry by evaporation at the room temperature. In cases where seminal stains are not mixed with blood or pus, spermatozoa with their characteristic refractile heads and long tails will be seen in fair numbers and sometimes in clusters. If no entire spermatozoa are found, the other slides are carefully dried, fixed by passing slowly over a flame two or three times and stained in the usual way by means of methylene blue or methyl green and eosin. Stained with methylene blue or methyl green for about fifteen to thirty minutes and counterstained with eosin for about two minutes the posterior half or one-third of the head assumes a deep red or pink colour,

1. *Brit. Med. Jour.*, 1906, *Vol. II, p.* 1261.

while the anterior half or two-thirds of the head will appear to be unstained or faintly stained with the basic dye. The tail is also stained pink.

Ganguli[1] has devised a modified method of Hankin for staining spermatozoa with erythrosine and malachite green. It is the best method for staining spermatozoa, especially in India, but it has also the disadvantage of being too lengthy. However, it must be adopted in those cases where seminal stains are very dirty. By this method the head, especially the posterior third, is stained dark red and the tail is stained green. It is better to examine the slide under the 1/12th oil-immersion lens, although it is easy to identify spermatozoa under a dry lens.

A human spermatozoon varies from 50 to 55 microns in length, and consists of a head, a neck and a tail. The head is ovoid and flattened when viewed in front and pearshaped when viewed in profile. It is about

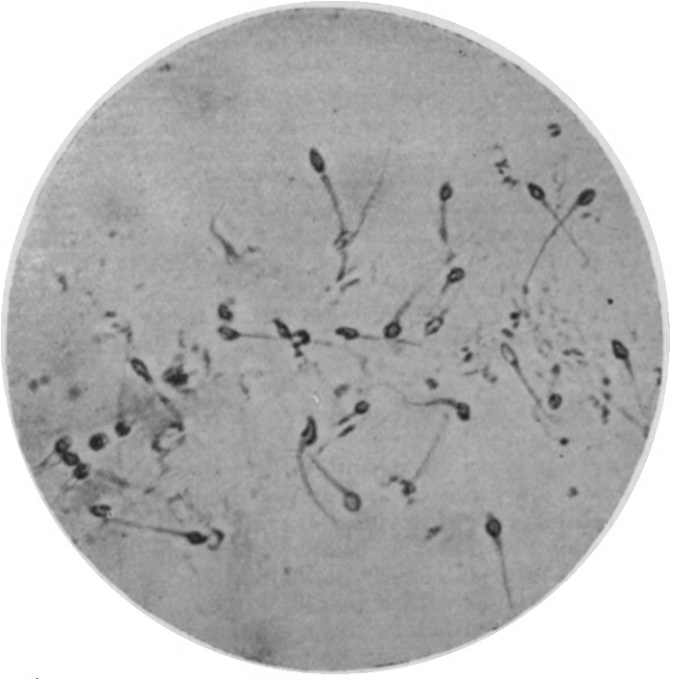


Fig. 20.—Microphotograph of Human Spermatozoa × 900
(*Rai Bahadur K. N. Bagchi*)

5 microns in length or about one-tenth of the total length of the spermatozoon and is about 3.5 microns in its greatest diameter or about one-half of the diameter of a red blood corpuscle. The neck is very short. The tail is the longest part of the spermatozoon and consists of a long, slender filament which tapers to a point at its end and has a vibratile ciliary motion which gives the spermatozoon its motile power. Owing to the vaginal acidity spermatozoa disappear from the vagina within forty-eight hours,

1. *Ind. Med. Gaz., July,* 1936, *p.* 400.

but may retain their activity in faintly alkaline secretion of the uterus and Fallopian tubes for about ten days after emission. Spermatozoa lose their activity in the mediums of acids, strong alkalies, metallic salts, alcohol, glycerine and urine, or when heated above 50° C.; but they retain their characteristic form for a long time if not disintegrated by decomposition. In properly preserved garments they have been identified in stains of from five[1] to eighteen[2] years' standing. It is interesting to note that the spermatozoa can withstand the action of concentrated sulphuric acid but cannot resist the action of the bacteria which produce decomposition. Their disintegration may be complete in less than twenty-four hours at temperatures obtaining in India, especially during the rainy season if the stains are kept damp. Hence it is necessary to dry the clothes suspected to bear seminal stains at air temperature, before they are wrapped up and sent for medical examination. That the presence of moisture in the garments having suspected seminal stains is not only conducive to the growth of moulds and bacteria which are frequently noticed but also to the growth and development of certain insects may be realised from the following case:—

In connection with a case under section 376, I.P.C., the clothes of the accused suffering from itches were received for the detection of seminal stains. While searching for spermatozoa in the suspected seminal stains the eggs of Sarcoptes Scabiei (Itch mites) in various stages of development with a few beautiful hexapod larvæ with active movements were detected under the microscope. The clothes were kept packed and sealed for over a month. It appeared that the moisture originally present in the stains was sufficient to keep the eggs fresh for further development.[3]

The detection of even one entire spermatozoon is quite sufficient for an experienced examiner to give a definite opinion. If no spermatozoa are found, but if the Florence test is positive, there should be no difficulty in affirming that a particular stain is due to semen. If Florence test is negative and if a few detached heads of spermatozoa are found, it may be assumed that the specimen has perhaps been badly handled and a careful examination of another specimen from the same garment is likely to reveal entire spermatozoa. It may also be mentioned that during the decomposition of a seminal stain the tail of a spermatozoon is the first to suffer and to disappear, but the head resists for some time; hence it is quite possible to find only a few heads of the spermatozoa in a decomposed seminal stain. An experienced examiner is not likely to miss the characteristic appearances of these heads and his opinion in such a case is quite valid. However, an inexperienced examiner is not justified to base his opinion on the finding of heads alone, as certain spores of fungi and some bacteria may resemble the heads of spermatozoa, although a well stained slide should leave no room for any doubt.

Some medical jurists believe that there can be no semen without the presence of spermatozoa, but this is not true, inasmuch as cases of aspermia, i.e., semen without spermatozoa, or of oligospermia, i.e., semen with a few spermatozoa, are occasionally seen. These conditions may be found

1. *In his annual report for* 1924, *the Chemical Examiner, U. P., mentions that some dried films of semen on glass slides, which were kept in his laboratory untreated and unstained for five years showed the spermatozoa in good condition and quite intact. Scrapings from these failed to respond to Florence test.*

2. *Witthaus and Becker, Med. Juris., Forens. Med. and Toxic., Ed. II, Vol. III, p.* 859.

3. *Beng. Chem. Exam. Annual Rept.,* 1936, *p.* 16.

in the very young, in the very old, or in those suffering from chronic epididymitis and other testicular diseases. Chronic venereal disease, excessive sexual intercourse or onanism, or some constitutional causes may produce these conditions even in healthy young men.

A man, aged 20 years, married a girl, 19 years old, but as he had no issue by her, he married again at the age of 30 years. He had no issue even by this wife. At the age of 37 years while he was thinking seriously of marrying for the third time, his semen was examined and found to be absolutely aspermic. In another case a man married at the age of 32 and had a child within a year and a half, but he had no other issue during the next twelve years. His seminal fluid was examined and was found quite free from spermatozoa. There was a history of excessive sexual indulgence in both these cases. In a third case a young man, aged 25, who had been addicted to excessive self-abuse since the age of 14 years, sought the advice of his doctor for scanty semen, as he thought that it was an indication of impending impotency. A fresh specimen of his semen obtained in the laboratory did not contain any spermatozoa, and the quantity was only half a cubic centimeter—about ¼ of the normal. It did not give a satisfactory positive reaction with the Florence test.[1]

Biological Examination.—In 1901, Farnum[2] proposed a biological test for human semen based on the same principles as the precipitin test for blood. He used human semen or testicular emulsion for the antigen, and injected 5 to 10 c.c. of it into the peritoneal cavity of a rabbit from five to eight times at intervals of from six to eight days. He found that the serum obtained from the blood of the rabbit thus treated gave a precipitate with both recent and old emulsions of human semen which had been dried and kept for thirty-four days. In 1928, Hektoen and Rukstinat[3] showed that an antiserum produced by immunising rabbits with human semen is both species specific and semen specific, that is, it gives a positive reaction with human blood and also with human semen. In order to demonstrate the semen specific property of the antiserum its species specific property is at first exhausted completely by precipitation with human blood serum, and then the residual or semen specific property is tested with human semen.

This test has evidently a bright future, and is likely to be of much practical value in those cases where it has to be determined whether a seminal stain is of human origin or derived from an animal. It must, however, be remembered that the bacterial action which produces disintegration of spermatozoa in seminal stains in the tropics, is equally effective in decomposing or digesting the protein constituents of semen which, acting as the antigen, produce antibodies. Such seminal stains with their completely disintegrated protein constituents cannot possibly give a positive precipitin reaction and, therefore, offer the greatest difficulty in giving a definite opinion.

The group specific agglutinogens, when present, occur in a highly concentrated form in the seminal fluid, and it may be possible to ascertain the group of the individual by performing the test for detecting the presence of these agglutinogens in the seminal stains in the same manner as with blood stains.

1. *For cases of aspermia and oligospermia* vide *also Currie and Lisstmore, Brit. Med. Jour., Jan.* 28, 1939, *p.* 189.
2. *Jour. Amer. Med. Assoc., Dec.* 28, 1901, *p.* 1721.
3. *Arch. Path.,* 1928, 6, *p.* 96.

HAIR

The detection of hairs upon weapons, blood stains, or upon the clothing or person of an assailant or a victim forms not unfrequently a very important chain in the evidence of cases of alleged assault, murder, rape, and unnatural offence. The examination of hairs also becomes very necessary in identification, particularly when unknown bodies or fragmentary remains have been sent for medical inspection.

While examining hairs the following points have to be determined :—

1. The nature of hairs.
2. The source of hairs.
3. The character of hairs showing the manner of extraction.

1. **The Nature of Hairs.**—Human hairs have to be distinguished from those of lower animals as also from fibres of cotton, linen, silk and wool. For this purpose hairs should be washed in water, alcohol, ether and oil of cloves successively, and mounted in Canada balsam, and then should be examined under the microscope.

Under the microscope the human hair is seen covered with a thin cortical layer of very fine imbricated scales, and a medullary portion of different shades of colour, and frequently containing a narrow axial band of spheroidal cells.

Distinction between human and lower animal Hairs.—To distinguish between the hairs of human beings and those of lower animals the microscopic features represented by the cuticle, medulla and cortex should be observed.

In animal hairs the imbricated scales of the cuticle are very large, and marked with step-like or wavy projections. The medulla of the human hair is narrow and in some cases absent ; while in the animal hair the medulla is conspicuous and, when seen under low power, is found to contain round or oval and prominent cuboidal epithelial cells. The cortex forms the bulk of the shaft in the human hair, and is, as a rule, four to ten times as broad as the medulla ; while in that of the lower animals the cortex is rarely more than twice as broad as the medulla, and often presents only a thin shell enclosing the medullary cells.

Before giving a decisive opinion it is advisable to compare under the microscope the specimen of the hair sent for medical examination with a sample taken from the same part of the individual or animal whence it is alleged to have been derived.

Fibres.—Cotton fibres are flattened bands having spiral twists. Linen fibres are round and transparent, and possess jointed markings at unequal distances. Silk fibres are cylindrical, and have no markings on the surface. They refract light powerfully, and hence exhibit well-defined boundary lines. Wool fibres are coarse and curly and the surface is striated showing the cortical cells.

A rough physical test[1] to distinguish between vegetable and animal fibres is to burn them in a flame. Vegetable fibres burn very readily

1. *J. M. Matthews, The Textile Fibres, Their Physical Microscopic and Chemical Properties, Ed. IV.*

without producing any disagreeable odour, while animal hairs burn with some difficulty and emit a disagreeable odour resembling that of burning feathers. Vegetable fibres burn off sharply at the end, whereas animal hairs fuse to a rounded, bead-like end.

Thymol and sulphuric acid give a violet colour to cotton and linen fibres, but no colour to silk or wool. Sodium nitro-prusside (2 grammes in 100 c.c. of water) produces a violet colour with woollen fibres but not with cotton, linen or silk fibres.

2. **The Source of Hairs.**—It is extremely difficult to determine whether the hairs sent for examination belong to a particular individual or not, though it may be easy to ascertain the source (part of the human body) from which they are derived. This may be easily done by observing the following characteristic features :—

Hairs from the head are usually long and soft, and taper gradually from root to point. The hairs from the female head are generally thinner and much longer than those from the male head.

Hairs from the beard and moustache are usually thicker than those derived from any other part of the body.

Hairs from the chest, axillæ and pubic region are short, stout and curly. Those from the axillæ and pubic region also show split ends.

Hairs from the eyebrows, eyelashes and nostrils are stiff and thick, taper to a point and are $\frac{1}{4}$ to $\frac{1}{2}$ inch long.

Hairs from the body surface are generally fine, short and flexible, and do not show pigment cells in the cortex. The medullary canal is also apt to be relatively small, or may be altogether absent. The downy hairs of the new-born infant have no medullary canal or pigment cells.

3. **The Character of Hairs showing the Manner of Extraction.**—When examined under the microscope, hairs cut by a sharp weapon will not show the roots, and the cut ends will exhibit a more or less regular section. Recently cut hairs show a sharply cut edge with projecting cuticle and a few loose fibres. After a week the end becomes square, smooth, and rounded, but blunt. After three to four months the end becomes elongated, but not similar to the original uncut end, and the medulla is always absent from such ends.

The root should be examined to determine whether a hair has fallen out, or has been pulled forcibly. The root of a hair that has fallen out spontaneously is round and solid, but atrophic, while the root of a hair that has been extracted forcibly, has a hollow, concave surface, which covers the papilla of the corium.

CHAPTER VI

DEATH IN ITS MEDICO-LEGAL ASPECT

Definition.—Death is classified as *somatic* or *systemic* and *molecular*. Somatic death is that state of the body in which there is complete cessation of the functions of the heart, lungs and brain, which maintain life and health, and are, therefore, called " the tripod of life." Molecular death means the death of the tissues and cells individually, which takes place some time after the stoppage of the vital functions, and is accompanied by cooling of the body, the temperature of which is reduced to an equilibrium with the external world.

MODES OF DEATH

In all kinds of death, whether natural or accidental, there are three primary modes of death ; *viz.*,

1. Syncope.
2. Asphyxia.
3. Coma.

SYNCOPE

In this, death occurs from the stoppage of the heart's action, the causes of which are as follow : —

1. Anæmia due to sudden and excessive hæmorrhage from wounds of the large blood vessels, or of the internal organs, such as lungs, spleen, etc., or bursting of an aneurysm or a varicose vein.

2. Asthenia from deficient power of the heart muscle as in fatty degeneration of the heart, aortic regurgitation and certain poisons.

3. Shock inhibiting the action of the heart from sudden fright, blows on the head or on the epigastrium, drinking a large quantity of cold water when in a heated condition, extensive injuries to the spine or other parts of the body, or from the sudden evacuation of natural or pathological fluids from the body.

4. Exhausting diseases.

Symptoms.—These are pallor of the face and lips, dimness of vision, dilated pupils, cold perspiration, feeling of sinking and impending death, great restlessness, air hunger, noises in the ears, gasping respirations, nausea and possibly vomiting. The pulse is slow, weak and fluttering in anæmia, and rapid in asthenia. Slight delirium, insensibility and convulsions precede death. In collapse the patient retains consciousness, though the condition is attended with failure of the heart's action.

Post-mortem Appearances.—The heart is contracted and the chambers are empty when death has occurred from anæmia, but both the chambers

are found to contain blood in the case of death resulting from asthenia. The lungs, brain and abdominal organs are usually found pale.

ASPHYXIA

Death is said to have taken place from asphyxia when the respiratory function stops before the heart ceases to act.

Causes.—1. Mechanical obstruction to the air passages, *e.g.*, foreign bodies, exudations, tumours, suffocation and drowning by blocking their lumen from within; strangulation and hanging by their compression from without; and spasm of the glottis from mechanical irritation and irritant gases.

2. Absence of sufficient oxygen, as in high altitudes or the presence of inert gases in the atmosphere.

3. Stoppage of movements of the chest resulting from exhaustion of the respiratory muscles due to cold or debility; paralysis of the respiratory muscles from disease or injury of the medulla or phrenic or pneumogastric nerves, mechanical pressure on the chest or abdomen, and tonic spasm due to tetanus or poisoning by strychnine.

4. Collapse of the lungs from penetrating wounds of the thorax, and diseases, such as pleurisy with effusion, empyema, or pneumothorax.

5. Non-entrance of blood into the lungs, as in embolism plugging the pulmonary artery.

Symptoms.—These are divided into three stages: (1) The stage of exaggerated breathing, (2) the stage of convulsions, and (3) the stage of exhaustion.

In the first stage the face bears an anxious look, and the patient complains of heaviness in the head and ringing in the ears. The lips are livid, the eyes are prominent, and the respirations are deep, hurried and laboured, the extraordinary muscles of respiration being called into play.

In the second stage the expiratory muscles of respiration become more active with spasmodic movements, which are followed by convulsions of nearly all the muscles of the body. At the same time there is relaxation of the sphincters.

In the third stage the respiratory centres are paralysed. The muscles become flaccid, there is insensibility, the reflexes are lost, and the pupils are widely dilated. Prolonged sighing inspirations occur at longer and longer intervals until they cease altogether, and death ensues. The pulse is scarcely perceptible, but the heart may continue to beat for some minutes after respirations have quite ceased.

The three stages last for about five minutes before death takes place. They may be prolonged for two or three times as long. Occasionally asphyxia may bring about death almost instantly.

Post-mortem Appearances.—External.—The face is either calm and pale in slow asphyxia, or distorted, congested and blue in cases of sudden asphyxia. The lips and nails are livid. Cadaveric lividity is more marked. The tongue is protruded in most cases, and frothy and bloody mucus

comes from the mouth and nostrils. Rigor mortis is usually slow to commence, but may be rapid in some cases.

Internal.—The mucous membrane of the trachea and larynx is cinnabar red due to its injection and contains froth. The lungs are dark and purple in colour, and gorged with dark venous blood; on being cut they exude frothy, dark fluid blood. The air-cells are distended or even ruptured due to emphysema. The right cavity of the heart is full, containing dark-coloured, imperfectly clotted blood, and so are the pulmonary artery and the venæ cavæ. The left cavity, the aorta and the pulmonary veins are empty. In many cases, both sides of the heart are found to be full, if examined soon after death but, after rigor mortis has set in, the heart is found contracted and empty, or the tension in the abdomen presses on the inferior vena cava, and drives blood up into the heart. Similarly, the lungs are found heavier with blood collected in the dependant parts if examined sometime after death, or the tension in the abdomen or contraction of the heart muscle will drive more blood into the lungs, irrespective of the cause of death.

The brain is congested but not so much as in death from coma. The abdominal organs are found congested. Numerous small petechial hæmorrhages or ecchymoses, known as *Tardieu's spots,* are seen under the serous membranes of various organs due to rupture of the capillaries caused by intra-muscular pressure. These are usually round, dark and well-defined, varying in size from a pin's head to a small lentil. They are found under the pleuræ, pericardium, thymus, meninges of the brain and cord, conjunctivæ, and even under the skin of the face and neck. They are, sometimes, seen in deaths occurring from scurvy or purpura.

COMA

Coma means insensibility resulting in death from some cause preventing the action of the brain.

Causes.—1. Compression of the brain resulting from injuries or diseases of the brain or its membranes, such as concussion, effusion of blood on, or in, the brain substance due to depressed fractures of the skull bones, inflammation, abscess or new growth of the brain, or embolism and thrombosis.

2. Poisons, such as opium, alcohol, carbolic acid, etc., having a specific action on the brain and nervous system.

3. Poisons acting on the brain after they are generated in the body in certain diseases of the liver and kidneys, *e.g.,* cholæmia, acetonæmia, uræmia, etc.

Symptoms.—First of all, there is a condition of stupor from which the patient may be roused temporarily for a few seconds or more. In this condition the reflexes are usually present, or are exaggerated, and the patient may be able to swallow fluids. This is followed by complete unconsciousness from which the patient cannot be roused. In some cases sudden insensibility supervenes without an initial stage of stupor. During the comatose condition the reflexes are lost, the sphincters are relaxed, and the pupils are dilated or contracted, and insensible to light. The skin is generally covered with cold perspiration, and the temperature is

sub-normal or normal, except in the lesions of Pons Varolii, where it is high. The pulse is usually full and bounding, but slow. The breathing is slow, irregular and stertorous. Mucus collecting in the air passages causes the sound which is known as " the death rattle."

Post-mortem Appearances.—Injuries of the skull bones or of the brain and consequent effusion of the blood into the cranial cavity may be present. The brain and its membranes are found congested. Hæmorrhages within the cranium due to disease are found within the membranes or in the brain substance, but when due to injury, are commonly found in clots between the skull bones and the membranes, or on the surface of the brain. The right side of the heart is usually full and the left empty. The lungs and the venous system are gorged with blood, but not so much as in death from asphyxia.

SUDDEN DEATH

Sudden or unexpected death occurs from unnatural causes, such as violence or poison, as well as from natural causes. Unnatural deaths have always to be investigated by the police, but very often natural deaths form the basis of medico-legal investigations if they have occurred suddenly and under suspicious circumstances. In such cases medical men should not certify to the cause of death without holding a post-mortem examination even if there is strong evidence of disease.

Causes.—The natural causes producing sudden death are—

1. Diseases of the heart, especially fatty degeneration, angina pectoris, aortic regurgitation, rupture of the heart or of its valves, and diseases of the pericardium.

2. Diseases of the blood-vessels, especially arteriosclerosis, thrombosis, embolism and rupture of aneurysms or varicose veins.

A case[1] is recorded in which an enema caused fatal cardiac embolism. Two weeks after a suprapubic cystostomy was performed under laughing gas anæsthesia by Henry an orderly gave the patient, aged 72, a simple soap water enema. He suddenly gave a gasp and ceased to breathe. The autopsy showed that the pressure of the water dislodged some blood clots in the prostatic venous plexus, and that the patient died of an embolism in the right side of the heart.

3. Cerebral and cerebellar apoplexy caused by bursting of intracranial aneurysms even in children and young persons. Chronic alcoholism and syphilis largely predispose to this condition.

4. Fright, dread, anger or any other emotional excitement may lead to such a degree of shock as to result at once in a fatal termination. This will be more so in those persons who have an unstable nervous system or who have some organic disease, especially of the heart or large blood vessels.

A woman, who was brushing her teeth, accidentally swallowed a mouthful of harmless mouth-wash. She cried out that she had swallowed poison and immediately died.[2]

1. *C. P. Henry and Harisbury, Atlantic Med. Jour., Nov.*, 1927, *p.* 77.
2. *A. Robertson, The Practitioner, Aug.*, 1923, *pp.* 115, 116.

A young woman walking with her sweetheart along a country road received such a fright from a horse pushing its white head through a hedge by her side that she collapsed in her companion's arm and died.[1]

5. Certain diseases of the respiratory organs producing asphyxia, such as œdema of the glottis, membranous deposit in the larynx or trachea, or tumour pressing on the trachea, spasm of the vocal cords, air embolism, pneumo-thorax, hæmo-thorax, pleuritic effusion, hæmoptysis in the course of pulmonary tuberculosis, œdema of the lungs, asthma and whooping cough.

6. Rupture of chronic ulcers of the stomach, duodenum, or other parts of the alimentary canal. Large draughts of cold liquids drunk when overheated.

7. Rupture of the impregnated uterus, extra-uterine gestation, uterine hæmatocele, or uterine appendages.

8. Rupture of the over-distended urinary or gall bladder or enlarged spleen.

9. Acute hæmorrhagic pancreatitis.

10. Certain diseases, such as Addison's disease, diabetes and epilepsy ; laryngismus stridulus and status lymphaticus occurring in young persons usually during the first stage of chloroform inhalation.

11. Trivial procedures may, sometimes, induce syncope and lead to death, *e.g.*, vaginal examination, vaginal and uterine douching, or passing of a uterine sound.

Vipert[2] reports the sudden death of a young woman, four months pregnant, while a small cannula was being inserted into the uterus to produce abortion.

Even slight compression of the larynx has induced fatal inhibition.

A little boy noticing a very prominent pomum Adami in an old woman, gave it a gentle flick with his finger. The old woman died immediately.[3]

12. Catheterization of a distended bladder and sudden withdrawal of large quantities of fluid from the pleural, pericardial or peritoneal cavities may lead to death by rapidly lowering the blood pressure.

13. Zymotic diseases, such as cholera and influenza.

SIGNS OF DEATH

The signs of death are—

1. Entire and continuous cessation of circulation and respiration.
2. Changes in the eye.
3. Changes in the skin.
4. Cooling of the body.
5. Cadaveric lividity, hypostasis, suggilation or post-mortem staining.

---

1. *A. Robertson, The Practitioner, Aug.*, 1923, *pp.* 115, 116.
2. *Annales d' Hygienes publiques*, 1890, *xxiv, p.* 541 ; *Peterson, Haines and Webster, Leg. Med. and Toxic., Ed. II, Vol. I, p.* 213.
3. *A. Robertson, The Practitioner, Aug.*, 1923, *p.* 119.

6. Cadaveric changes in the muscles.
7. Putrefaction or decomposition.
8. Adipocere.
9. Mummification.

1. ENTIRE AND CONTINUOUS CESSATION OF CIRCULATION AND RESPIRATION

Ordinarily these signs are considered sufficient to determine that death has actually taken place, but these alone should not be relied on as absolute signs to avoid premature burial or cremation, inasmuch as persons like hibernating animals are known to have been resuscitated to life after having remained for some time in a condition in which the action of the heart and lungs was in abeyance and the muscles stiff and motionless. This state of *suspended animation* lasting from a few seconds to half an hour or more may be found in cases of trance, *yog*, catalepsy, hysteria, as well as in cholera, sunstroke, concussion, drowning, hanging, tetanus, convulsions, chloroform poisoning and the so-called still-born infants.

As an illustration of suspended animation, Guy and Ferrier[1] quote the case of Colonel Townshend, related by Cheyne in his *English Malady*, who possessed the power of voluntarily suspending the action of his heart. He sent for Drs. Cheyne and Baynard and Mr. Skrine, whom he told about the peculiar sensation he had observed and felt in himself for some time. They all three felt his pulse first; it was distinct, though small and thready, and his heart had its usual beating. He composed himself on his back and lay still for some time, while Dr. Cheyne held his right hand, Dr. Baynard laid his hand on his heart, and Mr. Skrine held a clean mirror to his mouth. Dr. Cheyne found his pulse sink gradually till at last it was quite imperceptible. Dr. Baynard could not detect any motion of his heart, nor could Mr. Skrine discern any dimness on the bright mirror which he held over the mouth. They failed to discover any symptoms of life in him. After Colonel Townshend had been in this state for half an hour, all the three were about to leave him thinking him dead, when they observed some motion about the body, and on examination found both his pulse and respiration gradually returning. This happened in the morning, and he died the same evening. On examination of the body all the viscera were found perfectly healthy except the kidneys which had been diseased and for which he had been under medical treatment.

Waddell[2] cites from Tebb's "Premature Burial" the case of a well-known *yogi*, of Delhi, who used to remain in a state of trance for a long time. Once when he was under the influence of Yog Dr. H. C. Sen and his brother, Mr. Chandra Sen, examined him and could not feel the pulse, nor could they detect the heart beats. The Yogi was then carried to the underground masonry-built cell, the door of which was locked and sealed by the City Magistrate. At the end of thirty-three days the cell was opened and the Yogi was found at the same place with a death-like appearance, the limbs having become stiff as in rigor mortis. He was removed from the cell, and the mouth rubbed with honey and milk, and the body massaged with oil. In the evening he showed signs of life and was fed with a little milk, and in three days was able to take his usual food and lived for several years afterwards.

Major N. C. Kapur[3], I.M.S., reports a case of resuscitation after cessation of the vital functions for over fifteen minutes. A Hindu male, 80 years old, was brought to the Medical College Hospital, Calcutta, at 10 p.m. on July 13, 1925, suffering from severe dyspnœa, the result of laryngeal obstruction due to a malignant growth of the larynx. As his case was urgent, he was taken straight to the operating theatre for the performance of tracheotomy. When the patient was placed on the table, he

1. *Forens. Med., Ed. VI, p.* 214.
2. *Lyon's Med. Juris., Ed. VIII, p.* 84.
3. *Ind. Med. Gaz., Dec.,* 1925, *p.* 582.

suddenly stopped breathing. On examination, the heart sounds were found absent, the pupils were dilated and the eyes were fixed. Artificial respiration was immediately started and tracheotomy was performed when the patient was apparently dead. The patient's chest was continuously flicked with a cold wet towel. For fully fifteen minutes there was no response. There was complete cessation of breathing, heart sounds were absent, there was no pulse at the wrist, and the patient's face had the usual cadaveric characters. Just when all hopes seemed to have been lost, the patient's chest was flicked in a forcible manner, and to the surprise of everybody present, the patient took a shallow breath. The flicking was continued and after a minute the patient took another breath. The pulse was now perceptible at the wrist and the heart sounds could just be heard. The respiration gradually established itself.

Dr. H. M. Shelley[1] relates the case of a young woman, who recovered temporary consciousness several hours after apparent death. Her throat had been cut, and her body was removed to the mortuary in the town of Zomba, as there was no evidence of any cardiac or respiratory action. Three hours later preparations were made for a necropsy, when it was found that the woman was breathing slowly and deeply, and a rapid, thready pulse was palpable in the radial artery. The cut vessels were quickly ligatured, a tracheotomy tube inserted, the skin sutured and intravenous saline administered. Within thirty minutes from the beginning of the operation the woman regained consciousness, and was removed to the hospital, where she gave an account of the assault to the police. Four hours later she died from asphyxia, due to the blockage of the tracheotomy tube. The injuries included a linear incision across the front of the neck, 4½ inches long, which divided the left carotid artery, the left internal and external jugular veins, the left superior thyroid artery, and the trachea completely. The woman had been apparently dead for nearly six hours before receiving treatment, and yet in the space of a few moments regained full consciousness. Dr. Shelley's explanation is that strangulation had preceded the injury causing asphyxia with the result that the blood remained fluid much longer than usual. In preparing the body for the necropsy pressure was exerted upon the woman's chest, allowing the lungs to take in a fresh supply of air and at the same time causing a flow of blood, which possibly reached the brain and stimulated the respiratory centre. The day was very hot, so that the woman could not have experienced any great loss of heat.

A careful examination of the heart and lungs with the stethoscope lasting for five minutes, and repeated at short intervals if necessary, will enable an opinion to be formed as to whether the circulatory and respiratory functions have ceased or not. In a case of doubt this may be supplemented by the under-mentioned tests.

The tests to determine the stoppage of circulation are—

(*a*) *Magnus's Test.*—This is one of the most reliable tests, and consists in tying a ligature tightly round the base of a finger, sufficient to cut off the venous channels without occluding the arteries. The finger remains white, if circulation has entirely ceased, otherwise the seat of the ligature is marked by a bloodless zone, and the portion beyond it becomes gradually blue and swollen.

(*b*) *Monte Verde's Test.*—The subcutaneous injection of ammonia causes a dirty-brown stain indicating dissolution.

(*c*) *Cloquet's Needle Test.*—A bright needle plunged into the biceps muscle and left there for ten seconds shows on withdrawal no signs of oxidation if circulation has stopped.

(*d*) *Diaphanous Test.*—During life the webs of the fingers appear scarlet or very red and translucent if the hand with the fingers abducted

1. *Kenya Med. Jour., Nairobi, East Africa, Sept.*, 1926, *p.* 174; *Brit. Med. Jour., Nov.* 13, 1926, *p.* 918.

is held against a strong light, artificial or natural, while they appear yellow and opaque after death. The hand may, however, appear red in carbon monoxide poisoning, and yellow in anæmia or syncope.

(*e*) *Icard's Test.*—The hypodermic injection of a solution of fluorescin does not produce any discoloration of the skin, if circulation has stopped; but it renders the neighbouring skin yellowish-green, if circulation still goes on. The substance may also be detected in the blood drawn by pricking the skin at some distance from the seat of injection. If some white silk threads are immersed in the blood, and then boiled in a test-tube containing distilled water, the threads will become greenish in colour. The solution of fluorescin is obtained by dissolving 1 gramme of resorcin-phthalein, and 1 gramme of sodium bicarbonate in 8 c.c. of water.

(*f*) On the application and withdrawal of pressure to the finger nail it does not assume alternately a white and pink colour as in life.

(*g*) The application of heat, *e.g.*, a burning match or melted sealing-wax, to the skin will not produce a true blister with a red line of demarcation if circulation has stopped.

(*h*) If a small artery is cut, there will be no jerky flow of blood if circulation has stopped.

The tests to determine the stoppage of respiration are—

(*a*) The surface of a cold, bright looking-glass held in front of the open mouth and nostrils becomes dim, due to the condensation of warm moist air exhaled from the lungs, if respiration is still going on, but not otherwise. This test is useful in the cold weather.

(*b*) There will be no movement of a feather or cotton fibres held in front of the mouth and nostrils if respiration has stopped, but this is not a reliable test as the slightest draught of air or nervousness on the part of an observer will move the feather or cotton fibres.

(*c*) *Winslow's Test.*—There will be no movement of an image formed by reflecting artificial or sun light on the surface of water or mercury contained in a saucer and placed on the chest or abdomen if respiration has ceased. Similarly, water will not be spilt from a vessel filled to the brim and placed on the chest or abdomen if respiration has stopped.

2. CHANGES IN THE EYE

Soon after death the eye loses its lustre. The cornea loses its reflex action and becomes opaque, and looks like dimmed glass. Such a condition may be present before death in uræmia, narcotic poisoning and cholera, while the cornea may retain its transparency for some time after death from apoplexy and from poisoning by hydrocyanic acid or carbon monoxide. The pupils are usually moderately dilated and are insensible to strong light, but react to solutions of atropine or eserine probably for an hour after death, but not longer. The pupils also change their form, and become oval, triangular or polygonal, when pressure is applied by the fingers on two or more sides of the eyeballs of a really dead person,

but they retain their round form in a living person, or in one who is apparently dead.[1]

3. CHANGES IN THE SKIN

After death the skin of the whole body assumes a pale and ashy-white appearance especially in fair bodies, and loses its elasticity ; hence incised wounds will not gape if caused after death. But the edges of ulcers and wounds caused during life retain their red or blue colour after death, and so do ecchymoses. Further the icteric hue produced in jaundice or phosphorus poisoning and tattoo-marks are not at all affected by this change.

4. COOLING OF THE BODY

After death the body commences to lose its animal heat and gradually attains the same temperature as that of its surrounding medium. But it must be borne in mind that this loss of heat cannot be considered as a certain sign of death until the body has lost 15 to 20 degrees of the normal heat, *viz.*, 98.4° F., for a rectal temperature of 90° to 94° F. may be observed in the algid state of cholera and severe cases of collapse, and a much lower temperature of 75° or 76° F. may be noted in cases of long exposure to cold.

The rate of the lowering in temperature is almost proportional to the difference between that of the body and the surrounding air. The average rate of heat that the body loses in temperate regions is 4° F. during the first three hours and then about one degree per hour. Thus, the body takes fifteen to twenty hours to reach the temperature of the surrounding air, but much less time in a country like India. From observations[2] made in 1902 at the famine hospital in Bombay, where the temperature is seldom above 98.6° F., it was found that, in those cases where the body temperature was normal at the time of death, the average rate in the fall of temperature during the first two hours was one half of the difference between that of the body and that of the air. During the next two hours the temperature fell at about half this rate, and during the next two hours at half the last mentioned rate or about a quarter of the initial rate. Thereafter the cooling took place at a much slower rate, the body attaining the temperature of the air at from twelve to fifteen hours after death. In one case in which the temperature recorded at death was 105.8° F., the body temperature came down to that of the air and then rose 13 degrees above the air-temperature in thirteen hours and a half after death.

The rate of cooling of the body may be influenced by such causes as age, condition of the body, manner of death, and surroundings of the body.

Age.—Middle-aged bodies cool more slowly than the bodies of children and old people.

1. *L. Tonelli, Il Policlin.*, 1932, *sez. prat., XXXIX, pp.* 205-210 ; *The Med.-Leg. and Criminolog. Review, April,* 1933, *p.* 132.
2. *Collis Barry, Legal Med., Vol. II, Ed. II, p.* 2.

Condition of the Body.—Fat and well nourished bodies retain heat much longer than lean and weakly bodies.

Manner of Death.—Cooling of the body is more rapid in deaths occurring from severe hæmorrhage or chronic and wasting diseases than in deaths occurring suddenly from accident, acute disease or apoplexy; whereas, the body keeps warm for a long time when death has resulted from asphyxia as in hanging, lightning, suffocation or poisoning by carbon dioxide.

Surroundings of the Body.—A dead body cools more slowly when kept in a small room with still air than when kept in a large room with access of cold draughts of air from outside. Similarly, a body covered with clothes and lying in bed, or in a cesspool or dung-heap, cools less rapidly than a naked body lying on a stone flag in the open air; while a body immersed in water, especially in running water, cools more rapidly than when exposed to the air. Cooling is delayed when the temperature of the atmospheric air or water is high.

Post-mortem Caloricity.—This term is applied to a rise of temperature observed for the first two hours or so in bodies dead from cholera, small-pox, yellow fever, rheumatism, cerebro-spinal meningitis, liver abscess, peritonitis, nephritis, injuries to the nervous system, tetanus and poisoning by alcohol and strychnine. This post-mortem rise of temperature is due to the action of micro-organisms in the still living fluids and tissues of the body, and to the chemical changes going on after death.

5. CADAVERIC LIVIDITY, HYPOSTASIS, SUGGILATION OR POST-MORTEM STAINING

This is a discoloration of the skin due to the accumulation of the fluid blood into the capillaries and small veins of the *rete mucosum* in the most dependant parts of the body according to its position, as the body after death, like all other inert matter, obeys the law of gravitation. If the body is lying on the back, the staining will be seen on the posterior parts of the head, ears, neck, trunk and extremities, except on those parts which actually come into contact with the surface on which the body is lying. Similarly, it is not seen on those parts which have been compressed by tight clothing or tight wrapping of a sheet, but occurs as stripes or bands called *vibices*, which often resemble the marks produced by flogging. Again, a white band on the neck produced by a tight collar or necklace may look like a mark of strangulation.

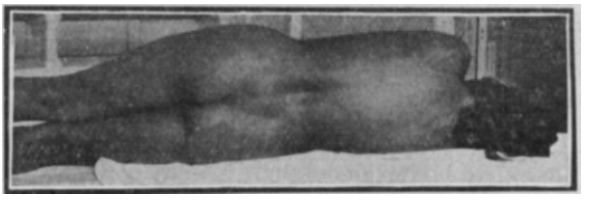


Fig. 21.—Post-mortem Staining.

In Northern India, post-mortem staining begins to form within an hour after death, and is well-marked in four to twelve hours. It is formed

after every kind of death, but it is more marked in the bodies of fair people than in those of dark individuals. It consists of small irregular patches on the skin having a coppery red or purple colour. At first they are single, and scattered on the surface, but later increase in size and unite together forming a large uniform area of discoloration. These patches will disappear and new ones will form on the dependent parts on altering the position of the body if the blood is still fluid, but they will remain permanent, and no more will form, if the position is changed after the blood has coagulated.

It is impossible to give the exact time at which the blood begins to coagulate after death. About four hours after death is the usual period when coagulation of the blood commences. Coagulation does not occur, and the blood usually remains fluid after death from asphyxia, and in cases where a large quantity of saline infusion has been injected intravenously in the treatment of acute hæmorrhage. On the contrary, coagulation occurs readily after death from acute infectious fevers, such as pneumonia.

The colour of the post-mortem staining may, in certain cases, indicate the cause of death. Thus, the colour is intensely bluish violet and purple in asphyxia, and is cherry red or pink in poisoning by carbon monoxide or hydrocyanic acid and, sometimes, in burns or in cold and exposure. On the contrary, the colour of the post-mortem staining is chocolate or coffee-brown in poisoning by potassium chlorate, potassium bichromate or aniline, and is usually dark brown in poisoning by phosphorus.

Rarely, hypostatic congestion resembling post-mortem lividity may be seen a few hours before death in cases of cholera, plague, uræmia, morphine poisoning, typhus, and asphyxia.

Post-mortem lividity or staining has, sometimes, been mistaken for bruises caused by violence during life, and consequently innocent persons have been prosecuted for murder, but acquitted afterwards, when the charge could not be proved. Dead bodies have occasionally been forwarded to me for post-mortem examination with a report from the police that as a result of violence there were bruises on the back, but, on inspection, the so-called bruises were found to be nothing else than the post-mortem stains.

The following are the points by which they can be differentiated :—

1. Post-mortem staining occurs on an extensive area of the most dependant parts of the body, and usually involves the superficial layers of the true skin ; a bruise may occur anywhere on the body, usually takes the shape of the weapon used, is limited in area, and generally affects the deeper tissues.

2. Post-mortem staining does not appear elevated above the surface, but has sharply defined edges ; a bruise appears raised above the level of the surface, and its edges are not sharply defined.

3. The colour of the post-mortem staining is uniform ; it may become green when the body begins to putrefy ; whereas a bruise exhibits the usual changes of colour, especially if a few days old.

4. In the case of post-mortem staining there will be no abrasion of the cuticle, but in the case of a bruise there may be an abrasion of the cuticle.

5. Post-mortem staining, on being cut, does not show any effusion of coagulated or liquid blood into the subcutaneous tissues, but may show minute drops of blood exuding from the divided ends of the distended capillaries and small veins; a bruise, on the other hand, shows infiltration of the tissues either with coagulated or liquid blood.

Along with the appearance of the external post-mortem staining internal hypostasis also takes place in the dependent portions of the visceral organs. Thus, if a body has been lying on the back, post-mortem staining is frequently found in the veins of the cerebral and spinal piamater, in the lateral and occipital sinuses, in the posterior cerebral lobes, in the lower posterior surfaces of the lungs, in the posterior surfaces of the liver, spleen and kidneys, and in the posterior parts of the stomach and of the intestines, especially those lying in the pelvis. Post-mortem staining does not occur in the heart, but it may contain the so-called "cardiac polypi" which are post-mortem fibrinous clots.

Hypostasis in internal organs, such as the brain, lungs, stomach, kidneys and intestines, has to be distinguished from congestion or inflammation of those organs.

Difference between Post-mortem Staining and Congestion in an Organ.—Post-mortem staining in an organ is irregular, and occurs on a dependent part; redness caused by congestion is generally uniform and all over the organ. The mucous membrane in post-mortem staining is dull and lustreless, but not so in congestion.

In post-mortem staining inflammatory exudations or constriction will not be seen, and areas of redness alternating with pale areas will be found if a hollow viscus is stretched out and held in front of light.

6. CADAVERIC CHANGES IN THE MUSCLES

After death the muscular tissues of the body pass through three stages: (1) Primary relaxation or flaccidity, (2) Cadaveric rigidity or rigor mortis, (3) Secondary relaxation.

(1) **Primary Relaxation or Flaccidity.**—Soon after death the whole muscular system commences to relax except in those cases where the muscles have been in a contracted condition before death; hence we notice that the lower jaw of a dead body falls, the eyelids lose their tension, the extremities become soft and flabby, and the joints are flexible. But the muscles are contractile, and react to external stimuli, mechanical or electrical, owing to their retaining molecular life after somatic death.

This stage lasts from three to six hours, but the average is two or three hours. One hour and fifty-one minutes is the average period of duration in Bengal as found by Mackenzie.[1]

1. *Ind. Med. Gaz., June,* 1889, *p.* 167.

(2) **Cadaveric Rigidity or Rigor Mortis.**—This phenomenon, which is also known as death stiffening, comes on immediately after the muscles have lost the power of contractility, and is due probably to the coagulation of myosin within the sarcolemmas of the muscle-fibres by the formation of sarcolactic and other weak acids, which are no longer removed from the system on account of molecular death. It is in no way connected with the nervous system, and occurs whilst the body is cooling. Owing to the setting in of rigor mortis all the muscles of the body become stiff, hard, opaque and contracted, but they do not alter the position of the body or the limb. A joint rendered stiff and rigid after death, if flexed forcibly by mechanical violence, will remain supple and flaccid, but will not return to its original position after the force is withdrawn; whereas a joint contracted during life in cases of hysteria or catalepsy will return to the same condition after the force is taken away.

Rigor mortis first appears in the involuntary muscles, and then in the voluntary. In the heart it appears, as a rule, within an hour after death, and may be mistaken for hypertrophy, and its relaxation for dilatation, atrophy or degeneration. The left chambers are affected more than the right. Post-mortem delivery may occur owing to contraction of the uterine muscular fibres.

In the voluntary muscles rigor mortis follows a definite course. It first occurs in the muscles of the eyelids, next in the muscles of the back of the neck and lower jaw, then in those of the front of the neck, face, chest and upper extremities, and lastly extends downwards to the muscles of the abdomen and lower extremities. It passes off in the same sequence.

Time of Onset.—This varies greatly in different cases, but the average period of its onset may be regarded as three to six hours after death in temperate climates, and it may take two to three hours to develop. In India, it usually commences in one to two hours after death, and takes one to two hours to develop.

Duration.—In temperate regions rigor mortis usually lasts for two to three days, though it may last several days (nine days according to Casper,[1] and three weeks according to Taylor[2]). In Northern India, the usual duration of rigor mortis is twenty-four to forty-eight hours in winter and eighteen to thirty-six hours in summer. According to the investigations of Mackenzie[3] in Calcutta the average duration is nineteen hours and twelve minutes, the shortest period being three hours, and the longest forty hours. When rigor mortis sets in early it passes off quickly and *vice versa.*

Cases[4] have occurred in which rigor mortis developed and disappeared within an hour and a half after death. In a case where death occurred from exhaustion after a prolonged illness of enteric fever, rigor mortis was evident everywhere on the body in three minutes and a half

1. *Forensic Medicine translated by Balfour, Vol. I, p.* 28.
2. *Princ. and Pract. of Med. Juris., Ed. IX, Vol. I, p.* 220.
3. *Ind. Med. Gaz., June,* 1889, *p.* 167.
4. *Bomb. Famine Hosp. Rep.,* 1901.

after death, disappeared in a quarter of an hour and in less than an hour after death putrefaction had appeared in the limbs.[1]

Circumstances modifying the Onset and Duration of Rigor Mortis.—

(*a*) *Age.*—Rigor mortis is said not to occur in the body of an immature fœtus of less than seven months. A case[2] is, however, recorded in which strongly marked rigor mortis was present in a five months' fœtus. Rigor mortis is commonly found in the bodies of still-born infants at full term. Tarleton[3] relates a case where rigor mortis was seen in a well-developed female child, which died during delivery. Cases of ante-natal rigor mortis, although rare, are recorded. This condition usually interferes with delivery. Dr. Jitendra Desai of Ahmedabad reported to me that in October, 1938, he delivered a quadripara, aged 28, of a full-term dead female child, which was in a state of rigor mortis. The labour was tedious and prolonged as compared to her previous labours.[4]

In adolescent and healthy adult bodies the occurrence of rigor mortis is slow, but well marked, while it is feeble and rapid in the bodies of children and old people.

(*b*) *Muscular Condition.*—The onset is slower, and the duration longer, in those cases where the muscles have been healthy and at rest before death than in those cases where the muscles have been feeble and exhausted, and thus have lost a greater degree of muscular irritability.

(*c*) *Manner of Death.*—Rigor mortis sets in early, and disappears soon in deaths from diseases causing great exhaustion and wasting of the muscles, as in cholera, plague, typhus, typhoid, phthisis, cancer, uræmia and chronic Bright's disease. Its onset is delayed in deaths occurring from pneumonia, apoplexy, asphyxia and nervous diseases causing paralysis of the muscles. In cases of strychnine and other spinal poisons the onset is rapid and the duration longer if death has occurred in a short time after the symptoms first appeared when the muscles had not been exhausted owing to convulsive fits.

(*d*) *Atmospheric Conditions.*—Rigor mortis commences slowly, but lasts for a long time in dry, cold air. On the other hand, its commencement is rapid, and duration short, in warm, moist air. It comes on rapidly and disappears late in bodies immersed in cold water.

Conditions Simulating Rigor Mortis.—The conditions which simulate rigor mortis are (a) heat stiffening, (b) cold stiffening and (c) cadaveric spasm or instantaneous rigor.

(a) **Heat Stiffening.**—The phenomenon known as heat stiffening is seen in the hardening and stiffening of the muscles in a body exposed to a temperature exceeding 75° C. This is due to the coagulation of other albuminates besides myosin, which coagulates ordinarily at a lower temperature, say 50° C.

1. *Savory, On Life and Death, p.* 196 ; *Taylor, Princ. and Pract. of Med. Juris., Ed. IX, Vol. I, p.* 220.
2. *Bomb. Famine Hosp. Rep.,* 1901.
3. *Brit. Med. Jour., June* 13, 1908, *p.* 1424.
4. *For other cases of ante-natal rigor mortis see Brit. Med. Jour., Vol. I, pp.* 1014 *and* 1312 ; *Jour. Surg. Gynæc. and Obstet., May,* 1925, *p.* 725.

Heat stiffening is commonly observed in the body of a person who has met his death from burning or from sudden immersion in a boiling fluid, or in a body which has been burnt soon after death while the muscles were still warm. The body assumes an attitude, called " pugilistic attitude " with the lower limbs and arms flexed and the hands clenched.

(b) **Cold Stiffening.**—The stiffening of the muscles occurs in a body from solidification of its fat when it is exposed to a freezing temperature. If the body is moved to a warm atmosphere, the stiffening rapidly disappears and normal rigor mortis develops, but it lasts only for a short time.

(c) **Cadaveric Spasm or Instantaneous Rigor.**—This is a phenomenon in which the muscles that have been in a state of contraction during life become stiff and rigid immediately after death without passing into an initial stage of relaxation; hence the attitude of the body adopted at the time of death is maintained for several hours afterwards. It is due to the fact that the last voluntary muscular contraction of life does not stop after death, but is continuous with an act of cadaveric rigidity and thus occurs in cases where there have been great muscular exertion and mental excitement before death, as observed among soldiers killed on a battle field. It is also found in sudden asphyxial deaths, and in deaths from irritation of the medulla. It is quite different from cadaveric rigidity or rigor mortis. In the case of cadaveric spasm, a weapon held in the hand before death is firmly grasped, and can only be removed with difficulty; whereas in cadaveric rigidity the weapon placed in the hand before rigor mortis has set in is not grasped, but drops down from the hand on the slightest touch. For practical purposes it is not possible for a murderer to imitate this condition.

Medico-legally the condition of cadaveric spasm is very important, inasmuch as the finding of a weapon, hairs, pieces of clothing, etc., firmly grasped by the fingers of a dead body may lead to the detection of a case being suicidal or homicidal. It must, however, be remembered that a heavy weapon may drop down from the hand of a suicide, unless it becomes glued down by clotting of the effused blood.

A razor was found firmly grasped in the right hand of a man who committed suicide by cutting his throat in June, 1915. A European widow, aged 40 years, who shot herself with a five-chambered revolver while driving in a victoria, was found with the revolver gripped in her right hand.[1] On the other hand, an army Major who committed suicide on the 1st December, 1922, by shooting himself with a revolver through the mouth was seen reclining against a wall in a bathroom at the Royal Hotel, Lucknow, with the head drooping forwards and the revolver lying between his legs with the right thumb and index finger loosely touching the trigger. In the case of *King-Emperor* v. *Navnidh Singh*, a piece of cloth found grasped in the hand of a murdered person, Phuloo Singh, was proved to have been torn from the vest of the accused at the time of murder.[2]

(3) **Secondary Relaxation.**—With the disappearance of rigor mortis, the muscles become soft and flaccid, but do not respond to a mechanical or electrical stimulus as in the first stage of relaxation. This is probably due to myosin being dissolved by the excessive production of acid during the stage of rigor mortis.

1. *Leader, Feb.* 12, 1928, *p.* 11.
2. *All. High Court, Cr. App. No.* 25 *of* 1925.

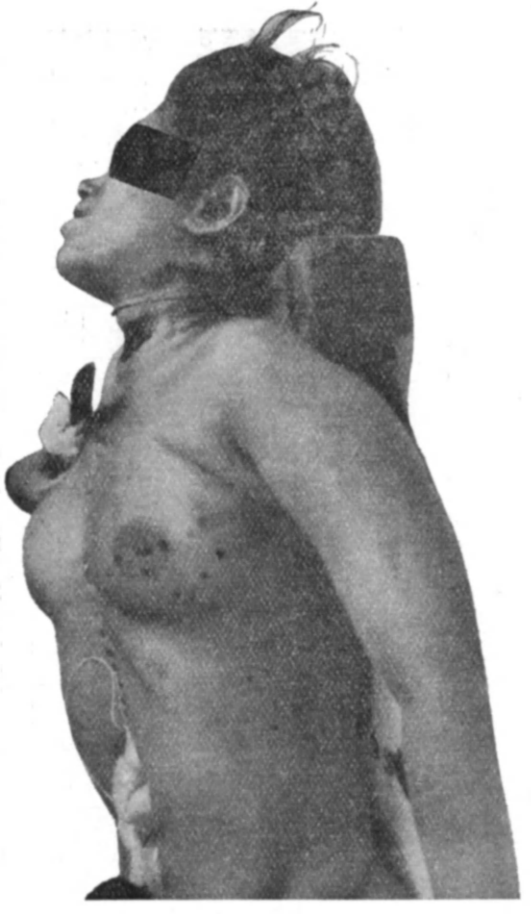


Fig. 22.—Cadaveric Spasm : The razor is firmly grasped in the hand. A case of suicide. (*From a photograph kindly lent by Dr. H. S. Mehta*).

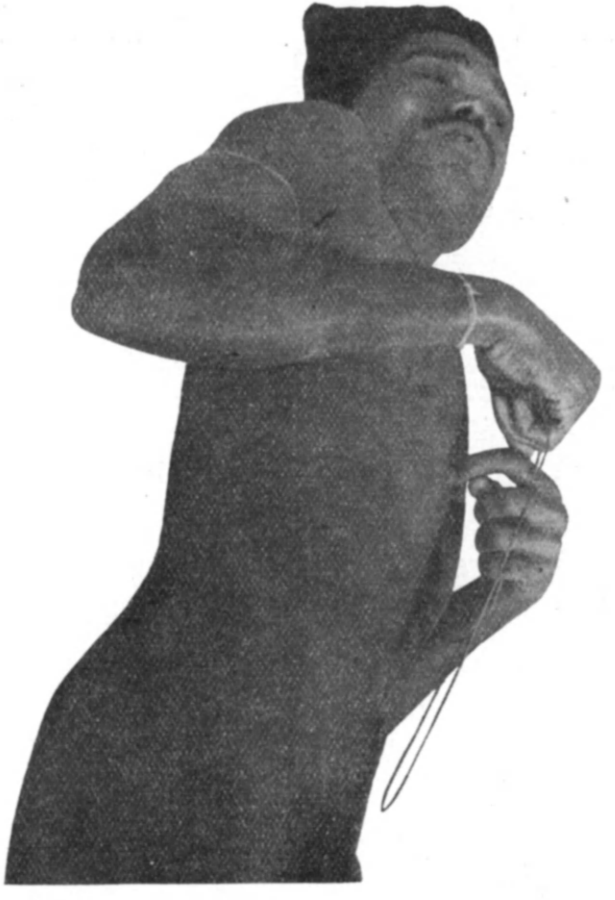


Fig. 23.—Cadaveric Spasm : Note the electric wire firmly grasped in the hands. A case of accidental death from electricity. (*From a photograph kindly lent by Dr. H. S. Mehta*).

7. PUTREFACTION OR DECOMPOSITION

This is absolutely a certain sign of death. It is a slow process and is brought about by the action of ferments produced by living saprophytic micro-organisms, which resolve the complex organised tissues of the body into simpler, inorganic compounds. These micro-organisms are both ærobic and anærobic and, during life, are found in large numbers in the alimentary canal but within a short time after death are found scattered in all the tissues, organs and even in the blood. As a result of their action the dead body invariably putrefies unless special means are taken to prevent their access or the tissues are rendered unfit for their use.

External Phenomena.—It is said that putrefaction follows the disappearance of rigor mortis, but this is not always the case; since, in Northern India, especially during the hot months from April to October, it commences before rigor mortis has completely passed off from the lower extremities. This fact has been observed by me in a large number of dead bodies in Agra and Lucknow. India being a vast country, the climatic conditions vary so much in different parts that it is impossible to give the exact time when the putrefactive process develop in a dead body.

The two characteristic features of putrefaction are the *colour changes* and the *development of foul-smelling gases.*

Colour Changes.—The first external evidence of putrefaction in a body exposed to the air is the formation of greenish discoloration of the abdominal skin over the iliac fossæ. This discoloration is due to the conversion of hæmoglobin of the blood pigment into sulphmethæmoglobin by the action of sulphuretted hydrogen diffusing from the intestine into the tissues, and occurs from one to three days after death in winter, and six to twelve hours in summer. This patch of green discoloration is more evident on a fair skin than on a dark one. About the same time the eyeball becomes soft and yielding, the cornea becomes white and milky and is either flattened or compressed. Later on, the eye collapses and the cornea becomes concave.

From twelve to eighteen hours after death in summer the green colouration spreads over the entire abdomen and the external genitals. Green patches also make their appearance successively on the chest, neck, face, arms and legs. These patches gradually deepen in colour, and later become purple and dark blue. They are at first separate and distinct, but later on coalesce together, and the whole skin of the body appears discoloured.

Soon after the discoloration of the skin has commenced the superficial veins look very prominent like purplish red or green streaks owing to the decomposed blood setting free the colouring matter of the red blood corpuscles, which stains the walls of the blood vessels and infiltrates into the tissues, which also appear coloured. The clotted blood becomes fluid; hence the position of post-mortem staining is altered, and the fluid blood collects in the serous cavities, especially in the pleuræ and pericardium.

Development of Foul-Smelling Gases.—Side by side with the appearance of the greenish patch on the abdomen the body begins to emit a nauseating and unpleasant smell owing to gradual development of the gases of decomposition, some of which are sulphuretted hydrogen, marsh gas, carbon dioxide, ammonia and phosphoretted hydrogen.

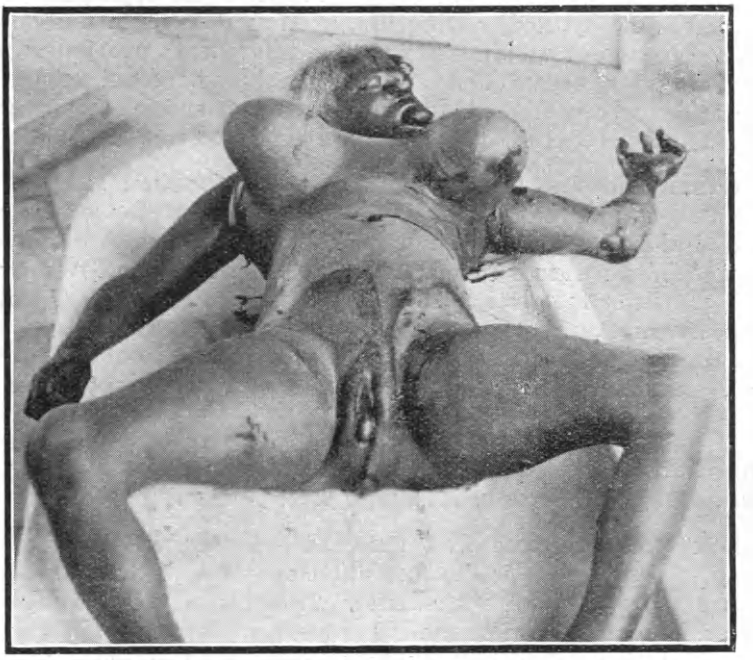


Fig. 24.—Body of a female undergoing decomposition.

From twelve to eighteen hours after death in summer these gases collect in the intestine; consequently the abdomen swells up. The sphincters relax and the urine and fæces may escape.

From eighteen to thirty-six or forty-eight hours after death the gases collect in the tissues, cavities and hollow viscera under considerable pressure with the result that the features become bloated and distorted, the eyes are forced out of their sockets, the tongue is protruded between the teeth, and the lips become swollen and everted. A frothy, reddish fluid or mucus is forced from the mouth and nostrils. Ultimately the features become obliterated and unrecognisable. The abdomen becomes greatly distended; hence on opening the cavity the gas escapes with a loud explosive noise. Owing to the pressure of the gases the stomach contents are forced into the mouth and larynx and are seen running out of the mouth and nostrils. The breasts of female bodies are greatly distended. The penis and scrotum become enormously swollen. The cellular tissues are inflated throughout, so that the whole body appears stouter and older than it actually is.

Owing to the formation of these gases under the skin blisters containing a reddish coloured fluid form on the various parts of the

body. When these burst, the cuticle being softened peels off easily. Bruises and abrasions may become unrecognisable when the cuticle is denuded. Wounds, whether caused before or after death, begin to bleed

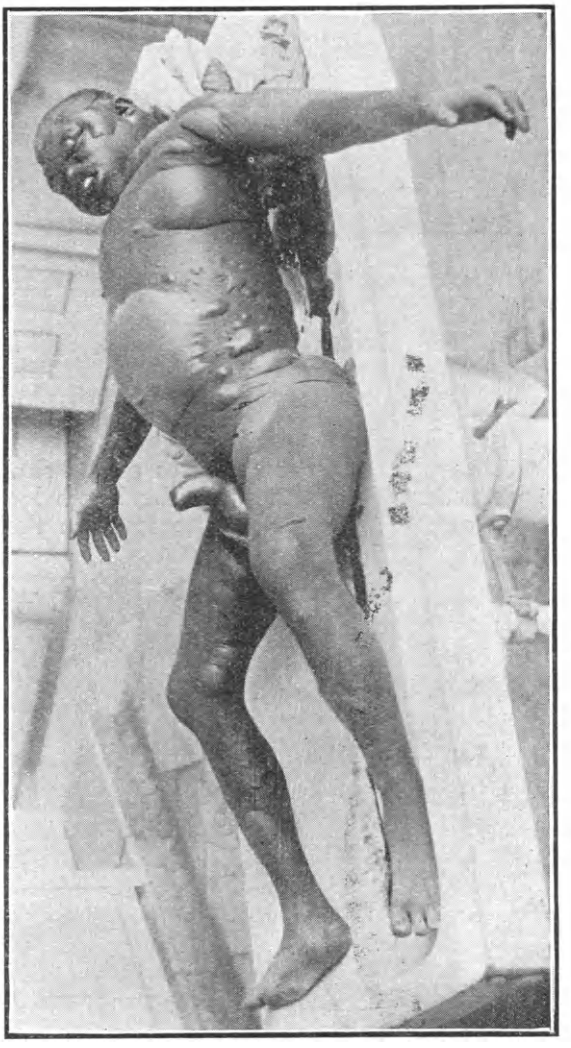


Fig. 25.—Decomposed body of a male showing especially blisters.

once more owing to the pressure of gas within the heart and blood-vessels. Wounds also become so altered in appearance that it may be difficult to form an opinion as to whether they were caused before or after death, unless the presence of the clotted blood can be distinctly made out.

Flies are attracted to the body, and lay their eggs, especially in the open wounds and natural orifices. The eggs hatch out very rapidly in four to eighteen hours into maggots, which crawl into the interior of the

body, and help in destroying the soft tissues. Sometimes, maggots appear even before death, if the person has ulcers on him.

Fig. 26.—Decomposed body of a female showing maggots.

From forty-eight to seventy-two hours the rectum and uterus protrude. The gravid uterus may expel its contents. The hair becomes loose, and is easily pulled out. The nails are also loose, and easily detached.

In three to five days or more the sutures of the skull, especially of children and young persons, are separated, the bones loosened, and the liquefied brain runs out. The teeth become loose in their sockets, and may fall off.

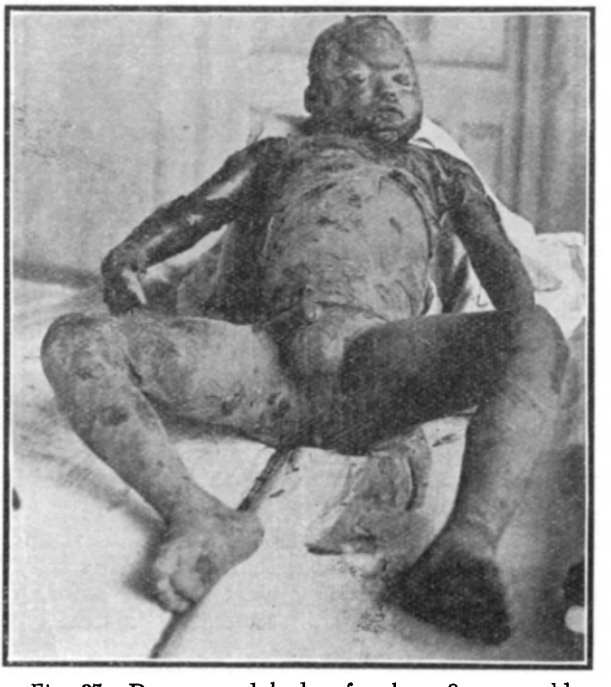


Fig. 27.—Decomposed body of a boy, 8 years old. The cuticle has peeled off at places.

The next stage of putrefaction is known as *colliquative putrefaction* which begins from five to ten days or more after death. During this stage the walls of the abdomen become softened, and burst open protruding the stomach and intestine. The thorax, especially in children, bursts. The diaphragm is pushed upwards.

If the putrefactive processes still go on, the tissues become soft, loose and are converted into a thick semifluid black mass. They ultimately separate from the bones, and fall off. The bones are consequently exposed, and the orbits are empty. The cartilages and ligaments are similarly softened, and ultimately the bones are destroyed, so that after some years no trace of the body is left. The time taken up by these changes varies considerably with the temperature and the medium in which the body lies.

The conclusions arrived at by Mackenzie[1] from his observations of dead bodies in Calcutta are given below in a tabulated form :—

	Average.		Minimum.		Maximum.	
	hr.	m.	hr.	m.	hr.	m.
Muscular irritability	1	51	0	30	4	30
Onset of rigor mortis	1	56	0	40	7	0
Duration of rigor mortis	19	12	3	0	40	0
Cadaveric lividity	14	33	1	38	31	30
Green discoloration	26	4	7	10	41	30
Ova of flies	25	57	3	20	41	30
Maggots	39	43	24	18	76	0
Formation of bullæ	49	34	35	0	72	0
Evolution of gases	18	17	5	50	54	30

Table[2] showing the chronological sequence of the putrefactive changes occurring in the temperate regions.

Putrefactive Changes.	Time.
1. Greenish colouration over the iliac fossæ. The eyeballs, soft and yielding.	1 to 3 days after death.
2. Green colouration spreading over the whole abdomen, external genitals and other parts of the body. Frothy blood from mouth and nostrils.	3 to 5 days after death.
3. Abdomen distended with gas. Cornea fallen in and concave. Purplish red streaks of veins prominent on the extremities. Sphincters relaxed. Nails firm.	8 to 10 days after death.

1. *Ind. Med. Gaz., June,* 1889, *p.* 167.
2. *Casper, Forensic Med., Balfour's Eng. Transl., Vol. I, p.* 38.

Putrefactive Changes.	Time.
4. Body greenish-brown. Blisters forming all over the body. Skin peels off. Features unrecognisable. Scrotum distended. Body swollen up owing to distension. Maggots on the body. Nails and hair loose and easily detached.	14 to 20 days after death.
5. Soft parts changed into a thick semi-fluid black mass. Skull, abdomen and thorax burst. Bones exposed. Orbits empty.	2 to 5 months after death.

Internal Phenomena.—The changes of discoloration similar to those described in the external phenomena of putrefaction are observed in the various visceral organs, such as the liver, spleen and kidneys, but the colour is usually dark red changing to black instead of a greenish colour. This discoloration should not be mistaken for the greenish-yellow or black colouration imparted to the neighbouring organs by the bile soaking through the gall bladder. The pathological changes are still evident, hence it is necessary to go on with the examination. The viscera subsequently become greasy and softened, so that it is difficult to remove them entire.

The rate of putrefaction in the internal organs varies greatly owing to the differences in their structure as regards firmness, density and moisture. From his long experience Casper[1] has drawn up the following table showing the order in which the internal organs putrefy :—

Those which putrefy soon.	Those which putrefy late.
1. The Larynx and Trachea.	9. The Heart.
2. The Brain of Infants.	10. The Lungs.
3. The Stomach.	11. The Kidneys.
4. The Intestines.	12. The Bladder.
5. The Spleen.	13. The Œsophagus.
6. The Omentum and Mesentery.	14. The Pancreas.
7. The Liver.	15. The Diaphragm.
8. The Adult Brain.	16. The Blood Vessels.
	17. The Uterus.

The Larynx and Trachea.—The decomposition of these organs coincides almost with the appearance of the greenish colouration over

1. *Forensic Med., Balfour's Eng. Transl., Vol. I, p.* 44.

the abdomen. Their mucous membrane appears first uniformly brownish-red without any vascular injection, and later becomes greenish and softened. Lastly, the cartilages separate from one another, but this change takes place after some months.

The Brain of Infants.—Owing to the thinness of the skull bones and the presence of the fontanelles the brain of infants very rapidly becomes soft and pulpy, and soon turns into a greyish fluid so that it flows out on removing the cranial bones.

The Stomach.—Owing to the presence of the fermenting food, digestive ferments and bacteria, and a large amount of blood supply, the stomach, as a rule, putrefies much sooner after death. It putrefies usually from twenty-four to thirty-six hours in summer and from three to six days in winter, but it may, sometimes, begin to putrefy much earlier. As a consequence of putrefaction dark-red, irregular patches are first seen on the posterior wall, and then appear on the anterior wall. These patches may be mistaken for the effects of irritant poisoning, but can be readily distinguished as putrefactive changes involve the whole thickness of the stomach wall, while the effects of irritant poisoning are observed usually in the mucous membrane only. Afterwards blebs form on the inner surface of the walls, which become softened, dark brown and ultimately change into a dark pultaceous mass.

The Intestines.—The putrefaction of these organs follows that of the stomach. The intestines are rapidly inflated with the formation of gases in the interior, and the mucous membrane undergoes exactly the same changes as are observed in the stomach. Owing to the walls being softened the intestines burst and discharge their contents.

The Spleen.—In some cases the spleen decomposes earlier than the stomach and intestines, especially if it is swollen and hyperæmic from an acute infectious disease, or enlarged from chronic malaria, but it may resist putrefaction longer, if it happens to be firm and comparatively bloodless. Owing to putrefaction the spleen becomes soft, pulpy, greenish-steel in colour, and within two to three days in summer it may be reduced to a diffluent mass.

The Omentum and Mesentery.—These withstand putrefaction for a long time, if they are free from fat, but decompose sooner, if loaded with fat. In that case they appear greyish-green and dry.

The Liver.—Owing to the effects of decomposition the liver usually becomes softened and flabby in consistence during summer from twelve to eighteen or twenty-four hours after death, and owing to the evolution of gas in its substance it becomes studded with blisters from twenty-four to thirty-six hours. Later, the usual greenish discoloration appears on the upper convex surface, and gradually extends to the whole organ, which ultimately becomes coal-black. The liver putrefies earlier in new-born children than in adults. The gall-bladder is recognisable for a long period owing to its resisting action against putrefaction, but bile-pigments may diffuse early through the adjacent tissues.

The Adult Brain.—The putrefaction of the adult brain first begins at its base, and then proceeds to the upper surface. It is hastened if any injury to the brain or skull is present. The brain becomes soft and pulpy within twenty-four to forty-eight hours in summer, and becomes a liquid mass from three to four days.

The Heart.—The heart putrefies much later than the stomach, intestines and liver. The organ first becomes soft and flabby, and the cavity appears dilated, and is usually empty containing a few gas bubbles. The organ itself can be recognised for several months.

The Lungs.—These organs putrefy at about the same time as the heart or little earlier in a few cases. The first sign of putrescence in the lungs is the formation of gaseous bullæ under the pleural membranes. These are at first pale-red, small, and scattered over the various parts of the lungs, and later on they coalesce. The colour of the lungs does not change with the development of these bullæ, but it then changes to dark, black and green as putrefaction progresses. Later on, the lungs become soft, collapse, and are reduced to a small black mass, which is ultimately completely destroyed. The diaphragm resists putrefaction for a long time, and may be recognisable even after six months.

The Kidneys.—The kidneys become brown and greenish, but retain their consistence for long, so that diseases, such as nephritis and cancer, can be detected for a long time after death.

The Bladder.—This organ, if empty and contracted, resists putrefaction for a long time, but undergoes decomposition rapidly if it has been distended and inflamed. Within forty-eight hours after death the urine in the bladder may usually contain albumin owing to the transudation of serum albumin and globulin from the blood.

The Oesophagus.—The œsophagus withstands putrefaction for a very long time, and may be recognised long after the stomach has entirely disappeared.

The Uterus.—The virgin uterus is the last organ to putrefy, and may be useful in determining the sex long after the complete destruction of the external genitals from advanced decomposition. Casper[1] records a case where the uterus in a body, which had lain in a cesspool for nine months and which was in an advanced stage of decomposition, was found "of a bright-red colour, hard and firm to feel and to cut, its form perfectly recognisable and normal, its size that of a virgin uterus, its cavity unimpregnated and empty." In another case he found the uterus in a well-preserved condition in a mature fœtus which had lain in the earth for about a year. It should, however, be remembered that the impregnated uterus or the gravid uterus soon after delivery rapidly undergoes putrefaction. I have seen some cases in which the uterus was found decomposed in three to four days after death and completely destroyed by maggots in four to five days after death, especially during summer.

1. *Forensic Med., Balfour's Eng. Transl., Vol. I, pp.* 53, 54.

The Blood-vessels.—The blood-vessels, particularly large arterial trunks, resist putrefaction for a long period. The aorta can be recognised after a burial of even fourteen months.

Putrefaction in Water.—The rate of putrefaction of a body in water is more reliable than that of a body exposed to the air or interred, as the temperature of the water is more uniform, and the body is protected from the air, as long as it remains submerged in water. Ordinarily a body takes twice as much time in water as in air to undergo the same degree of putrefaction. Putrefaction is retarded when a body is lying in deep water, and is well protected by clothing. It is accelerated, when once a body has been removed from water, as the tissues have imbibed much fluid. In such a body decomposition is so rapid, that the changes occurring in twenty-four hours' exposure to the air will be more marked than those ordinarily resulting from a fortnight's further submersion.

The colour changes of decomposition are first noticed over the face on the eyelids and ears instead of on the abdomen as in ordinary putrefaction. These changes then gradually spread downwards from the chest to the upper extremities, and lastly from the abdomen to the lower limbs.

The following table drawn up from the observations of Devergie shows the putrefactive changes occurring at different periods of time in a body submerged in water :—

Putrefactive Changes.	Time.
1. Very little change if water is cold. Rigor mortis may persist.	First four or five days.
2. The skin of the hands and feet becomes sodden and bleached. The face appears softened and has a faded white colour.	From five to seven days.
3. Face swollen and red. Greenish discoloration on eyelids, lips, neck and sternum. Skin of hands and feet wrinkled. Upper surface of brain greenish in colour.	One to two weeks.
4. Skin wrinkled. Scrotum and penis distended with gas. Nails and hair still intact. Lungs emphysematous and cover the heart.	Four weeks.
5. Abdomen distended, skin of hands and feet comes off with nails like a glove.	Six to eight weeks.

The above table applies to bodies immersed during winter in temperate regions. Bodies immersed in summer undergo the same changes from three to five or six times as rapidly as in winter, or even more quickly than that.

If fish and crabs happen to be present in water, they destroy the soft parts, and expose the bones in a very short time. On the 2nd June,

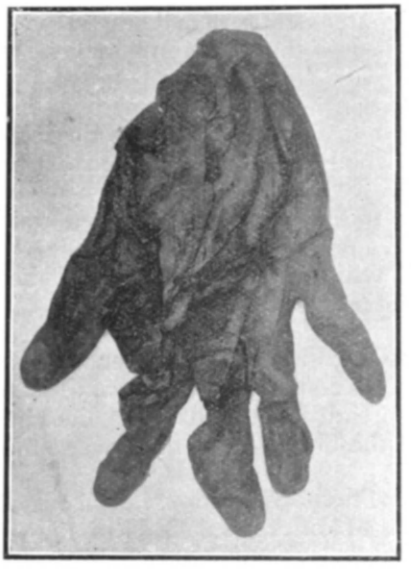


Fig. 28.—Skin from hand after three to four days' immersion in water.

Fig. 29.—Skin from foot after three to four days' immersion in water.

1919, a boy, about twelve years of age, was drowned in the Gomti at Lucknow. On the 4th June, when the body was recovered, almost all the soft parts had disappeared leaving the bones bare.

Floatation of a Body.—The gases of decomposition developed within the submerged body cause it to rise to the surface, unless it is entangled in weeds, ropes, or any other impediment. In India, the body comes to the surface within twenty-four hours in summer, and two to three days or more (rarely more than a week) in winter.[1] In England, the body floats in ten to fourteen days in summer, and in six to eight weeks in winter. The power of floatation of a decomposing body is so great, that it may come to the surface in spite of its being weighted with a heavy stone.

Hehir[2] records the interesting case of a woman who was murdered on a Friday night in September, 1883, and whose body was thrown into a well about midnight. On the following Sunday at about 8 or 9 a.m. the body was found floating with a heavy stone attached to it. The woman was a slight figure and short stature and, while alive, did not probably weigh more than 100 to 105 lbs. The stone, the specific gravity of which was 2.7, weighed 92 lbs. It appears that decomposition in thirty hours was so rapid as to generate gas capable of raising not only the body itself but the dead weight attached to it. The stone was attached to the waist and the body, when found, was lying horizontally on the surface of the water on its side. The water in the well was from ten to twelve feet in depth.

1. *Mackenzie, Ind. Med. Gaz., May,* 1889, *p.* 131 ; *Chevers, Med. Juris., p.* 640.
2. *Outlines of Med. Juris., Ed. V, p.* 56.

The period of floatation depends on the age, sex, condition of the body, season of the year and water.

Age.—The bodies of newly-born infants, if fully developed and well nourished, float rapidly.

Sex.—Owing to the lightness of the bones and a greater proportion of fat the bodies of women are of less specific gravity than those of men, and therefore float sooner.

Condition of the Body.—Fatty bodies float quicker than lean and thin bodies, as fat has a lower specific gravity. Bodies wearing loose clothes will soon come to the surface.

Season of the Year.—The moist, hot air of summer is favourable to putrefaction ; hence dead bodies float quicker in summer than in winter.

Water.—Dead bodies float in the shallow and stagnant water of a pond sooner than in the deep water of a running stream, as the water of a pond, being warmer from the action of the sun's rays, favours putrefaction. Bodies float more readily in sea water than in fresh water, the specific gravity of the former being higher.

Circumstances modifying Putrefaction.—These may be divided into external and internal.

External Circumstances.—These are warmth, moisture, air, and the manner of burial.

Warmth.—Putrefaction commences at a temperature above 50° F., and is most favoured between 70° F. and 100° F., and even upto 115° F. The rapidity of the change considerably lessens as the temperature advances above 100° F. It is altogether arrested below 32° F., and above 212° F. A higher temperature accompanied by dry air generally retards putrefaction.

Moisture.—This is very essential for the occurrence of putrefaction, as the micro-organisms, which are the causative agents of decomposition, thrive well in both heat and moisture. Hence the organs which contain water decompose more rapidly than dry ones.

Air.—The presence of air promotes, and its absence retards, putrefaction. Closely fitting clothes on the body retard decomposition by excluding the air. Similarly, bodies placed in air-tight, lead or zinc coffins resist putrefactive processes for a long period. Moist air favours putrefaction by diminishing evaporation, while dry air retards it. In the same way still air helps putrefaction, and air in motion retards it. Putrefaction is also delayed in bodies completely submerged in water to the entire exclusion of air, and it has been ascertained that at the same temperature the putrefactive changes observed in a body exposed to the air for one week will almost correspond to those in a body submerged in water for two weeks, or buried in a deep grave for eight weeks.[1]

1. *Casper, Forens. Med., Balfour's Eng. Trans., Vol. I, p.* 37.

The Manner of Burial.—Putrefaction is hastened in a body buried in a damp, marshy, clayey soil, or in a shallow grave, where the body will be exposed to constant changes of temperature. It will also be hastened in the case of a body buried without clothes or coffin in a porous soil impregnated with organic matter. Putrefaction is, however, retarded, if a body is buried in a dry, sandy or gravelly soil on high ground, or in a grave deeper than six feet, and also if the body is well covered with clothes and placed in a tightly fitting coffin. Lime and charcoal, when sprinkled on the body, do not hasten or retard putrefaction, but act as deodorizers to some extent, as they have the power of absorbing gases emanating during decomposition.

Internal Circumstances.—These are age, sex, condition of the body, and the cause of death.

Age.—The bodies of children putrefy more rapidly than those of young adults. The bodies of old people do not decompose rapidly, probably owing to a less amount of moisture.

Sex.—Sex has no influence on putrefaction, but the bodies of females dying soon after child-birth decompose rapidly, especially if death has been due to septicæmia.

Condition of the Body.—Fat and flabby bodies putrefy more quickly than thin and emaciated ones. Those parts of the body which are the seats of bruises, wounds or fractures, or which have been mutilated, decompose very early.

Cause of Death.—The bodies of persons who have died from acute infectious fevers and chronic diseases terminating in septicæmia or general anasarca decompose more rapidly than those of healthy persons who have died suddenly from accident or violence. Putrefaction is hastened after death occurring from asphyxia as in lightning, strangulation, and suffocation by smoke, coal gas, hydrogen sulphide or sewer gas and certain poisons, *e.g.*, hydrocyanic acid and opium. On the other hand, putrefaction is retarded after death occurring from wasting diseases attended with emaciation and anæmia, and also from certain poisons which act on the body tissues as antiseptics, *e.g.*, arsenic, antimony, zinc chloride, phosphorus, and mineral acids, especially sulphuric acid. Death by chronic alcoholism generally hastens decomposition, while death by chloroform and strychnine poisoning delays it.

8. ADIPOCERE

Under certain conditions the progress of putrefaction in a dead body is checked, and is replaced by the formation of adipocere, which is a waxy-looking substance having a greasy feel and a pure white or faint yellowish colour. It cuts soft, and melts at a flame, or burns with a feebly luminous flame giving off a dull cheese-like, but by no means a disagreeable, smell. Its specific gravity being less than that of water, it floats when placed in the latter. It is more or less permanent lasting

for several years, but becomes hard, brittle and yellow, when exposed to the air. The results of chemical analysis of my cases (see below) go to support the following remarks of Lucas[1] regarding the chemical composition and formation of adipocere :—

"It is evident that adipocere is composed almost entirely of fatty acids, but that it contains a certain amount of calcium soap and probably in the early stages of its formation some ammonia soap and therefore from its chemical composition there can be little doubt that adipocere is the residue of that fat pre-existing in the body, the greater part of which has undergone slow hydrolysis by water but some small part of it has been saponified by ammonia (derived from decomposing nitrogenous tissue), this ammonia being ultimately replaced by lime."

Sydney Smith[2] believes that adipocere is not connected with the formation of soaps, but it is formed by a gradual hydrogenation process in which pre-existing fats in the body are converted into higher fatty acids.

Adipocere commences first in the subcutaneous fat, and then in the skin, muscles and organs. It occurs soon in the female breasts, cheeks, buttocks and other parts of the body, where large accumulations of fat are found. As fat is distributed extensively throughout the body, nearly all parts may undergo this change.

It is rare for the whole body to be converted into adipocere, but Guy[3] saw whole bodies completely transformed among the large number of bodies disinterred while laying the foundations of King's College Hospital. Glaister[4] also mentions having seen the whole body so transformed in more than one case when the pits into which bodies had been buried were dug up.

Water is necessary for the formation of adipocere, so that the process takes place in bodies which have been submerged in water, buried in damp, clayey soils, or thrown into cesspools. It may, however, occur in bodies exposed to the air, especially in hot and damp climates. Vaughan[5] reports two cases of such an occurrence. In one case the body of a Hindu female lay in a dry bed covered with bedding and heavy pillows in a room, and in the other case the body of a man had lain absolutely naked on the ground in a hut in a plantation of trees and shrubs.

Time of Formation of Adipocere.—The time required for the formation of adipocere varies according to the climate. In Europe, it ranges from three months to one year, though the change may occur in five weeks, or may be delayed to three years. It is more rapid when a body is submerged in water than when it is buried in the earth. In India, Dr. Coull

1. *Forensic Chem., p.* 253.
2. *Forensic Medicine, Ed. VI, p.* 32.
3. *Forensic Med., Ed. VI, p.* 226.
4. *Med. Juris. and Toxic., Ed. VI, p.* 126.
5. *Ind. Med. Gaz., May,* 1906, *p.* 161.

Mackenzie[1] found it occurring within three to fifteen days after death in bodies drowned in the Hughli or buried in the damp soil of Lower

Fig. 30.—The body of a child converted into adipocere.

Bengal. I have observed adipocere taking place in seven to thirty-five days after death in bodies submerged in wells or buried in shallow graves. Professor Powell[2] records its formation in three days and twenty-two hours after death in the body of a healthy male buried in a gravelly and sandy soil.

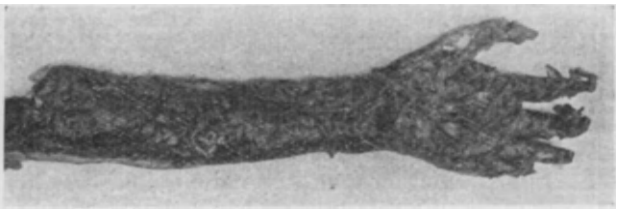


Fig. 31.—A forearm converted into adipocere.

Of twelve cases of adipocere which came under my observation during a period of six years between 1917 and 1922, I quote the following typical cases :—

1. On February 26, 1922, a report was made at Police Station, Malihabad, Lucknow District, that Ramadhin, Brahmin, 55 years old, resident of Ramgarh was missing. On the 2nd April, the body of the said Ramadhin was found in a well in village Hamirpur. A post-mortem examination was held on the following day. The body was well preserved. There was no disagreeable smell. The eyeballs had been disintegrated and the teeth loosened. Saponification had taken place in the soft tissues of the trunk, scrotum, penis and extremities. There was an incised wound, measuring four inches by two inches, across the right side of the neck cutting the larynx below the thyroid cartilage, and the fifth cervical vertebra. The brain was liquefied. The lungs were decomposed and disintegrated to a small black mass. The abdominal fat and mesentery were saponified. The liver appeared to be undergoing saponification. The spleen was reduced to almost a liquid mass. The kidneys were reduced to a small putrefied mass, but the bladder was normal and empty.

2. The body of Musammat Jasoda, a girl of 4 years, was taken out of a well on the 26th March, 1923. On examination externally the buttocks were found

1. *Brit. Med. Jour., Vol. I,* 1917, *p.* 842.
2. *Ind. Med. Gaz., Feb.,* 1889, *p.* 42.

saponified and internally the mesentery and omentum. The girl was robbed of her silver ornaments and thrown into the well about 10 days ago.

Pathological Examination.—In these two cases pieces of saponified tissues, *viz.*, muscle, mesentery, omentum and liver, were sent to the Pathological Department of the King George's Medical College for section cutting and examination. Dr. Mukarji, Reader in Pathology, furnished me with the following report :—

"Microscopically the tissues appeared to be yellowish-white, disintegrated masses. Under the microscope no definite structure was seen, but masses of acicular crystals and round bodies about three times the diameter of a red blood cell were detected. When treated with xylol most of the above were dissolved leaving a hazy round outline. In all probability the round bodies consisted of neutral fat and soap."

Chemical Analysis.—The following results of analysis were obtained in the laboratory of Mr. D. N. Chatterji, Chemical Examiner for the United and Central Provinces at Agra :—

Case No. 1.

Pieces of saponified muscle, mesentery, fat and liver were removed from the body and placed in a bottle with rectified spirit. These were forwarded for chemical analysis. The fatty substance got completely dissolved in the alcohol.

The total amount of fatty substance recovered from the alcohol at the time of analysis in November (about seven months after the post-mortem examination) was 63 grammes. It was practically soluble in ether, and contained only traces of mineral matter. The other tissues on analysis were found to contain—

Ash	5.6 per cent.
Ethereal extract	53.4 per cent.

The quantity of unsaponified matter in the fatty substance recovered from the preservative spirit and the other tissues was 2 per cent.

Soap was present, but in a very small quantity ; this soap was probably a lime soap. Most of the adipocere consisted of free fatty acids, *viz.*, stearic and palmitic acids. No ammonia was present. The ash from the tissues contained lime, soda and potash compounds.

Case No. 2.

Pieces of saponified buttock and mesentery were sent for chemical analysis preserved in rectified spirit.

The following is the result of analysis made in July, 1923 :—

The total amount of fatty matter which was dissolved in the alcohol was 23.4 per cent. It contained only 0.2 per cent. of mineral matter and 0.5 per cent. of unsaponifiable matter. The results of the analysis of the tissues are—

	Ash %.	Ethereal extract %.	Unsaponifiable matter %.
Buttock,	0.4	7.35	23.1
Mesentery,	0.7	80.0	48.7

No soap was found. The fatty matter obtained from the preservative spirit consisted of free fatty acids, *viz.*, stearic and palmitic acids. No ammonia could be found. The ash from the tissues sent contained lime, soda and potash compounds.

9. MUMMIFICATION

Fig. 32.—A mummified body. (*From a photograph kindly lent by Dr. G. B. Sahay*).

The term, mummification, is applied to a peculiar desiccation of a dead body, whereby its soft parts shrivel up, but retain the natural appearance and even the features of the body. The skin is dry, leathery and rusty-brown in colour, and adheres closely to the bones. The odour is more like that of old cheese than that of a decomposed body. The internal organs either disappear altogether, or blend together and get transformed into a thick mass of a dark-brown, dry substance, from which they cannot be separately distinguished.

Mummification occurs in bodies buried in shallow graves in the dry, sandy soils of Rajputana, Sind and Baluchistan, where evaporation of the body fluids is more rapid owing to hot, dry winds prevailing in the summer season. It is observed also in the bodies kept perched up on trees, or rafters of a roof, as also in those kept closed in steel trunks. Arsenic or antimony poisoning is said to favour the process of mummification in dry, warm climates.

Time of Mummification.—The time taken by a dead body to mummify is not exactly known, but it may be regarded as varying from three months to a year or two.

The artificial method of mummifying or embalming dead bodies was known to the ancient Egyptians, and specimens of their mummies are to be found in the British Museum of London in a very well preserved condition after thousands of years. At present it is resorted to in medical schools and colleges to preserve dead bodies for the purpose of dissection by injecting solutions of arsenic, lead sulphide and potassium carbonate into the femoral artery or into the aorta. The process has, sometimes, to be adopted when dead bodies have to be taken from one country to another for burial, and when the time taken in transit is so much as would ordinarily lead to putrefaction.

TIME OF DEATH

It is very important from a medico-legal point of view that a medical jurist should always be prepared to give an opinion as to the time which

elapsed since death, when a body is brought to him for post-mortem examination. The points to be noted in ascertaining the time are warmth or cooling of the body, the absence or presence of cadaveric hypostasis, rigor mortis and the progress of decomposition. All these points have been discussed at full length, but it must be remembered that the conditions producing these changes vary so much in each individual case, that only an approximate time of death can be given.

In addition to these, the time of death can be ascertained with some possibility from the degree of digestion of the stomach contents, and from the condition of the bladder and intestines as regards their contents.

It has been ascertained by physiologists that a mixed diet containing more of animal food and less of vegetable food as ordinarily taken by a European leaves the stomach in four to five hours after it is completely digested, while a vegetable diet containing mostly farinaceous food as usually taken by an Indian does not leave the stomach completely within six to seven hours after its ingestion. But this cannot always be relied upon in determining the time of death, inasmuch as the power of digestibility may remain in abeyance for a long time in states of profound shock and coma. I have seen the food in the stomach remaining undigested in persons who received severe head injuries soon after their meal and died within twelve to twenty-four hours afterwards. In one case the food consisting chiefly of rice and *dal* (pulse) remained in the stomach for about forty hours without undergoing digestion. It must also be remembered that the process of digestion in normal, healthy persons may continue for a time after death.

In some cases the time of death may be calculated by examining whether the bladder or intestines are empty or not. Thus, in the case of a murder of an individual having taken place in bed at night, one can state that the individual had lived for some time after going to bed if the bladder was found full of urine, since people usually empty their bladder before going to bed. Similarly, one can give an opinion that the death occurred some time after he had got up in the morning if the large intestine was found empty of fæcal matter.

PRESUMPTION OF DEATH

The question of presumption of death may arise at the time of inheritance of property or in obtaining insurance money, when a particular person has gone abroad and has not been heard of for a considerable time, or when he is alleged to have been dead and the body is not forthcoming. Under the Evidence Act[1] of India, if it is shown that a person was alive within thirty years, and there is nothing to suggest the probability of his death, there is a presumption that he is still alive unless proof be given that the same person has not been heard of for seven years by those friends and relatives who would naturally have heard from him had he been alive. But there is no legal presumption that he died at any particular time during the seven years. The onus of proving it lies on the person who asserts such fact.

1. *Sections* 107 *and* 108, *Appendix V.*

PRESUMPTION OF SURVIVORSHIP

The question of presumption as to survivorship may arise in connection with the devolution and distribution of property, when two or more persons, natural heirs of each other, lose their lives in a common disaster, such as earthquake, shipwreck, battle, conflagration, etc. Section 184 of the Law of Property Act of England, 1925, provides that in all cases where two or more persons have died in circumstances rendering it uncertain which of them survived the other or others, such deaths shall (subject to any order of the Court), for all purposes affecting the title to property, be presumed to have occurred in order of seniority, and accordingly the younger shall be deemed to have survived the elder. It is, however, open to the parties interested to show by evidence in Court that one in fact survived the other or others. The law of India does not recognise any presumption regarding the probabilities of survivorship among persons whose death is occasioned by one and the same cause, and the Courts are influenced in establishing the survivorship in such cases by the facts and evidence, where available. In the absence of such evidence the following conditions should be taken into consideration in determining the question of survivorship with a reasonable certainty :—

Injury.—Wounds, even if small and insignificant, inflicted on the vital organs or main blood vessels, are likely to produce death much earlier than injuries, even though extensive, inflicted on those parts of the body which are not vital.

Age.—Adults have the power of resistance against a common danger more than the young and the old, and it is, therefore, presumed that the former will survive the latter but much will depend on the mode of death.

Sex.—Males, being stronger, are presumed to survive longer than females, but when there is a question of physical endurance females will live longer than males, as the former can withstand severe physical strain better than the latter.

Constitution.—Vigorous and healthy individuals are ordinarily presumed to live longer than the weak and those debilitated from disease.

Mode of Death.—The following modes of death should be particularly discussed :—

Drowning.—Females may be presumed to survive longer than males, as the former are more likely to faint from dread, which delays asphyxia. However, in cases where there has been a struggle for life, men being stronger will probably survive women, and those who know swimming will live longer than those who do not. In cases where bodies are recovered from water the presence of severe injuries is likely to be regarded as a plea against survivorship, and evidence of an attempt to save others, as shown by the position of two bodies, will be strong proof of survivorship.

Suffocation.—In a common accident such as that occurring from the debris of a fallen roof, persons who have least injuries, and who are nearer the surface and consequently not buried deep under the debris, are presumed to have died last.

Asphyxia from Want of Oxygen or from Irrespirable Gases.—Women consume less oxygen and are, therefore, supposed to live longer than men in an atmosphere containing a less amount of oxygen. Again, an individual will be required to consume more oxygen if he were to make a muscular effort to escape the danger, as he will be more liable to the danger than one who is inactive and makes no exertion.

Starvation.—Fatty persons have a better chance of outliving the lean, as they can live on their fat for some time. Again, one deprived of food alone will live longer than one deprived of both food and water, as water alone enables a person to live for many days. In the case of children, adults and old people exposed to starvation, children will die first, then adults and lastly the old, as the old require less nourishment than adults, and adults less than children. In the same manner women consume less food than men and can bear starvation longer and better.

Cold.—Ordinarily adults are presumed to live longer than the young and the old, as the former endure cold better than the latter. Men generally bear cold better than women, but this hypothesis should be modified by the amount and kind of clothing, the physical condition of the body, and the habit of using alcohol or other intoxicating drugs.

Heat.—Adults do not bear heat so well as children and old people, and the former are, therefore, supposed to die before the latter if exposed to a common danger of heat.

Burns.—Children die sooner from the effects of extensive burns than adults, as the former are very susceptible to shock; the same is true of old people as compared with adults.

Delivery.—When mother and child die during delivery without witnesses, there is a strong presumption that the mother survived the child, but, if she died of hæmorrhage, it would be presumed that she died first. But it should be remembered that in cases of survivorship of a child it will be necessary to prove that the child was born alive.

In addition to the above considerations, the medical man should note the presence of the degree of warmth and rigor mortis to ascertain which died first, if several bodies meeting with death in the same accident were sent to him for post-mortem examination.

CHAPTER VII

DEATHS FROM ASPHYXIA

Violent deaths resulting chiefly from asphyxia are: Hanging, Strangulation, Suffocation and Drowning.

HANGING

Fig. 33.—Suicidal Hanging.

Definition.—Hanging is a form of death produced by suspending the body with a ligature round the neck, the constricting force being the weight of the body. The term, "partial hanging", is used for those cases in which the bodies are partially suspended or for those in which the bodies are in a sitting, kneeling, reclining, prone, or any other posture. In all such cases death is inevitable if there is enough force upon the ligature to constrict the neck.

The Nature of the Ligature used.—Any substance that is available at the time of the impulse has been used by suicides as a ligature for hanging, *e.g.*, cotton, hemp or *moonj* rope of any thickness, *newar, dhoti, saree,* turban (*safa*), bed-sheet, sacred-thread, neckerchief, neck cloth (*dupatta*), etc. When a material with which an individual is alleged to have been hanged is sent for medical examination the medical jurist should see if the mark on the neck corresponds with its thickness, and if it is strong enough to bear the weight of the body or the sudden strain. He should also note its texture and length, and after labelling it with some distinctive mark for future identification should return it in a sealed packet to the police constable who brought it.

Symptoms.—The first symptoms are the loss of power and subjective sensations, such as flashes of light, and ringing and hissing noises in the ears. These are followed by loss of consciousness, which is so very rapid that hanging is regarded as a painless form of death. Owing to this rapid unconsciousness, an effort at saving oneself is not possible in accidental or suicidal hanging. In the case

of judicial hanging convulsive movements of the limbs may be seen. Respiration stops before the heart which may continue to beat for about ten minutes.

Causes of Death.—1. *Asphyxia.*—In most cases this is the true cause of death. The ligature is usually situated above the thyroid cartilage, and the effect of its pressing the neck in that situation is to force up the epiglottis and the root of the tongue against the posterior wall of the pharynx. Hence the floor of the mouth is jammed against its roof, and occludes the air passages.

2. *Apoplexy or Cerebral Congestion.*—This is due to congestion of the venous blood in the brain from compression of the large (jugular) veins in the neck caused by a ligature round the neck.

3. *Combined Asphyxia and Apoplexy.*—This is supposed to be the commonest cause of death, as in most cases the air passages are not completely blocked by the ligature passed round the neck.

4. *Syncope.*—This results from pressure on the large arteries of the neck, which prevents blood from going to the brain thus causing anæmia.

5. *Shock.*—This occurs from pressure on the pneumogastric nerves.

6. *Fracture or Dislocation of the Cervical Vertebræ.*—In judicial hanging, a sudden drop of five to seven feet produces fracture or dislocation of the cervical vertebræ which, compressing or lacerating the spinal cord, causes instantaneous death. Usually the first and second vertebræ are injured, but in a few cases the third and fourth vertebræ may be found fractured or dislocated.

Fatal Period.—Death is almost instantaneous, if the cervical vertebræ are fractured as in judicial hanging. It may occur instantaneously or rapidly in cases of asphyxia, but usually in five to eight or ten minutes if the blocking of the air passages is partial only. Death is, as a rule, slow in cases of apoplexy.

Treatment.—The first and the most important thing to do is to let the individual down, and to remove constriction of the neck by cutting the ligature. Artificial respiration should then be used after pulling out the tongue, and wiping the froth from the mouth and nostrils. This may be supplemented by ammonia vapour to the nose and tickling the fauces. Cold affusion may be applied to the head, and the galvanic battery may be used if the body is warm but if the body is cold warmth should be restored by friction and mustard plaster on the chest, abdomen and calves. If the patient is able to swallow, stimulants should be given by the mouth, otherwise they should be given hypodermically or per rectum. It may be necessary to perform venesection to relieve distension of the right side of the heart and pulmonary circulation or cerebral congestion. The patient should be watched for some time after respiration has been established, as death may occur from a relapse of the symptoms.

The secondary effects of hanging in subjects, who have recovered, are, sometimes, hemiplegia, epileptiform convulsions, amnesia, dementia, bronchitis, hæmoptysis, cervical cellulitis, parotitis and retropharyngeal abscess.

In August, 1919, a Hindu woman, aged thirty, who hanged herself while under the influence of opium was cut down. On the 3rd day she died of meningitis. On post-mortem examination the brain and its meninges were congested and the lungs were congested and œdematous.

Fig. 34.—Attempted Suicide by Hanging.

Post-mortem Appearances.—These are external and internal.

External Appearances.—External appearances are those due to the ligature on the neck and those peculiar to the mode of death.

Ligature-Mark. — This depends on the nature of the ligature used, and the time of suspension of the body after death. If the ligature be soft, and the body be cut down immediately after death, there may be no mark. Again, the intervention of a thick and long beard or clothes on the neck may lead to the formation of a slight mark only.

Situation of the Mark.—The mark is usually situated above the thyroid cartilage between the larynx and the chin, and is directed obliquely upward following the line of the mandible (lower jaw) and interrupted at the back, reaching the mastoid processes behind the ears. The mark may be found on or below the thyroid cartilage, especially in cases of partial suspension. It may also be circular if a ligature is first placed at the nape of the neck and then its two ends are brought horizontally forward and crossed, and carried upward to the point of suspension from behind the angle of the lower jaw on each side. The mark will be both circular and oblique if a ligature is passed round the neck more than once.

Character of the Mark.—The mark varies according to the nature of the material used as a ligature and the period of suspension after death. It is a superficial and broad mark if a cloth or a soft rope is used; while it is well-defined, narrow and deep, if a firm string is used. The mark is a groove or furrow, the base being pale, hard, leathery and parchment-like, and the margins red and congested. The colour becomes reddish-brown or chocolate brown if seen after some hours of suspension. Ecchymoses and slight abrasions in the groove are rare, but may be found in some cases, *e.g.,* in judicial hanging. Ecchymoses alone have no

significance as to whether hanging was caused during life or not, but abrasions with hæmorrhage are strongly suggestive of it having taken place during life.

Other Signs.—The neck is found stretched and elongated, and the head is always inclined to the side opposite to the knot. The face is usually pale and placid, but may be swollen and congested if the body has been long suspended. The eyes are closed or partly open, and the pupils are usually dilated. The tongue is drawn in, or caught between the teeth, or protruded and bitten. It is usually swollen and blue, especially at the base. Bloody froth is, sometimes, seen at the mouth and nostrils. Saliva is often found running out of the angle of the mouth down on the chin and chest. This is a sure sign of hanging having taken place during life, as the secretion of saliva being a vital function cannot occur after death. The hands are often clenched, especially in violent hanging. Turgescence of the genital organs generally occurs in both sexes from hypostasis. Seminal fluid is, sometimes, present at the urethral meatus, but it is not a diagnostic sign of hanging, as it has often been observed by me in those who suddenly died from mechanical violence. Escape of urine and fæces is often found from relaxation of the sphincters; it is also of no diagnostic value, as it is seen in other forms of death. Post-mortem staining will be seen on the lower parts of the body if suspension has been continued for some time after death.

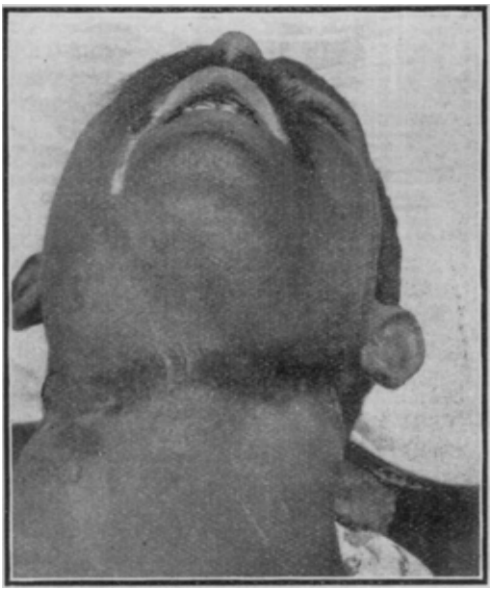


Fig. 35.—Front view showing a ligature mark of hanging in the neck and dribbling of saliva from the right angle of the mouth.

Internal Appearances.—On dissection the subcutaneous tissue under the ligature mark is usually dry, white and glistening—more marked if the body has been suspended for a long time. The muscles of the neck, especially the platysma and sterno-mastoid, are likely to be ruptured only when considerable violence has been used in hanging. The inner and middle coats of the carotid arteries may be found lacerated with an extravasation of blood within their walls, if there has been a sudden drop as in judicial hanging. Similarly, fracture-dislocation of the cervical vertebræ together with compression or laceration of the spinal cord may be noticed. In rare instances, the processes of the thyroid cartilage may be fractured, but the hyoid bone is, as a rule, not fractured.

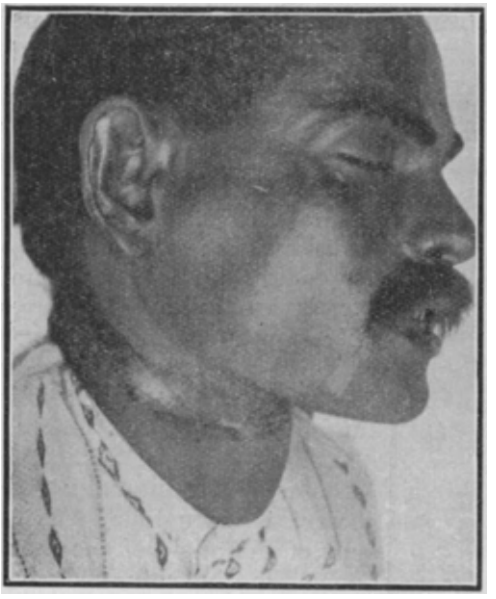


Fig. 36.—Side view showing the ligature mark of hanging in the neck and dribbling of saliva from the right angle of the mouth.

The epiglottis is frequently congested. The mucous membrane of the trachea is found to be red and congested containing a fine bloody froth in some cases. Very rarely, it is found to be lacerated.

In the case of constriction occurring at the end of expiration the lungs are congested, œdematous and exude bloody serum on being cut, but are pale if constriction occurred at the end of inspiration. Subpleural ecchymoses are very rare. The right side of the heart, the pulmonary artery and venæ cavæ are full of dark fluid blood, and the left side is empty. The abdominal organs are usually congested. The brain is usually normal, but may be pale or congested according to the mode of death.

Medico-Legal Questions.—The medico-legal questions likely to arise in a case of hanging are—

1. Whether death was caused by hanging.

2. Whether the hanging was suicidal, homicidal or accidental.

1. **Whether Death was caused by Hanging.**—In India, it is a common practice to kill a victim, and then to suspend the body from a tree or a rafter to avert suspicion. It is, therefore, necessary to find out if hanging was the cause of death in a suspended body.

The presence of a ligature-mark alone is not diagnostic of death from hanging, inasmuch as, being a purely cadaveric phenomenon, it may be produced if a body is suspended after death. Casper[1] has illustrated by experiments that a mark similar to one observed in persons hanged alive can be produced if suspended within two hours or even a longer period after death. Besides, a similar mark may also be produced by dragging a body along the ground with a cord passed round the neck soon after death. However, one can safely say that death was due to hanging if, in addition to the cord mark, there were trickling of saliva from the mouth, ecchymoses and slight abrasions about the ligature-mark, laceration of the intima of the carotid arteries with an extravasation of blood within their walls and the post-mortem signs of asphyxia, and if there were no evidence of the signs of a struggle, fatal injuries, or poisoning.

1. *Forens. Med., Eng. Transl., Vol. II, p.* 173.

2. **Whether the Hanging was Suicidal, Homicidal or Accidental.**—Hanging is usually suicidal. Of thirty-two cases of hanging that came under my observation during a period of over six years thirty were suicidal. One was suspended after murder and in the other there was a presumption of homicide.

Blindness or age is no bar to suicidal hanging. A blind man of seventy-five committed suicide by suspending himself from the branch of a tree in Lucknow. After chastisement or some other violence children are known to have committed suicide by hanging from shame or grief. A case occurred in Jubbulpore, where a Muslim lad, 12 years old, quarrelled with his elder brother one night, and committed suicide by hanging himself from the ceiling of his house the next day.[1] Sometimes, hanging is adopted as a last resort, after other forms of suicide, *e.g.*, cutting of the throat or ingestion of poison, have failed to produce the desired effect. In December, 1916, a Hindu girl, aged 16 years, first took poison, then tried to cut her throat and lastly, gathering courage, hanged herself.

Homicidal hanging, though rare, has been recorded. Usually more than one person are combined in the act, unless the victim is a child, or very weak and feeble, or is rendered unconscious by some intoxicating or narcotic drug. In a case where resistance is offered, marks of violence on the body and marks of a struggle or footprints of several persons at or near the place of the occurrence are likely to be found.

"Lynching" is the form of homicidal hanging which is confined to the southern States of America. When a negro is accused of having committed some serious offence, *e.g.*, rape upon a white girl, the mob get enraged, take him from the police custody and hang him by means of a rope from a tree or some similar object.

Accidental hanging is extremely rare. It has occurred among children during play while imitating judicial hanging, or among athletes who are in the habit of exhibiting hanging.

ILLUSTRATIVE CASES

Suspension after Murder.—In June, 1916, the body of a Hindu male, aged 30 years, was brought to the Agra Medical School Mortuary with a police report that the deceased committed suicide by hanging. The post-mortem examination showed ecchymosis of the skin of the scrotum, especially on the left side and effusion of clotted blood round about the left testicle, which appeared to have been crushed. There was a ligature mark between os hyoides and thyroid cartilage, interrupted at the back. There was no congestion or abrasion about its margins. Death was found to be due to forcible crushing of the left testicle, and the body was suspended after death.

Homicide.—1. A prisoner who had been sentenced to three years' hard labour was being brought from Seona to Patiala Central Jail escorted by an elderly police constable. On the way the prisoner struck the constable on the head with the handcuffs on his wrists with the result that he fell down unconscious. The prisoner then took the key of the handcuffs from the constable's belt and set himself free. He then tied a turban round the constable's neck and hanged him from a branch of a tree.—*The Times of India, September* 8, 1937.

1. *Hindustan Times, Jan.* 8, 1933, *p.* 3.

2. A girl, aged 18, was found hanging from the parapet of a bridge over a stream in a kneeling posture. She was 3-5 months pregnant. The girl was pregnant by a man, aged 23, who had tried to poison her by the contents of a so-called Indian "poison bladder" which contained in one 2 c.c. ampoule 0.3 gm. of hydrocyanic acid in solution. The girl inhaled the acid and immediately suffered from sickness and vomiting. The knot in the rope was made in the same way as that of the halter of the cows at the accused's home. The accused confessed that he had wound round the girl's neck the rope he had previously prepared and pushed her from the bridge and fastened the rope to the parapet.—*W. Schwarzacher, Beitr. Gerichrl. Med.* 1931, *XI*, 48-53; *abstr.; Deuts. Zeits. f. d. ges. gerichtl. Med.*, 1932, *XIX*, 27; *The Med.-Legal and Criminolog. Rev., Jan.*, 1933, *p.* 85.

3. After terrorising a rich merchant in village Dholera the robbers removed all cash and ornaments, burnt the account books and hanged the merchant from the ceiling of the roof of the house where he was found dead.—*Leader, April* 29, 1934.

4. The following case came under my notice in which there was presumption of homicide:—

On the night of October 23, 1910, a Hindu male went to sleep on a *charpoy*, after taking his evening meal. The next morning he was found dead in his bed. At the necropsy saliva was seen trickled down the right angle of the mouth and an oblique ligature mark was seen in the upper and front part of the neck from ear to ear with congestion in the internal organs. It was presumed that some one entering the house passed a cord round his neck and pulling up the head, gave it a downward jerk while the deceased was in sound sleep.

A Case of Lynching.—A mob of about a thousand persons including women and children burst into the country gaol at Princess Anne, Maryland, defying a barrage of tear gas and the truncheons of twenty police, and carried off a negro who was accused of assaulting an aged white woman. He was stripped of all his clothes, a rope was tied round his neck, and he was dragged through the town behind a motor car with the mob howling and cursing him. Then he was hanged from a tree next to the Judge's house. Later, petrol was poured over the body and it was burned in a public square.—*Leader, Nov.* 27, 1933.

Accident.—1. In 1840, Scott, the American diver, was in the habit of making public exhibitions of hanging but, on the last occasion, the ligature apparently shifted, for, he was asphyxiated; he was, however, allowed to hang for thirteen minutes by the spectators who thought he was prolonging the experiment for their gratification.—*Taylor, Prin. and Pract. of Med. Juris., Ed. IX, Vol. I, p.* 675.

2. A boy, aged one year and a half, was swinging by two ropes attached to two posts; the ropes became twisted round his neck and he was consequently hanged.—*Harvey, Ind. Med. Gaz.*, 1876, *p.* 3.

3. In order to punish her child a woman tied the arms above the elbows with a stocking fixed to a string, which in turn was fixed to a ring in the wall of a dark closet. At the end of three hours the child was found dead, having been suspended by the string, as the stocking accidentally slipped up round the throat and constricted it.—*R. v. Montagne, Dublin Assizes,* 1892.

STRANGULATION

Definition.—Strangulation is a violent form of death, which results from constricting the neck by means of a ligature without suspending the body. It is called throttling when constriction is produced by the pressure of the fingers upon the throat. Strangulation may also be brought about by compressing the throat with a foot, knee, elbow, or some other solid substance.

A form of strangulation, known as *Bansdola,* is, sometimes, practised in Northern India. In this form one strong bamboo or *lathi* (club) is placed across the throat, and another across the back of the neck. These

are strongly fastened at one end; a rope is passed round the other ends, which are bound together, and the unfortunate victim is squeezed to death. The throat is also pressed by placing a *lathi* (club) or bamboo across the front of the neck and standing with a foot on each end of the *lathi* or bamboo.

In the case of *K. E.* v. *Jeevan and Cheda* charged under Section 302, I.P.C., with having murdered one Duja, 25 years old, it was proved in evidence that Jeevan threw down Duja and put a stick on his neck, each of the accused pressing it down with his foot at either end till Duja was dead.[1]

On the 16th April, 1927, one Khemkaran was lying on a *charpoy* (bedstead) when one Mansa thrust a stout piece of bamboo, about four feet long, forming the bottom part of a yoke, under the wood of the *charpoy* on one side. He had this length of a bamboo pressed against Khemkaran's throat and kept it down at the other end with his foot till death. He then threw the body into a well.[2]

On August 30, 1934, one Wanarse of a village known as Humgaon, District Satara, who was suspected to have set fire to Mahangade's house, was seized and made to lie flat on the ground; a bamboo stick was placed across his neck and Mahangade and Bhilare stood on either end of the stick till the man was strangled to death. The body was then taken to another village where it was weighted with stones and thrown into a disused well.[3]

Symptoms.—If the windpipe is compressed so suddenly as to occlude the passage of air altogether, the individual is rendered powerless to call for assistance, becomes insensible and dies instantly. If the windpipe is not completely closed the face becomes cyanosed, bleeding occurs from the mouth, nostrils and ears, the hands are clenched and convulsions precede death. As in hanging, insensibility is very rapid, and death is quite painless.

Causes of Death.—Death is usually due to asphyxia, but it may be due to other causes as in hanging, *viz.*, cerebral congestion or apoplexy, asphyxia and apoplexy combined, or shock. Very rarely, the cervical vertebræ may be fractured.

Treatment.—This is hardly necessary as most cases are homicidal, but in case of necessity the constriction of the neck should be removed, and artificial respiration should be at once started. This may be assisted by the application of ammonia to the nostrils, and galvanism or inhalation of oxygen. Venesection should also be resorted to, if necessary. Hot bottles may be applied to the body if it is cold. If no serious injury has occurred to the neck, the prognosis is favourable provided this treatment is adopted within five minutes. After recovery the patient may die from any of the secondary complications, such as convulsions, paralysis, lesions of the larynx and lungs, or from abscesses.

Post-Mortem Appearances.—These are external and internal.

External Appearances.—The external appearances are those due to the constricting force applied to the neck, and those due to asphyxia.

Appearances on the Neck.—These vary according to the means used.

1. *All. High Court, Criminal Appeal No.* 197 *of* 1923.
2. *K. E.* v. *Mansa, Chief Court of Oudh, Crim. App. No.* 461 *of* 1927.
3. *The Times of India, March* 6, 1935.

1. **Ligature-Mark.**—This is a well defined and slightly depressed mark corresponding roughly to the breadth of the ligature, usually situated low down in the neck below the thyroid cartilage, and encircling the neck horizontally and completely. The marks are multiple if the

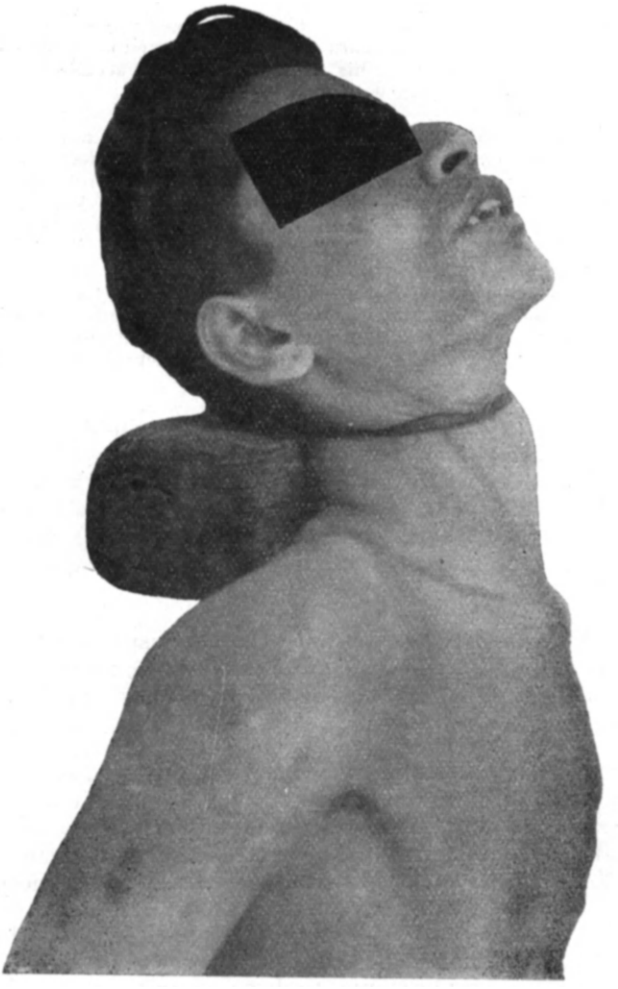


Fig. 37.—Strangulation : Ligature mark on the neck.
(*From a photograph kindly lent by Dr. H. S. Mehta*).

ligature is twisted several times round the neck. The mark may be oblique as in hanging, if the victim has been dragged by a cord after he has been strangled in a recumbent posture, or if the victim was sitting and the assailant applied a ligature on the neck while standing behind him, thus using the force backward and upward. The base of the mark, which is known as a groove or furrow, is usually pale with reddish and ecchymosed margins. It becomes dry, hard and parchment-like several hours after death, if the skin has been excoriated. Very often there are

abrasions and ecchymoses in the skin adjacent to the mark. In some cases the mark in the neck may not be present at all, or may be very slight, if the ligature used is soft and yielding, and if it is removed soon after death.

A Mahomedan boy, aged 15 or 16 years, was strangled to death by means of a loin cloth (*dhoti*) tied round the neck on the 19th December, 1925. At the post-mortem examination held on the next day at 12 noon I found no ligature mark round the neck, but found an effusion of blood in the soft tissues along the front of the trachea, the mucous membrane of which was congested and covered with froth.

2. If the fingers are used (throttling), marks of pressure by the thumb and fingers are usually found on either side of the windpipe. The thumb mark is ordinarily higher and wider on one side of the front of the neck, and the finger marks are situated on its other side obliquely downwards and outwards, and one below the other, but are, sometimes, found clustered together, so that they cannot be distinguished separately. These marks look like soft, red bruises, if examined soon after death, but they look brown, dry and parchment-like some time after death. The position of these marks may definitely indicate whether the left or

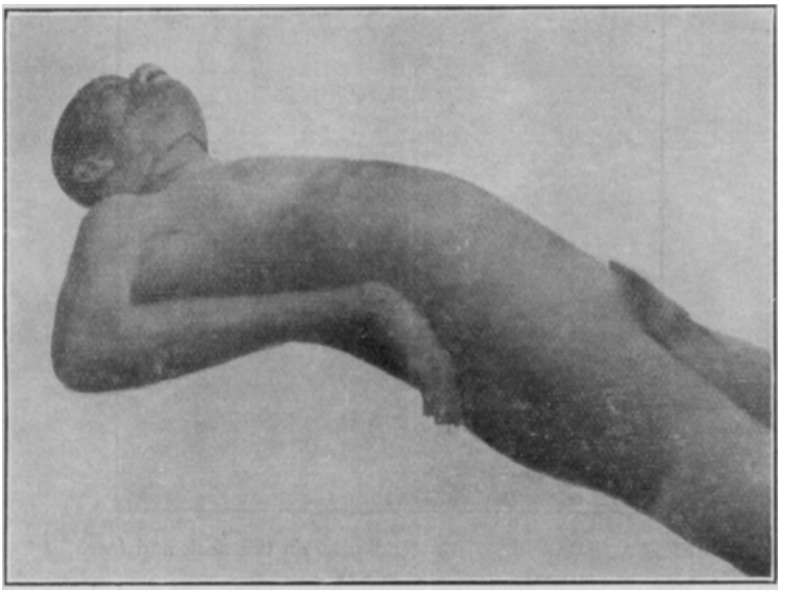


Fig. 38.—Strangulation: Ligature marks on the neck.

right hand was used, as also the size of the hand. Crescentic marks produced by the finger-nails are occasionally present if the finger tips are pressed deeply into the soft tissues of the neck. When both hands are used to grasp and compress the throat, the thumb mark of one hand and the finger marks of the other hand are usually found on either side of the throat. Sometimes, both thumb marks are found on one side and several finger marks on the opposite side. If the throat is compressed between

two hands, one being applied to the front and the other to the back, bruises and abrasions may be found on the front of the neck, as well as on its back.

Besides these marks, there may be abrasions and bruises on the mouth, nose, cheeks, forehead, lower jaw or any other part of the body, if there has been a struggle. Similarly, fractures of the ribs and injuries to the thoracic and abdominal organs may be present, if the assailant kneels on the chest or abdomen of his victim while pressing his throat.

In the case of throttling of one Hindu, Din Dayal, aged about 40, of Police Station Mohanlalganj, on or about the 18th day of December, 1926, I found at the autopsy blood issuing from the mouth and nostrils, eight bruises and abrasions on both the sides of the thyroid cartilage, and two contused wounds, three lacerated wounds and thirty bruises on various parts of the body, especially the face, head and chest. The second and third ribs on the left side, the second to the tenth ribs

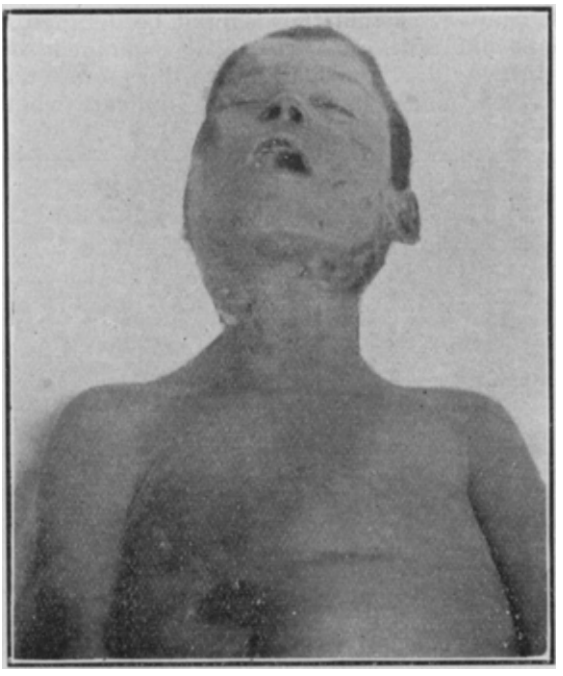


Fig. 39.—Throttling: Finger marks on the neck and face.

on the right side, and the sternum were fractured. The third phalanx of the left middle finger was also fractured. There were three lacerations across the front of the left lung below its apex, and four contusions at the root of the right lung. There was a contusion of the right auricle in front, and a contusion of the right ventricle in the middle on its front. There was a laceration of the aorta at its commencement from the heart. There were five lacerations on the right lobe of the liver, which was, otherwise, normal.

3. If a stick or a foot is used, there is a bruise in the centre of the front of the neck, generally across the windpipe, corresponding in width to the substance used. There will be a similar mark on the nape of the neck if two sticks are used. In such a case severe local injury will be evident.

Appearances due to Asphyxia.—The face is swollen and cyanosed, and marked with petechiæ. The eyes are prominent and open. In some cases they may be closed. The conjunctivæ are congested, and the pupils are dilated. The lips are blue. Bloody foam escapes from the mouth and nostrils, and sometimes pure blood issues from the mouth, nose and ears, especially if great violence has been used. The tongue is often swollen, protruding and dark in colour, and occasionally bitten by the teeth. The hands are usually clenched. The genital organs may be congested and there may be discharges of urine, fæces and seminal fluid.

Interal Appearances.—There is an extravasation of blood into the subcutaneous tissues under the ligature mark or finger marks, as well as in the adjacent muscles of the neck. Sometimes, there is laceration of the sheath of the carotid arteries, as also of their internal coats with an effusion of blood into their walls. The cornua of the hyoid bone may be fractured, but the fracture of the cervical vertebræ is extremely rare.

A Hindu male, aged 40 years, resident of Budayun District, was murdered by violent pressure on the neck and chest. Among several injuries inflicted on the body there was an extensive bruising of the larynx and trachea with fracture of the right cornu of the hyoid bone.[1]

The hyoid bone was also fractured in the case of a boy, 5 years old, who was strangulated with a piece of cloth tied round the neck with two knots in it for the sake of gold and silver ornaments.[2]

In a case of strangulation, which occurred on the 16th September, 1915, I found a fracture-dislocation of the first and second cervical vertebræ together with the usual cord-mark. In addition to these injuries, the right humerus, the left femur, and the first and second ribs of both sides were fractured. The fracture-dislocation of the spine was either caused by forcibly twisting the neck during the act of strangulation, or by a violent blow with a blunt weapon across the nape of the neck.

In another case in which a man was murdered by pressure of his neck with a stout *lathi* the hyoid bone was fractured and the first and second cervical vertebræ were fractured and dislocated.[3]

The larynx and trachea are congested, and contain frothy mucus. The cartilages of the larynx or the rings of the trachea may be fractured when considerable force is used.

It should be noted here that the hyoid bone is not, as a rule, fractured by any other means than by strangulation, although the larynx and trachea may, in rare cases, be fractured by a fall. Jungmichel[4] reports the case of a labourer who fell from a roof and sustained a longitudinal fracture of the thyroid cartilage, a fracture of the left ramus of the mandible and a compound fracture of the left humerus. Chatterji[5] relates the case of a boy, aged 10, who fell from a chair, striking his neck against the back of the chair, and sustained a small rupture at the posterolateral aspect of the trachea on the left side at its junction with the cricoid cartilage.

The lungs are usually congested, showing the appearance of red hepatization, and exuding dark fluid blood on section. They may show

1. *K. E.* v. *Premsing and Mohansing, All. High Court, Cr. App. No.* 796 *of* 1923.
2. *K. E.* v. *Shiamlal, Ibid., No.* 175 *of* 1923.
3. *All. High Court, Criminal Appeal No.* 281 *of* 1923.
4. *Medizinische Klinik, Feb.* 8, 1929, *p.* 219; *Jour. Amer. Med. Assoc., May* 4, 1929, *p.* 1565.
5. *Calcutta Med. Jour., Sept.,* 1925, *p.* 66.

emphysematous patches on their surface due to the rupture of the air-vesicles. The bronchial tubes usually contain frothy, bloody mucus. The right side of the heart is full of dark fluid blood, and the left empty. The right ventricle is found contracted and empty like the left, if the heart has continued to beat after the stoppage of respiration. Sometimes, both the cavities are found full if the heart stopped during diastole. The abdominal organs are darkly congested. The brain is also congested.

Fig. 40.—Throttling: Effusion of blood in the epiglottis, larynx and soft tissues.

Medico-Legal Questions.—The questions that are raised in a Court of law in connection with strangulation are—

1. Whether death was caused by strangulation.

2. Whether the strangulation was suicidal, homicidal or accidental.

1. **Whether Death was caused by Strangulation.**—No inference should be drawn simply from a ligature mark, for it may be indistinct or absent if a soft ligature like silk is used, and may be produced by the application of a ligature to the neck even after death. A similar mark may be produced by a collar or neckband worn loosely round the neck when it compresses the tissues which are swollen and distended by putrefaction.

The natural folds of the skin especially of a stout person rarely produce marks on the neck which may look like those found after strangulation.

Abrasions and finger marks may be produced on the neck by a person gasping for air in an intoxicated condition or in an epileptic or hysterical fit.

In 1859, a man was dead in a prostitute's house at Mymensingh. There were three marks on the neck produced by finger-nails and it was thought by the Civil Surgeon who held the post-mortem examination that he died of strangulation. But during the trial it was proved that the deceased was subject to epilepsy and the marks were probably produced by the deceased himself.[1]

To come to the conclusion that death was due to strangulation it is necessary, therefore, to note the effects of violence in the underlying tissues in addition to the ligature mark or bruise marks caused by the fingers or

1. *Chevers, Med. Juris., Ed. III, p.* 580.

by the foot, knee, etc., and other appearances of death from asphyxia. At the same time the possibility of other causes of death should be excluded.

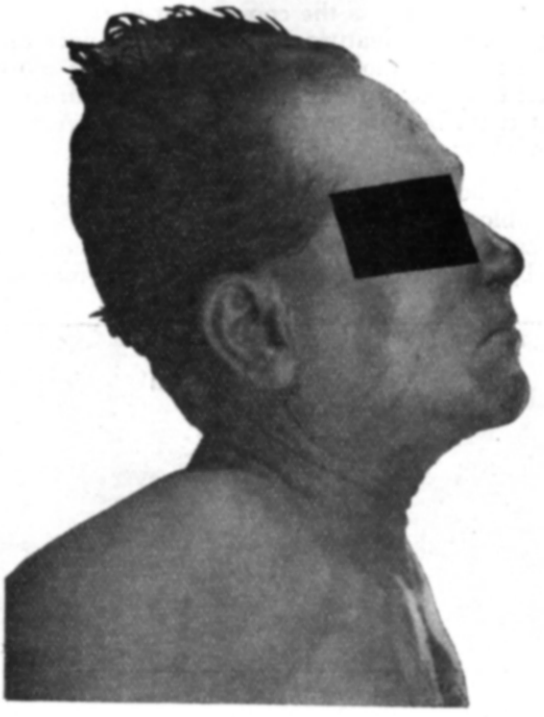


Fig. 41.—Natural folds of the skin simulating ligature marks on the neck. (*From a photograph kindly lent by Dr. H. S. Mehta*).

2. **Whether the Strangulation was Suicidal, Homicidal or Accidental.** Suicidal strangulation is not very common, though sometimes met with. In these cases some contrivance is always made to keep the ligature tight after insensibility supervenes. This is done by twisting a cord several times round the neck and then tying a knot, which is usually single and in front or at the side or back of the neck, by twisting the cord tightly by means of a stick, stone or some other solid material, or by tightening the ends of the cord by tying them to the hands or feet or to a peg in a wall or to the leg of a bed. In such cases injuries to the deep structures of the neck and marks of violence on other parts of the body are, as a rule, absent.

It is not possible for any one to continue a firm grasp of the throat after unconsciousness supervenes; hence throttling by the fingers cannot possibly be suicidal, although Binner[1] records the case of a woman, aged 40, who committed suicide by throttling. She was suffering from melancholia, and was found dead, crouched in her bed with both hands compressing her throat; the elbows were supported on the knees, and the

1. *Zeitschr. f. Med-Beamte,* 1888, *i., pp.* 364-368; *Dixonmann, Forensic Med. and Toxic., Ed. VI, p.* 175.

back leaned against the wall; there were marks of her finger-nails on both sides of the throat.

Homicidal strangulation is the commonest of the three forms. Usually there is a single turn of a ligature round the neck with one or more knots. Sometimes there may be more turns, in which case more ligature marks will be found on the neck. In addition to the ligature mark or the finger marks there is a probability of evidence of a struggle, and marks of violence on the other parts of the body.

A person may be first rendered helpless by being bound, or rendered unconscious by blows on the head or by intoxicating drugs, and then strangled by a small amount of compression. In some cases strangulation and suffocation by closure of the mouth and nostrils may both be attempted.

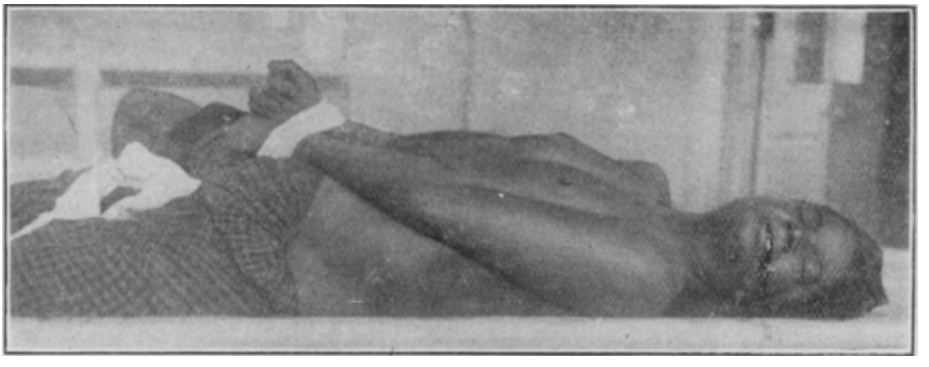


Fig. 42.—Throttling: The woman was murdered by throttling after her hands and feet were tied together.

A case came under my observation in which one Mt. Dulari, aged 30 years, of Police Station, Mohanlalganj, was murdered by throttling on the night of the 25th February, 1928, after her hands and feet were tied together with a piece of white cloth.

In the case of *King-Emperor* v. *Dhani Ram*,[1] charged under Section 302, I.P.C., with the murder of Ganeshi it was brought out in evidence that Dhani Ram, accused, took hold of a rough piece of wood, about 3½" long and 2" or more in diameter, and struck Ganeshi with it. Ganeshi fell down. He then put the wood across the neck of Ganeshi and stood on it until he died of strangulation.

Mt. Sukhdevi and Angun[2] were convicted under Section 302, I.P.C., of the offence of having committed murder by causing the death of Pancham, the husband of Mt. Sukhdevi. At the trial Angun confessed that on the night of the 20th May, 1921, he went to the house of Mt. Sukhdevi, with whom he was carrying on an intrigue when he found Pancham lying drunk in the courtyard. They took him into the room (*kothri*) and Mt. Sukhdevi sat on his chest and held both his hands while he pressed the throat with a brick.

Sometimes, a ligature is passed round the neck and over the body, and then tied to the hands and feet to simulate suicidal strangulation. In such cases the manner of tying should be examined carefully to see if it could have been done by a suicide. A ligature is occasionally placed

1. *Chief Court of Oudh, Crim. App. No.* 650 *of* 1927.
2. *Oudh Jud. Commissioner's Court, Crim. App. No.* 414 *of* 1921.

round the neck after throttling to simulate suicide, but on removal of the ligature finger marks on the throat accompanied by injuries to the deeper structures will be visible, thus suggesting murder.

Lastly, it must be borne in mind that strangulation may be committed without any noise or disturbance; even if other persons are in close vicinity, they may not be aware of the act.

An old woman was strangled in her shop by an apprentice in so short a time and with such facility that her husband, who was only separated from her by a small partition, heard no noise or disturbance during the act.[1]

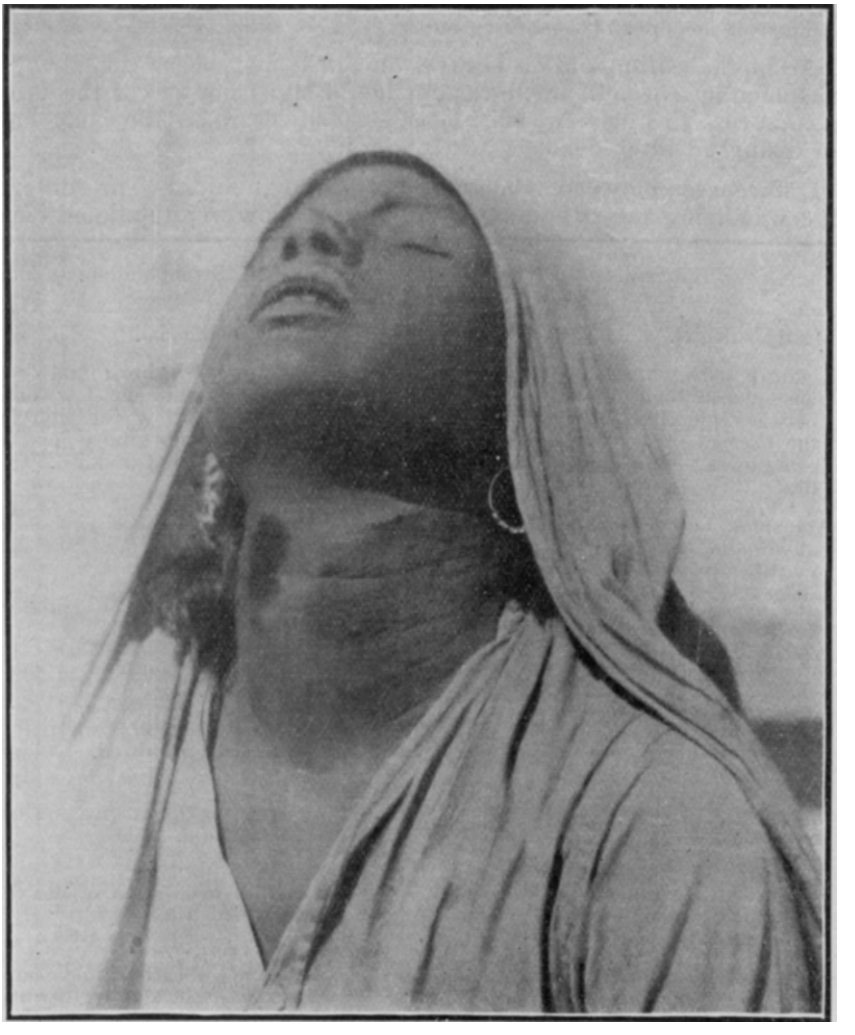


Fig. 43.—Feigned strangulation caused by the application to the neck of the juice of a marking nut.

1. *Ann. d' Hyg.*, 1859, 1, *p.* 157; *Taylor, Princ. and Pract. of Med. Juris., Ed. IX, Vol. I, p.* 646.

Homicidal strangulation is, sometimes, feigned by an individual, who wishes to bring a false charge against his enemy, or wishes to exculpate himself after he has embezzled some money entrusted to his charge. Hysterical women, sometimes, feign it without any obvious motive whatsoever.

On or about the 9th November, 1926, Mt. Masuman reported to the Sub-Divisional Magistrate of Mohanlalganj, District Lucknow, that her husband had attempted to murder her by strangulation. On examination I found bruise-like marks on both sides of the front of the neck. The skin round about these marks was inflamed and covered with tiny blisters. She had similar marks on the palms and tips of the fingers. On further examination these were found to have been caused by the application of the juice of a marking nut.

Accidental strangulation is rare, though it may occur if a string used in suspending a weight on the back should slip from across the forehead and compress the neck. This is easy to conjecture, if the body has not been disturbed after death.

Differences between Hanging and Strangulation.—The differences between hanging and strangulation are given below in a tabulated form :—

Hanging.	Strangulation.
1. Mostly suicidal.	1. Mostly homicidal.
2. Ligature mark, oblique, noncontinuous, placed high up in the neck between the chin and the larynx, the base of the groove or furrow being hard, yellow and parchment-like.	2. Ligature mark, horizontal or transverse, continuous, low down in the neck below the thyroid, the base of the groove or furrow being soft and reddish.
3. Abrasions and ecchymoses round about the edges of the ligature mark, rare.	3. Abrasions and ecchymoses round about the edges of the ligature mark, common.
4. Subcutaneous tissues under the mark, white, hard, and glistening.	4. Subcutaneous tissues under the mark, ecchymosed.
5. Injury to the muscles of the neck, rare.	5. Injury to the muscles of the neck, common.
6. Carotid arteries, internal coats ruptured in violent cases of a long drop.	6. Carotid arteries, internal coats ordinarily ruptured.
7. Fracture of the larynx and trachea, very rare and that too in judicial hanging.	7. Fracture of the larynx and trachea, often found.
8. Fracture-dislocation of the cervical vertebræ, common in judicial hanging.	8. Fracture-dislocation of the cervical vertebræ, rare.
9. Scratches, abrasions and bruises on the face, neck and other parts of the body, usually not present.	9. Scratches, abrasions and bruises on the face, neck and other parts of the body, usually present.
10. Face, usually pale and no petechiæ.	10. Face, congested, livid and marked with petechiæ.
11. Neck, stretched and elongated in fresh bodies.	11. Neck, not so.

Hanging.	Strangulation.
12. External signs of asphyxia, usually not well marked.	12. External signs of asphyxia, very well marked.
13. Bleeding from the nose, mouth and ears, very rare.	13. Bleeding from the nose, mouth and ears, may be found.
14. Saliva, running out of the mouth down on the chin and chest.	14. Saliva, no such running.
15. Emphysematous patches on the surface of the lungs, not present.	15. Emphysematous patches on the surface of the lungs, may be present.

ILLUSTRATIVE CASES

Suicide.—1. A middle aged woman was brought into the Hotel-Dieu, labouring under insanity. Soon after admission she strangled herself by passing a handkerchief twice round the neck and then tying it in front. The woman had lost four fingers of her right-hand from an early period, and yet she managed to tie the kerchief round the neck with great firmness and dexterity.—*Ann. d' Hyg.*, 1833, 2, *p.* 153; *Taylor, Princ. and Pract. of Med. Juris., Ed. IX, Vol. I, p.* 643.

2. A Hindu lunatic in a jail strangled himself by passing two or three coils of stout thread around his neck, attaching the ends securely to his wrists and then extending his arms to the utmost limit. This occurred during ten minutes when his attendant was absent.—*Surgeon-General Francis, Med. Times and Gaz., Dec.* 2, 1876, *p.* 634.

3. A woman in Madras nearly succeeded in strangling herself by means of her long hair.—*Chevers, Med. Juris., Ed. III, p.* 589.

Homicide.—I quote the following cases from my note-book:—

1. On October 14, 1912, a Hindu boy, aged 13 years, resident of Agra, was murdered for the sake of his gold ear-rings and a *dhoti* was tied round his neck. The autopsy revealed finger marks on both sides of the windpipe with ecchymosis of blood in the soft tissues and signs of asphyxia. In this case the Sub-Inspector of Police propounded a very queer theory that the image of the murderer would have been impressed on the lenses, and he requested that the eyeballs should be preserved. I need not say that he never came to trace the culprit by examining the eyes even though they had been preserved for six months.

2. In December, 1917, Mt. Munia, aged 60 years, was said "to have been dead of compression of the throat with pincers." On examination two bruises were seen on the front of the neck with an extravasation of blood underneath in the soft tissues, *viz.*, one, 1" × ½", across the right side of the neck 2" to the right of the middle line, and the other, ½"×½", across the left side of the neck 2" below the angle of the left lower jaw.

3. On November 17, 1918, the body of a Hindu male, 30 years old, was found tied up in a steel box lying near the Iron Bridge in Lucknow. At the post-mortem examination the face was found flushed, the eyes were closed and congested, and the lips were blue. A transverse, brownish, hard and parchment-like ligature mark, ½" broad, was found encircling the neck and passing over the windpipe. There were abrasions about the margins of the mark. A similar mark, 5" × ½", was detected across the left side of the neck, above the first mark and a third mark, 2" × ½", was seen 1" below the second mark. The larynx and trachea were congested. The lungs were congested and exuded dark, fluid blood on section. The left chamber of the heart was empty and the right was full of dark fluid blood. The body was identified to be that of a Brahmin of Hardoi by the head that had been preserved and four persons were prosecuted for having killed him by strangulation.

4. On the 11th December, 1918, the body of Sarju, 50 years old, was forwarded to the King George's Medical College Mortuary with a report from the Station Officer of Police Station, Goshainganj, that he was murdered by dacoits. At the post-mortem examination I found bruises on both sides of the windpipe and fractures of the rings of the trachea in its upper part. There was an extravasation of blood in the muscles of the neck in front. Blood was also found issuing from the mouth and nostrils.

5. A Hindu widow, 70 years old, was seen sitting at her door between 8 a.m. and 9 a.m. on July 25, 1921. At noon she was found dead in her house with a twisted *moonj* cord, 82 inches long, tied round the neck. At the autopsy no cord mark was seen on the neck, but two finger marks, each ½" × ½", were found on each side of the windpipe with an extravasation of blood in the underlying tissues. The lining membrane of the windpipe was congested with hæmorrhagic points and was covered with frothy mucus. The left second and third ribs were fractured. I gave the opinion that the deceased died of asphyxia due to throttling, and the cord was tied round the neck probably to simulate suicide.

6. On the 7th September, 1921, the body of a Hindu girl, aged 13 years, was found covered with mud and a *dhoti* tied round the neck. The post-mortem examination revealed a fracture of the right parietal and temporal bones and a soft depressed ligature mark, ½" broad, encircling the neck in its middle with extravasated blood in the underlying soft tissues, especially the front of the windpipe, which was found congested. There were also small bruises about the angles of the mouth.

7. On the 19th December, 1924, the body of Mt. Kailasha, 50 years old, of Police Station, Goshainganj, was found strangled with a *dhoti* (loin cloth) round the neck. On examination two incised wounds were found on the face. There were several bruises on both sides of the throat with an extravasation of blood in the soft tissues. It appeared that she was struck with a sharp cutting weapon, and thrown down. She was then throttled, and the cloth was tied round the neck afterwards.

8. On the night of the 19th March, 1926, Swami Kundana Nand Rishi, about 40 years old, was found murdered by compression of the throat by means of a heavy iron *belcha*, 25" long and 3" to 4" in diameter. At the autopsy an incised wound, 1½" by ½", was found obliquely across the inferior jaw below the chin and exposing the bone. There were three bruises, varying from 3" to 4" by ½" to 1½", across the upper part of the middle of the neck in front. There were some bruises on the face. The thyroid cartilage and the upper two rings of the trachea were fractured. There was also an effusion of clotted blood in the muscles of both sides of the neck in front.

9. At 11 a.m. on the 21st April, 1926, I held a post-mortem examination on the body of Mt. Maharania, aged 40 years, and resident of Police Station, Malihabad. Blood was issuing from the mouth and nostrils. There were small bruises with crescentic scratches on both sides of the throat. Three upper rings of the trachea were fractured.

10. On the morning of the 25th July, 1927, the body of Mt. Sitala, aged 70 to 75 years, was found in her house by the *Mukhya* of her village in Police Station, Goshainganj, and it was believed that "the deceased was strangled in the night by some one to take away her belongings." On examination on the next morning a bruise, 1½" by ¼", was found along the front of the neck 1" to the right of the thyroid cartilage, and four bruises, varying from ¼" to 1" by ¼" to ½", with two crescentic nail-marks were found obliquely across the left side of the neck 1½" below the angle of the left inferior jaw and situated one below the other. The thyroid cartilage was fractured, and the left cornu of the hyoid bone was dislocated.

11. On the 8th January, 1932, I held a post-mortem examination on the body of Mst. Patari Devi, aged 25 years, which was found lying in a park within the jurisdiction of Police Station, Hazrat Ganj. The face was flushed. In addition to a reddish, transverse ligature mark, ½" broad, encircling the neck over Adam's apple, the following external injuries were found on the body :—

1. A bruise, ¼" × ¼", over the lower margin of the left cheek bone.
2. 2 bruises, one ¼"×¼", and the other, ½"×1/4", along the left mandible.
3. A crescentic bruise, ¼"×1/6", across the right cheek.

4. An abrasion, $\frac{3}{4}$"$\times\frac{1}{4}$", along the outer side of the right thigh in its upper part.

5. 3 abrasions, varying from $\frac{1}{4}$" to $\frac{1}{2}$"$\times\frac{1}{4}$" to $\frac{1}{2}$", over the back of the left elbow joint.

6. An abrasion, 1"$\times\frac{1}{4}$", across the upper part of the left shoulderblade.

7. An abrasion, 1"$\times\frac{1}{4}$", across the middle of the right buttock.

There was an effusion of clotted blood in the soft tissues of the neck under the skin. There was an ecchymosis in the submucous coat of the larynx. There was bloody froth in the larynx, trachea and bronchi, the mucous membrane of which was deep red and injected. The lungs were bulky, deep red and congested with venous engorgement. The womb was enlarged and contained a fœtus of about three months of intra-uterine life. A corpus luteum was visible in the right ovary.

A case is recorded in which one Malla, under sudden and grave provocation, killed his wife by strangulation effected by twisting her long hair round her throat. —*Criminal Law Jour., July,* 1924, *p.* 519.

Accident.—1. An ingenious young man having nearly lost the use of his arms used to move a heavy weight by a cord passed round his neck.. One morning, soon after he went to his room, his sister found him sitting in a chair quite dead with the cord twisted round his neck. The deceased must have tried to move the weight in the usual way, but it had slipped behind and so strangled him.—*Gordon Smith quoted by Guy and Ferrier, Forensic Med., Ed. VI, p.* 261.

2. In July, 1839, Elizabeth Kenchan, an extremely dissipated woman, went to bed intoxicated, with her bonnet on, and in the morning was found strangled in its strings. She had fallen out of bed, her bonnet became fixed between the bedstead and the wall, and she, being too drunk to loosen the strings, was strangled.—*Ibid., p.* 262.

3. A girl was accustomed to carry fish in a basket on her back, supported by a leathern strap passing round the front of her neck above her shoulders. One day she was found dead, sitting on a stone wall; the basket had slipped off while she was resting, and had thus raised the strap which firmly compressed the windpipe.—*Taylor, Princ. and Pract. of Med. Juris., Vol. I, Ed. IX, p.* 640.

SUFFOCATION

Definition.—The term, suffocation, is applied to that form of death which results from the exclusion of air from the lungs by means other than compression of the neck.

Causes.—The causes of suffocation are—

1. Smothering or closure of the mouth and nostrils.
2. Obstruction of the air-passages from within.
3. Pressure on the chest.
4. Inhalation of irrespirable gases.

1. **Smothering or Closure of the Mouth and Nostrils.**—Infants are often accidentally smothered by being overlaid by their mothers when they are drunk. This is more common among the lower classes of women in England. In India, such cases are rare, as infants are generally not allowed to sleep in the same bed with their mothers, but are placed in separate cradles; however, they are, sometimes, smothered by inexperienced *girl mothers* who press them too closely to the breast when suckling. A common method of killing infants and children is to close the mouth and nostrils by means of the hand, bed clothes, soft pillows or mud.

Cases have been recorded of adults being accidentally smothered by plaster of Paris at the time of taking a cast or mould, or by falling face downwards into vomited matter, flour, cement, sand or mud, especially when drunk or during an epileptic fit.

2. **Obstruction of the Air-passages from within.**—This may be due to—

(*a*) The presence of foreign bodies, such as a piece of meat, potato skin, fruit-stone, corn, button, coin, cork, rag, India-rubber teat, live fish, round-worm, loose artificial teeth, mud, cotton, leaves, etc.

It is not necessary that a foreign body should be of such a size as to block the air passages completely. Even a small object blocking the lumen partially may cause death by spasm.

On the 26th September, 1912, a Mahomedan girl, about 10 years old, was standing in a street in Agra with her infant sister, one year old, in her arms, when a boy playfully gave her a push from behind. The infant girl fell and died immediately. On post-mortem examination a split gram (*chaneki dal*) was found to be obstructing the lumen of the larynx. It appears that the infant girl had a parched gram in her mouth at the time of the fall, and it got into the larynx during the involuntary inspiratory movement.

On the 10th April, 1929, a Hindu male, aged 60 to 65 years, died all of a sudden while trying to hire an *ekka* for proceeding to his house. At the post-mortem examination I found a carious tooth lying in the glottis, and death was caused by suffocation due to spasm of the glottis brought on by the tooth lying there.

Vomited matter may regurgitate into the larynx and, by inspiratory efforts, be aspirated into the smaller bronchi and cause suffocation. This is especially common in acute alcoholism, and occasionally occurs during a fit of epilepsy or in a case of badly administered anæsthesia. It must be remembered that the contents of the stomach fall into the larynx and trachea after death owing to pressure of the gases of decomposition, but they cannot reach the smaller bronchi.

(*b*) Diseases, such as tumours pressing on some portion of the air-passages, or a false membrane as in diphtheria, or œdema of the glottis, or effusion of blood from hæmoptysis, epistaxis and wounds of the throat, or of pus from an abscess in the tonsils or caseating glands ulcerating into the trachea. A foreign body in the pharynx or œsophagus may cause obstruction pressing on the windpipe from behind.

3. **Pressure on the Chest.**—This may occur accidentally through the chest being pressed violently in crowds at big fairs, or by being trampled upon in the rush of such crowds. Pressure on the chest may also occur in railway, motor-car or other vehicular accidents or by burial under the debris of a falling wall or roof. Cases of compression of the chest homicidally are also met with in India.

4. **Inhalation of Irrespirable Gases.**—Inhalation of gases, such as carbon dioxide, carbon monoxide, hydrogen sulphide, or smoke from a burning house, will produce suffocation.

Mode of Death.—Usually death is due to asphyxia, but it may be due to shock, when the heart stops by reflex action through the vagus nerves.

Fatal Period.—Death occurs on an average from four to five minutes after complete withdrawal of air from the lungs, although cases have occurred in which death was almost instantaneous when the windpipe was blocked by a foreign body. Recovery may occur if treated within four minutes.

Post-mortem Appearances.—These appearances are external and internal.

External Appearances.—These may be due to the cause producing suffocation, or to asphyxia.

Appearances due to the Cause producing Suffocation.—In homicidal smothering effected by the forcible application of the hand over the mouth and nostrils, bruises and abrasions are often found on the lips and angles of the mouth, and alongside the nostrils. The inner surface of the lips may be found lacerated from pressure on the teeth. The nose may be flattened, and its septum may be fractured from pressure of the hand, but these signs are, in my experience, very rare. There may be bruises and abrasions on the cheeks and malar regions, or on the lower jaw, if there has been a struggle. Rarely, fracture or dislocation of cervical vertebræ may occur, if the neck has been forcibly wrenched in the attempt at smothering with the hand. No local signs of violence will be found, if a soft cloth or pillow has been used to block the mouth and nostrils.

In compression of the chest, external signs of injury may not be present, but the ribs are usually fractured on both sides. In homicidal compression of the chest brought about by the hands or knees of a murderer or by some other hard material, bruises and abrasions, symmetrical on both sides, are usually found on the skin together with an extravasation of blood in the subcutaneous tissues. The ribs are also fractured symmetrically on both sides, and the sternum is fractured, though rarely.

Appearances due to Asphyxia.—The face may be pale or suffused. The eyes are open, the eyeballs are prominent, and the conjunctivæ are congested. The lips are livid, and the tongue is, sometimes, protruded. Bloody froth comes out of the mouth and nostrils. The skin shows punctiform ecchymoses with lividity of the limbs. Repture of the tympanum may occur from a violent effort at respiration.

Internal Appearances.—Mud or any other foreign matter may be found in the mouth, throat, larynx or trachea when suffocation has been caused by the impaction of a foreign substance in the air-passages. It may also be found in the pharynx or œsophagus. The mucous membrane of the trachea is usually bright red, covered with bloody froth and congested. The lungs are congested and emphysematous. They may be lacerated or contused even without any fracture of the ribs, if death has been caused by pressure on the chest. Punctiform sub-pleural ecchymoses (Tardieu's spots) are usually present at the root, base, and lower margins of the lungs, and are characteristic of death by suffocation, though they may be present in asphyxial deaths from other causes. They are also found on the thymus, pericardium, and along the roots of the coronary vessels. The lungs may be found quite normal if death

has occurred rapidly. The right side of the heart is often full of dark fluid blood, and the left empty. The blood does not readily coagulate; hence wounds caused after death may bleed. The brain is generally congested, and so are the abdominal organs, especially the liver, spleen and kidneys.

Medico-Legal Questions.—These are—

1. Whether death was caused by suffocation.

2. Whether the suffocation was suicidal, homicidal or accidental.

1. **Whether Death was caused by Suffocation.**—Sometimes, it is not easy to state whether death is due to suffocation, inasmuch as the signs of asphyxia may be altogether absent, or these signs may be present in deaths from epilepsy, tetanus, or strychnine poisoning. To come to a definite conclusion it is, therefore, very essential to look for evidences of violence in the shape of external marks surrounding the mouth and nostrils, or on the chest, or the presence of foreign bodies in the throat. Again it cannot be positively affirmed from the presence of Tardieu's spots that death occurred from suffocation, since they are found in deaths from apoplexy, heart disease, pneumonia, etc.

Circumstantial evidence should always be taken into consideration to establish the proof of death from suffocation.

2. **Whether the Suffocation was Suicidal, Homicidal or Accidental.**—Suicidal suffocation is very rare, though a few cases of suicide among prisoners and insane persons have been recorded. They are said to have stuffed their throats with rags, pieces of blanket, hay, etc.

Homicidal suffocation is common, and is usually resorted to in murdering infants and children by forcing mud, etc., down their throats, or by smothering them by the hands, clothes, etc. In adults it is only possible if the victim is weak and feeble, or is unable to resist, having been previously stupefied by intoxicating or narcotic drugs. Homicidal suffocation by pressure on the chest is, sometimes, resorted to in India, but in the case of adults it is often combined with smothering or throttling, and it is usually an act of more than one person. "Burking" is a method of suffocation adopted by Burke and his associate, Hare, for killing their victims. They used to throw their victims down on the ground, and kneeling on the chest, used to close the mouth and nostrils with one hand, and firmly hold the lower and upper jaws together with the other hand, thus effectually blocking the air-passages.

A form of homicidal suffocation practised in Northern India is known as "Bans-dola", although it is not so common now as it used to be formerly. In this form the victim's chest is squeezed so forcibly between two strong wooden planks or bamboos, one being placed across the upper part of the chest and the other across the back of the shoulders, that the respiratory act is interfered with, the muscles are lacerated and the ribs are fractured. If the force applied is very severe, the lungs may be crushed and lacerated.

Burying alive used to be resorted to in India as a form of punishment, and lepers used to be, sometimes, buried alive.

In the case of infants dying under suspicious circumstances and afterwards exhumed, a question may arise as to whether they had been buried alive. The presence of fine dust in the œsophagus and stomach is a convincing proof of the infant having been buried alive. In a burial after death fine dust may be found in the upper air-passages, but not in the œsophagus or stomach.

Accidental suffocation is frequent and is produced as described above.

ILLUSTRATIVE CASES

Suicide.—1. An epileptic was found in bed lying on his back, with a round pebble in each nostril and a rolled up strip of flannel stuffed in the throat.—*Sankey, Brit. Med. Jour.*, 1883, *Vol. I, p.* 88.

2. A maniac committed suicide by forcing a roll of flannel down his throat.—*Macleod, Ibid.*, 1882, *Vol. II, p.* 1246.

3. A middle aged man was brought to the workhouse by the police with a history of delusions. On examination, no definite signs of insanity were found, but it was thought desirable to keep him under observation. On the evening of the same day he suddenly became excited and violent and was, therefore, placed in a padded room. After a short time he quieted down and went off to sleep. At 9-20 p.m. he was found dead. Upon examination the body was found lying on its back with the arms outstretched. The face was placid, and no marked cyanosis was present; there was, however, lividity of the lips, ears and finger and toe nails. Inside the mouth a piece of flannel, about 1" by 1", was found and behind this there were two similar strips. The last of these two was so firmly packed down over the epiglottis that it was withdrawn with some difficulty. He obtained these strips by tearing up his blanket and had evidently suffocated himself by packing them down his throat.—*J. Mill Renton, Brit. Med. Jour., Feb.* 29, 1908, *p.* 493.

Homicide.—The following are a few of the cases of homicidal suffocation that came under my observation:—

1. A boy, 5 years old, was murdered at Hardoi by his mouth being stuffed with leaves.

2. Dacoits killed a young *Vaish* at Agra by sitting on his chest and penetrating his lungs at several places by sharp iron nails.

3. A Hindu boy, about 14 years old, was murdered by closure of the mouth and nostrils in a village of Police Station, Mohanlalganj of District Lucknow. On examination of the body at noon on the 31st January, 1919—48 hours after death—bruises were found across the tip of the nose and the lips. The larynx and trachea were congested and contained froth. The lungs were congested with sub-pleural ecchymosis.

4. A Hindu girl, 10 years old, was found dead in her house on the morning of March 26, 1919. On examination it was found that she died of asphyxia due to suffocation caused by the blocking of the mouth and nostrils and by pressure on the windpipe and chest. The eyes were closed but congested. Frothy liquid was issuing from the nostrils. The lips were blue and several small bruises were seen about the angles of the mouth, on both sides of the windpipe and on the right side of the upper part of the chest. There was an extravasation of blood in the soft tissues of the neck and on the right side of the chest under the bruises. The lining membrane of the larynx and trachea was congested and contained fine froth. The lungs were congested with patches of sub-pleural ecchymosis and exuded dark fluid blood on section.

5. The body of Kalka Chaukidar of a village in Police Station, Banthra, was removed from a well and forwarded to the college mortuary. At the post-mortem examination held on the 14th April, 1919—about 60 hours after death—several small bruises were found on the face and on both sides of the throat. There were patches of extravasated blood in the soft tissues on both sides of the chest and the sternum was fractured transversely in its middle. The third, fourth, fifth, sixth and

seventh ribs were fractured on both sides. Both the lungs were congested and lacerated at several places. Opinion: The deceased died of suffocation caused by pressure of the chest producing fractures of the ribs and lacerations of the lungs.

6. The body of a Hindu girl, 10 years old, was found buried in sandy mud on the bank of a canal at 1 p.m. on the 3rd September, 1923. The post-mortem examination was held at 11 a.m. on the 4th September, 1923. The eyes were found congested. The lips were livid. A frothy muddy liquid was issuing from the nostrils, which contained sandy mud. A thick coat of similar mud was found on the tongue, especially at its base and the pharynx. The œsophagus was full of sandy mud, and its lining membrane was congested. Sandy mud was also present at the cardiac end of the stomach mixed with food which consisted of undigested maize and rice weighing about half a pound. The larynx and trachea were red in colour and contained froth and sandy mud as deep as the upper and lower bronchus of the left lung and the terminal bronchioles of the right lung. The lungs were œdematous, and turgid with blood with patches of sub-pleural ecchymosis. Both sides of the heart contained blood and the pulmonary arteries were congested. The abdominal organs were congested. There was an extravasation of blood in an area of 2"×2" on the under surface of the scalp corresponding to the upper part of the left side of the forehead and a similar extravasation in an area of 2" × 1" on the left side of the back of the skull below the top. The brain and its membranes were congested. There was no injury to the vagina. The hymen was intact. The cause of death was asphyxia due to suffocation by the deceased having been buried alive. It appeared that the girl had been thrown down and held firmly by pressure on the left forehead while she was being buried.

7. On the 22nd December, 1923, the body of a Mahomedan boy, aged 16 years, was found lying on the roof of a bungalow situated on Mall Road, Lucknow, five or six days after death. On examination the following external injuries were found on the body:—

1. A bruise, ¼" by ¼", on the left nostril.

2. A bruise, ¾" by ¼", across the left lower lip.

3. A bruise, ½" by ¼", on the left upper lip.

4. A semi-circular and curved bruise, 3" by ½", obliquely along the face extending between the left malar bone and the left angle of the mouth.

5. Six bruises, varying from ¼" to ½" by ¼" to ½", on the left elbow.

6. Two small abrasions on the right knee.

The inside of the mouth and the lips were livid and congested. The lungs were congested, and exuded dark frothy blood. There were patches of sub-pleural ecchymosis on the pericardium and lungs. Death occurred from suffocation by closure of the mouth and nostrils.

Accident.—1. The body of a Hindu male, 30 years old, was brought to the Agra Medical School Mortuary on the 21st September, 1910, with a police report that it was not known whether the deceased died from snake bite or had committed suicide. At the post-mortem examination it was found that the man died of asphyxia by suffocation due to food accidentally getting into the larynx and trachea.

2. A Mahomedan male, aged 35, was found dead in a street of Agra on the 23rd October, 1918. Upon examination no external mark of injury was found on the body. The larynx and trachea were injected and particles of a whitish substance were found adherent to the mucous membrane. The lungs were engorged with dark blood and the finer divisions of the bronchi contained a whitish substance (not muco-pus) similar to that found in the trachea. The stomach contained 14 ounces of whitish material similar to that found in the trachea and lungs. Death was due to suffocation brought about by particles of food accidentally getting aspirated into the trachea and bronchioles. The viscera were preserved but no poison was detected on analysis. There was a history of intoxication.—*Agra Med.-Leg. Post-mortem Rep., No.* 53.

DROWNING

Definition.—Drowning is a form of death in which the atmospheric air is prevented from entering the lungs by submersion of the body under water or other fluid medium. It is not necessary that there should be complete submersion. Death is sure to occur, even if the face alone is submerged so that air is prevented from entering the respiratory orifices.

Stages of Drowning.—When an individual in perfect possession of his senses falls into water, he sinks to a depth proportional to the momentum obtained during the fall, but immediately rises to the surface owing to the struggling movements of his limbs, though the specific gravity of the body is slightly higher than that of water. If he is not a swimmer, he cries and shouts for help, when, his mouth being at the level of the water surface, he draws water into the stomach and lungs. The water in the lungs excites coughing and, during violent expiratory efforts due to cough, a certain amount of air is expelled from the lungs, and its place is taken up by water which is drawn into the lungs. The weight of the body increases and he sinks. He rises again to the surface by the involuntary movements of his limbs, and draws more water into the lungs in an effort to keep above water, and consequently goes to the bottom. This alternate rising and sinking goes on longer than the proverbial three times, until all the air has been expelled from the lungs and its place has been taken up by water. He then becomes insensible and sinks to the bottom to die. Sometimes, convulsions precede death.

When an individual is rendered unconscious by shock or syncope at the time of immersion, he goes to the bottom, and may rise once to a certain height but usually sinks without a struggle. In such a case little water enters the respiratory tract.

Symptoms.—The subjective symptoms felt by a drowning person are auditory and visual hallucinations, and return to memory of past events, which had already been forgotten. In some cases there is mental confusion.

Mode of Death—Asphyxia.—This is a common cause in the majority of cases, as water getting into the lungs gets churned up with air and mucus, and produces a fine froth which blocks the air vesicles.

Shock.—This is brought about by fright or terror, or it may be caused during a fall, the water striking against the chest and pit of the stomach. Again, if water is very cold, it may induce shock through the recurrent laryngeal or trigeminal nerves, which reflexly inhibit the action of the heart and lungs. Shock may also be induced through the cutaneous nerves.

Concussion.—This may occur by falling into water on the head or buttocks from a height and striking against some hard solid substance or even against the water itself.

Syncope.—This may occur in persons suffering from heart disease or epilepsy by falling suddenly into cold water.

Apoplexy.—Cerebral vessels, especially if they happen to be diseased, may be ruptured by a sudden rush of blood to the brain from cold, excitement, or the first violent struggles to keep above the surface of the water.

Exhaustion.—This results from continued efforts to keep above the surface of the water.

Injuries.—Fracture of the skull bones and fracture-dislocation of the cervical vertebræ may result, if a man falling from a height into shallow water or into a narrow deep *pucca* well strikes his head forcibly against some hard solid substance.

In August, 1918, a Mahomedan girl, 2½ years old, fractured her right parietal and temporal bones by falling accidentally into a well.

On January 10, 1923, a Hindu male, while jumping into a well with a view to commit suicide, struck his head against a wooden board fixed in the well about the surface of water. The autopsy revealed three contused wounds on the head and rupture of the left middle meningeal artery.

Fatal Period.—Asphyxia supervenes within two minutes after complete submersion, and the heart stops in two to five minutes afterwards. It has been found from observations that even expert divers cannot hold their breath under water for two minutes continuously. It is, however, recorded that Miss E. Wallenda remained submerged in a tank at the Alhambra Music Hall for four minutes, forty-five seconds and a half. Death is almost sure after complete submersion for five minutes, unless water was prevented from entering the lungs on account of shock or syncope caused at the time of the fall. Such cases are possible of resuscitation even after an immersion of ten to twenty minutes.

Treatment.—In the case of persons rescued from drowning, an attempt at resuscitation should be made without delaying for a moment, and should be continued for at least an hour, unless it is certain that death has occurred.

The chief object in the treatment of an apparently drowned person should be the removal of water from the lungs and the introduction of air instead. This can be best accomplished by stripping the person naked to the waist, freeing the mouth and nostrils from mud or sand and froth, turning the body with the head lowered downwards so as to allow the water to drain from the upper respiratory passages and then starting artificial respiration. This should be supplemented by the administration of carbogen (a mixture of 95% of oxygen and 5% of carbon dioxide), by friction to promote heat of the body, by alternate splashes of hot and cold water to the face and chest, and by hypodermic injections of strychnine, atropine sulphate, adrenaline chloride or coramine.

There are five methods of artificial respiration; *viz.*, (1) Schafer's method (prone posture), (2) Sylvester's method, (3) Howard's method, (4) Marshall Hall's method, and (5) Laborde's method. But Schafer's method is the best and simplest to perform, and has been recommended by the authorities of the Humanitarian Society of England.

It is carried out in the following manner:—

Lay the patient, face downwards, on the ground, with one arm stretched straight forward and the other bent under the head which is turned to one side. Place a folded piece of clothing under the lower part of the chest. Kneel either astride or by the side of the patient about the level of the hips, facing his head. Place the palms of your hands flat

over the back on the lowest ribs, one on each side, and gradually throw the weight of your body forward, so as to produce firm pressure on the patient's chest. By this means, the air, and water if any, are driven out of the patient's lungs. Immediately thereafter raise your body slowly so as to remove the pressure, leaving the palms of the hands in position. Repeat this forward and backward movement from twelve to fifteen times per minute and continue it for at least an hour or until the natural respirations are restored.

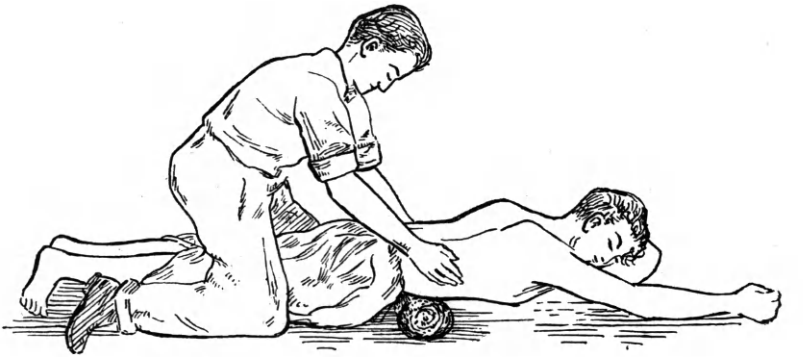


Fig. 44.—Schafer's Method of Artificial Respiration. Expiration.

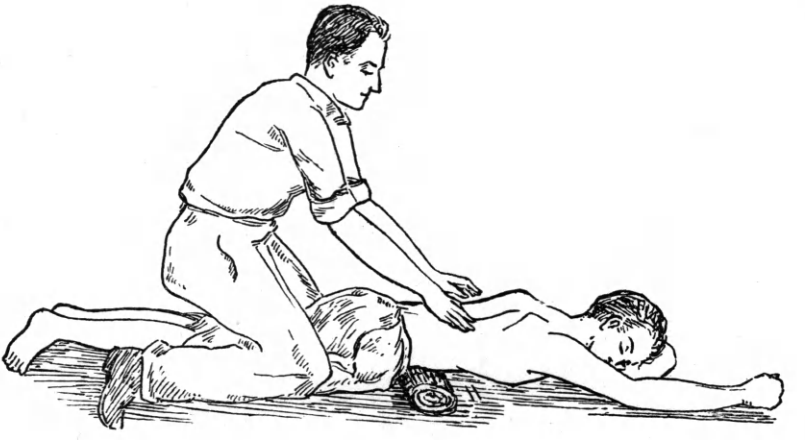


Fig. 45.—Schafer's Method of Artificial Respiration. Inspiration.

After respiration has been established, the patient should be covered with warm blankets, put to bed with hot-water bottles at the sides, and given a little brandy in warm water or warm milk, if he can swallow it. The patient should be carefully watched for some time as after recovery, death may occur from exhaustion or pneumonia.

Post-mortem Appearances.—These are the signs of asphyxia unless death occurred from shock, syncope or concussion supervening immediately upon submersion. The appearances are external and internal.

External Appearances.—The clothes on the body are wet unless examined a long time after removal from the water. The face is pale,

the eyes are half open or closed, the conjunctivæ are congested, and the pupils are dilated. The tongue is swollen and sometimes protruded.

A fine, white, lathery froth or foam, rarely tinged with blood, is seen at the mouth and nostrils. If not visible, it may appear on compression of the chest. This is regarded as a diagnostic sign of drowning. Froth of almost a similar nature is seen in cases of opium poisoning and in deaths occurring from slow asphyxia. It also appears after putrefaction has set in. But the froth in all these cases is not of such a lasting nature and of such a large quantity as in drowning, and the bubbles are also much larger.

The granular and puckered appearance of the skin, known as *cutis anserina, goose-skin* or *goose-flesh,* is found on the anterior surface of the body, particularly the extremities, especially during winter when water is cold. It is caused by the contraction of the muscle fibres, termed *arrectores pilorum* connected with the hair follicles, and is considered as an important sign showing that drowning had occurred during life, but it may occur on submersion of the body in cold water immediately after death, while the muscles were still warm and irritable. It is not confined to drowning alone as, being the result of nervous shock, it is found in other violent and accidental deaths. It may also develop after death as a result

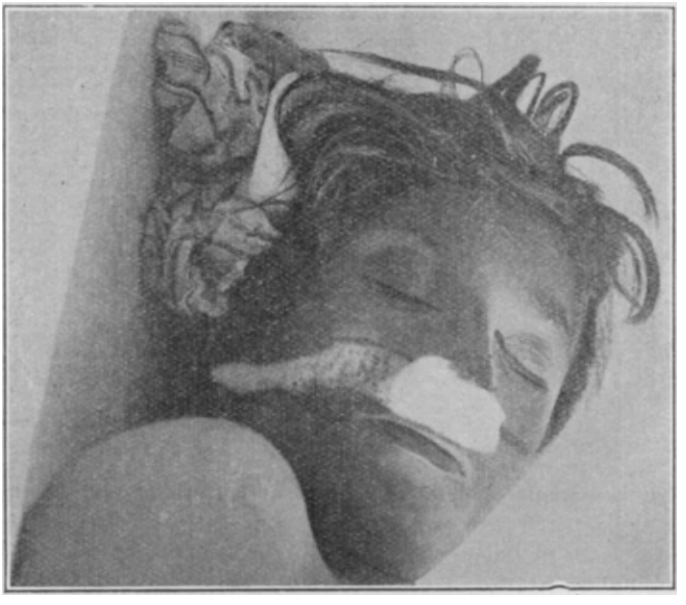


Fig. 46.—Drowning: Froth at the nostrils.

of rigor mortis of the muscle fibres of the *arrectores pilorum,* especially during winter. Moreover, *cutis anserina* is rarely seen in India as the water, being usually warm, does not produce the contraction of the muscle fibres of the *arrectores pilorum.* Of 110 cases of drowning examined by me during a period extending over eight years I found marked *cutis anserina* only in 4 cases.

The penis and scrotum are found retracted and contracted, especially during winter, when water is cold. Casper[1] lays great stress on this point as a sign suggestive of death from drowning, but the penis may, sometimes, be found semi-erect, and the retraction and contraction of the genital organs may be encountered, if the body is thrown into the water after death. The reverse condition of the genitals is usually found if the body is taken out early, and if the water is not cold ; hence in a tropical country like India no reliance can be placed on this sign.

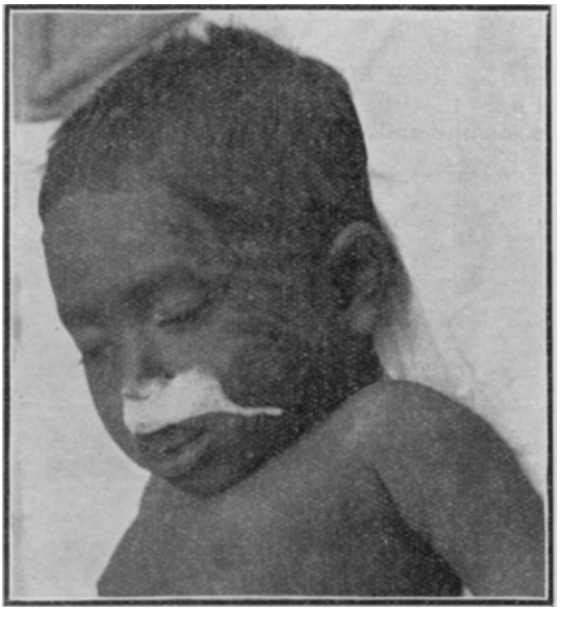


Fig. 47.—Drowning : Froth at the nostrils coming out on compressing the chest.

Grass, gravel, sticks, weeds, twigs or leaves may be found firmly grasped in the hands as the result of cadaveric spasm. The presence of

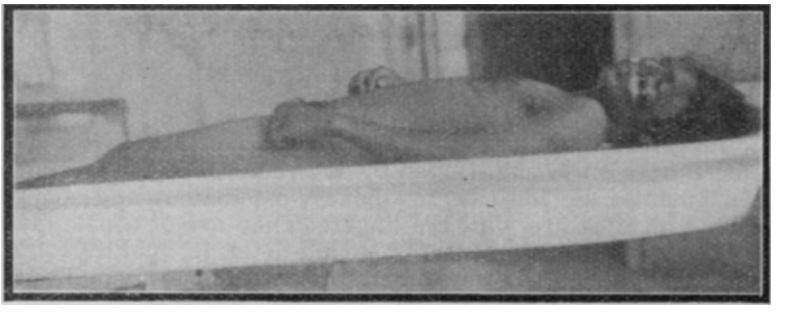


Fig. 48.—Opium Poisoning : Froth at the nostrils.

1. *Forensic Medicine, Eng. Trans., Vol. II, p.* 236.

this sign is indicative of death from drowning as it shows the struggle of the victim for his life. Mud or sand may be found under the finger-nails, and abrasions may, sometimes, be found on the fingers and toes.

On or about the 9th March, 1926, a Hindu male, aged 60 years, was found drowned with shoes firmly grasped in his hands in the Kokrel Nala at Lucknow. It appears that he tried to ford the Nala with the shoes in his hands, but was drowned on going into deeper water. The post-mortem examination revealed the presence of water in the stomach and duodenum. The lungs were found bulky, œdematous and congested, and on section frothy liquid blood poured out.

The skin of the hands and feet shows a bleached, corrugated and sodden appearance, after the body has lain in water for ten to twelve or more hours. This condition of the skin is known as the *washerwoman's hand.* It proves only that the body has remained in water for some time without reference to the cause of death.

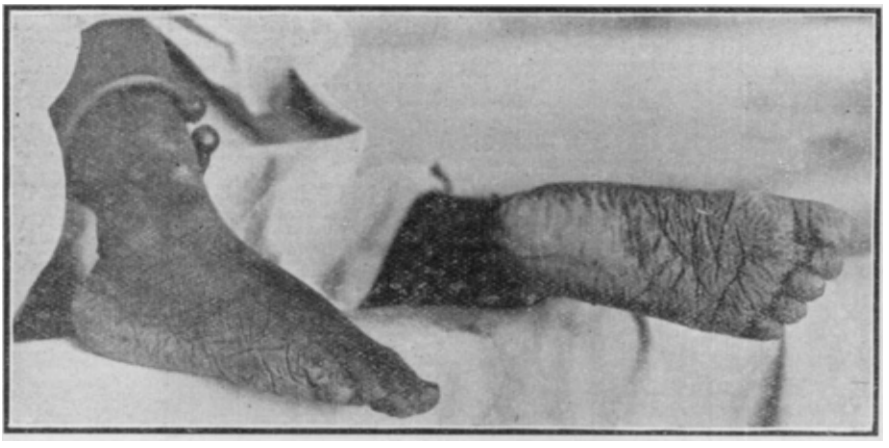


Fig. 49.—Corrugated skin of the feet in a body removed from a well.

Post-mortem lividity is most marked in the head, neck and chest, since blood gravitates to these places, which are usually the most dependent parts, when a body is immersed in water.

Rigor mortis appears early, especially when a violent struggle has taken place before death.

Internal Appearances.—The brain is hyperæmic, and the vessels of its membranes are injected.

The lungs are distended like balloons, overlap the heart and protrude out of the thorax on laying it open by the removal of the sternum. They are often indented by the ribs, are heavy, œdematous and spongy to the feel, and pit on pressure with the finger. They are of a pale grey colour with reddish stains, and on section exude a large quantity of a forthy, bloodstained fluid. Minute, punctiform sub-pleural and pericardial ecchymoses noted in other forms of asphyxia are seldom found in cases of drowning.

The larynx, trachea and bronchial tubes usually contain a fine, white froth, and may contain some foreign matter, such as sand, mud or

fragments of aquatic plants. Their mucous membrane is usually red and congested. Froth appears within two minutes of submersion, and its quantity varies according to the length of submersion and the violent respiratory efforts. Particles of food may be found regurgitated in the air-passages owing to the vomiting set up by the imbibition of water especially if the stomach was full at the time of drowning.

During putrefaction the watery fluid from the lungs may transude into the pleural cavities.

The heart presents the appearance usually found in death from asphyxia ; the left side is usually empty, the right side is full, and the large veins are gorged with blood which is dark in colour and unusually fluid owing to admixture of water.

Alexander C. Gettler[1] suggests the determination of the chloride content in the blood of the right and left chambers of the heart as a specific test for drowning. This test is based on the fact that the water enters into the lungs during drowning, and dilutes the chloride content of the blood in the heart by osmosis. Normally the chloride content of the right and left chambers of the heart is almost the same, the greatest difference being 5 mg. in 100 c.c. of blood. In cases of drowning the difference is always much more than 5 mg., ranging from 19 to 294 mg. This difference depends on the amount of water going into the lungs, and also on the time interval between the entrance of water into the lungs and death. The longer this time interval, the more water passes to the left chamber of the heart. In cases where drowning occurs in salt-water, the left chamber of the heart shows a higher chloride content and in cases where drowning occurs in fresh water the left chamber shows a lower chloride content. It must be remembered that persons who die of shock immediately after submersion in water may not show this difference in the chloride content. It must also be borne in mind that water cannot get into the left side of the heart if the body is thrown into the water after death.

The presence in the stomach of a certain quantity of water is regarded as an important sign of death, particularly if the water possesses the same characteristics as that in which the body was found immersed, and contains sand, mud, algæ, weeds, fine shells, etc. It is almost impossible for water to get into the stomach, if a body is submerged after death. In rare cases it is possible that the water found in the stomach may have been drunk by the deceased immediately before submersion occurred. On the other hand, water may not be present in the stomach, if the person died from syncope or shock, or became unconscious immediately after falling into water, so that he could not struggle and swallow water in the act of drowning. There will also be no water in the stomach, if the body has undergone putrefaction, for water, even if it is present in the stomach, is forced out by the pressure of the gases of decomposition. In Northern India, it is not usual to find water in the stomach, as the bodies taken out of water are brought to the Sadar mortuary in a more or less decomposed condition. In my experience at Agra and Lucknow I have found water in the stomach in about 30 per cent of cases of drowning.

1. *Jour. Amer. Med. Assoc., Nov.* 19, 1921, *p.* 1650.

The small intestine, especially the duodenum and jejunum and rarely the ileum, contain water mixed with mud, sand, etc. This sign is regarded as positive evidence of death by drowning, as the passage of water into the intestine is only due to the peristaltic movement, which is a vital act. But water is not always present in the intestine. I have found it in about 20 per cent of cases.

The liver is usually gorged with dark fluid blood, which pours out from the cut surfaces, on an incision being made into its substance. The spleen and kidneys are dark in colour and deeply congested.

Water may be found in the middle ear on aspiration by a small pipette. It is possible for a small quantity of water to be forced into this cavity, especially in the violence and confusion of inspiration and swallowing in the process of drowning, when the mouth is full of water. It is impossible for water to force its way into the middle ear after death; hence its presence is regarded as an important sign of drowning.

Medico-Legal Questions.—These are—

1. Whether death was caused by drowning.
2. Whether the drowning was suicidal, homicidal or accidental.

1. **Whether Death was caused by Drowning.**—In Northern India, it is a common custom to throw dead bodies into running streams, and the fact of finding a dead body in water does not, therefore, lead one to presume that death was caused by drowning. Again, victims are often murdered or poisoned first, and then their bodies are thrown into water to avoid the detection of crime. In my experience I have found that 21 per cent out of the bodies removed from water were such as were thrown into a well, pond, lake, canal or river after the deceased had been destroyed by wounds inflicted on the head or neck or by strangulation, suffocation, poisoning, etc. It is, therefore, very essential to examine the body carefully for the evidence of external and internal injuries—ante-mortem or post-mortem—and for the signs of poisoning.

After excluding these possibilities, an opinion as to the cause of death from drowning should be given from a number of the following characteristic signs:—

1. The presence of a fine, white, lathery froth about the mouth and nostrils.
2. The presence of some object firmly grasped in the hands.
3. The presence of a fine, white froth in the air-passages.
4. The bulky and œdematous condition of the lungs which exude a copious, frothy, blood-stained fluid on section.
5. The presence of water, mud, weeds, etc., in the stomach or small intestine, or both.
6. The presence of water in the middle ear.

It must be remembered that these typical signs of drowning are seen only in the body of a drowned person when it is removed from water within a few hours after death and examined immediately. They are modified by the lapse of time and disappear altogether when the body is

recovered from water after putrefaction is well advanced. Moreover, putrefactive changes in the body advance so rapidly after its removal from water that a short delay in holding a post-mortem examination even of a recent case of drowning is likely to obliterate the signs to a great extent. In doubtful cases, where a definite opinion cannot be given, it is safer to preserve the viscera for chemical analysis.

2. **Whether the Drowning was Suicidal, Homicidal or Accidental.**—In India, drowning occupies the first position of all the modes of committing suicide. In two hundred and thirty-one cases investigated by me during a period extending over eight years suicide was effected in the following modes:—Drowning, 90 (34 males, 56 females) ; Opium, 73 (55 males, 18 females) ; Irritant poison, especially arsenic, 30 (17 males, 13 females) ; Hanging, 28 (13 males, 15 females) ; Cut throat, 6 (5 males, 1 female) ; Gunshot, 2 (1 male, 1 female) ; Burns, 2 females.

Females, even on the least provocation, commit suicide by jumping into a well or tank in the neighbourhood of their house or village. Sometimes, a woman falls into the water with her child. If she survives and the child dies, she is tried under Section 309, I.P.C., for the offence of having committed an attempt at suicide, and may be tried under Section 302, I.P.C., for having committed the murder of her child or under Section 304-A, I.P.C., for causing the death of her child by negligence.

A case occurred at Agra where a woman jumped into a well with a view to commit suicide together with her daughter, 3 years old. She was saved, but her daughter died. During the trial the woman put up a very ingenius plea of defence that while playing in the vicinity of the well her daughter climbed up its parapet and fell down accidentally; to rescue her, she at once plunged into the well, but could not save her daughter. The plea was, however, not accepted by the Court, as the parapet was too high for the child to climb, and the woman was sentenced to death under section 302, I.P.C.

A woman, who was harassed and ill-treated by her husband, in a fit of disappointment and annoyance jumped into a well with the object of drowning herself. At the time when she jumped into the well she had her child tied at her back but she was not conscious of the fact and the result was that although she escaped the child died. It was held that the woman was guilty of attempting to commit suicide, that it could not be said that her act came within the purview of clause 4 of section 300 of the Indian Penal Code, and that, therefore, she was guilty of a negligent omission, that is the omission to put the child down before jumping into the well and that the death of the child having been caused by such negligence, the accused was guilty of the offence under section 304-A., I.P.C.[1]

Sometimes, suicides tie their hands or feet together or attach heavy weights to their person, before jumping into water. Dr. Godfrey Carter[2] records the case of a man who bound himself with a rope in a way that completely inhibited the movement of his arms and legs and then threw himself in a canal a few miles from his house. In such cases it would be necessary to determine whether the knots or folds of the rope or ligature were such as could have been made by the suicide himself. For a murderer often ties the hands and feet of his victim before he throws him into water.

1. *K. E.* v. *Supadi, Bombay High Court, Cr. Appeal No.* 14 *of* 1925 ; *Crim. Law Jour., Aug.* 2, 1925, *p.* 1016.
2. *Brit. Med. Jour., Aug.* 13, 1932, *p.* 321.

Injuries are generally absent, but they may be found on the body coming accidentally into violent contact with a hard substance during a fall. Rarely, suicides inflict severe wounds upon themselves either by a cutting weapon or by a firearm before they take the fatal jump into the water.

Homicidal drowning is rare except in the case of infants and children. It is a common practice to rob children of their ornaments and then throw them into a well or tank. It is not possible to throw an adult of average strength and in full possession of his senses into water so as to drown him unless he is attacked unawares or he has been previously stupefied by some narcotic drug. In the case of *Rex* v. *George Joseph Smith* (popularly known as " the brides of the bath "), Joseph Smith was convicted in 1915 of the offence of having committed the murder of three women by drowning them in a bath a few days after he had married each. During the trial it was proved, from the experiments carried out by Inspector Neil in full as well as empty baths, that it was possible to submerge an unsuspecting person all of a sudden into a bath and keep the head under water for five or ten minutes for death to occur without producing any injury on the head or body. Under the circumstances the person would be unable to offer any resistance, as unconsciousness would supervene immediately by sudden submersion, when water would rush up the nose.

Accidental drowning is not uncommon in India. It occurs occasionally among swimmers due to their rashness in swimming, but it occurs mostly in non-swimmers who venture to go beyond their depth in the sea, rivers, canals, lakes, etc. It also occurs among persons at bathing places while bathing in deep water. In such cases the body is usually naked with only a loin cloth, having no weight attached to it. Females may fall accidentally into a well while drawing water from it. It must be remembered that before jumping into water with a view to commit suicide an Indian woman generally tucks up the lower ends of her garments and passing them between her legs fastens them at the loin so that the garments may not be dishevelled and expose nakedness after death when the body comes up to the surface.

Children may also accidentally fall into ponds or lakes while playing near their banks. They may even fall accidentally into domestic vessels of water, such as buckets, wash tubs, etc.

Accidental drowning in shallow water is very rare except when the individual happens to be intoxicated, insane or epileptic.

ILLUSTRATIVE CASES

Suicide in Shallow Water.—A male inmate in a lunatic asylum in the neighbourhood of Glasgow committed suicide by submerging his head in a sink filled with water. The Medical Superintendent found the upper part of his chest resting against the front of the sink, the head sunk just under the level of the brim of the basin and the legs bent under the body ; but the knees were not touching the floor.—*Glaister, Med. Juris. and Toxic., Ed. VI, p.* 156.

Homicidal Drowning—Weeds in the Air Passages.—The body of a child was discovered in a tank at a considerable distance from his own house. At the post-mortem examination the fauces, larynx and trachea contained small portions of green vegetable matter, and the right bronchus was almost completely filled with a large piece of an aquatic weed doubled together. It was afterwards proved distinctly

that no weed of this type grew in the tank where the body was found. Further inquiry led to the discovery that the boy's body had been found by a woman in a tank near his home, in which similar weeds grew abundantly. This woman had conveyed the corpse to the more distant tank which belonged to a person against whom she had a grudge.—*Chevers, Med. Juris., Ed. III, p.* 644.

Accidental Drowning in Shallow Water.—1. On October 31, 1920, a British soldier was found drowned in a shallow *nala* (ditch) near the second Rajput Lines, Lucknow Cantonment. The face was submerged but most of the body was above the surface of water. The deceased had been drinking and was on the verge of delirium tremens.

2. (*a*) One Patu, aged 20, who had been liable to epileptic fits, went to work in some muddy rice land on May 14, 1890. An hour later he was found dead, lying with his face downwards in a shallow pool. The water was so shallow that his mouth, nose and the right side of his face were immersed, the left eye and side of the face being above the surface. The rest of his body from the neck downwards was on dry ground. At the post-mortem examination the mouth, nasal cavities and air-passages contained mud and green water weeds.

(*b*) One Muzli, aged 26, an epileptic subject, was found dead, face downwards, in an almost dry drain on August 23, 1890. Water was two inches deep except at a depression where it was three inches and a half and where the head was lying. Upon dissection the air-passages contained sand and muddy water with a few blades of grass.—*Powell, Ind. Med. Gaz., Aug.*, 1897, *p.* 300.

CHAPTER VIII

DEATH FROM STARVATION, COLD AND HEAT

STARVATION

Starvation or inanition results from the deprivation of a regular and constant supply of food, which is necessary to keep up the nutrition of the body. Starvation is regarded as *acute* when the necessary food has been suddenly and completely withheld, and *chronic* when there is a gradual deficient supply of food.

Symptoms.—In the protracted absence of food the acute feeling of hunger lasts for the first thirty to forty-eight hours and is succeeded by pain in the epigastrium, relieved by pressure, and accompanied by intense thirst. After four or five days of starvation general emaciation and the absorption of fat begin. The eyes appear sunken and glistening, the pupils are widely dilated, and the bony projections of the face become prominent. The lips and tongue are dry and cracked, and the breath is foul and offensive. The voice becomes weak, faint and inaudible. The skin is dry, rough, wrinkled and boggy, emitting a peculiar, disagreeable odour. The pulse is usually weak and frequent, but sometimes becomes slow. The temperature is usually subnormal, the diurnal variation reaching 3.28° F. instead of 0.3° F. to 1° F. as in the normal body. The abdomen is sunken, and the extremities become thin and flaccid with loss of muscular power. There is at first constipation, the motion being dry and dark, but later towards death diarrhœa or dysentery generally supervenes. The urine is scanty, turbid and highly coloured. Loss of weight is most marked and constant. According to Chossat a loss of two-fifths or forty per cent of the body weight ordinarily ends in death.[1] The intellect remains clear till death though, in some cases, delusions and hallucinations of sight and hearing may be met with. Occasionally delirium and convulsions or coma precede death. It should be remembered that in cases where there is a gradual deprivation of food, death may occur after a prolonged period from some intercurrent disease.

Major-General Sir Patrick Hehir[2] describes the effects of the siege of Kut which lasted 148 days with food scarcity. Towards the end of this period the daily rations yielded 1110 to 1850 calories for one man each day. The average loss of body weight was from 10 to 14 per cent. The muscles decreased in size and about 80 per cent of the usual adipose tissue disappeared. The pulse rate was slow, it being 30 to 40 per minute among some people doing duty. The temperature was much below normal varying from 94° F. to 97.4° F. In some cases 90° F. was recorded before death. Choleraic diarrhœa and dysentery were widely spread. He believes that degenerative changes in the digestive organs ensue in prolonged starvation, leading to failure to assimilate food properly. Some of the officers took 18 months to 2 years to regain their normal weight and to eat and digest an ordinary diet.

Fatal Period.—Death occurs in ten to twelve days if both water and food have been totally deprived. Guy,[3] however, gives an account of a

1. *Witthaus, Med. Juris., Vol. I, p.* 981.
2. *Brit. Med. Jour., June* 3, 1922, *p.* 865.
3. *Forens. Med., Ed. VI, p.* 312.

shipwreck, where the crew lived various periods of from eleven to twenty-eight days without provisions or fresh water. If food alone is withdrawn life may be prolonged for a long period, say from six to eight weeks or even more since some Jain Sadhus are reported to have fasted for two to three months without taking anything but boiled water. It is reported that a Jain Muni[1] broke his fast after having fasted for nearly one hundred and sixty-seven days to bring about unity between two sections of Jain Sadhus at Jodhpur. Mayor Mc. Swiney abstained from food in Brixton prison for seventy-five days before he died, while Jatindra Nath Das, accused in the Lahore conspiracy case, died in Borstal jail after sixty-one days' hunger strike. This is, however, influenced by certain conditions, such as age, sex, condition of the body and its environments.

Age.—Children suffer most from want of food. Old people require less nourishment than young adults, and can, therefore, stand the deprivation of food better, but not for a longer period owing to the weakening of their vital functions.

Sex.—Females can withstand starvation for a longer period than males, as they have a relatively greater amount of adipose tissue in their bodies, and ordinarily consume less food.

Condition of the Body.—Fat stored up in the body is utilized as food for the maintenance of life during starvation. It is, therefore, natural that fatty, healthy people are likely to endure the withdrawal of food better and longer than thin, lean and weakly persons.

Environments of the Body.—The effects of starvation are not felt very much so long as the body temperature is maintained by suitable clothing. Exposure to cold tends to shorten the period of life. Exposure to excessive heat also accelerates the onset of death, if a sufficient quantity of water is not available. Starvation is well borne by those persons in whom the activity of their vital functions is lowered, as in the cataleptic. On the other hand, active physical exertion during starvation hastens death.

Treatment.—In persons suffering from prolonged starvation the digestive processes have become very feeble; hence caution should be observed in the administration of food. Solid food should not be given at once, as it is likely to set up an attack of serious indigestion and even death. It is advisable to give at first sips of hot water, and then to add gradually small quantities of milk. Feeds should consist of small quantities at a time, and should be repeated at frequent intervals. The simplest and most easily digestible liquid foods should be given, and solid foods should be added gradually and with care, when the stomach has regained the digestive power. Warmth of the body should be maintained by the application of hot water bottles, and by rubbing the surface gently with stimulating lotions. Diffusible stimulants may be given hypodermically or by the mouth.

Post-mortem Appearances—External.—The body is greatly emaciated, and emits a disagreeable, offensive odour. The eyes are dry, red and open, the eyeballs being sunken. The cheeks and temples are hollow. The tongue is dry and coated. The skin is dry and shrivelled, and is, some-

1. *Times of India, Aug.* 1, 1934.

times, excoriated or ulcerated. Bed-sores are often present. The muscles are pale, soft and wasted, and fat is almost completely absent in the subcutaneous and intracellular tissues, as well as in the omentum, mesentery and about the internal organs, although some fat may be present in cases where death has occurred rapidly from the sudden withdrawal of both food and water. It should also be remembered that the entire absence of fat throughout the body is never seen in wasting diseases, such as tuberculosis, diabetes, etc.

Internal.—The brain is usually normal, although it is, sometimes, pale and soft. The meningeal vessels are congested, and there is frequently some serious effusion in the ventricles. The heart is small in size, and the muscle is pale and flabby. The chambers are generally empty. The lungs are pale and collapsed, and exude very little blood, when cut. At times the lungs may be œdematous, and may show hypostatic congestion at the bases. The stomach is small, contracted and empty. It may contain undigested food, if it had been given to the deceased shortly before death in order to avert a suspicion of wilful starvation. The mucous membrane of the stomach and upper part of the small intestine is more or less stained with bile. The intestines are empty and contracted, but the lower portion of the large intestine may, sometimes, contain hard, scybalous fæcal matter, and may often present more or less evidence of inflammation. These hollow viscera show an extensive thinning and translucency of their walls indicating thereby that no food has passed the stomach for a considerable time. Sometimes, ulcerations are found in their walls; these are very likely due to irritation resulting from the injudicious ingestion of substances to appease hunger. The liver, spleen, kidneys and pancreas are small and shrunken. The gall bladder is usually much distended, and contains dark, inspissated bile. The urinary bladder is empty.

On the 23rd November, 1928, I examined the body of one Musammat Samita, aged about 20 years, who died on or about the 25th day after having received an injury in the neck which completely divided the larynx and pharynx, so that no food could pass down the throat. The body was thin and emaciated, and reduced almost to a skeleton. There was no fat about the breasts which had all atrophied, leaving only the dark nipples. Internal examination showed that the brain was normal and its membranes were congested. The lungs were congested at the bases, and the root of the right lung was pneumonic showing grey hepatisation. The chambers of the heart were empty. The stomach was corrugated and empty, weighing 4 ounces. Its mucous membrane was rough, corrugated and congested at places. The small intestine was shrunken and empty. The mucous membrane was pale except at the lower part where it was congested. The large intestine contained dry fæcal matter in its lower part. The liver was small, weighing 34 ounces. The spleen was shrunken and weighed 3 ounces. The kidneys were congested, each weighing 2 ounces. The gall bladder contained dried bile, and the urinary bladder was empty.

Medico-Legal Questions.—

1. Whether death was caused by starvation.
2. Whether the starvation was suicidal, homicidal or accidental.

1. **Whether Death was caused by Starvation.**—One must always bear in mind that there are certain pathological conditions, *viz.*, the malignant disease of the alimentary canal, progressive muscular atrophy, Addison's disease, diabetes mellitus and tuberculosis, which lead to

progressive wasting and emaciation of the body. It is, therefore, very necessary to examine carefully all the internal organs and to search for the existence of any of these diseases while holding a post-mortem examination, before one can give the opinion that death occurred from starvation. In the Penge murder case of 1877 in which Louis Staunton, Patrick Staunton, Mrs. Patrick Staunton and Alice Rhodes were sentenced to death for having killed by starvation one Harriet Staunton, aged 35, the wife of the first-named accused, an agitation was started later in the medical press that death was not due to starvation, but was due to tuberculosis, as at the post-mortem examination "a slight deposit of a tubercular substance" was found "on the membranes of the brain," and there was also "a tubercular deposit about 2 inches square at the apex of the left lung." It was also urged that the post-mortem examination had not been thorough, inasmuch as the urine had not been examined for the presence of sugar, nor had the œsophagus and suprarenal glands been examined. These criticisms led the Home Secretary to re-open the case. A free pardon was granted to Alice Rhodes and the sentence passed on the three Stauntons was commuted to penal servitude for life. Dr. Halliday Sutherland[1] reviewed this case before the Medico-Legal Society, London, at their meeting held on the 15th February, 1921, and proved from the medical evidence given during the trial that death was due to starvation.

2. **Whether the Starvation was Suicidal, Homicidal or Accidental.**— Suicidal starvation is rare, though it may be seen among lunatics or prisoners, who may go on "hunger strike." In this connection it must be remembered that the forcible feeding of prisoners, when they refuse to take any food on account of passive resistance, is not an assault but is quite lawful.

In India, sometimes, young hysterical women imagine that they are possessed by deities, and say that they can live without food for a prolonged period, or they do so to practise deception on their friends and relatives. When people watch them, the fraud is exposed, but in some cases they actually abstain from food, and prefer to die rather than that their imposture should be detected. Persons watching them must be very careful, as they are criminally responsible for abetting suicide if death results from this enforced fasting.

Bai Prembai, a Hindu woman of Bombay, who professed to live without food and to pass neither urine nor fæces, undertook to allow a watch to be kept upon her movements. A committee of medical men and one lady doctor undertook this duty and selected eight nurses to conduct the watch. After four days' watching a packet of food was found to have been concealed upon her person and she was thus exposed. —*Barry, Legal Med., Vol. II, Ed. II, p.* 244.

Homicidal starvation is met with in the case of old, helpless, or feeble-minded persons and children or infants. Illegitimate infants are, sometimes, done to death by depriving them of proper food, and at the same time exposing them to cold. Rarely mothers-in-law in the lower classes in India starve their little daughters-in-law to death. Two such cases came to my notice at Agra. Both were sisters and were married in the same house. They were seven and eleven years old respectively, were burnt at several places and were not given sufficient food, until they died

1. *The Transactions of the Medico-Legal Society, Vol. XV, p.* 54.

from inanition. The Bombay Children Act, 1924, as modified upto the 15th December, 1928, provides that whoever having the actual charge of, or control over, a child or young person abandons, exposes or wilfully neglects or ill-treats such child or young person in a manner likely to cause such child or young person unnecessary suffering or injury to his health shall be punishable with imprisonment of either description for a term which may extend to six months or with fine which may extend to two hundred rupees or with both. For the purpose of this section injury to health includes injury to, or loss of, sight or hearing and injury to limb or organ of the body and any mental derangement, and a parent or other person legally liable to maintain a child or young person shall be deemed to have neglected him in a manner likely to cause injury to his health if he wilfully fails to provide adequate food, clothing, medical aid or lodging for the child or young person (*Vide* Part III, Section 9). According to this Act a "child" means a person under 14 years, and a "young person" means a person who is 14 years of age or upwards but under the age of 16 years.

Accidental starvation may occur during famines, among shipwrecked seamen and persons entombed in mines or pits by falls of rock. It may also occur from obstruction to the passage of food into the stomach from disease, such as ankylosis of the jaws, stricture or cancer of the œsophagus or stomach, etc.

COLD

Children and old persons having little reserve of thermotaxic power are very susceptible to the bad effects of cold. Individuals, whose vitality has been lowered from faitgue, want of food, indulgence in alcoholic drinks and previous ill-health, are less able to withstand the effects of cold than healthy, well-nourished adults of temperate habits. Owing to a greater deposit of subcutaneous fat—a non-conducting material—women are likely to endure cold longer and better than men. Dry cold air is less harmful in its effects than moist cold air.

Symptoms—Local.—These appear on the skin in the form of erythematous patches called frost-bites (frost-erythems) and chilblains produced by constriction of the cutaneous vessels which deprives the tissues of their nourishment. The exposed parts, such as the ears, nose, fingers and toes, are usually affected. The condition of frost-bite being a vital action can never be produced after death.

General.—There are no bad effects from moderate cold. On the contrary, it invigorates the body, and produces appetite and hunger; but exposure to severe cold continued for a long time produces deleterious effects, especially if a person is not properly clothed to keep up the body heat, and does not get sufficient food or exercise. The skin becomes pale and numb; sometimes it assumes a dusky, reddish and livid hue, with the formation of vesicles. The muscles become so stiff, rigid and heavy, that the patient is unable to move or raise his limbs. This condition is followed by general lethargy, drowsiness and an inclination to sleep which, if not controlled, passes gradually into stupor, coma and ultimately death. Sometimes, convulsions, hallucinations and delirium occur before death.

In rare cases the temperature of the body has fallen to 75° F. without causing death.[1]

Cause of Death.—Death occurs from a lesser supply of oxygen to the nervous centres and tissues, as hæmoglobin is unable to part with it at a lower temperature.

Treatment.—This consists in covering the patient with woollen garments and placing him immediately in a warm bed. Hot water bottles should be applied to the surface, and the warmth of the body should be gradually restored by rubbing the limbs with flannel or hot towels. Hot coffee or tea and other stimulants, such as strychnine, digitalis and alcohol, should be administered. Enemata of warm normal saline are very beneficial. It may be necessary to treat nephritis and other inflammatory conditions, if they arise after the reaction has set in.

Post-mortem Appearances—External.—The surface of the body is usually pale, marked with irregular, dusky red patches of frost-erythems, especially on the exposed parts, such as the tips of the fingers and toes, nose, lips and ears. These do not appear on the dependent parts as in post-mortem staining. Rigor mortis is slow to appear and hence lasts longer. If a body buried in snow is found in a condition of commencing decomposition, death is very likely not from cold, which prevents decomposition.

Internal.—The brain is congested, with effusion of serum into its ventricles. The heart contains fluid blood in both the chambers. The lungs and other organs are congested. Owing to the combination of oxygen with hæmoglobin, the blood is bright red in colour except in the heart, where it appears dark when viewed *en masse.*

Medico-Legal Aspect.—Death from cold is mostly accidental, though very rare in India. Drunkards may be found dead in the streets, when exposed to cold on a wintry night. Death from cold may form a case for medico-legal enquiry, as a newly born infant is, sometimes, murdered by exposure to cold by depriving it of the necessary clothes. Questions of responsibility as to homicide may arise in cases where insane, aged, sick or wounded persons have died from exposure to cold.

A newly born male infant, two or three days old, was found dead from exposure at night in the compound of a bungalow at Agra.

HEAT

The effects produced by exposure to excessive heat may be considered under one group, *viz.,* heat stroke, which manifests itself in three distinct types, namely—

1. Mild thermic fever.
2. Heat exhaustion.
3. Sunstroke.

Exposure to the direct rays of the sun is not necessary. An individual may be affected while working in a closed, hot, and badly ventilated room or factory. People, however, from a prolonged habit, can bear very high temperatures for a considerable period. Even persons unaccustomed to

1. *Renieke, Brit. and For. Med. and Surg. Rev., April,* 1876.

very much heat can bear it for some time if there is not much moisture in the atmosphere, and if they keep on drinking a lot of water, so that they may perspire freely.

The predisposing causes are ill-nourishment, over-exertion to a fatiguing point, such as long marches, over-indulgence in alcohol and previous attacks of the disease.

1. **Mild Thermic Fever.**—This affects persons working in small, closed rooms with open ovens, as in glass works.

Symptoms.—These are general lassitude and insensibility to light or sounds. The skin becomes hot, with a temperature of 103° or 104° F. Severe headache, prostration and delirium then follow. The condition may last from three to ten days in temperate climates, but in India these symptoms may be followed by coma, death occurring in three to four days.

2. **Heat Exhaustion.**—The attack may come on suddenly or gradually. In a sudden attack the patient falls down, and dies immediately or within a short period. When the attack is gradual, the first symptoms are giddiness, nausea, headache of a throbbing character, and dim vision with dilated pupils. Collapse then supervenes, with a subnormal temperature, rapid and feeble pulse and sighing respirations. Death occurs from heart failure, or reaction sets in after some time, followed by recovery. Throughout the course, consciousness is, as a rule, not lost.

3. **Sunstroke (Heat Apoplexy, Insolation or "Coup de Soleil").**—The symptoms supervene all of a sudden in a person exposed to very great heat in the summer months, especially if he has been fatigued by prolonged and extreme exertion, but in some cases prodromal symptoms, such as a feeling of heat, headache, giddiness, nausea and vomiting, may be experienced. Insensibility soon sets in, and the patient may be struck down with a temperature of 104° F. The temperature rapidly rises very high, even upto 112° or 115° F., and the skin is hot and dry. The face is flushed, and the pupils are first dilated and insensitive to light, but become contracted towards death. The pulse is full and bounding, and the respirations are hurried and stertorous. The urine which is usually offensive contains indican. Death may occur from syncope, but usually results from asphyxia and coma, followed often by convulsions and delirium. The shortest fatal period is five minutes[1]; it may be prolonged to three days.[2]

Treatment.—In heat exhaustion give a hot bath, and rub the body with hot towels or apply mustard plaster to the præcordium, and to the soles of the feet, if the temperature is below normal. Administer enemata of hot salines containing alcohol or some other stimulant. Give without delay hypodermic injections of ether, camphor in oil, strychnine, digitalis or ammonia. Administer chloroform inhalation or morphine hypodermically for the convulsions.

In true sunstroke measures should be taken to reduce the temperature as rapidly as possible. The patient should be unclothed and placed

1. *The Lancet*, 1870, *Vol. II, p.* 316.
2. *Katzenbach, New York Med. Jour., Vol. XIII.*

immediately in a bath containing water having a temperature of 70° to 80° F. He should be kept in the bath until his temperature has fallen to 101° F. After removal from the bath, if the temperature is found rising, an ice bag should be applied to the head, and ice should be rubbed over the body, or the patient should be again immersed in the cold bath. Ice-water enemata may also be employed with benefit. Venesection may be necessary, when the symptoms of intense asphyxia are evident.

After-Effects.—After recovery from heat stroke the patient becomes very susceptible to variations of temperature and usually complains of headache, loss of memory, mental confusion and nervous irritability. Sometimes, he may suffer from epilepsy or insanity for the rest of his life.

Post-mortem Appearances—External.—Rigor mortis is well marked, but comes on early and passes off rapidly, putrefaction following immediately after. Petechial and livid patches are found on the skin.

Internal.—The brain and its meninges are congested, and the ventricles contain serum. The lungs are congested and œdematous. The right side of the heart and pulmonary arteries are dilated, and gorged with dark fluid blood, and the left side is empty and contracted. The abdominal organs are congested.

Medico-Legal Importance.—There is no medico-legal importance attached to deaths occurring from heat stroke, as they are all accidental, but the medical man may have to hold a post-mortem examination on such a body if found lying dead on the road side or in a railway carriage, as it sometimes happens on hot summer days, and the police are bound to send such cases for autopsy.

In the case of *K. E.* v. *Lieut. Clark, I.M.D., Mrs. Fulham and others* charged under Section 302, I.P.C., with having murdered Mr. Fulham and Mrs. Clark, it was proved from the letters produced that under instructions from Lieut. Clark Mrs. Fulham so simulated heat apoplexy in her husband by the judicious administration of poison (a mixture of belladona or atropine and possibly cocaine) that the medical officers of the military hospital at Meerut were completely deceived and they treated him as a case of heat apoplexy. It may be mentioned that at the time some fatal cases of heat apoplexy had already occurred in the military hospital, and the knowledge of these cases led Mrs. Fulham to write to Clark at Agra to send her some poison which, when administered to her husband, would produce symptoms simulating heat apoplexy. The book of medical jurisprudence found in the possession of Clark at the time of his arrest was brought to me during the course of the trial at Agra. The symptoms described under certain poisons, such as arsenic, belladonna, cocaine, gelsemium, etc., had all been underlined with red pencil suggesting that he had made a special study of these poisons, most of which were alleged to have been administered to Mr. Fulham on different occasions.

CHAPTER IX

DEATH FROM BURNS, SCALDS, LIGHTNING, AND ELECTRICITY

BURNS AND SCALDS

Definition.—Burns are injuries produced by the application of flame, radiant heat or some heated solid substance, to the surface of the body. Injuries caused by friction, lightning, electricity, X-rays and corrosive chemical substances are all classified as burns for medico-legal purposes.

Scalds are injuries produced by the application to the body of liquids at or near their boiling points, or their gaseous forms, such as steam.

Scalds are usually not so severe as burns, as the liquids producing them run off the surface of the body, and rapidly cool on account of their evaporation, but they resemble burns very much in severity when produced by oils or other sticky substances, which boil at a much higher temperature than water. Scalds produced by molten metals cause great destruction of the tissues, as they adhere to the parts struck.

Burns resulting from X-rays are generally due to faulty exposure, and vary from mere redness of the skin to dermatitis with shedding of the hair and epidermis and pigmentation of the surrounding skin. Severe exposure may produce vesicles or pustules, which often form sloughing ulcers after they have burst, and take a long time to heal. The cicatrix formed is radiate in shape with the surrounding skin marked with pigmentation or permeated with numerous capillary vessels. Persons employed in the X-ray department and constantly exposed to the influence of the rays have, sometimes, suffered from chronic, intractable dermatitis and cancer of the parts exposed. Burns caused by radium are very similar to X-ray burns. The chemical rays of light, *e.g.*, ultra-violet rays, may produce erythema of the exposed part, or acute eczematous dermatitis. These burns are rarely seen now, as the operator uses special protective measures for himself and for his patient.

Burns produced by chemical corrosive substances, such as strong acids and caustic alkalies, are usually uniform in character, and the resulting eschars are soft and moist, and readily slough away. In these burns the red line of demarcation is absent, the hairs are not scorched, nor are the vesicles formed. But Greek fire, which is formed by dissolving phosphorus in carbon bisulphide, produces vesication by the rapid oxidation and burning of the phosphorus.

The characteristic stains found on the skin and clothing usually assist in determining the nature of the corrosive used. Chemical analysis of the clothing is also of importance in establishing the identity of the substance used.

These burns do not, as a rule, result in death, but may constitute grievous injuries involving loss of sight or permanent disfigurement from unsightly scars on the head or face.

The Classification of Burns.—Dupuytren has classified burns into the six following degrees according to the nature of their severity :-

First Degree.—This consists of erythema or simple redness of the skin caused by the momentary application of flame or hot solids, or liquids much below the boiling point. It can also be produced by mild irritants. The redness and swelling of the skin, marked with superficial inflammation usually disappear in a few hours, but may last for several days, when the upper layer of the skin peels off. At any rate, they disappear after death due to the gravitation of blood to the dependent parts. There being no destruction of the tissues, no scar results from this kind of burn.

Second Degree.—This comprises acute inflammation and formation of vesicles produced by the prolonged application of flame, liquids at a boiling point, or solids much above the boiling point of water. Vesicles can be produced by the application of strong irritants or vesicants, such as cantharides. Vesicles may also be produced on the part of the body which is allowed to soak in a decomposing fluid, such as urine or fæces, and subject to warmth, especially in the case of a patient who is bed-ridden from some nervous disease or old age, and is not properly nursed. If burns are caused by flame or a heated solid substance, the skin is blackened, and the hair singed at the seat of lesion, which assumes the character of the substance used. No scar results as only the superficial layers of the epithelium are destroyed. Some slight staining of the skin, however, may subsequently remain.

Third Degree.—This refers to the destruction of the cuticle and part of the true skin, which appears horny and dark, owing to its having been charred and shrivelled up. The nerve endings are exposed in this form of burn, and hence it is the most painful. This leaves a scar, but no contraction, as the scar, which forms after healing, contains all the elemnts of the true skin, and consequently the integrity of the part is retained.

Fourth Degree.—This means the destruction of the whole skin. The sloughs which form are yellowish-brown and parchment-like, and separate out from the fourth to the sixth day, leaving an ulcerated surface, which heals slowly, forming a scar of dense fibrous tissue with consequent contraction and deformity of the affected parts. On account of the complete destruction of the nerve endings this kind of burn is not very painful.

Fifth Degree.—This includes the penetration of the deep fascia and implication of the muscles, and results in great scarring and deformity.

Sixth Degree.—This involves charring of the whole limb, and ends in inflammation of the subjacent tissues and organs, if death is not the immediate result.

Effects of Burns.—Burns and scalds vary in their effects according to the following conditions:—

Fig. 50.—Extensive burns from clothes catching fire.

1. The degree of heat applied.
2. The duration of exposure.
3. The extent of the surface.
4. The site.
5. The age of the patient.
6. The sex.

1. **The Degree of Heat Applied.**—The effects are much more severe if the heat applied is very great.

2. **The Duration of Exposure.**—The symptoms are also more severe if the application of heat is continued for a long time.

3. **The Extent of the Surface.**—The involvement of one-third to one-half of the superficial surface of the body is likely to end fatally.

4. **The Site.**—Extensive burns of the trunk, even though superficial, are much more dangerous than those of the extremeties. Burns of the genital organs and the lower part of the abdomen are often fatal.

5. **The Age of the Patient.**—Children are more susceptible to burns, but stand prolonged suppuration better than adults. Aged people bear burns well.

6. **The Sex.**—Sensitive and nervous women are more susceptible to burns than strong women, and women generally do not bear burns so well as men.

Causes of Death.—1. Shock.—Severe pain from extensive burns causes shock to the nervous system, and produces a feeble pulse, pale and cold skin and collapse, resulting in death instantaneously or within twenty-four to forty-eight hours. In children it may lead to stupor and insensibility deepening into coma and death within forty-eight hours. In order to avoid the suggestion that coma was due to the drug it is advisable not to administer opium in any form for the alleviation of pain.

If death does not occur from shock, it may subsequently occur from toxæmia due to the absorption of certain poisonous substances, such as protein and histamine, from the burned area. In this condition the temperature rises perhaps to 104° F., the pulse rate increases in frequency, and restlessness supervenes and passes into unconsciousness and death.[1]

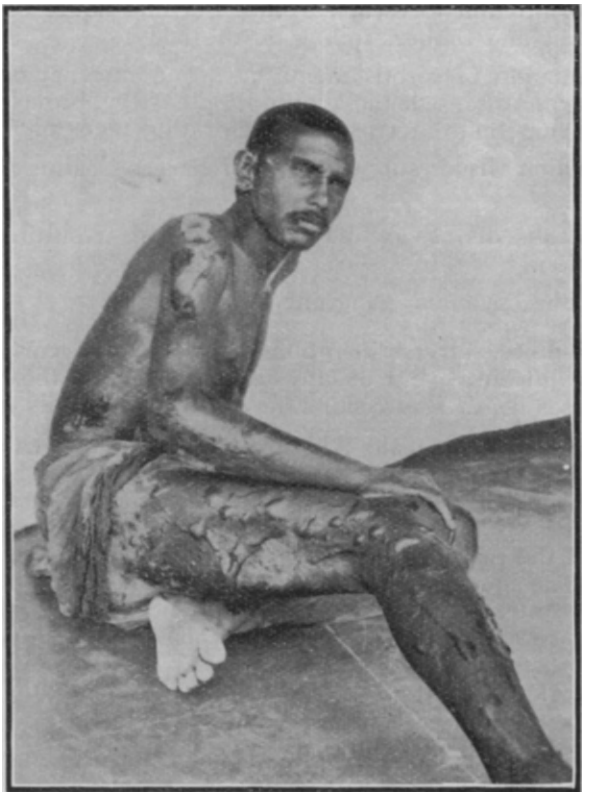


Fig. 51.—Scalds from a fall into a cauldron of boiling *ghee* (clarified butter). Note the blisters.

2. **Suffocation.**—Persons removed from the houses destroyed by fire are often found dead from suffocation due to the inhalation of smoke, carbon-dioxide and carbon-monoxide—the products of combustion. In such a case the burns found on the body are usually post-mortem.

On or about the 12th January, 1917, a lunatic in the asylum at Agra was suffocated in bed from smoke produced by the quilt with which he had covered his face catching fire and the extensive superficial burns found on the body appeared to have been caused after death.

Between 1 a.m. and 3 a.m. on the 6th January, 1922, some dacoits broke into the house of one Kusher Lodh, aged 50 years, and, finding him and his son, 20 years old, sleeping in a room, chained it from outside. On leaving the house they set fire to rubbish lying at the door with the result that the father and the son died in the room. The post-mortem examination of both the bodies afforded clear evidence

1. *Lloyd, Brit. Med. Jour., Aug.* 1, 1931, *p.* 177.

of death from suffocation. The larynx and trachea in both were congested with a deposit of soot along the interior. The lungs were congested and exuded frothy blood on section. The brain vessels were found engorged with blood. There was general venous engorgement. Externally the bodies showed a few small superficial burns on the face, thighs and legs with singeing of the hair of the head.

3. **Accidents or Injuries.**—Death may result from an accident occurring in an attempt to escape from a burning house or from injuries inflicted by walls and timbers falling on the body.

4. **Inflammation** of serous membranes and internal organs, such as meningitis, peritonitis, œdema glottidis, pleurisy, bronchitis, broncho-pneumonia, pneumonia, enteritis and perforating ulcer of the duodenum.

5. **Exhaustion** from suppurative discharges lasting for weeks or months.

6. **Lardaceous** disease of the internal organs resulting from suppurative exhaustion.

7. **Erysipelas**, pyæmia, gangrene and tetanus.

Fatal Period.—As already mentioned, death may occur within twenty-four to forty-eight hours, but usually the first week is the most fatal. In suppurative cases death may occur after five or six weeks or even longer.

The Nature of Burns in the Absence of Death.—In a case where death has not occurred, burns will constitute simple or grievous hurt as the case may be. Burns of the first and second degree, if not extensive, are mostly simple. Burns are grievous, if they cause scars causing permanent disfiguration of the head or face, permanent loss of sight of either eye, or permanent impairment of a member or a joint owing to the formation of a cicatrix and contraction, if a joint and its neighbouring parts have been severely burnt. Lastly, burns are grievous if the individual has suffered from shock so as to endanger life, or if he has been in severe bodily pain or bed-ridden, and unable to follow his ordinary pursuits for twenty days.[1]

Sections 324 and 326 of the Indian Penal Code deal with the punishments to be awarded for simple and grievous hurt caused voluntarily by means of fire or any heated substance, or by means of any corrosive substance, or by means of any explosive substance.[2]

Post-mortem Appearances—External.—The external appearances of burns vary according to the nature of the substance used to produce them. Thus, the skin is whitened when a burn has been caused by radiant heat.

Burns produced by flame may or may not produce vesication, but singeing of the hair and blackening of the skin are always present.

A highly heated solid body or a molten metal, when momentarily applied to the body, may produce only a blister and reddening, corresponding in size and shape to the material used, but will cause roasting and charring of the parts when kept in contact for a long time.

Burns caused by explosions in coal mines or of gunpowder are usually very extensive, and are accompanied by blackening and tattooing due to the driving of particles of unexploded powder into the skin.

1. *Appendix VII, Section* 320, *Indian Penal Code.*
2. Vide *Appendix VII.*

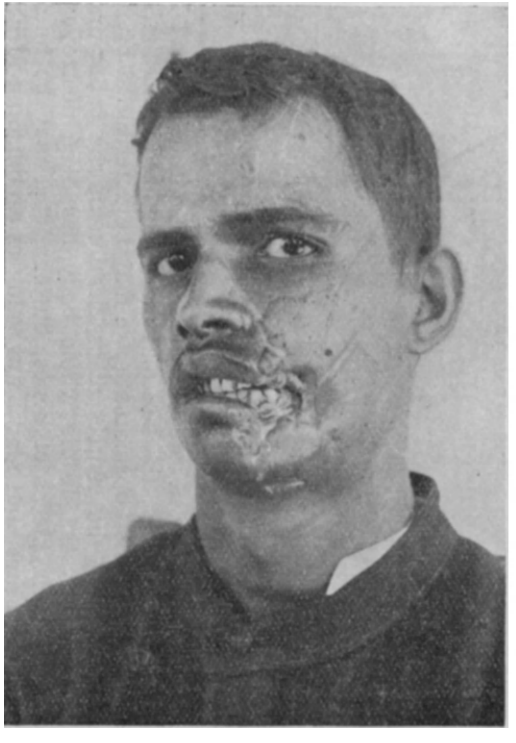


Fig. 52.—Effects of burning caused by the explosion of a cracker placed in the mouth and chewed in mistake for a *laddoo* (sweetmeat) during the state of intoxication. (*From a photograph lent kindly by Dr. G. B. Sahay*).

Fig. 53.—Burns on the face, abdomen and hands from the explosion of a bomb.

Burns caused by kerosene oil are usually very severe, and are known from its characteristic odour and the sooty blackening of the parts.

When a body has been exposed to great heat, it becomes so rigid with the limbs flexed and arms fixed that it assumes an attitude of defence, called the "pugilistic" or "fencing" posture. This stiffening occurs on account of the coagulation of its albuminous constituents. If the heat applied is very great cracks and fissures resembling incised wounds often occur in the skin and tissues, but no blood clot, nor infiltration of the blood, is found in the cellular spaces, and the blood-vessels are seen stretching across the fissures, as they are not usually burnt. Sometimes, the skin, being hard and brittle due to the effect of heat, cracks easily when an attempt is made to remove the body from a house destroyed by fire.

Scalds caused by boiling water or steam produce reddening and vesication, but do not affect the hairs, and do not blacken or char the

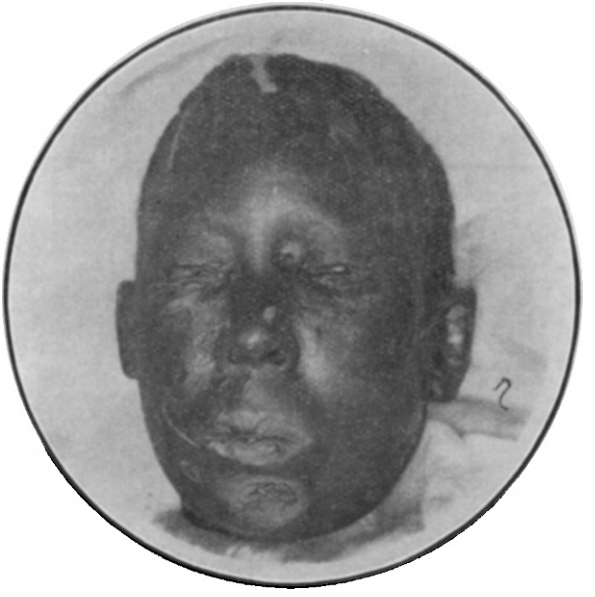


Fig. 54.—Burns on the face from a kerosene oil lamp.

skin. Superheated steam soddens the skin, which has lost its elasticity, and has a dirty white appearance.

It is difficult to identify a badly charred or incinerated body. If the skeleton has remained intact, the sex may be recognized from the shape of the pelvic bones, and the approximate age may be determined by noting the teeth and observing the centres of ossification in the bones and the condition of the epiphyses. If the whole body has been destroyed and reduced to ashes, teeth, pieces of bones, buttons, etc., may be found on carefully sifting the ashes, and may be of value in establishing identity.

Internal.—The skull bones are found fractured or burst open if intense heat has been applied. There is an extravasation of blood, usually brick-red in colour, upon the upper surface of the dura mater. The brain

is sometimes shrunken though its form is retained. In a case of death from accidental burning on the 30th November, 1921, I found the membranes adherent to the skull cap and the brain shrunken and dried up. If death has occurred from suffocation the larynx, trachea and bronchial tubes may contain sooty particles, and their mucous membrane may be congested and covered with frothy mucus.

The pleuræ are congested or inflamed, and there may be serous effusion into their cavities. The lungs are usually congested; they may be shrunken and rarely anæmic. The chambers of the heart are usually full of blood. The blood is cherry red in colour if death has occurred from suffocation. The mucous membrane of the stomach and intestines is frequently reddened. There may be inflammation and ulceration of

Fig. 55.—Scalds caused by accidentally falling into a pail of boiling water. Note the blisters.

Peyer's patches and solitary glands of the intestines. Ulcers may also be found in the duodenum when the patient dies some time after receiving the burns. These ulcers are more common in women than in men, whereas the idiopathic ulcer in the duodenum is more frequent in men.

The kidneys may show signs of nephritis, and on section the straight tubules may be filled with debris of the blood corpuscles giving the appearance of reddish-brown markings.

The uterus and testicles resist the action of fire, and may show very little change even when the body is almost consumed.

Distinction between Ante-mortem and Post-mortem Burns.—People sometimes produce burns on a dead body to support a false charge of murder, and at other times the police remove a dead body while in the act of burning on a cremating pyre and send it to the medical officer for

post-mortem examination, when they suspect that the body is being hurriedly cremated to conceal the crime of murder. In both cases the medical officer should be prepared to tell the difference betwen ante-mortem and post-mortem burns.

The three main points to differentiate between ante-mortem and post-mortem burns are—

1. The line of redness.
2. Vesication.
3. Reparative processes.

1. **The Line of Redness.**—In the case of a burn caused during life a line of redness involving the whole true skin is formed round about the injured part. It is a permanent line, persisting even after death, but

Fig. 56.—Body of a male burned and charred after death from throttling.

redness or erythema, which is found beyond this line of redness due to distension of the capillaries, is transient, disappears under pressure during life and fades after death. The line of redness being a vital function separates living from dead tissue, and is always present in burns caused

during life, though it takes some time to appear. Hence it is possible that it may be absent in the case of a person of a very weak constitution who dies immediately from shock due to burns.

2. **Vesication.**—Vesication caused by a burn during life contains a serous fluid consisting of albumen and chlorides, and has a red, inflamed base with raised papillæ. The skin surrounding it is of a bright red or coppery colour. This is known as *true* as compared with *false* vesication which is produced after death. False vesication contains air only, but may contain a very small quantity of serum comprising a trace of albumen, but no chlorides as in a person suffering from general anasarca. Again, its base is hard, dry, horny and yellow, instead of being red and inflamed.

3. **Reparative Processes.**—Reparative processes, such as signs of inflammation, formation of granulation tissue, pus and sloughs, will indicate that the burns were caused during life. Burns caused after death have a dull white appearance with the openings of the skin glands coloured grey. The internal organs are roasted, and emit a peculiar offensive odour.

The Period of Burns.—In the case of a burn the question is raised as to when it was caused, and in the case of several burns on the same individual a further question is raised as to whether they were inflicted simultaneously. Both these questions may be answered by examining carefully their condition as regards the different stages of reparative processes.

Redness and vesication form immediately or within a few hours, say two to three, after receiving burns. Pus forms in two to three days but not before thirty-six hours. The formation of granulation tissue, and separation of sloughs, occur after several days. The last result is the formation of a cicatrix and deformity.

Suicidal, Homicidal and Accidental Burns.—Suicidal cases are rare among men. A case[1] occurred at Hapur, where a treasurer of the local branch of the Imperial Bank of India committed suicide at midnight by putting himself on a pyre of charcoal and wood and throwing kerosene oil on it and then setting fire to it. Occasionally women, disappointed in love or tired of domestic worries or some acute or chronic ailment, commit suicide by soaking their clothes first with kerosene oil and then setting fire to them. The pernicious custom of dowry among certain Hindu castes, sometimes, leads young maids to commit suicide in this manner with a view to relieve their poor parents of the anxiety to raise sufficient money for the dowry at their marriages.

A Hindu woman, aged 40 years, who was suffering from phthisis, put an end to her life by setting fire to her clothes after soaking them with kerosene oil. On post-mortem examination held at 9-30 a.m. on the 22nd April, 1923, 18 hours after death, the body was found to have assumed the pugilistic attitude. The arms were extended from the shoulders, and the forearms partly flexed. The thighs were almost perpendicular to the abdomen and the legs partly flexed at the knees. There were extensive burns of the whole body including the anus and private parts. The hair of the head, eyebrows and eyelashes was singed. The eyes were closed and congested. The mucous membrane of the larynx and trachea was congested and covered with froth mixed with soot. The brain and its membranes were congested.

1. *Leader,* March 7, 1934.

Burns are, sometimes, self-inflicted for purposes of false accusations.

In August, 1923, a Mahomedan woman, about 18 years old, filed a complaint at the City Magistrate's Court at Lucknow that she was burnt by her husband with a pair of tongs. She had several small marks of superficial burns causing redness and vesication on the wrists, forearms, legs and thighs. Some of these had the shape of the knob of the tongs. During the trial it was suggested that they appeared to have been self-inflicted, inasmuch as they were on the places easily approachable by the woman herself. It was afterwards discovered that they had been self-inflicted and the woman had brought a false accusation as she wanted divorce from her husband.

Homicidal cases are fairly common in India. Burns are often caused by a mother-in-law on the body of her infant daughter-in-law for very trifling faults. The substances selected are generally a pair of hot tongs (*chimta*) or *karchi* and the sites selected are usually the arms, hands, thighs and private parts. I have seen several such cases with three deaths —two in Agra and one in Lucknow. Among grown-up females burns are produced usually on the pudenda, as a punishment for adultery. When a master becomes angry with his servant for disobedience or petty theft, he, sometimes, produces burns on his body with a heated solid substance, such as a hot pipe or *chilum*. Robbers and dacoits often inflict burns as a torture to extort information about valuables hidden in the houses of their victims. Sometimes, they burn their victims to death by pouring kerosene oil over their clothes and then setting a light to them.

Cases.—1. Musammat Hardei owing to domestic quarrels with her daughter-in-law burned her to death by throwing kerosene oil over her clothes and then setting fire to them. The oil fell over the clothes of her child, one and a half years old, who also died.[1]

2. On the night of the 21st May, 1922, a gang of dacoits went to the house of Bihari Lal at Uchasia in the Bisalpur police circle. Bihari Lal was away, but they got hold of his mother, Musammat Indo, his sister, Musammat Kamli and his wife, Musammat Rampa. They poured kerosene oil over Musammat Indo, and set a light to it. Musammat Kamli protested; hence they poured a great deal of oil over her and burnt her so badly that she died a few hours later. They had torches, and after robbing the inmates they went away.[2]

3. At Trivandrum a servant, harbouring ill feelings against his master, poured petrol over the latter at night, when he was lying in a chair and set fire to him. Seeing his master roll frantically over the floor, the servant poured more petrol over the victim. The man sustained serious burns and died in the hospital a few hours after admission.[3]

4. One Mani Ram[4] caused the death of his daughter-in-law, aged 9 years, by burning her all over the body with a heated *karchul*. He sat on her legs and gagging her mouth with cloth in order to prevent her from crying for help, he deliberately branded her with the *karchul* several times each time withdrawing it from the fire and placing the hot metal against the body and then heating it again. The burns were mostly on the chest, abdomen, back, buttocks, private parts, thighs, cheeks, right orbit and left hand. The reason why the man branded the girl was that she had eaten some of the bread which he had kept for himself.

Sometimes, murderers kill their victims by some other means, and then set fire to their bodies or to their houses in order to conceal all evidence of the crime. In such cases fatal injuries, such as fractures of

1. *King-Emperor* v. *Mt. Hardei, Chief Court of Oudh, Crim. App. No.* 64 *of* 1927.
2. *King-Emperor* v. *Shib Singh and Kandhari Singh, All. High Court, Criminal Appeal No.* 636 *of* 1924.
3. *Times of India, October* 9, 1930.
4. *King-Emperor* v. *Mani Ram, Oudh Chief Court, Crim. App. No.* 234 *of* 1931.

skull bones, etc., as a result of mechanical violence, or signs of strangulation, suffocation or poisoning, may be found on the bodies, if they are not completely destroyed by fire. It must, however, be remembered that injuries on the body, such as lacerated wounds or fractures of bones, may be produced by beams, walls, etc., of a burning house falling on a living or dead person.

Accidental cases are very common, especially among women and children, on account of their loose garments catching fire, while sitting near an *angethi, chula,* Primus stove or an open lamp. Lately, cases of accidental death by burns sustained from Primus stoves have become so frequent among the Gujarati women of Bombay that the Coroner has, on several occasions, passed strong strictures against their husbands or parents and warned them not to allow the use of these stoves in their houses.

A number of persons may die from burns when a fire breaks out in an inhabited house or when an explosion occurs in a factory of gunpowder or fireworks. In such cases wounds caused by the falling of rafters, bricks, etc., in addition to the burns, may be seen on the bodies.

Children and feeble, epileptic, blind or intoxicated persons may fall in fire or in cauldrons of boiling water, oil or *ghee.*

Children may be scalded by trying to drink from the spout of a kettle containing boiling water, or by the kettle accidentally falling upon them.

Spontaneous or Preternatural Combustion.—The possibility of spontaneous combustion of a human body may be raised as a plea in defence, but it must be remembered that it has not yet been proved scientifically that a body can be consumed without the application of fire or flame, though a few unauthentic cases have been recorded. It is even difficult to believe that a body composed of seventy-five per cent of water could catch fire from a spark or flame and be reduced to ashes without the surrounding objects being set on fire. It is, however, possible that the combustibility of a body may increase if the individual be fat, bloated, and has passed middle life, and is saturated with alcohol on account of intemperate habits. It is also possible for a body to burn spontaneously owing to the evolution of inflammable gases during the process of decomposition. Even an individual may eliminate an inflammable gas in breath during life. Beatson[1] records the case of a man who, while attempting to blow out a match, woke his wife from sleep owing to a loud noise due to the explosion caused by the combination of his breath (marsh gas) with the flame of the lighted match. Martin[2] mentions a case in which a man used to get frequent eructations of foul gas due to a severe form of flatulent dyspepsia. One morning just as he was lighting his pipe with a match, he was obliged to eructate. The gas coming in contact with the lighted match exploded with a sparkling flash scorching the hair of his beard and eyebrows.

1. *Brit. Med. Jour., Vol. I,* 1886, *p.* 295.
2. *Lancet, Vol. II,* 1902, *p.* 991.

LIGHTNING

During thunderstorms people are, sometimes, struck down by lightning or atmospheric electricity in the open fields or in their houses, especially near open doors and windows, through which it enters. It is attracted by the highest points; hence it is dangerous to stand near tall trees during thunderstorms. Similarly, it is dangerous to have a good conducting material on the body or in its vicinity. Wet clothes and wet skin are also good conductors, while dry clothes and dry skin are bad conductors.

Symptoms.—Death occurs immediately from shock, or subsequently from the effects of burns and lacerations after some days or even weeks. In non-fatal cases the individual complains of giddiness, ringing in the

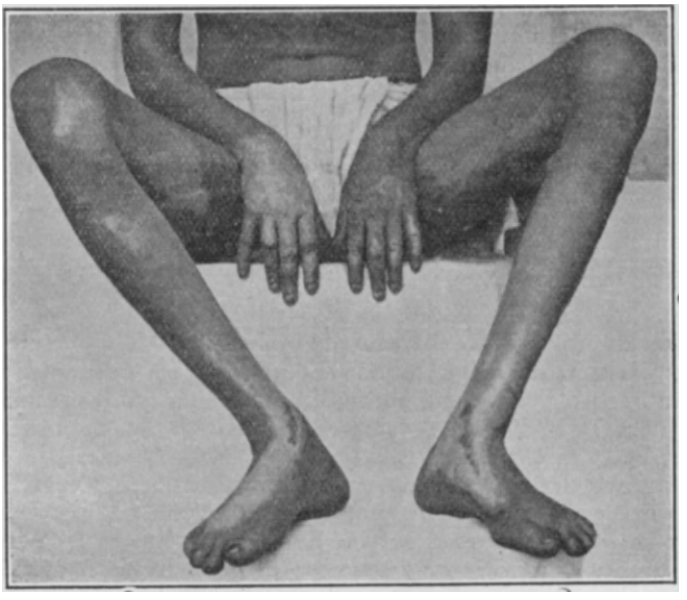


Fig. 57.—Burns caused by lightning.

ears and headache. These symptoms pass off very soon or hysteria and nervousness may supervene, when the lightning dicharge is very slight, though in severe shock the patient may suffer from loss of memory, paralysis, tetanic convulsions, delirium, cardiac affections, blindness, deafness or dumbness.

Post-mortem Appearances—External.—The lesions produced by a lightning stroke on the skin near the points of its entry and exit are ecchymoses, contusions and lacerations. Wounds of all varieties and fractures of bones may also be present. In addition to singeing of the hair, blisters, fissures and even charring caused by burns, reddish-brown arborescent (tree-leaf) markings are often seen on the body. It was thought that these markings were due to the photographs of the neighbouring trees imprinted on the body by the discharge of lightning, but in

reality they are the result of the passage of lightning stroke along the skin and consequent rupture of the superficial vessels.

The wearing apparel is usually burnt or torn at the point where the lightning strikes and enters the body and at the point where it leaves the body. In some cases the wearing apparel may be wholly stripped from the body and thrown to some distance. One or both of the boots or shoes may be burnt or torn to pieces. Sometimes, the soles may be torn off the upper leathers or a large hole may be torn in them.

It must be remembered that it is possible that a person may be killed by lightning, while the clothing is not damaged. On the contrary, the clothing may be burnt and torn off the body without causing any injury to the person.

Glass and metallic articles carried about the person are fused and articles of steel, such as a pen-knife, are magnetised ; these may leave their impressions on the skin.

Internal.—The internal signs are not very characteristic. There may be extensive hæmorrhage of the brain. The blood is usually fluid but may be found clotted. The cavities of the heart are either empty or full. The blood-vessels may be found ruptured, and the internal organs torn.

The following points should be taken into consideration before an opinion is given that death was caused by lightning :—

1. History of a thunderstorm in the locality.

2. Evidence of the effects of lightning in the vicinity, *e.g.*, damage to houses or trees, death of cattle, etc.

3. Fusion or magnetisation of metallic substances.

4. Absence of wounds and other injuries indicating homicidal death.

Illustrative Cases.—1. A farm labourer was struck by lightning whilst in the act of urinating under a tree. His boots were found at the bottom of the tree. They had completely lost their laces, some of the eyelets had burst, the leather was torn and the sole of the right boot was torn. He was found lying on the back at a distance of a couple of yards with nothing on the body except a portion of the left arm of his flannel vest. He was quite conscious. His clothes were torn to shreds and scattered all over the field. The buckle of his belt was burnt out. His watch had a hole fused through it, and the chain was almost destroyed. The hair on the face was burnt, and the body was covered with marks of burning, which were superficial on the chest, but deeper on the abdomen and the right thigh. There was a compound fracture of the right tibia, and there was a comminuted fracture of the os calcis with a lacerated wound of the right heel. The man eventually recovered completely.—*Wilks, Trans. Clinic. Soc., London, Vol. XIII,* 1880.

2. A sepoy, aged 25, was struck by lightning while walking across a meadow carrying an open umbrella in his right hand and a brass *lota* in his left hand. When seen twenty-six hours after the accident he was collapsed ; the right hand which had carried the umbrella was blackened and vesicated, the rest of the arm up to the shoulder being uninjured. There was a deep lacerated wound, four inches long, in front of the right coracoid process, the capsule of the process being almost laid bare. The whole of the clavicular portion of the deltoid, as well as the outer fibres of the pectoralis major, were completely divided, the wound having a charred and blackened surface ; the right side of the face, chest and loins was charred ; the whiskers, moustache, eyebrows and eyelashes were burnt, and the inner sides of both thighs were denuded of epidermis. The pupils were slightly contracted.—*Ind. Med. Gaz., Nov.* 1, 1886 ; *Collis Barry, Leg. Med., Vol. II, Ed. II, p.* 28.

3. Two men were standing in the door-way of a cowshed while a violent thunderstorm was in progress. A flash of lightning struck them, throwing both to the ground, one on top of the other. They were seen about half an hour later. Both had arborescent markings on the left side of the face and neck of an erythematous nature. The marks lasted for about twenty-four hours.

During the fall one struck his head on the brick floor and developed a traumatic cataract. Both men complained of pain in the supra-clavicular region radiating down the arm. Weakness of the affected limb and sensations of pins and needles were present for two or three weeks, but this condition cleared up, leaving no disability.—*Lloyd Williams, Brit. Med. Jour.*, Oct. 13, 1923, *p.* 659.

4. Edwards describes a case in which he saw a young woman said to have been struck by lightning on the night of July 27, 1925. Over the right hip she had a large bruise about six inches in diameter, with a central area of scorching. She was also suffering from a mild degree of shock. Her clothes were quite uninjured. At the time of the accident she had been standing close to the "lead in" of the wireless aerial. This had completely fused, and it seemed to be the flash from the fusing wire which had done the damage. She described it as seeming as though the whole of her right side had caught fire.

Considerable damage had been done to the window frame where the wire entered the house, and bricks had been dislodged from the wall, although the aerial was "earthed" by a switch inside the house. Apparently this common form of protection against lightning is useless. The aerial, which was of seven-strand copper wire, was fused in several places.—*Brit. Med. Jour., Aug.* 15, 1925, *p.* 294.

5. A woman, while working in a field, was struck by lightning. Examination revealed a mark of the size of a penny at the top of the head where the lightning had entered the body. The hair on the occiput was singed. Along the entire course of the spinal column a mark from 25 to 30 cm. wide was noticeable, and it was continued on the back of the thigh. A second track of lightning branched off at the neck, and a mark from 2 to 3 cm. wide was visible along the left breast and on the left side of the abdomen, and from the pubic symphysis it passed over to the right thigh. The pubic hair was singed. The mark of lightning showed several skin defects and coagulation necrosis, 4 cm. in diameter. Around these, areas of black discoloration and numerous small black holes were noticeable. The heart sounds were weak. The other internal organs were normal. The patient was unconscious for forty-eight hours. There was a tear across the tympanic membrane of each ear. When the woman regained consciousness, the retrograde amnesia with regard to the accident continued. She complained of severe pain in the region of the burned areas and of chills. She recovered in 20 days after the accident. On the sixth day a lumbar puncture was made on account of severe headache. Burns were treated by tannic acid ointment.—*Dengl, Munchener Medizinische, Wochenschrift, Munich, Vol.* 78, *Jan.* 2, 1931, *p.* 27; *Jour. Amer. Med. Assoc., April* 11, 1931, *p.* 1274.

6. The following case was reported to me by Major P. N. Basu, I.M.S., Superintendent of District Jail, Bareilly :—

On May 1, 1933, about 6 p.m. lightning struck the undertrial ward of the district jail, Bareilly, in which about a dozen undertrial prisoners were sitting in a row of two in the middle line of the barrack facing the iron grated door.

The lightning-flash struck the arch of the front iron grated door of the barrack, travelled down to the floor for a distance of about 29 feet, scattering in several sparks, and then upwards to an iron rod, 6 feet long, hanging from the roof. It melted the rod, and then passed to the floor of one of the side iron grated windows. Here it fused two iron cups lying in the vicinity and finally passed into the earth.

All the twelve undertrial prisoners, who were sitting in the barrack, were simultaneously struck, and were thrown upon its floor in varying states of insensibility. One of them suffered from a severe type of shock, was unconscious for twenty-four hours after which he recovered. Another person developed paralysis of the left upper and lower limbs, but recovered. Two were very severely burnt mostly in the abdomen, chest, buttock, scrotum, penis and thighs. One of them died and the other recovered.

The characteristic features of the injuries of all these cases were as follows :—

1. They were mostly on the lower part of the body.

2. The burns varied in degrees from mere discoloration to scorching and destruction of the skin. The burns never went deeper.

3. Eczema similar to one noticed after X-Ray burns developed in most of these burns.

4. Shock was the predominant symptom and injection of gum and pituitrin had a marked beneficial effect.

5. Some of these patients developed a fright complex after recovery.

ELECTRICITY

Cases of injury or death from electric shocks occur in those cities where electricity is used for lighting and motive purposes. The electrical main may break, and the two ends may fall on a person, thus making a short circuit, or the workman may grasp the ends of a live wire, or may stand on one with the other in his hand.

The Effects of Electricity.—The chief effect of electricity is shock produced by its current. It varies in accordance with—

1. The nature of the current.
2. The resistance of the body.

1. **The Nature of the Current.**—Currents generated at high voltages are dangerous to life. Alternating currents are considered more dangerous than continuous currents, probably because they are usually generated at high tensions; but continuous currents of high tensions are equally dangerous under similar circumstances. Battelli[1] considers that alternating currents of low periodicity are dangerous even at low voltages, but the danger diminishes with the increase of frequency even when generated at high voltages.

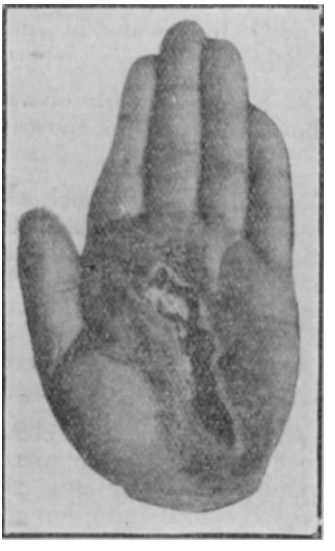


Fig. 58.—Lesion of hand from contact with a live current of 250 volts. (*From Pathological Museum, King George Medical College*).

In addition to high voltage, long duration and close contact are the essential features of danger from electric currents.

In continuous currents shock is produced at the moment of the opening and closing of the circuit. In alternating currents it is also produced at each reversal of the machine.

Many of the fatal accidents have occurred from currents carrying more than 1,000 volts, but cases are recorded, where death has resulted from currents of 200 or even lower volts. In such cases the victims were mostly standing in water or on damp ground, and were thus well "earthed." Professor Jellinek mentions the case of a man who was killed by a current of 95 volts, when standing with wet boots in a mixture of potash and sugar in a factory at Prague. On the other

1. *Lewis Jones, Medical Electricity, Ed. VII, p.* 315.

hand, recovery has occurred after the passage of a current of 2,500 volts and even of 5,500 volts. Fitzsimons[1] records the case of a man, through whose body a current of 20,000 volts passed at a colliery. He recovered consciousness after twenty minutes' artificial respiration. He was none the worse after a few days, except for a large piece of flesh which sloughed from the sole of one of his feet. He was standing at the time of the shock on a dry concrete floor, but totally unprotected as regards gloves or rubber shoes.

The danger of a person in a bath touching a defective electrical switch, in consequence of the water and bath furnishing a perfect "earth" for the current, is well known.

A girl aged 17 years, on going to her bath took an electrical heater with her to warm the bath-room. After a few minutes her mother heard screams and forced the door open. She found the girl unconscious in the bath with the heater on the top of her. She was removed to a hospital where she died. A woman, 23 years old, took a book and an electrical hair drier to her bath-room. Her father found her with the drier in her right hand and the book at the end of the bath. When he touched her, he got a shock himself, the handle of the drier being saturated and the current still on.[2]

Judicial electrocution is the form of execution employed in the United States of America. For this purpose the condemned criminal is seated and strapped in a strong chair, and an alternating current of 7½ amperes at a pressure of 1,700 to 2,000 volts is, as a rule, passed three times for about thirty seconds through the body by means of metal electrodes placed over the head and round one leg.

According to the regulations of the Board of Trade currents of more than 250 volts are not supplied to dwelling houses for the purposes of lights and fans.

2. **The Resistance of the Body**.—The effects of electric shock vary with the amount of resistance offered to the flow of its current. The human body is a bad conductor of electricity, though the resistance varies in different tissues. The skin offers very great resistance and when perfectly dry is impervious to currents of great strength. Hard and oily skins are also resistant, but moist, soft and perspiring skins are less resistant. The resistance diminishes with the continuance of the current. It also diminishes in kidney diseases, Baesdow's disease and hysteria.

Symptoms.—The symptoms produced by the passage of an electric current are local lesions at the points of entrance and exit of the current, pallor of the face, suffused eyes, dilated pupils, cold, clammy skin, stertorous breathing, and insensibility. Signs of cerebral irritation may, sometimes, be present. In severe cases insensibility occurs immediately followed by a few gasps and death.

According to Professor Jellinek[3] the lesions produced by the electric current are not burns, and none of the surrounding structures is altered. They heal without infiltration, suppuration or pyrexia, but when tendons, joints and large areas are involved, there may be some aseptic necrosis

1. *Brit. Med. Jour., Nov.* 15, 1924, *p.* 932.
2. *London Correspondent, Jour. Amer. Med. Assoc., March* 11, 1933, *p.* 752.
3. *Lancet, Nov.* 5, 1927, *p.* 1002; *Medizinische, Klinik, Sept.* 23, 1927.

and it is presumably this change which accounts for hæmorrhages which sometimes complicate recovery.

After recovery from electric shock, there may be paralysis due to degenerative changes in the nervous system, or death may occur later from extensive destruction of the tissues of a limb or limbs. Rarely, optic atrophy and other intraocular lesions may develop several months afterwards.[1]

Lucas[2] reports the case of a boy, aged 15 years, who, while standing on a discharged transformer so as to dust a ledge, slipped and clutched at a cable carrying a current of 10,000 volts. Respiration at once ceased and the limbs were burnt, swollen and stiff. The boy recovered consciousness after artificial respiration, but the limbs became gangrenous. On the third day the right arm was amputated through the shoulder-joint. Arterial thrombosis was found upto the origin of the superior profunda. Both legs were amputated on the sixth day. Toxæmia set in, and the boy died on the ninth day after the accident.

Causes of Death.—Death from electric shock may occur from sudden stoppage of the action of the heart, or from paralysis of the respiratory centre due to nervous inhibition. According to the experiments of Langworthy[3] death from fibrillation of the cardiac ventricles is believed to be more common after contact with low voltage circuits, while circuits at high voltages cause death from respiratory failure due to a central inhibition in the nervous system.

Treatment.—The current should be switched off at once, or the patient should be removed from the vicinity of the live wires, but the person trying to remove him should guard himself against its effects by wearing India-rubber gloves, by standing on hay, or by using a long stick to remove the wires. The treatment to be adopted after removal is stimulation, warmth, friction, artificial respiration and venesection, if necessary. Pometta[4] recommends that artificial respiration should be continued for at least five hours except in those cases where the injuries are so severe that the patient cannot possibly be alive. Carbon dioxide is a valuable respiratory stimulant, and can be given from a soda-water syphon when no cylinder is available. The syphon is half emptied and a rubber tube is attached to its nozzle ; it is then inverted and the fluid is blown out of the glass tube. Gas is admitted to the patient's air-passages through one nostril.

Post-mortem Appearances—External.—The face is generally pale, the eyes are congested and the pupils are dilated. Local lesions are found at the points of entrance and exit of the electric current. Professor Jellinek[5] has pointed out that the micropathological changes, as observed in the skin at the site of an electrical lesion, are a compression of the horny layer into an homogeneous plaque, and an ironing out of the underlying papillary process. Occasionally fissures and hollows appear between the corneum and germinativum, but this is not invariable, and the surest sign that an electric current has passed is the coalescence into a star-shaped or rod-like structure of the basal cells in each group of the rete Malpighii.

1. *Bainbridge, Brit. Med. Jour., Dec.* 6, 1930, *p.* 955.
2. *Brit. Med. Jour., Jan.* 21, 1905, *p.* 134.
3. *Jour. Exper. Med., June* 1, 1930, *p.* 943 ; *Brit. Med. Jour., Aug.* 30, 1930, *Ep., p.* 38.
4. *Schweiz Med. Woch.*, 1930, *Vol. IV, p.* 82 ; *Lancet, Feb.* 8, 1930, *p.* 310.
5. *Lancet, Loc. Cit.*

Internal.—The lungs are often found œdematous, and the other internal organs are congested. Minute hæmorrhages are seen in the meninges, and Tardieu's spots are found on the pleuræ, pericardium and endocardium. Ecchymoses may be noticed along the path of the current.

In the case[1] of a young man, about 21 years of age, who was killed by a direct current of 220 volts, the following post-mortem appearances were found :—

There was no sign of electric burn on the skin. There was intense vascular congestion of the dura mater, more noticeable on the left than on the right. The cerebral vessels were similarly congested, free blood being present with clots around the medulla, between the cerebellum and tentorium and over the island of Reil. Both lateral ventricles were full of blood-clot.

Medico-Legal Questions.—Deaths by electric currents are mostly accidental. Recently, a case occurred in one of the suburbs of Bombay, in which a milkman was accidentally killed by touching the door of the house where he had gone early in the morning to sell milk. The owner of the house had attached a live wire to the door to prevent the entrance of thieves.

The danger of flying kites in the vicinity of overhead electric supply lines is illustrated by a fatal accident which occurred in Jullundur city. While flying a kite with the ordinary string, a boy happened to touch a live electric wire with his kite, and was burnt badly and rendered unconscious. He succumbed eventually to his injuries. On the day of the occurrence, the ground was wet with rain and the string appears to have been moistened by contact with it.[2]

Suicide by electric currents is rare, but a few cases have been reported. A man,[3] named Paul Thiebault, with a view to commit suicide, deliberately took hold of the electric conductors at the works of M. Chertemps in Paris, and met with an instantaneous death. A case[4] is recorded where a young man committed suicide by attaching to himself an electric installation, operating a potential of 200 volts, and timed to make contact during his sleep.

Homicide by electricity, though extremely rare, is quite possible. In January, 1927, certain colliery proprietors of Cardiff were charged with manslaughter of a collier, who was electrocuted during a ratting expedition. It was alleged that a copper wire in the fence was electrified from the powerhouse to protect the coal bunkers.[5]

1. *Lancet, April* 14, 1928, *p.* 756.
2. *Times of India, Feb.* 12, 1936.
3. *Brit. Med. Jour., March* 14, 1885, *p.* 550.
4. *M. Critchley, Brit. Med. Jour., Jan.* 13, 1934, *p.* 71.
5. *Sydney Smith, Forens. Med., Ed. VI, p.* 236.

CHAPTER X

MECHANICAL INJURIES

For medico-legal purposes mechanical injuries are divided into contusions or bruises, abrasions and wounds.

CONTUSIONS

Contusions or bruises are injuries which are caused by a blow with a blunt weapon, such as a club (*lathi*), iron bar, stone, ball, fist, etc., or by a fall, or by crushing or compression. These are accompanied by a painful swelling and crushing or tearing of the subcutaneous tissues, without solution of continuity of the skin. The swelling is due to the rupture of the subcutaneous blood vessels producing in the cellular tissues an extravasation of blood, which is known as ecchymosis or effusion of blood.

Ecchymosis makes its appearance over the seat of injury in one or two hours after the injury. It may appear even in less time if the skin injured is very thin, as in the eyelids and scrotum. When ecchymosis has occurred into the deeper tissues or under tense fasciæ, it appears on the surface at an interval of one or two days or even more at some distance from the seat of injury following the line of least resistance and in obedience to the law of gravity; *e.g.*, the appearance of a black eye in the case of a contusion on the forehead or on the head. Sometimes, ecchymosis may not appear until after death, when a contusion has been caused within a few hours or a day or two before death. According to Sir Bernard H. Spilsbury[1] this is not due to any appreciable addition to the blood in the contused area after death, but to a more rapid hæmolysis of the stagnant blood as a part of post-mortem changes; there is no circulation to carry away the pigment and the tissues are dead and cannot deal with it. The pigment diffuses locally, producing a stain in the surface, dark red at first, but changing sometimes to a bright red colour from absorption of oxygen through the skin; or an area of a dark green putrefactive discoloration appears over a deep bruise before the skin around it is changed.

The extent of ecchymosis depends, under ordinary circumstances, upon the nature and severity of the force used, the vascularity of the part struck, looseness of the underlying cellular tissue and the condition of the assaulted victim. Thus, it will be extensive in the eyelids, scrotum and vulva and very little in the scalp where the skin is tense. Again, it may not appear in the abdomen even if a cart-wheel were to pass over the body and cause death from the rupture of an internal organ. No evidence of ecchymosis is present if the weapon used is a yielding one, such as a sand-bag.

---

1. *Lancet, Feb.* 28, 1925, *p.* 421.

Ecchymosis is easily produced in children, flabby women and old people even by slight violence; on the other hand, it will be very slight if a person happens to be strong and muscular.

In certain pathological conditions, such as scurvy, purpura, erythema nodosum, hæmophilia, malignant cases of infectious diseases, rashes due to the continuous use of drugs, and in the aged with sluggish circulation,

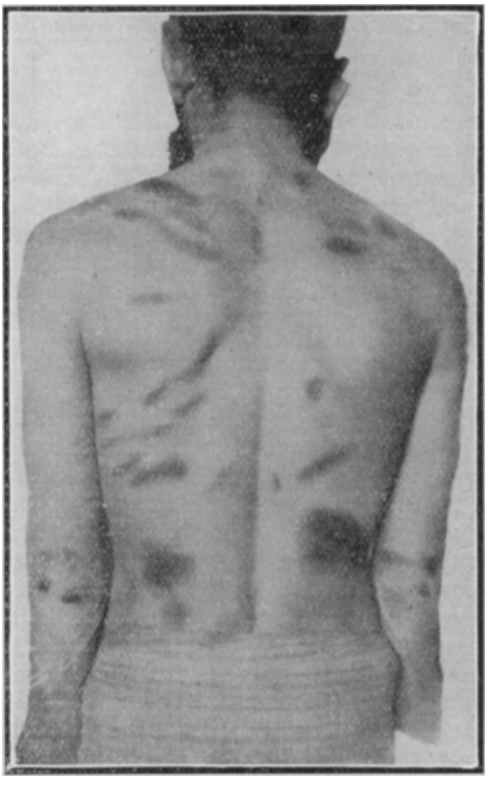
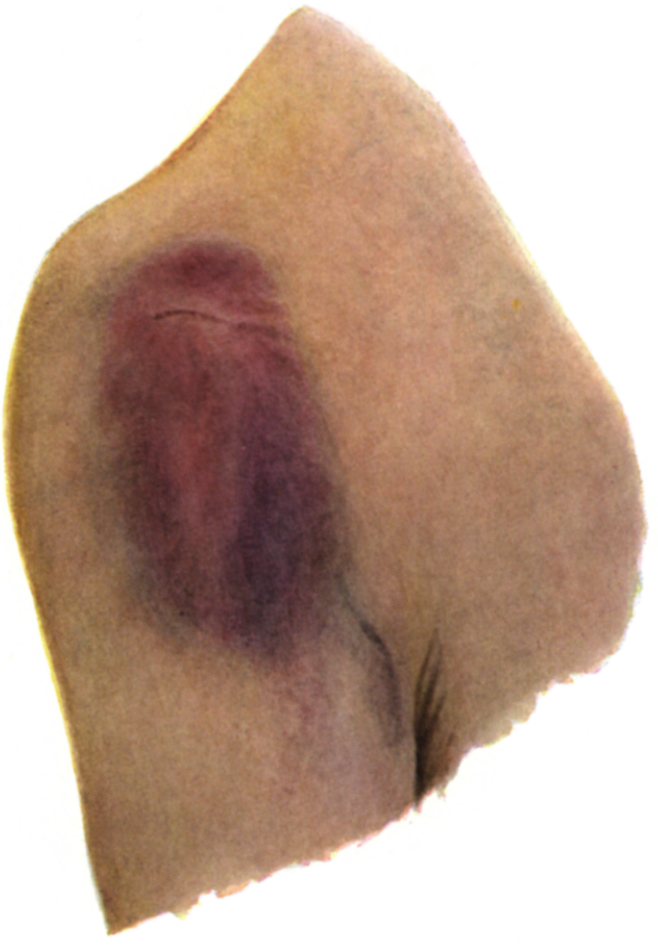


Fig. 59.—Contusions caused by blows with a blunt weapon (stick).

a slight blow or pressure may produce an extensive ecchymosis. In such cases subcutaneous hæmorrhages may occur spontaneously and may be mistaken for ecchymosis, but they can be easily distinguished from their number, size, and symmetrical situation (generally on the legs), and from the absence of abrasions over the spots. Subcutaneous hæmorrhages may also result from great muscular exertion as in epileptic seizures. These are usually numerous, but smaller in size.

Subconjunctival ecchymoses due to the rupture of small vessels may occur directly from a blow to the eye or indirectly from a blow or fall on the head. They are often seen in children suffering from whooping cough, and may, sometimes, result from severe straining during sneezing, coughing, vomiting or lifting heavy weights, especially in old people.

Plate III.



Bruise on shoulder showing colour changes after four days.

[*To face page 219*].

Sometimes, blebs and bullæ may form over the injured part, especially when ecchymosis is caused by a fracture or by an oblique and glancing blow.

The Result of Bruises.—Contusions are, as a rule, simple injuries. They are seldom fatal unless accompanied by rupture of an internal organ, or by extensive crushing of the tissues and large extravasations of blood, producing sloughing and gangrene of the parts. However, several bruises, though trivial individually, may cause death from shock.

In June, 1910, Musammat Bullo, 13 years old, was beaten to death by her husband and father-in-law for neglecting the household duties. The post-mortem examination showed that death occurred from shock due to twenty-nine simple bruises inflicted on various parts of the body.

The Age of a Bruise.—The age of a bruise may be ascertained from the colour changes which its ecchymosis undergoes during absorption. These colour changes are due to the disintegration of the red blood cells and staining of the hæmoglobin thus set free. They commence at the periphery and extend inwards to the centre. They are red at first, but during the next three days they appear blue, bluish-black, brown or livid red, and become greenish from the fifth to the sixth day, and yellow from the seventh to the twelfth day. This yellow colour slowly fades in tint till the fourteenth or fifteenth day, when the skin regains its normal appearance. Moreover, its disappearance is more rapid in healthy persons than in sickly and old people with feeble circulation. It also depends on the nature of the violence used. An ecchymosis caused by slight force will disappear in about a week or two, while an extensive one caused by considerable force will disappear in about three to four weeks. It must be remembered that the colour changes are not seen so well on dark skins as on fair skins.

Ecchymoses situated in the deeper tissues do not exhibit any gradations of superficial colour changes during their absorption. Subconjuctival ecchymoses do not undergo the usual colour changes; they are at first bright red, and then become yellow in colour before they disappear.

Difference between Accidental, Homicidal and Self-inflicted Bruises.—The usual question that a defence pleader puts to a medical witness in the case of bruises is whether they were caused accidentally by a fall or homicidally by mechanical force. The reply to this question is not easy in all cases; however, the position and arrangement of the bruises may help the witness to give a definite reply. In the case of a fall, he should look for the evidence of sand, gravel or mud on the body. Again, the shape and size of a bruise generally correspond to the weapon used in inflicting the injury. Thus, a bruise caused by a blow from a fist or butt end of a club (*lathi*) is usually rounded in appearance. A bruise inflicted by the length of a club or stick is, as a rule, elongated and irregular. A soft cane or whip usually produces two parallel bruises with an intervening space almost equal to the diameter of the weapon. A bruise caused by a whip may also encircle a limb or part of the body, and may present an abraded surface at the end.

Bruises caused by a blunt weapon are not, as a rule, self-inflicted. During my long practice of twenty-eight years as a medico-legal officer I have not come across a single case of this nature. But, with a view to

support a false charge of assault, bruises are, sometimes, simulated by the application of some irritant substance, such as the juice of *Bhilawa* (marking nut) or the root of *Chitra* (plumbago zeylanica) or *Lal Chitra* (plumbago rosea). The marks produced by these substances appear like

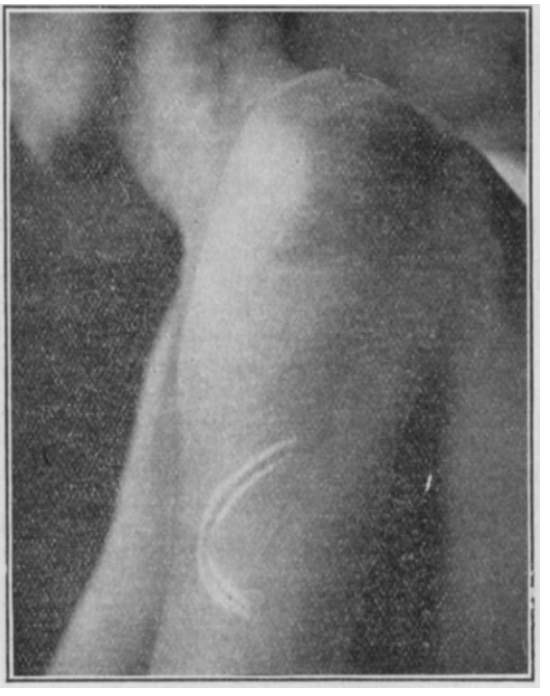


Fig. 60.—Bruise caused by a shoe heel.

bruises, but they are dark-brown in colour with the margins usually covered with tiny vesicles, and the surrounding skin is red and inflamed. The scrapings of the marks, if recent, will respond to the tests of the substance used. Owing to the irritation caused by the application of these substances it is very difficult to avoid scratching the part with the fingers; hence similar marks are usually found on the tips of the fingers and under the free edges of the finger-nails.

In November, 1926, a woman complained that she was beaten with a club. On examination I found four marks of dark-brown pigmentation, varying from 1" to 4" by 1" to 3", obliquely across the back and outer side of the left thigh in its lower half. The skin around the marks was red and inflamed, and the edges were covered with tiny vesicles. The tips of the fingers showed similar marks of dark-brown pigmentation. The scraping of the marks on the thigh and fingers gave the chemical tests of the juice of marking nut.

In October, 1932, a woman was sent to me by the City Magistrate of Lucknow with a report that she had been beaten with a cane. She had two vertical dark-brown patches along the upper part of the left shoulderblade towards its inner border. These were almost parallel with an intervening space of half an inch. Their margins were wavy. The first one of them was $2\frac{1}{2}" \times \frac{1}{4}"$, and the other was $2" \times \frac{1}{2}"$. She had also a similar patch, $2" \times \frac{3}{4}"$, along the back of the left forearm

towards its outer side and 1 1/3" below the left elbow. All these patches appeared to have been caused by sulphuric acid.

Dutt[1] also reports cases in which a rectangular shiny patch produced by caustic on the chest was claimed to be the result of a blow, and the mark left by a heated rupee on the back was attributed to a blow from a *lathi* (club).

Difference between an Ante-mortem and Post-mortem Bruise.— A certain amount of swelling and the colour changes are found in a bruise caused during life. There is usually coagulation of the effused blood into the subcutaneous tissues and infiltration of the blood in the muscle fibres. These signs are absent in a bruise caused after death. A bruise is likely to be disfigured by putrefaction, and it is difficult to differentiate between

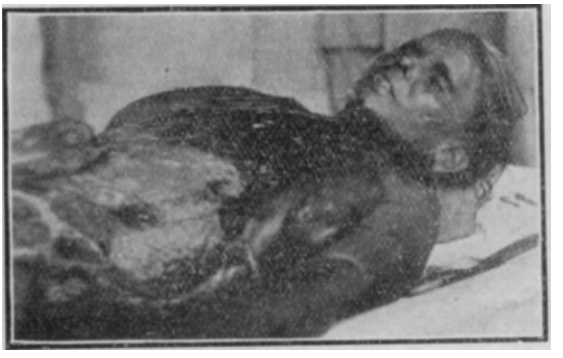


Fig. 61.—Effused, coagulated blood in the subcutaneous tissues of the chest due to ante-mortem contusion.

a bruise caused during life and that caused immediately after death. Sir Robert Christison proved by experiments that it was possible to produce a bruise within two hours to three hours and a quarter after death which it would be difficult to distinguish from one caused during life ; but he found that very great violence had to be used and even then the resulting bruise was much smaller than what would have been produced by similar means during life. However, Sir Bernard H. Spilsbury[2] has pointed out that two minutes after death no appreciable bruising occurs, inasmuch as the development of a bruise depends upon the maintenance of the circulation, which slows down owing to the fall of the arterial blood, and is soon completely arrested as soon as the heart's action is stopped in death.

ABRASIONS

Abrasions are injuries involving the loss of the superficial layers of the skin, and are produced by a blow or a fall on a rough surface, by scratching with the finger-nails or by teeth-bites. They are of very little significance from a surgeon's point of view, but are of great importance from a medico-legal point of view.

1. *Ind. Med. Gaz., May,* 1927, *p.* 296.
2. *Lancet, Feb.* 28, 1925, *p.* 421.

Abrasions resulting from friction with a rough surface during a fall are mostly found on bony parts, and are usually associated with contusions

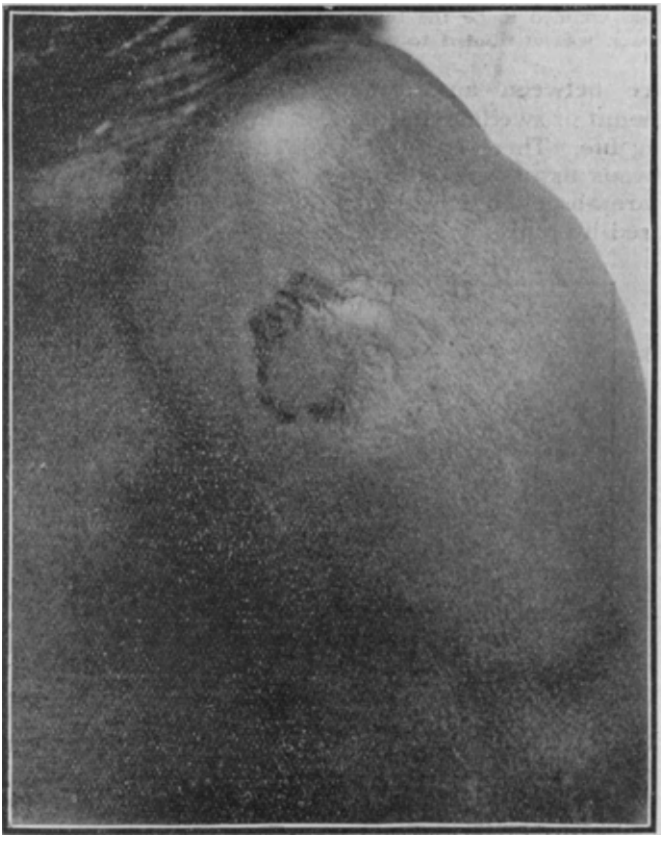


Fig. 62.—Abrasions on arm caused by teeth-bite.

or lacerated wounds and sometimes with very serious injuries. The abrasions may also be covered with mud, straw, etc.

Abrasions caused by the finger-nails indicate a struggle and an assault, and are usually seen on the exposed parts of the body, such as the face, neck, fore-arms, hands, etc. They may be crescentic in shape, especially if the finger-nails have been pressed with violence into the skin. In such cases there will be an ecchymosis in the underlying tissues.

Abrasions caused by teeth-bite are elliptical or circular in form, and are represented by two or four separate marks caused by the upper front teeth on one side and the same or less number of marks by the lower front teeth on the opposite side. The intervening space between the marks is often bruised. Sometimes, the marks coalesce together, and form a single mass of abrasions.

Difference between Ante-mortem and Post-mortem Abrasions.—Abrasions caused during life appear as bleeding surfaces or scratches, and are soon covered with reddish-brown crusts or scabs owing to coagulation of the blood. They generally heal in about ten to fourteen days without leaving permanent scars, but in cases where the abrasions involve the whole thickness of the skin and destroy the epithelial cells capable of forming a new skin, they take a longer time to heal, and leave obvious scars unless the surgeon has performed an operation of grafting.

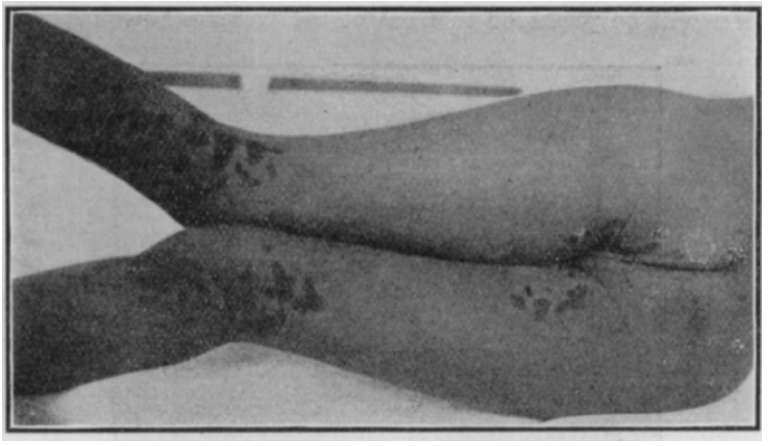


Fig. 63.—Marks simulating abrasions caused by ant-bites after death.

Owing to the drying and hardening of the underlying skin abrasions produced after death are dark-brown and parchment-like in appearance, and look like abrasions caused during life, but they are distinguished by complete absence of bleeding and injection of vessels in the underlying tissues. It must be remembered that ants sometimes attack a dead body lying on the ground, and produce marks which simulate ante-mortem abrasions. The marks caused by their bites have, however, irregular margins, and are usually seen on the eyes, nostrils, angles of the mouth, ears, armpits, groins, scrotum and anus.

WOUNDS

A wound is defined as the forcible solution of continuity of the soft tissues of the body including the skin or mucous membrane. Medico-legally, wounds may be classified as—

1. Incised wounds.
2. Punctured wounds.
3. Lacerated wounds.
4. Gunshot wounds.

1. **Incised Wounds.**—An incised wound is produced by a sharp, cutting instrument, such as a knife, razor, sword, *gandasa* (chopper), axe, hatchet, scythe, *kookri,* or any object which has a sharp, cutting edge.

Character of an Incised Wound.—An incised wound is always broader than the edge of the weapon causing it owing to the retraction of the divided tissues. It is somewhat spindle shaped and gaping, its superficial extent being greater than its depth. This gaping is greater in deep wounds when the muscle fibres have been cut transversely or obliquely. Its edges or margins are smooth, even, clean-cut, well-defined and usually everted. The edges may be inverted, if a thin layer of muscular fibres is closely united to the skin, as in the scrotum. They may be irregular in cases where the skin is loose or the cutting edge of the weapon is blunt, as the skin will be puckered in front of the weapon before it is divided.

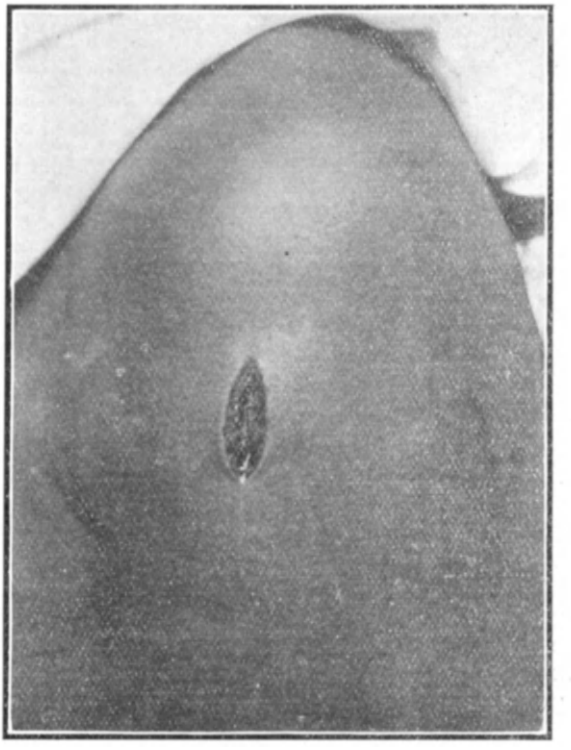


Fig. 64.—Incised wound inflicted by a knife.

The edges of a wound made by a heavy cutting weapon, such as an axe, hatchet or shovel, may not be as smooth as those of a wound caused by a light cutting weapon, such as a knife, razor, etc., and may show signs of contusion. Such a wound is, as a rule, associated with extensive injuries to deep underlying structures or organs.

While describing an incised wound it is always necessary to note its direction. The commencement of the wound is deeper, and it gradually becomes shallower and tails off towards the end, but no direction is noticeable when the weapon has not been drawn while inflicting a wound.

Hæmorrhage in the case of incised wounds is usually much more than in the case of other wounds, and it may be so severe as to cause death, especially if a main artery has been cut.

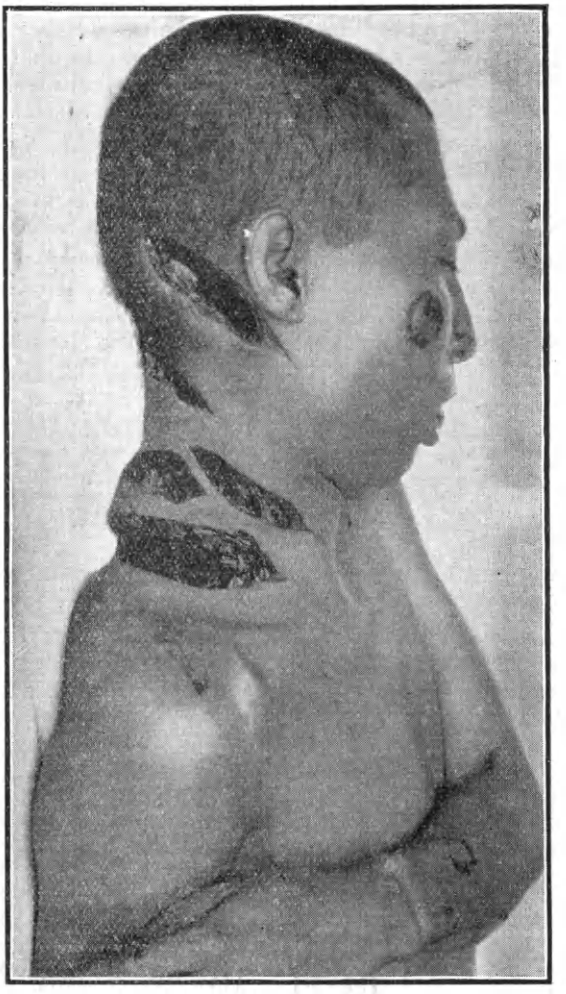


Fig. 65.—Incised wounds inflicted by a butcher's knife.
Note also teeth bites on face and forearm.

2. **Punctured Wounds.**—These are popularly called stabs and are termed penetrating wounds when, passing through the tissues, they enter a cavity of the body. These wounds are produced by a piercing or stabbing instrument, such as a knife, scissors, bayonet, spear, dagger, pick-axe, arrow, needle, pin, etc. The point of the instrument may be sharp or blunt.

A punctured wound caused by a sharp-pointed and cutting instrument has clean-cut margins, which are almost parallel but slightly curved to each other and have sharp angles at the two extremities. This is

commonly the case if the instrument has two cutting edges, and may be so with an instrument having one cutting and one blunt edge. The wound is generally wedge-shaped, if it is produced by an instrument with a thick, broad back and only one cutting edge.

A sharp-pointed and cylindrical or conical instrument produces a wound having a slit-like opening. A blunt-pointed instrument requires considerable force to puncture the skin and penetrate the soft tissues. It causes a punctured wound with lacerated margins.

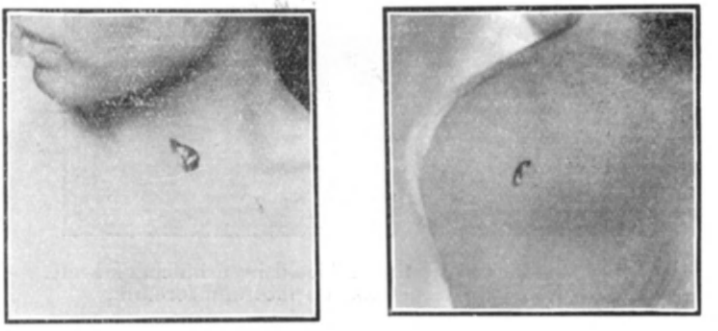
The aperture of a punctured wound in the skin is usually a little smaller in length than the breadth of the weapon used owing to the elasticity of the skin, although it is, sometimes, larger, as the weapon enlarges the wound, if it is withdrawn by lateral movements.

The depth of a punctured wound is much larger than its length or width, and may be equal to, or less than, the length of the blade of the instrument causing it. In some cases the depth may even be greater than the length of the blade owing to the fact that the force of the blow may depress the tissues of the part struck, allowing the point to reach the deeper tissues.

On February 21, 1923, a cobbler killed a Hindu Fakir, aged 45 years, by stabbing him on the chest with an awl, 8 inches long. On examination of the body two days later I found among other injuries a punctured wound in the post-axillary line on the left side of the chest which, passing through the substance of the lower lobe of the left lung, had traversed the left chamber of the heart 2 inches above its apex.

Great care should be taken in probing a punctured wound. If necessary, a blunt probe or catheter should be used.

External hæmorrhage is not necessarily any criterion of the danger to life. There may be very little external hæmorrhage and yet profuse hæmorrhage may take place internally owing to some vital organ having been penetrated.



A B

Fig. 66.—Punctured wound perforating the chest caused by a dagger.
A—Wound of entrance. B—Wound of exit.

In the case of a punctured wound perforating a part of the body there are two wounds, one a wound of entry and the other, a wound of exit. The wound of entry is usually larger with inverted margins and the wound of exit is smaller and has everted margins. The margins of the entrance wound may be found everted when the weapon used is rough and rusty.

In some cases two or more punctures may be found in the soft parts with only one external orifice. This shows that the instrument had been partially withdrawn after it pierced the tissues, and thrust again in a new direction.

Sometimes, it is argued that a punctured wound may have been caused by a fall on a sharp pointed piece of an earthenware pot or broken glass. In that case the edges of the wound are irregular and more or less bruised, and fragments of such articles may be found embedded in the soft tissues.

3. **Lacerated Wounds.**—These are produced by blows with blunt objects and missiles, by violent falls on sharp and hard projecting surfaces, by machinery and railway accidents, by the wheels of a vehicle, by the claws, teeth or horns of animals and by projecting nails. These wounds do not generally correspond in shape or size to the weapon producing them. Their margins are torn, jagged, irregular and swollen or contused. The tissues are torn and the skin beyond the seat of injury is ecchymosed and the bones which are near the surface are likely to be fractured. Foreign bodies, such as earth, grease, machine oil, cinders, hair, fibres of clothing, etc., are frequently found in the wounds.

When produced by a blunt weapon, such as a club (*lathi*), crowbar, stone, brick, etc., the lacerated wound is usually accompanied by a considerable amount of bruising of the surrounding and underlying tissues, and has inverted and irregular margins.

Fig. 67.—Lacerated wound caused by the leg being crushed under a cart wheel.

Hæmorrhage in lacerated wounds is, as a rule, not extensive owing to the fact that the arteries are not cut evenly, but are torn across irregularly so as to facilitate clotting of the blood. But in lacerated wounds of the scalp the temporal arteries often spurt as freely and forcefully as when cut cleanly. These arteries being firmly bound are unable to contract and. may, therefore, spurt and continue to bleed for a long time.

In a quarrel with her husband a woman sustained several injuries on her face and head. One of these was a lacerated wound on the right temple. Blood stains were found on the ceiling at a distance of four feet from her bed. These were caused

by the spurting of the divided right anterior temporal artery. A young man had been struck on the right temple causing a lacerated wound. Blood spurted to a distance of three feet and a quarter from the place where he was standing at the time of the assault.[1]

Occasionally, wounds produced by a blunt weapon or by a fall may look like incised wounds when inflicted on tense structures covering the bones, such as the scalp, eyebrow, etc., or by a fall on the knee or elbow when the limb is flexed. But the margins of such wounds will be found irregular with a certain amount of bruising, and small strands of tissue may be seen at the bottom bridging across the margins, if examined with a lens. In the case of wounds of the scalp the hairbulbs will be found crushed if they are inflicted with a blunt weapon, but will be found cut if produced by a cutting weapon. Similarly, wounds produced by pieces of glass, broken crockery or sharp edges of stone have the characteristics of incised wounds, but the margins are found irregular, inverted and contused if examined carefully with a lens.

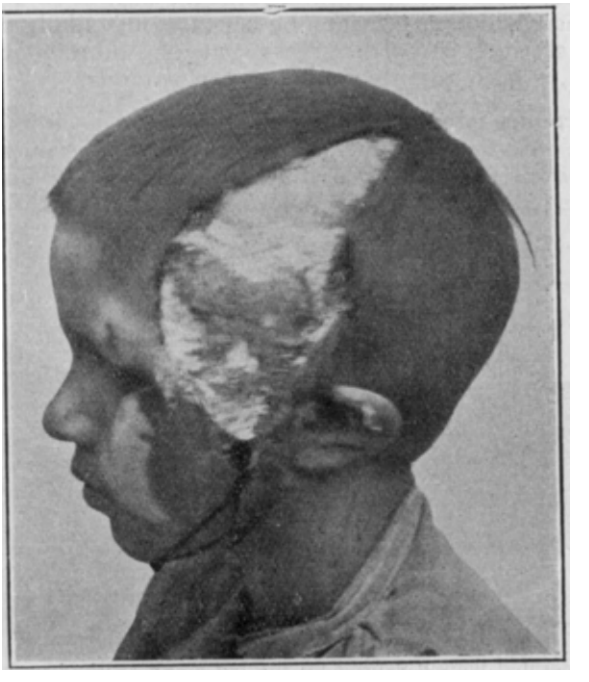


Fig. 68.—Lacerated wound of scalp caused by an *ekka* wheel.

4. **Gunshot Wounds.**—These are injuries produced by projectiles discharged from firearms, and present the characteristics of lacerated wounds, but their appearances vary according to the nature of the projectile, the velocity at which it was travelling at the moment of impact, the distance of the firearm from the body at the moment of discharge and the angle at which it struck the part of the body.

1. *Peterson, Haines and Webster, Leg. Med. and Toxic., Ed. II, Vol. I, p.* 294.

Gunshot wounds generally produce two wounds or apertures, *viz.*, one of entrance and the other of exit of the projectile. When the wound of entrance is present, but not the wound of exit, it means that a bullet is lodged in the body, except in those cases where a bullet has been coughed up after entering the respiratory passages or lost in the stool after entering the intestinal tract and also where a hard bullet by coming in contact with a bone is so deflected as to pass out by the same orifice as it entered. If a bullet is lodged in the body it must be taken out if death has occurred, and must be forwarded to the Superintendent of Police in a sealed envelope containing its description in the medical officer's handwriting, as it forms

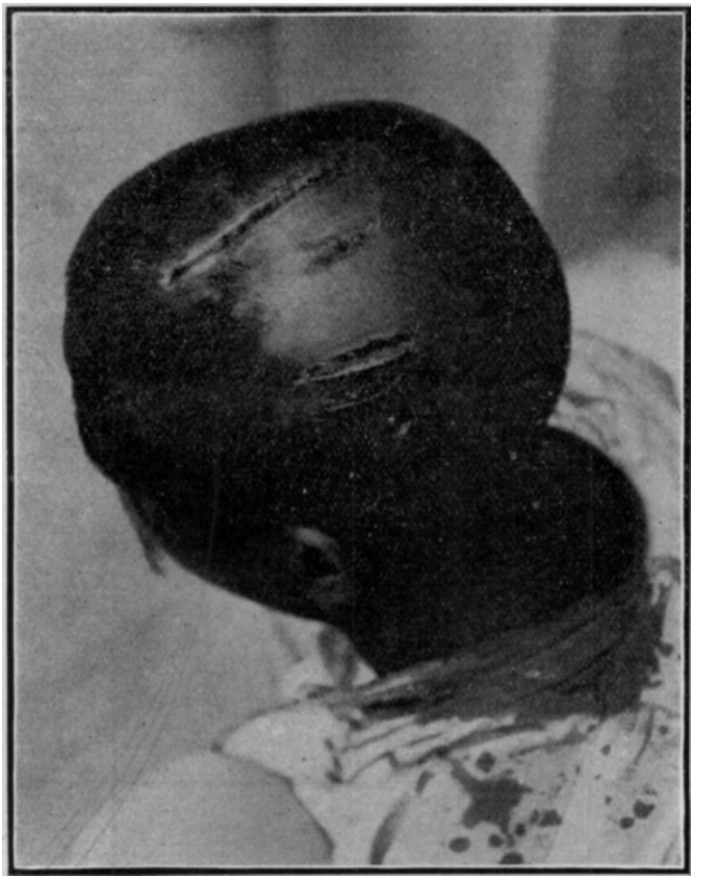


Fig. 69.—Wounds on the scalp inflicted by a blunt weapon (*lathi*).

inherent evidence of the greatest value. While searching for a bullet it must be borne in mind that it takes a very erratic and circuitous course while passing through the body.

In a case of suicide a bullet entered the mouth, and was found lodged under the left scapula after a good deal of dissection at the post-mortem examination. In another case, a man who was working in a field was shot in mistake for a black buck while in a squatting position. On post-mortem examination the bullet was found to have entered the outer side of the left-arm and come out at its inner side.

It again entered the body at the second left intercostal space and the left lung, passed out of it at its root, entered the right lung near its root, passed out at its base, and lodged itself in a flattened condition on the inner side of the right eighth rib causing its fracture.

In a case where death has not occurred, the bullet should be located by means of X-rays, if available.

The medical officer may be asked to determine whether the bullet found within the body of the victim was fired from the weapon alleged to have been used. It must be borne in mind that the interior of the barrel of a weapon is marked by a series of spiral grooves, which vary in number, depth, width and direction in weapons of different manufacturers. As the bullet passes through the barrel of a weapon, it receives on its surface impressions of these grooves, and also scratches, known as secondary marks, caused by any irregularities in the barrel. Hence it is possible to determine whether a particular bullet is fired from a particular weapon by comparing the impressions and secondary marks of the bullet to those produced on similar bullets fired from the alleged weapon for the purpose of an experiment. These marks offer the best evidence for the identification of weapons, inasmuch as " no two guns, whether of the same or different makes, have exactly the same marking, and the bullets fired from them will also vary." [1]

The wound of entrance is usually smaller than the projectile from the elasticity of the skin, and is rounded when the projectile strikes the body at right angles and oval when it strikes the body obliquely. The

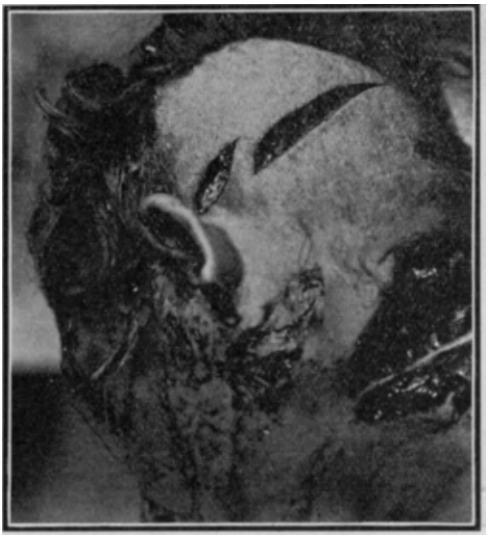


Fig. 70.—Wounds on scalp inflicted by a cutting weapon (*gandasa*). The skull bones have also been cut.

margins of the wound are inverted and ecchymosed. Wadding, pieces of clothing or other debris may be found lodged in the wound, and the

1. *Ralph W. Webster, Leg. Med. and Toxic.*, 1930, *p.* 155; *For further details* vide *Sydney Smith and Glaister. Recent Advances in Forens. Med.. Ed. II. Chapter II.*

skin surrounding it will be scorched and tattooed with particles of unconsumed gunpowder, if the firearm is discharged at close range. The wound of exit is often larger than the wound of entrance, and its margins are irregular and everted, but free from scorching and tattooing. The margins of both the wounds of entrance and exit may be everted in fatty persons due to protrusion of fat in the wounds, and in decomposed bodies from the expansible action of the gases of putrefaction. The margins of the wound of exit may be very ragged and torn, if the projectile was discharged at close quarters, had passed through the bone or was deformed by striking elsewhere at first (wound by recochet). These characters of the wound are due to the wobble of the projectile, to its deformed condition or to laceration of the skin by fragments of bone expelled from the body with the projectile or by the splintered pieces of the projectile itself.

The Nature of the Projectile.—Large bullets cause greater damage to the structures than small ones. Round bullets produce larger wounds

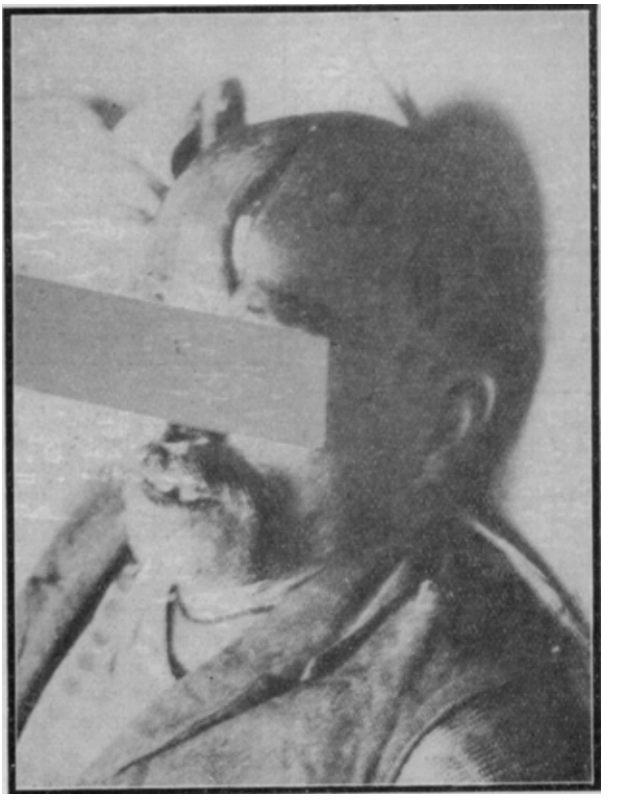


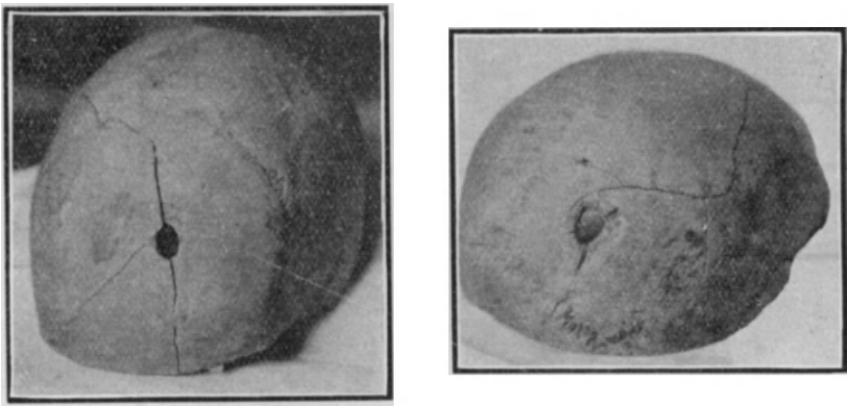
Fig. 71.—Wound on forehead caused by a fall from a height.

than conical. They cause extensive laceration of the tissues and comminuted fractures of the bones if they strike the body at a right angle; but their course is deflected if they strike the body at a different angle

and sometimes their course is arrested by coming in contact with buttons or other hard articles carried in the pocket. Berg[1] reports a case in which a metal trouser button was hit by a bullet, and while the bullet itself after hitting the button fell to the ground, the button was drawn into the abdomen.

Conical bullets produce much less laceration than round ones, and the wounds produced by them are punctured in appearance. Conical bullets rarely split in the tissues, though round ones often do.

Modern, steel-jacketed bullets used in army weapons have the shape of an elongated cone and owing to their great velocity usually pass straight and direct through the body without any deflection or deviation, and without causing much damage. The wounds of entry and exit are almost circular and similar in appearance without any bruising or laceration of the surrounding parts. Such wounds also heal very rapidly. Even the wounds caused by such bullets in the brain, lungs, or intestines often run a perfectly normal course, and heal without any difficulty.



A B

Fig. 72.—A. Entrance, B. Exit wounds of a revolver bullet in skull bones. The margins of the entrance wound are clean-cut, while those of the exit wound are bevelled externally.

Expanding, grooved, Dum-dum bullets are very destructive in character, and produce extensive wounds with ragged margins.

Fragments of shell are also destructive and cause extensive wounds.

Irregular missiles, such as pieces of stone, iron, *kankar*, beads of brass or nickel anklets or wristlets, seeds, etc., used in muzzle loading guns produce several irregular, lacerated wounds, and the exit wounds are larger than the entrance wounds. It is possible for a single pellet of shot to cause death.

In one case several *kankars* penetrated the lungs of an old woman who was shot with a muzzle loading gun. The woman died of gangrene of the lungs after three months in Lady Lyall Hospital at Agra. In another case, a woman, 50 years old, was shot by dacoits. There were three gunshot wounds on the right side of the chest, internal to the right nipple, and on dissection four small *kankars* were found embedded in the right lung and in the right ventricle of the heart. In a third case

1. *Med.-Leg. Jour., Vol.* 46, *Nos.* 3-4, 1929, *p.* 79.

an old man received a charge of shot in the left side of the chest from a gun fired by a dacoit, and died immediately. On dissection a pellet of shot was found lodged in the pericardial sac having traversed the right chamber of the heart in its lower part.

Wadding or gunpowder may cause frightful laceration and may produce death by penetrating the internal organs of the body even if a blank cartridge is discharged close to the body.

Velocity of the Projectile.—A bullet travelling at high velocity produces a clean, circular, punched-out aperture or slit as in a stabbing wound, and usually perforates the body. It is not deflected from its path by striking a bone, but may cause its comminution or splintering. On the other hand, a bullet of low velocity causes contusion and laceration of the margins of the wound of entrance. It is easily deflected and deformed by striking some hard object, and often lodges in the body. The track made by the bullet widens as it goes deeper. This is the reverse of a punctured wound.

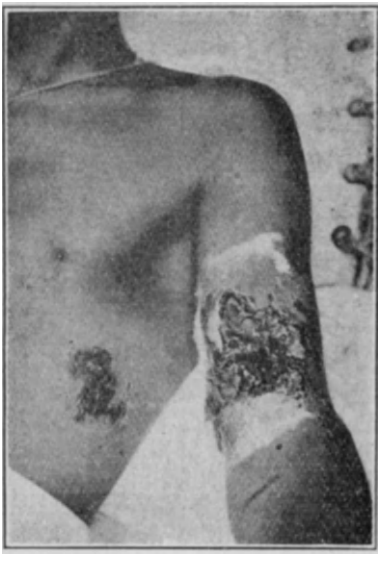


Fig. 73.—Gunshot wounds from close quarters.

Distance of the Firearm.—If a firearm is discharged very close to the body, or in actual contact, subcutaneous tissues over an area of two or three inches round the wound of entrance are lacerated and the surrounding skin is usually scorched and blackened by smoke and tattooed with unburnt grains of gunpowder. The adjacent hair is singed, and the clothes covering the part are burnt from the flame of the gas. If the powder is smokeless, there will be no blackening of the skin, but there may be a greyish or white deposit on the skin round the wound. No blackening or scorching is found if the firearm is discharged from a distance of more than four feet. Moreover, these signs may be absent even when the weapon is pressed tightly against the skin of the body, as the gases of the explosion and the flame, smoke and particles of gunpowder will all follow the track of the bullet in the body.[1]

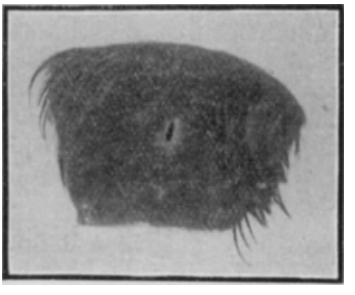


Fig. 74.—Wound of the scalp caused by the bullet of a rifle from a long distance.

1. *Spilsbury, Lancet, Feb.* 28, 1925, *p.* 421.

Stewart[1] reports a case of suicide by gunshot wounds in which there were two punctured wounds in the forehead, both measuring in diameter three-sixteenths of an inch. One was situated in the mid-line, and the other in the upper margin of the right eyebrow in a line with the outer angle of the right eye. There was no burning or scorching around either of the wounds, nor was there any singeing of the eyebrow. The wound in the middle of the forehead penetrated the whole depth of the tissues, and at its base, lying against the bone, was found a flattened bullet. There was no injury to the bone at this point. The other wound over the right eye penetrated the skull. The bullet had made its way diagonally across the brain and in a slightly downward direction. It was found in the brain substance at the tip of the left occipital bone. There was no injury to the base of the skull. The weapon with which the wounds were inflicted was a Marlin repeating rifle, calibre 0.22. He carried out experiments with the rifle on dead skin from a post-mortem examination. At a distance of 3 feet the edges of the wound were irregular. At the distance of 1/10" there was absence of scorching and singeing. In the original wounds the margins were regular and slightly inverted. There were marks which closely resembled tattooing. Section of these marks showed them to be sub-epithelial petechial hæmorrhages; there was no destruction of the squamous epithelium. Microscopic examination showed abundant deposit of unspent gunpowder in the deeper layers of the tissues although there was no tattooing around the wounds of the deceased.

The effects produced by small shot fired from a shot-gun vary according to the distance of the weapon from the body. A charge of small shot fired very close to, or within a few inches of, the body enters in one mass like a single bullet making a large irregular wound with scorched and contused edges, and is followed by the gases of the discharge which greatly lacerate and rupture the deeper tissues. Particles of unburnt powder expelled from the weapon behind the missile are driven to some distance through the wound, and some of them are found embedded in the wound and the surrounding skin which is also blackened by the smoke of combustion. At a distance of one to three feet small shot make a single aperture with irregular and lacerated margins corresponding in size to the bore of the muzzle of the gun, as the shot enter as one mass, but are scattered after entering the wound and cause great damage to the internal tissues. The skin surrounding the wound is blackened, scorched and tattooed with unconsumed grains of powder. On the other hand, at a distance of six feet the central aperture is surrounded by separate openings in an area of about two inches in diameter made by a few pellets of the shot which spread out before reaching the mark. The skin surrounding the aperture is not blackened or scorched, but is tattooed to some extent. At a distance of twelve feet the charge of shot spreads widely and enters the body as individual pellets producing separate openings in an area of five to eight inches in diameter, but without causing blackening, scorching or tattooing of the surrounding skin. This scattering of shot depends upon the size of the gun, the charge of the powder and the distance of the gun from the body.

In conclusion it must be noted that it is not easy to give a definite opinion about the distance from which a firearm was discharged. According to Taylor[2] no general rule can be laid down. Experiments must be done with the weapon and cartridges (or loading) similar to those which are alleged to have been used.

1. *Lancet, Dec.* 27, 1930, *p.* 1397.
2. *Princ. and Pract. of Med. Juris., Ed. IX, Vol. I, p.* 498.

The Time when a Weapon was fired.—Sometimes, a medical man is asked to find out when a particular weapon was fired. If he is not a sportsman and not conversant with different weapons, he should never hazard an opinion. But he should remember for the purpose of rough calculation that after recent discharge a black deposit of potassium sulphide

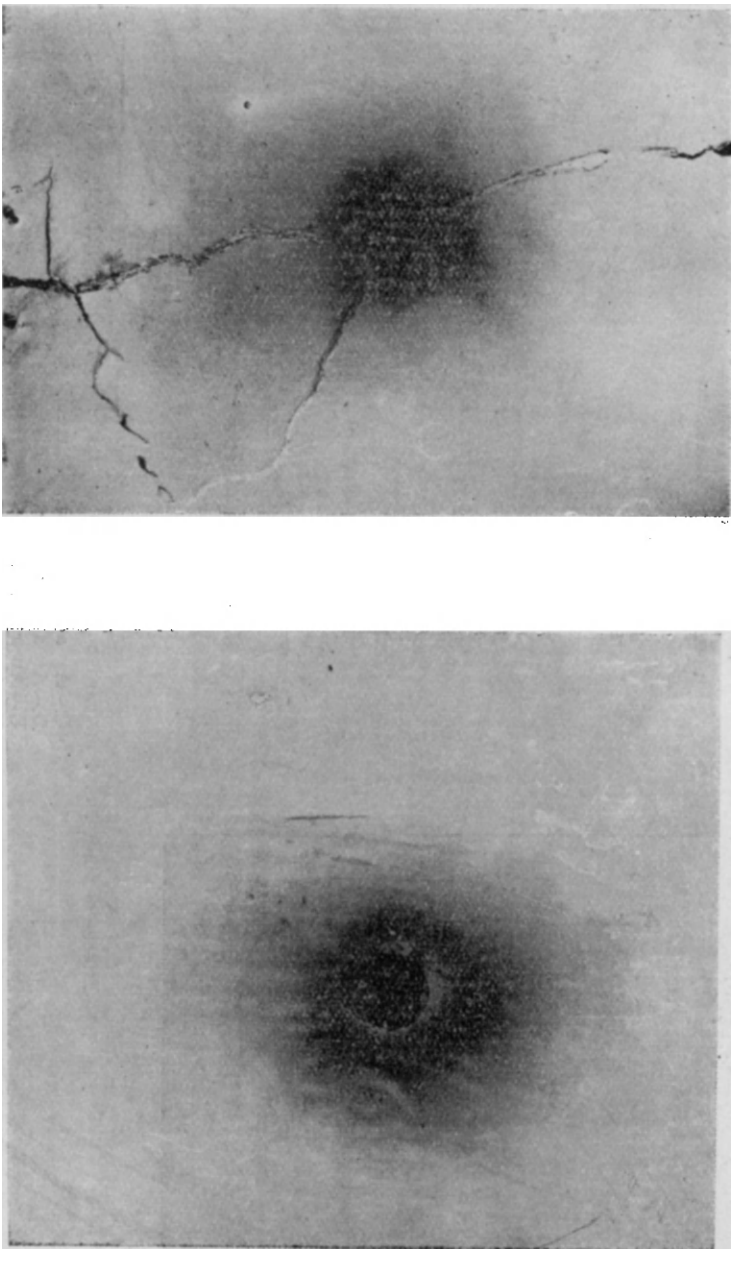
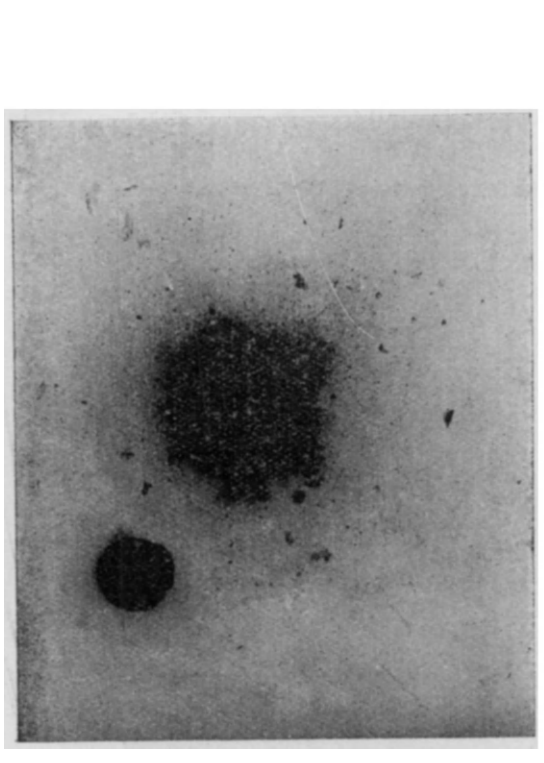
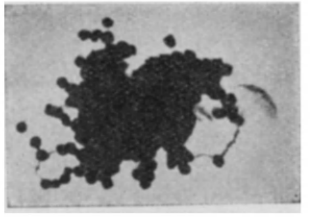


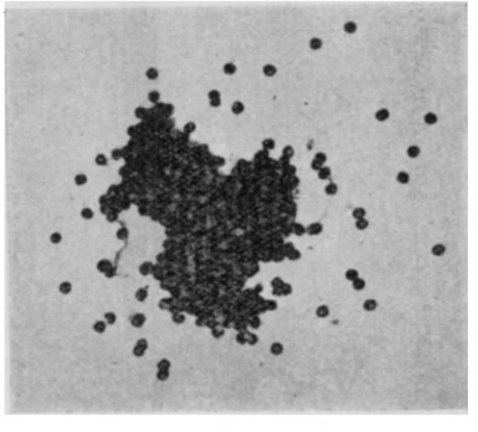
Fig. 75.—Effects of Shot No. 4 on card-board targets fired from a 12-bore shot-gun at varying distances.
A.—At a distance of 6 inches × ½.
B.—At a distance of 12 inches × ½.



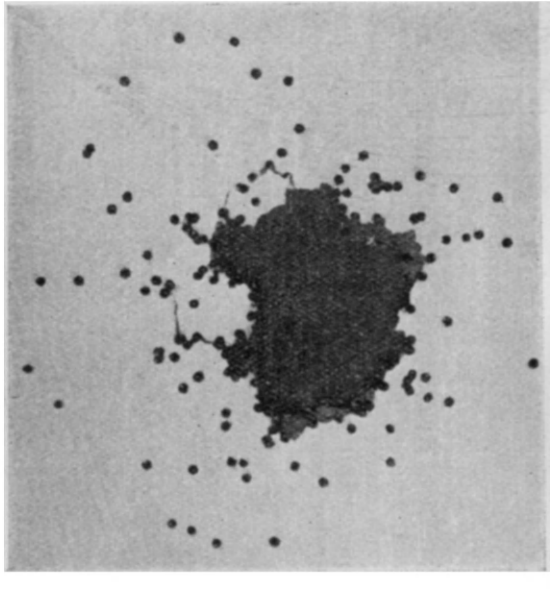
C



D



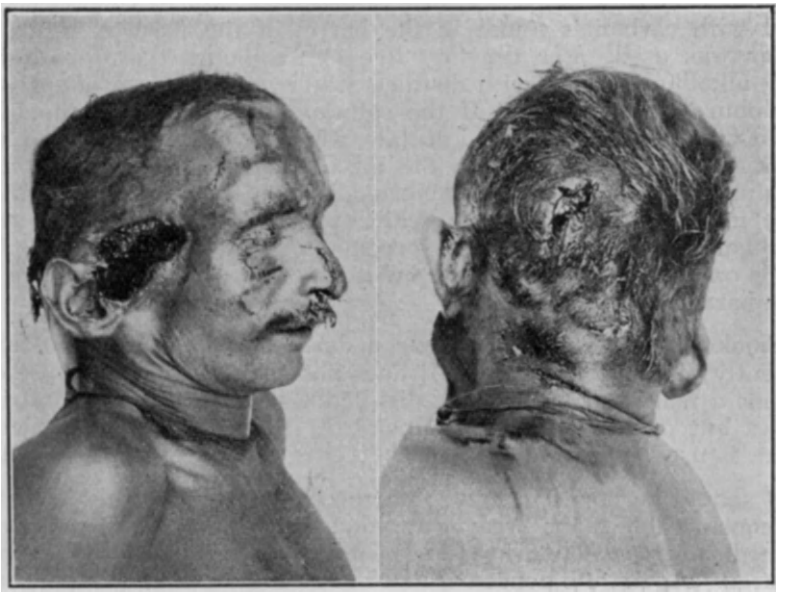
E



F

Fig. 76.—Effects of Shot No. 4 on card-board targets fired from a 12-bore shot-gun at varying distances.

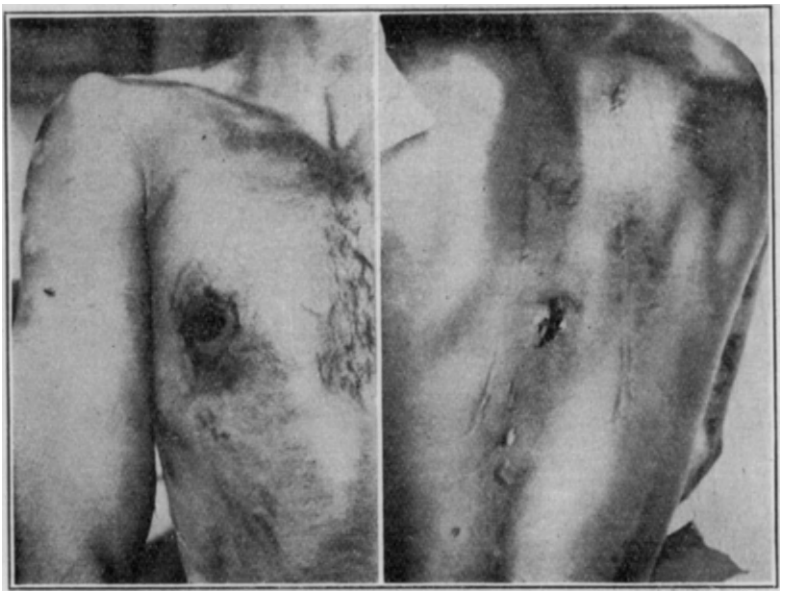
C.—At a distance of 3 feet × ½.
D.—At a distance of 6 feet × ½.
E.—At a distance of 9 feet × ½.
F.—At a distance of 12 feet × ½.



A B

Fig. 77.—Homicide by shooting with a gun.

A. Entrance-Wound showing the characters of a near discharge.
B. Exit-Wound.



A B

Fig. 78.—Suicide by shooting with a gun.

A. Entrance-Wound showing blackening and tattooing of the surrounding skin.
B. Exit-Wound.

mixed with carbon is found in the barrel of the firearm, if black gunpowder was used. For the first five or six hours this deposit forms a strong alkaline solution with distilled water and emits an offensive odour of sulphuretted hydrogen. If the solution is filtered, and the filtrate is treated with a solution of lead acetate, a black precipitate of lead sulphide is formed. After exposure to the air and moisture for a few days the potassium sulphide becomes converted into thiosulphate, thiocyanate and finally into potassium sulphate, which forms a neutral solution with distilled water and gives a white precipitate with lead acetate. At later periods oxides of iron (iron rust) with traces of iron sulphate are formed in the barrel.

Smokeless nitro-powders leave a dark grey deposit in the barrel of a recently discharged firearm. It does not change with the lapse of time. It forms a neutral solution with distilled water, and contains nitrites and nitrates but does not contain sulphides. If the chromate or bichromate powder is used, the residue in the barrel is usually of a greenish tint.[1]

It should be borne in mind that the composition of the deposit would vary considerably if the firearm was dirty at the time of its discharge, and the medical practitioner has no means to know its condition prior to discharge. Again, the deposit would not be found, if the weapon had been thoroughly cleansed after discharge.

Direction from which the Weapon was fired.—The question regarding the direction from which the weapon was fired may arise in a case where it is alleged that it was fired from a certain point in a quarrel. To ascertain this it is necessary to know the position of the victim at the time of the discharge of the bullet when a straight line drawn between the entrance and exit wounds and prolonged in front should indicate the line of direction. In some cases it is difficult to determine the direction as the bullet is so often deflected by the tissues that its course is very irregular.

1. *For further details of the examination of the weapon and the residue the reader is referred to Sydney Smith and Glaister's Recent Advances in Forensic Medicine, Ed. II, Chapts. III and IV.*

CHAPTER XI

THE MEDICO-LEGAL ASPECT OF WOUNDS

EXAMINATION OF THE INJURED PERSON

The medical officer is supplied by the Police Superintendent or the Magistrate with a printed form which he is required to fill up after examining the injured person (*Vide* Appendix IV). He should fill up this form very carefully. First of all he should write the name of the injured person and the name and number of the police constable accompanying him, and should note the mark or marks of identification to enable him to recognise the injured person in Court. He should then note the exact time of the examination, *viz.*, hour, date, month and year, and proceed with the examination proper as below :—

Nature of Injury.—While describing the injuries in columns 1, 2 and 3 of the form he should carefully note their nature and number, the character of their margins, their size as regards length, breadth and depth, the line of direction and their situation. If necessary, he should use a magnifying lens. All the injuries should be measured with a tape-measure, and the exact measurements in inches must be given ; they should never be guessed. While mentioning the exact situations a reference to some bony prominences or anatomical landmarks should be made, as for example, so many inches above or below the front or back of the left or right wrist, elbow, etc. In describing these points technical terms must be avoided as far as possible.

Wounds of the chest or abdomen ought not to be probed, lest they be converted into penetrating wounds ; but in doubtful cases, they may be enlarged, under proper precautions, to find out the condition of the underlying bone or organ.

Simple, Grievous or Dangerous Injury.—In column No. 4 it must be mentioned whether the injury is simple, grievous or dangerous to life.

A simple or slight injury is one which is neither extensive nor serious, and which heals rapidly without leaving any permanent deformity or disfiguration.

Grievous injuries as described in the foot-note of the form and in Section 320, I. P. C., are as follows :—

1. Emasculation. 2. Permanent privation of the sight of either eye. 3. Permanent privation of the hearing of either ear. 4. Privation of any member or joint. 5. Destruction or permanent impairing of the powers of any member or joint. 6. Permanent disfiguration of the head or face. 7. Fracture or dislocation of a bone or tooth. 8. Any hurt[1] which endangers life or which causes the sufferer to be, during the space of twenty days, in severe bodily pain, or unable to follow his ordinary pursuits.

---

1. *Hurt means bodily pain, disease or infirmity caused to any person* (vide *section* 319, *I. P. C., Appendix VII*).

It must be remembered that a mere stay in a hospital for twenty days does not constitute a grievous injury as some doctors and even lawyers are inclined to believe. It must be proved that during that period the injured man was in severe bodily pain or unable to follow his ordinary pursuits. An injured man may be quite capable of following his ordinary pursuits long before twenty days are over, and yet may prolong his stay in a hospital by interfering with the healing of his wound or for the sake of permanent recovery or greater ease or comfort may be willing to remain as a convalescent in a hospital, especially if he is fed at the public expense.[1] I remember a case in which a man who had received some bruises over his arms and back as a result of *lathi* blows stayed in a cottage-ward of a hospital for over a month, and yet it was held that the injuries were simple.

Danger to life should be imminent before the injuries are designated " dangerous to life ". Such injuries are extensive, and implicate important structures or organs, so that they may prove fatal in the absence of surgical aid. For instance, a compound fracture of the skull, a wound of a large artery, or rupture of some internal organ, such as the spleen, should be considered " dangerous to life ". But the injuries which prove fatal remotely by intercurrent diseases, such as tetanus, erysipelas, etc., should not be considered as dangerous.

If an opinion as regards the nature of a particular injury cannot be formed at the time of the examination, as in the case of an extensive swelling of a limb when its fracture cannot be detected, or in the case of a head injury where the symptoms are obscure, the injured person must be kept under observation until a definite opinion can be formed, and the police should be notified of the fact in police form No. 238 (Appendix IV).

The Kind of Weapon.—In the fifth column the kind of a weapon by which the injury was inflicted should be mentioned. This can be inferred from examining the injury, for example, the margins, ends and shape in the case of a wound, but sometimes it is difficult to give an opinion as to whether a particular injury, especially a contusion, or a lacerated wound, was caused by a blunt weapon or a fall. In that case it is better to give a guarded opinion, mentioning the possibility or probability, as the case may be. While forming an opinion the medical practitioner should not always depend on the statement of the injured person, which is often false. Again, as a precautionary measure it is better to mention the fact if he found that the injuries were such as could not have been caused in the manner suggested by the police or the injured person. This is important to avoid unnecessary cross-examination at the time of giving evidence in Court.

If a weapon alleged to have been used in producing the injuries is sent by the police it should be examined for marks of blood-stains or fragments of hair, etc., adherent to it and should be returned to the police after it is properly labelled and sealed. When any foreign body, such as a piece of broken glass, a splintered piece of a bamboo staff, a broken point of a cutting instrument or a pellet, bullet or wadding of a firearm, is found

1. *Vasta Chela,* (1894) 19 Bom. 247; *Ratanlal and Thakore, Law of Crimes, Ed. XIV, p.* 799.

lodged in a wound or in its surrounding tissue, it should be carefully preserved, and sent to the Superintendent of Police or Magistrate in a sealed packet containing its description with full particulars in the handwriting of the medical examiner. These articles help one materially in

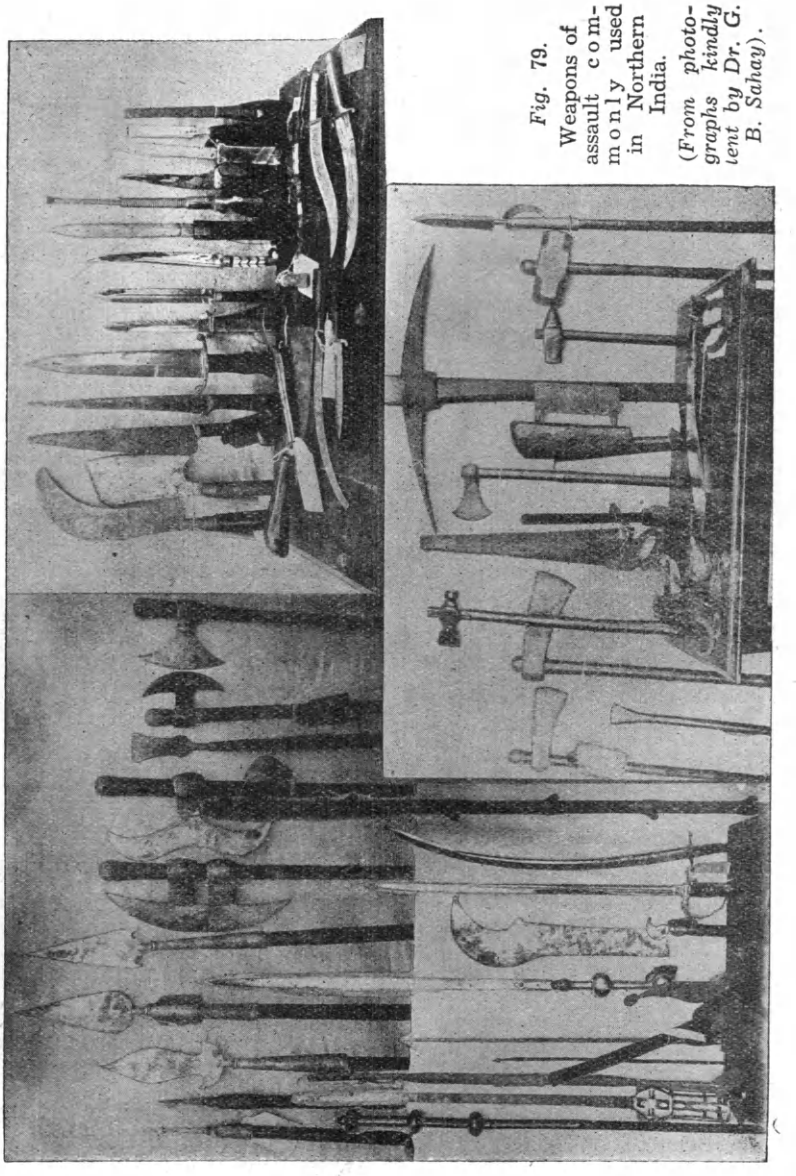


Fig. 79. Weapons of assault commonly used in Northern India. *(From photographs kindly sent by Dr. G. B. Sahay).*

judging as to whether a particular weapon had caused the alleged injuries or not. The clothes should also be examined for the presence of cuts, rents, tears or burns coinciding with the wounds on the underlying parts of the body, but these might not coincide with the wounds if the garment

worn at the time of the assault was very loose and was disarranged during the struggle. The clothes should then be properly marked, sealed and handed over to the police.

Dangerous Weapon.—The sixth column of the form refers to the description of the weapon as to whether it is dangerous or not. It need not be filled up, as a foot-note No. II is given at the bottom of the form which describes a dangerous weapon as any instrument for shooting, stabbing or cutting, or any instrument which, used as a weapon of offence, is likely to cause death.[1]

The Age of Injury.—In the column of remarks the age of the injury should be noted. It is frequently found that medical practitioners do not mention in their report the time when an injury was inflicted, but it is not fair to do so, inasmuch as the guilt or innocence of a person charged with criminal wounding or with robbery, burglary, or dacoity may be proved from the injury found on the body of his victim or on his own body, for its appearance may or may not correspond to the time when it is alleged to have been inflicted according to the prosecution theory. Moreover, it is also possible that all the injuries found on a person might not have been inflicted on the same day.

On July 9, 1931, I examined a Mahomedan woman alleged to have received certain injuries during a quarrel which took place four days ago. I found an incised wound, 1" by 1/6" by 1/6", over the crown of the head ¾" to the right of the middle line and 4" above the forehead. It was quite fresh and bleeding and appeared to have been self-inflicted on that very day. She had also a bruise, ¾" by ¼", on the palmar surface of the right middle finger below its second joint, and an abrasion with a dried scab, ½" by ¼", over the left shoulderblade towards its lower part. These injuries appeared to be about four days old. It appeared that she received only the latter two injuries during the alleged quarrel, but to make the offence more serious she herself inflicted the wound on her head on the day of the examination.

It is not easy to give the exact time of infliction of any injury, but an approximate time can be given from the data given below. Hence it is always necessary to mention "about" when giving the period of an injury.

Data to Ascertain the Age of Injury.—

1. The age of a contusion may be ascertained from the colour changes which its ecchymosis undergoes. These changes commence from eighteen to twenty-four hours after its infliction.

2. A superficial cut, such as that made with a razor during a shave, heals and scabs over in ten to twenty-four hours.

3. Vascular congestion and inflammatory swelling of a wound appear in less than forty-eight hours.

4. Pus appears in about thirty-six to forty-eight hours in a wound which has been dirty, or which has not been properly treated.

5. The margins of a small wound join together by the exudation of serum within forty-eight hours, when it commences to heal, and heals completely in four to seven days.

1. Vide *Sections* 324 *and* 326, *I. P. C., Appendix VII.*

6. In a wound which heals by the formation of granulation tissue, it generally appears after about a week.

7. A contused wound on the head, as ordinarily observed, heals in a week's time but, if the margins are lacerated very much it may take about ten to fourteen days, if properly treated, and if no pus has formed.

8. A wound may not heal for several days, or even weeks, if it has suppurated, especially if the injured person happens to be flabby or anæmic.

9. In the fracture of a bone the reparative process will enable one to fix its approximate time. The signs of inflammation and exudation of blood in the soft parts and round about the fractured ends are noticed from the first to the third day. Inflammation slowly subsides, and granulation tissue, known as the soft provisional callus, is formed from the third to the fourteenth day. This callus binds together the fractured ends of the bone. The formation of the amount of callus depends on the mobility or immobility of the fractured ends. It is less if the ends are immobile and impacted. It begins to ossify from the fourteenth day to the fifth week, and six to eight weeks is the average period taken by the callus to be absorbed completely, so that the fractured ends may be entirely united with the formation of bone.

The repair of fractures of the skull is usually attended with a very slight amount of callus, probably owing to the absolute rest of the fragments. The edges of a fissured fracture are usually glued together within a week, or gradually smoothed within three to four weeks, and are united by the formation of bone within two to three months or more. Bony formation does not occur in comminuted fractures, the line of fracture remaining permanently visible. Gaps left in the skull due to much loss of bone from injury or operation are filled with fibrous tissue. Infection interferes with the process of repair, and causes necrosis of the bone.

10. In the dislocation of a joint the time can be judged from the colour changes of a bruise which usually accompanies it, when caused by violence.

11. When a tooth has been knocked out, bleeding from its socket stops in about twenty-four hours, but sometimes on probing it the blood may come out even after two or three days. The cavity of the socket usually fills up in seven to ten days, and the alveolar process becomes quite smooth after fourteen days.

CAUSES OF DEATH FROM WOUNDS

Before discussing the causes of death it is necessary to point out that an assailant is not responsible for the death of his victim occurring on receiving an injury, if it can be proved from the post-mortem examination that it was due to natural causes, such as apoplexy, heart disease, phthisis or any other pathological condition or poisoning, and that the infliction of the injury did not operate in any way, immediately or remotely, to cause the death which might have occurred at the time even if the injury had not been inflicted. To substantiate a charge of murder or culpable homicide, it is, therefore, necessary to determine that the injury inflicted on the

deceased was actually the cause of death, and that it was such as was likely, or sufficient in the ordinary course of nature, to cause death (*Vide* Sections 299 and 300, I. P. C., Appendix VII). In such a case it is the duty of a medical officer to hold a thorough post-mortem examination and to examine the various organs and blood vessels for the presence of any morbid condition. It should be noted that a person can be convicted of culpable homicide, if he causes bodily injury to another who is labouring under a disorder, disease or bodily infirmity, and thereby accelerates the death of that other person (*Vide* Explanation 1, Section 299, I. P. C., Appendix VII). But the intention or the knowledge with which the act which caused death was committed is a necessary ingredient to bring about a conviction under these offences. In such cases even if the medical officer has proved from the post-mortem examination the existence of the disease which caused the death and its relation to the injury alleged to have accelerated it, the Court will have to be satisfied from the evidence before giving a decision (1) that the death at the time when it occurred was not caused solely by the disease, and (2) that it was caused by the bodily injury to this extent that it was accelerated by such injury.

On the 13th April, 1925, I was called upon to examine the body of one Sadiq Hussain, aged 45 years, who was alleged to have died from the injuries inflicted on him about seven days ago. Abrasions were found on the knees, elbows and the first kunckle of the right index finger. A small contused wound was found on the head and an incised wound, $\frac{1}{4}$" by $\frac{1}{4}$", along the back $1\frac{3}{4}$" to the right of the spine and 4" above the loin. Internally the right kidney was found lying in a pool of blood. It was enlarged, and was about three times the normal size. On section a sarcomatous growth was found round the kidney between its substance and the capsule. In the lower segment a cyst, about the size of a rupee, was found affecting the whole substance of the kidney and containing clotted blood. The hæmorrhage appeared to have taken place from the anterior surface of the kidney having given way and leading to the cyst. It was held that the hæmorrhage occurred from a fall due to the assault, and the accused was sentenced to rigorous imprisonment for eighteen months.

The causes of death from wounds are *immediate* or *direct* and *remote* or *indirect.*

Immediate or Direct Causes.—These are—

1. Hæmorrhage.
2. Injury of a vital organ.
3. Shock.

1. **Haemorrhage.**—This may be external or internal. External hæmorrhage may produce syncope and cause death either rapidly if a large blood vessel, such as the carotid or femoral artery, has been wounded or slowly if a number of small vessels has been injured. The amount of hæmorrhage required to cause syncope varies according to circumstances. A sudden loss of blood is more dangerous than the same quantity lost slowly. According to Watson[1] the loss of blood from five to eight pounds in adults is almost enough to end fatally, but children, women and old persons die from the loss of a much smaller quantity. Persons with hæmorrhagic diathesis or hæmophiliacs may die of hæmorrhage even from a trifling injury.

1. *Witthaus and Becker, Med. Juris., Vol. II, p.* 43.

Internal hæmorrhage may occur in penetrating and gunshot wounds. It need not be profuse for a fatal result; for a small quantity of hæmorrhage in the brain or into the pleural cavities or pericardium may prove rapidly fatal by disturbing the functions of the brain, lungs or heart from mechanical pressure on them. Blood flowing into the windpipe may cause death mechanically by asphyxia.

2. **Injury of a Vital Organ.**—Severe injury of a vital organ, such as crushing of the brain, heart, lungs, etc., is, as a rule, rapidly fatal.

When death occurs from a slight injury inflicted on a previously diseased organ, such as the rupture of an enlarged spleen, perforation of a chronic intestinal ulcer, or bursting of an aneurysm, etc., the assailant inflicting such an injury cannot be charged with culpable homicide, but he is convicted of simple or grievous hurt, if it be proved that his intention was not to kill his victim, that he could not possibly have known the existence of that disease and that the same injury could not have proved fatal when inflicted on an ordinarily healthy individual. In a case where a man received two or three simple injuries, one being on the head, was knocked down and died shortly afterwards on account of the rupture of the spleen which was enlarged owing to disease, it was held that where a person dies as the result of simple injuries owing to the fact that his spleen is diseased and it is not shown that the accused had the knowledge of this fact, he can only be convicted of causing simple hurt.[1] In another case where the accused went to a place where a cart was standing, and presuming that it belonged to a man who was sleeping on a cot close by, roused him and told him to let him have the cart. The man explained that the cart did not belong to him and remarked at the same time that he was ill. The accused, thereupon, got irritated and pulled the cot about, causing the man to fall out of it, kicked him and struck him on the side or on the ribs with a stick. Owing to the injuries he had received, the man died very soon after. It was held that as the deceased was suffering from a diseased spleen the accused was guilty of causing grievous hurt.[2]

3. **Shock.**—Death may occur from shock without any visible injury from paralysis of the heart by a blow on the cardiac region, or from the inhibitory action of the solar plexus caused by a blow on the pit of the stomach in the upper part of the abdomen.

Shock may be produced from exhaustion resulting from several injuries combined, though each one of them separately may be very slight. Shock may also result from fright due to vagus inhibition of the heart or from pain felt in flogging. Another instance of shock is concussion of the brain resulting from a severe blow on the head.

Shock usually appears immediately after receiving the injuries, but it may supervene after some time if the individual at the time of receiving injuries was in a state of great excitement and mental preoccupation.

Remote or Indirect Causes.—It is necessary to know the remote causes of death due to injury as the assailant, under the law of England, is responsible for the death of his victim if it occurs within a year and a day

1. *Criminal Law Journal*, 1923, *Vol.* 24, *p.* 427.
2. *Obrien*, (1880) 2 All. 766; *Ratanlal and Thakore, Law of Crimes, Ed. XIV, p.* 698.

after the infliction of the injury. But there is no such statutory limit in Indian law.

The remote causes of death operating secondarily from the injury are—

1. Inflammation in the internal organs, such as meningitis, cerebritis, pleurisy, pneumonia, peritonitis, etc.

2. Septic infection of a wound causing septicæmia, pyæmia or exhaustion from prolonged suppuration.

3. Gangrene or necrosis resulting from severe crushing of parts and tearing of the blood vessels.

4. Infective diseases, such as erysipelas and tetanus, which may develop through the entrance of the causal organisms through a wound. Erysipelas occurs from three to seven days, and is commonly associated with septic wounds of the scalp. It is more common in cold and temperate climates than in India and the tropics. In India, tetanus occurs usually from three to ten days after receipt of a wound or even an abrasion. It may occur within a few hours of receipt of the injury, but in temperate countries it usually manifests itself in two to three weeks. Cases are on record, where the disease developed from the twenty-sixth to the thirtieth day.[1]

5. Supervention of a disease from a traumatic lesion. For instance, a wound of the abdomen may, after healing, be followed by a strangulated hernia with fatal results. An injury affecting the lower portion of the spinal column or cord may cause paraplegia which may end fatally from septic cystitis or bed-sores and general exhaustion after an interval of some weeks or months.

6. **Neglect of the Injured Person.**—Death may occur from complications arising from a simple injury owing to the negligence of the injured person in its proper care and treatment. In this connection it may be mentioned that a person is not bound to submit himself to medical treatment for injuries received during an assault.

In all these cases an assailant is liable to be indicted for manslaughter[2] according to English law, if the cause of death is directly and definitely traceable to the injury, and the relation between cause and effect is not obscured by the action of concurrent causes. In a case where a wound, not in itself mortal, caused death from gangrene owing to neglect or want of proper applications it was held that the party by whom the wound was inflicted was guilty of murder. For, though the fever or gangrene, and not the wound, be the immediate cause of the death, yet the wound being the cause of gangrene or fever, is the immediate cause of the death, *causa causati*.[3] To justify a conviction of murder against an assailant in India it is necessary at the same time to prove that the act was committed with

1. *The Med. and Surg. History of the War, Part III, Surg.*, 1883.
2. *Unlawful killing of another without any malice, either expressed or implied, which act may be either brought about by sudden passion or by improper action of another.*
3. *I. Hale P. C.* 428; *Ryan*, (1868) 16 W. R. (Eng.), 319; *Ratanlal and Thakore, Law of Crimes, Ed. XIV, p.* 702.

the intention of causing such bodily injury as the offender knew that it was likely to cause death or was sufficient in the ordinary course of nature to cause death (*Vide* Section 300, I. P. C., Appendix VII). If these conditions are not fulfilled the assailant may be convicted under the offence of culpable homicide not amounting to murder or grievous hurt, or even simple hurt according to the circumstances of the case. The nature of a weapon used is also taken into consideration for deciding whether the crime falls under section 300 or any other section of the Indian Penal Code.

In November, 1928, I held a post-mortem examination on the body of one Must. Sumarta who died from starvation and pneumonia about 25 days after her throat was cut with a scythe (*hansia*). The accused, the uncle of the deceased, was found guilty of murder and sentenced to death. In a case where the accused savagely attacked and wounded their cousin with a hatchet, who was laid up with fever in consequence of the wounds for about 40 days and ultimately died of blood poisoning, it was held that the accused were guilty of murder, the wounds inflicted by them being the cause of death.[1] In another case the accused stabbed the deceased with a dagger in the back. The wound, though not severe, was in such a part of the body that it was considered dangerous; but the dagger did not penetrate to any great length. The wound healed in about seven days, at the end of which time symptoms indicative of tetanus were observed and the deceased died from that cause on the following day. It was held that there was no reasonable doubt that tetanus was due to the wound inflicted by the accused, who were found guilty of murder.[2]

Fazla, Mohamad Din and others made a murderous assault upon one Jalal which resulted in the fracture of a number of his ribs and other injuries. Jalal was removed to the hospital where he developed pneumonia and died after ten days. It was held that the perpetrators of the attack upon the deceased were guilty of murder.[3]

Seven persons made a deliberate attack on one man, and ultimately caused his death by fracturing his left ninth, tenth and eleventh ribs and rupturing his spleen and inflicting three injuries on his head which left him unconscious and which might, in the opinion of the medical officer, have also resulted in his death. They also inflicted twenty-four injuries on the deceased, about twenty of them being inflicted after the deceased had fallen down and was unconscious. It was held that the accused had no intention of committing wilful murder of the deceased, still the fact of the infliction of the injuries showed that they beat him with the intention of causing such bodily injuries as were likely to cause his death, or with the knowledge that they were likely by such acts to cause his death and that in the circumstances of this case all the accused were clearly guilty of the offence of culpable homicide punishable under section 304, I.P.C.[4]

An incised wound which by itself was not grievous or dangerous was inflicted on the right calf on the 22nd August, 1922. Tetanus set in on the 31st August, 1922, and this caused the death of the victim on the 3rd September, 1922. The assailant was sentenced to two years' rigorous imprisonment under section 324, I.P.C.[5]

7. **Result of a Surgical Operation.**—If death follows a surgical operation performed for the treatment of an injury, the assailant is responsible for the result, if it is proved that death was inevitable even without the operation, and that the operation was thought necessary and was performed by a competent surgeon with reasonable care and skill. It should be noted that the liability of the offender is, in no way, lessened even though life may have been preserved but for the refusal of the deceased

1. *Nuro*, (1913) 7 S. L. R. 83 ; 15, *Crim. Law Jour.*, 1914, *p.* 376 ; *Ibid.*, *p.* 716.
2. *Nga Dwe*, (1904) 10 Bur. L. R. 171 ; *Loc. Cit.*, *p.* 717.
3. *Crim. Law Jour., Aug.*, 1928, *Vol.* 29, *p.* 678.
4. *Oudh Chief Court, Cr. Appeal No.* 401 *of* 1934, *K. E.* v. *Sat Narain and others ; Cr. Law Jour.*, 1935, *Vol.* 36, *p.* 573.
5. *Crim. Law Jour.*, 1925, *Vol.* 26, *p.* 294.

to submit to a surgical operation (*Vide* Explanation 2, Section 299, I. P. C., Appendix VII). Similarly, where an injury is inflicted on a person by a blow, which in the judgment of competent medical practitioners renders an operation advisable, and as a preliminary to the operation chloroform is administered to the patient, who dies during its administration, and it is agreed that the patient would not have died but for its administration, the person causing the injury is liable to be indicted for manslaughter.[1] On the contrary, if the hurt or wound is not mortal, and if it is clearly proved that the death of the victim is caused by the application of unwholesome salves or medicines by himself or those about him, this cannot be regarded as homicide.

During a quarrel over grazing cattle one Sobha struck a blow over the head of his uncle and caused a wound over the top. The injury was thought to be simple but death occurred three weeks later from sepsis consequent to the bad handling of the wound and application of wrong village remedies. Sobha and his associate were convicted under section 304, I.P.C. On an appeal in the Chief Court of Oudh it was held that the accused had no intention of causing death or such bodily injury as he knew to be likely to cause death, nor could it be held that the accused must have had the knowledge that the blow he was dealing was likely to cause death. The conviction under section 304, I.P.C. was, therefore, set aside and the accused was convicted under section 325, I.P.C.[2]

WHICH OF THE SEVERAL INJURIES CAUSED DEATH ?

In the case of multiple injuries inflicted on a person by more than one accused either at the same time or at different times it is very essential to discover the injury which proved fatal, and whether it was the result of one or more blows ; for, the defence pleader may admit death, but may plead that it was not due to the wound attributable to his client. This can be ascertained by examining the wounds individually and noting which of them involved injury to some vital organ or large blood vessel, or led to secondary results causing death. For instance, there may be several wounds on the scalp, but only one may cause fracture of the skull ending in death. Again, fractures of more than one skull bone may result from only one blow. It must, however, be noted that, even if he fell dead at the hands of one of them, all the accused are responsible for having caused the death of their victim, if they started with the common object of intentionally causing such hurt as would be likely to end fatally.[3] For example, in the case of *Emperor* v. *Chandan and two others*[4] where all the three accused in furtherance of a common intention beat one Kallu Jat with *lathis,* and one of the blows caused fracture of the skull, which resulted in death on the following day, the accused were found guilty under section 304, I. P. C., even though it was not ascertained as to who dealt the fatal blow. In the case of *Emperor* v. *Bukshan and others*[5] where a woman was murdered it was held that though there was no evidence as to who actually committed the murder, the four persons having taken

1. *Davis,* (1883) 15 *Cox* 174 ; *Ratanlal and Thakore, Law of Crimes, Ed. XIV, p.* 702.
2. *Oudh Chief Court Crim. Appeal No.* 173 *of* 1935, *K. E.* v. *Sobha and another,* 36, *Crim. Law Jour.,* 1935, *p.* 1262.
3. Vide *Section* 34, *I.P.C., Appendix VII.*
4. *Criminal Law Journal,* 1926, *Vol.* 27, *p.* 619.
5. *Criminal Law Journal,* 1926, *Vol.* 27, *p.* 1265.

the woman out with the knowledge and with the purpose that one of them should murder her, the murder was committed in furtherance of the common intention of all, and all the four accused were guilty of murder. On the other hand, four persons attacked another with *lathis* with the result that the latter received a single blow on his head which caused his death due to fracture of the skull. There was no other grievous hurt on his body, and there was no evidence as to which of the assailants had struck the fatal blow. It was held (1) that it was impossible to hold that the assailants had any common intention to cause death, nor could it be said that each one of them knew that death was likely to be caused; (2) that the common intention of the assailants was to give the deceased a good thrashing and they must have known that grievous hurt was likely to be caused; (3) that as it was not known which of the assailants had struck the fatal blow, they could only be convicted of causing grievous hurt.[1]

THE POWER OF VOLITIONAL ACTS IN A VICTIM AFTER RECEIVING THE FATAL INJURY

Sometimes, the prosecution sets up a theory that the victim, after receiving mortal injuries involving a vital organ, such as the brain, was able to speak and mentioned or wrote down the name or names of his assailant or assailants. Similarly, the defence may try to prove an alibi if the accused was seen with the victim a moment before his death at a particular spot, and the victim had afterwards moved to some other place on the ground that he could not have walked after having received the fatal injury. In both these cases the medical witness is required to state whether a person is capable of speaking, walking or performing any other volitional act, which would involve bodily and mental power for some time after receiving a fatal injury. A very guarded reply should be given, seeing that a few cases have been recorded in which the victims were able to perform some act as that of walking or climbing requiring some exertion, and survived for some hours or days after receiving very grave injuries, which would ordinarily have proved rapidly fatal.

Cases.—1. One evening, while walking in Bow Bazaar in Calcutta a young Hindu, aged about 18 years, was struck on the head with a piece of wood and knocked down by the violence of the blow. He got up and, after some delay, proceeded to the police station in Lower Circular Road and laid a charge against his assailant, whose name was not known, but who was arrested and identified by some of the eye-witnesses. From the police station he walked to the Medical College Hospital, and was then found to have sustained a lacerated wound on the scalp, situated on the left side of the vertex in the frontal region. The wound was dressed, and the injured person went to a friend's house, where he spent the night. Next morning he got into a hackney carriage to go to his uncle; during the drive he began to show signs of compression, and becoming unconscious, was removed to the Campbell Hospital, where he died. The post-mortem examination revealed, besides the external wound of the scalp, a fracture which extending vertically through the temporal region and through the middle fossa of the base terminated at the posterior part of the sphenoid. —*Ind. Med. Gaz., Jan.*, 1894, *p.* 32.

2. A man received several extensive fractures of the skull with abundant subdural hæmorrhage, and rupture of the diaphragm with hernia of the stomach.

1. *Criminal Law Journal*, 1925, *Vol.* 26, *p.* 381.

The stomach was ruptured and nearly a litre of its contents was contained in the left pleural cavity. Notwithstanding all this, he was able to walk about for an hour or so and answer several questions. He died only after several hours. Another man, crushed by a carriage, received a large rupture of the diaphragm, complete rupture of the jejunum, and rupture and crushing of the kidney. Yet he walked nearly 5 miles, and did not die until the next day.—*Vibert quoted by Witthaus and Becker, Med. Juris. and Toxic., Vol. II, p.* 40.

3. A man received a cut in the carotid (it is not mentioned whether external or common) artery late at night. After receiving the cut he mentioned that he had seen the persons whom he named stealing his *goor*, that he had seized one of them, that the other cut him on the neck with a *dhao* or knife and that both escaped. He was able to identify them, when the neighbours were sent for and confronted with him. He died the following day.—*Chevers, Med. Juris., Ed. III, p.* 427.

4. At noon on the 23rd May, 1923, a Mahomedan male, aged 40 years, was stabbed in the stomach with a knife, and was able to walk about two furlongs and a half, when his strength gave out and he lay down. He was then taken on a bed to the police station when he was in his right senses and made a report. He was sent to the hospital for medical examination, where his dying declaration was recorded as he proved to be in a dangerous condition. He died at 10 p.m. on the following day.—*K. E.* v. *Kallankhan of District Bijnor, All. High Court, Criminal Appeal No.* 757 *of* 1923.

5. Gurdeen of Police Station, Mohanlalganj, aged 30 years, who was assaulted with *lathis* and a sharp cutting instrument on the 9th August, 1926, walked a distance of 70 to 80 paces and gave the names of his assailants before he died. At the post-mortem examination on the next day I found the nostrils cut off with a portion of the septum removed, two lacerated wounds on the head and eleven bruises on various parts of the body. There was also a fracture of the right parietal bone extending into the right side of the frontal bone. The coronal and sagittal sutures were separated, and the temporal bones were fractured.

6. At about 8 p.m. on the 24th March, 1928, Ali Bakhsh, 50 years old, received an incised wound, 1" × ½", in the middle of the left side of the neck causing injuries to the big vessels of the neck, and tried to run after his assailant but fell after a few yards. He was removed to the police station where he was able to make a report of his assault. From there he was taken to the hospital, and his condition was so grave that the doctor took down his dying declaration at 10-30 p.m. He died at midnight.—*King-Emperor* v. *Chhote, All. High Court Crim. Appeal No.* 636 *of* 1928.

7. At about 9 or 10 p.m. on the 21st August, 1928, Sheo Narain, aged 45, of District Cawnpore, was assaulted by his brother with a *kanta*, and received an incised wound, 6" long, along the left side of the chest, severing completely the left 8th, 9th, 10th and 11th ribs, penetrating into the left pleural cavity and cutting the diaphragm to an extent of about 4" in length. The stomach, spleen and a part of the intestines were protruding ouside the chest wall through the wound. The spleen had also a superficial wound, 2" long. At 4-30 a.m. on the next day he was taken to the police station, where he lodged a complaint. From the police station he was sent to the nearest dispensary, but was afterwards removed to the District Hospital at Cawnpore, as his wound was very serious. At about midnight he made a dying declaration before a Deputy Magistrate in the hospital, and died in the morning of the 23rd August.—*King-Emperor* v. *Manna, Allahabad High Court Appeal No.* 239 *of* 1930.

8. On the morning of December 14, 1931, Mr. Stevens, Collector of Comilla, was shot by a girl with a .45 revolver, while he was standing on the threshold of his office and on the left of his sub-divisional officer. He fell against the sub-divisional officer and said "I am hit", then turned and ran through the office up through the dining room into the pantry and shut the folding doors, before he fell dead on the floor. The post-mortem examination showed that the bullet had gone through the heart and out into the right lung.—*Leader, Dec.* 25, 1931.

DIFFERENCE BETWEEN WOUNDS INFLICTED DURING LIFE AND AFTER DEATH

In India, the practice of inflicting wounds on a dead body to support a false charge against an enemy is so common that every medical officer who has done medico-legal work must have come across such cases during his professional career.

The following are the principal points by which a wound inflicted during life can be recognised : —

1. Hæmorrhage.
2. Retraction of the edges of the wounds.
3. Signs of inflammation and reparative processes.

1. **Haemorrhage.**—There is more or less copious hæmorrhage in all wounds, except in lacerated wounds, when it may be very little. The effused blood is forced into the tissue interspaces in the vicinity of the wounds, and is found infiltrated in the cellular and muscular tissues. There is consequent staining of the edges of the wounds and the neighbouring tissues, which cannot be removed by washing, but the staining caused by the blood effused from post-mortem wounds is easily removed by washing.

There will be clots of the effused blood in the wounds and tissues, and in the neighbourhood of the body. Clotting of the blood occurs normally in about five to ten minutes.

There will also be signs of spouting of arterial blood on the body, clothing, or in its vicinity.

In a contusion there will be the presence of ecchymosis, absorption changes of its colour and a swelling of the neighbouring tissue. On dissection coagulated blood will be found in the subcutaneous tissues.

2. **Retraction of the Edges of the Wounds.**—Owing to the vital reaction of the skin and muscular fibres the edges of a wound inflicted during life retract, and cause the wound to gape. On the other hand, in the case of a wound inflicted long after death when the body heat has passed off the edges do not gape, but are closely approximated to each other, as the skin and other tissues have lost their contractility.

3. **Signs of Inflammation and Reparative Processes.**—The signs of inflammation, the formation of granulation tissue, and the presence of pus or scab are very strong proofs that the wound was inflicted some days before death.

The absence of the above signs will show that the wound was inflicted after death ; however, it must be borne in mind that hæmorrhage and retraction of the edges may take place in a wound caused within one to two hours after death during the molecular life of the tissues, when the body is still warm. In such a case hæmorrhage is slight, unless a large vein is cut and there is no arterial spouting or formation of a firm clot which rarely occurs ten minutes after death.

Table showing the Distinction between Ante-mortem and Post-mortem Wounds

Ante-mortem Wounds.	Post-mortem Wounds.
1. Hæmorrhage, more or less copious and generally arterial.	1. Hæmorrhage, slight or none at all, and always venous.
2. Marks of spouting of blood from arteries.	2. No spouting of blood.
3. Clotted blood.	3. Blood is not clotted; if at all it is a soft clot.
4. Deep staining of the edges and cellular tissues, which is not removed by washing.	4. The edges and cellular tissues are not deeply stained. The staining can be removed by washing.
5. The edges gape owing to the reaction of the skin and muscle fibres.	5. The edges do not gape, but are closely approximated to each other, unless the wound is caused within one or two hours after death.
6. Inflammation and reparative processes.	6. No inflammation or reparative processes.

DIFFERENCE BETWEEN SUICIDAL, HOMICIDAL AND ACCIDENTAL WOUNDS

In the case of death occurring from wounds, the question is often raised as to whether they were the result of suicide, homicide or accident. The answer is not always easy, but it can be given to some extent by a medical practitioner by noting the following points: —

1. The situation and character of the wounds.
2. The number, direction and extent of the wounds.
3. The condition of the locality, and the surroundings of the wounded person.

1. **The Situation and Character of the Wounds.**—Suicidal wounds are usually on the front or on the sides of the body, and affect the vital organs. They are usually incised, punctured or gunshot wounds. Suicidal incised wounds are generally situated on the front of the body, in easily accessible positions, especially, the throat or chest. These may be found in unusual regions. For instance, a woman[1] cut the walls of her vagina, and when the intestines protruded she pulled down several feet, and cut them off. Incised and punctured wounds situated on the back, or in such a position as cannot be easily reached by a suicide, are homicidal, though an insane person may produce wounds on himself which may have the appearance of being homicidal. A woman[2] in an asylum took a blunt knife, and cut her head almost off from behind.

Incised or punctured wounds may be caused accidentally by falling upon a sharp cutting weapon held in the hand or upon a sharp pointed

1. *A. Robertson, Med. Juris. and Toxic., Ed. IV, p. 138.*
2. *Ibid.*

object. Such wounds may be situated at such places as may give rise to a suspicion of homicide, if there was no eye-witness at the time of the accident. A. S. Dawson[1] reports the case of a Burmese male, who was descending a bamboo ladder in his house when he suddenly slipped and fell a distance of 12 or 13 feet. At the time when he was descending the ladder he had in his hand a long sharp knife, or *dao,* his hand resting on his left shoulder : as he fell this slipped off and struck him on the back over the apical region of the left lung causing a gaping incised wound, 3" long, and penetrating the pleura and lung. The knife was extracted by his relatives. The patient ultimately recovered. If death had taken place, the question as to possible homicide might have been raised, since it would be difficult for a person to stab himself in that position.

Cuts on the fingers and palms are produced during attempts by the injured person to seize the weapon, and are, therefore, indicative of

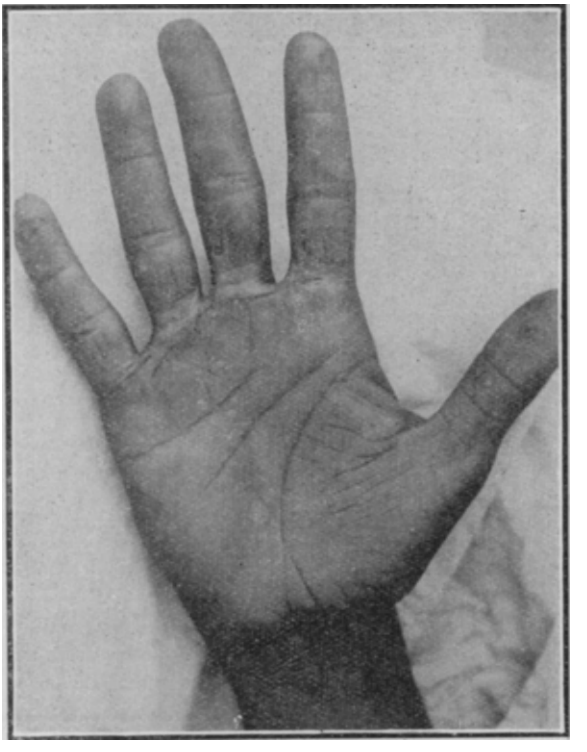


Fig. 80.—Cuts on the fingers and palm caused by grasping the blade of a knife and indicating homicide.

homicide. Incised or lacerated wounds inflicted on the backs of the hands, wrists and forearms during an endeavour to ward off blows on the head or other parts of the body are strongly suggestive of murder.

Incised wounds on the nose, ears and genitals are usually homicidal, and are inflicted on account of jealousy or revenge in cases of adultery.

1. *Ind. Med. Gaz., April,* 1928, *p.* 201.

A case is recorded where a man had been carrying on an intimacy with a widow. The cousin of her deceased husband was much aggrieved over it. Hence he waylaid the lover of the woman, tied him to a tamarind tree with a big rope, and cut off his genital organ practically at the root, severing it completely from the body. Not being content with this he inflicted a wound, 8" by 1", right round the scrotum, his obvious intention being to remove it altogether.[1] In another case[2] a Brahman, aged 23 years, armed with a sickle (*hansia*) cut off the vagina and uterus of his wife,

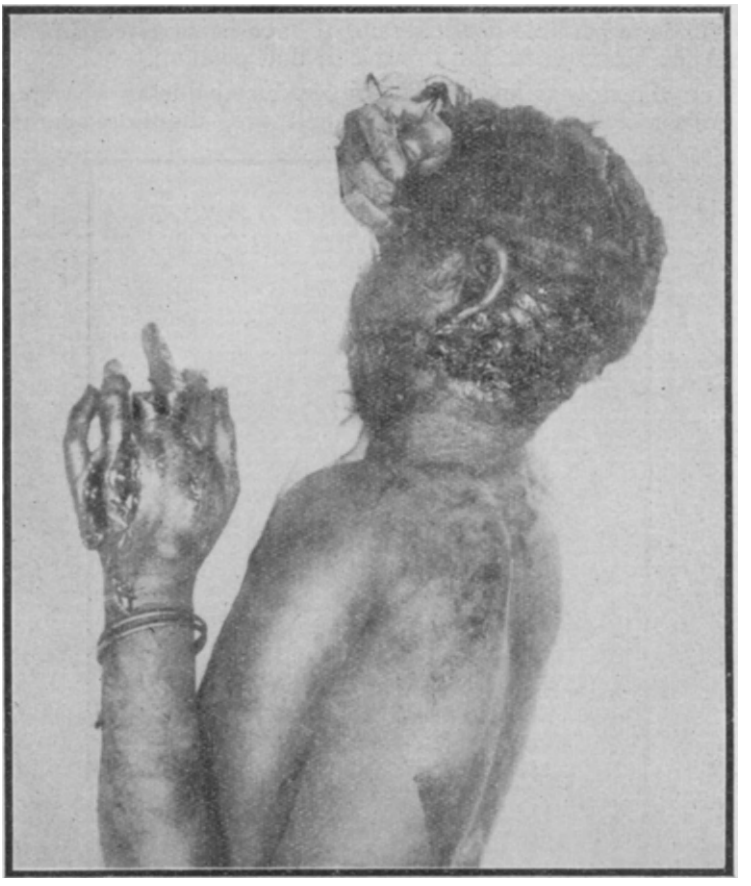


Fig. 81.—Homicide: Incised wounds on back of hand and fingers caused by a heavy cutting weapon during attempts to ward off the blows over the head.

aged 18 years, and disembowelled her by wounds extending from the level of the breast to the anus, cutting the heart, liver, lungs, stomach and intestines. The motive for the crime was jealousy as he saw her lying with another man at midnight.

1. *Leader, Dec.* 7, 1930.
2. *K. E.* v. *Baldeo Prasad, All. High Court Crim. Appeal No.* 377 *of* 1929.

It should be noted that incised wounds on the genital organs are, sometimes, produced after death. I saw a case in which the penis was cut off after the deceased was done to death by a stab in the heart. In another case the penis was almost severed after the neck was cut off with a *gandasa*. In a third case a *kulchal* was thrust into the vagina of a Hindu woman after she was killed by inflicting several wounds on the head and neck with a heavy cutting weapon.

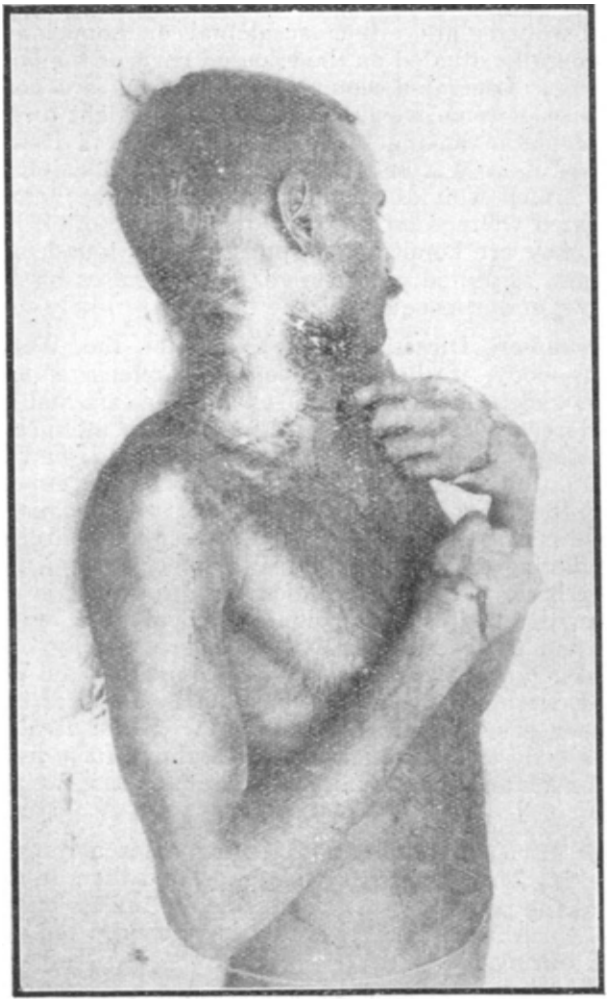


Fig. 82.—Homicide : Incised wounds on wrists due to attempts to ward off assailant's knife.

Incised wounds of a trifling nature on the genitals may be self-inflicted. I came across a case where an adult male inflicted a superficial cut across the root of the penis over its dorsum and directed from left to right, and brought a charge against his enemy who had assaulted him with a *lathi* (club).

Gunshot wounds inflicted in the mouth, or on the forehead, temple or heart, are, as a rule, suicidal when the skin in the neighbourhood is blackened, scorched and tattooed. In such cases the hand used to steady the weapon at the muzzle end may be blackened or scorched from the discharge of the same, and may be stained with spirting of the blood from the injured arteries. Gunshot wounds situated on the back and on the occipital region are usually homicidal. Those situated on the sides and front may be accidental or homicidal.

Lacerated wounds are either accidental or homicidal. Accidental wounds are generally situated on the exposed parts of the body and mostly on the same side. Lacerated wounds of the vertex are homicidal unless there is a history of some weight falling from a height on the top of the head or of the victim having fallen head downwards from a height, in which latter case there will be abrasions and lacerations on other parts of the body. Lacerated wounds on the forehead may be homicidal or accidental. Lacerated wounds on the occiput are more often homicidal than accidental. If they are homicidal, wounds may be found on the backs of the fingers as the assaulted person involuntarily raises his hand to guard against the blow, and, consequently, the fingers are likely to be injured.

2. **The Number, Direction and Extent of the Wounds.**—Several injuries on the body, if they are deep and extensive, are, as a rule, homicidal if we except accidents from falls, motor cars and other vehicles. In India, murderers select a heavy cutting instrument, such as a *gandasa* (chopper), *banka, khurpi, kulhari* (axe), sword, etc., and inflict several deadly wounds on the head cutting the skull bones and exposing the brain tissue, or on the neck cutting the larynx, œsophagus, large blood vessels, vertebræ and even the spinal cord. They are not generally satisfied by inflicting only one wound, but inflict several mortal wounds, and sometimes hack the body so much that the head is either severed altogether from the trunk, or remains connected to it by a mere tag of skin. In addition to these, several wounds are usually inflicted on the trunk and limbs. In one case twenty-six wounds on the body of a boy, three years old, were inflicted with a *gandasa* by a girl of twelve years of age. In another case of murder one hundred and five wounds were inflicted on the body of a Hindu woman. Of these, forty-five were on the head, face and neck and the remaining on the forearms, wrists, fingers and shins.

It must be borne in mind that in some cases a murderer kills his victim by inflicting one or more fatal wounds, and then, in order to divert the attention of the police to possible suicide, he inflicts on the dead body other wounds which in themselves would have caused the death had they been produced during life.

A case[1] occurred at Agra, where a Lumbardar killed a boy, aged 17, by inflicting wounds on the face and neck with a sword, and then fired a rifle from a distance of a few feet, causing further wounds on the face and head which resulted in the splintering of the face and skull bones and laceration of the brain substance. Afterwards he placed the rifle over the corpse to make it look as if it were a case of suicide. During the trial the defence suggested that the boy committed suicide by firing the rifle with the muzzle in the mouth. But the medico-legal officer who held the post-mortem examination proved to the satisfaction of the Court that the rifle

1. *All. High Court, Crim. Appeal No.* 1202 *of* 1929.

was not fired within the mouth and that some of the injuries on the neck and face were such as could not have been caused by the firing of a rifle, but were caused by a cutting weapon and might have been caused by a sword. The Lumbardar was convicted of the murder of the boy under section 302, I.P.C., and was sentenced to death.

On the other hand, several severe injuries on the body may, sometimes, be suicidal.

Wm. Alexander[1] reports a case where an officer was found lying on a couch with two deep incised wounds on the front of the abdomen and one similar wound on the back near the spine. Twenty-six incised wounds were found about the left breast, some of them penetrating the thorax and others leading along the ribs; both hands were dreadfully mutilated. Lying close by the officer was a sword covered with blood and bent to an angle of about 45 degrees. He lived for several hours. and mentioned how he had transfixed himself by placing the hilt of the sword against the wall and then pressing forward on it; but failing to effect his object he made a second attempt. This time the blade, impinging on the spine, was bent, so that he had great difficulty in withdrawing it, his hands being cut severely in the effort. As death did not ensue, he then tried to perforate the heart, but without success.

On August 27, 1933, a Mahomedan male, 60 years old, was admitted to the King George's Hospital, Lucknow, as a case of suicide with multiple injuries inflicted with a razor. On examination Mr. Mathur, Reader in Surgery, found the following injuries :—

1. An incised wound, 3" × 2½" × ½", round the base of the penis and the scrotum cutting off both the structures with the testicles from the body.

2. An incised wound, 4" × ¾" × ½", along the middle line of the abdomen towards the right and directed from above downwards.

3. An incised wound, 6" × 2½", across the abdomen at the level of the navel. A loop of the small intestine, 4 feet long, was protruding out of the wound. One foot of the intestine was slit longitudinally and then divided transversely into two.

The abdominal cavity was full of blood, and the stomach was also found divided vertically into two parts in its middle.

In his statement he mentioned that he felt heat in his head and, therefore, inflicted the wounds with a view to end his life.

The presence of a large number of superficial wounds is presumptive evidence of self-infliction.

Douglas J. Kerr[2] mentions the case of a healthy young man who, under the influence of a delusion that he had killed his sister, had made over 440 cuts on various parts of his body, *e.g.*, on the forehead, chin, front of the neck, chest, abdomen. scrotum and the dorsal and palmar surfaces of the fingers, hands and wrists. These had been inflicted with a blunt pen-knife, and were chiefly superficial though in a number of cases the underlying muscles were divided.

Suicidal wounds caused by a cutting instrument on the neck are generally single and are situated either above the hyoid bone and open directly into the mouth or are situated below the hyoid bone and involve the thyroid or cricoid cartilage, or the large blood vessels of one side. However, extensive wounds in the neck involving the large blood vessels of both sides and reaching the spine, though rare, are seen in suicidal cases.

In June, 1915, a Hindu male, aged 22, committed suicide by cutting his throat with a razor. On inspection an incised wound, 4½" by 2", was found across the front

1. *Lancet, Jan.* 24, 1885, *p.* 178.
2. *Brit. Med. Jour., Feb.* 12, 1927, *p.* 278.

of the neck above the hyoid bone cutting all the structures down to the spine. Taylor[1] quotes a case observed by Marc, where a suicide divided all the muscles of the neck. windpipe, and gullet grazing tne anterior ligaments of the spine, and opened the jugular vein and both carotid arteries. Sydney Smith[2] describes the following case :—

An adult Egyptian male was seen to cut his throat in the street, after which he staggered along for about 50 yards and fell dead. On examination seven transverse

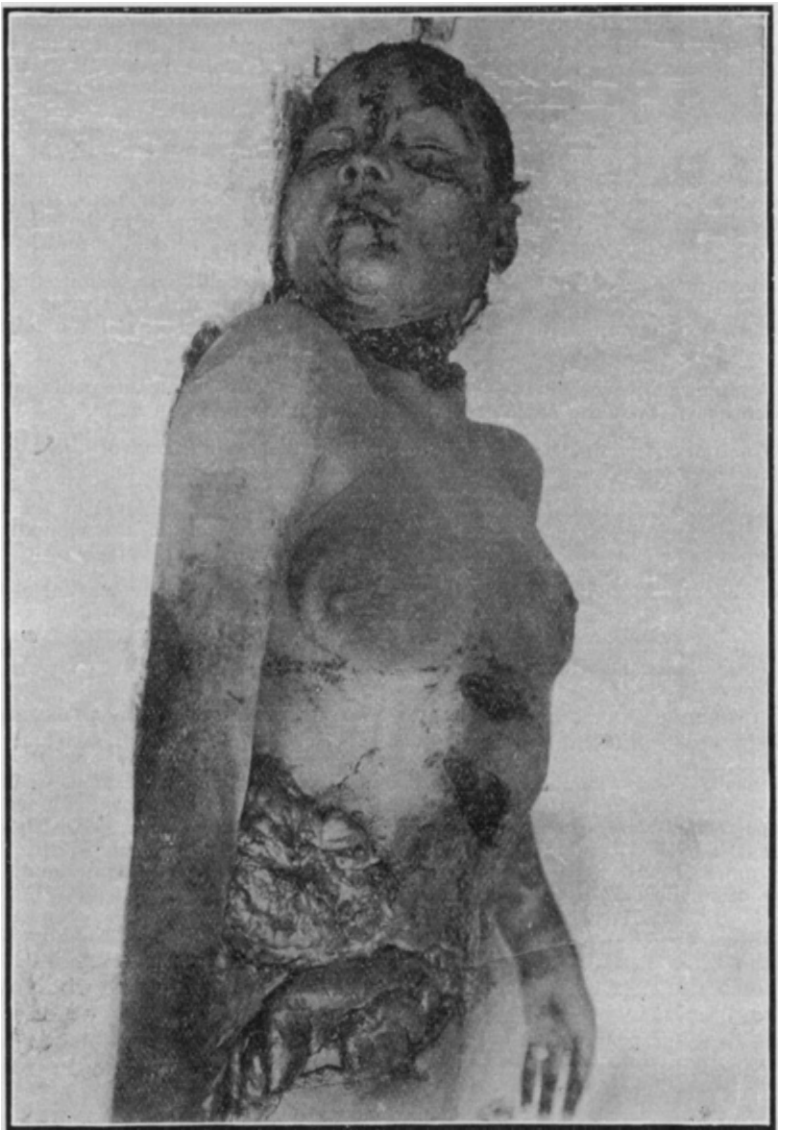


Fig. 83.—Homicidal cut-throat with multiple injuries.

1. *Pric. and Pract. of Med. Juris., Vol. I, Ed. IX, p.* 374,
2. *Lancet, Dec.* 24, 1926, *p.* 1163.

wounds of the throat were found. These were all superficial and separate on the right side of the neck but joined together into one deep incision on passing to the left. Three of them, deeper than the others, started behind the level of the right sternomastoid; the other four which were lower down and much more superficial, commenced midway between the sternomastoid and the middle line. They all passed almost horizontally across the throat and ended in a jagged incision on the anterior margin of the left sternomastoid. Considerable damage was done to the deeper tissues of the neck and in the cartilaginous structures four well-defined incisions were observed. The highest of these passed horizontally through the thyroid cartilage and completely divided the larynx. The second passed completely through the larynx between the thyroid and cricoid cartilages. The third passed through the middle of the cricoid in the same horizontal position as the others. The fourth passed through the lower part of the cricoid in front, but sloped downwards at an acute angle and reached the upper rings of the trachea posteriorly. This incision cut the left common carotid artery and the left external jugular vein. There was also a superficial cut on the right wing of the thyroid cartilage which could be produced only by a cut commencing on the right and indicated that the victim was left handed. Other structures divided included the thyroid gland in two places; the superior thyroid artery and the crico-thyroid branch; the sterno-hyoid, sterno-thyroid and omo-hyoid muscles, while the sterno-mastoid muscle on both sides was severely injured.

Douglas J. Kerr[1] reports the suicidal case of a cut-throat with a razor, in which the thyroid cartilage was found to have been hacked to pieces, there being seven distinct cuts through the cartilage. The œsophagus had been opened into on the right side, both jugular veins had been severed, the carotid artery on the left side had been opened and the left transverse process of the fifth cervical vertebra had been cut through, the severed part lying free in the tissues of the neck.

In a criminal case[2] of reference before the Patna High Court where one Sheocharan Das was accused of murdering two young boys by inflicting wounds on their throats and then having attempted suicide by cutting his own throat the Honourable Judges held that all the three boys had attempted suicide, two successfully and one unsuccessfully, and acquitted the accused of the charge of murder but convicted him of the offence under section 309, I.P.C. The injuries on these three boys were as follows:—

First boy: Two incised wounds, 5" × 2", on the right side of the neck cutting the trachea, larynx, œsophagus and the right cornu of the hyoid bone, grazing upto the vertebræ and cutting the intervening muscles and the external carotid artery.

Second boy: Two incised wounds on the neck, one of these, 1" × ½", skin deep, and the other, 5" × 1½", muscle deep, on the right side of the neck cutting the larynx and trachea laterally, and the platysma and the muscles of the right side of the neck. The external carotid artery was also divided and the vertebra was grazed.

Third boy: Three incised wounds on the neck; of these two were superficial, and the third was 5" × 1½", on the front of the neck, cutting the larynx and œsophagus and fracturing the right cornu of the hyoid bone.

Sometimes, there are two or more simple cuts at the commencement of the wound, when the suicide is still hesitating or nervous, and then makes a deep cut, after plucking up courage to destroy himself. In India, suicidal wounds of the throat are rare. During a period of sixteen years from 1907 to 1923 I saw only nine cases of suicidal cut-throats with four deaths. Of these nine cases two were among females and seven among men. In one of the cases the wound was inflicted by a *kulchal* across the right side of the neck cutting the thyroid and cricoid cartilages.

1. *Brit. Med. Jour., Dec.* 6, 1924, *p.* 1042.
2. *K. E.* v. *Sheocharan Das, Patna High Court Cr. Ref. No.* 5 *of* 1937; *Cr. Law Jour., Jan.*, 1938, *Vol.* 39, *p.* 66.

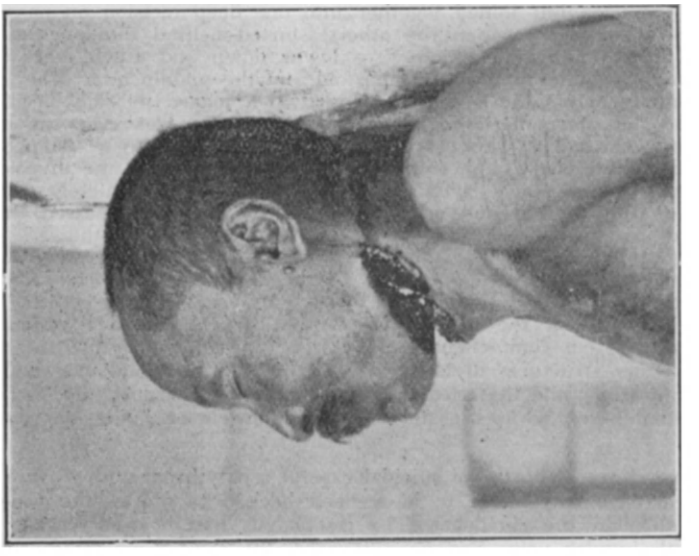


Fig. 85.—Suicidal cut-throat.

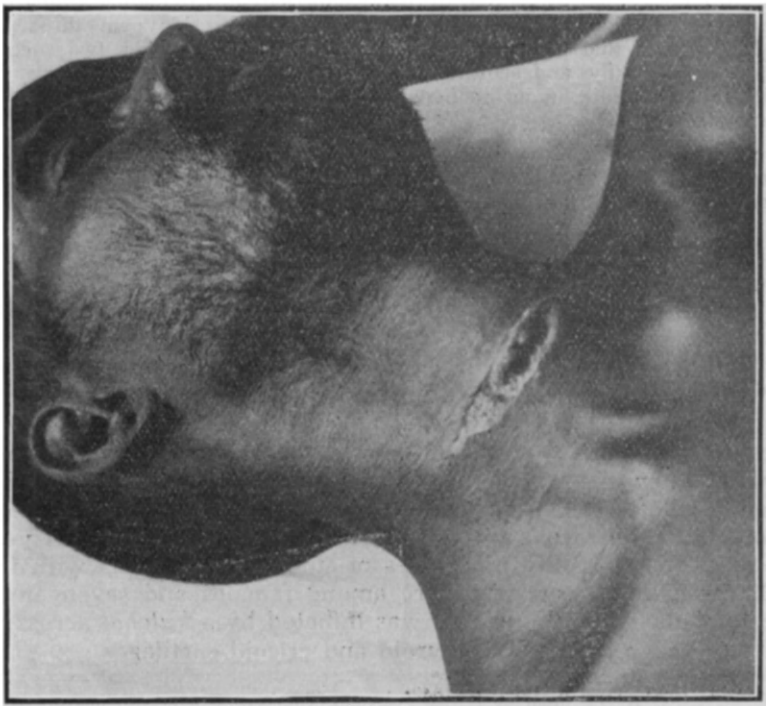


Fig. 84.—Suicidal cut-throat.

Suicidal wounds of the throat inflicted by a right-handed person are usually high up in the neck and are directed obliquely from a higher to a lower level and from left to right, while homicidal wounds of the throat, when inflicted from the front by a right-handed person, are, as a rule, horizontal and directed from right to left; but the reverse is the case if the assailant happens to be left-handed. Again, a homicidal wound on the throat may resemble a suicidal one, if the assailant has inflicted it from behind the victim, or by standing on the right when the victim is lying. It is difficult to decide in the case of ambidextrous persons, who can use both hands.

Suicidal wounds of the chest are usually on the left side, and directed downwards and inwards, unless the person happens to be left-handed. Homicidal wounds of the chest are usually distributed over a wider area and are more horizontal, and most of them may be deadly owing to the vital organs being injured. They may be directed from below upwards which is rarely seen in suicidal wounds.

Suicidal wounds on the arms are usually directed from above downwards, and those on the lower limbs from below upwards.

Self-inflicted wounds simulating homicidal wounds are usually produced with a view to support a false charge of assault against an

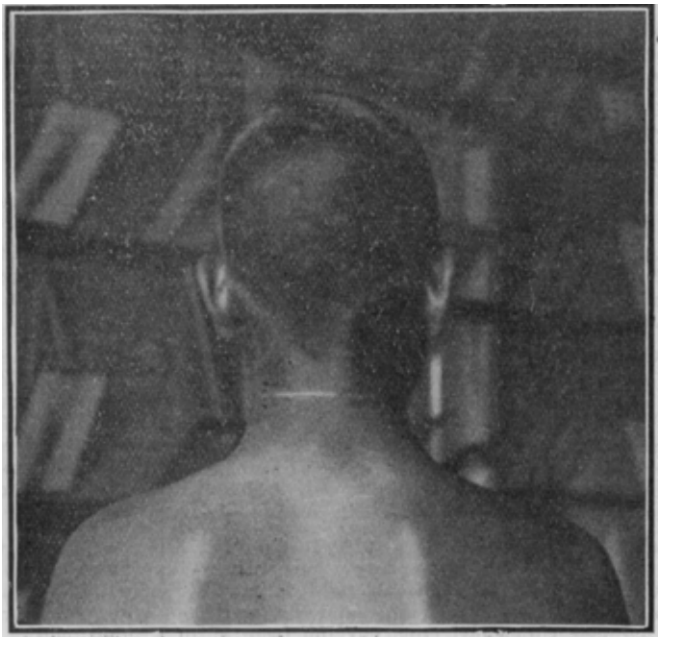


Fig. 86.—Self-inflicted incised wound across the back of the neck (*Vide* case 3 on p. 262).

opponent, to augment the seriousness of the injuries which one has already received during a quarrel, to prove self-defence in an accusation of assault or murder, or to substantiate a charge of violence and robbery in a case where one has appropriated money or valuables placed in one's charge.

Such wounds are commonly on the front of the body, but may be on those parts of the back which can be easily reached by the hand. They are several superficial cuts or scratches made with a knife, razor or some pointed instrument. They are often parallel with straight regular margins. I have seen several cases of fabricated wounds, but the following are characteristic :—

1. In 1916, a student of the Agra College inflicted twenty wounds on the abdomen, twenty-eight on the right thigh and thirty on the left, after he had inflicted some nasty wounds with a razor on his room fellow. They were very characteristic and suggestive of self-infliction. The wounds on the abdomen were mostly transversely oblique, and directed from left to right. None had gone deeper than the muscles. Those on the right thigh were all supreficial and directed from below upwards, and from within outwards, while those on the left were directed from below upwards, and from without inwards. Thus, all the wounds were caused by the right hand. They varied in length from three to eight inches.

2. In May, 1919, a Mahomedan male of Police Station, Saadatganj, District Lucknow, received three simple bruises on the right forearm, right shoulder, and left cheek bone during a quarrel. He went home, inflicted some injuries on his body and lodged a complaint against his opponent at the police station. On examination I found the following injuries which, from their position and appearance, did not leave any doubt of their being self-inflicted :—

(*a*) A superficial cut, half-an-inch by quarter of an inch, tapering into a linear tail, three inches long, obliquely along the middle of the front of the left forearm directed from below upwards and from without inwards.

(*b*) Three superficial incised wounds, varying from three-quarters to two inches long, and from one-eighth to one-fourth inch broad, obliquely along the right side of the chest directed from above downwards and outwards.

(*c*) Two vertical linear cuts, each half-an-inch long, on the left side of the chest above the left nipple.

(*d*) Three horizontal superficial cuts, varying from half-an-inch to two inches by one-eighth to one-fourth inch, and parallel to each other, below the right nipple. They all ended in linear tails of varying lengths and were directed from left to right.

(*e*) Two vertical superficial incised wounds, each measuring one inch by one-fourth inch, along the upper part of the right thigh on its inner side. One of these was directed from below upwards and from within outwards and the other, from above downwards and from without inwards.

3. One Ramavtar of Police Station, Mohanlalganj, complained that he was assaulted by a man with a *gandasa* (chopper) on the 12th May, 1927, and received a wound on the back of the neck. On examination I found a transverse incised wound, 3" by $\frac{1}{4}$" (in its widest part) by 1|6", across the back of the neck in its lower part, commencing from 2" to the left of the spinal column and directed towards the right side ending into a linear, superficial, transverse scratch, $1\frac{1}{2}$" long. I gave my opinion that it appeared more like a self-inflicted wound than a homicidal wound, and the accused was discharged by the Sessions Judge.

4. On the 15th November, 1928, I examined Must. Rukmani who complained that she was struck with a *gandasa*. She had a superficial cut with a dry scab, $\frac{3}{4}$" by 1|10", across the back of the right foream $3\frac{1}{2}$" below the right elbow, ending into a linear superficial cut, 1" long, and directed from within outwards. There were three more linear superficial cuts, varying from $\frac{3}{4}$" to 1" long, across the back of the same forearm below the first cut.

5. On or about the 26th January, 1929, one Swami Din killed a Mahomedan male by inflicting several injuries on his body with a heavy cutting weapon, and then caused cuts on some parts of his own body to bring forward a plea that in self-defence he killed the Mahomedan as the latter wanted to take his life. On examination I found several linear cuts and scratches on his forehead, right temple, sides of the front of the neck and back of the left hand. Most of these were almost parallel, and some were crossing one another.

6. On the 28th December, 1930, I examined a sweeper who complained that he was struck with a razor by his opponent. He had several superficial cuts, varying from 1" to 4" by 1/8" to 1/6", obliquely across the back of his right forearm in its upper half and directed from within outwards. I gave my opinion that the cuts appeared to be self-inflicted.

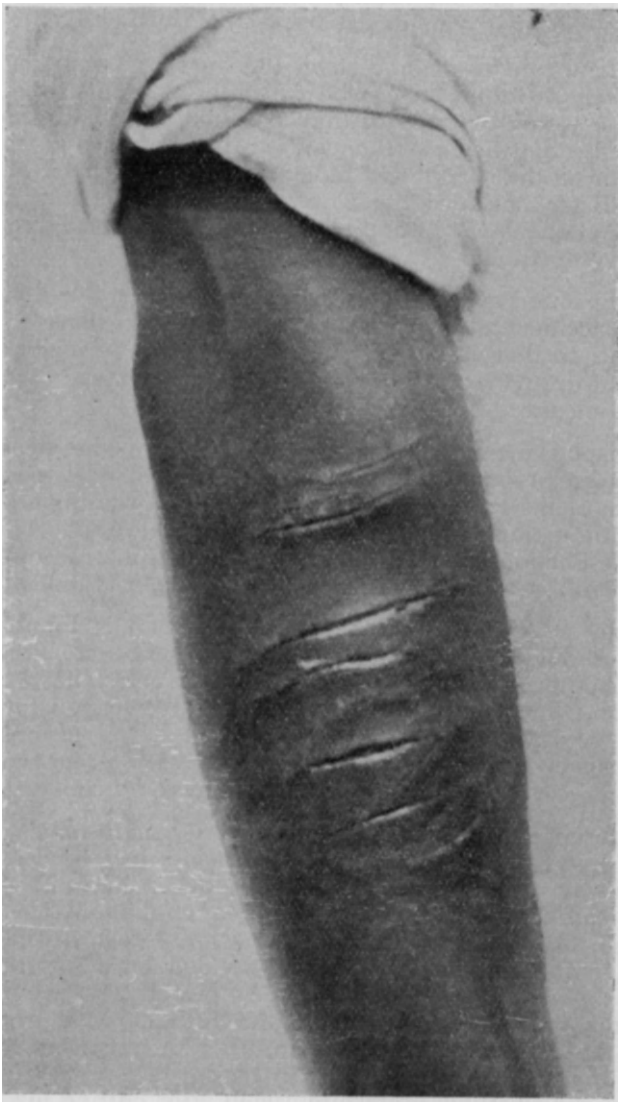


Fig. 87.—Self-inflicted wounds on forearm (*Vide* case 6 on this page).

3. **The Condition of the Locality and Surroundings of the Wounded Person.**—The finding of a body in a room with the door and windows locked on the inside points to suicide. If only the door is locked, the windows should be carefully examined for the presence of bloody finger-marks or other evidence that some one has escaped through them. The

finding of a farewell letter or evidence of a design is strongly presumptive of suicide. A disordered state of the clothing and a disarranged condition of the furniture in a room indicate a struggle having taken place, and are, therefore, greatly in favour of homicide. It should, however, be remembered that cases are on record where lunatics upset and damaged the furniture owing to the maniacal frenzy before they committed suicide.

Foot-prints in blood or dirt on the floor or verandah of the room in which a body is found should be carefully examined and compared with those of the victim or those of the suspected person in order to determine if it is a case of suicide or homicide. Blood-stained finger-marks on the furniture or on the corpse will indicate homicide, if they do not correspond with the finger-marks of the victim. These foot-prints and finger-prints should be photographed so that they might be used for identifying the assassin in the future.

A body found at the foot of a precipice, or on a railway line points to suicide or accident; but it may have been placed there to conceal the act of homicide. In that case a careful search should be made for the presence of marks of dragging the body on the ground, marks of blood-stains, and foot-prints on the ground and in the vicinity.

A weapon firmly grasped in the hand of the deceased person is strongly suggestive of suicide. In such a case blood is generally found on the outside of the hand and fingers, or between the fingers, but not on the palm and the palmar aspect of the fingers. There may be blood-stains on the wrist. Portions of hair, fragments of clothing, or some other foreign material firmly grasped in the hand of the corpse is indicative of homicide.

Suicide is generally suspected if a weapon is found lying near the body. It should be examined for the presence of blood-stains, and it should be determined whether the wounds could have been caused by the weapon; for, it is quite possible that the weapon found may not be that with which the injuries were inflicted. It is also possible that the weapon may be quite clean if it was wiped with a piece of cloth or towel, which would, very likely, be found lying in the vicinity.

The absence of a weapon in the vicinity of the body is suggestive of homicide, but not necessarily, for the suicide may conceal the weapon or throw it away after inflicting a fatal injury on himself. T. H. G. Shore[1] reports a case of suicide, where a sergeant inflicted two cuts on the left side of the neck, which joined into one large gash above his larynx and extended to the right side. He had divided both internal jugular veins and both superior thyroid arteries. The œsophagus, prevertebral muscles and the discs between the fourth and fifth cervical vertebræ were all injured. After inflicting all these injuries he put away his razor into its case, and that into its usual place in his kit-bag. I saw a case where an old man threw away his knife into a well after cutting his throat, and then jumped into it.

All the articles found on or near the body and likely to be of any value in detecting the crime should be carefully examined and then sent to the Superintendent of Police or Magistrate in sealed packets.

1. *Lancet. July* 24, 1920. *p.* 182.

CHAPTER XII

REGIONAL INJURIES

HEAD

Scalp.—Injuries of the scalp are either accidental or homicidal. Lunatics are, however, reported to have produced suicidal injuries on the scalp by means of a heavy weapon. A patient[1] of the Glasgow Lunatic Asylum, Gartnavel, battered his head with a hammer and spike so that the brain-substance protruded, but he recovered. A man,[2] aged 28, who had been arrested for a particularly cold-blooded murder, took out a nail from the wall of his cell and committed suicide by driving it into his skull. A 50-year-old woman, who was suffering from insomnia and climacteric disturbances committed suicide by striking blows on the crown of her head with a hatchet.[3]

In India, most scalp injuries are homicidal, and are generally produced by a blunt weapon, *e.g.*, a *lathi*, a stone or a wooden pestle (*musal*) and occasionally by a cutting instrument, such as a *gandasa*, a *khurpi*, an axe

Fig. 88.—Murder: Wound made with axe seen sticking into the head (*From a photograph lent kindly by the Superintendent of Police, Ahmedabad*).

or a sword. The injuries are consequently contusions and lacerated wounds, as well as incised and punctured wounds. The swelling and

1. *Glasgow Med. Jour.*, 1858; *Glaister, Med. Juris. and Toxic., Ed. VI, p.* 291.
2. *Brit. Med. Jour., Aug.* 13, 1932, *p.* 321.
3. *Munck, Willy, Deut. Zeit. f. Ges. Gerichet. Med., Bd.* 27, *Hft.* 5, *s.* 308, *Jan.*, 1937; *The Med.-Leg. and Criminol. Rev., Vol. V, Part II, April,* 1937, *p.* 233.

inflammation are not usually very much as the scalp is a dense tissue. It must be remembered that an oblique blow generally causes a large wound and a direct blow, a small wound. These wounds may be simple or complicated with fractures of the skull. While examining them it is always advisable to find out if there is any fracture.

In the case of a contusion the effusion of blood is, sometimes, so great that it forms a hæmatoma (cephal-hæmatoma), which may readily be mistaken for a depressed fracture owing to the sensation of crepitus which it imparts to the fingers on palpating it. The diagnosis is not easy in such cases. In a hæmatoma there is pitting on pressure and there may be a pulsation if any large artery is involved. Its ridge is raised above the surface of the skull, and, if subcutaneous, is movable on its surface, while in a depressed fracture the edge is at or about the level of the rest of the skull and is sharper, more irregular, and less evenly circular than in a hæmatoma.

Wounds of the scalp usually heal very rapidly, though in rare cases fatal results may follow from the supervention of cellulitis or erysipelas, or suppuration may set in, and travel into the brain through the blood vessels or through necrosis of bone resulting from cellulitis, or through an unnoticed fissured fracture. Thus, cases have occurred in which scalp wounds have apparently healed, and yet death has occurred from septic meningitis or brain abscess after a few days or weeks.

The following are a few of the cases brought to my notice :—

1. In 1912, a Hindu male, aged 45 years, went walking to the Thomason Hospital at Agra, three or four days after receiving a contused wound on the head. He was admitted to the surgical ward, where he died after four days. At the necropsy the cause of death was found to be septic meningitis due to a fissure in the right temporal bone.

2. A Hindu male, 22 years old, received seven contused wounds on the head on the 24th May, 1919. Four days afterwards he was admitted to the Police Hospital at Lucknow, from where he was discharged at his own request as the wounds had almost healed with the exception of two which were infected with pus. On the 18th June, he got an attack of paralysis and was, therefore, removed to a dispensary at Malihabad, where he died on the 28th June. On examination of the body necrosis of the left parietal bone in an area of one inch by three-quarters of an inch and a fissure in the right parietal and temporal bones were found. There was a collection of pus between the dura mater and the skull under the fissured fracture. That portion of the dura mater was almost blackened and pus was seen on the upper surface of the brain, especially on the right side.

3. A woman, 70 years old, was injured on the head by dacoits on the 12th March, 1921, and on the 22nd March, she succumbed to the injuries. On post-mortem examination on the following day a contused wound, two inches by three-quarters of an inch, covered with pus, was found along the left side of the crown of the head, one inch and a half above the forehead, exposing the bone which was denuded of its periosteum. The skull bones were intact. The membranes of the brain were congested, and covered with a deposit of lymph. Pus had collected in an area of one inch square on the left upper surface of the brain under the wound.

4. On the 22nd June, 1924, a Hindu male of Police Station, Malihabad, District Lucknow, 45 years old, was admitted into the King George's Hospital, Lucknow, for the injuries inflicted on his head with a blunt weapon, and died on the 14th July, 1924, at 6-35 p.m. The post-mortem examination on the next day revealed a comminuted fracture of the left frontal bone and a fracture of the right anterior and middle fossæ of the base of the skull. The brain substance had sloughed away in an

area of 3" × 3" on the under surface of the frontal lobe on the left side. There was pus underneath the slough in an area of 2" × 2" × ½."

5. On August 20, 1932, a Hindu male, aged 38, was struck on the head with a heavy cutting weapon, while he was asleep, and he sustained a linear fracture of the right temporal bone with an incised wound on the right side of the head. He was quite conscious and able to answer questions rationally till August 31, when he developed the signs of cerebral irritation which deepened into coma, and died on September 9. The post-mortem examination revealed an abscess of the middle and posterior portions of the right hemisphere of the brain in addition to the fracture of the skull bone.

6. A Hindu male, 50 years old, sustained a contused wound, 2½" by ¼", across the crown of the head to the right of the middle line and 2" above the forehead as a result of a blow of a blunt weapon on the 13th November, 1932. He was almost unconscious and was suffering from cerebral irritation and partial paralysis of the right upper limb. On the 22nd November, he regained consciousness, and was able to speak, although not rationally. He died from pneumonia on the 28th November. At the autopsy I found a fissured fracture of the right parietal and right temporal bones with an effusion of clotted blood over the membranes which were congested. There was a contusion, 1" by 1", with softening over the right temporo-sphenoidal lobe. The left lung was pneumonic.

Skull.—Fractures of the skull are, sometimes, caused without any contusion or wound on the scalp, though there may be an extravasation of blood on its under surface.

During free *lathi* fights the skull is, sometimes, smashed into several pieces, as if it was a cocoanut shell. Thus, in a case where a man, 35 years old, was struck with *lathi* blows, there was a comminuted fracture of the frontal, left parietal and temporal bones and of the occipital bone. The base of the skull was fractured in

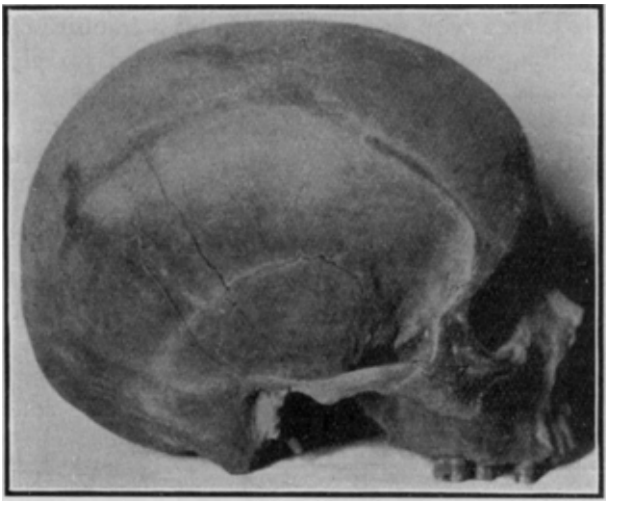


Fig. 89.—Fissured fracture. This skull bone was removed with other bones from a blind well.

the left anterior, middle and posterior fossæ. In another case where a woman, 40 years old, was murdered with *lathi* blows, there were comminuted fractures of the left temporal, parietal and frontal bones, and a simple fracture of the right temporal bone. There was also a separation of the right parietal and temporal sutures, with comminuted fractures of the middle and posterior fossæ of the base of the skull.

The varieties of the fractures of the skull that are usually met with are fissured, partial (outer or inner table, though the inner table is more commonly fractured), stellate or radiating, depressed, elevated, punctured and comminuted. Sometimes, the sutures are separated with or without the fracture. The temporal bone and the orbital plate of the frontal bone are easily fractured. In old age the bones become thin, brittle, and are more fragile.

Vault.—Fracture of the vault occurs at the place of contact by direct violence or at its opposite side by *contre coup* (counter side), when the head is not supported. An extensive fracture running parallel to the two points of contact (bursting fracture) will occur if mechanical force is applied on one side of the head when it is pressed on the other side against a hard substance, such as a wall, while the individual is standing, or against the hard ground or floor when he is in a lying posture. In such cases the fracture may extend transversely even to the base of the skull.

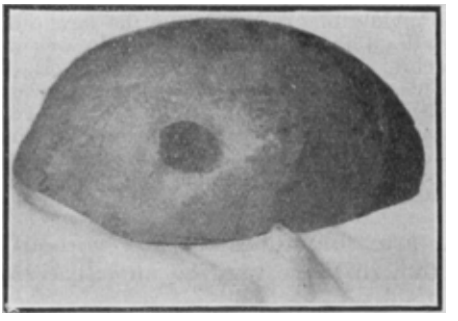


Fig. 90.—Localised depressed fracture of skull bone caused by a mallet.

If not associated with an external wound a fracture of the vault is not always easily diagnosed. In such cases it is best to rely on the general symptoms resulting from injury to the meningeal vessels, cerebral sinuses and brain.

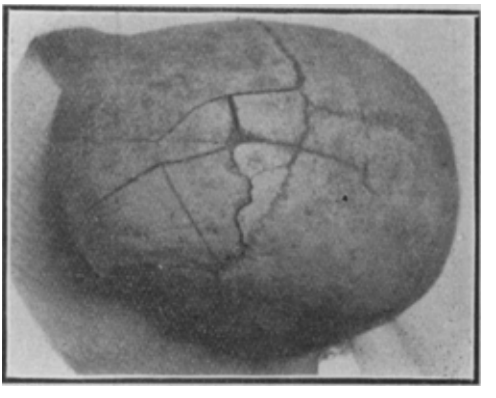


Fig. 91.—Depressed comminuted fracture of skull caused by a blunt weapon.

Fractures of the vault, though dangerous, do not always end in death. I have seen cases in which recovery occurred after the vault of the skull was fractured.

A boy, ten years old, was hit on the head with a *lathi* and sustained a contused wound, 3" by ¼", along the right side of the crown of the head with a fissured fracture of the right parietal and occipital bones and partial paralysis of the left upper limb. After nine days he was admitted into the King George's Hospital, Lucknow, and was discharged cured after three weeks. Dawson[1] relates the case of

1. *Ind. Med. Gaz., Feb.*, 1926, *p.* 65.

a girl, about 8 years old, who was knocked down by a motor lorry and sustained fractures of the parietal, temporal and frontal bones with a fracture extending into the base of the skull. At the operation it was found that the dura mater was torn through which the brain matter was escaping. The patient recovered in a month's time. Tichborne[1] also relates the case of a woman who was brutally assaulted by her husband, and who received five wounds on the head with complete compound fractures of the vault of the skull. The fractures were situated on the left temporal, frontal, right and left parietal and occipital bones, the fractures on the occipital bone being 5" long and 1½" gaping. All these fractures communicated with the surface of the brain, and in all cases the cerebral meninges were exposed. After three days she was taken to hospital, where the severed muscles and the torn scalp seemed sloughing and a general septic state prevailed. The patient did live and attended the Court three months later.

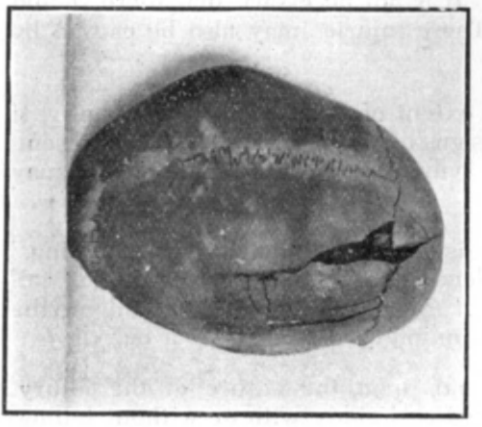


Fig. 92.—Fractures of skull caused by a banka (cutting weapon).

The Base of the Skull.—Fracture of the base of the skull is generally caused by a blow or fall upon the vertex as the head is pressed on the other side of the spinal column. It may be caused by a direct blow with the point of an umbrella or stick thrust through the roof of the orbit or up the nose through cribriform plate, by a violent blow on the chin or by a gunshot wound through the roof of the mouth. It may also result from extension of a fracture of the vault, or may be caused indirectly by a heavy fall upon the feet or nates.

The symptoms observed in fractures of the base are—

(1) Signs of concussion or compression of the brain.

(2) Effusion of blood in the subconjunctival tissue, or in the suboccipital and mastoid regions.

(3) Bleeding, or discharge of cerebro-spinal fluid, from the nose, mouth, or one or both ears.

(4) Lesions of the nerves issuing from the base of the skull giving rise to paralysis or loss of sensation of the parts supplied by them.

The result is not always fatal. Sometimes recovery takes place, though headache, deafness, or other nervous derangements may persist for a long time.

Brain.—Injuries to the brain may occur even without any fracture of the skull, and may result in immediate or remote consequences.

1. *Lancet, Sept.* 22, 1928, *p.* 599.

Contusion and Laceration of the Brain.—A fall or a blow on the head may produce a contusion and laceration of the brain on the same side or on the opposite side by *contre coup*. It is not necessary that there should always be a fracture of the skull. These injuries may also be caused by penetrating or gunshot wounds.

The symptoms depend upon the extent of the injury to the brain. If laceration is slight or superficial, the signs of cerebral irritation are present. In severe lacerations there may be symptoms of concussion, which may be followed by compression.

Concussion of the Brain.—This is popularly known as "stunning," and may be produced by direct violence on the vertex, by a violent fall upon the feet or nates from a height, or by an unexpected fall on the ground, when pushed forcibly by a running cart or even by a bicycle.

Symptoms.—The symptoms depend upon the nature of the injury. Thus, the patient may become confused and giddy with or without falling, if there is a slight injury, and recovers in a short time.

With severe injury the patient falls down and becomes unconscious, though he can often be partially roused by shouting. The muscles are relaxed and flaccid, but there is no paralysis. The sphincters are relaxed with involuntary passage of urine and fæces. The face is pale, and the pupils are equal and usually contracted reacting to light but, in more severe cases, are dilated and insensible to light. The skin is cold and clammy with subnormal temperature. The pulse is rapid, weak, small and hardly perceptible. The respirations are slow, irregular and sighing. Death occurs rapidly from syncope, or recovery follows, with the setting-in of nausea or vomiting. The skin becomes hot and dry, the pulse is full and strong, and the respirations are increased in rate. After apparent recovery in some cases death may result after some days from inflammation or compression of the brain.

In severe cases there is often complete loss of memory of the accident and even of the events occurring before and after it extending over a period of from a fortnight to a month or more. An old man narrated the incident and mentioned the names of his assailants soon after he received four lacerated wounds on the head, five incised wounds on the face and twenty-nine bruises and abrasions on various parts of the body on the morning of July 1, 1932, but in the afternoon of the next day he completely forgot the assault, and vividly described the accident which had occurred to him about seven years ago.

Post-mortem Appearances.—In most cases there may be nothing more than slight congestion of the brain with minute capillary hæmorrhages in its substance. In some cases there may be a cotusion or laceration of the brain substance with an extravasation of blood on its surface.

On the 30th November, 1921, a Pasi, 65 years old, received three small contused wounds on the head with a blunt weapon and died on the 19th December, 1921.

The post-mortem examination revealed a contusion on the left side of the brain with a deposit of lymph on the membranes. The ventricles were full of a dirty reddish fluid.

Fig. 93.—Laceration of brain.

Dr. Syam reports a case in which a man, 38 years old, was struck on the head with a stick at about 2 a.m. on the 8th April, 1922, and died on the 21st April. At the post-mortem examination a lacerated wound, $\frac{3}{4}$" $\times \frac{1}{4}$" $\times \frac{1}{4}$", with a contusion, 2" $\times \frac{1}{4}$", round it was found on the left side of the head. There was no fracture of the skull. On removing the skull cap the whole of the left hemisphere of the cerebrum was found compressed by a blood clot under the dura mater, and the left temporo-sphenoidal lobe was lacerated to the extent of one-and-a-half inches.—*Ind. Med. Gaz., May,* 1923, *p.* 214.

The body of a Mahomedan male, 25 years old, was brought to me with a history that it was found near a railway line. The post-mortem examination revealed an effusion of blood on the upper surface of the brain which was lacerated in an area of 2" $\times$ 2" along the frontal lobe. There was no fracture of any skull bones and no external injury except a few abrasions across the upper part of the forehead.

On August 17, 1932, a Mahomedan male child, 8 or 9 years old, died from a motor car accident. At the post-mortem examination on the following day I found congestion of the membranes and effusion of clotted blood over the surface of the brain which was lacerated in an area of 1" by $\frac{1}{2}$" by $\frac{1}{2}$" along the upper surface of the left temporo-sphenoidal lobe. There was no fracture of the skull bones, but there were abrasions externally over the face, head and chest.

Compression of the Brain.—This may result from a depressed fracture of a skull bone pressing on the brain or from intra-cranial hæmorrhage. It may also result from the pressure of inflammatory exudation or pus on the brain tissue.

Symptoms.—These may come on immediately, or may be delayed for some hours or days, after receiving the injury. The symptoms are those of coma. There is complete loss of consciousness. The patient cannot be roused by shouting or even by shaking. The face is flushed, and the pupils are dilated and insensible to light, but they may be contracted or unequal if there is a small degree of compression over a limited area of the brain. The temperature of the body is normal or subnormal, but may be above normal. The pulse is full and slow, but becomes rapid and irregular towards death. Breathing is slow,

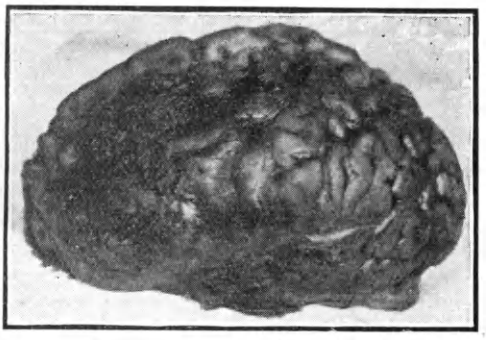


Fig. 94.—Effusion of blood upon the surface of brain.

laboured and stertorous with the lips and cheeks being puffed in and out. There is paralysis of the muscles and extremities according to the area of the brain involved. The reflexes are lost, and retention of urine occurs from paralysis of the bladder. Fæces pass involuntarily owing to relaxation of the sphincter ani, although marked constipation is usually present. Sometimes, convulsions precede death.

In some cases partial recovery may occur, owing to the arrest of blood in an injured artery of the brain by the formation of a clot, but death may take place later on when the clot is disturbed and fresh hæmorrhage takes place owing to the heart being excited by exercise or indulgence in alcohol.

Permanent recovery may occur when the compressing factor, such as a depressed piece of a fractured cranial bone, is removed by trephining. In such cases, however, remote effects, *e.g.*, headache, loss of memory, epilepsy, paralysis, or insanity, may supervene from permanent damage to the brain tissue.

Post-mortem Appearances.—Effusion of blood between the skull and the dura mater upon the surface, or in the substance, of the brain, or at the base of the skull. This effusion is usually due to a fracture of the skull but it may be due to rupture of the middle meningeal artery or of the venous sinuses without any fracture. The brain and its meninges are also found congested. An injury to the brain, such as a contusion or laceration, may, sometimes, be found.

On the morning of the 3rd November, 1920, a Hindu male, 27 years old, was attacked in his shop by another man, who gave him a slap on the face and dashed his head against a wall. He soon became unconscious and died at 5 p.m. on the same day. On examination of the body at 11-30 a.m. on the next day the left middle meningeal artery was found ruptured with an effusion of blood with a few clots between the membranes and the surface of the brain. There were only two small abrasions on the head.

On the night of the 11th October, 1927, a Hindu woman, about 30 years old, was struck with a *lathi* on the head and died soon after. At the post-mortem examination held on the next morning I found a laceration, 2" by 2", along the under surface of the scalp over the left crown of the head and 4" above the left ear. The skull bones were intact, but there was an effusion of blood on the surface of the brain due to rupture of the left middle meningeal artery.

Medico-Legal Questions.—The questions that are usually raised in Court are—

1. Whether the effusion of blood found at the post-mortem examination was due to mechanical violence, or disease, or to excitement during a quarrel.
2. How old the effusion was.

1. When due to violence, the effusion of blood is almost always extradural from rupture of the venous sinuses or the middle meningeal artery as a result of fracture of the skull, but it may be subdural and in the substance of the brain, especially when it is lacerated. The hæmorrhage is usually found under the point injured or directly opposite to this. It is not necessary that there should always be external signs of injury in such cases, for the blood vessels in or on the brain may be ruptured by a blow on the head without causing any injury to the bone. It should be noted that a disease, known as pachymeningitis hæmorrhagica interna, may cause subdural hæmorrhage, but this condition can easily be ascertained on post-mortem examination.

When due to disease, the effusion of blood generally occurs in individuals over forty years of age, and is ordinarily produced by the diseased condition of the arteries, such as arterio-sclerosis, atheroma, or aneurysm. There may also be evidence of chronic heart or kidney diseases, or of syphilis. Sometimes, there may be a history of scurvy, purpura or hæmophilia. Again, pathological hæmorrhage occurs most frequently in the internal capsule due to rupture of the lenticulo-striate and optic arteries known as Charcot's " arteries of cerebral hæmorrhage."

It must be borne in mind that a slight injury on the head may cause cerebral hæmorrhage in a person previously predisposed to it from age or disease, and that the head may be injured during a fall from cerebral hæmorrhage caused by disease.

It is possible for the diseased cerebral arteries to rupture from mere excitement caused by alcohol or struggle, but it is rare in the young and healthy, unless such excitement is associated with extreme congestion of the cerebral vessels. The spontaneous rupture is, however, contra-indicated if there is any evidence of violence, such as a bruise or a wound on the scalp or a fracture of the skull.

2. It is difficult to give the exact date of the effusion of blood, but an approximate idea may be formed from its colour and consistence as to whether it is recent or old. The colour of a recent effusion is red, which changes to chocolate or brown after some days, and turns to an ochre colour generally in from twelve to twenty-five days. The consistence of the coagula becomes firmer and more or less laminated with the progress of time, and the compressed lymph may be between the laminæ or around the coagula. Owing to the blood clot resting upon the surface of the brain a depression equal to its size and shape is formed on the brain substance. Wilfred Trotter[1] quotes a remarkable case where an area of the brain that had been bruised by the glancing contact of a bullet with the skull no less than 4 years earlier showed a bruise that appeared as fresh as if it had been inflicted within a few weeks.

FACE

Wounds of the face heal, as a rule, rapidly owing to its great vascularity, but they are grievous if they are severe and cause permanent disfiguration or deformity.

Face Bones.—The nasal bones are often fractured by a blow with a fist or a blunt weapon such as a *lathi*. When causd by considerable force they may involve the fracture of the ethmoid bone and its cribriform plate forming part of the base of the skull, and may cause death by meningitis. Fractures of the superior maxillæ, malar bones and the mandible (inferior jaw) are produced by a blow with a blunt weapon, such as a heavy stone. Sometimes, in addition to the fractures of these bones the whole face is reduced to a pulpy condition when struck with a heavy stone slab.

A young Mahomedan woman was beaten to death by her husband with a heavy brick. The face was pulverized owing to the bones having been fractured into several pieces. The right eyeball was dislocated, and the brain substance was exposed.

1. *Lancet, May* 10, 1924, *p.* 936.

Eyes.—Injury to the eye, *e.g.*, a lacerated wound produced by a blunt weapon or by throwing a brickbat may damage the tissues so severely as to necessitate the enucleation of the eyeball. A blow on the eye with a blunt weapon may cause a permanent injury to the cornea, iris or lens,

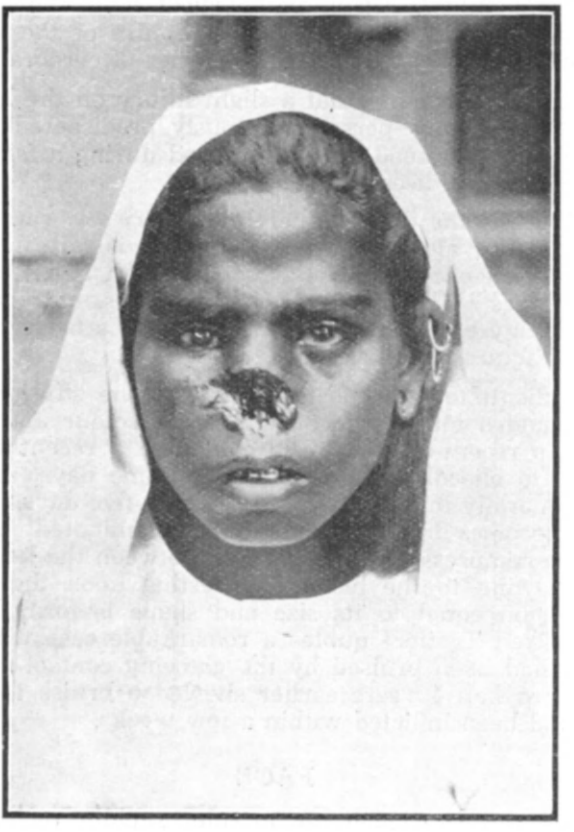


Fig. 95.—Nose cut off by a knife.

hæmorrhage into the vitreous or a detachment or rupture of the retina and even traumatic cataract. The injury may prove fatal from the inflammation of the orbital tissues extending into the brain, and the consequent formation of pus. Similarly, a penetrating wound of the orbit may prove fatal by setting up meningitis through penetration of the thin orbital plates. Neuralgia and temporary or permanent amaurosis may result from paralysis of the upper eyelid, when there is a wound of the eyebrow.

The eyes can be gouged out with the fingers, but in this connection it should be remembered that birds of prey generally attack first the eyes of a dead body when exposed in a field or jungle.

One Sunjoo *Fakir* of Nuddea tied both the hands of his wife, a child of about 12 years, and then gouged out her eyes by thrusting his fingers, as she protested

when he attempted to have connection with her under a tree in some waste land away from his house. He was sentenced to sixteen years' imprisonment.[1]

Dr. A. N. Verghese,[2] Medical Officer of Palghat, reports a remarkable case of gouging out of a right eyeball. In an altercation that arose over a pack of playing cards on the 28th April, 1924, two brothers attacked one Gopal Krishna Menon, aged about 27 years. One held the victim tight above the waist keeping the extended arms in the hold, while the other got behind, fixed the victim's head with his left arm, thrust his right index finger in, and pulled out the right eye. On examination the right eyeball was found pulled out of its socket breaking the optic nerve and tearing asunder the muscles. It hanged out on a few shreds of the external portion of the conjuctiva and the rectus muscle. The socket was filled with blood clots.

It is said that insane persons, sometimes, gouge out their own eyes by enucleating them with their fingers.

A Sadhu (ascetic), known by the name of Shambhu Bhola Baba and residing in a cottage on the banks of the river Narbudda near Jubbulpore, gouged out both his eyes. On being asked by his disciples as to why he tormented himself in this fashion, and deprived himself of his eye-sight, the Sadhu replied that since the eyes were the cause of all sorts of mental and physical sins, he did not think it wise to keep such sinful things with him.[3]

Goodhart and Savitsky[4] report a case of self-mutilation in chronic encephalitis in a girl, aged 16 years, of Russian-Jewish parentage, consisting of avulsion of the eyeballs and extraction of teeth, all but seven of which she pulled out in the course of two years.

Fig. 96.—Nose bitten off by teeth.

Nose.—In India, the nose is technically considered a symbol of honour and reputation. Hence during a quarrel it receives the first attention of an opponent. The nose is also cut off or bitten off through enmity, vengeance and sexual jealousy, the victim being usually a female and occasionally a male. Wounds of the nose are grievous if they leave permanent difigurement or deformity. A blow on the head sometimes causes bleeding from the nose due to partial detachment of its mucous membrane without any injury to the nose. An extensive lacerated wound of the head may lead to loss of the sense of smell, and a penetrating wound of the nose caused by thursting a sharp pointed instrument up the nostril may result in death by injuring the brain through the cribriform plate of the ethmoid bone, though no sign of any external injury is visible.

1. *Chevers, Med. Juris., Ed. III, p.* 484.
2. *Madras Med. Jour.; Medico-Legal Jour., Vol.* 41, *No.* 6, *Nov.-Dec.*, 1924, *p.* 164.
3. *Bombay Sentinel, Aug.* 26, 1937.
4. *Amer. Jour. Med. Science, May*, 1933; *Brit. Med. Jour., Epitome, Sep.* 23, 1933, *p.* 49.

The left nostril or the septum of a female is liable to be injured by pulling at the nose ring worn by he

Ears.—A blow over the ear may produce rupture of the tympanum leading to temporary or permanent deafness. A police constable complained that he was slapped over his left ear by a station master on May 9, 1933. On examination of his ear on the next day the tympanic membrane was found ruptured and the surrounding surface was congested. If a blow over the external ear is very severe, it may also injure the labyrinth. During a quarrel the ears may be bitten off or cut off, and their lobes may be torn by pulling at the earrings either with the intention of causing hurt or committing theft. The injuries are grievous if they produce permanent dsifiguration.

Lips.—Injuries to the lips are caused by a blow with a fist, a shoe, or a blunt weapon, or by teeth bite. Sometimes, a half of the upper lip

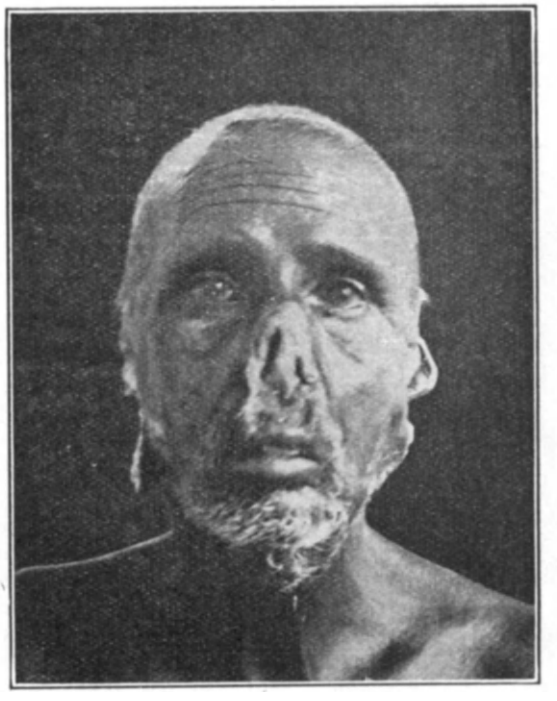


Fig. 97.—Nose and ears of a man cut off by dacoits: Front view. (*Dr. Manglik's case*).

along with a portion of the moustache is cut off, the motive being sexual jealousy. Such injuries are grievous if they cause permanent disfigurement.

Teeth.—The teeth are dislocated or fractured either by a fall or by a blow with a blunt weapon, such as a fist, a shoe, the butt end of a *lathi,* etc. When their dislocation or fracture is caused by mechanical violence, contusions or lacerations are, in all probability, found on the

lips or on the gums or sockets. In India, false reports about the loss of a tooth are often made with a view to charge the accused with the offence of grievous hurt, especially when an assaulted person happens to be old, and has already lost some teeth or has got some shaky teeth. It is, therefore, necessary that the following points should be taken into consideration when reporting on a person who alleges to have his tooth knocked out :—

1. The condition of the neighbouring and other teeth as to whether they are firm, shaky, or diseased.

2. The number of the teeth present in each jaw.

3. The condition of the socket of the missing tooth, as to whether there is any stump left if a tooth is fractured, whether there is any bleeding and whether there is any laceration.

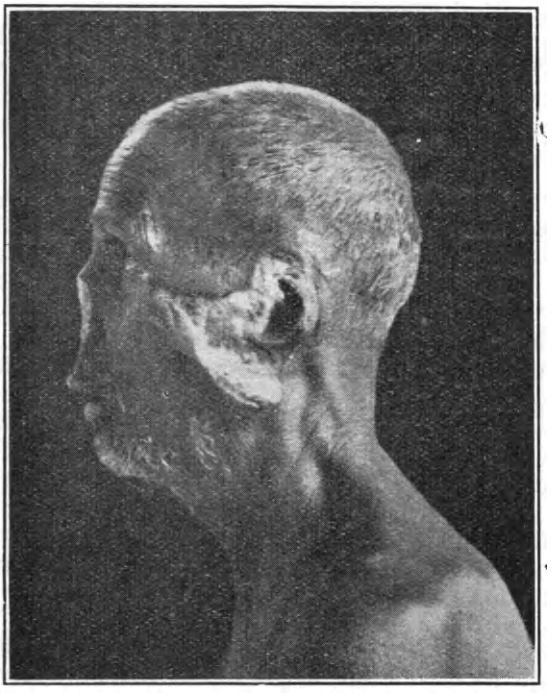


Fig. 98.—Nose and ears of a man cut off by dacoits : Side view. (*Dr. Manglik's case*).

4. The condition of the lips and gums as regards the presence of injury.

5. If a tooth is sent with the injured person, it should be examined to ascertain if it corresponds to the missing tooth. Its fangs should be especially examined to find out if fracture or dislocation has occurred. After examination the tooth should be returned in a sealed packet to the police constable accompanying the injured person.

NECK

Wounds of the neck are mostly incised. In India, they are more often homicidal than suicidal, and very rarely accidental. They are supposed to be instantly fatal, if the large blood vessels of the neck are cut, but this is not always so, as some cases have been recorded in which persons ran a certain distance after the carotid arteries as well as the internal jugular vein had been cut. Wounds of the larynx, trachea and œsophagus are not necessarily fatal, if the large blood vessels are not injured. They may, however, cause death by suffocation due to the flow of blood into the air-passages, though most of it is coughed up. They may also cause death from subsequent œdema or inflammation blocking the air-passages or from septic pneumonia. A woman, 17 or 18 years old, received an incised wound across the upper part of the front of the neck, involving the larynx and œsophagus. The wound healed resulting in stenosis of both. Both tracheotomy and gastrostomy were later performed, but she died of broncho-pneumonia three months after the wound was inflicted on her neck. G. D. Scott[1] reports the case of a man, æt. 47, who was successfully operated for a cut throat involving the complete severance of both the trachea and the œsophagus caused by glass from the windshield in an automobile wreck.

In the case of a wound of the larynx, speech is possible if the wound is above the vocal cords, even if it is gaping. But in a wound of the larynx below the vocal cords, and in that of the trachea, no speech is possible. In such a case one may be able to speak in a whisper if the wound is not gaping sufficiently to allow air to pass into the mouth. Prof. Harvey Littlejohn[2] describes the case of a woman, æt. 45, who after making a transverse incision, 2½" long, in the front of the neck cutting the trachea completely through 2" below the vocal cords, was found sensible, and said that she had torn the tumour out of her neck because it was choking her, and that she wanted to die. On the bed was a small tumour which was encapsuled and consisted of the right lobe of the thyroid gland, hypertrophied, and of fibrous consistence. She was removed to hospital, but was dead on arrival. At the post-mortem examination the upper end of the divided trachea projected from the wound, along with the œsophagus. The protruding œsophagus measured 6¾".

Wounds of the sympathetic and pneumogastric nerves may be fatal, and those of the recurrent laryngeal nerves cause aphonia. A forcible blow on the front of the neck may cause unconsciousness or even death by a reflex inhibitory action or by a fracture of the larynx usually involving the thyroid and cricoid cartilages, and consequent suffocation from hæmorrhage or œdema of the larynx.

A man was brought to me on the third day of his receiving a blow over Adam's apple. On examination a swelling was found over the right side of the thyroid cartilage, and the laryngoscopic examination revealed the presence of submucous hæmorrhage in the larynx on the right side involving the right vocal cord and ventricular band as also the epiglottis.

1. *Jour. Amer. Med. Assoc., March* 3, 1928, *p.* 689.
2. *Forensic Medicine,* 1925, *p.* 196.

SPINE AND SPINAL CORD

Wounds and injuries affecting the spine and the spinal cord are generally accidental, are occasionally homicidal and are rarely suicidal.

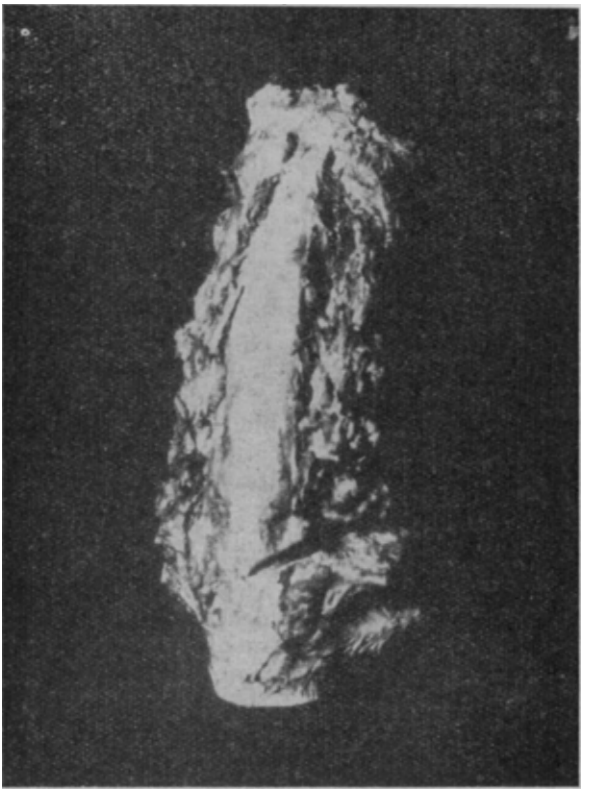


Fig. 99.—Cervical vertebræ showing a cut by a *gandasa* (chopper).

Fractures of the Spine.—These are produced by (1) direct violence, *e.g.,* a blow on the back with a heavy blunt weapon or a fall from a height on the back over some hard projecting substance, and (2) indirectly by forcible bending of the body or by a fall on the head. They may, in certain cases, be produced by a sudden forcible twisting of the neck, as during wrestling. They may also be caused by a very slight twist especially if a person happens to be suffering from Pott's disease.

In January, 1912, a Hindu male, about 20 years old, took a somersault in wrestling and died immediately. On examination a dislocation of the fifth cervical vertebra was found. There was no external injury, nor was there any disease of the vertebræ. A similar case occurred in August, 1912, where a Mahomedan male, 20 years old, died from a fracture-dislocation of the third cervical vertebra caused by a sudden powerful muscular contraction of the neck during wrestling.

These fractures are generally associated with dislocations except in injuries of a minor degree, such as fractures of the spinous processes,

laminæ, etc. Owing to the displacement of the parts they cause compression, laceration or crushing of the cord, which produces paralysis of the body below the seat of injury. In such cases hæmorrhage occurs in the substance of the cord, or around it, between or outside its membranes. These cases are very rare. In the Agra District during twelve years, out of about one thousand medico-legal autopsies death was found to be due to the fracture of the spine in only five cases.

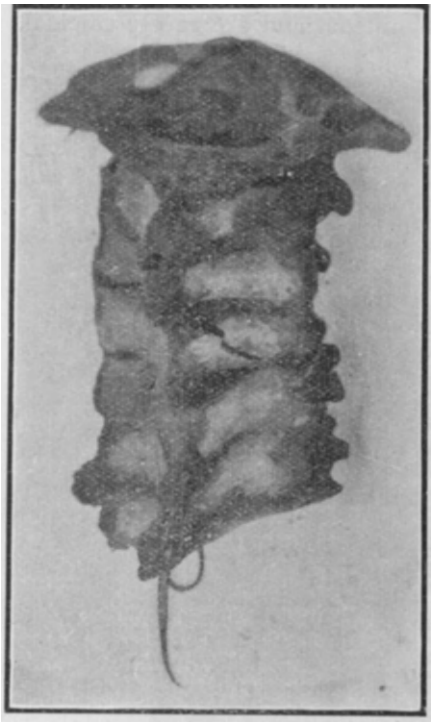


Fig. 100.—Cervical vertebræ showing fracture of the 4th cervical vertebra.

Spinal injuries are, as a rule, immediately fatal, owing to implication of the phrenic nerves, if the fracture occurs above the fourth cervical vertebra, though death may be delayed a few hours even after fracture of the odontoid process of the axis with forward displacement of the atlas. Death usually occurs within twenty-four hours if the three lower cervical vertebræ are injured. In rare cases death may not occur for some months, but the trunk and the limbs will be paralysed, if the spinal cord is compressed by displacement of the fractured portions.

A boy, 10 years old, regularly attended school, and took part in games for five weeks dislocating his neck. He merely complained of stiff neck and it was only when he made a sudden movement with his neck that the cervical cord became compressed, and he died immediately.—*W. G. A. Robertson, Practitioner, Aug.*, 1923, *p.* 121.

A Mahomedan male, aged 60 years, who was knocked down by a motor car on September 17, 1932, sustained a dislocation of the third cervical vertebra from the fourth cervical vertebra with a transverse fracture of the body of the latter, suffered from loss of sensation and paralysis of all the limbs, and died on November 20, 1932.

When the dorsal vertebræ are injured, the patient becomes bed-ridden on account of paralysis of the lower limbs. He also suffers from paralysis of the bladder and rectum, and is always in danger of getting bed-sores and septic infection of the bladder and kidneys, which generally hasten death. Thus, death may occur after two or three weeks if the upper dorsal vertebræ have been injured; while life may be prolonged for years with partial paralysis of the limbs if the lower dorsal or the lumbar vertebræ have been fractured.

Alfred Master[1] describes an unusual case of a fractured spine caused by a sudden muscular strain. A clerk, aged 47, was helping unload some heavy bags of money from a taxi outside his office one morning in April, 1932. While lifting a particularly heavy bag from ground, he suddenly felt something snap in his back, and collapsed on the floor. At the time he felt "the use go out of his limbs" below the waist for a few moments, but this passed off, though he still complained of a severe pain in the lumbar region of the spine. After resting for a little while in the office he was able to travel home in bus unattended, having fastened his belt firmly round his waist. He continued in bed for three weeks, and after another month's convalescence he returned to duty. He continued his office work for twelve months, apparently in good health, except for occasional pain in the lumbar region and a jarring sensation down the spine on walking downhill. Owing to the persistence of the pain he sought medical advice, and the X-ray examination revealed an ununited fracture of the body of the third lumbar vertebra.

Concussion of the Spine.—This condition may occur without any evidence of an external injury to the spinal column. It may follow a severe blow on the back, or a jar, or a fall from a height. This is the most common form of injury met with in railway collisions, and is then known as "railway spine."

The symptoms may develop immediately or may be delayed for some weeks. The patient becomes restless, excitable and emotional and generally suffers from nerve prostration or neurasthenia. He complains of pain and tenderness over the spine and weakness in the limbs. Hence he is unable to walk. He also complains of amnesia, and derangement of the special senses. These symptoms are exaggerated very much by any kind of mental excitement, *e.g.*, during the time of medical examination. Most of the symptoms being subjective, it is difficult for a medical practitioner to determine whether the patient is feigning or not. It has often happened that the symptoms have abated immediately after a civil suit for damages, brought by the patient against his employer or a railway company, has been decided in Court.

Being well protected by anatomical structures, incised or punctured wounds of the spinal cord are rare except between the first and third cervical vertebræ, where they are more exposed owing to the narrowness of the laminæ. A punctured wound caused in this region even by a small needle proves almost instantaneously fatal, as it injures the medulla and the upper part of the cord which contain the respiratory and other vital centres. The process of killing in this manner is called *pithing* and the wound caused is so very small, that it may be overlooked altogether if the weapon is thrust obliquely.

CHEST

Injuries of the chest are mostly accidental, occasionally homicidal, and rarely suicidal.

Traumatic Asphyxia.—This results from severe compression of the chest and abdomen sufficient to prevent respiration for an appreciable length of time, as when an individual is crushed in a dense crowd or under a heavy object, or caught between two buffers of a railway carriage. In such cases the face and neck are deeply cyanosed, accompanied by ecchymoses of the skin and conjuctivæ. This discoloration is brought about

1. *Brit. Med. Jour., Nov.* 24, 1934, *p.* 976.

by mechanical overdistension of the smaller veins and capillaries with stasis of deoxygenated blood. It extends to the root of the neck, and rarely passes down beyond the level of the clavicles owing to the absence of competent valves in the jugular and facial veins. The discoloration may disappear in ten to fourteen days without passing through the colour changes of a bruise, if it is not associated with severe injuries. Coullie[1] describes the case of an epileptic young man, who suffered from traumatic asphyxia caused by the unyielding collar-band of his shirt compressing the jugular veins, together with the partial asphyxia, high blood pressure, and fixation of the chest caused by the epileptic fit.

Wall.—Contusions and abrasions of the chest wall may be caused by a blunt weapon, fall or crush under a heavy weight as in vehicular accidents. These may be accompanied by fractures of the ribs or sternum, or associated with grave visceral injury. Even when not accompanied by such injuries severe blows on the chest wall may produce concussion of the chest causing considerable shock followed by death.

Simple contusions and abrasions of the chest wall may be followed by pleurisy or pneumonia.

Wounds of the chest wall are not dangerous unless the cavity is penetrated and a vital organ is injured. In non-penetrating wounds there may be free hæmorrhage from the divided mammary and thoracic arteries.

Ribs.—Fracture of the ribs results from direct violence, as by blows or stabs, and from indirect violence as in compression of the chest or very rarely from muscular contraction during violent coughing. When due to direct violence it is more dangerous, as the splinters are driven inwards, and are likely to injure the underlying pleura, lungs, heart, large vessels, liver, or diaphragm while, in indirect violence, the fracture occurs at the most convex parts of the ribs near their angles, and the fragments are driven outwards. The ribs that are most frequently fractured are the middle ones, *viz.*, the fourth, fifth, sixth, seventh and eighth, as they are most prominent and fixed at both ends. The upper ribs are not usually fractured, unless very great force is used, when the lesions of the viscera, as a rule, occur. The lower ribs often escape on account of their greater mobility. Owing to diminished elasticity and increased brittleness, fracture of the ribs takes place more easily in the old than in the young and healthy.

Symmetrical fractures of the ribs on both sides are often met with when a person sits on the chest and compresses it considerably by means of the knees or elbows, by trampling under feet, or by means of two bamboos, a process known as *bans dola.* They may also occur in accidents as in a fall from a height, or when run over by a heavy bullock cart or motor car or when caught between railway buffers. In such cases the ribs are often fractured in front near the costal cartilages, where the compressing force is applied, and near the angles at the back, the force travelling along the ribs. These are not always accompanied by external injuries or ecchymoses of blood in the soft tissues over the ribs.

1. *Brit. Med. Jour., Sep.* 29, 1928, *p.* 569.

Sternum.—Fracture of the sternum is rare. It is ordinarily due to direct violence, and usually occurs transversely either between the manubrium and gladiolus or a little below this level. If depressed it becomes serious, as it is liable to damage the viscera behind it.

Lungs.—Wounds of the lungs may be immediately fatal from profuse hæmorrhage, or from suffocation due to respiratory embarrassment on account of the presence of blood in the pleural cavity or in the air-passages, or may result in death subsequently from septic pneumonia. They may be produced by the penetrating wounds of the chest caused by a cutting or stabbing instrument, by the sharp fragments of a fractured rib, or by a projectile from a firearm. The hæmorrhage is recognised by the escape of bright red and frothy blood from the mouth, and from an external wound if present.

Contusions or lacerations of the lungs may be produced by blows with a blunt weapon or by compression of the chest even without fracturing the ribs or showing marks of external injury. These may cause instantaneous death or may result in pleurisy, traumatic pneumonia or hæmothorax.

In June, 1919, a girl, 3 years gold, was run over by an *ekka* and died immediately. At the autopsy there was no external mark of injury to the chest, nor were the ribs fractured, but the left lung was found lacerated.

In February, 1922, a Mahomedan girl, 15 years old, received a kick on the chest from her husband, and died within an hour. On examination no external injury was visible, but there was a laceration of the lower lobe of the left lung which was fibroid from disease.

The body of a Hindu female was found lying near the railway line near Alambagh on or about the 11th November, 1925. The post-mortem examination showed a bruise, 3" × 1", obliquely across the left side of the chest 3" below the left collar bone, but no fracture of any ribs. The right lung was lacerated in front 1" below the apex, and a contusion, 2" × 2", was found on the base of the left lung. These appeared to have been caused by compression of the chest.

A Hindu male, 25 to 30 years old, was crushed under a machinery in a workshop on January 3, 1929, and died on the next day. The post-mortem examination did not show any marks of external violence on the chest or fracture of the ribs. The chest cavity contained blood, and the right lung had four contusions on its anterior aspect, while the left lung showed a contusion of its root and a tear, 2½" long, over its lower lobe. There was also a dislocation of the fourth cervical vertebra.

Heart.—Wounds of the heart are produced by a cutting or stabbing instrument, a bullet or a sharp end of a fractured rib or sternum. These wounds are commonly instantly fatal from shock and hæmorrhage except in a few cases, where the individual has been able to walk some distance, and has prformed some other volitional acts after receiving the injury. Strassman[1] reports a case where a man was stabbed in the left fourth intercostal space with a knife which, penetrating the thorax, caused a wound, ¼" wide, in the left ventricle. He lived for four days, and on the day following the receipt of the wound he lifted heavy weights. Coats[2] reports the case of a girl, ten years old, who survived nine days after receiving a penetrating wound in the right auricle through the fourth

1. *Lehrb. der ger. Medicin,* 1895 ; *Dixonmann, Forens. Med. and Toxic., Ed. VI, p.* 232.
2. *Glasgow Med. Jour.,* 1891 ; *Ibid., p.* 232.

costal cartilage on the right side of the chest as the result of a fall on an iron railing. Magnus C. Peterson[1] reports the case of a man, aged 26, who survived eighteen months after a safety pin was thrust in his heart with a view to commit suicide. The pointed end of the brass pin was protruding into the pericardial sac from the left atrium on the surface of which it formed a shallow depression. The pin, which was about 12 cm. in length and 1.2 mm. in diameter, penetrated the left atrium and curved over on the right side of the vertebral column in a slightly downward dirction. Chevers[2] also mentions the remarkable case of a soldier who was wounded by a shot in the heart on the 14th April, 1852, and died on the 24th June. On the other hand, some cases have been successfully treated by surgical operation. E. M. Freese[3] describes a case of recovery from a stab wound through both ventricles of the heart. A coloured man was stabbed about 8 p.m. on the 2nd September, 1920, and was taken to Grant Hospital in half an hour. He was unconscious, the respirations were feeble and very shallow, and the pulse was not perceptible, either in the radial or carotid artery. The pupils were dilated, and the skin was bathed in cold perspiration. On examination a wound, ½" long, was revealed in the fourth intercostal space 2" to the left of the sternum. On opening the chest the pericardium was filled by a clot which produced almost a complete tamponade. When the clot was scooped out, the heart began at once to beat violently, spurting a stream of blood on the anæsthetist and over the field. Recovery occurred after the wounds were sutured. S. S. Sen[4] describes the case of a young Mahomedan male who was stabbed in a street and was at once brought to the General Hospital, Rangoon. On opening the chest the pericardium was found to be cut, and a large amount of blood clot was removed from the pericardial sac. It was then found that the weapon had also penetrated the left ventricle, where a large blood-clot had fortunately prevented the escape of serious quantities of blood. This clot was removed and the heart-muscle wound sutured with fine catgut; the pericardium was also closed, and the wound in the skin sutured in layers. The patient was treated on the usual lines for shock, and he made an uneventful recovery.

It is possible that foreign bodies, such as bullets, sharpnel, or fragments of shells, may remain embedded in the myocardium for months or years without the production of symptoms. In such cases it is probable that the original injury was relatively slight and that the missile, by acting as a plug, effectively checked any severe hæmorrhage. Gilchrist[5] describes two cases in which missiles were found embedded in the muscle of the left ventricle of the heart by X-ray examination thirteen years after wounding. Both men were in good health and fit for active work.

Danger to life depends upon the nature of the wound. If it is small and passes obliquely through the wall so as to act as a valve-like flap or if a weapon happens to plug the orifice, life may be prolonged for some hours, days or even months. On account of their thinner muscular walls, wounds of the auricles are more dangerous than those of the ventricles.

1. *Jour. Amer. Med. Assoc., May* 11, 1929, *p.* 1599.
2. *Med. Juris., p.* 474.
3. *Jour. Amer. Med. Assoc., Feb.* 19, 1921, *p.* 520.
4. *Ind. Med. Gazette, Sept.*, 1931, *p.* 508.
5. *Brit. Med. Jour., April* 20, 1929, *p.* 723.

The right ventricle is more likely to be wounded, as it exposes its widest area on the front of the chest.

On September 22, 1919, the body of a Hindu male, 26 years old, was brought to the King George's Medical College Mortuary. On examination an arrow was found sticking in the left side of the chest, causing a penetrating wound, 1" × ¾", obliquely across the fourth intercostal space 1" below and internal to the left nipple, and through the wound a small portion of the lung was protruding. On opening the chest the arrow was found to have lacerated the left lung and penetrated the right ventricle, having pierced through the pericardium and the left ventricle above the apex of the heart.

At the post-mortem examination on the body of one Chitaoo, aged 45 to 50 years, on January 28, 1934, thirty-six hours after death, I found among other injuries an incised wound, 1" × ¼", across the chest wall in the left second intercostal space and to the left of the middle line. On opening the chest the blunt end of an arrow head was seen projecting out of the wound, 1" × ½", through the upper portion of the left lung behind the external wound. On further examination the sharp blade of the arrow head was found to have traversed through the pericardium, left pulmonary artery, and left auricle, and had penetrated into the substance of the left lung below its root.

Rupture of the heart is usually caused by a blunt weapon, by a carriage wheel running over the chest or by a fall over a hard projecting surface. In such cases the heart is usually ruptured on its right side and towards its base, and the ribs and the skin over them are damaged. But in rare instances rupture of the heart may occur without leaving any external mark of violence or causing fracture of the ribs.

Surgeon Major Gibbons[1] reports a case where a cooly, aged about 30 years, died in three hours and ten minutes from rupture of the heart over the apex caused by a blow with a bamboo stick. The rupture was ¼" long and irregular in shape, and communicated with the right ventricle. Externally there was an elliptical abrasion, 2¼" × ¾", over the left fourth and fifth ribs below and inside the left nipple but no fracture.

Ingle[2] also describes a case of a motor car accident in which a young motor car driver sustained two tears admitting the tip of the index finger over the posterior aspect of the right ventricle. There was a contusion, 3½" by 3", over the front of the chest, but there was no fracture of the ribs or vertebræ.

The following cases are given from my notes :—

1. An old Hindu widow was run over by a cart on the 2nd July, 1922, at Lucknow. The post-mortem examination showed an irregular rupture of the right ventricle of the heart without any fracture of the ribs or external injury on the left side of the chest. The second, third and fourth ribs were, however, fractured on the right side.

2. A male child, 1½ years old, was run over by a tonga on the 7th October, 1922, and died immediately. The autopsy revealed a contusion of the pericardium and the right chamber of the heart and lacerations of both lungs, but no external injury on the chest or fracture of the ribs.

3. A Hindu male child, aged 8 years, was run over by a bicycle wheel on the 19th October, 1927, and died immediately. On examination a bruise, 5" × ½", was found obliquely across the right side of the chest, and a contusion, 1" × ⅛", with a rupture, ½" × ⅛", was seen across the front of the right auricle of the heart.

The heart may also rupture spontaneously from sudden exertion if it has already been diseased. Patients are, as a rule, elderly, and the

1. *Ind. Med. Gaz., Dec.*, 1897, *p.* 443.
2. *Ind. Med. Gazette, Aug.*, 1934, *p.* 450.

rupture in such cases occurs mostly in the left ventricle at its apex, though it may occur in the right ventricle or in the auricles. According to Nuzum and Hagen[1] spontaneous rupture of the heart frequently follows obstruction of a coronary artery. Coronary thrombosis invariably results in an infarction of that portion of the heart wall supplied by the obstructed vessel. Pulvertaft[2] reports the following case where a rupture occurred in the right ventricle in a young female of 19 years of age, in whom there was no evidence of cardiac or vascular disease :—

The female had committed suicide in a fit of temper by putting her head in a gas oven. The usual signs of poisoning by carbon monoxide were present ; in particular Tardieu's spots were prominent on the diaphragm and the visceral layer of the pericardium. On opening the pericardium, about 12 ounces of uncoagulated blood were found. A tear, $\frac{3}{4}$" long, was found on the anterior aspect of the right ventricle, about 1" from the apex, and $\frac{1}{4}$" lateral to the interventricular septum. The coronary arteries were normal. There was no chronic endocarditis or myocarditis.

I quote below three cases of spontaneous rupture of the heart from my case book :—

1. In Agra, a Hindu hawker of about 70 years went to a prostitute's house to purchase empty bottles, and after ascending a staircase sat down on a *charpoy* and asked for a glass of water, but he expired before it was brought to him. At the autopsy I found that death was due to rupture of the left ventricle owing to thinning of the heart muscle on account of chronic ulceration.

2. A Hindu woman, aged 65, died all of a sudden. Upon examination, the left ventricle was found ruptured, the muscle being thin with a deposit of fat. There were calcareous ulcerations in the aorta.

3. A Hindu male, 60 to 65 years old, was found dead in a third class compartment of a railway carriage on the thirteenth December, 1933, and his body was removed to the Medical College Mortuary, Lucknow. The post-mortem examination showed that there was no external mark of violence on any part of the body. The pericardium contained liquid and clotted blood. The heart was found contracted and empty. The right auricle was lacerated, the tear being 1" $\times$ $\frac{1}{4}$", along its posterior surface towards the lower part. The opening caused by the tear was covered with a blood clot. The wall of the right auricle was quite thin. The valves of the left chamber were thickened and the aorta was dilated and had atheromatous ulcers. The lungs were bulky and congested. They exuded frothy serum from cut surfaces. The bronchial tubes were dilated.

Lewelyn Hodge[3] records a case in which an unmarried woman, 57 years old, while in bed suffering from a right sided ovarian cyst with free ascitic fluid, complained of sudden nausea and slight precordial pain. She tried to sit up in bed, but collapsed. Her face was pale and clammy, but there was no cyanosis ; she became unconscious in about five minutes and died in ten minutes after the onset of the symptoms. At the necropsy death was found to have been due to a rupture of the right ventricle. The pericardium was full of blood. The heart was small, and was coated with bright yellow fat, which also covered the aorta. At the apex of the right ventricle there was a definite perforation, the size of a small knife-stab. The musculature of the heart in general was extremely thin, that of the apex of the right ventricle practically consisting of the endothelial lining of the heart with an outer covering of about three-quarters of an inch of fat. The walls of the auricles were exceptionally thin. The aortic valves showed slight atheromatous changes, but the other valves were normal.

In all the cases of rupture of the heart that came under my observation death occurred immediately except in the case of a Hindu female, about 50 years old, who died within two to three hours after she was run over

1. *Amer. Jour. of Med. Sciences, Philadelphia, Feb.*, 1926, *p.* 185.
2. *Lancet, Aug.* 6, 1932, *p.* 289.
3. *Brit. Med. Jour., June* 18, 1927, *p.* 1096.

by a motor car on the 4th October, 1928. At the post-mortem examination I found that all the ribs except the twelfth were fractured, and the right ventricle of the heart was lacerated in front, the laceration being 1"×1/6". Both lungs were also lacerated. Leslie Pearce Gould[1] describes the case of a chief boatswain, aged 47, who fell a distance of twenty feet, and sustained an injury to the heart. He died within about six hours. During the interval he was quite conscious and rational, and answered questions intelligently. The examination showed a fracture of the first segment of the gladiolus of the sternum which was running obliquely downwards from right to left. A very small tear was found in the anterior wall of the right ventricle, close to the semilunar valves, large enough to admit a lead pencil into the cavity of the ventricle. Corin[2] records a similar case which survived six days. O'Neill[3] records the case of a boy who died after five days. He had a slit, 3 mm. long, at the auriculo-ventricular valve. Howat[4] mentions a case of delayed traumatic rupture of the heart, described to him by his colleague, Dr. J. Donaldson, as occurring in his practice. A bricklayer, aged 68, unusually deep chested, was working beside a large iron pipe close to which were the rails on which bogey trucks ran. On the approach of a truck he stood with his back pressed close against the pipe in order that the truck might clear him. The truck squeezed the front of his chest, scraping the skin, but causing no further apparent local injury. He was in bed for two weeks and resumed work after three weeks. After three days' resumption of work he felt unable to continue, his chief complaint being pain in the chest. He was confined to bed again for two weeks, during which his pulse-rate rose gradually from 30 to 60. He appeared to be progressing favourably when he suddenly died. The post-mortem examination revealed no injury of any part of the chest wall, except the scraping of the skin. The pericardium was intact and full of blood. No disease of the heart substance was found. The left ventricle alone was injured. Its wall was bruised in five places, three in front and two behind. The largest bruise, the size of a shilling, and extending through the greater part of the wall's thickness, was found near the apex. Here the ventricle wall was ruptured.

Gunewardene[5] reports the case of a boy, aged 9, who survived ten days after sustaining injury to the heart owing to his chest having been pressed against a wall by the back of a double-bullock cart. No external mark of injury or fracture of the ribs was evident at the time of the accident. He felt quite well the following morning, and was, therefore, allowed to go to school, where he took part in the usual games. On the tenth day, while playing at school, he complained of precordial pain and fell down dead. The autopsy revealed a hæmopericardium. On removal of the clot a rupture of the anterior surface of the left ventricle, was seen. The slit was blackish-grey, roughly circular, and about 1" by ¾" in diameter. The rupture was probably due to the contusion of the heart wall at the time of the accident and yielding of the muscle so damaged.

1. *Lancet, Oct.* 13, 1917, *p.* 567.
2. *Bulletin de l' Acad. Royale Belgique ; 4 Med. Series,* 1911, *p.* 562.
3. *Jour. Amer. Med. Assoc.,* 1914, *Vol. LXII, p.* 697.
4. *Lancet, June* 19, 1920, *p.* 1313.
5. *Brit. Med. Jour., Nov.* 24, 1934, *p.* 942.

Blood Vessels.—Wounds of the aorta or the pulmonary artery are rapidly fatal. Wounds of the smaller arteries may prove fatal on account of profuse bleeding. Wounds of the large veins especially of the neck, chest, axilla or groin, may result in death from the air entering the blood and consequently passing into the right side of the heart.

Rupture of the aorta may be traumatic or spontaneous. When due to trauma the rupture is often localized just above the valves, and is more frequently transverse than longitudinal.

A thin man, aged 45, died immediately after he was hit with a *lathi* over the left side of the chest. The aorta which was almost completely calcified, was found ruptured at the junction of the transverse and descending parts. There was no injury to the chest wall or to the ribs.

Spontaneous rupture may occur from local diseases of the aorta, or even when there is little or no change in the aorta, especially in those cases where the aorta has only two valves or there is stenosis of the aortic isthmus.[1]

A case[2] is recorded of a healthy man, aged 48, in whom two large ruptures were found in the aortic arch with a dissecting aneurism and profuse hæmorrhage in the left pleural cavity. The whole of the aortic arch showed areas of necrosis, which were most marked at the site of the rupture. The ruptures had occurred with an interval of a month in each; the last which proved fatal took place when the patient was asleep in bed. There was a history of high blood pressure for a year, but there was no evidence of syphilis or other inflammations.

Diaphragm.—Wounds of the diaphragm are liable to be produced by penetrating wounds of the chest or of the abdomen. They are not rapidly fatal unless the important organs in contact with it are also wounded. In non-fatal cases diaphragmatic hernia may subsequently occur after the wound has healed and a cicatrix has formed.

Rupture of the diaphragm may occur from a blow with a blunt weapon, a violent kick, or a fall on the abdomen when the stomach and intestines are full. Rupture may also take place, when the trunk is compressed under a wheel of a heavy bullock cart, motor car or railway carriage. It is the central tendon that is ruptured most frequently, and death generally occurs from shock.

ABDOMEN

The Abdominal Parietes.—Injuries of the abdominal parietes may be contusions, abrasions, and non-penetrating or penetrating wounds.

Contusions of the abdominal parietes are produced by a blunt weapon a kick, a carriage wheel passing over the abdomen, or by a fall. It is not necessary that they should show any external mark of injury on the skin. Sometimes, an effusion of blood may be seen in the tissues or muscles under the spot where violence was used; but it must be borne in mind that an effusion of blood in the muscles may occur spontaneously as a result of disease without any external violence.

1. *Brit. Med. Jour., March* 31, 1923, *Epitome, p.* 52; *W. M. De Vreis, Nederl, Tijdschr.* v. *Gencesk, Dec.* 16, 1922, *p.* 2713.
2. *Brit. Med. Jour., Aug.* 13, 1932, *Ep.*, p. 29.

Abrasions on the abdominal parietes are generally caused by vehicular accidents or by falls from a height.

Contusions and abrasions of the abdominal parietes are, as a rule, simple, unless accompanied by lesions of the visceral organs, when they prove fatal from shock, hæmorrhage, or from peritonitis. In some cases peritonitis may occur without evident injury to any of the abdominal organs. Besides, it has already been mentioned that a blow on the epigastric region (pit of the stomach) may cause death by its inhibitory action on the heart through the reflex action on the solar plexus. Post-mortem examination would reveal nothing except the signs of shock to account for such a sudden death.

Wounds of the abdomen are produced by a cutting or stabbing instrument, a firearm, the horns or claws of an animal, or by a fall on an iron railing or on a sharp projecting point. They are of two kinds, non-penetrating and penetrating. Non-penetrating wounds are usually simple and heal rapidly, but may be serious from hæmorrhage from some large blood-vessel, such as the epigastric artery, or from septic infection, which, extending to the deeper tissues, may involve the peritoneum and cause peritonitis. A ventral hernia, may, sometimes, occur from the cicatrix left after the healing of the wound.

Penetrating wounds are, as a rule, dangerous, and may cause death immediately from shock or internal hæmorrhage, or subsequently from septic peritonitis. They may occur with or without injury or protrusion of the abdominal viscera. Those wounds in which the subjacent viscera are not damaged usually heal readily, unless they are extensive and the abdominal contents are exposed to the air.

Stomach.—The stomach, especially when distended with food or diseased from ulcer or cancer, may be easily bruised or even ruptured by a blunt weapon, a crush or a fall on the epigastrium, without leaving any mark of external injury on the abdominal wall. The pyloric end and the greater curvature of the stomach are the usual sites of rupture.

At 10 a.m. on the 6th October, 1923, a boy, 8 years old, was run over by a carriage and died immediately. On post-mortem examination there was no external mark of injury, but on opening the abdomen the peritoneal cavity was found to contain a dirty reddish fluid consisting of undigested rice; the stomach was found ruptured across its lower border and greater curvature, half an inch above the pyloric end.

It should be borne in mind that its spontaneous rupture may occur when there is an ulcer in the stomach or sometimes even when there is no evidence of disease. Thus, in one case the stomach was ruptured during a fit of violent vomiting, and in another the rupture occurred when the deceased had been to the water closet. The stomach in both cases was quite healthy.[1] Penetrating or stabbing wounds of the stomach are generally fatal, and very often involve the adjoining viscera, such as the liver or the spleen.

On the 23rd October, 1921, a Hindu male was wounded in the stomach, and he died on the 26th October. A penetrating wound passed through the stomach into the right lobe of the liver.—*Oudh J. C. Court, Cr. App., Reg., No. 2, 1921.*

1. *Taylor, Princ. and Pract. of Med. Juris., Vol. I, Ed. IX, p. 445.*

Intestines.—Rupture of the intestines occurs frequently from violent blows, kicks, falls, crushes or compressions. In many cases no mark of injury on the abdominal wall is visible though in addition to the rupture a great deal of contusion and laceration of the intestines may be present. Like the stomach the intestines may rupture spontaneously from chronic ulceration or from very slight force, if they are diseased or distended. Moir[1] describes a case where a patient sustained a rupture of the small intestine 3½ feet from the ileo-cæcal valve when he attempted to reduce an inguinal hernia by using considerable force.

When caused by injury the rupture may take place at the point of impact, or in some cases, at a distance from it. In the former case the margins of the rupture are clean cut, and in the latter they are usually ragged and irregular. Rupture usually occurs at the commencement of the jejunum, and in the lower three feet of the ileum, but very rarely in the large intestine. However, it must be remembered that rupture of the large intestine at the junction of the sigmoid with the rectum may occur from straining at stool without the presence of chronic ulceration or any other disease.

Death occurs immediately from shock or subsequently from peritonitis owing to the expulsion of the contents into the peritoneal cavity. In three out of ten cases of mechanical violence to the small intestine that came under my observation, death occurred from peritonitis on the third, fourth and sixth day respectively after the rupture. In one case the intestine was diseased and it was the chronic ulcer that had given way.

If a puncture is very small the mucous membrane becomes everted and closes the little opening and thus prevents the escape of the intestinal contents. The power of locomotion or other muscular exertion may be preserved after these injuries.

Rectum.—Fatal injuries of the rectum are, sometimes, produced by the forcible thrusting of a blunt weapon through the anus, a method of torture, which is occasionally resorted to in India for adultery and theft.

Roy Chowdhury reported to me a case, in which he examined the body of one Bhogola, aged 11 years, and he found that a bamboo, 12 inches long, had been thrust through the anus into the abdominal cavity, where it produced a tear of the transverse colon and then entered the pleural cavity tearing through the diaphragm.

Severe injuries of the rectum may also occur accidentally from falls on an iron railing, or any projecting point, or from forcibly sitting upon a piece of a broken bottle or broken china.

Pillai[2] describes the case of a Burman male who sustained a penetrating wound of the anus by falling from a paddy heap, 14 feet high, on to a forked stick used for supporting a country cart. The wound involved the anus and surrounding skin. Two loops of the small intestine, each about 1¾ feet long, with a piece of omentum, about 1½ feet long, were protruding through the wound and lying loose over the perinæum. The gut was lacerated, gangrenous and offensive. On examination by the fingers a gaping wound was felt on the right side of the rectum, extending into the abdominal cavity. The abdomen was tympanic and tender. Recovery took place after an operation.

1. *Brit. Med. Jour., Oct.* 8, 1921, *p.* 563.
2. *Ind. Med. Gaz., Sep.,* 1933, *p.* 519.

It must be remembered that a column of air under pressure rushing from the nozzle of a compressed air hose which does not touch the body may enter the bowel through the anus and cause fatal injury. In such cases the sigmoid is usually injured, the anus and rectum escaping.

Block and Weissman[1] cite a case where a man, aged 45, received the following injuries when one of his fellow workers, as a practical joke, placed the nozzle of the air hose about an inch from his rectum :—

An irregular perforation with a diameter of about 25 mm. of the intestine slightly above the junction of the sigmoid with the rectum on the left side together with a laceration of the mesosigmoid about 50 mm. long.

Fæcal matter escaped into the peritoneal cavity through this opening. The patient recovered after the necessary operation.

An apprentice rivetter after unscrewing his hammer from his compressed air pipe, blew air at a boy, aged 15, as a joke. Another apprentice had his arm on his shoulder, but could not say whether he was bending or not. The boy said " Oh " and collapsed. The jury was satisfied that the boy died by a blast of compressed air entering the anus from a distance of 6". The air pressure in the nozzle of the pipe used was one hundred pounds to the square inch.[2]

Pancreas.—Wounds of the pancreas are extremely rare. They may occur from direct violence applied to the epigastrium or from penetrating wounds of the abdomen. They are usually accompanied by injuries of the other abdominal organs. But when the stomach is empty, the pancreas alone may be ruptured by being pressed against the spinal column by the object struck. MacLeod and Harvey[3] each mentions a case of rupture of this viscus caused probably by kicks or trampling with the feet. In one case contusions, though not visible externally, were present on both sides of the spine. Mocquot and Constantini[4] describe a case of complete rupture of the pancreas in a bicycle accident. The shock was intense, and the youth died on the twenty-fourth day. In a case in which a young man was run over by a *tonga* and died on the eighth day I found a contusion of the pancreas towards its tail. There were also contusions of the bases of the lungs and of the front of the transverse colon. The left kidney was ruptured in about its middle, lacerating its substance with the capsule intact on its posterior surface. There were three small abrasions externally on the chest but no injury on the abdominal wall.

Gray and Hodgson[5] report the case of a man, aged 20, who while playing football, was charged by another player and fell to the ground with his opponent on top of him. The latter's knee hit him on the abdomen. Within fifteen minutes of the accident he suffered from shock and died in forty hours. The post-mortem examination revealed the presence of a tear of the pancreas anterior to the vertebral column. There was no tear of any other organ or no bruise of the abdominal wall. On the other hand, Brown and Barlow[6] describe a case of complete division of the

1. *Jour. Amer. Med. Assoc., March* 22, 1926, *p.* 1597.
2. *Jour. Amer. Med. Assoc., Dec.* 8, 1928, *p.* 1816.
3. *Lyon's Med. Juris., Ed. IX, p.* 216.
4. *Revue de Chirurgie, Paris,* 61, 1923, *p.* 21 ; *Jour. Amer. Med. Assoc., Aug.* 11, 1923, *p.* 510.
5. *Brit. Med. Jour., Feb.* 14, 1931, *p.* 264.
6. *Jour. Amer. Med. Assoc., May* 28, 1932, *p.* 1882.

pancreas, followed by recovery through an operation, from an automobile accident without serious injury.

Liver.—Owing to its size, its fixed position and its friable consistence, the liver is frequently wounded from a stab in the abdomen, or is often ruptured from a blow, kick, crush, or fall, or even from a sudden contraction of the abdominal muscles. It may also be lacerated by the fractured end of a rib perforating the diaphragm. There may or may not be signs of external injury. Fifteen cases of rupture of the liver came to my notice during a period extending over eight years. Out of these, external marks of injury were visible in three only. One of these cases was very remarkable. A young Mahomedan male of twenty-two years was run over by a motor car, and died within an hour. At the post-mortem examination which I held on the day after the death I found no trace of external or internal bruising of the abdominal wall, but the liver was crushed and the right lobe was almost pulverized.

Ruptures usually involve the right lobe, and occur at the anterior surface and the inferior border. They are ordinarily directed antero-posteriorly or obliquely, rarely transversely, and are generally one or two inches deep, but rarely pass through the entire substance of the organ. The liver is lacerated more easily if it is enlarged and fatty. In ruptures of the liver death occurs immediately from shock and hæmorrhage, especially if the portal vein or vena cava is injured, or it may occur within forty-eight hours. Sometimes, life may be prolonged for days if the liver substance alone is injured. A case occurred in Agra in which a man survived five days after the liver was ruptured. In a case reported by Wilks death occurred on the tenth day after the rupture of the liver.[1] Sometimes, recovery occurs after slight wounds or lacerations. It is also possible that the rupture may lead to the formation of liver abscess through septic infection. From his observations Bauer[2] thinks that the abscess may occur from eleven days to one year after the central rupture of the liver as the result of compression. In rare cases, it may occur even four and ten years later. He describes a case in which a man fell into a hole and fractured his right fifth rib. For months he complained of local pain and pain on breathing, and held himself so bent that he was ordered a corset. A year later he was suddenly taken ill with rigors and high fever, and died two weeks later. At the necropsy a liver abscess was found.

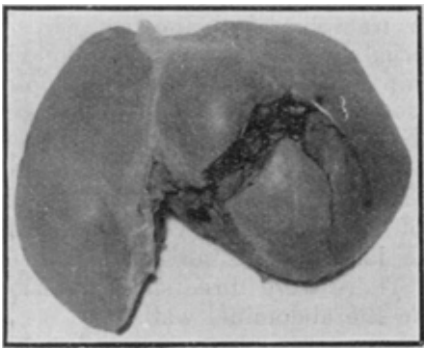


Fig. 101.—Rupture of Liver.

1. *Med. Chir. Rev.*, 1836, *p.* 296.
2. *Brit. Med. Jour., March* 12, 1921, *Epitome, p.* 43.

Gall-bladder.—Wounds and ruptures of the gall-bladder may result from penetrating wounds or from a blow, kick, or compression with the knee but, on account of the small size of the gall-bladder and the deep situation of the hepatic veins, these injuries are rare.

In January, 1919, a case occurred, in which the upper surface of the gall-bladder was lacerated by an *ekka* passing over the body.

The gall-bladder may rupture spontaneously, when distended with gall-stones. Braithwaite[1] records such a case in which a circular perforation about a third of an inch in diameter occurred on the posterior surface of the fundus of the gall-bladder. Death occurs from peritonitis owing to the effusion of blood and bile into the peritoneal cavity.

Spleen.—On account of its situation, rupture of a normal spleen is very rare unless caused by considerable compressing force, such as the passing of a carriage or motor car over the body, or by a crush in a railway accident, or by a fall from a very great height; in such cases it is usually associated with injuries to other solid organs and to the ribs overlying the spleen. A normal spleen may, sometimes, be ruptured by the broken ends of a rib which may be fractured by a severe kick or by a blow with a blunt weapon.

A young Mahomedan male died after receiving a kick from a horse. On post-mortem examination on the 21st February, 1926, fourteen hours after death, there was no external injury, but the left ninth rib was fractured, the fractured ends projecting inwards. The spleen, which was normal, was lacerated and divided almost into two parts across the middle of its outer surface, with triradiate tears on its inner surface. The left kidney was also lacerated in two places on its outer surface.

An enlarged spleen becomes softened and brittle. Hence it is liable to rupture from a fall or from violence of a very slight degree. In such cases the abdominal wall may not show any external mark of injury. During a period of eight years, I met with thirty-six cases of rupture of the spleen as a result of falls and blows. Of these cases six showed marks of bruising on the abdominal wall over the splenic region and in one there was a fracture of the left ninth and tenth ribs. Rupture usually takes place at its concave or inner surface, and causes death rapidly from hæmorrhage owing to its great vascularity. There may be frequently more than one rupture from one single blow, and its substance may rupture leaving the thickened capsule intact. In such a case death may be delayed for some days, as the capsule limits the rupture or prevents excessive bleeding, and the small quantity of blood, which has already effused under the capsule forms into a clot, and presses on the rupture and prevents further bleeding. But with sudden muscular exertion or excitement, the clot is disturbed, further bleeding occurs and death takes place immediately. Thus, an old *punkah* cooly in the Agra Fort, who had his spleen ruptured by a kick on the left side of the abdomen, appeared to be all right for three days, and died on the fourth day, when he went to his village in a jolting *ekka*. Crawford[2] records a case in which a Mahomedan boy, aged fifteen years, survived for seventeen days after the spleen and left kidney were ruptured, and three bones of the skull were fractured. Chevers[3]

1. *Brit. Med. Jour., Dec.* 12, 1908, *p.* 1746.
2. *Ind. Med. Gaz., June,* 1902, *p.* 219.
3. *Med. Juris., Ed. III, p.* 461.

mentions the case of a soldier, who died on the eighteenth day after receiving the blow which ruptured the enlarged spleen.

Rupture of an enlarged spleen from very slight violence is a very common occurrence in malarious districts, and every medical jurist is familiar with such cases. Sometimes, the enlargement is so great that its length is more than fourteen inches and its breadth more than eight inches, while the weight is often more than four pounds. Of all the enlarged spleens that were seen ruptured in the Agra District between 1909 and 1910 (the years of a high malarial incidence) the smallest spleen measured 6" × 5" × 2" and weighed nine ounces; while the largest measured 13" × 7" × 2½" and weighed four pounds and eight ounces.

An enlarged spleen may, sometimes, rupture spontaneously from the contraction of the abdominal muscles during the act of sneezing, coughing, vomiting, or straining, and some cases of this nature have been recorded (see cases below). Rare cases have also been reported in which it is claimed that a perfectly healthy normal spleen has ruptured spontaneously. It is difficult to believe that a normal spleen can rupture suddenly without apparent cause. It is, however, possible that occasionally, in certain individuals and at certain phases, a healthy normal spleen may rupture from minimal trauma. If the capsule is intact in such a case, the symptoms may be delayed for hours or days, and when eventually the patient collapses, he has forgotten the original and causative injury so that the spleen appears to have ruptured spontaneously.

Fig. 102.—Ruptured spleen.

Patey[1] reports the case of an apparently spontaneous rupture of a healthy normal spleen in a healthy muscular man, aged 50, who suddenly and without apparent cause experienced one evening acute pain in the epigastrium, which within a few minutes became so severe that he had to stop work. After two days he was removed to a hospital where an abdominal operation was performed, and a rupture was found under the capsule on the outer convex surface of the perfectly normal spleen communicating with a similar rupture on its concave side round the posterior border. At the time he denied having received any injury, but shortly after his discharge from the hospital he mentioned that at 10-30 a.m. on the day of the onset of his illness he was leaning over a ledge in order to open his shop window, when he slipped and fell on to his left side against the edge of the ledge. He had only slight discomfort and, thinking nothing of the injury, dismissed it from his mind until the repeated questionings recalled the incident. Undoubtedly this forgotten trauma was the primary cause of the ruptured spleen.

Another case[2] is also recorded in which a married woman, aged 34, had received a blow on the left side in the small of the back just below the ribs about a fortnight ago and the blow brought her to the knees and she spent a sleepless night with the pain which also persisted through the next day. After that, however, she felt quite all right, and indeed went on foot to the skating rink with a friend and had no further trouble until June 23, 1930, when on waking up in the morning she did not feel very well and was seized with very severe pain in the gastric region and died

1. *Brit. Med. Jour., May* 18, 1929, *p.* 898.
2. *Dawson-Walker, Lancet, March* 7, 1931, *p.* 523.

after a few hours. The post-mortem examination revealed a transverse tear, 2" long, on the convex surface of the normal spleen, which weighed 4½ ounces and measured 4¼"×3¼".

Wounds of the spleen are rare but may be caused by a stabbing or cutting instrument. In his reported cases Crawford[1] found one case of wound to every fifty cases of rupture.

In March, 1924, a Hindu male, 30 years old, resident of Police Station Mohanlalganj, District Lucknow, died from the effects of a stabbing wound in the left side of the chest caused with a spear. At the post-mortem examination I found an incised wound, 1¾"×¾", obliquely along the left post-axillary line in the interspace between the eighth and ninth ribs piercing the diaphragm and penetrating the external surface of the spleen to an extent of ¾"×1/6" three inches above its inferior border.

The body of a Hindu male, aged 40, said to have died of wounds was examined by me on the 29th May, 1929, thirty-six hours after death. In addition to several wounds on the body there was an incised wound, 1¼"×¼", obliquely along the back over the left eleventh rib 4" to the left of the spine and penetrating the abdominal cavity by cutting through the rib. There was an incised wound, 1"×¼"×½", along the outer surface of the spleen in its lower part and near its interior border.

A slight wound may cause death from shock by profuse hæmorrhage from the spleen. Thus, a case occurred to Dr. J. N. B. Wise, in which death occurred from puncture of the enlarged spleen made with a needle, apparently by way of treatment.[2]

Cases of Spontaneous Rupture of the Spleen.—1. A fine looking old Mahomedan, aged about 50 years, and apparently in good health, suddenly became faint and fell down in the Deputy Commissioner's Court at Umballa, where he was watching the proceedings of a case in which he was the complainant. He was immediately carried out of Court and died in the compound. On dissection the spleen, which was soft, friable and enlarged, weighing 3 lbs. 13 ozs. was found ruptured in its inner surface. The rupture had occurred spontaneously from excitement due to the law suit, for there was no furniture or projecting angles against which the body could have impinged during a fall.—*James, Ind. Med. Gaz., June*, 1902, *p.* 222.

2. On the evening of the 9th February, 1904, a Nepalese employed as a naik driver in a Government Yak Corps lay down to sleep in a tent after he had finished his work. At about 7-20 p.m. he called to his brother, who was a driver in the corps, and said that he had pain in his side. The Havaldar and the man's brother went to the tent when they found him in great distress and complaining of intense pain "near his heart". The Havaldar at once called a British officer who came to the tent and looked at the man who still complained of the pain and weakness but made no accusation of anybody having struck him, nor could he in any way account for the pain. He grew rapidly weaker and died a few minutes before eight—about half an hour after the pain commenced. On post-mortem examination the body was found to be well-developed and powerful and the age of the deceased appeared to be between twenty-five and thirty years. There was no external mark of injury. On opening the peritoneal cavity a large quantity of blood gushed out; the peritoneum was found perfectly healthy and no adhesions were found in any part of the abdomen. A large rupture was found in the spleen extending through the anterior angle of the hilum. The organ was enlarged to double its normal size and was very soft. The other abdominal organs were quite healthy.—*Davys, Ind. Med. Gaz., June*, 1904, *p.* 218.

3. A Hindu male, aged about 20, was admitted into the Calcutta Medical College Hospital on the 30th April, 1903, with a history of abdominal pain of ten hours' duration. There was constipation but no vomiting. The abdomen was generally tender, the tenderness being most marked in the left iliac region; it was slightly distended. On the 1st May, the abdomen was more distended, the distension being more marked in the epigastric region; the tenderness was greater and free fluid was

1. *Ind. Med. Gaz., June*, 1902, *p.* 220.
2. *Ind. Med. Gaz., June* 20, 1902, *p.* 220.

present in the abdominal cavity. He complained of much thirst. Colotomy was performed but no obstruction was present. He died at 9 a.m. on the 3rd May. At the post-mortem examination the spleen was found slightly, if at all, enlarged and its substance was largely occupied by blood clot; on the outer surface towards the lower end of the organ, was a small rupture of the substance which had caused hæmorrhage beneath the capsule with the formation of a cyst-like cavity containing serous fluid; this had burst into the peritoneum setting up peritonitis. A portion of the omentum was adherent to the ruptured cyst-like cavity. There was no free blood in the abdominal cavity. The other organs were normal, there being nothing to suggest that an impact had occurred in the spleen.—*Owen Thurston, Ind. Med. Gaz., Oct.*, 1904, *p.* 379.

4. A Hindu male, aged about 45 years, and residing in Hardoi, who was addicted to drinking alcohol to excess and smoking *charas*, fell down unconscious in a lane while walking on the afternoon of the 13th August, 1906, and died immediately. At the post-mortem examination I did not notice any external mark of injury over the abdomen on the part corresponding to the region of the spleen, but the abdominal cavity was full of blood, and the spleen was ruptured. The spleen was so soft, friable and pultaceous that not an inch of the solid substance could be taken out entire. It was lying in a thickened capsule which showed as if it was a bag containing the pultaceous mass. The rupture was spontaneous probably due to contraction of extraordinary muscles brought to head in a fall, for there was no history of the deceased having received a blow or having been hit against a hard substance in the splenic region during the fall.—*Ind. Med. Gaz., Oct.*, 1906, *p.* 423.

5. The following is a case of spontaneous rupture of the spleen during an attack of malaria :—

J. K., a British soldier, aged 33, was admitted into hospital on the 6th January, 1924, at 2-30 p.m. with a temperature of 103.6° F., and was vomiting a good deal of blood-stained fluid. The next morning the temperature dropped to 99.8° F. The pulse was 82 per minute and he felt better. He spent the morning in bed reading. At 1 p.m. he became restless, and complained of pain over the stomach, and made several attempts at vomiting without any results. Morphine, $\frac{1}{4}$ grain, was injected hypodermically. At 2-45 p.m. he looked very ill and pale, complained of severe gastric pain and a sense of dizziness, and was found leaning over the cot attempting to vomit. At 3-10 p.m. he was found reclining in bed looking desperately ill. The face was very pale, the hands and chest were cold and clammy, and he was perspiring. The respirations were frequent and long drawn, and the pulse was weak and 80 per minute. There was no abdominal tenderness, but he rapidly collapsed and died at 3-40 p.m. On examination no evidence of external injuries was found. On opening the abdomen about a pint of pure blood escaped, and the spleen was found ruptured on the upper and posterior part. The spleen was enlarged, deeply congested and of a violet colour, and weighed 453.5 grammes.—*P. Savage, Ind. Med. Gaz., Nov.*, 1926, *p.* 552.

Kidneys.—Owing to their deep situation in the abdomen rupture of the kidneys is rare by direct violence from blows, even if they are diseased, unless the force used is very considerable and inflicted on the loin.

During the Hindu-Mahomedan riot at Agra in 1913, a young Hindu of about 20 years of age was hit by one of the rioters with a *lathi* in the left lumbar region and died immediately. On post-mortem examination the left kidney, which was quite healthy, was found ruptured.

The kidneys may be accidentally ruptured when an individual is run over by a heavy vehicle, such as a motor car or a country cart or when he is crushed between the buffers of a railway carriage. They are also apt to be injured when the body is violently flexed forwards at the lumbar region. Rupture may prove rapidly fatal from collapse or hæmorrhage, or more slowly from peritonitis or suppuration caused by extravasation of urine. A slight rupture may result in recovery.

E. W. Riches[1] reports the case of a man, aged 34, who lived for eleven days after rupture of the right kidney sustained by him when a lorry knocked one of the handles of his wheelbarrow violently into his sides. Fowler Ward[1] cites even a case of recovery from a ruptured kidney without an operation.

Bladder.—Rupture of the bladder may be produced generally at the posterior and upper surface by blows, crushes or kicks on the hypogastrium, especially when it is distended with urine. Sometimes, very slight violence may rupture the bladder without any external sign of injury. Rupture may also occur from a fall, from a fracture of the pubic bone, or from a sharp weapon penetrating through the vagina or rectum.

In June, 1923, a woman, 25 years old, was run over by a tonga, and died in a few hours. On examination there was no external mark of injury, but on opening the abdomen an oblique transverse ecchymosis in an area of three inches by two inches was seen in the substance of the muscle in the left iliac region. The pelvic cavity contained blood. The bladder was found lacerated to an extent of two inches in the upper surface and was covered on the inside with clotted blood.

On the 11th November, 1923, a woman was run over by a bullock cart and died immediately. There was no external mark of injury to the abdominal wall, but there was an extravasation of blood in the muscles of the abdomen across its lower part above the pubes with rupture of the bladder in its upper part and fracture of the pubic and iliac bones.

Spontaneous rupture of the normal bladder is rare, almost impossible, though it may occur at its base from over-distension when it is diseased or ulcerated, or when there is an obstruction in the urethra from stricture, enlarged prostate or some such condition. Similarly, it is liable to rupture in females during parturition, owing to the pressure of the child's head, if the bladder is over-distended. Persons who are habituated to excessive indulgence in alcohol or opium are apt to go about with a distended bladder owing to the depressed effect on the nerve centres. In such a condition slight pressure or an accidental fall on the abdomen may be sufficient to rupture the bladder.

The symptoms of rupture of the bladder are pain, tenderness in the abdomen, rigidity of the abdominal muscles, inability to pass urine and the presence of blood in the urine. In some cases the symptoms may be delayed for eight hours or more. The patient may be able to walk for some time after receiving the injury.

Death may occur suddenly from shock, but usually occurs in three to seven days from peritonitis due to the extravasation of urine into the peritoneal cavity, or from suppuration and sloughing due to urine being extravasated into the cellular tissue if the bladder is ruptured at its extraperitoneal portion. Taylor[2] cites a case in which death did not take place until the fifteenth day.

Uterus.—The non-gravid uterus is not ordinarily injured unless involved in the injuries of the pelvic organs, but the gravid uterus is likely to be ruptured by a blow, kick, or trampling on the abdominal wall, or by the passage of a sharp instrument per vagina to procure abortion. Death may result from hæmorrhage, peritonitis, or septicæmia. The

1. *Proceedings of the Royal Society of Med., Feb.*, 1931, *p.* 470.
2. *Princ. and Pract. of Med. Juris., Vol. I, Ed. IX, p.* 445.

pregnant organ may also be ruptured during obstetrical operations or spontaneously during parturition.

Partial or complete separation of the placenta caused by a blow on the abdomen during pregnancy may cause fatal hæmorrhage.

Case.—Chevers quotes a case of rupture of the gravid uterus caused by a kick. At the post-mortem examination there was no external mark of injury, but there was an extravasation of blood among the muscles above the mons veneris. An enormous quantity of fluid blood was effused into the peritoneal cavity. The uterus was ruptured at its fundus, and through the laceration the membranes of a fœtus between the second and third month protruded.—*Med. Juris., Ed. III, p.* 477.

Urethra.—The male urethra may be ruptured by a kick in the perinæum, by a fall astride some projecting substance, such as a fence or beam, or by a fractured piece of the pubic bone. The seat of rupture is usually in front of, or behind, the triangular ligament, just where the urethra passes under the pubic arch. Death may occur from extravasation of urine, but the rupture may heal without any serious effects if the tear is a slight one, and if immediate surgical treatment is undertaken.

The female urethra may be ruptured by an act of rape. I saw a girl of eight years, whose anterior urethral wall was lacerated to an extent of $\frac{1}{4}$" × $\frac{1}{8}$" by an act of rape committed by a grown-up man. Schepetinsky[1] also reports the case of a woman, 23 years old, who had been raped by an intoxicated man. Her urethra was abnormally wide, and there was a bleeding rupture, 3 or 4 cm. long, on the posterior wall, and she was suffering from gonorrhœa.

Penis.—Wounds of the penis and its total extirpation, if not fatal by shock and hæmorrhage, are not dangerous. Cutting off the penis with a knife or razor is one of the usual modes of punishment for adultery in India. In order to avenge himself on one Raghubar Dayal who had committed adultery with his wife, one Murli of Kheri District amputated his penis at its root, placed it in his mouth and then killed him.[2] Sometimes, the victim is first killed, and then his genital organ is cut off.

Mutilation of the penis and even castration are occasionally self-inflicted by lunatics or by individuals who want to be eunuchs or wish to dedicate their lives to a goddess, *viz., Bahucharaji,* in the Gujarat Province. On September 17, 1932, I saw one Hijra, called Pancham *alias* Ilaichi, aged 28 years, who had cut off his penis and scrotum with the testicles by one sweep of a knife, causing an incised wound, 4" × 4", below the pubes and directed from above downwards. When he was brought to the hospital, the wound was covered with curd and a piece of a dirty rag. He was discharged cured after ten days.

Superficial incised wounds of the penis are, sometimes, produced to fabricate a false charge of assault.

Rupture of the penis, as also its dislocation into the scrotum, may be produced by a squeeze or crush.

---

1. *Zentralblatt fur Gynakologie, Leipzig, Oct.* 4, 1930, *p.* 2530; *Jour. Amer. Med. Assoc., Jan.* 10, 1931, *p.* 155.
2. *Chief Court of Oudh, Crim. Appeal No.* 164 *of* 1928.

In a quarrel that took place at Delhi a man seized another man by the penis and dragged him along until the prepuce gave way and was torn off.—*Nizamat Adawlat Rep., N. W. P., Sep.,* 1854, *p.* 415.

Biondi reports the case of a man, aged 64 years, whose penis was pulled off by an energetic young woman with whom he had attempted sexual intercourse. The case was brought into Court and each party gave a different version as to how the thing actually occurred. The woman's story was that the man tried to seduce her and that in self-defence she pulled forcibly at the penis, which came off in the struggle. From certain experiments made at the cadaver with a view to elucidate the question it was found that the nature of the injuries sustained in the case was exactly similar to that which was produced when the penis was forcibly avulsed and the amount of force required to tear off the penis in a flaccid state was far in excess of that which any ordinary person would be likely to possess. But when the penis was erect, the resistance was very much reduced, so that a comparatively moderate amount of force, quite within the possibility of a woman of average strength, was quite sufficient to completely avulse the penis.—*La Clin. Mod., An.* 12, *N.* 18; *Brit. Med. Jour., Aug.* 18, 1900, *Epitome, p.* 26.

Testicles.—Contusion of the testicles results from blows, kicks and squeezes, and is accompanied by severe pain of a sickening character, which may produce a fatal shock. The squeezing of a testicle is a common practice of assault in India, and sometimes the squeezing is so very forcible that the testicle is protruded out of the scrotum. It may also be accidentally protruded through a lacerated wound of the scrotum caused in jumping over a barbed wire.

Vulva.—Injuries to the vulva may be caused homicidally by a blow or kick in front, or from behind when a female is bending forward. They may also be caused accidentally when a female forcibly sits on a broken chamber pot while urinating or falls on a projecting sharp substance.

Owing to the underlying pubic bone wounds of the vulva caused by a blunt weapon may look like incised wounds, but minute and careful examination of the wounds will reveal the difference. These wounds may prove fatal from excessive hæmorrhage.

Case.—A woman, aged 36, while in a stooping posture, received a kick from her husband in the lower part of the abdomen. She died within an hour from loss of three to four pounds of blood. On inspection there was no injury to the vagina or uterus. A wound, about an inch long and three-quarters of an inch deep, was found at the edge of the vulva, extending from the pubes along the ramus of that bone. The left crus clitoridis was crushed throughout its length, so as to exhibit its cavernous structure. From this the fatal bleeding had proceeded.—*Taylor, Princ. and Pract. of Med. Juris., Vol. I, Ed. IX, p.* 449.

Vagina.—The vagina may be lacerated by the introduction of an abortion stick for procuring criminal abortion or by the forcible thrusting of a foreign body, such as a blunt weapon as a form of torture or punishment. Lacerations caused by thrusting a foreign body into the vagina are, sometimes, multiple involving the pelvic organs and cause death. The following two cases are illustrative :—

1. A *dhak* stick had been forcibly thrust by Saktu, accused, through the vagina of a girl, 14 years old, so that its upper part had been bored over for four inches. The stick had perforated the vagina, torn the bladder and displaced the uterus which was almost lying loose. It had passed into the abdominal cavity as far as the stomach. The abdominal portion of the stick was fifteen inches long. The peritoneal cavity contained about two ounces of clotted blood, and the omentum was torn and

congested. The upper part of the rectum was perforated, and the uterine ligaments were torn.[1]

2. A woman was killed by a *lathi* being thrust into her vagina, which, lacerating the orifice and tearing the posterior fornix, entered the peritoneum making an opening, 2"×2". The surrounding structures were blue and congested.[2]

The vaginal walls may be lacerated during parturition, and the laceration may extend into the bladder or rectum.

The vagina may be injured by violent sexual intercourse especially by a strong healthy adult with a small girl, and fatal results may follow from profuse hæmorrhage or from pelvic cellulitis.

I saw a case in Agra, where a girl of thirteen years died from septic cellulitis caused by a lacerated wound in the posterior wall of the vagina, the result of sexual intercourse by her husband, who was a strong young man. J. Alfred Gaynor[3] records the case of a married woman, 28 years old, who immediately after the first attempt at coitus, had a severe vaginal hæmorrhage losing about three pints. On examination a dense central adhesion was found between the anterior and posterior vaginal walls, leaving two small lateral apertures. This was situated about midway between the hymen and the vault of the vagina. A deep tear in the vaginal wall, about 1½ inches long, extended from the lateral aperture on the right side towards the hymen.

The usual practice of punishment for adultery in India is to brand the vulva with a heated solid substance or to introduce a bruised marking nut (*Bhilawa*), or chillies into the vaginal cavity.

MUSCLES

Contusions and sprains of the muscles may occur from a blow or from a fall. They are generally simple in nature, but an abscess may form in the contused part of the muscle, or paralysis and subsequent atrophy of the muscles may occur if the nerve supplying these muscles is damaged. Similarly, a person may become lame from a sprain of the gastrocnemius and soleus owing to their contraction.

Lacerations and crushing of the muscles due to a heavy cart or a railway or machinery accident may necessitate the amputation of a limb, or may cause death indirectly from gangrene or tetanus.

Sometimes, it so happens that owing to its elasticity, the skin, especially of the chest and abdomen, remains intact, but the underlying muscles are torn by kicks, blunt weapons or by street accidents causing protrusion of a portion of a viscus behind the skin.

In a carriage accident a boy, aged 11 years, sustained injuries in his chest by the front end of a pole of an *ekka* hitting him on the right side. On examination there was no mark of external injury on the chest, but a portion of the lung was found protruding through the torn muscles in the fourth intercostal space of the right side of the chest.

A woman, aged 60 years, was struck in the abdomen by a bullock with his head, and died 3 days later. At the autopsy the abdominal wall was found intact without any external mark of injury, but a piece of the omentum was found pro-

1. *K. E.* v. *Saktu, Oudh Jud. Com. Court, Crimin. Appeal Reg.* 138 *of* 1922.
2. *K. E.* v. *Patey Singh and Sarup Singh, Allah. High Court, Crimin. Appeal No.* 97 *of* 1931.
3. *Brit. Med. Jour., Dec.* 10, 1927, *p.* 1080.

truding behind the skin through a tear, 1½"×1", of the abdominal muscles on the right side in the upper part. There was also a tear in the small intestine towards its lower part on the right side.

BONES

Contusion of a bone and of its periosteum due to a blow or a fall is a simple injury and, under ordinary circumstances, subsides in a few days, though acute infective periostitis or necrosis may occur in the case of debilitated, syphilitic or rheumatic people.

Fractures of bones may occur from blows, falls or from muscular contraction. A case is recorded in which a healthy man fractured his humerus by muscular exertion in throwing a cricket ball.[1]

Fractures are not ordinarily dangerous unless they are compound, when death may occur from loss of blood, if a big vessel is wounded by the split end of a fractured bone, or from embolism, septicæmia, gangrene or tetanus.

In children and young persons the bones are tough and elastic, hence a green-stick or partial fracture occurs more frequently; while in old people the bones, being brittle owing to the increase of their inorganic constituents, are easily fractured even with very slight violence. The bones are more fragile in certain diseases, such as syphilis, arthritis, osteomalacia, rickets, sarcoma, cancer, scurvy, and those nervous diseases which produce trophic changes.

There is a peculiar brittle condition of the bones called *fragilitas ossium,* in which fracture may occur from a trivial trauma or a slight exertion, such as a misstep in walking or moving around in bed. This condition is hereditary and found in persons apparently in good health. It is also found in people suffering from locomotor ataxy, syringomyelia, and general paralysis of the insane, and in workers in phosphorus.

In criminal cases the defence often admits the fracture of a bone, but raises the plea that it was due to an accident and not to direct violence. A fracture caused by direct violence can be judged from its position and the presence of a bruise or wound of the skin or subjacent tissues accompanying the fracture. It should, however, be noted that in some cases no bruise or wound is associated with a fracture. But such a fracture is generally transverse and sometimes comminuted. When due to an accident, such as a fall, the fracture occurs at the weakest part of the bone, is usually spiral or oblique and is, generally, not accompanied by a bruise or wound.

Distinction between Ante-mortem and Post-mortem Fractures.— Fractures caused during life show the signs of effusion of blood, laceration of muscles, pouring out of lymph, and formation of callus, but these signs are absent in fractures produced after death. However, it is difficult to distinguish if a fracture is caused immediately after death when the body is still warm, though the effusion of blood about the torn muscles and

1. *Lond. Med. Gaz., Vol.* **16**, *p.* **659.**

fractured ends will be very little. Besides, it should be remembered that with ordinary force it is not possible to fracture a bone after death, as it loses its tonicity and elasticity.

DISLOCATIONS

Dislocations are caused by falls, blows, or muscular action. They are not common in old people and in those persons whose bones have become brittle, as well as in children, in whom the separation of epiphysis is more common. They are not dangerous unless they are between the vertebræ, or are compound when death may result from secondary complications.

Dislocations may occur spontaneously when the joints are diseased. It is easy to diagnose a dislocation before it is reduced. Owing to swelling, ecchymosis and limitation in the movement of a joint it may be easy to find it out even after it is reduced. But it is quite difficult to do so, after these effects have passed off, unless there is paralysis or muscular atrophy due to the involvement of a nerve as in the dislocation of a shoulder joint.

After death they may be recognised by the effusion and coagulation of blood, and by the laceration of the soft tissues in the vicinity of the joint. Old dislocations may be ascertained by scar tissue in and about the capsule.

CHAPTER XIII

IMPOTENCE AND STERILITY

Definition.—Impotence is defined as physical incapacity for accomplishing the sexual act, and is applied to the male more than to the female, as the latter is a passive agent in the act of copulation. While sterility means inability for procreation of children, and is referable more to the female than to the male.

It should be remembered that an impotent individual need not necessarily be sterile, nor a sterile individual impotent, though both conditions may, sometimes, be combined in the same individual.

QUESTIONS RELATING TO IMPOTENCE AND STERILITY

Impotence and sterility in either man or woman may form the basis of medico-legal investigation both in civil and criminal cases. The civil Court may call on the medical jurist to determine this point in suits of adoption, contested paternity, nullity of marriage and divorce. The criminal Court may have to decide this question with the aid of the medical jurist in accusations of alleged adultery, rape and unnatural offences, in which the accused pleads impotence as an excuse in defence, and in cases where an injured individual asserts that he has become impotent from wounds or injuries received by him, especially if they happen to have been inflicted on the head, neck or loin.

When asked by the Court to examine a particular male as to whether he is capable of sexual intercourse, the medical jurist must give an opinion in the negative form, and must answer that from the examination he finds nothing to suggest that the male examined is not capable of sexual intercourse if he happens to be a healthy, normal individual. Casper[1] states that "the possession of virility and procreative power neither requires to be, nor can be, proved to exist by any physician, but is rather, like every other normal function, to be supposed to exist within the usual limits of age." It is, therefore, necessary for the medical jurist to ascertain by an examination of the individual in a case of disputed potency, if there is any abnormal condition which is likely to interfere with the normal function of copulation.

Under the law of England, marriage is a contract which may be declared "null and void" if it can be proved that either party was, at the time of contracting marriage, impotent, *i.e.*, incapable of fulfilling the rights of consummation of marriage. But this incapacity must be permanent and incurable by an operation, even if the individual is willing to submit to it. The acquirement of impotence subsequent to marriage, or sterility alone, is not a sufficient ground to grant a decree of divorce.

1. *Forens. Med., Engl. Trans., Ed. III, Vol. III, p.* 241.

Again, it cannot be set aside if marriage was contracted with full knowledge with an impotent person or with one who, from advanced age, might be inferred to be incapable of sexual intercourse.

CAUSES IN THE MALE

The causes of impotence and sterility in the male are—

1. Age.
2. Malformations.
3. Local Diseases.
4. General Diseases.
5. Psychical Influences.

1. **Age.**—Impotence is generally observed at the extremes of age. Boys are considered to be sexually potent at the age of puberty which usually occurs at the fifteenth or sixteenth year. Sexual intercourse is, however, possible at about the thirteenth or fourteenth year, as the power of coitus commences earlier and ceases later than the power of procreation. The changes which occur in a boy at puberty are the development of the genital organs, the ability to secrete semen, the growth of hair on the pubes, axillæ and chin, and the increase in size of the larynx leading to the deepening of the pitch of the voice. When examining an individual for sexual capacity the medical jurist should depend more on physical development than on age alone.

Rarely, sexual development may occur at a very early age. Ram Chandra Row[1] reports the case of precocious development of a boy, aged about 22 months, whose penis and testicles were highly developed simulating those of an adult, and the pubic region was covered with long and dark hair. He possessed a very shrill voice, and his brother, 4 years old, was terribly afraid of him on account of his rough behaviour. A case[2] is recorded in which a boy, aged four years and a half, attempted sexual intercourse with his sister, aged two years. Another case[3] is recorded in which a boy, 13 years old, impregnated a young woman. Gemmell[4] reports a case in which a boy, aged 14 years, impregnated a girl, 12 years and 11 months old, after a single coitus, and the girl at the age of 13 years and 8 months gave birth to a baby weighing 6½ pounds.

Sexual development may be delayed till late in life. Curling[5] quotes the case of a man, whose sexual organs at the age of twenty-six were like those of a child of eight. At twenty-eight his organs assumed their nomal development. He married and became the father of a family.

As age advances the power of sexual intercourse and procreation diminishes, but no limit can be assigned at which this power ceases, as men of eighty years and over have been known to have begotten children. Casper relates a case in which spermatozoa were found in a man, ninety-

1. *Ind. Med. Gaz., Feb.,* 1926, *p.* 70.
2. *Lond. Med. Gaz., April,* 1872.
3. *Brit. Med. Jour., April* 23, 1887.
4. *Ibid., May* 7, 1927, *p.* 862.
5. *On Sterility in Man; Collis Barry, Leg. Med., Vol. II, Ed. II, p.* 45.

six years old,[1] and that he himself had observed them in a man of sixty-nine.[2] In October, 1924, I referred a case to Dr. Mukarji, where he found spermatozoa in a man of about ninety years of age. Seymour and others report a case in which a man, aged 94, had a child by his wife, aged 27. His seminal fluid contained motile spermatozoa of normal conformation and of average size.[3]

2. **Malformations.**—The absence or non-development of the penis renders a man impotent, but the man is not sterile if semen can be deposited into the vagina with the partially developed penis. The penis adherent to the scrotum cannot be a plea for divorce if it can be remedied by a surgical operation. The presence of double penis, although a rare occurrence, may cause difficulty in sexual intercourse, but coition is, nevertheless, possible in some cases.

A case[4] is recorded in which a man, aged 26, had two penes lying side by side. The right penis was attached at the normal site in the midline, had a foreskin and was normal in all respects, except for a slight hypospadias, while the second or accessory organ was attached 5 c.m. to the left of the other, was smaller and had no urethra or foreskin. There was a small meatus from which a slight mucous discharge came out on sexual excitement.

Hypospadias, a congenital deformity of the penis, which is characterised by the urethral orifice being situated on the under surface, does not, as a rule, produce incapacity for sexual intercourse, unless it is associated with marked deformity of the penis which may interfere with its intromission into the vagina. Sterility in such a case depends upon the position of the urethral orifice, and it is assumed if the urethral orifice is so placed as to prevent the deposition of semen into the vulva. Spermatozoa, if deposited into the vulva, can certainly travel upwards into the vaginal canal owing to their mobile power. A case[5] is recorded in which a married man with the deformity of hypospadias had two daughters by his wife.

Epispadias, a deformity in which the urethra opens on the dorsum of the penis, is extremely rare, and is often associated with the rudimentary and stunted penis and extroversion of the bladder rendering sexual intercourse impossible.

The congenital absence of the testicles produces sterility and impotence, but it is possible for a man to impregnate a woman after double castration if semen had already been present in the vesiculæ seminalis before the operation; he becomes permanently sterile after this stock of semen has been exhausted.

Sir Astley Cooper[6] knew a man in whom both the testicles had been extirpated for twenty-nine years. During the first year, this man, when

1. *Forens. Med., Eng. Trans., Vol. III, p.* 292.
2. *Ibid., p.* 258.
3. *Jour. Amer. Med. Assoc., Nov.* 2, 1935, *p.* 1423.
4. *Seth and Peacock Urolog. and Cut. Rev., Sep.,* 1932, *p.* 590.
5. *West Lond. Med. Jour., Sep.,* 1911; *Glaister, Med. Juris. and Toxic., Ed. VI, p.* 330.
6. *Observations on the Structure and Diseases of the Testicle, London,* 1830; *Casper, Forensic Med., Eng. Trans., Vol. III, p.* 256.

satisfying his sexual desire, had regular seminal emissions. Subsequently he had erections, though but rarely, and satisfied his sexual desire without any ejaculations; after two years the erections were more seldom and less perfect.

Monorchids, *i.e.*, those who have one testicle only are physiologically quite potent; whereas cryptorchids, *i.e.*, those who have undescended testicles, are usually, but not invariably, impotent and sterile. Some may be quite potent and fertile. A case[1] is recorded in which a cryptorchid who was married at the age of eighteen had five children born to him, till he was thirty-four years old.

3. **Local Diseases.**—A large hydrocele or scrotal hernia, elephantiasis, phimosis, paraphimosis and adherent prepuce may cause temporary impotence by mechanical obstruction to coitus, as these conditions can be remedied by proper surgical treatment. Marked diseases of the penis or of the testicles, such as syphilis, cancer and tuberculosis, may lead to impotence or sterility or both. Inflammatory affections of the testicles, epididymis, prostatic gland and seminal vesicles of gonorrhœal origin are the frequent causes of impotence and sterility. The ejaculatory ducts may be obliterated by chronic gonorrhœa, so that the seminal discharge may be prevented from flowing into the urethra. Atrophy of the testicles occurring after mumps may produce impotence or sterility. An operation of lithotomy, sometimes, causes sterility from injury to the ejaculatory ducts.

General Diseases.—Endocrine disturbances may produce sexual infantilism, rendering an individual impotent. Certain general diseases, such as diabetes, pulmonary tuberculosis, chronic nephritis, etc., which occasion extreme debility, may produce impotence, temporary or permanent, through the weakness to which they give rise, though the genital organs are apparently quite normal. It is difficult to say which of the nervous and mental diseases weaken the sexual power. Inflammation of the brain and its meninges generally produces more or less paralysis of the genital organs. It is said that hemiplegia, paraplegia and locomotor ataxy produce impotence, but this is not always the case. Guy[2] mentions two cases where men, within two to three weeks of a well-marked attack of hemiplegia, had fruitful sexual intercourse with their wives. Curling[3] quotes the case of a man who, during paraplegic condition lasting for eight years, held intercourse with his wife and begot two children. In the case of *Bagot* v. *Bagot* tried in the Irish Probate Court in 1878 Dr. Radcliffe gave evidence of his having seen cases of locomotor ataxy where sexual capacity and fruitfulness were retained.[4]

Some forms of mental disease, especially general paralysis of the insane, increase the sexual power in the beginning, though at a later advanced stage totally abolish the sexual instinct. Blows on the head or spine may produce temporary or permanent impotence by affecting the brain and spinal cord. A condition of temporary azoospermia (complete

1. *Taylor, Princ. and Pract. of Med. Juris., Vol. II, Ed. IX, p.* 22.
2. *Forensic Med., Ed. VI, pp.* 49, 50.
3. *Diseases of the Testes, Ed. IV, p.* 443.
4. *Guy, Forensic Med., Ed. VI, p.* 49.

absence of spermatozoa in semen) unattended with any loss of sexual power is observed in individuals, who attend in the X-ray department without proper protection. The excessive and continued use of some drugs, such as alcohol, opium, cannabis indica, tobacco, cocaine and bromides, may render a man impotent.

5. **Psychical Influences.**—A temporary absence of desire for sexual intercourse may result from fear, timidity, aversion, hypochondriasis, excessive passion, and sexual over-indulgence. Sometimes, an individual may be impotent with one particular woman, but not with another. It should be noted that in a divorce suit the question to be decided is the incapacity of the husband to sexual intercourse with his married partner; his capacity for intercourse with other women is of no consequence in deciding the case.

Lord Birkenhead, the Lord Chancellor, granted a divorce to a woman who instituted a suit for nullity of marriage aften ten years of married life on the ground that the husband was unable to consummate the marriage. It followed that although physically normal, he had always been incapable of consummating this particular union with this particular woman (impotence *quoad hanc*).[1]

In an appeal from a divorce suit of *Ibrahim* v. *Musammat Altafan* heard before Mr. Justice Kanhaya Lal at the High Court of Allahabad in 1923, it was contended that no consummation of marriage had taken place although the parties had been married for years. Medical evidence proved beyond doubt that the husband had no malformation of, or defect in, the male organ and that he was normally capable of performing the sexual act. The woman was also medically examined, and certified to be a virgin, who had had no sexual intercourse with any man. This was a case of a man who might be impotent *quoad* his wife, but the learned Judge allowed him one year more to prove his potency with his wife.

CAUSES IN THE FEMALE

The causes which prevent sexual intercourse and conception in the female are the same as those of impotence and sterility in the male; *viz.*,

1. Age.
2. Malformations.
3. Local Diseases.
4. General Diseases.
5. Psychical Influences.

1. **Age.**—Puberty in the female usually commences at the thirteenth or fourteenth year in India. It is generally believed that puberty commences at an early age in the tropics than in the temperate regions, but I do not think that there is any difference in the age of puberty and Professor Crew[2] expressed the same opinion at a meeting of the Social Hygiene Congress in London. From observations made in 479 cases amongst Indian women representing many different castes and races, Miss Curjel[3] has come to the conclusion that the average age of the onset of puberty (catamenia) in an Indian girl is 13.63 years.

1. *Jour. Amer. Med. Assoc., July* 23, 1921, *p.* 297.
2. *Lancet, Sep.* 21, 1929, *p.* 619.
3. *Ind. Jour. of Med. Research, Oct.*, 1920, *p.* 306.

The signs of puberty in a girl are the development of the external and internal genitals, the appearance of menstruation, the growth of hair on the pubes and axillæ, and the development of the breasts. There is a change in her tastes, and the girl no longer looks like a child but is more bashful and retiring. Luxurious living and early stimulation of the mental faculties tend to bring on menstruation at an earlier age, while feeble health and poor diet tend to retard it.

In exceptional cases menstruation may appear very early or late in life. A female child, from a few days after her birth, menstruated at regular intervals of twenty-three or twenty-four days until she died at the age of four. At the autopsy the sexual organs were found as much developed as those in a girl at the age of puberty and the breasts were also as much developed as in a woman of twenty years.[1] Arnold Gesell[2] reports the case of a girl who began to menstruate at the age of three years and seven months. Powell[3] describes the case of a child, aged four, who used to have a discharge of blood from the vagina every six or eight weeks. The labia were large and the breasts as large as the halves of a moderate sized orange. C. Worster-Drought[4] reports the case of a girl, aged 5 years, who began to menstruate at the age of $2\frac{1}{2}$ years and continued to menstruate regularly for 12 months, the period lasting each time for three days. Menstruation stopped for 18 months and then reappeared. Since then it has been more or less regular. The breasts were noticed to be prominent at birth, but there was a sudden increase in size at the onset of menstruation. Pubic hair appeared at the age of 4 years and six months. P. M. Sen Gupta[5] also records the case of a girl who began to menstruate at the age of $3\frac{1}{2}$ years. At first the flow came on every month, then the intermenstrual periods lengthened to about two months and the last interval was over six months. At the age of 5 years she was quite intelligent, her breasts were considerably developed and there was slight growth of pubic and axillary hair.

Cases of delayed menstruation have also occurred. I have known a family, where girls did not menstruate till they were eighteen years old. Powell[6] has known women of twenty who had not menstruated. A married woman did not menstruate till she was thirty years old, and did not bear children.[7] In one case menstruation appeared for the first time at the age of forty-seven.[8]

It is generally assumed that the power of fecundity commences with the first flow of menstruation, and lasts till the menopause, which usually occurs from the forty-fifth to the fiftieth year.[9] For obvious reasons such

1. *Whitmore, North Jour. of Med., July,* 1845, *p.* 70; *Taylor, Princ. and Pract. of Med. Juris., Ed. IX, Vol. II, p.* 14.
2. *Jour. Amer. Med. Assoc., March* 17, 1928, *p.* 840.
3. *Ind. Med. Gaz., June,* 1902, *p.* 233.
4. *Proceedings of the Royal, Soc. of Med., Aug.,* 1931, *p.* 1338.
5. *Ind. Med. Gaz., June,* 1937, *p.* 368.
6. *Ind. Med. Gaz., June,* 1902, *p.* 233.
7. *Camps, Med. Gaz., Vol.* 32, *p.* 409.
8. *Camps, Ibid; Taylor, Princ. and Pract. of Med. Juris., Ed. IX, Vol. II, p.* 15.
9. *According to Miss Curjel the average duration of menstrual life (reproductive) among Indian women is* 32.14 *years, and this does not appear to differ materially from European races.—Ind. Jour. of Med. Research, Oct.,* 1920, *p.* 566.

a view is not tenable in the case of babies and small girls who menstruate prematurely. Cases have, however, occurred where girls became pregnant at a very early age. A Mahomedan unmarried girl,[1] 6 years and 8 months old, who had never menstruated, was delivered of a full term female child by Cæsarian section in the Zenana Hospital at Delhi. She was able to nurse her child. Dodd[2] records the case of a girl who began to menstruate at the age of one year, became pregnant at eight years and ten months and was subsequently delivered of a living child which weighed seven pounds. Chevers[3] quotes Dr. Chuckerbutty who knew a girl who became a mother at the age of ten. E. D. H.[4] reports a case where the operation of Cæsarian section had to be performed on a little girl, both at the birth of her twin babies when she was ten years old, and at the birth of her living child before she was eleven years of age. Curtis[5] mentions the case of a girl who became pregnant by a boy, aged 15 years, twenty-four days before she reached the age of ten, and was delivered of a healthy child at ten years and eight months. In his *Essays on Midwifery* Robertson quotes the case of a factory girl who became pregnant when she was eleven years old. Pregnancies, on the contrary, have occurred at the advanced ages of fifty-five, fifty-seven, sixty-six and seventy-two, several years after the menopause.[6]

2. **Malformations.**—Congenital malformations, such as the total occlusion of the vagina, adhesion of the labia and the tough, imperforate hymen, are barriers to coitus, and consequently lead to sterility, but these malformations are such as can be remedied by surgical interference. The congenital absence of the vagina will ordinarily render a female completely and permanently impotent and sterile. Cases have, however, been reported where an artificial vagina had been formed by operation. Hodgson[7] records a case in which he formed an artificial vagina in a married woman, 32 years old, who had no vagina from her birth. Coitus was subsequently carried out satisfactorily.

The conical cervix and the absence of the uterus, ovaries or Fallopian tubes produce sterility, though allowing the gratification of sexual intercourse.

3. **Local Diseases.**—The female merely plays a passive role in the act of coitus, hence the local diseases of the genital organs do not, ordinarily, prevent sexual intercourse provided the vagina is normal, but they may produce sterility. Thus, the inflammatory affections of gonorrhœal infection involving the cervix, uterus, ovaries and Fallopian tubes, often produce sterility. Removal of both the ovaries owing to pathological conditions may not render a woman sterile, if a healthy portion of an ovary is left intact. Displacements and tumours of the

1. *Jour. Ind. Med. Assoc., Aug.*, 1932, *p.* 535 ; *Keane, Brit. Med. Jour., Sept.* 23, 1933, *p.* 567.
2. *Lancet,* 1881.
3. *Med. Juris., p.* 673.
4. *Times of India, March* 15, 1926.
5. *Boston, Med. and Surg. Jour.*, 1863.
6. *Dixonmann, Forens. Med., Ed. VI, p.* 76.
7. *Brit. Med. Jour., May* 13, 1933, *p.* 822.

uterus may be considered as causes of sterility, but not in all cases. Owing to the painful and spasmodic contraction of the constrictor muscle of the vagina at the time of coitus, vaginismus may lead to temporary impotence. Further, rectovaginal fistula, ruptured perinæum, disorders of menstruation, leucorrhœa and acid discharges from the vagina, may contribute towards sterility.

4. **General Diseases.**—General diseases and a bodily deformity in women are not barriers to sexual intercourse or conception if the generative organs and menstruation are normal. Thus, a woman suffering from paraplegia can become pregnant.

5. **Psychical Influences.**—Hatred, fear, passion, neurotic temperament, etc., may produce a hysterical fit on an attempt at copulation, and may thus render a woman temporarily impotent especially if she happens to be a virgin. It is possible for a woman to be sterile or impotent with a paritcular man, and quite the opposite with another.

CHAPTER XIV

VIRGINITY, PREGNANCY AND DELIVERY

VIRGINITY

The question as to whether a woman is a virgin (*virgo intacta*) arises in cases of nullity of marriage, divorce, defamation and rape.

SIGNS OF VIRGINITY

The signs of virginity in a healthy woman are seen in the genitals and breasts.

Genitals.—The labia majora are firm, elastic and well-rounded, and lie in close contact with each other so as to cover completely the labia minora or nymphæ and clitoris. The labia minora are soft, small and rose-coloured, and the clitoris is small. The vestibule is narrow. The posterior commissure and the fourchette are intact and crescent shaped. They are rarely destroyed by sexual intercourse, but are not infrequently lacerated in the attempts at sexual intercourse on children. The vagina is narrow and tight with rugose walls, but the rugosity of the vagina cannot be considered as a diagnostic proof of virginity, as it is only removed by the first birth, and not merely by sexual intercourse ; besides, in some cases it may be absent even in a virgin.

The hymen is the most reliable sign of virginity. It is a thin fold of mucous membrane situated at the orifice of the vagina. It is generally annular with a central opening which may be round or elongated. It is usually semilunar or crescentic with the opening anteriorly. Its free margin is, sometimes, fimbriated, having numerous notches which may be mistaken for artificial tears, but these natural notches are usually symmetrical, occur anteriorly and, as a rule, do not extend to the vaginal wall. The mucous membrane over the notches is also intact. On the other hand, tears caused by sexual intercourse or by introduction of any foreign body are usually situated posteriorly at one or both sides, or in the median line, and usually extend to the points of attachment of the hymen at the edge of the vagina.

The hymen is, sometimes, divided by a bridge of tissue into two equal or unequal openings, and is then known as a septate hymen. It is occasionally cribriform, presenting numerous minute openings. It may form a complete septum across the lower end of the vagina, when it is called an imperforate hymen. Nasiruddin[1] cites the case of a Mahomedan girl of 18 years who had an imperforate hymen. Mc Ilroy and Ward[2]

1. *Ind. Med. Gaz., May,* 1926, *p.* 232.
2. *Proceedings of the Royal Soc. of Med., March,* 1930, *p.* 633.

report the cases of three sisters in one family who had an imperforate hymen. It is said that the hymen may be congenitally absent, but no authentic case has so far been recorded.

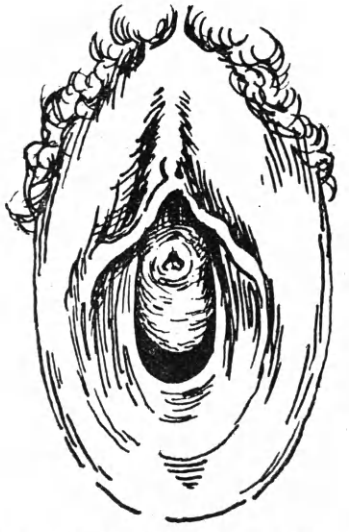


Fig. 103.—Semilunar or crescentic hymen.
(*From Peterson, Haines and Webster's Legal Medicine and Toxicology, Ed. II, Vol. I*).

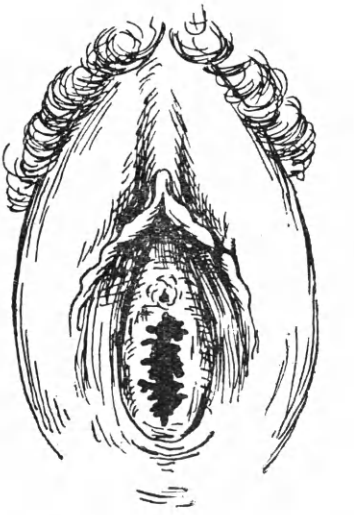


Fig. 104.—Fimbriate hymen.
(*From Peterson, Haines and Webster's Legal Medicine and Toxicology, Ed. II, Vol. I*).

The hymen is situated more deeply in children than in nubile girls, and so it more often escapes injury in attempted rape on children.

Normally the hymen is ruptured by the first act of coitus, though it may persist even after frequent acts of coitus if it happens to be loose, folded and elastic, or thick, tough and fleshy. Cases have been recorded in which the hymen had to be incised at the time of delivery, while even prostitutes have been known to possess an intact hymen.[1]

Cases of Persistent Hymen after Coitus.—1. A girl attended the out-patient department at the Broca Hospital in Paris for treatment of what was to all appearances an insignificant leucorrhœa. On examination the girl was found to be suffering from gonorrhœa and admitted that she had infected several of her customers, she being a clandestine prostitute of the purlieus of the Sorbonne. She had been in the town for over a year, and had entertained as many as five men in a single afternoon on a fete day. The hymen was, however, present, whose orifice was barely two millimetres in diameter. It was elastic, and admitted the passage of a large rectal bougie, returning to its obturator-like condition when this was withdrawn.—*Sutherland, Ind. Med. Gaz., June,* 1902, *p.* 245.

2. In October, 1920, an unmarried Mahomedan female of twenty eloped from her father's house with a young Mahomedan male. She stayed with him for about a week, and during this time she admitted to having had sexual intercourse with the man. On examination I found a superficial laceration, $\frac{1}{2}" \times \frac{1}{4}"$, along each side of the labia minora. The vaginal canal was dilated, but the hymen was intact, it being thick and fleshy.

1. *Guy and Ferrier, Forens. Med., Ed. VI, p.* 56.

Besides the act of coitus, the hymen may be ruptured by—

1. An accident, *e.g.*, a fall astride on a projecting substance, fence, or while playing at see-saw.

A child, 7 years old, while standing on an iron fence, fell striking the perinæum against a knob on the top of a post. The perinæum was lacerated, as also the vaginal wall as far as the cervix. The sphincter ani and the rectum were also lacerated. Hæmorrhage was very slight.—*Amer. Jour. of Obst.*, 1888, *Vol.* 21, *p.* 974.

Fig. 105.—Circular hymen presenting natural notches.

(From Peterson, Haines and Webster's Legal Medicine and Toxicology, Ed. II, Vol. I).

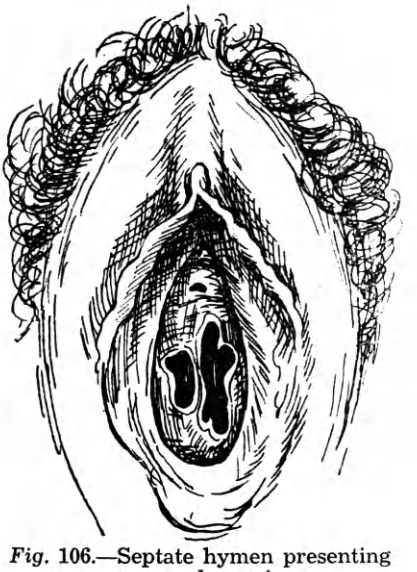


Fig. 106.—Septate hymen presenting unequal openings.

(From Peterson, Haines and Webster's Legal Medicine and Toxicology, Ed. II, Vol. I).

The plea that is usually brought forth by the defence pleader in a rape case in the mofussil Courts is that the hymen was ruptured by a fall on the stem of a plant, such as *arhar,* projecting a little above the ground in a field. In such a case the rupture of the hymen alone is highly improbable. Again, forcible separation of the thighs will not rupture the hymen, especially in children, unless the perinæum is ruptured. Owing to the situation of the hymen, its rupture is not possible by riding, jumping, dancing, etc.

2. Masturbation, especially if practised with some large foreign body. But the hymen is not destroyed in most cases, as the manipulation in little girls is generally limited to parts anterior to the hymen. In such cases the nymphæ are elongated and the clitoris enlarged by the continued practice of masturbation. The vaginal orifice may be dilated, and the edge of the hymen may show at the most a scratch produced by the finger nail. The hymen is, however, liable to be ruptured by forcible

introduction of a stick or finger constituting an indecent assault on small girls.[1] I saw a girl, six years old, in whom the posterior part of the hymen, the posterior wall of the vagina and the posterior commissure were lacerated by a thumb forcibly introduced into the vagina.

Fig. 107.—Hymen presenting two lateral lacerations.

(From Peterson, Haines and Webster's Legal Medicine and Toxicology, Ed. II, Vol. I).

Fig. 108.—Circular hymen torn in several places.

(From Peterson, Haines and Webster's Legal Medicine and Toxicology, Ed. II, Vol. I).

3. Introduction of instruments by medical men during examination or a surgical operation.

4. A foreign body, such as a sola pith, introduced purposely with a view to render very young girls fit for sexual intercourse (*aptœ viris*). This is, sometimes, resorted to by prostitutes.

Chevers mentions on the authority of Dr. S. C. Mackenzie that the bawds who train up girls to prostitution insert a piece of sola pith as large as the vagina will contain, and then make the unfortunate sit in water; a dilating action similar to that of a sponge tent is the consequence. They gradually increase the size of the plug.—*Med. Juris., Ed. III, p.* 689.

Casper relates the case of a mother, who, in order to fit her daughter, aged ten, for having painless intercourse with men, dilated the vagina by introducing two fingers at first, and then four fingers and lastly stuffed a longish stone into it. The vagina was wider than is usual in children at that age and there were several lacerations on both sides of the hymen.—*Forens. Med., Eng. Trans., Vol. III, p.* 318.

5. Ulceration from diphtheria, noma, or other diseases. In such cases the whole hymen is destroyed leaving a scar only. Sir Bernard

1. *Thoinot's Medico-Legal Aspects of Moral Offences, Eng. Trans., p.* 75.

Spilsbury[1] reports a case in which destruction of a child's hymen was due to threadworms, and until the cause was known suspicion of foul play was entertained. Persistent pruritus in children is likely to lead to injury of the hymen.

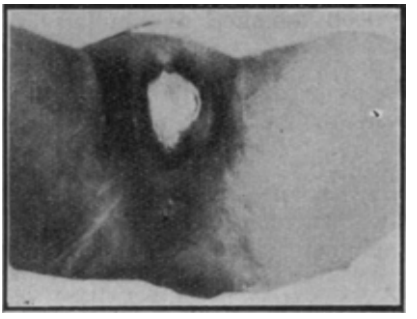


Fig. 109.—Intact hymen in a pregnant woman ; it had to be ruptured during labour. (*From a photograph kindly lent by Dr. G. B. Sahay*).

6. Clots of menstrual blood passing through the vagina, but this is highly improbable.

Breasts.—These are firm, elastic and hemi-spherical, with a small undeveloped nipple surrounded by an areola, which is pink in fair women and dark brown in dark women. The breasts become large and flabby by frequent handling and sexual intercourse, as well as by masturbation, but are not affected by a single act of coitus.

PREGNANCY

In Courts of law the question of pregnancy may be disputed under the following circumstances :—

(1) When a woman advances pregnancy as a plea to avoid attendance in Court as a witness in an important trial.

(2) When a condemned woman pleads pregnancy as a bar to hard labour or execution. Under section 382 of the Indian Criminal Procedure Code, the High Court is the only judicial court which can postpone the execution of a sentence of death confirmed by it, or commute it to transportation for life, after it is satisfied from the Civil Surgeon's certificate that the woman is pregnant. The usual certificate required from the Civil Surgeon in such a case is as to whether the woman is "quick with child" or not. In England by the Sentence of Death (Expectant Mothers) Act, 1931, sentence of penal servitude instead of sentence of death is to be passed on a woman condemned of an offence punishable with death, if she is found to be pregnant. The trial jury, without being re-sworn, will have to determine the question of pregnancy from the evidence adduced before it either on the part of the woman or on the part of the Crown.

(3) When a woman feigns pregnancy soon after her husband's death so as to defraud the rightful heir by producing a supposititious heir to an estate, the heir-at-law may apply to the Court to order an inquiry into the allegation.

(4) When a woman, who has filed a suit in Court for breach of promise of marriage or for seduction, claims to be pregnant.

1. *Lancet, May,* 7, 1932, *p.* 990.

(5) When a woman blackmails a gentleman, and accuses him that she is pregnant by him.

(6) When a widow, or an unmarried woman, or a married woman living separate from her husband, has been defamed or libelled to be pregnant.

(7) When a woman alleges that she is pregnant in order to secure greater compensation from some person or persons, through whose culpable neglect her husband has died.

(8) When pregnancy is alleged to have been a motive for suicide or murder of an unmarried woman or a widow. In such a case the dead body has to be examined for the proof of pregnancy.

SIGNS OF PREGNANCY

The signs of pregnancy in the living may be classified as *subjective* and *objective signs.*

SUBJECTIVE SIGNS

1. Cessation of menses.
2. Morning sickness.
3. Sympathetic disturbances.
4. Quickening.

1. **Cessation of Menses.**—This is the first sign of pregnancy, but it cannot be relied on as menses may be suspended in certain diseases, such as anæmia, phthisis, cancer and nervous excitement. Unmarried women without being pregnant may miss their menstrual periods for some time after illicit intercourse simply from fear and nervousness. In married women an intense desire for pregnancy may stop menstruation for some time. Pregnancy may occur in a woman who has never menstruated. It has already been mentioned that pregnancy has occurred in some cases even after the climacteric period. It may also occur in a woman during the amenorrhœa of lactation. When a woman suckles her child she does not usually menstruate for the first six months after delivery, but it is quite possible for her to be impregnated during this period. Digby[1] relates the case of a woman who was delivered of a healthy full-term female child on February 3, 1929. The child was breast-fed and the mother never menstruated. She was again delivered of another fully developed female child on December 1, 1929.

From his investigations Bonnar[2] has fixed the 14th day after delivery as the earliest at which a fresh impregnation may take place. Conception, however, took place on the fourth day after delivery in the following case recorded by Brasseur[3] :—

1. *Brit. Med. Jour., April* 11, 1931, *p.* 652.
2. *A Critical Inquiry regarding Superfœtation with cases,* 1865 ; *Guy and Ferrier, Forens. Med., Ed. VI, p.* 136.
3. *New York Med. Record ; Ind. Med. Gaz., April,* 1894, *p.* 153.

A woman, 22 years old, was delivered of her child on July 4, 1892. On July 8, she practised coitus, and was again delivered of a healthy child on March 10, 1893. Calculating from the date of coitus, the second pregnancy lasted two hundred and forty-three days. Ovulation must have existed in the woman on the fourth day after the delivery, and it was necessarily quite independent of menstruation.

In rare cases menstruation may occur for two or three periods after conception.

Lastly, a woman may practise deception on the medical jurist by denying the stoppage of the monthly course, and imitating the catamenia by blood, if she wants to conceal pregnancy. Similarly, she may conceal menstruation if she feigns pregnancy. In this case the fact of her having menstruated may be found by inquiring of her washerwoman (*dhoban*).

2. **Morning Sickness.**—Nausea or vomiting, usually as a sign of pregnancy, most frequently occurs soon after the woman rises from bed in the morning. It commences about the beginning of the second month, and lasts generally till the end of the fourth month. It may, however, commence soon after conception. It is not a reliable sign, as it may occur in gastric troubles or chronic alcoholism, irrespective of pregnancy.

3. **Sympathetic Diturbances.**—Salivation, perverted appetite in the form of longings or cravings for very strange and even disgusting articles of food, and irritable temper are a few of the conditions which are caused reflexly by pregnancy.

4. **Quickening.**—The first perception of the fœtal movements felt by the mother is known as " quickening." It is attributed to the uterus coming into contact with the abdominal wall, and usually occurs from the 14th to the 18th week, but sometimes as early as the 12th. It is peristaltic in nature, and when observed, the woman is said to be " quick with child."

In the case of spurious pregnancy a nervous and hysterical woman may experience these sensations. Again, an inexperienced primipara may not feel them at all.

None of the above signs are reliable, and the medical jurist should never venture an opinion on these signs alone.

OBJECTIVE SIGNS

These are—

1. Mammary changes.
2. Pigmentation of the skin.
3. Changes in the vagina.
4. Changes in the cervix uteri.
5. Softening and compressibility of the lower segment of the uterus.
6. Enlargement of the abdomen.
7. Intermittent uterine contractions.
8. Fœtal movements.
9. Uterine souffle.
10. Fœtal heart sounds.
11. Ballottement.
12. X-Ray examination.
13. The Biological Test.

1. **Mammary Changes.**—From the very commencement of pregnancy the breasts become full and tender, and by the second month begin actually to increase in size. The superficial veins are seen more distinct and enlarged. The nipples are harder, firmer and more prominent, and the areolæ surrounding them become wider and darker. By the third month a clear, transparent secretion can be squeezed out of the nipples on pressing the breasts. This contains the colostrum corpuscles or milk, as pregnancy advances. Milk has, however, appeared in the breasts of women who have not been pregnant. Jago[1] reports a case in which a woman, who had never been pregnant, had a copious flow of milk from her breasts, and suckled a child of another woman. David Krestin[2] also reports the case of an unmarried woman, aged 25, with hymen intact, who had enlarged breasts, which yielded milk on compression due to enlargement of the pituitary fossa.

After the sixth month silvery lines or striæ similar to the lineæ albicantes of the abdomen are seen especially in primiparæ on account of the stretching of the skin.

All these changes may, however, occur from various uterine and ovarian diseases. Sometimes, they also occur independent of pregnancy when women have reason to expect it shortly after marriage or after illicit intercourse.

Again, pregnancy may occur without any changes in the breasts, or the breasts may even diminish in size after the middle of pregnancy.

These changes are also of very little diagnostic value after the first pregnancy as the areolæ retain their colour permanently, and the secretion of milk from the breasts is more or less permanent.

2. **Pigmentation of the Skin.**—This is well marked in dark women. The abdomen, axillæ and pubes become darker due to the deposit of pigment, and a special dark band (linea nigra) is observed extending from the ensiform cartilage to the pubes.

3. **Changes in the Vagina.**—After the fourth week of pregnancy the normal pinkish colour of the mucous membrane of the vagina and vulva changes to violet, deepening to blue, as a result of venous obstruction owing to pressure of the gravid uterus. This is known as Jacquemier's sign, as it was first described by him. This sign may, however, be found just before, as well as immediately after, menstruation.

The anterior wall of the vagina is found flattened. This results from backward traction by the upward tilted cervix, and has been described by Dr. Barnes as a sign of pregnancy.

When introduced into the vagina the fingers may feel the pulsation of the vaginal arteries consequent on the high arterial tension of the pelvis.

4. **Changes in the Cervix Uteri.**—From the very first month of pregnancy the cervix which is normally as hard as the tip of the nose

1. *Sydney Smith, Forens. Med., Ed. VI, p.* 301 ; *Kenya and East Africa Med. Jour., July,* 1927.
2. *Proceedings of the Royal Soc. of Med., April,* 1932, *p.* 693.

begins to soften from below upwards, and is felt as soft as the lips. By the fourth month this softening can be very well felt by the fingers introduced into the vagina. This is a diagnostic sign of pregnancy, and is known as Goodell's sign, though certain morbid conditions, such as acute metritis, hæmatometra, etc., may produce softening of the cervix.

As softening continues, and involves the whole neck of the uterus, there is an apparent shortening of the cervix towards the last months of pregnancy. The orifice, instead of being transverse, bcomes circular, and admits the point of the finger more readily, and to a greater depth.

5. **Softening and Compressibility of the Lower Segment of the Uterus.**—This is known as Hegar's sign, and is elicited by bimanual examination. It is regarded as a valuable sign of early pregnancy from the second to the fifth month, but it may be found in soft uterine myomata. C. J. Gauss[1] published in 1920 a modification of Hegar's sign. The cervix presents in the first and second months of pregnancy an abnormal motility. It may easily be pushed to either side without entailing a corresponding movement of the uterus. This phenomenon is comparatively rare outside of pregnancy.

6. **Enlargement of the Abdomen.**—The abdomen begins to enlarge gradually after the third month. Up to the first three months the gravid uterus remains in the cavity of the pelvis, and about the fourth month rises just above the symphysis pubis and comes into contact with the abdominal wall. At the end of the fifth month it is midway between the symphysis and the umbilicus (navel). At the end of the sixth month it reaches the level of the umbilicus, and at the end of the seventh month it is midway between the umbilicus and the ensiform cartilage. At the end of the eighth month and in the early part of the ninth month it reaches the ensiform cartilage or epigastrium. During the last two months the uterus on account of its weight does not rise higher, but sinks deeper into the pelvis and tends to fall forward.

The enlargement of the abdomen may occur in ascites, ovarian cysts, ovarian and uterine tumours, and in phantom tumours.

7. **Intermittent Uterine Contractions.**—Throughout pregnancy, the uterus is subject to alternate contractions and relaxations, but before the third month it is difficult to observe them except by a very careful bimanual examination. After the fourth month the uterus can be easily felt as alternately contracting and relaxing by palpating the abdomen. The period of contraction and relaxation is variable, each contraction lasting from one to five minutes, and each relaxation from five to twenty minutes. This phenomenon is known as Braxton Hick's sign, and is considered as a valuable proof of pregnancy. It is present even when the fœtus is dead or degenerated. It may, sometimes, be present in cystic distension of the uterus, in large soft uterine myomata or in large intra-uterine polypoid growths.

1. *Zentralblatt ful Gynakologie, Leipzig, April* 3, 1926, *p.* 875 ; *Jour. Amer. Med. Assoc., June* 5, 1926, *p.* 1810.

8. **Foetal Movements.**—Fœtal movements are felt and seen through the abdomen after the sixth month. They may be felt on bimanual examination through the vagina at the commencement of the third or fourth month, and may be heard on auscultating the abdomen about the middle of the fourth month. The fœtal parts may also be palpated through the abdominal walls. This is a certain sign of pregnancy.

9. **Uterine Souffle.**—The uterine souffle is described as a soft, blowing murmur, synchronous with the mother's pulse, and heard towards the end of the fourth month on either side of the uterus just above Poupart's ligament. In some cases it may be heard as early as the ninth or tenth week. This sign is not infallible, because it may be heard in uterine or ovarian tumours.

10. **Foetal Heart Sounds.**—The sounds of the fœtal heart constitute by far the most important sign of pregnancy. They are usually heard for the first time in the course of the fifth month, generally from the eighteenth to the twentieth week, and are compared to the muffled ticks of a watch under a pillow. They vary in rate from one hundred and twenty to one hundred and sixty per minute, and are not synchronous with the mother's pulse. They are not heard when the fœtus is dead, when there is an excessive quantity of liquor amnii or when the abdominal walls are very fat.

11. **Ballottement.**—This is the name given to the sensation observed by moving the fœtus about in the liquor amnii. It can be felt internally per vaginam or externally through the abdominal wall. Ballottement can be tried from the fourth to the seventh month, but the test fails if the amniotic fluid is scanty. In practised hands it is a sign of great value.

12. **X-Ray Examination.**—This is useful in the diagnosis of pregnancy after the sixteenth week when the centres of ossification have become well developed. The X-ray examination is not harmful to mother or fœtus, as the exposure with the modern apparatus lasts only a few seconds. The X-rays are of great assistance particularly in a case of twins where one ovum is suspected to be bigger than the other and in cases of suspected hydatidiform mole, pregnancy with fibroids, and fœtal malformations.

13. **The Biological Test.**—From the above mentioned signs it is evident that before the sixteenth or eighteenth week there are no certain signs from which a medical man can give a definite opinion about the existence or otherwise of pregnancy. In such a case it is always a safe plan to wait till the definite signs develop or to perform a biological test devised by Aschheim and Zondek in 1928 for detecting pregnancy in its early stage. This test is popularly known as the Aschheim-Zondek test, and is based on the fact that in a pregnant woman an abnormal amount of the anterior-pituitary-like hormone of the chorionic villi is excreted in the urine, and that sortly after the puerperium this excessive excretion of the anterior-pituitary-like hormone stops. The presence of this hormone can be demonstrated by significant developmental changes in the sex organs of sexually immature female white mice, when small amounts of a pregnant woman's urine are injected subcutaneously. The ovaries are enlarged. The corpora lutea are formed and hæmorrhagic spots occur into the

follicles. There are often swelling and hyperæmia of the uterus. The technique for performing the test is as follows :—

Five sexually immature female white mice, three to four weeks old and weighing from six to eight grammes, are inoculated twice daily for three days with the catheterized morning urine of the suspected case of pregnancy in quantities of 0.2 c.c., 0.25 c.c., 0.3 c.c., 0.3 c.c. and 0.4 c.c., respectively One hundred hours after the commencement of the test the mice are killed and the ovaries are inspected with a hand lens or with the naked eye. A positive reaction is characterised by the presence of corpora lutea and hæmorrhages into the follicles of the enlarged ovaries.

If the urine is turbid, it should be filtered and its reaction made slightly acid if it is not already so. One drop of tricresol to each 30 c.c. of urine should be added if the specimen is not to be used at once or if it has to be sent by post.[1]

The Friedman modification of this test can be carried out by injecting 7 to 10 c.c. of the suspected morning urine into the marginal ear vein of a virgin female rabbit, 12 to 14 weeks old and weighing not less than 4 pounds. Twenty-four to thirty hours later a positive reaction will be indicated by the presence of corpora lutea and corpora hæmorrhagica in the hypertrophied ovaries of the rabbit. There will also be marked injection of the uterus and oviduct.

The Aschheim-Zondek test gives a positive reaction in 98 to 100 per cent of the cases of pregnancy. It is positive as early as ten to fourteen days after conception and two days after the first missed menstrual period. It remains positive throughout pregnancy and for a period of about seven days after parturition. It also gives a positive reaction in ectopic gestation, hydatidiform mole and chorion-epithelioma.

Signs of Pregnancy in the Dead.—In addition to some of the objective signs mentioned above, the diagnostic signs of pregnancy which are found in the dead body at the post-mortem examination are—

1. The presence of an ovum or fœtus.
2. Uterine changes.
3. The corpus luteum.

1. **The Presence of an Ovum or Foetus.**—The presence of an impregnated ovum, fœtus or placenta in the uterus after death is positive proof of pregnancy. In place of the ovum certain abnormal products of conception, such as sanguineous and vesicular moles, may, sometimes, be present. These moles develop so very rapidly that the uterus is usually larger than at the corresponding period of normal pregnancy, but more frequently it is not enlarged beyond its size at the fifth or sixth month of gestation.

2. **Uterine Changes.**—As a result of pregnancy the uterus is thickened, and increases in size, both in its length and width. The length increases from one-and-a-half inches to twelve inches, and the width from

1. *Dharmendra, Ind. Jour. Med. Res., July,* 1931, *p.* 239.

one-and-a-half to about nine inches. Its weight at the full term of pregnancy is twenty-eight ounces or more.

The nulliparous uterus weighs about an ounce, and that of the woman who has borne children weighs about an ounce and a half. The uterus also increases very much in its capacity, being five hundred or more cubic inches at its full development. The marks of the attachment of the placenta are also noticed.

3. **The Corpus Luteum.**—The corpus luteum is a cicatrix formed in the ovary after the escape of ova from the bursting of a Graafian follicle at the menstrual period. This corpus luteum develops in size for the first five or six days after the rupture of the follicle, remains quiescent for a few days more and then undergoes rapid absorption. In the event of pregnancy the corpus luteum continues to develop, and attains the largest size about the fourth month, forming a firm projection on the surface of the ovary. It then undergoes a slow retrogressive change, although it is usually well marked at the time of delivery, and may be evident for one or two months after.

The corpus luteum used to be regarded as a positive sign of pregnancy, but it has now no forensic value, inasmuch as it is seen as a result of over-congestion, as in fibroid tumours and other pathological conditions. It has also been found in the ovaries of women who were neither pregnant nor menstruating. Moreover, pregnancy has occurred without the formation of a corpus luteum.

DELIVERY

The cases in which the medical jurist is required to ascertain whether a woman has been delivered or not, are those of abortion, infanticide, concealment of birth, feigned delivery, legitimacy, and libel actions.

SIGNS OF DELIVERY

These signs are discussed under the following four headings :—

I. The signs of recent delivery in the living.
II. The signs of recent delivery in the dead.
III. The signs of remote delivery in the living.
IV. The signs of remote delivery in the dead.

I. SIGNS OF RECENT DELIVERY IN THE LIVING

The signs of recent delivery at full term are—

1. **Appearance of General Indisposition.**—For the first two or three days after delivery the woman wears a languished look, with the sunken eyes having a dusky pigmentation about the lower eyelids, and has a slight increase in the pulse and temperature. These signs may be absent in strong women, or may be found in any other illness or at the time of the monthly course. The intermittent contractions of the uterus are usually present for the first four or five days. These are termed after-pains when they are vigorous and painful.

2. **Breasts.**—The breasts are full, firm, knotty and enlarged, and contain colostrum or milk. The areolæ are dark and the nipples turgid.

3. **Abdomen.** The abdomen is slightly full, but more often lax and flabby. The skin is wrinkled and shows the lineæ albicantes, which are pinkish in the beginning, but subsequently become white in colour.

4. **Uterus.**—Just after delivery the uterus relaxes, and may be felt as a flabby mass extending to the umbilicus a few hours after delivery. It then diminishes in size, and is felt like a hard cricket ball for about two or three days in the lower part of the abdomen above the symphysis pubis, but its fundus can be felt just above or behind the symphysis pubis up to the fourteenth day.

5. **Vagina.**—The labia are tender, swollen and bruised or lacerated. The vagina is smooth, relaxed and dilated, and may show recent tears. The fourchette is usually ruptured, and the perinæum is sometimes lacerated.

6. **Cervix.**—The cervix is soft and patulous, and its edges are torn or lacerated transversely. The internal os begins to close during the first twenty-four hours. The external os is soft and patent, admitting two fingers for a few days. It admits with difficulty one finger at the end of a week, and closes in two weks.

7. **Lochia.**—The lochia is a discharge from the uterus and vagina, lasting for the first two or three weeks after delivery. It has a peculiar, sour, disagreeable odour. During the first three or four days the discharge is bright red (*lochia rubra*) consisting of pure blood mixed with large clots. It becomes serous and paler in colour (*lochia serosa*) during the next four days. About the ninth day the colour becomes yellowish-grey or slightly greenish (*lochia alba* or *green water*), and gradually diminishes in quantity, till it disappears altogether from the second to the third week.

From the above signs taken collectively it will scarcely be difficult to diagnose a case of recent delivery for the first fourteen days after parturition. These signs are more characteristic of a full-term delivery than of a premature one. They are likely to disappear within a week or ten days or even at an earlier date in a strong and vigorous woman, especially if she happens to be a multipara.

II. SIGNS OF RECENT DELIVERY IN THE DEAD

The diagnosis of recent delivery in the dead hardly presents any difficulty. In addition to the signs described above, the uterus is found flabby and nine to twelve inches long, containing large clots of blood, and its inner surface is lined by the decidua if a necropsy is held on the body of a woman who has died soon after delivery. The uterus, in course of time, becomes more and more contracted. In the first two or three days after a full-term delivery it is about seven inches long and four inches broad. At the end of a week it is between five or six inches long and about an inch thick. At the end of a fortnight it hardly exceeds five inches in length, and returns to the normal size in about six weeks. Soon after delivery the uterus weighs about twenty-eight ounces, twelve ounces

at the end of a week or ten days and about one and a half ounces by the end of a month.

The site of the placental attachment is of a dark colour. The openings of its vessels are well marked, and recognisable for two or three months.

The ovaries and the Fallopian tubes are usually congested, but may become normal in a few days. A large corpus luteum is usually found in one of the ovaries.

III. SIGNS OF REMOTE DELIVERY IN THE LIVING

1. **Abdomen.**—The abdominal wall is relaxed, and marked with white silvery streaks, called the lineæ albicantes, which result from over-distension. These lines also occur from ascites, ovarian tumours, etc.

2. **Breasts.**—These are soft and pendulous, marked with lineæ albicantes. The areolæ are dark. The nipples are prominent and larger than usual, unless the woman has not suckled her child.

3. **Vagina.**—The labia are more or less separated from each other. The vagina is somewhat capacious, its rugæ are absent, and its walls are relaxed, especially in a multiparous woman. The fourchette and posterior commissure are destroyed, and the perinæum may be found ruptured. The hymen is absent, or may be seen as separate nodules in the form of carunculæ myrtiformes.

4. **Cervix.**—The cervix is cleft transversely with ragged and irregular margins. The os is wider.

Most of the above signs may possibly be simulated by the passage of a large fibroid tumour per vaginam. Again, most of these signs may disappear in a woman who had had only one delivery short of the full-term several years before, and it is possible for the vagina and uterus to regain normal appearances as observed in a nulliparous woman. In exceptional cases no trace of a previous delivery may be found even on women who have borne several children.

Montgomery[1] reports a case in which he examined a lady who had borne five children and nursed three of them. He found that "her breasts were small, but neither flaccid nor pendulous; the nipple short, with not the least shade of brown colour in the areolæ, which exhibited only the delicate rose colour so often observed in that part of the virgin breast; there were neither lines nor spots of any kind on the abdomen; the os *uteri* was small and natural, the vagina contracted and the fourchette perfectly entire." This lady used to be delivered at the eighth month of her pregnancy.

Glaister[2] mentions the case of a married woman whom he examined in 1887. She was 30 years old, and had borne four children and nursed each of them for nine months, the last being three years old. Her breasts were virgin-like in appearance and there was a total absence of lineæ albicantes and pigmentation around the nipples.

1. *Cyclop. Pract. Med., Vol. IV, p.* 504.
2. *Med. Juris. and Toxic., Ed. VI, p.* 351.

IV. SIGNS OF REMOTE DELIVERY IN THE DEAD

In the dead body of a woman who has borne children, the uterus is larger, thicker and heavier than the nulliparous uterus. The walls are concave from inside, forming a wider and rounded cavity, while the walls of a nulliparous uterus are convex on the inner aspect, and form a cavity which is smaller in capacity and triangular in shape. The top of its fundus, as seen from the front or from the back, is convex and on a higher level than the line of the broad ligaments. The cervix is irregular in form and shortened, and its edges show cicatrices on account of previous tears and lacerations caused during delivery. The external os is enlarged, irregular and patulous so as to admit the tip of the finger, and the internal os is not so well defined as in the virgin or nulliparous woman. It must be remembered that the uterus undergoes atrophy in old age.

CHAPTER XV

LEGITIMACY

According to the law of England, a child born during lawful marriage (wedlock) or within a competent time after the dissolution of such marriage or after the death of the husband is presumed to be a legitimate child of the husband unless it is proved that the husband was impotent, or that the husband and the wife had no sexual access to each other at a time when conception could have taken place. Under section 112 of the Indian Evidence Act[1] there is a presumption in favour of legitimacy of a child born during the continuance of a valid marriage between his mother and any man, or within two hundred and eighty days after its dissolution, the mother remaining unmarried, and the presumption can only be rebutted if it is shown by competent evidence that the parties to the marriage had no access to each other at any time when the child could have been begotten. In England the presumption of legitimacy may be rebutted by proof of the impotence or sterility of the husband, but cannot be done so under the Evidence Act of India.

An *illegitimate* or *bastard* child is one which is born out of wedlock or not within a competent time after the cessation of the relationship of man and wife or born within wedlock when procreation by the husband is not possible. By the law of Scotland and by the Legitimacy Act of England amended in 1926, an illegitimate child becomes legitimate by the subsequent marriage of the parents, and inherits the property of its father.

The question of legitimacy may arise in the following cases :—

1. **Inheritance.**—A legitimate child born during lawful wedlock can inherit the property of its father. According to the law of England a monster, which has not the shape of mankind, is incapable of inheriting, but there is nothing specific on this point in Indan Law.

A monster generally does not live after it is born, but double monsters of the varieties of the Siamese twins may live to adult age. They are united mostly in the umbilical region or at the pelvis, and have some organs common to both.

2. **Tenancy by Courtesy of England.**—If a man marries a woman who owns estates, and has by her a child born alive, he shall, for his lifetime, become the tenant of the estates by the *Courtesy of England* after the death of his wife, but the child should be born during lawful wedlock. Thus, the husband cannot have any interest in the estates if the child was delivered alive by Cæsarian section after the mother's death, though such a child is regarded as a legitimate child. If she has had no child born alive, her estates pass to her next heir-at-law at her death.

The law of tenancy by courtesy is not tenable in India, for section 4 of the Indian Succession Act of 1865 enacts that a person marrying after

1. Vide *Appendix V.*

the 31st December, 1865, shall have no interest in the property of the person whom he or she marries.

It may also be mentioned that by the Administration of Estates Act, 1925, the old law as to inheritance and succession to property in England (for instance, heirship and tenancy by the courtesy) was abolished and replaced by a simple code for the devolution of property upon the death of a person intestate.

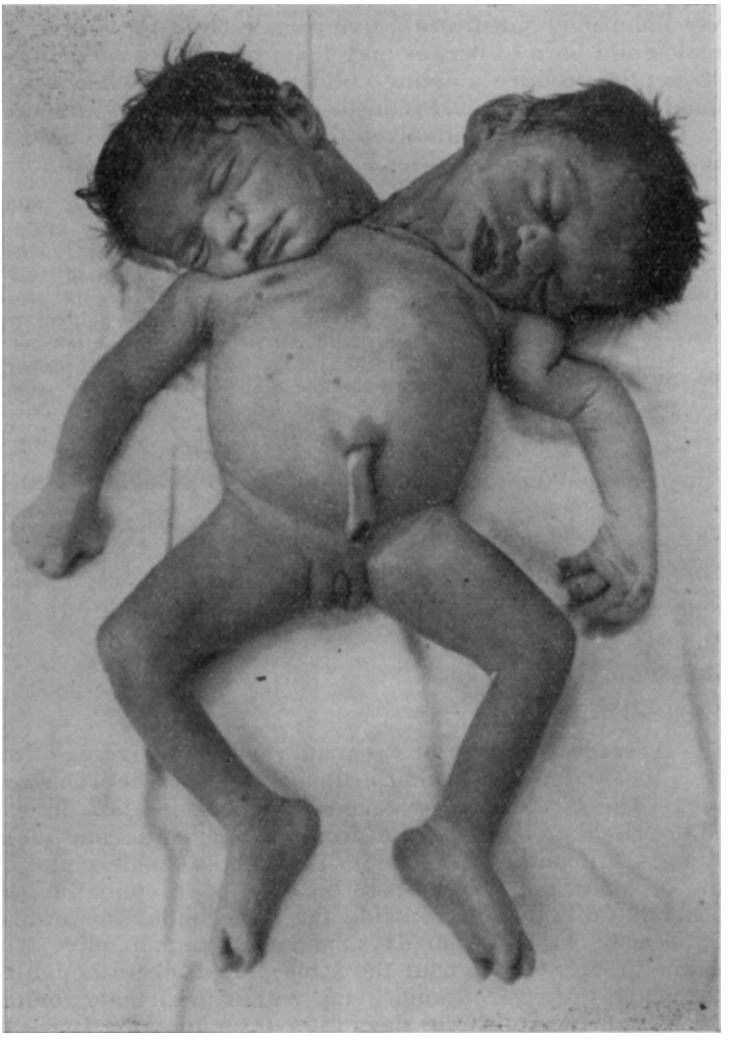


Fig. 110.—Monster: Front view showing the relation of the heads to the body. (*From a photograph kindly lent by Dr. H. S. Mehta*).

3. **Affiliation Cases.**—These are the cases which are brought before the Court for fixing the paternity of an illegitimate child upon a certain

individual, as he is bound, under section 488 of the Indian Criminal Procedure Code, to support his illegitimate child which is unable to maintain itself and which has not reached the age of majority. A Magistrate of the first class may make a monthly allowance of any sum not exceeding fifty rupees on the whole for the maintenance of such child. In determining the amount of maintenance, luxury is not to be taken into consideration but only the necessaries of life, *viz.*, food, clothing and lodging.

4. **Supposititious Children.**—A supposititious child means a fictitious child. A woman may substitute a living male child for a dead child or a living female child born of her, or may feign pregnancy as well as delivery and subsequently produce a living child as her own when she wants to extort money or to divert succession to property. Such cases occur when succession to large estates is involved or when money is to be extorted by blackmail.

In 1922, a case occurred at Ahmedabad, where a young widow abducted, with the help of a nurse, from the Victoria Jubilee Hospital, a newly born child which she passed off as her own, alleging that it had been born after her husband's death (*posthumous child*), and pretended delivery while in fact she had had none. In October, 1923, a Bhatia widow[1] of Bombay was sentenced to one year's simple imprisonment and a fine of Rs. 2,000, for having tried, with the help of two accomplices, to conceal the fact of her giving birth to female twins soon after her husband's death by substituting a male child and claiming a share in the property of her husband. The two accomplices were also sentenced to various terms of imprisonment.

MEDICO-LEGAL POINTS

The medico-legal points that have to be investigated in these cases are—

1. The average duration of pregnancy.
2. The maximum period of pregnancy.
3. The minimum period of pregnancy and the viability of a child.
4. Superfecundation.
5. Superfœtation.
6. Paternity.

1. **The Average Duration of Pregnancy.**—By the average duration of pregnancy is meant the period that ordinarily elapses between conception and delivery. The circumstances taken into consideration in estimating this period are the date of conception from a single coitus and the arrest of menstruation. But neither of these is reliable ; a single coitus does not fix the date of conception, but merely the date of insemination. Spermatozoa are capable of surviving outside the body under favourable conditions for a period of about ten days, and may remain alive within the Fallopian tube or uterus for about the same period, even though cases are recorded in which they were found living within the female genital organs for seventeen[2] and twenty-three[3] days after insemination. It is, therefore,

1. *Leader, Oct.* 20, 1923.
2. *Bossi, Gazetta degli Ospitali, April* 18, 1891, *Taylor, Princ. and Pract. of Med. Juris., Ed. IX, Vol. II, p.* 40.
3. *Duhrssen, Centrablatt fur Gynœk.*, 1893 ; *T. W. Eden, Trans. of Med.-Leg. Soc., Vol. XVII,* 1922-23, *p.* 159.

possible for conception to occur at any time within ten days after a single coitus has taken place. However, conception usually occurs three to four days after coitus.

The exact time of conception during the intermenstrual period is not known. It is generally assumed that ovulation occurs about fourteen days before the commencement of menstruation, and the ovule probably perishes in two or three days after it is shed unless fertilised. Hence fertilisation may occur if coitus has taken place two or three days before and about the same period after ovulation.

From the above points it is quite clear that the actual duration of pregnancy in the female is not known; however, the average period calculated from experience is two hundred and eighty days, or forty weeks, or ten lunar months. This is equivalent to ten times the normal intermenstrual period which is usually twenty-eight days. It has been observed that in women whose intermenstrual period is shorter than the usual time pregnancy has terminated at the eighth or ninth lunar month or even earlier, the child having attained full development. Sidney H. Waddy[1] describes a case in which a woman, aged 30 years, gave birth to a full-time daughter after gestation of 210 days—ten times three weeks—which was her normal intermenstrual period. The child cried lustily at birth, had a good crop of hair, was well coated with vernix caseosa, measured twenty inches in length, and weighed seven pounds. The finger and toe nails were fully developed and the child sucked vigorously on being put to the breast.

2. **The Maximum Period of Pregnancy.**—Sometimes, cases of disputed legitimacy arise in which it is necessary to determine how long gestation may be prolonged. In India, England and the United States the law does not lay down any fixed limit of gestation. Each case is decided on its own merit. The longest period of gestation allowed by the law in France, Italy and Scotland is 300 days and in Germany 302 days. In America the law has allowed pregnancies lasting 313 and 317 days in cases for the legitimacy of births.[2] Dr. Phillips reports the case of a young unmarried girl in whom gestation lasted 324 days after the cessation of her last menstruation and 311 days after the date of coitus.[3]

In the divorce suit[4] of *Gaskill* v. *Gaskill* the Lord Chancellor accepted 331 days as a period of protracted gestation. During the trial Eden said in his evidence that in cases of such prolonged pregnancy the child would be much above the average weight and dimensions at the time of birth. He cited six cases accepted as authentic in which the calculated period lay between 311 and 336 days, and the weights of the children varied from $12\frac{3}{4}$ to $13\frac{1}{4}$ pounds.[5] But in this particular case the child was not weighed or measured.

1. *Brit. Med. Jour.*, *Jan.* 14, 1928, *p.* 75.
2. *Witthaus, Med. Juris. and Toxic., Vol. II, p.* 514.
3. *Lancet*, 1900, *Vol. I, p.* 94.
4. *Brit. Med. Jour., Aug.* 6, 1921, *p.* 220.
5. *Transactions, Med.-Leg. Soc.*, 1922-23, *Vol. XVII, p.* 168.

D. Ropez[1] reports a case in which pregnancy lasted 352 days calculated from the last menstrual period and probably 344 days from the coitus which resulted in the conception. The last menstrual period was on February 25, 1920. By March 15, morning sickness started. In July, the fœtal heart sounds could be heard, and delivery took place on February 10, 1921. Tausch[2] also describes the case of a primiparous woman, 27 years old, who was delivered of a well-developed girl 343 days after her last menstrual period. The girl was considerably larger than the normal child, was 56 c.m. long and weighed 5,000 grammes.

3. **The Minimum Period of Pregnancy and the Viability of a Child.**— In a case of disputed ligitimacy, when a child is born within a short time after marriage, or within a short time of the husband and wife living together after some years' separation, an important question that is raised is whether it is possible for a fully developed child to be born before the termination of the usual period of gestation. This question can be answered by determining the intra-uterine age of the fœtus from its length, weight and other characteristics, and in most of these cases it will be found that the fœtus is not full term, and yet it is capable of living. The question, therefore, resolves itself into another, *viz.*, what is the shortest period of gestation at which a viable child can be born ?

Children born at or after two hundred and ten days or seven calendar months of uterine life are viable, *i.e.*, are born alive and are capable of being reared. Hubbard[3] records a case where an infant born at the beginning of the seventh month of pregnancy weighed only 15 ounces, and at the age of six weeks was in good health and weighed 32¾ ounces. It was fed on breast milk from a bottle with one feed daily directly from the breast. Children born after six calendar months or one hundred and eighty days of uterine life may be viable and capable of continuing an independent life apart from their mothers. Houlihan[4] reports the case of a primipara, who was delivered of a premature, living male infant on July 29, 1932, after 6½ months of gestation. At birth the infant was 14 inches long and weighed 23½ ounces. At the end of 12 weeks it weighed 90¼ ounces. Dr. Outrepont[5] of Bamberg records a case where a young woman was delivered of a viable child one hundred and seventy-five days (twenty-five weeks) after her last menstruation. In the case[6] of *Clark* v. *Clark* the President of the Divorce Court held that a child born after 174 days of intra-uterine life was able to live and was a legitimate child. In rare cases, children born in the fifth calendar month or even as early as the fourth month may survive for a short time, but they can never be conceived as having reached the period of viability. Richard H. Hunter[7] describes the case of a fœtus of 5 months of intra-uterine life who lived for 18 hours after birth. It was 30 c.m. long and weighed 512 grammes.

1. *Gaceta Medica de Mexico, Mexico City,* 57, *Sep.-Oct.,* 1926, *p.* 583 ; *Jour. Amer. Med. Assoc., Dec.* 24, 1926, *p.* 2038.
2. *Monatschrift f. Geburtshulfe u. Gynakologie, Berlin, Jan.,* 1933, *p.* 137 ; *Jour. Amer. Med. Assoc., March* 4, 1933, *p.* 704.
3. *Brit. Med. Jour., Vol. II,* 1928, *pp.* 878 *and* 1076.
4. *Practitioner, May,* 1933, *p.* 608.
5. *Guy and Ferrier, Forens. Med., Ed. VI, p.* 130.
6. *Lancet, March* 11, 1939, *p.* 593.
7. *Brit. Med. Jour., May* 27, 1933, *p.* 919.

Rodman[1] reports a case where a woman, who had already borne five children, was delivered of a living male infant after her period of gestation which was "rather under nineteen weeks" (one hundred and thirty-three days). The infant lived a year and nine months.

4. **Superfecundation.**—By superfecundation is meant the fertilisation of two or more ova of the same ovulation at two successive acts of coitus. This occurrence in human beings is proved by the fact that the same woman has, sometimes given birth to twins possessing physical peculiarities from which it was inferred that they were the children of fathers of different races. Thus, Dr. Mosley[2] mentions the case of a Negro woman, who brought forth two children at a birth, both of a size, one a Negro, the other a Mulatto. She stated that she suffered the embraces of a white man directly after her black husband had quitted her.

5. **Superfoetation.**—By superfœtation is meant the impregnation of an ovum belonging to a subsequent period of ovulation after the ovum discharged from a previous ovulation has been developing for a month or more. The occurrence of superfœtation is possible, though rare, inasmuch as ovulation may take place especially during the first three or four months of gestation until the decidua vera comes into apposition with the decidua reflexa and the decidual cavity is obliterated. Its occurrence in a bipartite or double uterus is certainly probable. The result of superfœtation would be the birth at the same time of two fœtuses showing different stages of development, or the birth of two fully developed fœtuses at different periods varying from one to three months.

The following case recorded by Tyler Smith[3] conclusively proves the occurrence of superfœtation:—

A young married woman, pregnant for the first time, miscarried at the end of the fifth month, and some hours afterwards a small clot was discharged enclosing a perfectly healthy ovum of about one month. There were no signs of a double uterus in this case. The patient had menstruated regularly during the period that she had been pregnant.

John M. Maury[4] reports that at the post-mortem examination on the body of a coloured woman, aged 35 years, who died from pulmonary tuberculosis, the uterus contained a well-formed fœtus of thirteen or fourteen weeks, and a much smaller embryo of six weeks was found in the left Fallopian tube.

Sussi[5] also reports a case of superfœtation. A primipara, aged 32, gave birth to a full term boy and twenty minutes later to a living female fœtus of about the sixth fœtal month. There was a great difference in the sizes and in weights of the fœtuses, the ratio being 7 : 1 and there was also a considerable difference in the two placentas.

Cases of supposed superfœtation may, however, be explained in other ways. If twins are born together of apparently very unequal development, this may be due simply to one of the twins having failed to obtain an equal share of nutriment during intra-uterine life. If the less developed

1. *Guy and Ferrier, Forens. Med., Ed. VI, p.* 131.
2. *Ibid., p.* 132.
3. *Man. of Obstetr., p.* 172.
4. *Jour. Amer. Med. Assoc., Jan.* 10, 1925, *p.* 139.
5. *Medizinische Klinik, Berlin, Oct.* 11, 1935, *p.* 1934; *Jour. Amer. Med. Assoc., Nov.* 30, 1935, *p.* 1814.

fœtus is not alive, it is almost certain that it is simply a case of blighted ovum retained without decomposition.[1]

6. **Paternity.**—In questions of illegitimacy, the paternity of a child may be determined from the blood grouping test and from the resemblance of its features, colour, voice, manner, etc., to those of the alleged father. Diseases or deformities may be transmitted from parent to offspring, and may serve as an important piece of evidence.

It is an accepted fact that where a woman marries a second time her children may not resemble their father, but the first husband of their mother. Again, children may not resemble their parents at all and, therefore the absence of likeness of features or of transmitted peculiarities does not disprove paternity, nor prove illegitimacy. Moreover, cases of atavism occur in which the child does not resemble its parents, but resembles its grandparents.

1. *Galabin, Man. of Midw., Ed. VI, p.* 266.

CHAPTER XVI

RAPE AND UNNATURAL OFFENCES

RAPE

Definition.—Rape is defined as the unlawful and carnal knowledge by a man of his wife under the age of thirteen years or of any other woman under the age of fourteen years, or above that age against her will, without her consent, with her consent, when her consent has been obtained by putting her in fear of death or of hurt, or with her consent, when the man knows that he is not her husband, and that her consent is given because she believes that he is another person to whom she is or believes herself to be lawfully married. Section 375 of the Indian Penal Code refers to the offence of rape, and section 376 refers to the punishment to be awarded for the offence (Vide Appendix VII).

An indecent assault by a male on a female committed with intent to outrage her modesty constitutes a less serious offence than that of rape and is punishable under section 354 of the Indian Penal Code (*Vide* Appendix VII). What constitutes an outrage on female modesty is not defined anywhere. This will differ according to the country and race to which the woman belongs. To place hands on the shoulder of a woman will be an outrage on the modesty of a Hindu or a Mohamedan woman, but not a European.[1] Where a teacher took indecent liberties with a female student, it was held that he was guilty of assault, though she did not resist.[2] Making a female patient strip naked under the pretence that the accused, a medical man, could not otherwise judge of her illness was held to be an assault.[3]

Consent.—According to the law of India a woman of and above the age of fourteen years is capable of giving consent to an act of sexual intercourse, but the consent must be free and voluntary and given while she is in full possession of her faculties. It should also have been obtained prior to the act. It is no defence to state that the consent was given after the sexual connection.[4]

The ingredients which are essential for proving a charge of rape are the accomplishment of the act by force, resistance and absence of consent if the woman happens to be of or above the age of consent. The consent of the woman is invalid if it is obtained by threat of physical injury or of death or by misrepresentation of facts, or if it is obtained from the woman who, from unsoundness of mind or intoxication, is unable to understand the nature and consequences of the act to which she gives her consent.[5]

---

1. *George Evans,* (1906) *Crim. Applicat. for Revision No.* 58 *of* 1906, *decided on April* 10, 1906. *Per Jenkins, C. J., and Aston, J. (Unrep. Bom.); Ratanlal and Thakore, The Law of Crimes, Ed. XIV, p.* 850.
2. *Peter Rozinski,* (1824) 1 *Mood. Cr. C.* 19; *Ratanlal and Thakore, The Law of Crimes, Ed. XIV, p.* 850.
3. *John Nichol,* (1807) *R. & R.* 131; *Mc. Gavaran,* (1852) 6 *Cox* 64; *Ratanlal and Thakore, The Law of Crimes, Ed. XIV, p.* 850.
4. 1 *Hawk. P. C., p.* 122; *Ratanlal and Thakore, The Law of Crimes, Ed. XIV, p.* 897.
5. Vide *Appendix VII, Section* 90, *I. P. C.*

A husband cannot be charged with rape against his own wife of, and above, the age of thirteen years, even though the act be committed against her will or without her consent. But he has no right to enjoy her person without regard to question of safety to her.[1] A husband can, however, be guilty of abetment of rape by another man on his wife. This was held in the notorious case of Lord Audley who held his wife by force while his butler ravished her.[2]

Under English law (the Criminal Law Amendment Act, 1885), the unlawful and carnal knowledge of any girl under the age of thirteen years is a felony, and the person committing the crime may be kept in penal servitude for life. An attempt at the same is a misdemeanour, and the punishment may be two years' imprisonment with or without hard labour. The unlawful and carnal knowledge of any girl between the ages of thirteen and sixteen years or of any female idiot or imbecile woman or girl is a misdemeanour punishable by imprisonment up to two years with or without hard labour. An attempt at the same is also a misdemeanour, and the same punishment may be awarded.[3] The age of consent is fixed at sixteen years. It is enacted by section 2 of the Criminal Law Amendment Act, 1922, that reasonable cause to believe that a girl was of or above the age of sixteen years shall not be a defence to a charge of rape under section 5 of the Criminal Law Amendment Act, 1885, provided that in the case of a man of twenty-three years of age or under, the presence of reasonable cause to believe that the girl was over the age of sixteen years shall be a valid defence on the first occasion on which he is charged with such an offence.

To constitute the offence of rape it is not necessary that there should be complete penetration of the penis with emission of semen and rupture of the hymen. Partial penetration into the vulva with or without emission of semen, or even an attempt at penetration, is quite sufficient for the purposes of the law. It is, therefore, quite possible to commit legally the offence of rape without producing any injury to the genitals or leaving any seminal stains. In such a case the medical officer should mention the negative facts in his report, but should not give his opinion that no rape had been committed.

English law does not regard an attempt by a woman to compel a young boy to hold sexual intercourse with her as an offence, but French and German laws punish the woman who attempts sexual intercourse with a boy under eleven years of age.

Age of the Male.—The law of England presumes that a boy under fourteen years of age is sexually impotent, and is, therefore, incapable of committing rape but he may be convicted of an indecent assault under the Criminal Law Amendment Act, 1885. The law of India does not presume

1. *Hurree Mohan Mythee,* (1890) 18 *Cal.* 49; *Ratanlal and Thakore, The Law of Crimes, Ed. XIV, p.* 900.

2. *Lord Audley's case,* (1631) 3 *St. Tr.* 401; *Ratanlal and Thakore, The Law of Crimes, Ed. XIV, p.* 900.

3. *Under the law of the Irish Free State it is a felony to have intercourse with a girl under* 15 *years and a misdemeanour to attempt it. It is a misdemeanour to have or to attempt intercourse with a girl above the age of* 15 *and under the age of* 17 *years.*

any such limit of age under which a boy is considered physically incapable of committing rape. In a charge of rape brought against a boy the Court decides the question of his potency from evidence in the case and is guided by sections 82 and 83 of the Indian Penal Code in awarding punishment. A case is recorded by Chevers in which a boy of ten was convicted of rape on a girl, three years old.[1] A case occurred at Poona in July, 1923, where a Chamar boy, aged ten years, was charged with the offence against a European girl, aged seven years. The Cantonment Magistrate found the accused guilty, and sentenced him to two years' rigorous imprisonment, ordering that the accusd be sent to the Dharwar Juvenile Jail.

Old men are known to have committed rape on small girls. A man of 60 years beckoned to a girl of seven or eight years and took her into a small room where he committed rape. She cried but he threatened to kill her with a knife in case she disclosed the secret. He also communicated to her the venereal disease from which he was suffering. He was sentenced to four years' imprisonment by the Magistrate of Amritsar.[2]

Age of the Victim.—No age is safe from rape. Chevers[3] records a case where a wretch was sentenced at Delhi to twenty years' imprisonment for rape committed on a woman of seventy years. However, it is comparatively easy for lusty brutes to commit rape on children, as they are ignorant of the world and are unable to offer resistance. In India, as in other countries, rape on children is common owing to the superstitious belief that gonorrhœa and syphilis are cured by sexual intercourse with virgins. To these may be added the cause of retaliation on the part of parents on account of previous enmity as a motive for rape on children. A case came under my observation at Agra, where a man committed rape on a girl of eight years, the daughter of his mistress, with whom he had had a quarrel. He infected the girl with spyhilis. A case[4] occurred in Banda District, where an old man of nearly 55 years of age ravished a girl of 14 years by way of revenge, because he harboured a grudge against the girl's father and uncle as they treated him as an outcaste and refused to dine with him.

The following table gives the age at which rape was committed in one hundred and thirty-four cases examined by me at Lucknow :—

From 2 to 4 years old	3
From 5 to 9 years old	40
From 10 to 14 years old	68
From 15 to 20 years old	22
30 years old	1
	134

EXAMINATION OF THE VICTIM

The female on whom rape is alleged to have been committed should be allowed to give her own account of the act without any questions being

1. *Med. Juris., Ed. III, p.* 674.
2. *Leader, June* 27, 1929.
3. *Med. Juris., Ed. III, p.* 678.
4. *Leader, April* 10, 1930.

put to her. She should never be examined without her written consent taken in the presence of a witness if she is of and over twelve years of age and is capable of understanding the nature and the implication of the examination, or without the written consent of her parent or guardian if she is a child under twelve years of age or a feeble-minded person.[1] The examination of a female without her consent is regarded in law as an assault. It must be remembered that the Police Court has no power of compelling a woman to submit the private parts of her person to the examination of a medical man. In a case where Gopal and two others[2] were prosecuted for abduction of one Nandkuvar the Sessions Judge of Ahmedabad held that force cannot be used by the Magistrate or his subordinate in the medical examination of the girl.

An attempt at undressing the woman should never be made, but she should be requested to undress herself. The exact time of the examination, and the date and month of the year should be mentioned, and then the examination proper should be commenced in the following order :—

1. **Clothes.**—If the clothes are the same as those worn at the time of the occurrence of rape, they should be carefully examined for the presence of blood or seminal stains, and whether these are on the front or on the back of the garments. Usually the seminal stains are on the front of the clothes and those of the blood are on the back, but no arbitrary rule can be laid down. It should also be noted if the clothes have been torn or soiled with mud. If there are any marks of suspicious stains, the clothes should be preserved with a view to forwarding them to the Chemical Examiner.

2. **Marks of Violence on the Body.**—The body, especially the face, chest, limbs and back, should be examined for marks of violence, such as scratches and bruises, as a result of struggle. If present, they should be carefully described as regards their appearance, extent, situation and probable duration. Such marks are more likely to be found on the bodies of grown-up women who are able to resist, than on the bodies of children who are incapable of offering any resistance. To substantiate false charges, marks of violence are, sometimes, self-inflicted. I saw a young woman of twenty, alleged to have been raped by a man. She had several marks simulating scratches made with a *kankar* on the forearms and the chest, which could be wiped off by rubbing them with a piece of wet cotton-wool.

In addition to these marks the female may experience difficulty in walking and pain in micturition or defæcation.

3. **The Genitals.**—To examine the genitals for the evidence of rape the female should be kept in the lithotomy position in good light, and the thighs should be well separated. In children the separation of the thighs is very painful, and it may, therefore, be necessary to apply cocaine solution to the parts, or to administer an anæsthetic.

During the examination the following points may be noted :—

1. In grown-up females, if the pubic hairs are found matted due to the presence of semen, some two or three should be plucked and examined

1. Vide *Section 90 of the Indian Penal Code, Appendix VII.*
2. *Times of India, October 2, 1924.*

for the presence of spermatozoa, if possible, or they should be preserved to be forwarded to the Chemical Examiner.

2. Recently effused or dried blood may be found upon the genital organs or in the neighbourhood, and in recent cases there may be bleeding from the vagina, which is usually very slight, unless there is some injury to the vagina itself. It should not be forgotten that the bleeding may be due to menstruation, which is possible to be induced by sexual intercourse, or the genitals and clothes may be intentionally soiled with blood to substantiate a false accusation. In his annual report for 1922, the Chemical Analyser of Bombay reports a case in which the *sari* worn by a woman alleged to have been raped was found to contain blood-stains of an avian origin. The Chemical Examiner, Bengal, also describes a case of alleged rape in his annual report for the year 1935 in which the cloth of the victim was covered with several stains of the blood of a ruminant animal with that of a bird (probably a fowl).

3. Bruising and laceration of the external genitals may be present with redness, swelling and inflammation.

4. In nubile virgins the hymen, as a result of complete sexual intercourse, is usually lacerated, having one or more radiate tears, the edges of which are red, swollen and painful, and bleed on touching if examined within a day or two after the deed. These tears heal within five or six days, and after eight to ten days become shrunken and look like small granular tags of tissue. Frequent sexual intercourse and parturition completely destroy the hymen, which is represented by several small granular tags of tissue, called carunculæ hymenealis or myritiformes.

In cases where the hymen is intact and not lacerated, it is necessary to note the distensibility of the vaginal orifice. The possibility of sexual intercourse having taken place without rupturing the hymen may be inferred, if the vaginal orifice is big enough to admit easily the passage of two fingers. In virgins under fourteen years of age the vaginal orifice is so small that it will hardly allow the passage of the little finger through the hymen.

The fourchette and posterior commissure are not usually injured in cases of rape, but they may be torn if the violence used is very great indeed. The amount of injury to the hymen and genital canal depends upon the degree of disproportion between the genital organs of both parties and the violence used on the female.

In small children the hymen, being situated high up in the canal, is not usually ruptured, but may become red and congested along with the inflammation and bruising of the labia, or, if considerable violence is used, there is often laceration of the fourchette and perinæum.

In grown-up married women accustomed to sexual intercourse, marks of violence, such as bruises, scratches, etc., may be found on the external genitals, perinæum, abdomen, thighs, hands and neck.

5. The mucous secretion of the vagina should be obtained by introducing a glass rod or pipette ; an ink pipette used for filling a stylographic pen will do very well. The secretion should then be examined

for the presence of spermatozoa, which is a positive sign of rape in the case of children and grown-up virgins. In grown-up married women it does not necessarily indicate rape, but it proves the occurrence of previous sexual intercourse.

6. **Signs of Infection of Gonorrhoea or Syphilis.**—A muco-purulent or purulent discharge of a greenish-yellow colour from the vagina and soiling the linen may be due to gonnorhœal infection or may arise from local irritative causes, such as uncleanliness, masturbation, threadworms, leucorrhœa or protozoal infection, such as Trichomonas vaginalis, and from diseases which enfeeble the general state of health. It is, therefore, very essential that a thin film from the discharge should be put on two or three glass slides, stained by Gram's method and examined under the high power of a microscope for the presence of gonococci (causative agents of gonorrhœa) which are kidney- or bean-shaped, intravellular, Gram-negative diplococci before a definite opinion is given. In the case of a negative result a decisive opinion must not be given unless the films from the discharge are examined on at least three successive occasions with intervals of one week, for in the later stages of the disease the gonococci may be found only with great difficulty or may not be found at all. In the case of adult females suffering especially from subacute and chronic gonorrhœa it is advisable to examine the discharge from the cervical canal, as large varieties of microorganisms other than gonococci are generally found in the vaginal canal, and these are apt to confuse an inexperienced medical practitioner, although they are not morphologically similar to gonococci. A non-pathogenic Gram-negative diplococcus which closely resembles the gonococcus is also frequently found in the genitals of female adults. It should, however, be remembered that these microorganisms are generally absent in the genitals of female children.

Owing to its peculiar situation and nearness to the vagina, the urethral canal in the female is likely to be infected early with gonorrhœa ; hence the microscopic examination of the urethral discharge will, sometimes, help the diagnosis.

In a purulent discharge from the male urethra the presence of kidney- or bean-shaped intracellular, Gram-negative diplococci in pairs with their concave borders facing one another is ordinarily taken as sufficient for the purpose of practical diagnosis. The Micrococcus catarrhalis which closely resembles the Gonococcus in morphology and staining reactions may occur in the urethra and cause confusion in the diagnosis. Hence, in a doubtful case it is necessary to resort to cultures on suitable media. The Micrococcus catarrhalis grows readily on ordinary media, where it forms large, white, dry colonies with irregular edges and elevated centres. Whereas the Gonococcus does not grow on ordinary media but grows readily on chocolate blood agar, where it forms minute, grayish and transluscent colonies.

The Meningococcus (Micrococcus meningitidis intracellularis) is also a Gram-negative diplococcus and morphologically resembles the Gonococcus, but it is usually present in the cerebro-spinal fluid and in the nasal discharge but is not found ordinarily in the urethral discharge or in the urine. It grows on Loffler's blood serum, where it forms colonies which are round, colourless or hazy, flat, shining and viscid looking.

The existence of a venereal disease in the female is not positive evidence of sexual connection. Gonnorrhœal infection of the genital tract particularly in young girls and infants may be conveyed through infected hands or other articles. Outbreaks of gonorrhœa in children in schools, boarding houses or hospitals have often been traced to the common use of the infected sponges, towels, bath tubs, etc. Syphilis may also be transmitted by means other than sexual intercourse, *e.g.*, kissing. In all such cases it is absolutely necessary to examine the accused for the presence of either of these diseases, for the finding of gonorrhœa or syphilis in both parties is strong corroborative evidence of sexual intercourse.

The period of incubation of gonorrhœa varies usually from two to eight days, although it may be as short as twenty-four hours and as long as two weeks. I have seen a few cases in which gonorrhœa appeared within twenty-four hours after the infection. The period of incubation of syphilis varies from two to eight weeks after inoculation, the average period being twenty-five days. If the accused is suffering from a venereal disease, and if the story of rape is true, the accuser (victim) is likely to suffer from the same disease within its period of incubation. But it must be borne in mind that the infection is not always communicated by sexual intercourse with one suffering from a venereal disease. In cases of rape on children and virgins, however, there is a greater probability of inoculation, as the delicate mucous membrane of their genitals is very susceptible to infection, and the hymen and other parts are usually abraded or lacerated.

ACCIDENTS FOLLOWING RAPE

Convulsions, epileptic fits, and mental derangements have been known to follow rape. Death may occur as a result of rape from shock due to fright and mental emotion, or from syncope due to excessive and severe injuries to the genitals and perinæum, especially among children. These injuries, if not immediately fatal, may produce sloughing, peritonitis and ultimately death. In some cases death has resulted from suffocation caused by covering the mouth and nostrils with the hand or by thrusting a piece of cloth down the throat to prevent the girl from crying for help. It is, therefore, necessary to examine the mouth for the presence of a foreign body when the body of a female who is alleged to have died from rape, is brought for post-mortem examination. Sometimes, a female is first raped, and then murdered to destroy the only reliable witness to the offence. Occasionally men with perverted passions, who are known as *sadists*,[1] are gratified by murdering a female without violating her even though very serious injuries caused by the hand may be found on the genitals. Such a murder is known as *lust murder*. In such a case the criminal may be impelled to an act of *anthropophagy* or *cannibalism* when the body is opened and the genitals or other organs are torn out and the flesh is eaten. Fortunately such a case has not been recorded in India.

Rarely, rape has been committed on a dead body (Necrophilia).[2] In such a case it would be difficult to obtain physical evidence, if the crime was not detected at the time. In the case of a virgin it might be possible

1. *Thoinot, Medico-Legal Aspect of Moral Offences, Eng. Trans., p.* 420.
2. *Ibid., p.* 448.

to find tears in the hymen, vagina and fourchette and scratches perhaps on the vulva. The presence of spermatozoa about the genitals would furnish corroborative evidence, but the possibility of a connection before death should not be lost sight of.

EXAMINATION OF THE ACCUSED

Before examining the accused his written consent should be taken after it is explained to him that the result of the medical examination may go against him. While writing the report the following points should be carefully noted :—

1. The exact time of the examination with the date and month of the year.

2. The age, development of the genital organs and physical powers of the accused as compared with those of the victim (accuser).

3. The presence of tears on the clothes or loss of any portion or buttons from them indicating the evidence of a struggle.

4. The presence of mud, blood or seminal stains on the clothes or on the body. The presence of blood-stains is an important piece of evidence, especially if the alleged victim be a child or virgin who has sustained some injury giving rise to hæmorrhage. The absence of stains does not negative the charge of rape as, although there may have been considerable loss of blood from the genitals of the victim, stains would not necessarily be found on the body or clothes of the ravisher, especially if he had had an opportunity of washing after the act. It is, therefore, necessary that the police should never allow the accused to go to the bath room alone under any pretext until the medical examination has been finished, if he is arrested soon after the crime.

The presence of seminal stains only on the body or clothes does not necessarily prove rape. It merely indicates a recent emission.

5. The presence of the marks of a struggle, such as bruises, scratches, and teeth bites on the body, especially on the face, hands, thighs and genitals.

6. The clotting of pubic hairs due to the emission of semen.

7. The presence of hairs similar to those of the female alleged to have been raped. For instance, the hair of the head may be found on the body of the accused, or the pubic hair of the victim may be found on or about the prepuce.

8. In addition to scratches or lacerations on the penis caused by the finger nails of the victim during a struggle, an abrasion or a laceration may be discovered on the prepuce or glans penis, but more often on the frænum, due to the forcible introduction of the organ into the narrow vagina of a virgin, especially of a child, but it is not necessary that there should always be marks of injuries on the penis in such cases. I have seen cases in which there was no injury to the penis of the accused, although there were lacerations of the hymen, posterior commissure, perinæum and even the vaginal walls of the complainant (victim).

9. If the accused is not circumcised, the existence of smegma round the corona glandis is proof against penetration, since it is rubbed off during the act of sexual intercourse. The smegma accumulates if no bath is taken for twenty-four hours.

10. The presence of a gonorrhœal discharge or of a syphilitic chancre. In such cases the female (victim) should be examined for the existence of either of these venereal diseases with due regard to their incubation period.

11. Lastly, the locality where the offence is alleged to have been committed should be examined, as it may reveal valuable clues in the shape of blood-stains, pieces of torn clothing, marks of the body on the ground, or the crushed and trampled condition of the grass in the vicinity.

MEDICO-LEGAL QUESTIONS

The following are the conrtoversial questions, which are likely to arise in a Court of law in cases relating to rape :—

1. **Can a healthy adult female be violated against her will ?**—Under ordinary circumstances it is not possible for a single man to hold sexual intercourse with a healthy adult female in full possession of her senses against her will, unless she is taken unawares, thrown accidentally on the ground and placed in such a position as to render her completely helpless, or unless she swoons away from fright or exhaustion after long resistance. The act may be accomplished if more than one man are concerned in the crime, or if the woman is too feeble to resist. In giving a definite opinion, the relative strength of the parties should be taken into consideration. However, in the majority of rape cases on adult women the charge is made with the object of blackmail, or the act is done with the consent of the woman, but when discovered, to get herself out of the trouble, she does not scruple to accuse the man of rape. If the complaint in these cases is made a day or two after the act, the case is probably one of concoction. It is also necessary to note the previous character of the female, and her relations with the accused.

2. **Can a woman be violated during natural sleep ?**—It is impossible for complete sexual intercourse to be accomplished on a nubile virgin during her natural sleep without her knowledge, as the pain caused by the first act of coitus would certainly awaken her from sleep. It is, however, possible, though indeed rare, for vulval penetration to occur in a virgin without awaking her from sleep. It is also possible, though highly improbable, for a woman to allow coitus during profound sleep without her being conscious of it, if the genital parts are large and accustomed to the intromission of the penis. Guy[1] mentions the case of a poor woman who complained of her sleep being so heavy that she was with difficulty roused and, by way of illustration, stated that her husband had assured her that he had frequently had connection with her during sleep.

3. **Can a woman be raped during unconsciousness ?**—There is no doubt that rape can be committed on a woman during syncope, coma or mesmeric or hypnotic trance, or during unconsciousness produced by the

1. *Guy and Ferrier, Forens. Med., Ed. VI, p.* 63.

administration of narcotic and intoxicating or anæsthetic drugs. In connection with anæsthesia it must be remembered that it is impossible to anæsthetise against his or her will a person who is awake. It is also impossible to anæsthetise a sleeping person without disturbance so as to substitute artificial sleep for natural sleep. Again, a woman, during the stage of anæsthesia, gets hallucinations that she has been raped, and insists on the belief after the effects of the drug have passed away, so that she brings an accusation against her medical attendant. As a precautionary measure against such an emergency, the medical practitioner should never administer an anæsthetic to a female without the presence of another person, preferably her near relation.

4. **Can a woman become pregnant from an act of rape ?**—A woman can certainly become pregnant from an act of rape, even if she was quite unconscious during the act. Formerly there was an erroneous belief that impregnation would not occur if the intercourse was not voluntary and followed by a pleasurable feeling, and that it would not follow the first act of coitus. But this belief is absolutely wrong, as it has been proved physiologically and experimentally that conception will occur in a healthy woman if the living spermatozoa can be deposited into the vaginal canal even by a glass syringe, and if they can meet in the uterus or Fallopian tube the ova ready to be fertilized.

ILLUSTRATIVE CASES

Rape committed by Misrepresentation of Facts.—1. A girl of 14 years consulted a physician for suppressed menstruation. He had connection with her stating that it was part of the treatment. She did not resist, being ignorant of the act owing to her youth. The physician was convicted of rape.—*R.* v. *Carr,* 4 Cox C. C. 223.

2. An epileptic girl, 19 years old, consulted a quack-doctor for her ailment. He told her that there were some internal adhesions which must be broken down by a surgical operation. The mother, who was present, gave consent to the operation, not understanding his motive, and allowed the doctor to take her daughter to a private room. Here she submitted to sexual intercourse believing it to be a part of the treatment.—*R.* v. *Flattery, L. R. Queen's Bench Div.,* 410.

3. One Must Ram, a *bairagi,* falsely personated himself as Basorey, the husband of one Mt. Khunia, who had left his home some twelve years ago, and whose whereabouts were not known to any of his relatives since then. The *bairagi* deceitfully made Mt. Khunia believe that he was her real husband and had returned from a long journey extending over a period of twelve years. He lived with her for some time, but his imposture was found out when he was persuaded to visit Lalua, the brother of the woman's real husband, who was a police *chaukidar.* He was subsequently charged with having committed rape on Mt. Khunia by falsely personating himself as her husband, and was sentenced by the Additional Sessions Judge at Banda under Section 376, Indian Penal Code, to four years' rigorous imprisonment and fifteen stripes.—*The Leader, Jan.* 20, 1928, *p.* 6.

4. By the representation that she was submitting to a spiritual obligation for the good of her soul, a shoemaker, 48 years old, persuaded a young woman of 25 years and of weak intellect to yield to sexual intercourse. He was sentenced to five years' penal servitude for this grave offence.—*News of the World, December* 12, 1928.

Injury to the Genitals of Nubile Virgins during Coitus.—1. A woman, 23 years old, got laceration of the posterior vaginal fornix during coitus in a sitting posture. The vagina was torn from the back of the cervix uteri for one inch and a half, and an artery being opened the wound bled freely. The peritoneum was not injured.—*Loof, Ann. de Gynec. et d' Obstet., March,* 1898 ; *Glaister, Med. Juris. and Toxic., Ed. V, p.* 532.

2. A newly married woman felt a sharp pain during the first act of coitus. This was immediately followed by copious bleeding owing to a tear in the vagina, which, commencing at its orifice, extended upwards to the left of the median line to Douglas' pouch, and then crossing it passed to the right side of the vagina.—*Mylott, Brit. Med. Jour., Vol. II,* 1899, *p.* 760.

Rape on Children.—Rape was committed on an infant of seven months. There were bruising and bleeding of the vulva. The hymen was intact. The vagina was not lacerated. Seminal stains were found on the person of the child.—*R. v. Harris, Bristol, Lent Assizes,* 1873.

The following six cases are picked up at random from my note-book :—

1. On the 25th June, 1920, a girl, 6 years old, was raped by a male. On examining her on the 28th June, the hymen, posterior commissure and perinæum were found lacerated. The accused was also examined at the same time. He had a laceration of the frænum of his penis.

2. A girl, 10 years old, alleged to have been raped, was examined on the 30th September, 1920. The hymen and perinæum were lacerated. The accused who was examined at the same time had no mark of injury on his genital organ.

3. A girl, 8 years old, was examined on the 22nd February, 1921, as it was reported that she had been raped by a young man. The hymen was found intact. There was a laceration of the fourchette with redness of the right labium minus.

4. On the 28th April, 1930, I examined a girl, 5 years old, who was alleged to have been raped on the previous day by a boy, 16 years old. The labia majora were swollen and stained with blood. The left labium minus was red, and there was a laceration along the whole length of the right labium minus ; this laceration was continuous downwards with a laceration of the posterior commissure. The hymen was red and congested. The boy had redness and swelling over the lower part of the urethral opening, and had an abrasion, 1/6" by 1/6", on the inner side of the prepuce (foreskin) near the corona glandis on the right side of the dorsum of the penis.

5. Musammat Sukhni, aged 12 years, was examined by me on November 29, 1932, twenty-four hours after she had been raped by a male, aged 35 years. Her labia minora were red and inflamed, her hymen was torn on the sides and on the posterior part, and the posterior wall of the vagina as also the posterior commissure were lacerated, each to an extent of $\frac{1}{2}" \times \frac{1}{2}"$. The accused had no mark of injury to his genital organ.

6. On December 31, 1932, a girl, 12 years old, of P. S. Alambagh, was brought to me with a police report that a rape had been committed upon her by her husband. She had almost circular marks of teeth-bites over her cheeks, breasts and the back of the right forearm, and bruises over the front of both the thighs. The labia majora and minora were red and swollen. The hymen was lacerated in the posterior part and the posterior wall of the vagina was lacerated to an extent of $\frac{3}{4}" \times \frac{1}{4}"$.

False Charge of Rape on a Child.—One Mt. Thakurdevi, aged 8 years, was brought to me as her father complained that rape had been committed on her. There was no mark of injury to the private parts. The hymen was intact. There was a slight redness of the labia minora, which was probably due to irritation from dirt present on the vulva.

Rape with Gonorrhoeal Infection.—1. In July, 1920, one Ghulam Husein was charged with having committed rape on Chhidami, a Brahmin girl of five years. On examination there were no marks of injury to the genitals of the girl. Her hymen was intact. The labia minora were red and inflamed. There was a purulent discharge from the vaginal orifice, which was found to be gonorrhoeal. The accused was found to be suffering from gonorrhoea. He was convicted and sentenced.

2. In February, 1923, Jhuman, a Mahomedan cook, was charged with having committed rape on Ruth Violet, a Christian girl of six years. The accused was found suffering from gleet, which, on examination, showed a few gonococci. On examining

the girl, the vulva, especially the lower part, was found red and swollen, and covered with a thick purulent discharge emanating from the vaginal orifice. On microscopic examination the discharge showed a large number of gonococci. The labia minora were red, inflamed and painful to touch. The hymen was intact. The accused was found guilty, and sentenced to rigorous imprisonment.

3. In May, 1927, one Din Mohammad was charged with having committed rape on one Mt. Kalpi, aged about 9 or 10 years. On examination of the girl I found that the hymen was intact, but the labia majora were swollen and the labia minora were red, tender and excoriated. There was a thick, whitish purulent discharge from the vaginal canal which, on microscopic examination, was found to be due to gonorrhoea. The accused was also examined by me and was found to have a chronic gonorrhoeal discharge from the urethra.

4. In December, 1932, one Mt. Jagdevi, 8 years old, was brought to King George's Hospital with a police report that she was alleged to have been raped about 2 days ago. On examination there were no marks of injury to her private parts, but there was a whitish discharge in the vaginal canal; it showed the presence of spermatozoa and gonococci under the microscope. The accused was a boy of 18 years, who had a urethral discharge, which also showed, under the microscope, a few gonococci as a result of chronic gonorrhoea.

In the June issue of the *Indian Medical Gazette*, 1902, page 231, Powell relates the following cases in which infection was not communicated by illicit intercourse with a person suffering from the venereal disease:—

1. Four men had connection with the same prostitute who had a copious gonorrhoeal discharge. Only one became infected.

2. Seven troopers had connection with a woman, who had gonorrhoea. Only two were infected.

3. A woman was suffering from mucous patches of the vulva. A gentleman, who had been "keeping" her for six months, was greatly alarmed when he discovered her condition. But he never developed any sore or symptoms of disease.

Presence of Smegma as Negativing Rape.—1. In July, 1921, Mt. Ramdevi, aged 15 years, made a report at the police station of Malihabad in the district of Lucknow that three young men, *viz.*, Pachu, Debi, and Jodha had committed rape on her. They were arrested and sent immediately to me for examination. None of them had any mark of injury on their genitals or anywhere else on their bodies. The first two had smegma on the glans penis covered by the foreskin; this proved that they could not have had sexual intercourse at least during the last twenty-four hours. The girl was also examined, and found to have been used to sexual intercourse, inasmuch as her hymen had old lacerations. She had no mark of injury to her private parts or to any other part of the body. The men were released.

2. On the 23rd February, 1923, a man complained at the police station of Mandiaon in the district of Lucknow that one Dhani had committed rape on his daughter. He was immediately arrested and sent to me for medical examination. I found a uniform layer of smegma covering the glans penis, and gave an opinion that he could not have had sexual intercourse during the last twenty-four hours. The man was released.

Death following Rape.—1. In the case of *Queen-Empress* v. *Hurree Mohan Mythee* the accused, a fully developed adult man, was charged with causing the death of his wife, a girl of 11 years and three months. According to medical evidence, the death was caused by hæmorrhage from a laceration in the upper part of the vagina to the right of the neck of the uterus, measuring one inch and a half long and one inch broad. The rupture was caused by the prisoner having sexual intercourse with the girl, who had not attained puberty. The Court held that in such a case, where the girl is a wife and above the age of ten years (age of consent at that time), and when therefore the law of rape does not apply, it by no means follows that the law regards the wife as a thing made over to be the absolute property of her husband, or as a person outside the protection of the criminal law. The prisoner was convicted

under Section 338 of the Indian Penal Code of the offence of having caused grievous hurt by an act so rashly and negligently done as to endanger life.—*Ind. Law Reports, Calcutta, Vol. XVIII*, 1890, *p.* 49.

2. Johir Sheikh, a well-built Mahomedan, about 35 years old, had forcible connection with his girl wife, aged about ten years, rupturing her genitals, which caused her death from hæmorrhage in about twenty-four hours. There was blood about the genitals and a clot of blood protruded from the vagina, which on extraction, was found to have completely filled the cavity. The hymen was torn, the fourchette ruptured, and the anterior part of the perinæum lacerated for a distance of half to three-quarters of an inch in length. Extending forwards from this the mucous membrane of the posterior vaginal wall was torn for a short distance. On the right lateral wall of the vagina there was a laceration, one inch long; below, near the vaginal orifice it was about quarter of an inch broad and tapered to a point above near the uterus. On the left lateral wall in a corresponding situation was a laceration, one inch long by one-fourth inch broad, somewhat spherical in shape. These lacerations extended through the mucous lining, and partly, but not completely, through the muscular tissues. There was an effusion of blood in these situations beneath the serous coat, which was, however, uninjured. The vagina was dilated; it had been distended by the blood clot. The husband was prosecuted and convicted under Section 376, I. P. C.—*Calvert, Ind. Med. Gaz., June*, 1895, *p.* 221.

Murder after Rape.—1. In January, 1923, Mt. Idia, aged 18 years, of the Meerut District had been raped first, and then done to death by throttling in a sugar-cane field. Medical evidence showed that her hymen was ruptured and there was bleeding from the vagina. The girl's *pyjama* and *kurta* were torn. A number of scratches were found on the accused's person apparently caused by finger nails. This indicated that the girl was ravished after a desperate struggle in which great violence was used. The accused was a tall man of very powerful physique, and 25 years old. The Chemical Examiner, U. P., detected blood and seminal stains on the *dhoti* of the accused.—*All. High Crt., Cr. App. No.* 423 *of* 1923.

2. On the 19th July, 1927, one Mt. Matri Pasin, a girl of 13 or 14 years of age, went to Thakurainganj to give some clothing to her *dhobi*. Her way lay across a *nala*. When she got to this *nala* on her way back, she was seized by one Sukhlal Teli, who had been cutting grass there, and was violated by him. When Sukhlal released her, she said that she would inform her cousin about his conduct. Thereupon he seized her and cut her repeatedly with his *khurpi* till she died.—*K. E.* v. *Sukhlal, Oudh Chief Court Crim. App. No.* 462 *of* 1927.

3. On the 16th February, 1931, one Thanna Lodha of Afzalpur, District Etah, was convicted of the offence of murdering one Musammat Katori, a *dhobi* girl, 14 years old, by throttling after he had committed rape on her. As a result of rape there was a slight bruise mark on the posterior part of the vaginal orifice at the site of the old lacerated hymen. The accused was examined soon after the occurrence, when it was noticed that he had an abrasion of the size of a *moong* on the groove behind the glans penis $\frac{1}{4}$" to the left of the frænum, and an abrasion, 1" long, on the inner side of the left forearm 2" above the wrist.—*Allahabad High Court, Crim. Appeal No.* 531 *of* 1931.

Rape on a Dead Body.—1. A man bribed a watchman to gain entrance to the death-chamber of a girl of sixteen belonging to a French family of social position. He was caught in his night dress by the mother springing from the bed where the body lay. It was a first thought that his object was robbery, but his real intentions were soon laid bare. It was found that the criminal had violated a number of bodies previously, and he was sentenced to imprisonment for life.—*De Boismont, Gazette Medicale, July* 21, 1859; *Witthaus and Becker, Med. Juris., Forens. Med. and Toxic., Vol. II, p.* 732.

2. In the case of *King-Emperor* v. *Bharat Sing*, a Lodha, aged 18 years, the accused admitted in his confession before a Magistrate that he committed sexual intercourse with one Musammat Ramdevi, aged 18, with her consent, but after the act she began to upbraid him in a loud voice that she would be dishonoured if she conceived and that she would defame him when she went back to the village. Fearing that she would certainly go to the village and defame him, he was very much enraged,

threw her down on the ground and killed her by giving three cuts with a *khurpi* on her neck. He had had sexual intercourse again with the dead body, and then dragging the body threw it in a *laha* field.—*Allah. High Court, Crim. Appeal No.* 519 *of* 1933.

Rape on Adult Women.—1. In the District of Agra five men, seeing a young married woman going alone on a road away from habitation, followed her and accidentally seizing her round the body threw her on the ground, and flinging her dress over her violated her. During the struggle she received some scratches and bruises on the body but she was overpowered. All of them were arrested and convicted under Section 376, I. P. C.

2. On the 29th October, 1922, Mt. Brij Rani, a married woman of 18 years, was carrying a bundle of hay on her head from one threshing floor to another, when Nanhe Sing, a strong powerful man, rushing up from behind, pulled the bundle off her head, and seizing her by the arm flung her down on the ground. He then pulled up her clothes and putting his hand on the mouth to prevent her from crying ravished her until he had satisfied his lust. Her stifled cries, however, brought some people to the place, and consequently the man ran away. The woman was examined by me on the following day. There were no marks of injury to the genitals. The hymen was lacerated, and showed carunculæ myrtiformes. She had been used to sexual intercourse. She had a linear scratch one inch and a half long, across the left cheek caused probably by a finger nail. The accused was arrested on the fourth day of the occurrence and was convicted and sentenced by the Additional Sessions Judge of Lucknow to three years' rigorous imprisonment.

False Accusation of Rape.—1. On the 12th February, 1923, Mt. Lekhraji, a robust *kahar* widow of thirty years, reported at the City Magistrate's Court of Lucknow, that on the previous evening she went in an *ekka* from Aminabad, and that near the Imperial Bank building Mehtab Ali, the *ekka* driver, stopping the *ekka* lifted her up bodily, put her on the ground in the compound and ravished her against her will and consent. She had no marks of injury on her body nor were there any on the person of the accused, who admitted that he held sexual intercourse with her consent, but that she brought a complaint against him as he refused to pay her more than the sum equivalent to his fare for the *ekka*. The Magistrate being convinced of the false accusation discharged the accused.

2. In July, 1923, a deaf and dumb woman was arrested by the police for having stolen some cloth from a cloth-merchant's shop. When she was taken to the City Magistrate, Lucknow, she indicated by gestures that she had been raped by the merchant, and the *dhoti* which she was wearing had been stained with blood. The woman was dressed like a man in a *dhoti* and *kurta*, and had cropped hair on her head. She was a strong woman of twenty-five years. There were no signs of injury to the genitals or any other part of the body. The hymen was absent and represented by carunculæ myrtiformes. The vaginal canal was patulous and capacious. She had a menstrual flow which apparently had stained her *dhoti*. It was afterwards ascertained that she was a regular prostitute, and was convicted for having committed theft.

3. On the 14th July, 1923, Mt. Dularia, 16 years old, of the Malihabad Police Station complained that Bajraj Sing and Mahesh Sing went to her house on the night of the 12th July, lifted her up from her *charpoy*, on which she was sleeping, carried her to a groove of trees and flung her down on the ground. Bajraj Sing caught her by the hands, and Mahesh Sing, stuffing her mouth with sand to prevent her from crying for help, violated her. On examination I found slight redness of the left labium minus and a small tear on the left side of the hymen which was otherwise intact. From evidence at the trial the jury was unanimously of opinion that the accused Mahesh Sing did have sexual intercourse with the complainant, but he did so with her consent. Accepting the verdict of the jury the Sessions Judge of Lucknow found the accused not guilty of the offence charged and acquitted them.

Rape During Natural Sleep.—A maid-servant became pregnant and denied being conscious of any act of sexual intercourse. Suspicion fell upon an ostler in the establishment, who acknowledged his belief that he had impregnated her having found her in a deep sleep (due to fatigue, after long continued exertion and loss of sleep for two or three nights in succession). He stated that he had connection with

her, and as far as he knew, without her knowledge, as she evinced no consciousness of the act at the time, nor recollection of it subsequently.—*Ogston, Med. Juris., p.* 121; *Collis Barry, Leg. Med., Vol. II, Ed. II, p.* 97.

Rape During Unconsciousness.—A girl, 22 years old, had for years laboured under hysterico-epileptic convulsions followed by a state of unconsciousness lasting from one to six or seven hours. One evening a labourer found her lying on a sofa in this condition. He was aware of her liability to these attacks and after being convinced of her perfect unconsciousness by tickling her nose with a straw and passing a burning lamp beneath her nose, he drew her from the sofa on to a stool and violated her, a companion looking on from the adjoining room. Speedily awaking, the girl felt pain and dampness about her genitals, and saw the labourer standing before her with his breeches unbuttoned, so that she had no doubt that she had been violated. The accused did not deny having had connection with the girl, but denied her unconsciousness and asserted that she was compliant. The accused was convicted by the jury Court and sentenced to three years' penal servitude.—*Casper, Forensic Med., Vol. III, (Eng. Trans.), p.* 307.

Rape During Mesmerism.—A girl, aged 18, visited a therapeutic magnetizer daily for some days. Four and a half months afterwards she found herself pregnant and lodged a complaint against the magnetizer. Medical evidence proved that her pregnancy dated back to the time of her visits and that it was possible to hold sexual intercourse with a young woman during magnetic sleep without making her conscious of the act.—*Gazette Medicale de Paris; Edin. Monthly Jour., Dec.,* 1860, *p.* 566.

Rape During Anaesthesia.—1. A dentist was convicted of the offence of rape upon a woman to whom he had administered ether by inhalation. The prosecutrix was not perfectly unconscious, but she was rendered wholly unable to offer any resistance.—*Med. Gaz., Vol.* 40, *p.* 865.

2. A young woman brought a charge of rape against a dentist. She stated that under ether given her for the extraction of a tooth, she felt the accused "entering her person", but was unable to cry out or resist. After this she again inhaled the ether for the extraction of the tooth and finally left making an appointment for another day. In the evening she related the incident of alleged rape to her friends. The defendant was tried and convicted. The woman was never examined by a medical man.—*Reese, Med. Juris. and Toxic.,* 1891, *p.* 559.

False Accusation of Rape During Anaesthesia.—A married lady, to whom a dentist administered chloroform, afterwards accused him of violating her whilst under the influence of the anæsthetic. Her husband who was present during the time she was unconscious, testified that his wife was under the strangest impression that she had been violated.—*Dixonmann, Forensic Med. and Toxic., Ed. VI, p.* 80.

UNNATURAL OFFENCES

Section 377 of the Indian Penal Code treats of offences relating to carnal intercourse against the order of nature with any man, woman or animal (*Vide* Appendix VII). Penetration is sufficient to constitute the carnal intercourse necessary to the offences which are punishable with transportation for life, or with imprisonment of either description for a term which may extend to ten years and also with fine. These offences may be classified as sodomy, tribadism and bestiality.

SODOMY

This is also called buggery, and means anal intercourse between man and man or between man and woman. It is termed pæderstia, when the passive agent is a boy (catamite). In order that the offence of sodomy be made punishable under Section 377 I. P. C., it is necessary that penetration, however little, should be proved strictly. Similarly an attempt to

commit this offence is punishable under Section 511, I. P. C., only when the attempt was made to thrust the male organ into the anus of the passive agent. A mere preparation for the operation should not necessarily be construed as an attempt.[1]

Buccal Coitus or *Coitus per os* (the sin of Gomorrah) falls within the provision of, and is punishable under, section 377, I.P.C. In a case[2] in which one Khanu was found guilty under section 377, I. P. C. of having committed the sin of Gomorrah (*Coitus per os*) with a certain little child, the innocent accomplice of his abomination, Kennedy, J. C., observed that " there is no intercourse unless the visiting member is enveloped at least partially by the visited organism, for intercourse connotes reciprocity. Looking at the question in this way it would seem that the sin of Gomorrah is no less carnal intercourse than the sin of Sodom."

Sodomy prevails all over the world, and is, sometimes, practised between two men who alternately act as active and passive agents. In India there is a particular community of *Hijras* who prostitute themselves as passive agents. They commonly dress as women and have their genital organs cut off usually in boyhood.

In a few cases that come up before the Court for trial, the active agent is usually a grown-up male, and the passive agent, a boy and occasionally a girl or a woman. Two cases of unnatural connection with a woman were brought to me by the police in 1932. In one case the husband had committed unnatural connection with his wife of 13 or 14 years of age. Her hymen was found intact, but there was a tear, $\frac{1}{4}$"$\times$1/6", obliquely along the posterior part of the anus to the left of the middle line and external to the sphincter ani. In the other case a woman of about 16 to 18 years complained that her husband was having an unnatural intercourse with her. On examination I did not find any injury on or about the anus. The sphincter was quite normal in its tone ; her hymen had old tears.

Both active and passive agents are guilty of the offence in the eye of the law if the act has been committed with consent. However, according to English law if one of the parties is under fourteen years of age, he is not held responsible for the offence. In Indian law there is no such fixed limit, but sections 82 and 83 of the Indian Penal Code, which deal with age in relation to responsibility for offences in general are also applicable for this offence.

For the investigation of this offence the examination of the passive and the active agent is necessary as in the case of rape. It may also be necessary to inquire if the active agent had obtained the consent of the passive agent for this purpose by means of physical force or fraud, or if the active agent, by reason of age or disease, was physically unfit to commit the offence. A grown-up passive agent may persuade a young boy to act as an active agent to practise the vice on him, but such instances are very rare indeed. I have seen only one case in which a passive agent

1. *Sind J. C.'s Court, Crimin. Appeal No.* 122 *of* 1934 ; 36, *Cr. Law Jour.,* 1935, *p.* 718.
2. *Sind Judicial Commissioner's Court Crim. App. No.* 15 *of* 1924 ; *Criminal Law Jour., July,* 1925, *p.* 945.

of 45 to 50 years of age was prosecuted for having persuaded a boy of 16 years to commit an unnatural connection with him.

In false accusations I have often heard a story that the accused was sleeping in the same bed with the victim, and he committed the unnatural offence on the latter while he was asleep. It should be borne in mind that it is not possible for an adult male to accomplish the act on a boy during sleep without awaking him or on another healthy male against his will.

EXAMINATION OF THE PASSIVE AGENT

As in rape, consent must be obtained before commencing the medical examination. The following signs may be discovered if the boy (passive agent) is not accustomed to sodomy : —

1. Abrasions on the skin near the anus with pain on walking, as well as during examination. These injuries are extensive and well-defined in cases where there is great disproportion in size between the anal orifice of the victim and the virile member of the accused. Hence lesions will be most marked in children, while they may be almost absent in adults, when there is no resistance to the anal coitus. These injuries, if slight, heal very rapidly in two or three days. In most of the cases brought before me I have seen superficial abrasions, varying from 1/6" to 1"×1/6" to $\frac{1}{4}$", external to the sphincter ani. In some cases there may be bruising of the parts round about the anus, and the abrasions may extend into the anus beyond its sphincter.

2. Owing to the strong contraction of the sphincter ani, the penis rarely penetrates beyond an inch, and consequently the laceration produced on the mucous membrane within the anus with more or less effusion of blood is usually triangular in nature, having its base at the anus and the sides extending horizontally inwards into the rectum. I have found lacerations internal to the sphincter ani in several cases, but a typical triangular wound only in a few cases.

It is difficult to differentiate between lacerations caused by the act of sodomy and those caused by the introduction of a blunt weapon into the rectum, but Dr. J. W. Johnstone mentions that lacerations produced during sodomy are situated at the left superior or right inferior angle, and those caused by a blunt weapon are generally found on the upper and lower surfaces, and are of a notchy, patchy character.[1] In some cases no signs would be perceptible if very little force was used.

3. Blood may be found around the anus, on the perinæum or thighs, and also on the clothes.

4. Semen may be found in or at the anus, on the perinæum or on the garments of the boy too young to have seminal emissions.

In his annual report of 1923, the Chemical Examiner of the United Provinces of Agra and Oudh reports a case charged under section 377 of the Indian Penal Code from Allahabad, in which spermatozoa were detected on the trousers of a boy, aged 2 years.

1. *Chevers, Med. Juris., pp.* 708-09.

5. Signs of struggle, such as bruises, scratches, etc., on his person, if he is a grown-up boy, and if he is not a consenting party.

6. Prolapse of the anus.

7. Gonorrhœal discharge, or the presence of a syphilitic chancre.

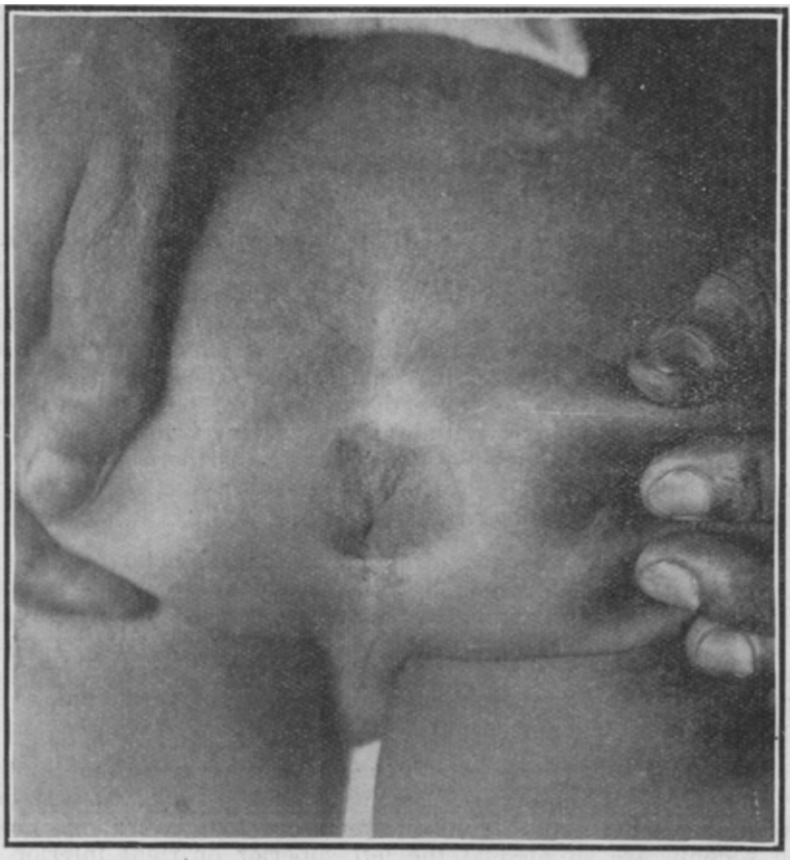


Fig. 111.—Sodomy on a boy of 6 years. The anus shows bruising round about its margins and an abrasion on its posterior part to the right of the middle line.

8. The presence of fæcal matter around the anus is a corroborative sign.

As in rape a passive agent is, sometimes, murdered after the act of sodomy.

On the 29th of January, 1911, a post-mortem examination was held by me on the body of a Hindu boy, twelve years old, residing at Tajganj in Agra, when it was found that the boy had a laceration in the anus and death was due to the effects of irritant poisoning (arsenic). He was either poisoned after the commission of sodomy, or being mortified with shame, committed suicide by taking the poison after the act,

EXAMINATION OF THE HABITUAL PASSIVE AGENT

The signs usually met with in a passive agent habituated to the act of sodomy are as follows :—

1. The shaving of the anal hair but not necessarily the pubic hair.

In a murder case that occurred in Lucknow on the 20th Dceember, 1918, a motive was ascertained for the murder by noticing at the autopsy the shaving of the anal hair and the presence of pubic hair on the body of the victim, a *sowar* (lancer), about nineteen years old, who was alleged to be a passive agent, and who was killed by his fellow *sowar* of the fifth cavalry.

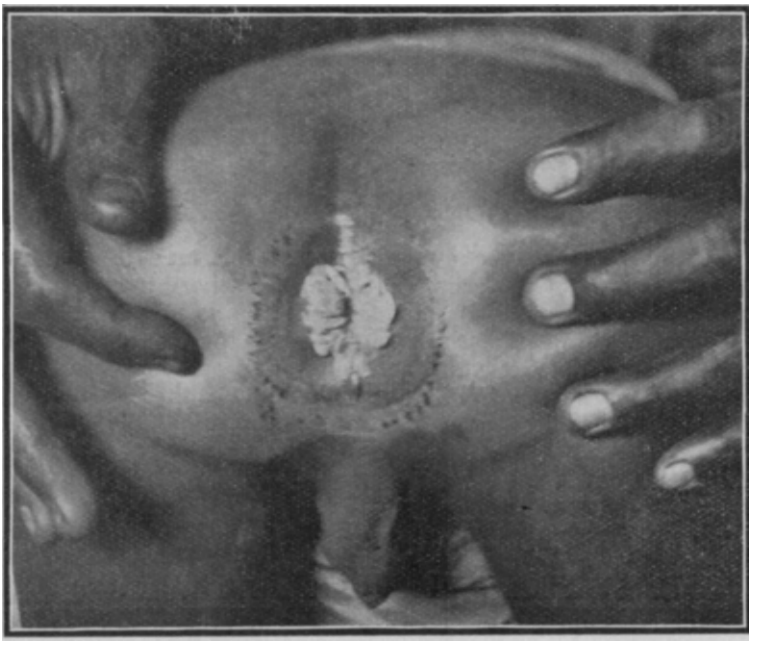


Fig. 112.—Case of a habitual passive agent. The anus shows condylomata with a sanious purulent discharge.

2. A funnel-shaped depression of the buttocks towards the anus. But this may be absent in strong healthy persons who are habituated to the act as passive agents.

A Brahmin, aged about 40, who, according to his own statement, had been a pathic agent for at least twenty years, had a typical Hunterian chancre, situated one inch in front of the anus, which he admitted to have contracted from one of his friends. The genitals were well formed and there was no deformation of the anal region, no infundibulum or loss of rugæ, and the tone of the sphincter was normal. —*Sutherland, Ind. Med. Gaz., June,* 1902, *p.* 245.

Again, this condition may be natural in thin individuals or old women.[1]

3. The dilated and patulous condition of the anus with disappearance of its radial folds and the prolapse of the rectal mucosa. In a dead body

1. *Thoinot, Medico-Legal Aspects of Moral Offences, Eng. Trans., p.* 216.

the anal orifice dilates from the relaxation of the sphincter and the protrusion of the rectum occurs from decomposition.

4. Cicatrices of old lacerations in the rectum near the anus.

5. The presence of a gonorrhœal discharge, chancre or condyloma. The active agent may be infected by the passive agent, who is already afflicted with gonorrhœa or syphilis.

On the 8th August, 1921, I examined a boy of Police Station Chowk, who was accused as a passive agent in a case of unnatural offence under section 377, I. P. C. He had an abrasion in the right posterior aspect of the anus, the sphincter of which was easily dilatable. There was some purulent discharge which was found to be gonorrhœal by the pathologist to the King George's Hospital.

In July, 1922, I examined a Hindu *Hijrah*, about 45 years old, who had received a superficial cut along the left side of the head above the right temple. On enquiry he admitted that a young man whom he had allowed to stay with him for the night, inflicted the cut on his head. I examined him, and found a condylomatous growth round about his anus.

EXAMINATION OF THE ACTIVE AGENT

No conclusive signs are evident, unless the man is examined soon after the commission of the crime. In that case there may be an abrasion on the prepuce, glans penis, or on its frænum, and stains of fæcal matter may be found on the penis, pubes, thighs, or on the clothes. The presence of blood and seminal stains is only corroborative evidence but not positive. There may be marks of violence on the body if the passive agent is a grown-up boy, and if the crime is perpetrated without his consent.

If the active agent is suffering from gonorrhœa or syphilis, the passive agent should be examined for the evidence of either of these diseases.

In males who are habitual sodomites the penis is, sometimes, elongated and constricted at some distance from the glans with the twisted urethra, probably owing to the constricting pressure exerted by the sphincter ani. These peculiarities may, however, be due to defective development. I have seen only one case in which a teacher who was charged with having committed an unnatural offence on his pupil of about ten years was found to have the body of his penis constricted in about its middle. He was proved to be a habitual active agent.

TRIBADISM

This form of mental aberration, which is known as Lesbian love, is practised by one woman on another and consists in friction of the genital organs by mutual bodily contact for the gratification of the sexual desire. It is said that in some instances an unduly developed clitoris is used as an instrument of passion, while in other cases some artificial contrivance is employed. Aristophanes[1] mentions the use of an artificial penis by Milesian females. This sort of sexual inversion is found among some women, though no such case has been brought before the Court. Such homosexual women are generally mental degenerates, and have very often natural antipathy and indifference towards individuals of the opposite

1. *Casper, Forensic Med., Eng. Trans., Vol. III, p.* 335.

sex. On the other hand, they are so jealous of the women with whom they are in inverted love that they are, sometimes, incited to commit even murder.

BESTIALITY

This means sexual intercourse by a man with the opposite sex of an animal. Very rarely, a woman resorts to this practice. Cases, though rare, do occur among villagers, who go out to graze cattle in fields far away from the gaze of the human eye. The animals, that have been selected for this purpose, are cows, mares, she-asses, goats, bitches and even hens.

It must be noted that sexual intercourse per nose with a bullock is regarded as an unnatural offence within the meaning of section 377, I. P. C.

In cases of bestiality the perpetrators of the crime are caught red handed ; medical evidence, therefore, is not required to prove the offence. But, as false accusations by village *chaukidars* and others are not uncommon in India, it is necessary that the accused should be examined by a medical man. The only important signs confirming the commission of the crime are the presence of human spermatozoa in the vaginal canal of the animal, and the presence of the animal hair, especially of the genitals, on the person or the clothing of the accused together with some suspicious stains or abrasions on his generative organ. Sometimes, lacerations of the genitals of the passive animal with an effusion of blood may be found.

Among half-a-dozen cases of bestiality reported to me in Agra during a period of eleven years, I could give a definite opinion only in one case from identifying by microscopic examination the hairs of the passive animal found under the prepuce, on the thighs and on the loin cloth (*dhoti*) of the accused.

A Mahomedan male, 23 years old, was caught committing an unnatural offence on a she-ass at 3 p.m. on the 29th October, 1927. He was medically examined in the King George's Hospital, Lucknow, at 12 noon on the next day. He had no signs of injury to his penis, but the smear taken of the urethral discharge was found to contain pus cells with very few gram-negative diplococci. The smears of the material taken from the vagina of the she-ass showed very few pus cells and a few human spermatozoa. The animal had no mark of violence on or about her genitals. It may be mentioned that there is a superstitious belief among illiterate people that they are cured of gonorrhœa by committing sexual intercourse with a she-ass.

1. *Khandu* (1934), 35 P. L. R. 73, 35, *Crim, Law Jour., p.* 1096 ; *Ratanlal and Thakore, Law of Crimes, Ed. XIV, p.* 906.

CHAPTER XVII

MISCARRIAGE

Definition.—Legally, miscarriage means the premature expulsion of the product of conception, an ovum or a fœtus, from the uterus, at any period of pregnancy before the full term is reached. Medically, three distinct terms, *viz.*, abortion, miscarriage and premature labour, are used to denote the expulsion of a fœtus at different stages of gestation. Thus, the term, abortion, is used only when an ovum is expelled within the first three months of pregnancy before the placenta is formed. Miscarriage is used when a fœtus is expelled from the fourth to the seventh month of gestation before it is viable, while premature labour is the delivery of a viable child possibly capable of being reared, before it has become fully mature.

CLASSIFICATION OF MISCARRIAGE

Miscarriage may be classified as *natural* and *artificial,* the latter being subdivided into *justifiable* and *criminal.*

NATURAL MISCARRIAGE

It must be remembered that miscarriages are naturally common among pregnant women, the proportion being one miscarriage to every five full-term deliveries. Miscarriages are most frequent within the first four months of pregnancy owing to the slight attachment of the ovum to the uterine wall. Within the first few weeks the ovum being very minute is cast off without being recognised or miscarriage being suspected. Very many cases, in which the woman goes one or two weeks over her time, and then has what is supposed to be merely a more than usually profuse period, are probably instances of such early miscarriages.

Causes.—The causes of natural miscarriage are classified as those which are directly referable to the mother, and those which affect the fœtus.

A. **Causes referable to the Mother.**—1. Poisons circulating in the blood, such as small-pox, plague, influenza, malaria, syphilis, streptococcal infection, lead, copper, and mercury. Among these syphilis is one of the most frequent causes of miscarriage, and is likely to act in successive pregnancies. It causes the death of the fœtus.

Streptococcal infection of a chronic nature is supposed to be the cause of cases of repeated abortion, where no other cause can be detected. Curtis[1] has isolated the streptococcus as the direct cause of abortion in several cases reported by him. He isolated the streptococci from the urine of a mother whose child was born dead, from the placenta, and also from the heart's blood of the still-born child.

1. *Peterson, Haines and Webster, Leg. Med. and Toxic., Ed. II, Vol. I, p.* 979.

2. Diseases affecting the circulation of the blood, such as anæmia due to excessive lactation or vomiting, jaundice, chronic Bright's disease, and heart and lung diseases.

3. Those acting through the nervous system, *e.g.*, sudden shock, fear, joy, sorrow, chorea gravidarum and reflex action from irritation of the bladder, rectum, or mammæ.

4. Local conditions, such as inflammations, chronic displacements and fibroid tumours of the uterus, old peritoneal adhesions, and excessive sexual cohabitation by inducing local hyperæmia.

5. Physical causes which separate the ovum. For instance, a blow or a fall or some other accident even of a trivial nature. Miscarriage from such causes usually occurs among women who are predisposed to it.

B. **Causes affecting the Foetus.**—*Death of the fœtus.*—Death of the fœtus occurring from a faulty development, syphilis and other diseases leads to secondary changes, and ultimately produces the uterine contractions which end in its expulsion.

2. Diseases of decidua, and inflammation and fatty degeneration of the placenta.

ARTIFICIAL MISCARRIAGE

1. **Justifiable Miscarriage.**—This is also known as therapeutic miscarriage, the induction of which is justifiable only when caused in good faith to save the life of the woman[2] if it is materially endangered by the continuance of the pregnancy, but not to save the family honour or for any other ethical reason. It is much better to defer the operation till the child has attained viability, if it is possible, so that the mother and the child may be saved. If miscarriage has to be induced before the child has become viable, the physician should never undertake the operation without a preliminary consultation with another medical practitioner, preferably one holding superior qualifications, or an obstetrician specialist, nor without the written consent of the woman and her husband or her guardian. If the consent is verbal, it should be duly attested.

A suggestion has been made that the procuring of abortion should be regarded as justifiable, if the mother's health is likely to be permanently damaged by the continuation of the pregnancy. Lord Riddell[1] thinks that the induction of miscarriage is not only justifiable but a duty when the continuation of the pregnancy indicates grave danger to the mother's health whether the result is likely to be permanent or not. Some physicians also think that therapeutic abortion is necessary and should be classed as justifiable when performed for eugenic considerations, *e.g.*, in cases of epilepsy, mental disease, abuse of intoxicating drugs and conception after rape but as the law in India stands at present, an abortion performed for such purposes is regarded as illegal unless the continuance of the pregnancy endangered the woman's life.

1. *Sections* 312, *and* 315, *I. P. C., Appendix VII.*
2. *Brit. Med. Jour., Jan.* 29, 1927, *p.* 188.

Dr. Aleck Bourne performed an abortion on a girl of about 15 years who became pregnant after she had been assaulted by some soldiers and raped under so revolting a set of circumstances as could be imagined. He thought that the girl would, in all probability, if she had gone to full-term, have suffered from grave and lasting nervous damage which would have expressed itself in psychoneurotic and physical illness perhaps for the whole of her life. He did not consult any of his colleagues, as in such cases he was accustomed to act as "the second opinion" himself. He was charged with unlawfully using an instrument with intent to cause miscarriage, and the case turned on the interpretation for the first time of the word "unlawfully". The trial took place on the 18th and 19th July, 1938, at the Old Bailey before Mr. Justice Macnaghten. The Judge, in summing up, said "that no line can be drawn between danger to life and danger to health; that no doctor knows whether life is in danger until the patient is dead; and that if on reasonable grounds based on adequate knowledge after consultation with colleagues a doctor forms an opinion that the probable consequences of the continuance of pregnancy would make the woman a physical wreck, then he is not only entitled, but it is his duty, to perform an abortion. To preserve a woman's life is not merely to save her from death; it is also to save her from illness which would destroy so much of her life that it would hardly be worth living." The jury returned the unanimous verdict of not guilty and the judge acquitted Dr. Bourne.[1]

Indications.—The indications for producing justifiable miscarriage are—

1. Obstruction to the passage of a fœtus owing to the contracted and deformed pelvis or the presence of a tumour.
2. Contraction of the soft parts and vagina due to cicatrices.
3. Incarceration of the retroflexed uterus.
4. Uterine hæmorrhage, hydramnios, and an ovarian or fibroid tumour.
5. Eclampsia, albuminuria, and chorea.
6. Severe vomiting, pernicious anæmia and diseases of the heart and lungs.
7. Threatened or incipient insanity.
8. Chronic phthisis.

2. **Criminal Miscarriage.**—In India, criminal miscarriage is resorted to mostly by widows who are prevented by rigid social custom from re-marriage, and in a few instances by unmarried women, to get rid of the product of conception from illicit intercourse. It is, sometimes, practised by married women to avoid additions to their families, but it is not so common in India as in Western countries.

Criminal miscarriage is generally induced during the earlier months of pregnancy, but occasionally between the fourth and fifth months of pregnancy, when the woman is certain of her condition. Sometimes, women, believing themselves to be pregnant, make efforts to produce abortion and suffer from consequent ill-effects, although pregnancy may be absent. Most of the cases which recover after criminal miscarriage are never detected. A case comes for investigation before the Court of law only when the death of the woman occurs as a result of criminal miscarriage or when some enemy of her family secretly communicates the information to the police.

1. *Brit. Med. Jour., July* 30, 1938, *p.* 225.

Legal Bearing.—Sections 312, 313, 314, 315 and 316 of the Indian Penal Code[1] refer to the offences of criminal miscarriage and punishments awarded for these offences. To constitute the offence under section 312 it is necessary that the woman should be pregnant and that miscarriage should be caused with her consent, inasmuch as the person procuring the miscarriage and the woman who causes herself to miscarry are both liable to punishment unless such miscarriage was caused in good faith for the purpose of saving the life of the woman. It is also necessary to prove whether the woman was " with child " or " quick with child " for, in the latter case, the offence is liable to enhanced punishment. If the means used, however, do not succeed, the offence is merely an attempt punishable under section 511. An additional punishment is awarded under section 313, if miscarriage is caused without the woman's consent, whether she was quick with child or not. Under this section the person who causes the miscarriage is alone punished. If a pregnant woman dies from an act intended to cause miscarriage, the offender is prosecuted under section 314 even though he did not know or intend that his act was likely to cause her death. The punishment to be awarded in such a case varies according as the act was done with or without the woman's consent.

A person commits the offence under section 315, if he causes the death of a child before or after its birth by any act intended to prevent the child from being born alive or to cause it to die after its birth, unless the act is done in good faith for the purpose of saving the life of the mother. Section 316 deals with offences against children *in utero* where the pregnancy has advanced beyond the stage of quickening, and where the death is caused after the quickening and before the birth of the child. A person would be guilty of culpable homicide, if he caused the death of a pregnant woman by an act which he knew that it was likely to cause her death. If his act injured the woman and did not cause her death, but caused the death of her unborn quick child he would be guilty of the offence defined under this section.

The law of England on criminal miscarriage differs from that of India in certain respects. The woman's consent is immaterial and does not in any way affect the crime. It is also immaterial whether the woman is pregnant or not, unless she is accused of doing an act with intent to procure her own miscarriage. The success or failure to produce the miscarriage does not matter. A mere attempt is what constitutes the offence. Further a man is guilty of wilful murder, if the woman dies as a result of his act to procure miscarriage even though he did not intend to cause death.

MEANS TO INDUCE CRIMINAL MISCARRIAGE

The means adopted to induce criminal miscarriage are—

A. The internal use of drugs.

B. Mechanical violence.

---

1. *Appendix VII.*

A. **The Internal Use of Drugs.**—There are practically no drugs which, when administered by the mouth, act on the healthy uterus, and expel its contents, unless they are given in very large doses so as to have deleterious effects on the woman herself.

The drugs that are generally administered for this purpose may be clissafied as—

1. Those acting directly on the uterus:
2. Those acting reflexly through the genito-urinary channel.
3. Those acting reflexly through the gastro-intestinal canal.
4. Those having poisonous effects on the system generally.

1. **Those acting directly on the Uterus.**—These are ecbolics and emmenagogues. Ecbolics increase the uterine contractions; the chief of these are ergot, quinine, and cotton root bark. Ergot is the most commonly used for procuring criminal miscarriage. It acts as a true ecbolic, and produces powerful uterine contractions, but acts better if administered when the uterus is contracting. It, however, frequently fails during the earlier months of pregnancy. Quinine produces contractions of the uterus, acting directly on the muscular fibres[1] and is commonly used as an abortifacient amongst certain classes. A multipara, aged 30, who had missed six weeks, took 15 grains of quinine and aborted in about six hours.[2] The cotton root bark or gossypium is supposed to resemble ergot in its action.

Emmenagogues promote the menstrual flow, but do not act as abortifacients, unless administered in large and frequently repeated doses. The chief of these, most frequently used criminally, is savin in the form of oil of savin or a decoction or infusion of its leaves. Its abortifacient action is doubtful. It often causes death from gastro-intestinal irritation. Borax is also frequently used, but it is very doubtful in its action.

2. **Those acting reflexly through the Genito-urinary Channel.**—These are diuretics in large doses, but they are very mild, and generally fail in their action.

3. **Those acting reflexly through the Gastro-intestinal Canal.**—These are emetics and purgatives given in large doses. The emetic that is chiefly used is tartar emetic, and the purgatives that are commonly used for this purpose are croton oil, gamboge, colocynth, elaterium and aloes. The last drug acts also directly on the uterine muscle fibres, and produces powerful contractions.[3]

4. **Those having poisonous effects on the System generally.**—These are animal, vegetable and metallic irritant poisons. Among the meatllic poisons, lead is the only drug which requires special mention. It is used in the form of pills made from diachylon paste consisting of lead oxide and olive oil. In England, these pills are largely used by women, especially of the working class, to procure abortion. In an outlying part of Nottingham they were sold as Mrs. Seagrave's pills, which, on analysis, were

1. *Cushny, Brit. Med. Jour., Nov.* 24, 1906, *p.* 1461.
2. *Eales, Brit. Med, Jour., Aug.* 5, 1905, *p.* 312.
3. *Cushny, Brit. Med. Jour., Nov.* 24, 1906, *p.* 1461.

found to consist of 50 to 70 per cent of diachylon and aloes with an outer coating of boric acid. Mrs. Seagrave *alias* Wardle was arrested for selling "noxious things" with intent to procure abortion and sentenced to eighteen months' hard labour.[1]

Diachylon acts successfully in producing abortion, but at the same time produces the symptoms of chronic lead poisoning. If abortion does not occur from the use of these pills, and if pregnancy is carried to full term, it often happens that the child dies shortly after birth.

The drugs that are chiefly used in India for the purpose of procuring criminal miscarriage are the seeds and the unripe fruit of Carica papaya (*Papita* or *Papayya*), the unripe fruit of pine apple, the seeds of Daucus carota (*Gajar ka bij*), the milky juice of Calotropis gigantea (*Madar, Ak*), the bark of Plumbago rosea (*Lal Chitra*), Randia dumentorum (*Main phal*), Cuscuta reflexa (*Ghagar bel*), Celastrus paniculata (*Malkangani*), Anethum graveolens (*Sowa*), Cucumis trigonus (*Karit*), Momordica charantia (*Karela*), Morynga pterygosperma (*Shajna, Saragwa*), Caryophyllus aromaticus (*Lavang*), Myristica fragrans (*Jayphal*), Crocus sativus (*Zafran, Kesar*), Trigonella fœnum-græcum (*Methi*), Cantharides, sal ammoniac, and copper, arsenic and mercury salts.[2]

B. **Mechanical Violence.**—This may be general or local.

General.—General violence acts directly on the uterus or indirectly by promoting congestion of the pelvic organs or hæmorrhage between the uterus and the membranes. The following methods are usually employed:—

1. Severe pressure on the abdomen by kneading, blows, kicks, jumping and tight lacing.

2. Violent exercise, such as riding on horseback, cycling, jumping from a height, jolting caused in driving on rough roads, long walks, running up and downstairs and carrying or lifting heavy weights.

3. Cupping, usually by placing a lighted wick on the hypogastric region and turning a brass mug (*lota*) mouth downwards over it. Traction is then made upon the mug, while it is firmly adherent, and probably a partial separation of the placenta, or possibly very severe injury to the uterine parietes is the result. This mode of procuring abortion is generally employed at advanced terms of pregnancy.[3]

4. Application of leeches to the pudenda, perinæum and the inner surface of the thighs.

5. Very hot and cold hip baths alternately.

Massage of the uterus through the abdominal wall is likely to result in miscarriage, but other kinds of violence, however severe they may be, do not often produce the desired effect. On the other hand, the slightest violence, such as the slipping of a foot, the fright of a cat or a dog or

1. *Brit. Med. Jour., Aug.* 11, 1906, *p.* 334.
2. *Chevers, Med. Juris., Ed. III, pp.* 712 *to* 720.
3. *Prof. T. W. Wilson quoted by Chevers, Med. Juris., Ed. III, p.* 720.

even the hearing of a noise, may cause miscarriage, especially in a woman who is predisposed to abort.

A woman[1] who was three months pregnant, was awakened by the noise of a collision of a motor car with the wall of her house and aborted the next day. She and her husband sued the motorist for damages. The doctor who attended the woman was clearly of opinion that the shock resulting from hearing a noise was enough to cause a miscarriage. The jury found a verdict for the plaintiffs. The husband recovered what he had paid on his wife's behalf and the wife was awarded £175.

Occasionally women are murdered to avoid further worry and disgrace.

On the 14th January, 1911, the body of a Hindu female, about 30 years old, was brought to the Agra Medical School Mortuary with a police report that she was found in a well in the jurisdiction of the Aharan Police Station. At the post-mortem examination a big, gaping, incised wound was found across the back of the neck cutting the third cervical vertebra and a twig of an *arhar* plant, about 3" long, with some stuff applied to one end was found lying in the os uteri. It appeared that an attempt was first made to procure abortion, but she was then murdered, and thrown into the well.

Local.—The commonest method of procuring miscarriage is to rupture the membranes by the introduction of an instrument, such as a uterine sound, catheter, douche cannula, knitting needle, hair pin, glass rod, etc., into the cavity of the uterus. Owing to the rupture of the membranes the liquor amnii flows away, and miscarriage frequently occurs from a few hours to two or three days, but occasionally may not occur for days or weeks. It is possible for a woman to pass a sound or other instrument into her own uterus, but it is difficult and fraught with danger.

An unscrupulous woman who wishes to abort will visit a medical practitioner either in his consulting-room or in the outpatients' department of a hospital and will get him to pass a sound by making a false statement that she suffers from displacement of the uterus and that on previous occasions it had been replaced with the aid of the uterine sound. In such a case the medical practitioner should never pass the uterine sound unless he is quite satisfied that his patient is not pregnant.

It is the usual practice of some abortionists to pass a sound into the uterus and then to direct the woman to go to her medical attendant as soon as pain and hæmorrhage have started in the hope that the medical attendant will treat her as a case of genuine abortion and will be held responsible for the occurrence of any untoward accident. The medical practitioner must always be on his guard in treating a case of threatened abortion and in a doubtful case must consult another practitioner.

In India, the so-called *Dhais* or abortionists who mostly practise this immoral and unlawful trade, introduce into the vagina or the os of the uterus a thin wooden or bamboo stick, from five to eight inches long, which is commonly known as an "abortion stick". This stick is wrapped round at one end with cotton wool, or a piece of rag, soaked with the juice of a marking nut, *madar* or euphorbium, or with a paste made of arsenious oxide, arsenic sulphide, and red lead. Instead of this stick a twig of some irritant plant, such as Calotropis gigantea (*Madar*), Nerium odorum (*Kaner*), Plumbago rosea (*Lal Chitra*) or Plumbago zeylanica

1. *Lancet, Dec.* 31, 1932, *p.* 1452.

(*Chitra*), is also used. The twig is frequently anointed with asafœtida (*Hing*) before its introduction.

In some cases, instead of the " abortion stick " the irritating juice is directly applied to the os, or a rag, in the form of a tampon, saturated with the irritating juice or paste, is introduced into the vagina.

The other methods are injections of soapy or hot fluids, or irritating lotions, such as corrosive sublimate and Condy's fluid, into the vagina or into the uterus. Electricity has been lately used to induce abortion, especially in the United States. The negative pole is applied to the cervix in the posterior vaginal cul-de-sac, and the positive pole is placed over the sacrum or lumbar vertebra. When the electric current is passed, the uterus contracts and may expel its contents. This kind of crime is difficult to be detected, unless there is a burn or mechanical injury.

ACCIDENTS FROM CRIMINAL MISCARRIAGE

When miscarriage has been caused by rupturing the membranes by the introduction of an " abortion stick ", excoriations, lacerations or perforations are usually produced in the upper part of the vagina or in the uterine walls. Death may occur immediately from shock and hæmorrhage from these injuries, or subsequently from septic pelvic peritonitis or septicæmia or even from tetanus.

In the case of death occurring from hæmorrhage the defence may raise a plea that the hæmorrhage was due to menstruation, and not the result of criminal miscarriage. The uterus and the pelvic organs are most probably found congested if death took place during a menstrual flow, but they are pale and anæmic if hæmorrhage occurred as a result of criminal abortion.

If death does not occur, the subinvolution of the uterus may result with concomitant symptoms of displacements, menorrhagia, leucorrhœa, etc.

When the act has been accomplished by injecting some fluid into the vagina or uterus, death may take place from shock due to the sudden distension of the uterus or from the sudden entrance of the air or fluid into the uterine sinuses. Death may also occur from subsequent septic peritonitis or septicæmia, or metritis may occur leading to the adhesions of the ovaries, tubes and uterus. Rupture of the uterus may, sometimes, occur from the forcible injection of a fluid into its cavity.

A healthy young woman,[1] aged 21 years, died from shock occasioned by unlawful injection of a fluid made up of soap and water for the sole purpose of procuring abortion.

Richter[2] describes the case of a woman, aged 28 years, found dead in the kitchen of her dwelling. Near her lay a syringe and a vessel containing soapy water. The clothing was not bloody or torn. She, supposing herself to be three months pregnant, had tried to bring about abortion by injecting soapy water. At the autopsy the pericardium was found distended and tympanitic. In the pericardium there was dark, fluid blood containing air. There was also foamy blood in the right heart, and in both ovarian arteries bubbles of air were found. There were also blood in the uterus, an embryo, 6 cm. long, and at the site of the attachment of the embryo, fluid blood containing air. He describes another case of a similar nature in which a woman

1. *Lancet, Feb.* 4, 1928, *p.* 255.
2. *Peterson, Haines and Webster, Leg. Med. and Toxic., Ed. II, Vol. I, p.* 96.

died after injecting a solution of boric acid with a view to procure abortion. At the post-mortem examination there were bubbles of air in the uterus and in the ovum, and blood containing air in the veins of the pelvis and lower abdomen.

When drugs have been used to produce miscarriage, death may result from their poisonous effects, as most of the reputed abortifacients are irritant poisons. If death does not occur, the woman may show signs of chronic gastro-intestinal disturbances, nervous prostration and chronic ill-health.

EVIDENCE OF MISCARRIAGE

The evidence of miscarriage can be determined by examining the woman alleged to have miscarried and the material alleged to have been expelled from the uterus.

Examination of the Woman.—(*a*) *During Life.*—The signs of recent delivery are found. These will depend upon the stage to which pregnancy has advanced, and the time that has elapsed since miscarriage at the time of the examination. In the earlier months of pregnancy the signs are likely to disappear very soon after miscarriage, and the woman should, if possible, be medically examined within a very few days after its occurrence. If septic infection has occurred at the time of miscarriage, the signs would persist for a longer time.

The usual sign in such cases is a bloody discharge from the vagina, which is relaxed and dilated. On examining the vaginal canal with the speculum, excoriations, lacerations or wounds of the mucous membrane of the vagina may be discovered. The os and cervix are patulous, with or without fissures, tears or lacerations. The uterus may be found enlarged by bimanual examination or by passing the uterine sound. The enlarged breasts and other signs of pregnancy are the valuable points for diagnosis.

(*b*) *After Death.*—In addition to the signs of pregnancy and the lesions caused by general violence, the vaginal canal should be carefully examined for the presence of punctures or lacerations, and the marks of inflammation and corrosion on its mucous membrane. The uterus and its appendages with the vagina attached should then be carefully dissected out, and laid on the table for minute inspection. The condition of the os and cervix should be examined as to the presence or absence of fissures, lacerations or the existence of a foreign body. The uterus should then be cut open, and its increased size, the attachment of the placenta and the presence of blood clots or of the product of conception should be noted. The ovaries should be examined for the existence of a corpus luteum. The alimentary and urinary organs should also be examined for evidence of irritant poisoning.

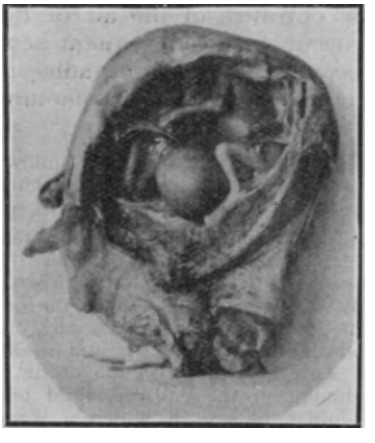


Fig. 113.—Uterus showing twins of about 3 months.

In all cases of criminal miscarriage the uterus and its appendages with any foreign matter found in the genital

canal, as well as the stomach, etc., should always be preserved for chemical analysis if there is the least suspicion of a drug having been used locally or internally.

Post-mortem Delivery.—The medical man should bear in mind the possibility of expulsion of a fœtus by the pressure of putrefactive gases generated in the abdominal cavity some days after the mother's death.

On March 18, 1920, a Hindu widow, 40 years old, finding that she had become pregnant, jumped into a well to conceal her shame. Four days later, the body was recovered from the well with a fœtus. At the post-mortem examination held by me on the 23rd March, the body was found to be decomposed. The face was bloated, and the hairs of the head had become loosened and were coming off. The abdomen was distended and the skin was peeling off at several places. The uterus was inverted and protruding from the vagina. The fœtus was a male of five months of intra-uterine life with the placenta and cord (ten inches long) intact, and attached to the umbilicus.

Brown[1] reports the case of a pregnant woman who had been dead about 60 hours before her body was found. There were signs of putrefaction in the skin and general emphysema. The vagina was not gaping. During the removal of the body from the hut, an eight months' fœtus, weighing 6 pounds, was spontaneously expelled. The inverted uterus prolapsed, showing the placenta still attached. There was no tear of the perinæum. The uterus was normal. The uterus showed slight peeling of the epidermis, but otherwise no signs of putrefaction.

R. Nagendran[2] reports the case of a widow, 35 years old, who died of drowning in a tank. At the end of three days the body floated to the surface and was removed by the police for investigation and examination. The post-mortem examination was held twelve hours after the inquest. The body was decomposed. Protruding through the vagina and hanging down was a fœtus with the umbilical cord, 13 inches long. The entrance to the vagina was plugged by the placenta. On further examination it was found that the uterus had prolapsed and completely everted, lying in the vaginal canal with the placenta still adherent to the fundus.

The material alleged to have been expelled from the Uterus.—When a substance alleged to have been expelled from the uterus as a product of conception is sent to the medical man for his opinion, he should thoroughly wash it in water to determine if it is a fœtus or merely a blood clot, a shred of the dysmenorrhœal membrane, a polypus, or a fibroid tumour. In a doubtful case a small portion of the suspicious material should be cut off, mounted on a slide in water or glycerine and examined under the low power of a microscope. I have often examined blood clots wrapped up in pieces of cloth alleged to be fœtuses, and brought by women, who reported to the police that they had miscarried as the result of an assault or a kick on the hypogastrium. In one case a woman complained that owing to the injuries inflicted on her abdomen during a quarrel she aborted, and brought for my examination a piece of cloth containing blood clots and a tissue alleged to be a fœtus of three months' pregnancy. On microscopic examination the tissue was found to be a piece of tumour. There was also no injury to the abdomen. In order to aggravate the offence women generally complain of miscarriage having occurred from an assault, when they are having their menses at the time of the struggle or when the menstrual flow has followed it.

1. *South African Med. Assoc. Jour., Cape Town, Feb.* 11, 1928, *p.* 64; *Jour. Amer. Med. Assoc., April* 21, 1928, *p.* 1336.
2. *Ind. Med. Gazette, October,* 1932, *p.* 571.

In the early months of pregnancy if the embryo is not found, the presence of chorionic villi found under the low power of a microscope will decide the fact of miscarriage. It should be remembered that during the first three months of pregnancy the fœtus is expelled with its membranes *en masse*, but after this period the fœtus is born first and then after a time the placenta is detached and expelled, a portion of which may remain adherent to the uterus. If the placenta is sent along with the fœtus, it should be examined to ascertain if it is entire or torn at any place, and if there are any degenerative changes on its surface.

If it is a fœtus, it is necessary to determine its probable intra-uterine age, its viability and the presence or absence of wounds or injuries inflicted on the body.

DEVELOPMENT OF THE FŒTUS AT DIFFERENT PERIODS OF GESTATION

First Month (Fourth Week).—At the end of the first month the ovum is greyish in colour, about ¾" in diameter and is roughly equal to a pigeon's egg in size. Its weight is about 40 grains. The embryo is about 1|3rd inch long, and is attached to chorion with a very short cord. The umbilical vesicle is present. It has two extremities, the head being a thick swelling and the tail slender and well-marked. Two dark spots indicate the eyes, the mouth is represented by a cleft, and the limbs by the bud-like processes. Being very small and minute, it can hardly be detected in abortions when surrounded by blood clots.

Second Month (Eighth Week).—At the end of the second month the ovum is 1¾ inches long, about the size of an hen's egg, and weighs two to five drachms. The embryo measures ¾" in length. The mouth and nose are separated, the umbilical vesicle has disappeared and the generative organs are apparent, but the sex is indistinct. The anus appears as a dark spot. The cord is longer, and the placenta has commenced to form. The centres of ossification have begun in the mandible (lower jaw), clavicle, ribs and bodies of the vertebræ.

Third Month (Twelfth Week).—At the end of the third month the fœtus is 3 to 4 inches long, and weighs about one ounce. The placenta is developed and chorionic villi have atrophied. The cord is much longer, and has a spiral twist. The head is more rounded and separated from the body by the formation of the neck. The eyes and the mouth are closed. The nails in the form of thin membranes appear on the fingers and toes. The sex is still indistinguishable. The heart is divided into two chambers, and the alimentary canal is situated within the abdominal cavity.

Fourth Month (Sixteenth Week).—Towards the end of the fourth month the fœtus is 4 to 6 inches in length, and is 2 to 4 ounces in weight. The sex can be differentiated. The skin is rosy and firmer. Down begins to be formed on the body. The head is one-fourth of the length of the body. The convolutions of the brain are commencing to develop. The membrana pupillaris is visible. The skull bones are partly ossified, but the sutures and fontanelles are very wide apart. The gall-bladder is forming, and meconium is found in the duodenum. The umbilicus is situated near the pubes. The centres of ossification are present in the lower segments of the sacrum, and the ossicles of the ear have ossified.

Fifth Month (Twentieth Week).—The fœtus of the fifth month is 7 to 10 inches long, and weighs about eight ounces. Light hair is seen covering the head which is about 1|3rd of the length of the fœtus. Lanugo is quite distinct on the body. The nails are distinctly marked, but are very soft. The germs of the permanent teeth begin to appear in the jaw. The position of the umbilicus recedes upward. The centres of ossification are present in the os pubis, os calcis, and ischium. Yellowish, bile-stained fluid is found in the small intestine, and meconium of a yellowish-green colour at the commencement of the large intestine.

Sixth Month (Twenty-fourth Week).—Towards the end of the sixth month the fœtus is 9 to 12 inches long, and weighs 16 to 24 ounces. The body is cinnabar red

in colour, and the skin has a wrinkled appearance for want of fat in the body, though a small degree of fat is beginning to deposit in the subcutaneous cellular tissue. Cerebral hemispheres cover the cerebellum. The eyelids are adherent, and the membrana pupillaris still exists. The eyebrows and eyelashes are beginning to form ; the umbilicus is situated farther from the symphysis pubis. The testicles are lying close to the kidneys. Dark meconium is found in the upper part of the large intestine. The centres of ossification are present in the four divisions of the sternum.

Seventh Month (Twenty-eighth Week).—The fœtus is 13" to 15" long, and weighs 2 to 4 pounds. The skin is dusky red, thick and fibrous, and covered with vernix caseosa, which is a white, fatty substance formed of epidermal scales, lanugo and the secretion of the sebacious glands. The eyelids are open, and the membrana pupillaris has almost disappeared. The nails are thicker, but do not reach the ends of the fingers. Meconium occupies nearly all the large intestine. The testicles are in the act of descent, and are found in the external inguinal ring. The centre of ossification is in the astragalus.

Eighth Month (Thirty-second Week).—At the end of the eighth month the length of the fœtus is about 15 to 17 inches, and the weight from 3 to 5 pounds. The skin is rosy, covered with soft hair, and is not wrinkled in appearance, as there is more subcutaneous fat under it. The hair of the scalp is denser, and the lanugo has almost disappeared from the face. The membrana pupillaris is no more visible ; the nails have reached the ends of the fingers. The left testicle has already descended into the scrotum, but not the right. The centre of ossification is found in the last vertebra of the sacrum. This is the most important month from a medico-legal point of view as the child becomes viable at the end of the thirtieth week, *i.e.*, at the 210th day.

Ninth Month (Thirty-sixth Week).—At the end of the ninth month the fœtus is about 18 inches long, and weighs 5 to 6 pounds. There is no more senile appearance of the face. The scalp is covered with hair, while the down on the body has disappeared. The scrotum contains the testicles, and is wrinkled. The vulva is closed. Vernix caseosa is found in the flexures of the joints. Meconium is found at the termination of the large intestine. The centre of ossification commences to form in the lower epiphysis of the femur from the commencement of the ninth month.

Tenth Month (Fortieth Week).—The fœtus presents the signs of a mature (full-term) child. The length is from 19 to 20 inches, and the weight from 6 to 7 pounds. The scalp is covered with hair about 1 to 2 inches long, which is generally dark. Lanugo is nowhere seen except on the shoulders. The skin is pale, and covered with vernix caseosa. The nails project beyond the ends of the fingers but reach only the tips of the toes. The cartilages have formed in the nose and ears. The umbilicus is situated in the central part between the pubes and the ensiform cartilage. The testicles are contained in the scrotum. The labia majora cover the nymphæ and clitoris. The rectum contains dark brownish, green or nearly black meconium, which is voided within a few hours after live-birth. The centre of ossification is found in the lower epiphysis of the femur and measures from $\frac{3}{4}$ths of a line to 4 lines in diameter. The centre of ossification is also present in the cuboid.

DISTINCTION BETWEEN NATURAL AND CRIMINAL MISCARRIAGE

When miscarriage is proved to have taken place, the defence cannot deny it, but may raise a point that it was induced spontaneously and not criminally. In such a case it is not always easy for the medical man to give a definite opinion simply from examining the woman or the contents of the uterus. Natural miscarriage occurs generally in women who are weak, irritable and unhealthy, and when the embryo or the fœtal membranes are diseased. The medical man is, therefore, justified in pronouncing it as criminal miscarriage if he finds lacerations in the vagina and on the cervix, or marks of violence on the abdomen of a healthy woman, or wounds on the fœtus or membranes, otherwise free from

disease. The occurrence of septicæmia is highly suggestive, but not conclusive, of criminal miscarriage. Septic peritonitis or metritis occurs more frequently in criminal than in natural miscarriage but it may occur in the latter if proper antiseptic precautions are not taken in its after-treatment, whereas it may not occur in criminal miscarriage if proper attention is paid to asepsis while inducing it.

The question whether the marks of injuries on the vagina and the cervix were due to criminal interference, or due to traumatism by the passage of the fœtus during spontaneous miscarriage can be determined by noting their site, extent and appearance, and the physical development of the fœtus. It may be mentioned that in a miscarriage of two to three months' pregnancy the fœtus is usually expelled without lacerating the cervical tissues.

CHAPTER XVIII

INFANTICIDE

Definition.—Infanticide means the unlawful destruction of a newly-born infant, and is regarded as murder in law. It is punishable under section 302, I. P. C., by death or transportation for life and also fine. In a case in which one Sundarbai, a Hindu widow, aged 22 years, was accused of infanticide, the Honourable Judges of the Bombay High Court pointed out that the law should be changed so that infanticide be regarded distinct from ordinary murder, especially when an infant was killed by the mother, while she was still under the effects of child birth so that the balance of her mind was disturbed. It should be brought on a line with other civilised countries, such as England, France, Germany and Italy. The punishment provided should be imprisonment for a few years.[1] By the Infanticide Act of England passed in 1922, it is provided that where a woman, by any wilful act or omission, causes the death of her newly-born child in circumstances which, but for the Act, would have amounted to murder, but at the time of such act or omission she had not fully recovered from the effect of the birth, and by reason thereof the balance of her mind was disturbed, she shall be guilty of the felony of infanticide, and shall be punishable as for manslaughter.

In most cases the crime of infanticide is generally committed at the time of, or within a few hours after, the birth of the infant. There is nothing definite in law regarding the period up to which an infant may be considered "newly-born". The Judges in England have usually held that an infant is newly-born up to fifteen days after its birth, but in the case of *R.* v. *Hale* Mr. Justice Humphreys suggested that Parliament should pass a short section specifying that in the Infanticide Act, 1922, "newly-born" means born within four weeks, the period indicated by Lord Dawson of Penn in his evidence, and also adopted by the Ministry of Health in dealing with newly-born infants in their mortality tables.

Lord Dawson's Infanticide Bill which was passed by the House of Lords on the 13th April, 1938, repeals the Act of 1922, and re-enacts it with the following important amendments:—

1. A child shall be deemed to have been recently born if born within twelve months before its death.

2. A woman who, by any wilful act or omission, causes the death of her recently born child while the balance of her mind is disturbed by reason of her not having fully recovered from the effect of giving birth to the child or by reason of the effect of lactation consequent upon the birth of the child, shall be guilty of the felony of infanticide and shall be liable to the punishment prescribed for manslaughter.

---

1. *See also Lahore High Court Cr. App. No.* 719 *of* 1937, *Mt. Talian* v. *K. E., Cr. Law Jour., Sep.,* 1938, *p.* 718.

The bill has not yet been before the House of Commons.

The legal bearing on infanticide is the same as in culpable homicide, except that the law presumes that every child is born dead, unless the contrary can be proved by medical and other evidence. To substantiate a charge of infanticide the prosecution is required to prove that the child was born alive, and that it died from criminal violence inflicted after its birth.

Owing to certain social customs prevailing in the different communities of India, infanticide, especially of female children, was formerly very common but, with the spread of education and restrictive action by Government, it is now rare except in cases of illegitimate children born of widows who are not allowed to remarry. Whereas, in Europe and other countries unmarried girls commit infanticide to get rid of children begotten of illicit intercourse, since they do not have early marriages among them as in India.

In cases of infanticide the medical officer is required to examine the woman—the alleged mother of the child—and the dead body of the child. He has to examine the woman to determine if she has been recently delivered of a full-term child. With reference to the child he is called upon to solve the following questions raised by the police, when the body is sent for post-mortem examination :—

I. Was the child still-born or dead-born ?

II. Was the child born alive ?

III. If born alive, how long did the child survive the birth ?

IV. What was the cause of death ?

I. WAS THE CHILD STILL-BORN OR DEAD-BORN ?

To avoid confusion, a distinction must be drawn between the terms, *still-born* and *dead-born*. Under the Births and Deaths Registration Act, 1926, of England and Wales, a still-born child is defined as one which "has issued forth from its mother after the twenty-eighth week of pregnancy and which did not at any time after being completely expelled from its mother, breathe or show any other signs of life." Still-births occur more frequently among illegitimate and immature male children than among legitimate, mature and female children, and more often in primiparæ than in multiparæ. A dead-born child is one which has died *in utero* and may show one of the following signs after it is completely born :—

1. Signs of maceration, which is the most usual change following the death of the fœtus *in utero*. This occurs when the dead child remains for some time in the uterus surrounded with liquor amnii, but with the exclusion of air. Hence, if a child died *in utero* twenty-four hours before it was born, the child may not show the signs of maceration, and in such a case it will be difficult to state whether the child died before or during birth.

The body of a macerated fœtus is soft, flaccid and flattened, and emits a sweetish, disagreeable smell, which is quite different from that

of putrefaction. The skin assumes a red or purple tint, but never green as in putrefaction. Large blebs resembling pemphigus and containing a red serous or sero-sanguineous fluid are raised, and the epidermis is easily peeled off leaving moist and greasy patches. The tissues are generally œdematous, and a turbid reddish fluid collects in the serous cavities. The sutures of the cranial bones are separated, and hence the skull bones are freely movable over each other. The brain substance is converted into a greyish-red, pulpy mass. All the viscera become infiltrated and lose their anatomical features, but the lungs and uterus remain unaffected for a long time. The umbilical cord is red, smooth, softened and lacerable. If the membranes are ruptured after the death of the fœtus, air gains admission into the liquor amnii, and the fœtus undergoes putrefaction instead of maceration.

2. Signs of mummification, by which the fœtus is dried up and shrivelled. Such a condition results when the death of a fœtus occurs from a deficient supply of blood, when the liquor amnii is scanty and when no air has entered the uterus.

II. WAS THE CHILD BORN ALIVE?

Live-birth, according to English law, means a child completely born external to the mother irrespective of the attachment or severance of the cord and manifesting some sign of independent life. Scientifically this definition does not seem to be correct, as it is absurd to call a child not born when one foot remains in the vagina, the rest of its body has been born and it has been breathing and crying for some time. To prove a charge of murder in such a case, it is not possible for a medical man to say definitely that the child was completely born before it was assaulted, unless he was present at the time of delivery, and thus there is always a chance of miscarriage of justice. To obviate this difficulty the British Parliament passed the Infant Life (Preservation) Act in 1929. It provides that any person who, with intent to destroy the life of a child capable of being born alive, by any wilful act causes a child to die before it has an existence independent of its mother, shall be guilty of the felony of child destruction, and shall be liable to penal servitude for life, provided it is proved that the act was not done in good faith for the purpose only of preserving the life of the mother.

The definition held by Indian law is more correct and appropriate. It constitutes live-birth, even if only a part of a living child is born, and the causing of death of such a child is regarded as culpable homicide.[1]

The Evidence of Live-Birth.—In civil cases, the cry, the feeling, seeing or hearing of the heart-beat or slight muscular movements, such as twitchings of the eyelids, are sufficient to establish the proof of live-birth. It is said that the mere "crying" of a child, though very strong evidence of live-birth, cannot be relied on as positive proof, for it is possible for the child to cry while the head is still in the uterus (vagitus uterinus), or in the vagina (vagitus vaginalis), and die before it is completely born. This can happen after rupture of the membranes, and is possible, if the

1. Vide *Explanation 3 of Section 299, I.P.C., Appendix VII.*

air has passed into the uterus or vaginal canal, and reached the child's mouth and nostrils.

Planchu and Reure[1] describe a case in which Reure heard loud crying of the child *in utero* for more than an hour, nine days before delivery. The patient complained of a watery discharge, and as labour had not set in, was ordered rest in bed and vaginal douches of hot water. A pear-shaped syringe was used which was afterwards shown to eject much air with the water. Reure was called because of the crying which was so loud on his arrival that he expected to find the child born. The cries lasted almost continuously for two hours and a half. On each of the following nine days the fœtal heart sounds were, on an average, 135 per minute. The child was born alive, breech first, but very much asphyxiated and was lost owing to the urgent need of attention to the mother who was bleeding.

Clouston[2] reports a case to which he was called out by a district nurse on November 10, 1931. The patient was in labour with her child and her previous pregnancies were normal and without difficulty. Labour had begun at 10 the previous night, and he arrived at 8-30 in the morning to find a brow presentation, the os almost fully dilated and the mother having no pains. The head was finally engaged and could not be moved. As he was withdrawing his hand, the child began to cry. It was the normal crying of a newly born infant, and was heard not only by the mother, nurse and himself, but also by a woman in the cottage in the room directly below the bed room. This loud crying persisted at frequent intervals for at least a minute.

Robert Watson[3] describes the case of a woman, 32 years old, who on October 25, 1932, was in labour but had made no progress. On examination he found a well-dilated os and a breech at the brim making no attempt to descend. The fœtus seemed very big, but the pelvis was well proportioned, so under chloroform he brought down a leg, having to reach the fundus to get a foot. The size and plumpness of the leg gave him furiously to think, and while he was arranging things for a hard job he heard just such a muffled cry as comes from the new born infant in a blanket. He whipped round, the nurse looked startled; they both bent over the anæsthetised woman and heard noises, unmistakable, familiar, from the woman's abdomen. A living male child, weighing 11 pounds and 12½ ounces, was delivered later on.

Westmoreland,[4] Harrison[5] and Douglas[6] have also recorded cases of vagitus uterinus.

It is also possible that a child may not utter a cry and yet may be born alive, if it happens to be immature or very delicate.

In criminal cases the Judge requires the medical witness to prove from the post-mortem examination that the child showed signs of life as a separate existence after it had wholly or partially emerged from its mother's womb. The most important sign is the establishment of respiration which can be determined from examining the chest and the lungs.

The appearances which show whether respiration has taken place or not are—

1. The shape of the chest.
2. The position of the diaphragm.
3. The changes in the lungs.
4. The changes in the stomach and intestines.

---

1. *Brit. Med. Jour., Jan.* 26, 1901, *Epitome, p.* 14.
2. *Brit. Med. Jour., Feb.* 4, 1933, *p.* 200.
3. *Brit. Med. Jour., Feb.* 25, 1933, *p.* 341.
4. *Brit. Med. Jour., Jan.* 26, 1901, *p.* 256.
5. *Jour. Amer. Med. Assoc., Oct.* 11, 1924, *p.* 1187.
6. *Brit. Med. Jour., Sep.* 11, 1937, *p.* 564.

5. The changes in the kidneys and bladder.
6. The change in the middle ear (Wredin's test).

1. **The Shape of the Chest.**—The chest is flat before respiration is established, but it expands and becomes arched or rounded after full respiration.

2. **The position of the Diaphragm.**—The abdomen should be opened before the thorax, and the position of the diaphragm should be noted by passing the finger up into its concave arch, the highest point of which is found at the level of the fourth or fifth rib, if respiration has not taken place; but the arch becomes flattened and depressed, and descends to the level of the sixth or seventh rib after respiration has been completely established. The position of the diaphragm may, however, be affected by pressure of the gases of decomposition developed within the thorax or abdominal cavity.

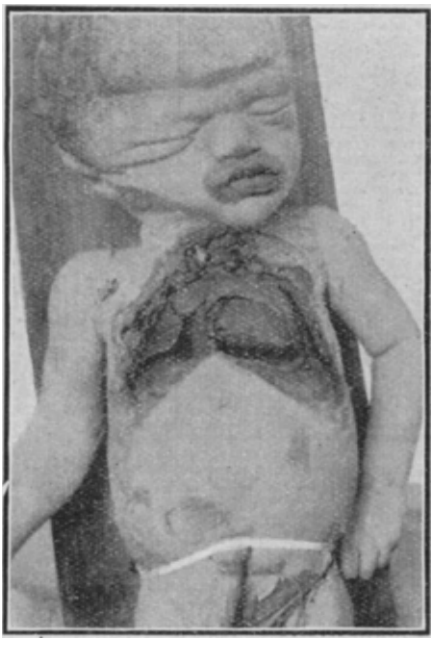


Fig. 114.—Unrespired lungs of a still-born, full-term infant.

3. **The Changes in the Lungs.**—These are considered with reference to their (*a*) volume, (*b*) consistence, (*c*) colour, and (*d*) weight.

(*a*) *Volume.*—Before respiration has taken place, the lungs are small with sharp margins, lie in the back part of the chest on either side of the vertebral column and are hardly seen on opening the chest, as the cavity is filled up by the heart and thymus. After complete respiration the lungs increase enormously in volume, have rounded margins and occupy the cavity covering more or less the thymus and heart.

(*b*) *Consistence.*—Before respiration the lungs are dense, firm, non-crepitant and liver-like. After respiration, they are spongy, elastic and crepitant.

(*c*) *Colour.*—Before respiration, the colour of the lungs is uniformly reddish-brown like that of the liver, but may become bright red at the margins from greater translucency owing to the thin walls. The surface of the lobules is marked with shallow furrows, but not with a mottled appearance. On section, little frothless blood exudes on pressing the cut surfaces. After respiration, the collapsed air-cells first become distended with air, usually on the edges and concave surface of the upper lobe of the right lung, and then on the remaining portions of the lungs. These air-cells are polygonal or angular in outline, arranged more often symmetrically in groups of four or five, though occasionally scattered irregularly, and are slightly raised above the surface. They are more

or less mottled or marbled in appearance with circumscribed rose-coloured patches. This mottled appearance is due to the blood vessels being filled with blood, and is characteristic of the lungs that have breathed. On section, frothy blood exudes from the cut surfaces on the application of very slight pressure. The fœtal lungs may assume a more or less rosy colour on exposure to the air after death, but the air-cells can never be

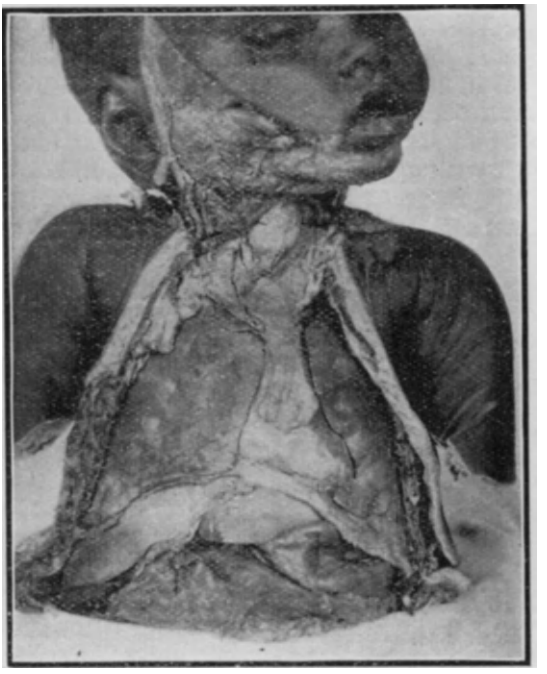


Fig. 115.—Respired lungs of an infant who survived after birth.

distended by the entrance of air into the lungs. This condition cannot be simulated by artificially inflating the lungs, as their colour is then cinnabar red, without any mottling.

(*d*) *Weight.*—As regards the weight of the lungs two tests are applied; *viz.*, the static test obtained by taking the absolute weight of the lungs, and the hydrostatic test which depends on their specific gravity.

Static Test.—In order to weigh the lungs they are removed along with the windpipe and bronchi after ligaturing the pulmonary vessels and separating them from the heart and thymus gland. The average weight of the fœtal lungs varies from 450 to 600 grains, while, owing to the increased flow of blood into the lungs, their weight is increased after respiration to 900 or 1000 grains. This varies in proportion to the weight and development of the child and according to the degree of respiration that has taken place. For obvious reasons it is not possible to weigh the lungs before and after respiration in any one case. Hence this test is worthless for medico-legal purposes, and another test has been devised in which the ratio of the weight of the lungs to that of the body is taken

into consideration to establish the fact of live-birth. This is known as *Ploucquet's Test* as Ploucquet was the first to ascertain that the proportion of the weight of the lungs to that of the body is 1 : 70 before respiration, and 1 : 35 after respiration, but this test also has no medico-legal value as the ratio of the weights is mostly variable.

Hydrostatic Test (*Docimasia Pulmonaris*).—This is the most reliable and valuable test and should, as a rule, be performed before an opinion is given as to whether respiration has taken place or not. It is based on the fact that the specific gravity of the unrespired lungs varies from 1040 to 1056, and that of the respired lungs is 940, owing to their volume being increased due to the presence of air. The fœtal lungs, therefore, sink in water, and those, that have breathed, float.

The Method of Test.—The method of performing the test is to remove the lungs as far as the trachea along with the heart and thymus after securing the large vessels, and to place them in a glass jar or vessel, about twelve inches high and eight to ten inches in diameter, filled with tap water or preferably with distilled water, and to note whether they float or sink. The lungs are then separated from the heart and thymus by tying a ligature on the bronchi, and dividing them above it, when each lung individually is placed into the vessel to note its buoyancy. Each lung is then cut into twelve to twenty pieces, which are again to be tested as regards their floatation. If these pieces float, they are each squeezed between the thumb and index finger under the surface of water to see if any bubbles of air are given off, and if they still persist to float; or they are taken out of water, wrapped in a piece of cloth and placed on the floor between two pieces of card board, when firm and equable pressure is applied by putting a heavy weight or by standing on the upper card board without any jerky movement. The pieces are once more placed in water, and if they continue to float after the application of this pressure the medical jurist is justified in affirming that respiration has been established. If the lungs sink separately, or if they float, but the pieces sink after pressure, it means that respiration has not taken place. If some of the pieces float while others sink it shows feeble respiration owing to the partial penetration of air.

Objections.—The two following objections have been raised against the hydrostatic test but, on close scrutiny, neither of them diminishes the value of the test:—

1. The expanded lungs may sink from disease or from atelectasis.

2. The unexpanded lungs may float from the presence of putrefactive gases or from artificial inflation.

1. *The expanded lungs may sink from—*

(*a*) *Disease.*—The disease may be catarrhal or crupous pneumonia, or it may be congenital tuberculosis or syphilis. In all these cases the pathological conditions characteristic of the disease can always be easily recognised, and it is not likely that both the lungs will be similarly affected. There will certainly be some portions of the expanded lungs which will escape the disease and consequently float in water. In cases of infanticide it is always necessary to examine the lungs for the evidence

of disease to exclude the possibility of their sinking in water due to this cause.

(*b*) *Atelectasis.*—Cases of atelectasis, *i.e.*, non-expansion of the lungs in children born alive and surviving for a few hours, though rare, have occurred, but there is no other test by which this condition can be determined, and so the test does not lose its value in its general application. Three explanations[1] for the non-expansion of the lungs have been given as mentioned below :—

1. Owing to the feeble respiration, air may not reach the alveoli, but the æration of blood may take place through the lining membrane of the trachea and bronchi.

2. The air which entered the lungs may have been entirely absorbed by the blood after respiration stopped, if circulation continued. It is a fact that the heart of a newly-born infant may continue pulsating for half an hour or more after the stoppage of respiration, or when the infant is in a state of asphyxia.

3. If the respiratory movements are very feeble it is quite possible for more air than what was taken during every act of inspiration, to be expelled from the lungs during expiration owing to the recoil of the lung tissue. Thus, the lungs may be emptied of all air, and may subsequently return to the fœtal condition.

Cases.—1. An inquest was held on the body of an illegitimate female child exhumed on the tenth day after its burial. There was ample evidence to show that the child lived five hours after its birth. At the necropsy the child was found to be a fairly well-developed full-time fœtus. The lungs were found collapsed, and were in a state of complete atelectasis. The lungs as a whole or when cut into separate pieces did not respond to the hydrostatic test. Dr. Dilworth, who examined the body, stated that but for the evidence heard by him at the inquest he would have no hesitation in affirming that the child had been still-born and had never breathed. He suggested that the child had lived a few hours by what little æration the blood received through the mucous membrane of the trachea and larger bronchi.—*Brit. Med. Jour., Dec.* 1, 1900, *p.* 1567.

2. A married woman, the mother of a family, was delivered of a seven months' child. The infant was washed and dressed; it did not cry, but the nurse said "it made a moaning noise." It lived a little over seven hours. An inquest was held, and at the autopsy the lungs were found collapsed and had all the appearance of those of a child who had never breathed. There were no developed air cells or vermilion spots. The lungs sank in water. They were then cut up into small pieces and every piece went to the bottom immediately it was thrown into the vessel. There was no doubt that the infant had lived and breathed though probably very feebly, for seven hours.—*Charles Randolph, Ibid., Jan.* 19, 1901, *p.* 146.

2. *The unexpanded lungs may float from—*

(*a*) *The Presence of Putrefactive Gases.*—For the putrefactive gases to be the possible cause of floating the lungs, there should be distinct signs of decomposition of the body as well as of the lungs. The putrefied lungs are soft and greenish in colour, and the putrefactive gases collect under the pleuræ and in the connective tissue in the form of air bubbles of different sizes, which can be squeezed from place to place, and can be expelled on the application of pressure after cutting the lungs into pieces,

1. *Dixonmann, Forens. Med., Ed. VI, p.* 116.

so that they will sink when placed in water, whereas the air due to the establishment of respiration cannot be expelled from the air vesicles after the application of pressure unless the force is so great as to disintegrate the lung tissue. However, the medical jurist should never venture an opinion if the lungs are too far advanced in putrefactoin.

(*b*) *Artificial Inflation.*—The fœtal lungs may be artificially inflated by blowing air through a tube, catheter or cannula passed into the trachea, by the mouth to mouth method or by Schultze's method (swinging the body) of resuscitation. It is quite easy to inflate artificially the lungs removed from the body, but it is extremely difficult to do so *in situ*. Elsasser[1] performed experiments on forty-five children born dead, but met with complete success only in one case and partial success in thirty-four. In such cases the stomach and intestines are apt to be filled with air, while they are airless in children born dead. Artificial inflation, if done forcibly, is likely to rupture some of the superficial alveoli and produce emphysema. If all the alveoli have not been inflated, air can be removed from them by pressure, and the pieces of the lungs thus treated will sink. However, if all the alveoli have been inflated without any rupture the cut pieces of the lungs will float even after pressure, but they can be distinguished from the normally expanded lungs by their bright cinnabar colour, the absence of the marbled appearance of the surface, and the absence of the escape of frothy blood on section.

The possibility of inflating the lungs artificially in criminal cases should never be countenanced, as inflation can only be practised by the medical man or by the mother to save the child, but one who wants to destroy a newly-born infant will try to prevent respiration rather than induce artificial inflation of the lungs.

In conclusion, the medical man is justified in affirming that the child had lived during and after its birth if he finds the following appearances on the post-mortem examination of the body of a newly-born infant :—

1. A full-term mature fœtus judged from its length, weight and other characteristics, especially the centres of ossification in the lower epiphysis of the femur and in the tarsal cuboid bone. The centre of ossification in the upper end of the tibia is usually found at full-term or shortly after full-term.

2. The diaphragm standing at the sixth or seventh rib.

3. The lungs occupying more or less the thoracic cavity and covering a portion of the heart and thymus gland.

4. The marbled or mottled appearance of the lungs.

5. Bloody froth exuding from the cut surface of the lungs on slight pressure.

6. The lungs responding to the hydrostatic test.

When is the Hydrostatic Test not necessary ?—The medical man need not perform the hydrostatic test, if he finds that—

1. *Casper, Forens. Med., Vol. III, Eng. Trans., p.* 65.

1. The fœtus is born at less than 180 days of intra-uterine life, when it cannot be viable.

2. The fœtus is a monster, which, owing to congenital malformations, is incapable of living a separate existence.

3. The fœtus shows signs of intra-uterine maceration.

4. The umbilical cord has separated and the umbilicus has cicatrized.

5. The stomach, on dissection, contains coagulated or half-coagulated milk as a result of the active digestive function.

4. **Changes in the Stomach and Intestines.**—During the process of respiration air is first swallowed in the stomach, and then gradually extends down the intestines. Hence when the stomach and intestines are removed from the body after tying double ligatures at each end of the stomach, at the end of the duodenum and also at some lower parts of the intestines, they will float when placed in water. They are then separated and tested separately for floating capacity. If respiration has not taken place, the stomach and intestines, being airless, will sink in water. This is known as *Breslau's second life test.* It is a corroborative test rather than a conclusive one. The practicability of this test is useful especially when air has been prevented from entering the lungs by foreign bodies or by occlusion of the bronchi. When breathing is impeded or imperfect, air enters and fills the stomach and intestines with a larger quantity than when breathing has completely and speedily taken place. The test is useless when the body has undergone decomposition, or when there has been an attempt at artificial inflation of the lungs.

On careful dissection under water so as not to allow its contents to escape, the stomach shows the presence of mucus with air bubbles and saliva, if respiration has been established ; whereas it will show the presence of only a glairy mucus if respiration has not taken place. The presence of blood, meconium and liquor amnii in the stomach indicates that the child was alive at or shortly before its brith and had swallowed these during the act of respiration. The presence of milk or farinaceous food in the stomach is very strong evidence that the child was not only born alive, but had lived for some time after birth. Any substance found in the stomach should be identified by microscopic examination. The absence of meconium from the bowels is not absolute proof of live-birth, as it may be voided in breech presentation even if the child is still-born. Under ordinary circumstances meconium is passed immediately, or within twenty-four hours, after birth.

It may be necessary to recognize the stains of meconium on the clothing. They are brownish-green and stiffen the fabric but do not penetrate deeply into its texture. When dissolved in water meconium forms a green solution which is acid in reaction, and is not affected by boiling.

5. **Changes in the Kidneys and Bladder.**—The deposit of uric acid in the form of brownish-yellow crystalline streaks found in the pelves of the kidneys has been regarded by some authorities as positive proof of live-

birth, but this sign is not reliable as the crystals have been found even in still-born children.

The absence of urine in the bladder is not at all diagnostic of live-birth, as urine may not pass for some hours after birth, or it may be passed mechanically during labour, and the child may subsequently be born dead.

6. **Change in the middle Ear (Wredin's Test).**—Dr. Wredin of Petrograd has observed that the gelatinous embryonic connective tissue, which fills the middle ear during fœtal life, disappears after birth, and is replaced by air, if respiration has taken place. This is not a valuable sign, since the gelatinous mass may disappear during fœtal life, or may not disappear until two or three weeks after birth.

III. IF BORN ALIVE, HOW LONG DID THE CHILD SURVIVE THE BIRTH ?

It is not possible to determine the exact length of time that a child has lived after its birth, but an approximate idea may be formed from carefully considering the following changes in the external and internal appearances of the body :—

1. **Changes in the Skin.**—The skin of a newly-born infant is bright red, and covered with vernix caseosa chiefly in the axilla, inguinal region and folds of the neck. The vernix is not easily removed, and persists for a day or two, but it is possible for a child to be born with little or no vernix. After birth the skin changes its colour, and becomes darker on the second or third day ; it then becomes brick red, and finally yellow. It assumes its normal colour in about a week's time. The exfoliation of the skin, chiefly on the abdomen, occurs during the first three days after birth. The exfoliation has to be distinguished from the detachment of the cuticle due to intra-uterine maceration.

2. **The Presence of Caput Succedaneum.**—The caput succedaneum is a valuable sign when present. It is formed on the presenting head during delivery. According to Powell[1] it is a bruise of the scalp and, when incised, contains an effusion of blood and not a serous fluid. Like ordinary bruises it undergoes the colour changes during absorption, and lasts about seven days.

3. **Changes in the Umbilical Cord.**—The changes in the umbilical cord begin to appear from the cut end to its base at the umbilicus soon after birth when it has been divided. Clotting occurs in the cut end after two hours. The portion of the cord attached to the child shrinks, and dries within twelve to twenty-four hours, and an inflammatory ring of redness forms at its base from thirty-six to forty-eight hours. This should not be confounded with a line of redness seen round the umbilicus at the time of birth. This line is merely red without any sign of a swelling or inflammation. By the second or third day it shrivels up, mummifies, and falls off on the fifth or sixth day leaving a slightly suppurating ulcer, which heals and cicatrizes within ten to twelve days. In rare cases the

1. *Lyon, Med. Juris., Ed. IX, p.* 406.

cord may drop off as early as the second day or as late as the tenth day. The mere mummification of the cord is not of any value as a sign of extra-uterine life, as it occurs in the dead body of a newly-born child if exposed to the air, but the separation of the cord with the formation of a cicatrix is a sure sign of survival of the child after birth.

The mummification of the cord does not occur if the child is submerged in water immediately after birth. Similarly, a cord which has already dried and withered may become soft and supple, though tough, if the body is lying in water, or wrapped in wet clothes.

4. **Changes in the Circulation.**—These occur after birth. The umbilical vessels, ductus venosus, ductus arteriosus and the foramen ovale, which were necessary to carry out the fœtal circulation, are no longer required to perform their functions after the birth of the child and are, therefore, obliterated. Thus, the umbilical arteries begin to contract in about ten hours after birth, and are completely closed by the third day. The umbilical vein and the ductus venosus are the next to contract. For the first three days the contraction is rather slow, but complete obliteration occurs on the fourth or fifth day. The ductus arteriosus begins to contract first at the aortic end, is reduced to the size of a crow-quill by the seventh day and usually closes completely by the tenth day. The closure of the foramen ovale generally occurs by the eighth or tenth day. Sometimes, it remains patent up to the second year, while in a few cases it remains open throughout life giving rise to cyanosis, a condition, known as *Morbus cœruleus*. In rare cases the foramen ovale has been closed at birth.

IV. WHAT WAS THE CAUSE OF DEATH ?

The death of the child may occur from natural, accidental or criminal causes.

NATURAL CAUSES

1. **Immaturity.**—If the child is prematurely born, it generally dies immediately after birth. In the case of the premature birth of a child the question may arise as to whether the birth was criminally induced or not, for, under the Indian Penal Code, the criminal induction of premature labour is an offence, but not culpable homicide, though under English law, a person is guilty of murder if he does an act by which a child is born prematurely so that it is not capable of living, and dies in consequence of its exposure to the external world.

2. **Debility.**—The child may be of full term, and yet may die after birth from debility due to the want of general development. In such a case no disease is detected, but some portions of the lungs may be found in a state of atelectasis from feeble respiration.

3. **Congenital Diseases.**—These are syphilis and specific fevers, such as small-pox, plague, etc., attacking the mother, or diseases of the child's internal organs, viz., the lungs, heart and brain.

Syphilis is the usual cause of the death of the fœtus. Specific fevers cause death from the toxæmic condition of the blood produced by the

attack on the mother, or from the attack on the child itself. Of the diseases of the internal organs, hepatization and tubercle of the lungs are common. The heart affections are rare, while diseases of the brain may destroy life without leaving any traces behind.

4. **Haemorrhage.**—This may occur from the umbilical cord, stomach, rectum or genitals.

5. **Malformations.**—These are acephalous and anencephalous monsters, or children born with congenital abnormalities of the blood vessels, heart, or alimentary canal. It must be remembered that monstrosity or malformation is no justification for taking the life of an infant. Again, it must be remembered that monsters do not necessarily die soon after birth.

6. **Disease of the Placenta.**—Disease of the placenta or its accidental separation from the uterine wall may cause the death of the fœus. This can be detected by examining the placenta or by examining the uterus, if the mother is dead and her body is available for the post-mortem examination.

7. **Spasm of the Larynx.**—This may occur from mucus or meconium being aspirated into the larynx or from the enlargement of the thymus gland.

8. **Placenta Praevia or Abnormal Gestation.**—Any of these conditions may cause the death of the fœtus.

ACCIDENTAL CAUSES

Accidents causing the death of the child may occur during or after birth.

During Birth.—1. **Protracted Labour.**—Prolonged labour may cause the death of the child by causing an extravasation of blood into the meninges or on the brain substance with or without fracture of the skull bones owing to severe compression of the head. In such cases it is not usual to find any external injury on the scalp. In this connection it should be borne in mind that the defective ossification of the cranial bones of a newly-born child may be confounded with fractures which may lead to dangerous mistakes. Casper[1] states that defective or retarded ossification commonly occurs in the frontal and parietal bones and rarely in the occipital bone of mature as well as immature children. He describes their characteristic appearances in the following terms :—" If the bone in question is held up to the light this is seen to shine through the opening, which is closed only by the pericranium. When the periosteal membrane is removed, the deficiency in the ossification is seen in the form of a round, or irregular opening, not often more than three lines in diameter, though frequently less ; its edges are irregular and serrated ; these edges *are never depressed as is the case in fractures ;* and neither they, nor the parts in their neighbourhood are ever observed to be ecchymosed." Sometimes, the child dies from exhaustion on account of prolonged and difficult labour.

1. *Forens. Med., Vol. III, Eng. Trans., pp.* 119, 120.

2. **Pressure on, or Prolapse of, the Cord.**—In such cases, death occurs from asphyxia and, on post-mortem examination, blood, meconium, liquor amnii, or vernix caseosa may be found in the bronchial tubes. These may be examined with a hand lens, or vernix caseosa may be stained with gentian and violet solution, and then examined under the low power of a microscope.

3. **Knots of the Cord or its Twisting round the Neck.**—The child is, sometimes, strangled before birth by the knots or loops of the cord being tightened, or the cord being coiled round its neck during delivery. A spasmodic contraction of the os uteri round the neck of the child may result in its death by suffocation.

4. **Injuries.**—Heavy blows on the abdomen of a pregnant woman with blunt weapons, kicks or falls from a height may kill the fœtus by causing concussion of the brain with or without fracture of the skull bones or rupture of the blood vessels or internal organs. Casper[1] quotes two cases of cranial fractures *in utero*.

One is the case of a primipara, aged 27, reported by Blot. The woman during labour and before the rupture of the membranes accidentally fell down in the court-yard, and fractured her femur with many severe contusions on the body. The child had now passed through the os uteri, much crepitation was felt on its head, and it was delivered with the forceps. There was no external injury to the scalp, but there were many ecchymoses in the subcutaneous cellular tissue in its anterior part and there was a fracture of each of the parietal bones.

The second case is recorded by Maschka. A woman towards the end of her eighth month of pregnancy leaped down from the second storey, fractured both her thigh bones, and died in six hours. On the fœtus *in utero* there were "several fractures of both parietal bones with extravasation of blood and coagula, both on the external surface and within the cranial cavity."

In such cases it is not necessary that there should be any external marks of injuries on the woman's abdomen. Sometimes, fractures of the long bones are caused by intra-uterine injuries and are recognized by the formation of callus.

In rare cases uterine contractions may be so powerful as to fracture the cranial bones of the fœtus.

Cater records the case of a woman, aged 32, who expected her first delivery about December 24, 1901. A month before the anticipated event there was hæmorrhage per vaginam following a "very bad dream," the patient leaving her bed during sleep. The child was then living and occupied the left dorso-anterior transverse position. The external os barely admitted the finger tip. The hæmorrhage ceased within forty-eight hours. On the 6th December labour pains commenced at 11 a.m. At 5 p.m. the doctor on his arrival found that the child was born and lying on its back with both legs and thighs flexed, the feet resting against the mother's left buttock. The cord was almost black and without pulsation, but there was no discoloration about the body. The eyes and tongue protruded, the head presenting the appearance of craniotomy forceps having been employed. The frontal bone was fractured, the fracture extending from above the left orbit to the right malar bone. The occipital bone was fractured into two unequal pieces. The child was full-term and weighed 6½ lbs. There was no such pelvic deformity as to be responsible for crushing of the child's head.[2]

1. *Forens. Med., Vol. III, Eng. Trans., p.* 114.
2. *Brit. Med. Jour., May* 17, 1902, *p.* 1207.

5. **Death of the Mother.**—When the mother dies in the act of delivery, the question arises as to how long a child may live *in utero* after her death. The time depends upon the cause of the mother's death. If death occurs slowly from hæmorrhage, there is very little chance of saving the child, but it may be saved if an attempt is made to extract it soon after the sudden death of the mother from some accident, if she was previously in good health. Dr. Harris[1] of Philadelphia, who has made many investigations regarding this point, is of opinion that a living fœtus may be extracted within two hours after the death of its mother.

Rosin[2] reports a case in which he delivered a full-time male child, weighing 7¾ pounds, by Cæsarian section in a state of asphyxia livida a quarter of an hour after the mother's death. Twenty minutes' artificial respiration and alternate immersions in hot and cold baths revived the child, who cried lustily.

After Birth.—1. **Suffocation.**—The child may die from suffocation after birth if it is born under a *caul, i.e.*, with membranes over the head, thus covering the mouth and nostrils. The child may also die from suffocation if its face is pressed accidentally in the clothes or submerged accidentally in the discharges, such as blood, liquor amnii or meconium.

2. **Precipitate Labour.**—In precipitate labour a child may be born without the mother's knowledge and may die from suffocation by falling accidentally into a privy pan containing fæces, or from drowning by falling into a chamber pot containing urine. If a woman is standing erect at the time the child may be forcibly shot down from her genital canal, and may die from a fall on a hard floor. In such a case one or both parietal bones may be fractured, and in some cases the fracture may radiate into the frontal, occipital or squamous portion of the temporal bone. Mud, sand or gravel may be found in the hair or injured scalp of the child, if the floor is covered with such material. The cord is either torn across, or the placenta is expelled with the child. Hæmorrhage from the torn cord, as a rule, stops owing to the contraction of the muscular wall of the umbilical arteries, but it may, sometimes, be so profuse as to cause the death of the child.

Precipitate labour is possible in multiparæ with large roomy pelves, but is extremely rare in primiparæ. Rensha[3] reports the case of a young primiparous woman who telling her mother that she was feeling queer stepped across the room and leaned on the mantel shelf, when without warning the child fell on the floor rupturing the cord, and almost immediately the placenta was expelled.

In connection with precipitate labour it will not be out of place to mention that the plea of unconscious delivery is, sometimes, raised in cases of infanticide; hence the medical jurist should bear in mind the possibility of such an event under certain conditions. There is no doubt that unconscious delivery may take place when a woman is under the influence of a narcotic or intoxicating drug, or suffering from syncope, asphyxia, apoplexy, coma, delirium or eclamptic convulsions. Cases have also been recorded, where women have been delivered unconsciously

1. *Witthaus, Med. Juris. and Toxic., Vol. II, p.* 453.
2. *Lancet, April* 16, 1927, *p.* 820.
3. *Brit. Med. Jour., March* 31, 1905, *p.* 777.

during profound sleep and hysterical fits. But these conditions should be such as to bring on deep lethargy and complete loss of sensation, or else the uterine pains of the expulsive stage of labour are likely to arouse the woman, especially if she happens to be a primipara. Chevers,[1] however, quotes a case of Dr. Wendell of Chicago in which a primipara was delivered of a child during sleep and woke up after delivery was completed. On the other hand, an easy and rapid delivery without any painful contractions is likely to occur in multiparous women who have roomy pelves and soft relaxed parts, especially if the fœtus is small.

CRIMINAL CAUSES

These may be—

A. The acts of commission, *e.g.*, the use of mechanical violence and poisoning.

B. The acts of omission or neglect.

A. ACTS OF COMMISSION

Mechanical Violence.—1. **Suffocation.**—This is the commonest form of infanticide. A newly-born infant is easily suffocated by pressing the face into some soft material, such as a pillow or bed cloth, or by closing the mouth and nostrils by a towel, handkerchief or some other cloth or by the hand. The mother may suffocate her child by intentionally overlaying it, or by forcing mud, rag, or cotton-wool into its mouth and throat. In one case I found a piece of white, blood-stained cloth, 13" long and 6" broad, stuffed into the throat and blocking the upper opening of the air passage. The mother may, sometimes, force her finger into the mouth of the child to prevent it from crying after birth and thus suffocate it to death. In such a case scratches or lacerations may be found about the mouth, tongue and throat. During the post-mortem examination of the body of a newly-born infant the mouth and throat should be examined for the presence of some foreign matter which, if detected, should be preserved, and sent in a sealed packet to the Superintendent of Police. The nose, lips and angles of the mouth should also be examined for the presence of bruising or other injury.

It should be remembered that infants are, sometimes, suffocated to death by pressure on the chest. Thus, in a case of infanticide I found the right fourth, fifth and sixth ribs fractured and in another case the left third, fourth and fifth ribs and the right third, fourth, fifth and sixth ribs were fractured.

2. **Strangulation.**—This is also a common form of child murder. During the act of strangulation far greater violence is used, than necessary, and severe marks of abrasions and contusions with extravasation of blood in the soft tissues are usually found on the neck. In one case a thick rope was used to strangle a child, and an *izarband* (a tape of pyjama) was used as a ligature round the neck in another case. This child with the *izarband* twisted round its neck and wrapped in a pyjama was found in the lavatory of a third class compartment of a railway train at the Agra

1. *Med. Juris., Ed. III, p.* 753.

Fort Station. Not unfrequently the child is murdered by passing the umbilical cord as a ligature round the neck. In such a case the plea put up by the defence is that the child was strangled accidentally by the cord coiling round the neck during delivery. In an accidental case of this nature there is most probably a broad continuous groove, livid or red in colour, without any excoriation, and the death being due to the stoppage of circulation, the lungs are generally found in a fœtal condition.

In October, 1921, the body of a newly-born infant was found lying in the grass farm at Naka Hindola. The umbilical cord was twisted round the neck, and the knot was tightened by fixing a piece of *madar* root in its loop and across the windpipe. The cord was twenty-five inches long with one end attached to the navel and the other cut with a knife or a pair of scissors. On removing the cord, a soft depressed mark, one inch by a quarter of an inch, was found encircling the neck over the windpipe. There was an extravasation of blood in the subcutaneous tissues under the ligature mark. The windpipe was congested. The lungs responded to the hydrostatic test and were congested.

Rarely, the natural folds of the skin in the neck of a fat child may resemble the cord marks caused by strangulation, but in that case no marks of abrasions or any extravasation of blood will be visible on the neck.

In cases of throttling, in addition to the bruises of the fingers or scratches of the finger-nails found on the neck, rupture of the muscles and fracture of the laryngeal or tracheal cartilages may be detected, as undue violence is used in throttling infants.

3. **Drowning.**—This is a rare form of child murder. As recorded by Chevers[1] submersion of the child's face into a cauldron of warm milk (*Dudh pita karna*) used to be a common method of infanticide in Benares and other neighbouring places. Now-a-days the usual custom is first to kill an infant by suffocation, strangulation, etc., and then to throw the body into a cesspool, well, tank or river with a view to conceal the crime.

The post-mortem appearances would be similar to those found in adults, if the chlid was drowned after respiration had been established. No signs would be evident if a woman was delivered in a bath and the child was drowned before respiration had taken place.

4. **Fracture of the Cranium.**—Fracture of the cranial bones results from a fall, from a blow on the head with a blunt weapon, or from the head being firmly pressed under the leg of a bedstead (*charpoy*). Excessive violence being used in such cases, depressed and extensive fractures of the skull bones with contusions or lacerated wounds of the scalp are usually noticed. Ordinarily a drop of 30 inches is sufficient to cause fracture of the skull bones, but a fall of 18 inches may fracture them as well.

A newly-born child which was found lying dead on a railway line near Achnera Station showed, on post-mortem examination, a fracture of the occipital bone with an extravasation of blood on the under surface of the scalp on its posterior aspect. It was presumed that the child was thrown out of the window of a railway carriage of a running train, as it was found soon after the train had passed.

1. *Med. Juris., Ed. III,* p. 753.

A newly-born female infant was discovered lying in a densely populated working class street in Leith, which had been thrown from a window on the first floor, a height of fourteen feet and three inches. The infant was removed to the parish hospital, where she died six hours later. At the necropsy no external marks of violence were found on the body, but the scalp all over felt soft. In the left parietal region there was a non-discoloured swelling of the size of half a walnut. On reflecting the scalp a copious effusion of dark coloured coagulated blood was found in the areolar tissue over almost its entire extent. The left parietal bone showed a somewhat depressed stellate fracture, radiating from a point situated about midway nearer its lower border. Of the three fissures one extended upwards to the sagittal sutures for a distance of two inches, a second reached forward to the frontal bone for one inch, and the third ran towards the occiput for one and a half inches. There was considerable congestion of the brain substance.—*Garland, Brit. Med. Jour., May* 18, 1907, *p.* 1182.

The defence is usually based on the plea of precipitate labour in those cases where the cause of death is due to fracture of the skull bones. Precipitate labour is likely to occur in a woman with a roomy pelvis and with an old laceration of the perinæum, or the woman may show a recent rupture of the perinæum if examined soon after delivery; but the perinæum may be ruptured in a primipara even if the delivery was normal. Again, in a case of precipitate labour the fœtal head will not show caput succedaneum and the placenta may be born along with the child, or the umbilical cord may show the torn and ragged ends due to its spontaneous rupture owing to the sudden jerk and strain. The rupture occurs more frequently at the fœtal end than at the placental end of the cord, but does not occur in its middle. Sometimes, the amniotic sheath of the cord remains intact, but the vessels contained in it may be torn at both ends. If the cord has been intentionally torn it is usual to find rupture of the liver caused by the forcible strain put upon the child's abdomen during the process of traction of the cord. The length of the cord, if available, should always be measured at the time of the post-mortem examination.

5. **Fracture and Dislocation of the Cervical Vertebrae.**—These injuries are produced by criminal violence applied to the neck. They are not usually caused by falls but may be produced accidentally by forcible rotation of the neck in an attempt made to correct a malposition of the fœtus or to extract the head in a breech presentation. It should, however, be remembered that the neck of a child is very short and is capable of considerable mobility.

6. **Wounds.**—The child may be killed by penetrating wounds into the heart, brain, medulla, or other internal organs caused by needles, pins or scissors. No external wound will be visible if a needle or pin is thrust through the fontanelles, through the inner canthus of the eye, up the nostrils, down the throat or up the rectum. Ogston[1] records several cases of infanticide by the employment of these methods. He also records the case of a child who had been suffering from convulsions and died accidentally. On dissection a pin was found sticking into the brain through the fontanelles.

Meixner[2] reports the case of an infant dying on the fourth day. The post-mortem examination showed that a needle was lodged in the right

1. *Med. Juris., p.* 267; *Collis Barry, Leg. Med., Vol. II, Ed. II, pp.* 176, 177.
2. *Encyclopaedia of Legal Medicine, Band* 48, *Supplement No. I; Peterson, Haines and Webster, Leg. Med. and Toxic., Ed. II, Vol. I, p.* 1013.

upper hemisphere of the brain through the anterior fontanelle. There was a very minute discharge of blood. The mother, an unmarried woman, had inserted the needle to kill the child. She had also given the child poison which produced its death, and the wound in the cranium was not the cause of the death.

Fig. 116.—Infanticide : Cut throat.
(From a photograph kindly lent by Dr. H. S. Mehta).

A Ucke[1] reports an extraordinary case of child murder. An illegitimate child, 20 days old, died in suspicious circumstances. It was found that the mother had ten days before the child's death passed four needles into the heart, lungs and liver. Death resulted from sepsis with purulent inflammation of myocardium and bilateral pleurisy. At the post-mortem examination the needles were found in the right heart and the liver, and had left behind a deposit of iron in the tissues and as a result of the damage to the right heart, congestion had been produced in the region of the vena cava, which had led to a typical atrophy of the liver cells.

1. *Deuts. Zeits. f. d. ges. gerichtl. Med.*, 1932, *XIX*, pp. 507-512 ; *The Medico-Legal and Crim. Review*, *April*, 1933, p. 153.

Poisoning.—Poison is rarely used for the purpose of infanticide though, sometimes, crude opium is put on the tongue of a child, or it is smeared on the nipple of the mother's breast, which is then given to the child to suck. Other poisons,[1] such as arsenic, *madar, datura* and tobacco, are also used for destroying newly-born infants. In suspicious cases, the stomach and other necessary viscera should be preserved for chemical analysis.

B. ACTS OF OMISSION OR NEGLECT

The law presumes that a woman who is about to be confined, should take ordinary precautions to save her child, after it is born. She is guilty of criminal negligence if she fails to do so. Thus, the acts of omission or neglect constituting the crime under the law are—

1. **Omission** to take the necessary help of a midwife or a skilled physician so that proper arrangements may be made to save the child after its birth. A married woman, or one who has borne children, is presumed by law to know her duty towards her new-born child. As soon as she gets labour pains she must inform her friends of her condition, and must send for medical aid. If she has failed to take any of these precautionary measures she may raise the following two points in defence :—

1. That she was not aware of her pregnancy till the birth of her child.

2. That she fainted away owing to the sudden onset of violent labour pains, and did not know what followed next.

In connection with the first point, it must be admitted that in rare cases it is possible for married women who become pregnant for the first time very late in life or have not conceived for many years after the birth of their last child to go to full term without being aware of their condition. They attribute the symptoms of pregnancy to some disease.

Beckers[2] records the case of a married woman, aged 30, the mother of three children, aged 3, 4 and 5, who found one morning that her bedclothes had been soaked by a clear vaginal discharge. On examination she was surprised to learn that she was at full term, as she had never suspected that she was pregnant. She had menstruated regularly as usual for four or five days at a time, and had felt no quickening, or had any digestive disturbance. Her abdomen and breasts had remained large since her last confinement. The woman gave birth to a normal infant the next day after expulsion of a considerable amount of amniotic fluid. Hobbs[3] describes a case in which he was called one night to see a young married woman said to be suffering from acute abdominal pain. On examination she was found to be in labour, but she was unaware of her condition. She was delivered of a healthy child of 7½ pounds in three hours and a half after her first pain. There was no evidence of quickening, and she felt perfectly fit and used to play tennis during the whole period of gestation without being conscious of the fact that she was pregnant. In fact she went out for a twelve-mile walk with her husband the day before the child was born. Dr. Robinson, Obstetric House Surgeon, King George's Hospital, Lucknow, described to me a case where a Hindu married woman, aged 42, who had five previous pregnancies, the last one being fifteen years ago, noticed a sudden escape of fluid from her vagina on the 7th October, 1930, at 7 a.m., and sought admission into the hospital on the next day at 4 p.m. after she had travelled 48 miles by a motor car to reach the

1. *Chevers, Med. Juris., Ed. III, p.* 764.
2. *Bruxelles Med., April* 19, 1925, *p.* 843 ; *Brit. Med. Jour., Aug.* 22, 1925, *Epitome, p.* 25.
3. *Brit. Med. Jour., March* 3, 1928, *p.* 382.

hospital. She did not think that she was pregnant, as she had had no amenorrhœa. During the last five months the periods were irregular and during the last three months the menstrual flow was scanty. She attributed it to the approach of menopause. On examination the fœtal heart sounds were heard on the left side, the vertex was presenting and the membranes had already ruptured with a partial dilatation of the os. At 7 p.m. the os was fully dilated and a living female child was delivered with forceps. It weighed 6¾ pounds.

However, cases in which there are distinct motives for pleading unconscious pregnancy require very careful examination. Such cases are probably unworthy of belief. This remark especially applies to a widow or an unmarried woman charged with infanticide, who is bound to consult a close friend or a medical practitioner on seeing the altered condition of her body, knowing fully well that she has exposed herself to the chances of pregnancy.

With reference to the second point it would be necessary for her to prove precipitate labour.

2. **Failure** to ligature the cord after it is cut may bleed the child to death. Fatal hæmorrhage may also occur if the cord is not tightly ligatured.

3. **Omission** to remove the child from the mother's discharges may result in suffocation. In the absence of a medical practitioner or any other attendant the question about the capacity of a woman after delivery may be raised. It is not easy to answer this question. Many women are known to have carried the child for a long distance soon after delivery, while other women may get so much exhausted as not to be able to move at all. It depends much upon the strength of the woman, and whether she is a primipara or a multipara. A weakly primaparous woman may faint away after delivery from mere exhaustion, or may be incapable of attending to the child from mere ignorance. The question has to be decided on circumstantial evidence.

4. **Omission** to protect the child from cold or heat. Exposure of a newly-born child to cold or heat may destroy its life without leaving any marks of violence suggestive of the cause of death except perhaps cerebral congestion.

At 8 p.m. on the 13th January, 1934, a newly-born male infant was found lying exposed on a grave in Shahmina, Lucknow. The infant was at once removed to Queen Mary's Hospital, where he was found very cold with a subnormal temperature and died at about 3 a.m. At the post-mortem examination held by me at 1 p.m. on the 15th January, 1934, the body was found to be that of a full-term male infant. There were no marks of external injury on the body. The brain, lungs and other viscera were congested.

5. **Omission** to supply the chlid with proper food according to its age. The starvation of a child constitutes slow death. The stomach and intestines must be examined for the presence of food. If the child is immature it is very difficult for the medical practitioner to say whether the death was due to natural causes owing to feebleness, or was due to starvation. The case depends chiefly upon circumstantial evidence.

THE ABANDONING OF INFANTS

When a newly-born infant, after it is born alive, is exposed in any place with the intention of abandoning it and death does not supervene, the parent or person responsible for the care of such infant is guilty under section 317, I.P.C., and may be punished with imprisonment of either description for a term which may extend to seven years, or with fine, or with both (see Appendix VII). The exposure need not be such as would put the child of tender years (under twelve years according to the section) in the immediate danger of health or life. The Madras High Court has held that it was not necessary that the exposure and abandonment must be under such circumstances as to endanger the life or the health of the child. The only ingredient required to complete the offence is an intention of wholly abandoning the child.[1] The offender may be tried for murder or culpable homicide, as the case may be, if the infant dies in consequence of the exposure (see Explanation to Section 317, I.P.C., Appendix VII).

CONCEALMENT OF BIRTH

In a case where infanticide is not proved, the mother is usually charged under section 318 I.P.C., with a lesser offence of concealment of birth by secretly disposing of the dead body of her newly-born child, and may be punished with simple or rigorous imprisonment for a period which may extend to two years, or with fine, or with both (see Appendix VII). It does not matter whether the child died before or after or during its birth, but there must be a secret disposal of the body. Leaving the dead body of a child in the compound of a house or in a public place where it can be easily seen does not constitute an offence under this section.

A woman, being pregnant with an illegitimate child, went to the village jungle for purpose of nature and there, in the presence of another woman, gave birth to a child, which died immediately. The dead body was left on the spot where the birth took place and was there discovered two days afterwards. It was held that mere leaving of the body where the birth took place did not constitute an offence under this section as it did not amount to a secret disposal.[2] But where a woman threw a child down a privy,[3] and where a woman placed a living child in a place of concealment, and on subsequently revisiting that place found the child dead and left it there,[4] it was held that this offence was committed.

For the purpose of this section a fœtus is considered a child if it has attained so much maturity as to be capable of maintaining a separate existence.

---

1. *Boya Sunkulamma*, (1890) 1 *Weir* 331, *Ratanlal and Thakore, Law of Crimes, Ed. XIV, p.* 790.
2. *Mt. Saraswati*, (1905) *I. N. L. R.* 89 ; *Ratanlal and Thakore, Law of Crimes, Ed. XIV, p.* 793 ; *Cal. H. Court Cr. Appeal No.* 801 *of* 1934, *K. E.* v. *Sailabaladasi, Cr. Law Jour.*, 1935, *Vol.* 36, *p.* 1460.
3. *Elizabeth Cornwall*, (1817), *R. and R.* 336 ; *Ratanlal and Thakore, Law of Crimes, Ed. XIV, p.* 792.
4. *Hughes*, (1850) 4 *Cox* 447, *Ibid., p.* 793.

CHAPTER XIX

INSANITY AND ITS MEDICO-LEGAL ASPECT

Definition.—It is not easy to give a succinct definition of insanity and the medical witness should never venture to do so in a Court of law, even though pressed for it by counsel, inasmuch as the law requires of him to affirm whether a particular individual, by reason of unsoundness of mind, is either incapable of looking after himself and managing his own affairs, or is dangerous to himself or to others. It appears that the law givers have used the term " unsoundness of mind " (*non compos mentis*) in the Indian Penal Code with a view to avoid the necessity of defining insanity. Unsoundness of mind covers a wider range, and is synonymous with insanity, lunacy, madness, mental derangement, mental disorder and mental aberration or alienation. All these terms are used for the disordered state of the mind in which an individual loses the power of regulating his actions and conduct according to the rules of society in which he is moving.

In cases of insanity brought before the Courts the following terms are often used in giving evidence ; hence the medical jurist should be well acquainted with the distinguishing points between them :—

1. **Delusion.**—A delusion is a false or erroneous belief in something which is not a fact. It is not always a sign of insanity. A normal man may have a delusion, but he corrects it by reasoning power, by applying his past experience, and by listening to the arguments of other people. A delusion in an insane person is a symptom of brain disease, is not in harmony with his education and surroundings, and cannot be corrected by any amount of logic, reasoning or argument. An insane person is guided by his own feelings and sensations, and does not care to listen to any arguments.

Delusions may be of grandeur or exaltation, of persecution, of depression, of reference, of jealousy, of infidelity, etc. Delusions of grandeur and delusions of persecution are often found together in the same person. For instance, a man who imagines himself to be very rich may also imagine that his enemies are conspiring to ruin him financially.

Delusions are very important from a medico-legal point of view, as they often affect the conduct and actions of the sufferer, and may lead him to commit suicide, murder or some other crime. The judge and the lawyer attach great importance to the presence of delusions as a sign of insanity. It is, therefore, necessary that a medical man, when called upon to examine the mental condition of a person, should carefully make a note of any insane delusions he has been able to elicit during the examination. It must be remembered that the delusions may not be evident in the beginning of the disease or in a form of insanity which is not characterized by delusions. In some cases the patient successfully conceals them, even though he be suffering from delusions.

2. **Hallucination.**—A hallucination is an erroneous sense perception without any external object or stimulus to produce it. It is due to some abnormal excitation in the brain cells, and may affect any or all the special senses, as also the cutaneous sensations. Hallucinations of sight and hearing are the most common. For instance, a man may imagine rats and mice crawling into his bed, when there are none, or may suspect a tiger coming to devour him, when there is no tiger. He may also hear the voices of persons in his room, when there is absolute silence.

Hallucinations occur in fevers and intoxications, as well as in insanity. They may be pleasant, but more often they are unpleasant. A person suffering from unpleasant and disagreeable hallucinations should be classed as a dangerous lunatic, and should be kept under proper restraint, for, owing to the delusions arising from these hallucinations, he may be incited to commit suicide or homicide.

3. **Illusion.**—An illusion is a false interpretation by the senses of an external object or stimulus, which has a real existence. For instance, a man may imagine a string hanging in his room to be a snake, or may, in the dark, mistake the stem of a tree on the roadside for a ghost. A sane man may experience illusions but, by closer investigation and his judging power, he is capable of correcting the false impression. An insane person cannot do so. He believes the illusion to be a reality and bases his conduct on that assumption. An illusion by itself is not a sign of madness but, owing to madness, the patient lacks the power or resolution to examine his illusion. Illusions of sight, hearing and other senses may occur in cases of mental disease.

4. **Impulse.**—This is "a sudden and irresistible force compelling a person to the conscious performance of some action without motive or forethought."[1] Normally when a man intends to do any act, he tries to realise its consequence and then decides whether he should accomplish it or not. If he finds that the consequences are unfavourable, he can restrain himself and will not undertake that act. An insane man has no balance of mind to use the reasoning faculty, and commits the act as soon as the idea occurs to him. He has no power to control it, however bad the consequences may be. It is possible that he may repent of his action afterwards. Under the influence of an irresistible impulse one may commit any form of crime, *e.g.*, suicide, homicide, theft, incendiarism or any perverted sexual act. Impulses are commonly met with in cases of imbecility, dementia, acute mania and epileptic insanity.

5. **Obsession.**—By obsession is meant "an imperative idea constantly obtruding itself upon the consciousness in spite of all efforts of the sufferer to drive it from his mind."[2] An obsessive idea arises from the emotional state, and the intellect protests against it. In fact it affords an excellent illustration of a border line between sanity and insanity. A man goes to bed at night after securely bolting the door of his room, but he immediately gets up to see if he has done so. If he repeats the process once or twice and then, being fully convinced of the security of his room, goes off to sleep, he is considered a sane person. On the contrary, if he

1. *Overbeck-Wright, Lunacy in India, p.* 12.
2. *Ibid., p.* 13.

does not sleep, and spends the whole night in frequently inspecting the security of the bolt, he is certainly to be considered insane, and requires to be placed under proper care and control.

Obsessive ideas generally occur among persons suffering from brain fag or nervous exhaustion. Very often they are unpleasant and annoying to the patients, who may wish to drive them from their minds, but cannot do so. These ideas are not infrequently accompanied by some sort of dread or fear.

Overbeck-Wright mentions the case of a woman who had been well-to-do, but came down in life after her husband's death. She had a daughter. Both of them were living with some distant relatives. At night the mother and child occupied one room. She was very much worried about the future of her daughter as she had no money and gradually felt the desire coming upon her to kill the child. Several times she asked her relatives to keep them separate, informing them of the reasons why she wished so. But they simply scoffed, and to emphasise their incredulity locked the mother and child at night with the result that she murdered the child. She was tried and sent to the Agra Asylum under Section 471 of the Criminal Procedure Code.[1]

6. **Lucid Interval.**—This is a period occurring in the course of mental disease, during which there is complete cessation of the symptoms of insanity, so that the individual can judge his acts soundly, and becomes legally liable for his deeds. In criminal cases it is, however, safer not to regard such an individual responsible for any offence, for it is, sometimes, very difficult to judge whether he was suffering from some mental aberration at the time of committing the offence. Lucid intervals are commonly met with in melancholia and mania.

CAUSES OF INSANITY

The causes of insanity are classified as **predisposing** and **exciting**.

Predisposing Causes.—The predisposing causes of insanity are—

1. Heredity.
2. Marriages.
3. Impaired vitality.
4. Absence of moral training and good breeding.

1. **Heredity.**—This is the most frequent cause of insanity. The tendency to inherit insanity depends largely upon the condition of the parent at the time of procreation. It is inherited more through the mother than through the father, and may show atavism as in other diseases. It is not necessary that the offspring of an insane person should show signs of insanity. It may only show some nervous disorder in the form of nervous excitement, hysteria, epilepsy, etc. It is also true that the parent of a nervous temperament may have an insane child.

2. **Marriages.**—Child marriages, especially among Hindus, are often conducive to insanity owing to the physical and nervous strain of premature sexual indulgence. Consanguineous marriages are apt to produce insanity as they contribute largely towards the perpetuation of bad hereditary influences.

1. *Lunacy in India, p.* 13.

3. **Impaired Vitality.**—The various causes which tend to impair the general vitality of the individual and consequently to render him more susceptible to the bacterial infection may be classed as the predisposing causes of insanity. Thus, mental worry, grief, physical strain, unhygienic surroundings, phthisis, syphilis, acute infectious fevers and the critical periods of life are all predisposing causes.

4. **Absence of moral Training and good Breeding.**—These also tend to predispose to insanity. Persons who have not received proper moral training and who have not been properly brought up according to their social status in life, are generally lacking in free will and self-control, and easily become victims of bad influences of undesirable associations during their life.

Exciting Causes.—The exciting causes of insanity may be divided into **physical** and **psychic.**

Physical Causes.—The physical causes leading to insanity may again be subdivided into two main groups, *viz.*, *non-toxic* and *toxic.*

Non-toxic Causes.—Among people predisposed by heredity or a neurotic temperament the non-toxic causes, such as exhaustion resulting from severe physical or mental strain, or serious illness, or cerebral hæmorrhage and injuries of the skull, may affect the brain, both pathologically and psychically, and may induce mental disorder.

Toxic Causes.—The toxic causes may be grouped under the following heads :—

1. Toxic substances resulting from excessive formation or deficient elimination of morbid waste products owing to disordered metabolism.

2. Toxines generated in the system by microbic infection.

3. Toxæmia produced by the excessive use of certain drugs, such as cannabis indica, opium, morphine, cocaine, alcohol, etc.

Psychic Causes.—Intense emotional disturbances, such as love, hatred, passion, disappointment, etc., are the psychic causes, which are liable to cause mental derangements, especially among those who are already predisposed to it.

INDICATIONS OF INSANITY

The onset of insanity is usually gradual, but it may be sudden in some cases. When the onset is gradual, the early physical symptoms of insanity are loss of appetite, constipation, dyspepsia and other digestive disturbances. Insomnia is almost a common symptom and in some cases there is a rise of temperature ; these are then followed by mental disturbances. The relatives and friends of the patient notice a change in his conduct and behaviour. He is not the same man as he used to be. He is quite eccentric in his dress, manners, habits and in his dealings with other people. Speech becomes involved, and the face bears a blank or vacant expression. He is gloomy, morose, listless, apathetic and does not care for the social conventionalities. At times he is very excitable and irritable even by trifling worries, which he will not otherwise mind. These are

followed by an alteration in his emotions. His affections for his wife and near relatives are changed into dislike and hatred. So far the intellect may not be impaired, and the patient may be quite capable of looking after himself and managing his business affairs. Later, his memory, however, fails him, and the power of self-control is lost. The power of reasoning is interfered with, and the judgment becomes weak and faulty. At this state the errors of perception of the special senses are evident in the form of hallucinations and delusions, which may lead the patient to perpetrate some crime or outrage.

In addition to these personal changes, the surroundings of the patient are often very characteristic. The house or room, in which he lives, is untidy and filthy, and the furniture is not unoften arranged in some fantastic fashion.

Stigmata of Degeneration.—In addition to the above symptoms, an insane person may exhibit physical peculiarities, known as stigmata of degeneration. These are—

1. *Changes in the Skull.*—The skull may be much smaller or larger in all its dimensions than that of the average healthy sane individual, or it may show inequality in the shape of both the sides. The frontal or occipital bone may also appear to be flattened and the vault may, therefore, look extended backwards and upwards in a very prominent manner.

2. *Changes in the Face.*—The face shows an irregular contour so that the nose appears to project on one side and the mouth to be tilted at one corner. In some cases the nose may be distorted, and stunted at the root. The ears are unusually large or small, and very often present a hæmatoma, which is known as the *asylum* or *insane ear*. The teeth are also set irregularly, and separated widely. The canine teeth are, sometimes, unduly prominent. The palate is highly arched, narrow, and unequal on both the sides. The pupils are irregular, and there is very often a high degree of myopia, or the presence of pterygium in one or both the eyes.

3. *Anomalies of the Extremities.*—These are manifested by unduly long arms, the occurrence of supernumerary fingers or toes, flat feet and the absence of hypothenar and thenar eminences.

4. *Sexual Abnormalities.*—There may be hairiness of the whole body and, due to the precocious development of the penis, the patient may get into the habit of self-abuse.

CLASSIFICATION OF INSANITY

The various forms of insanity may, for medico-legal purposes, be classified as—

1. Amentia.
2. Dementia.
3. Acute insanities.
4. Insanity associated with nervous diseases.

1. AMENTIA

Amentia is called "dementia naturalis" by lawyers, and results from arrested development of the brain before birth or in early childhood. It includes chiefly the types of insanity, known as idiocy, imbecility, feeble-mindedness and cretinism.

Idiocy.—This is a congenital condition due to the defective development of the mental faculties. All grades of this condition exist from the helpless life of a mere vegetable organism to one which can be compared to the life of young children, as far as mental development is concerned. An idiot is wanting in memory and will-power, is devoid of emotions, has

Fig. 117.—Case of Idiocy: Is listless and energic with marked mental confusion, and dirty in his habits. (*Dr. Benarsi Das' case*).

no initiative of any kind and is unable to fix attention on any subject. He is usually quiet, gentle and timid, though he can be easily irritated. He cannot express himself by articulate language, but he may be able to make himself understood by certain signs, cries or sounds. In some cases he is able to recognize his relatives, and learn with great difficulty. He is usually filthy in his habits, and has no concern as to what he eats or drinks. He is very often depraved in morals, and is, sometimes, cruel to weaker children as well as animals.

There is always some bodily deformity or peculiarity, such as a small (microcephalic), large (macrocephalic, hydrocephalic) or misshapen

head, cleft or highly arched palate, irregularly set teeth, enlarged tonsils, adenoids, curved bones, etc.

Imbecility.—This is a minor form of idiocy, and may or may not be congenital. Imbeciles are able to speak, though their command of language is very poor. Their memory is very feeble. In some cases it is highly developed, though not the intellect. They can mechanically repeat without any mistake what is taught them, but cannot understand its meaning. They are easily roused to passion, and may, consequently, become dangerous. They may commit theft or even murder. Owing to their repulsive manners and habits it is not possible to associate with them, but with a little patience and perseverance they can be taught to dress decently, to eat properly and to control their animal instincts.

A peculiar type of microcephalic imbeciles, commonly known as "Shah Daula's *chuha* (mice)" is prevalent in the Punjab. They are so named from their fanciful resemblance to mice owing to their flattened skull, and prominent ears. They are dedicated to the shrine of Shah Daula, whose tomb is in Gujrat (Punjab).[1] They have no other deformity except the peculiar shape of the head, though most of them are deaf-mute, and have a squint in the eye. They are capable of learning simple employments, and are usually modest and decent.

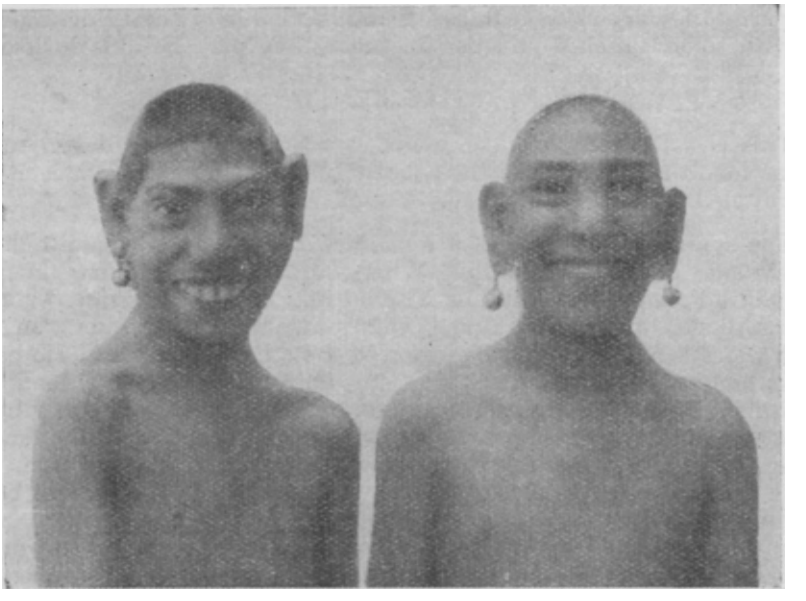


Fig. 118.—Microcephalic Imbeciles: Note the characteristic facial expression with the small head, low and flat forehead and prominent ears. (*By permission of Dr. N. J. Modi and the Editor of the* Indian Journal of Pediatrics).

Feeble-mindedness.—Under the Mental Deficiency (England) Act, 1913, feeble-minded persons or morons are defined as persons in whose case there exists from birth or from an early age mental defectiveness not

1. *Overbeck-Wright, Lunacy in India, p.* 322.

amounting to imbecility, yet so pronounced that they require care, supervision and control for their own protection, or for the protection of others, or, in the case of children, that they by reason of such defectiveness appear to be permanently incapable of receiving proper benefit from the instruction in ordinary schools. Feeble-minded individuals do not, as a rule, present bodily deformities and stigmata of degeneration, and are often capable of making their own living although they lack in initiative and ability for any work of responsibility. Such persons, however, develop vicious or criminal propensities, especially of a sexual nature, and are apt to commit assaults or even murders, as they are incapable of restraining their impulses.

Cretinism.—This is endemic, and is prevalent in the hilly districts. It is usually associated with goitre and other affections of the thyroid gland. Development of the body is generally arrested. The figure is squat and dwarfish with short thick limbs and clumsy movements. The complexion is sallow, the eyelids are swollen, and the lips and tongue are thickened. The skin is rough and pigmented. Such children learn to speak very late and that too, imperfectly. Some of them are deaf, and others blind.

Mentally cretins may look dull and stupid, or may be perfect idiots. Ordinarily they are slow in thought, and incapable of acquiring knowledge, but with some patience and perseverance they may be able to learn.

2. DEMENTIA

This is a form of insanity, which is produced by the degeneration of mental faculties, after they have been fully developed. Hence it is not congenital, but may occur at any period of life.

The symptoms appear all of a sudden in a previously sane individual, or they may appear gradually. When the attack is sudden, the patient passes into a condition of stupor without any emotional feeling or without any depression or delusion and becomes an imbecile or idiot. In a slow attack there is a gradual degeneration of the mental faculties. He becomes listless and apathetic, does not take any interest in his dress, food, family or business. He cannot fix his attention on any subject. Memory becomes feeble or is lost. Judgment is impaired, and his control over the emotional feelings is very much weakened. As the disease progresses from bad to worse, the common instincts of volition are abolished. The patient becomes irritable, incoherent, and begins to laugh or cry without rhyme or reason. He is mentally and morally depraved, and is unmindful of ordinary decencies of life. He sometimes resorts to masturbation in public.

Very often the appetite is voracious, but owing to impaired nutrition the patient becomes lean and thin.

Types of Dementia.—The following four types of dementia have been recognized :—Dementia præcox (Primary dementia), secondary dementia, senile dementia and organic dementia.

Dementia Praecox (Primary Dementia).—This is a psychosis, which usually occurs between fifteen and thirty years of age, and is characterized by a progressive mental deterioration. It is a term used by some

authorities to include the three conditions, (1) Katatonia, (2) Hebephrenia and (3) Paranoia, but this is not a scientifically correct term, as such conditions may appear in any age period and not only in youth.

Secondary Dementia.—This is a state of mental enfeeblement, which is the final result in all cases of acute insanity, which do not tend to recovery.

Fig. 119.—Case of Dementia præcox : Is dull, apathetic with childish delight, has impaired memory and is disinclined to answer. (*Dr. Benarsi Das' case*).

When a patient suffering from an attack of insanity passes into a state of weak-mindedness, he gets good appetite, sleeps well and improves in general health. However, general nutrition suffers in those cases where excitement and restlessness persist. Such patients suffer from insomnia and, owing to perverted appetite, will eat mud, rubbish or anything that comes to their hand. Owing to want of co-ordination their gait is unsteady and other movements are slow and clumsy.

As dementia develops, the patient becomes listless and apathetic, easily irritated and roused to passion, but by a little coaxing he can be appeased very soon. He is devoid of feelings and emotions. He is quite impulsive in his actions without any regard to consequences. He may be impelled to a suicidal or homicidal act, especially when delusions and hallucinations, the characteristic features of acute insanity, persist even after the patient has passed into a state of dementia.

Senile Dementia.—This condition results from the gradual decay of the body as well as the brain during old age, and depends upon the degenerative changes of the arteries. It affects those people, who have a hereditary taint of mental aberration, and who have led a strenuous life.

In this form the patient is forgetful, unable to fix attention on any subject, is dirty in his habits, and erotic in his tendencies. He begins to suspect his own near and dear relatives, and is often affected by hallucinations of sight and hearing and delusions of persecution. He imagines that he has become poor and destitute. He becomes melancholic and lastly becomes a perfect dement. Suicide is also common in such a condition. Maniacal excitement is very rare, though garrulity and continuous and aimless movements are sometimes seen.

Organic Dementia.—This condition is a result of some organic lesion of the brain. The lesion may be a localized one as a new growth, embolism, cerebral abscess or hæmorrhage, or it may be diffused as chronic meningo-encephalitis.

The symptoms vary according to the site and extent of the lesion. In a localized lesion the patient slowly becomes lethargic and somnolent. He speaks and thinks slowly and with great difficulty. His movements are slow and awkward. He does not seem to take interest in life, and has very few wants and desires. In acute cases the patient suddenly becomes restless and delirious, and suffers from visual and auditory hallucinations.

In the case of a diffuse lesion of the brain, the mental faculty is gradually diminished or abolished, accompanied by loss of memory and difficult speech. The patient is irritable, and is apt to get violent attacks of mania. Sometimes, convulsions occur, and exhaustion or syncope ends the scene.

In all cases of organic lesions of the brain if death does not occur soon, the patient becomes forgetful, loses perceptive faculties, and is incapable of fixing attention on present impressions. He is hopelessly indecent in his behaviour. He is unable to look after himself or manage his own business. Finally the patient becomes bed-ridden and passes into a state of complete dementia.

3. ACUTE INSANITIES

These are generally associated with some kind of toxæmia. Mania, melancholia, delusional insanity, exhaustion psychoses, katatonia and hebephrenia may be described under this heading. The first two disorders have been grouped together by Kræpelin under the term, *manic-depressive insanity,* but it is more convenient to describe them separately.

Mania.—This is a condition of exaltation affecting the emotions and the intellect and manifesting istelf in increased mental and physical activity.

For the convenience of description mania is subdivided into three forms, simple mania or hypomania, acute mania and chronic mania, although these forms merely represent the different stages of the same disease varying in degrees of intensity and duration.

Simple Mania or Hypomania.—This is the mildest form of mania, in which there is an exaggerated sense of self-importance. The symptoms manifested in this form result from the decreased inhibitions to the motor impulses. The general demeanour and conduct of the patient are greatly altered, although there is no real change in personality. He is quick-witted and entertaining in conversation, but owing to a lack of unity in

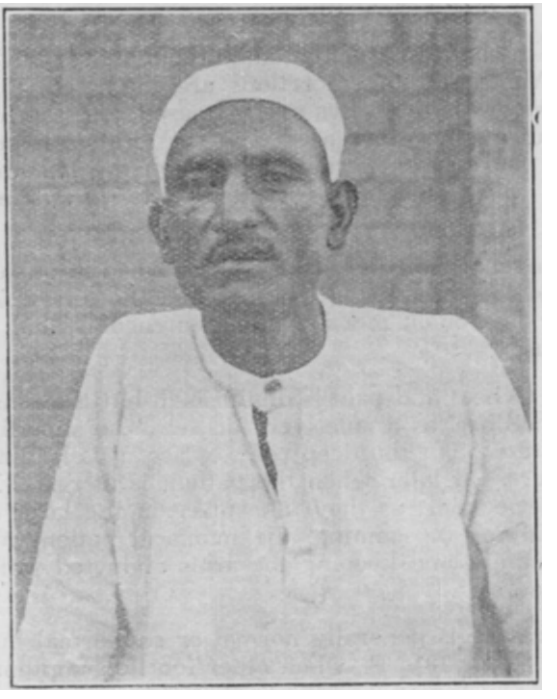


Fig. 120.—Case of Mania : Is excited, violent, abusive and destructive, talking incoherently and thinking himself a great man. (*Dr. Benarsi Das' case*).

the course of ideas he rapidly wanders from one subject to another. He is full of schemes and ideas which are never thoroughly worked out. Later, he becomes restless, irritable and interfering. He is always busy doing one thing or the other, but does not feel tired. He retains his memory and power of orientation, but lacks in moral control, as evidenced by his excessive indulgence in alcohol and sexual passions. There is no evidence of hallucinations or delusions. The patient often recovers from this form of the disease.

Acute Mania.—The attack of acute mania is usually gradual, preceded by a prodromal stage lasting two or three weeks. During this period there may be constant headache, general malaise, restlessness, insomnia, inability to concentrate and loss of weight. The patient is irritable and begins to dislike his friends and relatives. Sometimes, the attack commences suddenly without any prodromal symptoms.

The physical symptoms of acute mania are impaired general health, pale face, and bright and staring eyes with dilated pupils which react to light and accommodation. Gastric derangements are common. The breath is foul, the tongue is usually furred and the bowels are constipated. In the beginning appetite is impaired, but during the attack it becomes voracious. Anything that is eaten is digested. However, the patient, sometimes, refuses to take his food, and it becomes necessary to feed him with the nasal or œsophageal tube.

The pulse is slightly irregular and frequent, varying from 90 to 120 per minute. Almost all the secretions are increased. The amount and the amyolitic power of the saliva are increased, and the hydrochloric acid of the gastric juice is also increased. The perspiration is profuse and has a mousy odour. During the period of lactation the mammary secretion is increased, and may, sometimes, lead to the formation of mammary abscesses.

At the commencement the urine is diminished in quantity, but further in the course of the disease the quantity and the total solids of the urine are increased. In women menstruation is irregular, and the discharge is generally profuse.

Sensibility to heat and pain is diminished, but the sensations of touch, hearing and smell are, as a rule, very acute. The superficial reflexes are slightly exaggerated, but the deep reflexes are usually diminished at first, and may be increased later when the patient is at rest. Muscular movements are very peculiar, as they take place in the large proximal joints. Thus, while walking or running, the maniacal patient moves the trunk freely from the hips, and keeping the arms abducted waives them freely from the shoulders.

The temperature is generally normal or subnormal but, sometimes, it is raised to 100° F. or 101° F., when other febrile symptoms develop. The tongue is brown and furred, and the teeth and lips are covered with sordes. Constipation is very severe and complete insomnia is a marked symptom. The patient is unable to retain food even when given by the tube, and rapidly loses flesh and weight. The pulse is frequent, varying from 130 to 160 per minute, and the respirations are 30 to 40 per minute. Such a condition has been spoken of as *acute delirious mania.*

The prominent mental symptoms are excitement, loss of self-control, flight of ideas and great muscular activity. The patient is unable to fix his attention upon any one subject, and develops incoherent speech. He is happy in his mood, and has an exaggerated sense of well-being and power. He is very emotional. He begins to laugh, sing or shout, and then all of a sudden begins to weep or cry or gets angry. He gets violently excited, and has a tendency to tear or destroy his clothes, bedding or furniture. He is fantastic in his dress, and indecent in manners and talk, using obscene and profane language. He is dirty in his habits, and may defile his body and room with urine and fæces.

Owing to the flight of ideas the patient drops letters, omits words, phrases or even sentences, and is unable to keep up the chain of ordered reason, when he is writing a letter or is engaged in conversation.

The memory is, as a rule, good, but in severe forms of mental excitement there may be a certain clouding of consciousness with disorientation and great impulsiveness. At these times hallucinations of a visual and auditory nature are usually present, and are often associated with delusions. The delusions are usually of a grandiose type, in which the patient imagines that he possesses great wealth and power, or that he is the ruler of an extensive empire. These may be followed by delusions of persecution, when he may commit suicide or murder under the false belief of being persecuted or poisoned by others. It is, therefore, necessary that such a patient should be kept in restraint, so that he may not hurt himself or others. Not infrequently he becomes much more violent if any attempt is made to keep him under restraint.

The chief peculiarity of this disease is that the patient can continue to be boisterous and violent for days and nights without experiencing any sense of fatigue.

The acute form of mania may last for days, weeks and months. It may rarely last for years. Sometimes, the symptoms may subside, followed by a period of quiescence, called a *lucid interval.* The symptoms may again recur at a later period without any warning.

The acute symptoms of excitement often subside, and are followed by a stage of exhaustion, when the limbs are still and flaccid, and the patient sinks into a state of stupor. This stage lasts one to three weeks, after which recovery occurs. A few cases may pass into a state of chronic mania.

Chronic Mania.—This resembles acute mania, but the symptoms are less marked. It is characterized by incoherence, hallucinations and delusions, with occasional attacks of acute excitement. Each of these attacks leaves the patient weakminded. The memory is slowly affected, and the patient passes into a state of dementia, from which recovery never occurs.

Melancholia.—This form of insanity is characterized by difficulty of thinking, mental depression and inhibition of motor impulses. It affects women more than men, especially in early and advanced life. It may be described under three headings: simple melancholia, acute melancholia and chronic melancholia.

Simple Melancholia.—This is the mildest of the three forms of the disease, and is spoken of as simple retardation. It is characterized by mental depression without hallucinations or delusions. It is associated with apprehension of evil, loss of appetite, constipation, and sleeplessness, especially towards early morning. The face has an anxious expression, the forehead is wrinkled and the eyes are dull. There is a lack of interest in the surroundings with inability to attend to daily pursuits of life. Speech is slow and in whispers, and answers are given in monosyllables with great difficulty. There is a fear that the natural affection of relatives is lost. There is also a tendency to commit suicide. The thought processes are retarded, but there is no disorientation or clouding of consciousness, and memory and intellect are good.

Acute Melancholia.—In this form the three chief symptoms of melancholia are well marked. The onset is usually gradual, preceded by a

prodromal stage lasting one to three weeks. During this stage there are complaints of persistent headache, insomnia, gastric disturbances and irritability of temper, which are likely to be confused with neurasthenia or hypochondriasis. According to Overbeck-Wright the chances of recovery and the avoidance of the acute attack of the disease are very great indeed, if this prodromal stage be recognised, and the patient be promptly put under proper treatment.[1]

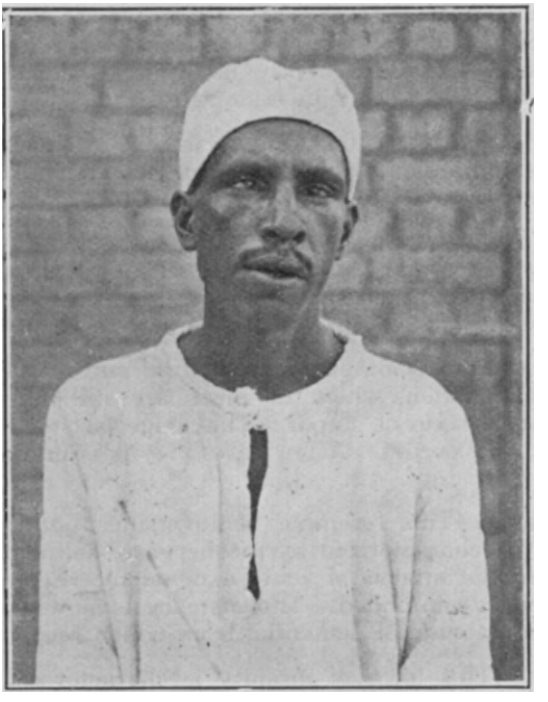


Fig. 121.—Case of Melancholia: Has a sad facial expression, is depressed and occasionally weeps without reason, has irregular sleep, does not reply to questions properly, and sometimes does not take food for a week. (*Dr. Benarsi Das*' case).

The physical condition of acute melancholia is manifested by marked anæmia and progressive loss of weight. The tongue is dry and coated with a thick white or brown fur, and appetite is lost owing to the marked deficiency in the secretion of the gastric juices, especially pepsin. The bowels are constipated owing to deficiency of the intestinal juices. The pupils are frequently dilated. The pulse is rapid, weak and irregular. The skin is dry owing to the diminution of perspiration. The hands and feet are blue and cold due to feeble circulation. The respirations are shallow, but normal in frequency. The temperature is usually sub-normal, but is often slightly raised in the evenings. The urine is diminished in quantity, and is passed at long intervals. It may even be

1. *Lunacy in India*, 1921, *p.* 218.

passed only once in twenty-four hours. In males impotence is usually observed. In females menstruation is generally absent, but reappears when recovery occurs, or when the disease becomes chronic.

The superficial and deep reflexes are often found exaggerated. Muscular movements are slow and weak, the larger proximal joints are rigid and the powers of fine inco-ordination are impaired. Sensation is, as a rule, normal, though sensitiveness to noise is a marked feature.

The mental symptoms generally appear along with the physical symptoms. Perception is normal, orientation is usually quite correct, and the memory and the intellectual faculties are well preserved, but volitional attention is generally poor and defective. There is paralysis of emotional reaction. Good or bad news or even a joke does not affect the patient, who feels gloomy and miserable, and experiences psychic pain. He has lost the social instinct. He sits apart, refuses to mix with his neighbours, or to take part in out-door games or social festivities.

Hallucinations and delusions are usually present. The hallucinations are often of an auditory type, in which the patient imagines that he hears voices accusing him of various misdeeds or threatening him of punishment. The delusions are generally of a hypochondriacal nature. The patient believes that he suffers from some incurable disease, *e.g.*, closure of the œsophagus, gangrene of the intestines or wasting of the brain, and that he will die a miserable death. The delusions may also be of the religious or persecutal character. The patient believes that he has committed the unpardonable sin against God, or that his food is being poisoned by some persons conspirnig to kill him.

Suicidal tendencies are common, though the patient may develop homicidal tendency, and may kill his wife and children to save them from the supposed utter ruin, or may kill some person, whom he believes to be giving him and his family all the imaginable trouble of the world.

Sometimes, the patient is afraid of some impending disaster, and he is so much agitated or excited in his anxiety, that he keeps on moving incessantly, wringing his hands, rocking to and fro and bemoaning his piteous plight. Such a patient often resists being fed, dressed or washed. He is unmindful of personal cleanliness, and passes urine and fæces in his garments.

On other occasions the patient passes, as it were, into a stuporous condition. He is apathetic, and sits silent and motionless in the same fixed attitude for a long time. He has to be spoon or tube fed, and his bladder and bowels have to be attended to.

Acute melancholia may alternate with an attack of mania with a lucid interval intervening between the two. This alternating form of the disease is known as circular insanity or *folie circulaire.*

An attack of acute melancholia, on an average, lasts from six to eight months and ends in recovery. If the attack is not followed by recovery within a year, it usually passes into a chronic condition. Death may occur in the acute stage, when the patient passes into a typhoid state.

Chronic Melancholia.—This form results from the acute form, and is characterized by some improvement in the physical signs but not in the mental symptoms. The patient becomes fat and increases in weight. His digestive powers also improve, and the bowels open regularly. The patient, however, remains persistently depressed, and suffers from hallucinations and delusions.

Ordinarily there is no recovery from the chronic form, but Stoddart has seen cases of recovery. One of his female patients recovered after eighteen years' duration, a male patient of his recovered from a previous attack of thirty-five years, and another male patient recovered from a previous attack of seven years.[1]

Delusional Insanity (Paranoia).—This is a form of insanity, which is characterized by fixed and systematized delusions, as also by hallucinations of various characters. It is called partial insanity by lawyers, and appeals to their legal mind, inasmuch as they believe that a delusion must be present to constitute insanity. The medical man, however, thinks that a delusion is merely a symptom, but not an essential element, of insanity.

Delusional insanity is mostly a disease of adults, affecting both the sexes equally. It exists in two stages: acute and chronic.

Acute Stage.—In the acute stage the disease commences with digestive disturbances and a rise of temperature, accompanied by melancholic depression and confusion of ideas. These are followed by hallucinations of hearing and delusions of persecution. The patient imagines voices dinning into his ears through the walls of a room. He becomes forgetful, loses the power of self-control, and is apt to commit some crime under a sudden uncontrollable impulse. Insomnia is a constant symptom throughout the disease. The patient may appear to have recovered, when a relapse may occur, or the acute condition may pass into the chronic stage.

Chronic Stage.—Suspiciousness is the characteristic symptom of this stage, in which the physical symptoms are also more prominent. The chief physical symptoms are a furred tongue, foul breath, irregular temperature, disordered nutrition and anæmia. Sleeplessness is a general complaint, which results from sensory disturbances producing impressions of electric shocks.

Auditory hallucinations, which occur very early in this condition, are first supposed to be sounds or noises in the ears, but are, afterwards, changed into abuses and insults. At first the patient is able to dismiss them by reasoning faculty. When the judging power fails, these hallucinations give rise to systematized delusions, which, he is, at times, able to defend with logical arguments. These delusions are, at first, indefinite, but gradually they become fixed and definite so as to lead the patient to believe that he is persecuted by some unknown person or some superhuman agency. The patient then develops hallucinations of taste and smell, so that he believes that his food is poisoned, or that some noxious gases are blown into his room. Disturbances of general sensation give

1. *Mind and its Disorder, Ed. V, p. 276.*

rise to hallucinations, which are attributed to the effects of hypnotism, magnetism, electricity, wireless telegraphy, etc. The patient gets very irritated and excited owing to these painful and disagreeable hallucinations and delusions.

In almost all cases the delusions of an exalted type referring to grandeur, power and wealth are seen, and the patient generally conducts himself in a haughty and overbearing manner. With the lapse of time the hallucinations of a grandiose character become less marked, and the patient becomes listless, apathetic and passes into a condition of melancholic depression. At this stage the patient requires to be carefully watched, lest he commit suicide.

During the chronic stage the patient usually retains his memory and self-control. He talks sensibly, and does not show any sign of insanity, until the conversation is directed to the particular type of delusion from which he is suffering. It is, however, always safe to keep such a patient under restraint, for he is often a source of danger to himself as well as to others. There is no motive or forethought for criminal offences, but sometimes premeditation and elaborate arrangement precede a criminal assault.

Exhaustion Psychoses.—These disorders result from fatigue and exhaustion of the nervous system at the late adult or senile period of life in men, but at an earlier age in women owing to prolonged lactation, especially in India.

Symptoms—Physical.—The onset is, as a rule, slow and insidious. Insomnia is a first symptom which is generally a disturbing factor in nerve exhaustion or neurasthenia. Sleep is either absent or is unrefreshing, disturbed and accompanied by nightmare. Hearing becomes so very acute that the slightest noise in a room, even the tickling of a clock or palpitation of the heart is enough to startle the patient from sleep and cause much annoyance. The other symptoms are loss of physical activity and vigour, dyspepsia, constipation, malnutrition, headache, giddiness, irritability, nervousness and anæmia. The pupils are widely dilated, but the visual field is not diminished and there is no abnormality of optical conditions. Circulation is feeble; the extremities are, therefore, cold and œdematous. Palpitation is a common symptom. The skin perspires readily. The temperature is generally subnormal. The superficial and deep reflexes are well-marked, and the muscles are in an irritable condition and react readily to the slightest external stimulus. The urine is normal.

Mental.—Irritability and loss of self-control are prominent mental symptoms. The power of attention is weakened, and memory either wanders or is incoherent in its associations. The power of thinking is lost. Any attempt of thought fags the brain. However, if persevered in it leads to mental confusion and depression. The speech is hesitating but not incoherent. Sometimes, the patient passes into a semi-stuporous condition, when he has a sad vacant look and sits listlessly in one position for a long time. He cannot be roused to answer questions.

Visual hallucinations very often occur, and the patient is, at times, troubled by painful obsessions of fear. For instance, the patient is afraid

of an empty space (*agoraphobia*) and cannot cross a street, especially if it is open and unoccupied. Similarly, some patients have a dread of being shut up in a closed space or apartment (*claustrophobia*). Some of these obsessions are apt to be associated with visceral sensations of a more or less oppressive nature. Thus, the patient may complain of an oppression at the chest, constriction of the heart, or may have a disposition to pass water or motion.[1] These obsessions may also lead to impulsive actions, which the patient is unable to control, though he is conscious of their evil effects. He may, later on, develop delusions of persecution or poisoning and consequently may develop suicidal tendencies.

Katatonia (Catatonia).—This is a disease which is associated with adolescence and occurs among Europeans, Anglo-Indians and Indians. The conditions which tend to impair the vitality and lower the resistance to infective diseases render the person liable to this disease. Heredity also plays an important part. For convenience of description the disease may be divided into four stages :

1. The prodromatous stage.
2. The acute stage of onset.
3. The stage of stupor.
4. The stage of excitement.

1. **The Prodromatous Stage.**—The onset is, as a rule, slow with gradual loss of energy and thinning of the body from malnutrition. The first mental symptoms are the development of hallucinations of an auditory nature followed by dreadful obsessions, delusions and loss of self-control.

2. **The Acute Stage of Onset.**—The physical symptoms of this stage are loss of appetite, nausea and vomiting. The heart is irritable, beating rapidly and irregularly. The arterial tension increases as the physical complaints become more acute. The skin is moist and greasy from frequent profuse sweats, and is often affected by pustular eruptions. Sleeplessness is almost a constant symptom. The senses of taste and smell are commonly affected, but not those of sight and hearing, though hallucinations of a visual and auditory nature are often present in this stage. The sensations of touch, pain and warmth are generally diminished. The pupils are sluggish and dilated. The visceral reflexes not being under control, the patient, as a rule, passes urine and fæces involuntarily ; hence he has to be watched constantly. The superficial and deep reflexes are markedly increased. The voluntary muscles are usually rigid and stiff. This condition may last from a few minutes to a few hours.

The mental condition is chiefly one of confusion and restlessness. The patient is largely affected by paroxysmal hallucinations of an auditory nature, characterized by dreadful persecutions from imaginary enemies, to avoid whom he may try to conceal himself or may commit suicide. During the periods when the patient is not disturbed by these hallucinations, he may close his eyes and lie quiet, listless and apathetic for hours without taking heed of his surroundings ; or he may appear sane, though confusion of thought, loss of memory and want of the power of fixing attention are generally present.

1. *Albutt, System of Med., Vol. VIII, p.* 749.

This stage lasts for four to six weeks, and is then followed by an attack of fever or increased leucocytosis which brings on the third stage. The acute stage may, however, end in death from exhaustion or from some toxæmic condition.

3. **The Stage of Stupor.**—During this stage the physical symptoms are the persistence of gastric disturbances with obstinate constipation. Sleep is excessive and causes the patient to be drowsy and heavy. The arterial tension is low. Circulation is slow and feeble; hence the extremities are cold, blue and frequently œdematous. There is an offensive odour from the skin, which is greasy. The muscles are very rigid, and the patient may assume uncomfortable attitudes. Strong resistance is encountered when an attempt is made to extend the contracted limbs. If a limb is, however, placed in a forced position, it may remain in that condition for an indefinite period.

Mentally the patient passes into a semi-conscious, stuporous condition. He sits or stands and does not show any signs of spontaneous movement. An attempt at feeding or dressing him is very strongly resisted by the patient. He does not even answer questions put to him. He generally assumes a state of mutism but may, all of a sudden, start repeating automatically some inarticulate syllable, word, number, phrase or sentence for hours (*verbigeration*). The patient sometimes repeats the words spoken to him or imitates the tone of one whom he has heard speaking (*echolalia*). Hallucinations of sight and hearing develop, and give rise to delusions of a varied nature. The patient may suddenly be attacked by a maniacal fit, when he may have a suicidal or homicidal tendency. During this period it is very essential to watch him closely.

The duration of the stage is uncertain; it may be from a few weeks to a few years. Recovery occurs in a very small number of cases.

4. **The Stage of Excitement.**—The stuporous condition is followed by the stage of excitement. In many cases there is an apparent recovery lasting for two to three years and then there is a relapse. Attacks of excitement generally come on in paroxysms. Durnig the periods of quiescence the patient appears to be sane. The patient takes his food freely. The general health improves and he gains in weight. Rhythmical stereotyped movements of the hands and verbigeration may be present. Confusion of thought is less marked, though memory and attentiveness are weakened. The patient becomes definitely weak-minded and passes steadily into a state of dementia. Hallucinations and delusions are present and generally affect the conduct of the patient, who should always be carefully watched.

Hebephrenia.—This is a disease, which occurs usually in the early adolescent perlod of life, and affects females more than males. Owing to marked leucocytosis, Dr. Bruce[1] thinks that the disease is due to toxæmia brought on by some bacterial infection.

Symptoms.—The onset is so slow and insidious, that the changes in the character and temperament of the patient are not noticed for a very

1. *Overbeck-Wright, Lunacy in India, p.* 262.

long time. The characteristic feature of this disease is an arrest of physical development. The patient is inactive, lethargic, and sits idle the whole day. He shuns society, avoids friends and, sometimes, wanders about aimlessly in streets. He is untidy and careless of his dress. He is very often cruel, mischievous and addicted to self-abuse. Obscene language and indecent actions are marked features of this disease. The patient is full of emotions, changing from time to time. Once he may be mute and depressive ; at another time he may become verbose, irritable and excited. This condition passes on steadily to dementia. The power of concentration and attention is lost. Memory is impaired. Visual and auditory hallucinations may be present and may lead to obsessions, impulsive acts and general restlessness.

INSANITY ASSOCIATED WITH NERVOUS DISEASES

There are certain forms of insanity, which are associated with nervous diseases. The chief of these are general paralysis of the insane and epileptic insanity, which will be described here.

General Paralysis of the Insane (Dementia Paralytica).—This is a chronic progressive disease, which is characterized by physical and mental symptoms terminating in paralysis and dementia due to degeneration of the brain and central nervous system.

It affects men more than women, and occurs in the prime of life between thirty and forty-five years of age, but it may occur in childhood or old age. Heredity plays a very minor part in the causation of this disease. Acquired or congenital syphilis is the chief factor causing this disease.

Symptoms.—In this disease there is always a prodromal stage lasting for months or years. During this period forgetfulness, irritability, restlessness, overfriendliness, intemperance in drinks and deterioration of the moral senses, are usually the first symptoms, which attract the attention of the friends and relatives of the individual. At this stage the feelings of self-satisfaction and expansiveness are the characteristic features of the disease. These are followed by ideas of grandeur which assume the nature of delusions of an exalted kind. The patient believes that he is the most powerful, and possesses enormous wealth. He squanders his money, undertakes business of a speculative nature, or orders the purchase of a large number of useless articles. At times, he steals articles which are of no use to him, or owing to perversion of the moral sense he may commit an indecent assault on a woman in public.

In place of excitability and a general sense of exhilaration, gloom, despondency, loss of energy and mental hypochondriasis may be the first mental symptoms to announce the commencement of the disease.

The physical symptoms usually follow the mental symptoms but they may precede or accompany them. Sometimes, the disease is ushered in by apoplectic or epileptic convulsive seizures or a temporary attack of aphasia lasting for a few hours or days.

The first physical symptoms that are generally observed are the tremors of the tongue and lips causing an embarrassed speech as if the

patient were intoxicated. The tremors slowly involve the muscles of the face causing loss of expression, and later the muscles of the hands, so that the handwriting becomes shaky and illegible, and the last letters of words are omitted. The finer and rhythmical movements of the fingers are also not properly executed. The pupils are frequently unequal, and sometimes small and contracted. The deep reflexes are exaggerated. Headache and neuralgic pains are often complained of. Insomnia is frequently a constant symptom, though drowsiness may be present in a few cases. Owing to the weakness and inco-ordination of the muscles of the legs the gait becomes slow and tottering. The temperature rises generally in the evening up to 101° or 102° F.

By the time that these symptoms have become prominent, the mental symptoms have become more pronounced. There is loss of memory with marked impairment of the intellectual faculties. The patient is no more restless or energetic, nor does he respond to external stimuli owing to the diminished activity of the general and special senses. Delusions of an exalted kind may persist in a few cases, but they do not generally affect the conduct of the patient, who is now quite manageable. He becomes listless, apathetic and careless about his dress and appearances. The patient passes into a state of complete dementia and, owing to the complete extinction of the mental faculties, he is unmindful of his surroundings, and leads more or less a vegetable existence without any interest in life. Complete paralysis supervenes, so that the patient is bedridden and passes urine and fæces involuntarily.

At times, there are spasmodic attacks of violent mania leading to destructive or homicidal tendencies. These are followed by remissions lasting from a few months to two or three years. Death occurs from exhaustion, some intercurrent disease or from blood poisoning. There is difficulty in swallowing and the patient may die by being choked.

Epileptic Insanity.—Epilepsy usually occurs from early infancy, though it may occur at any period of life. Individuals, who have had epileptic fits for years, do not necessarily show any mental aberration, but a great majority of them suffer from mental deterioration. Religiosity is a marked feature in the commencement, but the feeling is only emotional and perverted. Such patients are peevish, impulsive and suspicious, and are easily provoked to anger on the slightest cause.

The disease is generally characterized by short transitory fits of uncontrollable mania followed by complete recovery. The attacks, however, become more and more frequent. Lastly, there is general impairment of the mental faculties with loss of memory and self-control. At the same time hallucinations of sight and hearing occur, and are followed by delusions of a persecuting nature.

Epileptic insane persons are deprived of all moral sensibility, are given to the lowest forms of vice and sexual excesses, and are, sometimes, dangerous to themselves as well as to others. In many long standing cases there is usually feeble-mindedness leading to progressive dementia of a most degraded character.

True epileptic insanity is that which is associated with epileptic fits. This may occur before or after the fits, or may replace them, and is known

as pre-epileptic insanity, post-epileptic insanity and masked or psychic insanity.

1. **Pre-Epileptic Insanity.**—This is very common and may replace the epileptic aura, lasting in some cases for hours or even days. It is characterized by violent fits of maniacal excitement or by depressions, fussiness, suspiciousness and general malaise. Hallucinations of various kinds are experienced and, owing to delusions, the patient may commit violent assaults, or may bring false charges against innocent persons. Sometimes, the patient may refuse to take any food.

II. **Post-Epileptic Insanity.**—In this condition the stupor following the epileptic fit is replaced by automatic acts of which the patient has no recollections. The patient is confused, fails to recognize his own relatives, and wanders aimlessly about. He is terrified by visual and auditory hallucinations of a religious character and delusions of persecution, and consequently may commit crimes of a horrible nature, such as, thefts, incendiarism, sexual assaults and brutal murders. Such crimes are motiveless and unpremeditated. The patient never attempts to conceal them at the time of perpetration but, on regaining consciousness, may try to conceal them out of fear.

In a murder trial at Lancashire Assizes on October 30, 1928, evidence was given by mental specialists which suggested that the accused had killed his wife in a condition of post-epileptic automatism. He fired shots at his wife and killed her outright. There was evidence that at the time his manner was calm and deliberate and he appeared to know perfectly well what he was doing. In 1922, he had suffered from petit mal and he was suffering from a condition of post-epileptic automatism in which a man might do things about which he seemed quite able to reason, though entirely unconscious of what he had done. The jury found that the accused was guilty of the act charged but insane at the time.[1]

A case is recorded in which an accused murdered his mother and wounded his step-father in a fit of epilepsy without any apparent cause and then hid himself in a ravine. The medical evidence showed that the accused was subject to epileptic fits and he used to be completely unconscious during such time. It was, therefore, held that the evidence of this unprovoked attack upon his mother and step-father with whom he had no quarrel or trouble, and his hiding in the ravine were certainly consistent with the attack upon the deceased having taken place during or whilst recovering from an epileptic fit and that any other theory of the events was really untenable. It was found that the accused was guilty of the acts charged but not so as to be responsible in law for his actions. He was detained during His Majesty's pleasure.[1]

In some cases violent gesticulations or fits of mania may develop after sleep usually following the epileptic seizures. This condition is quite transitory, lasting for a few hours. In a very small number of cases mental depression may follow the epileptic fits, and may be accompanied by delusions of persecution leading to suicidal or homicidal tendencies.

III. **Masked or Psychic Epilepsy.**—In this variety the epileptic seizure is replaced by the transitory loss of consciousness and maniacal excitement. It is interesting from a medico-legal point of view, for a patient suffering from this condition is apt to commit an offence without

1. *Lancet, Nov.* 10, 1928, *p.* 990.
2. *Rangoon H. C., Cr. App. No.* 1397 *of* 1936, *NGA ANT BWE* v. *K. E., Cr. Law Jour.,* 1937, *Vol.* 38, *p.* 667.

any consciousness or premeditation and without any subsequent remembrance of the act.

Lastly, it should be remembered that epilepsy in childhood may arrest the growth of the mental faculties, and lead to idiocy and imbecility. Epileptic idiots are, as a rule, very impulsive and irritable, and are apt to injure their playmates, if not carefully watched.

DIAGNOSIS OF INSANITY

Sometimes, it is very difficult to form a correct diagnosis as to whether an individual is sane or not, especially when he has no permanent delusion, and when he is just on the border line between sanity and insanity. Under such circumstances it is always advisable to note carefully the following points before a definite opinion is given :—

1. **Family History.**—Insanity being mostly hereditary, it is very important to enquire into the mental condition of the patient's parents, uncles, grand-parents, brothers, sisters and other relatives as to whether any of them ever showed mental excitement or depression, or mental weakness, or suffered from nervous diseases, such as chorea, epilepsy, etc. It is also necessary to find out if any of them committed suicide, or were attacked by cerebral affections, gout, rheumatism or syphilis.

2. **Personal History.**—While listening to the history of the patient, the medical man should always try to be sympathetic, so as to win his confidence. The history should be as thorough and complete as possible, noting all the characteristic details from childhood likely to give a clue to the disease. Questions should be asked about his personal habits with reference to the excessive use of any intoxicating drugs, such as cannabis indica, alcohol, cocaine or opium, sexual excess, masturbation, any morbid propensity at the time of puberty, occupation, mental strain or shock, injury to the head or any brain disease, and chorea, epilepsy, convulsions, or any other nervous affections. It should be ascertained from his relatives and friends, if they noticed of late any change in his conduct and behaviour towards them, if he was cleanly in his habits or filthy and disgusting, and if he was restless and passed sleepless nights, or if he looked excitable or depressed at times. Lastly, it should be found out if this was the first attack, or there had been any attack previous to it.

3. **Physical Examination.**—The patient's manner of dressing and walking, as well as his bearing and gestures, should be carefully noted, when he comes to the physician. The presence of deformities and malformations in the head or body, as also the power of speech and articulation, should be observed. The pulse and temperature should be taken, as both might increase in insanity. The tongue should be examined to find out if it is foul or furred due to constipation. All the organs should be carefully examined. The skin would be dry, mottled and wrinkled, and the hands and feet would be moist with sweat.

4. **Mental Condition.**—The mental capacity should be found out by first testing his memory, and then the power of his reasoning and sound judgment.

While testing the memory the patient should be asked to give the dates of common incidents, that occurred in his family, or to recite the names of his relatives, or the days of the week, or answer such other simple questions. The questions put to him should not be too complex or difficult to be easily answered by an average man of his culture and education.

The power of his reasoning and sound judgment should be detected by discussing with him on various subjects. During discussion an attempt should be made to find out a delusion. An insane person tries to conceal his delusion ; hence it may be necessary to watch him for days before his condition can be certified.

Lastly, handwriting will show the mental confusion, the misspelling, the omission of letters or phrases and the muscular tremor, if an educated insane person is asked to write.

FEIGNED INSANITY

There is always some motive for feigning insanity. For instance, a criminal pretends insanity to escape punishment for his offence, especially when he is placed on trial. In civil practice an individual feigns insanity to try and avoid the results of business transactions or deeds, which he may have executed. Policemen, soldiers and sailors do so, when they wish to leave the service and are not allowed to do so, or when they know that they are likely to be punished very severely for some gross neglect of duty.

The detection of feigned insanity is one of the responsible duties of a medical officer. Ordinarily it is easy to detect the fraud but, at times, it becomes very difficult, when the individual should be detained under observation, before a definite opinion is given. It should be remembered that such a person cannot be kept under observation for more than ten days in the first instance but, with the permission of the Magistrate, he may be detained for further periods of ten days up to a maximum of thirty days.[1] During this period the medical officer has to watch him and make a careful note of all the symptoms exhibited by him, and has also to visit him daily at unexpected hours without the knowledge of the patient.

The following are the distinguishing features between feigned and true insanity : —

1. Feigned insanity always comes on suddenly, and not without some motive. True insanity may rarely develop all of a sudden but, in that case, some predisposing or exciting cause will be evident, if a careful history of the case is taken.

2. In feigned insanity there is no peculiarity in the facial expression, which is generally observed in the fully developed forms of insanity.

3. In feigned insanity the individual tries to pass off as insane by putting forward incoherent maniacal symptoms, especially when he knows

1. *Sec.* 16, *Act IV*, 1912 (*The Indian Lunacy Act*, 1912 *as modified upto the* 1*st October*, 1931).

that he is under observation. There is a total remission of all the symptoms, when he thinks that he is alone and unobserved.

4. In feigned insanity the symptoms are not uniform, indicating any particular type of insanity. Malingerers usually mix up the symptoms of one or two distinct types of insanity. Such a condition, may, however, exist in true insanity.

5. In feigned insanity violent exertion occasioned by imitating maniacal frenzy (which is generally imitated by impostors) will bring on exhaustion, perspiration and sleep, but a really insane person can stand such exertion for many days without sleep and fatigue.

6. A malingerer is not, as a rule, dirty and filthy in his habits. He may smear his room with fæces and other filth, if he has seen a true lunatic doing so. He will, however, keep a clear space for sleeping and will spare his person.

7. The dry, harsh skin and lips, the furred tongue, constipation, want of appetite and insomnia are, very often, physical manifestations of true insanity. These are, as a rule, absent in feigned insanity, as they cannot possibly be imitated by a malingerer.

RESTRAINT OF THE INSANE

If an insane person is dangerous to himself or to others, or if he is likely to injure or squander his property or that of others, he can be lawfully kept under immediate restraint under the personal care of attendants, or admitted into a mental hospital.

Immediate Restraint.—Immediate restraint under the personal care of attendants may be imposed either by the consent of a lawful guardian of the insane person, or without his consent, if there is no time to obtain it without fear of injury to his person or to the persons of others ; but the restraint should last so long as the danger exists. Such a restraint can also be imposed on persons suffering from delirium due to disease, or from delirium tremens. In this case the restraint must cease with the subsidence of the symptoms.

1. **Reception direct into a Mental Hospital.**—Under section 4 (1) of the Indian Lunacy Act, 1912 (Act IV, 1912), as modified upto the first October, 1931, any person in charge of a mental hospital may, with the consent of two of the visitors of the mental hospital on a written application from the intending boarder, receive and lodge as a boarder in such mental hospital any person who is desirous of submitting himself to treatment. Such a boarder should not be detained in the mental hospital for more than twenty-four hours after he has given to the person in charge of the mental hospital notice in writing of his desire to leave such mental hospital.

2. **Reception Order on Petition.**—The husband or wife of the alleged lunatic submits a petition for a reception order for his admission into a mental hospital to the Magistrate whithin whose jurisdiction the alleged lunatic ordinarily resides.[1] If there is no husband or wife or

1. *Sections* 5 *and* 6, *The Indian Lunacy Act*, 1912.

the husband or wife is prevented by reason of insanity, absence from India or otherwise from making the presentation, the nearest relative of the alleged lunatic who is not so prevented can make a petition.[1] If the husband or wife or the nearest relative in the absence of the husband or wife is unable to present the petition, any other person can present the petition which must contain a statement of the reasons why it is not so presented, and of the connection of the petitioner with the alleged lunatic, and the circumstances under which he presents the petition. It must be remembered that no person can present a petition unless he has attained the age of majority as determined by the law to which he is subject, and has, within fourteen days before the presentation of the petition, personally seen the lunatic.

The petition must be in the form (Appendix VIII, Form I) prescribed by the Indian Lunacy Act, 1912, as modified upto the first October, 1931, with the statement of prescribed particulars signed and verified by the applicant, and must be supported by two medical certificates (Appendix VIII, Form 3) on separate sheets of paper, one of which must be from a gazetted medical officer or a medical practitioner declared by Government to be a medical officer under Act IV of 1912 and the other from a medical practitioner holding a qualification to practise medicine and surgery registrable in the United Kingdom or declared by Local Government to be a medical practitioner under Act IV of 1912. If either of the medical certificates is signed by any relative, partner or assistant of the lunatic or of the petitioner, this fact should be noted, and where the person signing is a relative the exact manner in which he is related to the lunatic or petitioner should also be mentioned in the petition. Both the medical men should examine the alleged lunatic independently of each other and at different times and each should certify that the individual is " a lunatic and a proper person to be taken charge of, and detained under care and treatment " after he has formed an opinion from a statement given in the certificate of the facts indicating insanity observed by himself at the time of the examination and the facts indicating insanity communicated to him by others.

Every medical certificate made under the Indian Lunacy Act is a legal document, giving evidence of the facts therein appearing and of the judgment therein stated to have been formed by the person certifying on such facts, as if the matters therein appearing had been verified on oath.[2]

To avoid legal action for wrongful certification the medical practitioner must be very careful in giving a certificate of lunacy for admission into a mental hospital, as he is responsible for having an alleged lunatic sent to a mental hospital, but his responsibility ceases on the latter's admission into the mental hospital. It must be remembered that a reception order required to be founded on a medical certificate shall not be made unless the person who signs the medical certificate, or, where two certificates are required, each person who signs a certificate, has personally examined the alleged lunatic, in the case of an order upon petition, not more than seven clear days before the date of the presentation of the petition, and,

1. *Section 2, The Indian Lunacy (Amendment) Act, 1926 (Act No. V of 1926).*
2. *Section 18 (3).*

in all other cases, not more than seven clear days before the date of the order.[1] The Magistrate may in his discretion extend this period within which the alleged lunatic must have been medically examined.[2]

On receipt of the petition, the Magistrate holds inquiry in private, and personally examines the alleged lunatic, unless for reasons recorded in writing he thinks it unnecessary or inexpedient to do so. If he is satisfied, he forthwith issues a reception order (Appendix VIII, Form 2). If he is not so satisfied, he fixes a day for the consideration of the petition, due notice being given to the petitioner, and to any other person to whom in the opinion of the Magistrate notice should be given, and he makes such further inquiries concerning the alleged lunatic as he thinks fit. In the meantime he may pass necessary orders for the safe custody of the alleged lunatic until the inquiry is concluded.

On considering the petition, the Magistrate may grant a reception order, which holds good for seven days, or he may refuse the petition, when he has to give the reasons in writing, a copy of which has to be supplied to the petitioner.

No reception order can be made under petition, except in the case of a lunatic who is dangerous, and unfit to be at large, unless the Magistrate is satisfied that the person in charge of a mental hospital is willing to receive the lunatic, and the petitioner or some other person engages in writing to the satisfaction of the Magistrate to pay the cost of maintenance of the lunatic (Section 11, The Indian Lunacy Act, 1912, as modified up to the first October, 1931).

3. **Reception Orders otherwise than on Petition.**—When any European subject to the provisions of the Army Act or the Air Force Act has been declared a lunatic in accordance with the provisions of the military or air force regulations in force for the time being, he may be admitted into a mental hospital which has been duly authorized for the purpose by the Governor-General in Council, on a reception order signed by an administrative medical officer, if he thinks that the admission of the said lunatic into the mental hospital is necessary (Section 12, The Indian Lunacy Act, 1912, as modified up to the 1st October, 1931).

If an Indian soldier has been declared insane by a medical board, he has to be discharged from the Army, and handed over to his relatives. If the relatives be not at hand, or if the Indian soldier, by reason of insanity, be dangerous to himself or to others, he should be handed over to the civil authorities for disposal as a civilian.

Under section 13 (1) of the Indian Lunacy Act, every officer in charge of a police-station may arrest or cause to be arrested any person, who he has reason to believe, is a wandering or dangerous lunatic within the limits of his station. Any person so arrested must be taken forthwith before a Magistrate. The Magistrate shall examine such person, and if he thinks that there are grounds for proceeding further, shall order him to be examined by a medical officer (usually a civil surgeon), and may make such other inquiries as he thinks fit. If the Magistrate is satisfied

1. *The Indian Lunacy Act, Section* 19 (1).
2. *Ibid., Section* 11-B (2) (*d*).

that such person is a lunatic and a proper person to be detained, he may, on receipt of a certificate of lunacy from the medical officer, issue a reception order for the admission of such lunatic into a mental hospital. If any friend or relative desires that the lunatic be sent to a licensed mental hospital and engages in writing to the satisfaction of the Magistrate to pay the cost of maintenance of the lunatic in such mental hospital the Magistrate shall, if the person in charge of such mental hospital consents, pass a reception order for the admission of the lunatic into the licensed mental hospital. If any friend or relative of the lunatic enters into a bond with or without sureties for such sum of money as the Magistrate thinks fit, conditioned that such lunatic shall be properly taken care of, and shall be prevented from doing injury to himself or to others, the Magistrate, instead of issuing a reception order, may, if he thinks fit, make him over to the care of such friend or relative (Section 14, The Indian Lunacy Act, 1912).

Under section 13 (2) of the Indian Lunacy Act every officer in charge of a police station who has reason to believe that any person within the limits of his station is deemed to be a lunatic and is not under proper care and control, or is cruelly treated or neglected by any relative or other person having the charge of him, shall immediately report the fact to the Magistrate. It is enacted under section 15 (1), (2) and (3) that if it appears to the Magistrate, on the report of a police-officer or the information of any other person, that any person within the limits of his jurisdiction deemed to be a lunatic is not under proper care and control, or is cruelly treated or neglected by any relative or other person having the charge of him, the Magistrate may cause the alleged lunatic to be produced before him, and summon such relative or other person as has or ought to have the charge of him. If such relative or other person is legally bound to maintain the alleged lunatic, the Magistrate may pass an order for such alleged lunatic being properly cared for and treated, and, if, such relative or other person wilfully neglects to comply with the said order, the Magistrate may sentence him to imprisonment for a term which may extend to one month. If there is no person legally bound to maintain the alleged lunatic, or if the Magistrate thinks fit to do so, he may, on being satisfied that the person deemed to be a lunatic is a lunatic and a proper person to be detained under care and treatment and on receipt of a necessary certificate from a medical officer may make a reception order for the admission of such lunatic into a mental hospital.

When a reception order has been passed, the Magistrate may, for reasons to be recorded in writing, direct that the lunatic, pending his removal to a mental hospital be detained in suitable custody in such place as the Magistrate thinks fit.

Under section 17 of the Indian Lunacy Act the Commissioner of Police may, in place of the Magistrate, hold inquiries in cases of alleged lunacy, and issue a reception order in the Presidency-towns or Rangoon; and an officer of the police force not below the rank of an inspector in any of the Presidency-towns may perform all duties which an officer in charge of a police station is authorized or required to perform.

4. **Reception after Judicial Inquisition.**—Under Section 25 of the Indian Lunacy Act, 1912, a lunatic so found by judicial inquisition may be admitted into a mental hospital—

(*a*) in the case of a judicial inquisition under Chapter IV of the Indian Lunacy Act (Act IV of 1912), on an order made by or under the authority of the High Court;

(*b*) in the case of a judicial inquisition under Chapter V of the said Act on an order made by the District Court.

In such cases the High Court or the District Court, as the case may be, shall, on the application of the person in charge of the mental hospital pass an order for the payment of the cost of maintenance of the lunatic in the mental hospital, and may from time to time direct that any sum of money payable under such order shall be recovered from the estate of the lunatic or of any person legally bound to maintain him. If at any time the Court is satisfied that the lunatic has not sufficient property, and that the person legally bound to maintain such lunatic has no means to pay such cost, the Court shall certify the same instead of passing the order for the payment of the cost (Section 26, The Indian Lunacy Act, 1912).

5. **Reception of Criminal Lunatics.**—A criminal lunatic is to be admitted into a mental hospital on the order of the presiding officer of a Court in accordance with section 466 or 471 of the Code of Criminal Procedure, 1898 (Appendix VI), or according to section 30 of the Prisoners Act of 1900, or according to section 103 A of the Indian Army Act, 1911,[1] after the medical officer has certified to his lunacy.

Sections 464 to 475 of the Criminal Procedure Code, 1898 (Appendix VI) deal with the criminal lunatics who may be divided into three classes as under:—

1. Those who are unable to stand their trial by reason of being of unsound mind, and incapable of making their defence.

2. Those who committed the crime, but were acquitted on the ground of being of unsound mind at the time of committing the crime.

3. Those who contracted the disease after they were imprisoned in a jail.

When any person is detained in a mental hospital under the provisions of section 466 or 471 of the Code of Criminal Procedure, 1898, or under the provisions of section 103-A of the Indian Army Act, 1911,[1] the visitors of the mental hospital appointed by the Local Government or any two of them are authorized to visit him to ascertain the state of his mind; and they must visit him once at least in every six months so as to enable them to make a special report as to the state of his mind to the authority under whose order he is detained. When a criminal lunatic is detained in a jail, the Inspector-General of Prisons is authorized to pay such visits and make such a report (Section 30, The Indian Lunacy Act, 1912).

Discharge of Lunatics from a Mental Hospital.—The provision of the discharge of lunatics from a mental hospital is made in sections 31 to 34

1. *The Indian Lunacy Amendment Act*, 1923 (*Act No. XXIII of* 1923).

of the Indian Lunacy Act, 1912. According to these sections three of the visitors of a mental hospital, of whom one must be a medical officer, may, by order in writing, direct the discharge of any person detained in such mental hospital except criminal lunatics and European lunatics subject to the provisions of the Army Act or the Air Force Act. Notice of the discharge should be immediately communicated to the authority under whose orders the person was detained in the mental hospital.

A lunatic detained in a mental hospital under a reception order, made on petition, shall be discharged if the person on whose petition the reception order was made so applies in writing to the person in charge of the mental hospital provided that no lunatic shall be discharged if the officer in charge of the mental hospital certifies in writing that the lunatic is dangerous and unfit to be at large.

A European subject to the provisions of the Army Act or the Air Force Act, and detained in a mental hospital under the orders of a military administrative officer must be detained therein until he is discharged therefrom in accordance with the military or air force regulations in force for the time being, or until the officer making the order applies for his transfer to the military or air force authorities in view to his removal to England. Whenever it appears to the officer in charge of a mental hospital that the discharge of such a person is necessary either on account of his recovery, or for any other purpose, such person must be brought before the visitors of the mental hospital and on the visitors recording their opinion that the discharge should be made, the General or other officer Commanding the division, district, brigade, or force or other officer authorized to order the admission of such persons into a mental hospital shall forthwith direct him to be discharged, and such discharge shall take place in accordance with the military or air force regulations in force for the time being.

When a dangerous and wandering lunatic, or a lunatic cruelly treated or not under proper care and control is detained in a mental hospital and any of his relatives or friends is desirous that he shall be delivered over to his care and custody, he may apply to the authority, under whose order the lunatic is detained, and such authority, if it thinks fit, in consultation with the person in charge of the mental hospital and with the visitors or with one of them being a medical officer, may order the discharage of such lunatic from the mental hospital provided that the relative or friend making the application gives a sufficient undertaking that such lunatic shall be properly taken care of, and shall be prevented from doing injury to himself or to others.

When a person is admitted into a mental hospital on a Magistrate's reception order, and is subsequently found on a judicial inquisition to be of sound mind and capable of managing himself and his affairs, the person in charge of the mental hospital must forthwith, on the production of a certified copy of such finding, discharge the alleged lunatic from the mental hospital.

Escape and Recapture of Lunatics.—A lunatic escaping from a mental hospital may be retaken by any police-officer or by the person in charge of the mental hospital or any officer or servant belonging thereto, or any

other person authorized in that behalf by the said person in charge and conveyed to and readmitted into such mental hospital. But, except in the case of a criminal lunatic or a European lunatic subject to the provisions of the Army Act or the Air Force Act the power to retake such escaped lunatic is exercisable only for a period of one month from the date of his escape (Section 36, Act IV, 1912).

Illegal Detention.—Section 93 of the Indian Lunacy Act of 1912 provides that any unauthorized person who receives or detains a lunatic or alleged lunatic in a mental hospital, or for gain detains two or more lunatics in any place not being a mental hospital, is liable to imprisonment for a term not exceeding two years, to a fine, or to both.

CIVIL RESPONSIBILITY

Management of Property.—Chapters IV and V of the Indian Lunacy Act, 1912 (Act IV of 1912) provide for the legal proceedings to be followed in cases concerning the protection of the person and property of a lunatic. Chapter IV is applicable to those liable to the jurisdiction of the High Courts of the Presidency-towns of Calcutta, Madras and Bombay, and lays down that on the application of any relative of an alleged lunatic, or of the Advocate-General, the Court may direct an inquisition whether the person alleged to be lunatic is of unsound mind and incapable of managing himself and his affairs; the Court may also order inquiries concerning the nature of the property belonging to the alleged lunatic, the persons who are his relatives, the time during which he has been of unsound mind, or such other matters as seem proper. The Court may require the alleged lunatic to attend at some convenient time and place for the purpose of examination, and may authorize any person or persons to have access to the alleged lunatic for the purpose of a personal examination and a report on his mental capacity and condition. But if the alleged lunatic is a female, who cannot appear in public, such order will be regulated by the law and practice for the examination of such persons in other civil cases.

When a medical practitioner is called upon to give his opinion after the examination of the alleged lunatic in such cases, he should not simply mention that the individual is insane, but he should certify that insanity is of such a degree as to render him incapable of managing his own property. He must be very careful in giving his opinion, as an individual may be insane, and yet may be capable of looking after his own property. In a case of doubt it is always safer to give an opinion in favour of *sanity* rather than *insanity*.

If the alleged lunatic is not within the local limits of the jurisdiction of the High Court, and the inquisition cannot conveniently be made, the High Court may direct the inquisition to be made before the District Court within whose local jurisdiction the alleged lunatic may be.

When upon the inquisition it is found that the alleged lunatic is of unsound mind so as to be incapable of managing his affairs, but that he is capable of managing himself and is not dangerous to himself or to others, the Court issues an order for the appointment of a manager to look after his property, and by such order of appointment, or by any

subsequent order, grant such powers to the manager for the management of the estate as may seem necessary and proper to the High Court, provided that he will not, without the previous permission of the Court, mortgage, charge or transfer by sale, gift, exchange or otherwise, any immovable property of the lunatic, or lease any such property for a term exceeding five years. The Court may, if it appears to be just or for the lunatic's benefit, order that any property, movable or immovable, of the lunatic, and whether in possession, reversion, remainder, or contingency, be sold, charged, mortgaged, dealt with or otherwise disposed of as may seem most expedient for the purpose of raising money to be used for all or any of the following purposes : —

1. The payment of the lunatic's debts or engagements ;

2. the discharge of any incumbrance on his property ;

3. the payment of any debt or expenditure incurred for the lunatic's maintenance or otherwise for his benefit ;

4. the payment of or provision for the expenses of his future maintenance and the maintenance of such members of his family as are dependent on him for maintenance, including the expenses of his removal to Europe if necessary, and all the expenses incidental thereto ;

5. the payment of the costs for any judicial inquisition, and of any costs incured by order or under the authority of the Court.

The manager of the lunatic's estate shall, in the name and on behalf of the lunatic, have the power to execute all such conveyances and instruments of transfer relative to any sale, mortgage or other disposition of his estate as the Court may order. If it is subsequently reported to the Court that the unsoundness of mind for which the control of the lunatic's estate was taken away from his hands has ceased, the Court may order a second inquiry, and, on being satisfied that the lunacy has ceased, will order all proceedings in the lunacy to cease or to be set aside on such terms and conditions as may seem fit.

Chapter V is applicable to persons not subject to the jurisdiction of any of the High Courts of the Presidency-towns, and provides that the District Court within whose jurisdiction an alleged lunatic is residing may, upon an application made by any relative of the alleged lunatic, or by any public curator appointed under the Succession (Property Protection) Act, 1841, or by the Government Pleader, or by the District Collector on behalf of the Court of Wards, direct an inquisition for the purpose of ascertaining whether such person is of unsound mind and incapable of managing himself and his affairs. If the alleged lunatic resides at a distance of more than fifty miles from the place where the District Court is held to which the application is made, the said Court may issue a commission to any subordinate Court to conduct the inquisition. After the inquiry, if it is satisfactorily proved that the alleged lunatic is of unsound mind, and is incapable of managing his affairs, the Court may appoint a manager of the estate of the lunatic and a guardian of his person on the same terms and conditions as are mentioned in Chapter IV. But the manager has to submit an inventory of the estate belonging to the lunatic within six months from the date of his appoint-

ment and has to furnish an annual account of the income and expenditure within three months of the close of the year of the era current in the district. If any relative of the lunatic, or the Collector by petition to the District Court impugns the accuracy of such inventory or account, the Court may hold a summary inquiry into the matter, or refer such petition to any subordinate Court to the Collector if the manager was appointed by the Collector. The Court has power to remove a manager for any sufficient cause, and compel him to make over the property and to furnish accounts to any other person appointed in his place. The Court may impose a fine not exceeding five hundred rupees on the manager if he wilfully neglects or refuses to deliver his accounts or any property in his hands within the time fixed by the Court. The Court is also authorised, as in Chapter IV, to hold a second inquiry when it is reported that the lunacy has ceased.

Contracts.—Under section 12 of the Indian Contract Act (Act IX of 1872) a contract is invalid if one of the party at the time of making it was, by reason of insanity, incapable of understanding it, and forming a rational judgment as to its effect upon his interests.

A lunatic, however, is responsible for the payment of necessaries purchased by him in accordance with his social position and status, it being immaterial whether the vendor knew his condition or not; but he is not responsible if the order is grossly extravagant and beyond his means, or if the vendor has taken advantage of the fact of his insanity in selling those necessaries to him. Again, a person who is usually of unsound mind, but occasionally of sound mind, may make a contract when he is of sound mind. While a person who is usually of sound mind, but occasionally of unsound mind, may not make a contract when he is of unsound mind.

In a case where a person becomes lunatic after he has contracted to sell or otherwise dispose of his estate or any part thereof, the Court may direct the fulfilment of the contract, if it appears to the Court that the contract is such as ought to be performed. The Court may also order the dissolution of the partnership of a firm, if one of the partners is found to be a lunatic (*Vide* Sections 51 and 52, Act IV of 1912).

Marriage being regarded as a contract by the Divorce Act, 1869, may be declared null and void if it can be proved clearly and convincingly that one of the parties was, by reason of unsoundness of mind at the time of the marriage, unable to understand the nature and responsibilities of the contract of marriage. Unsoundness of mind developing subsequent to the marriage is no ground for divorce.

Mere weakness and imbecility of mind, eccentricity and partial dementia are not in themselves sufficient to void the marriage contract, but the mental defect or derangement must be such as prevents one party from comprehending the nature of the contract of marriage and from giving to it his or her free and intelligent consent.

In an appeal by Mst. Titli alias Tereza[1] from the decision of Mr. Young giving a decree of nullity of marriage at the instance of a European called Alfred Robert

1. *Leader, Nov.* 1, 1933.

Jones, a resident of Bhim Tal in Naini Tal district, who had prayed that his marriage with the appellant be declared null and void, one of the grounds being that he had been deficient in mentality since his very childhood and had to be looked after by his relations throughout his life, it was held that the marriage could not be declared as a nullity, as it was impossible to hold either that Mr. Jones was an idiot within the meaning of section 19 of the Divorce Act or that he was incapable of giving consent and did not voluntarily consent owing to force or fraud having been practised upon him after taking advantage of any imbecility of his mind. According to his own statement he understood what he was doing and realized what marriage meant.

Evidence.—Under section 118 of the Indian Evidence Act (Appendix V) a lunatic is not competent to give evidence if he is prevented by his lunacy from understanding the questions put to him and giving rational answers to them. However, he is competent to give evidence if an insane person is in the stage of a lucid interval, or if he is suffering from monomania, though it rests with the judge and jury to decide whether or not they should give credence to it.

Consent.—Section 90[1] of the Indian Penal Code provides that consent to certain acts is not valid, if such consent is given by a person who, " from unsoundness of mind or intoxication, is unable to understand the nature and consequence of that to which he gives his consent." The question of invalidity of consent may arise in cases of rape, causing death or grievous hurt, and abetment of suicide.

Consent to sexual intercourse given by a woman of imbecile or unsound mind is of no avail, and the act amounts to rape.

Exception 5 of section 300[1] of the Indian Penal Code provides that " culpable homicide is not murder when the person whose death is caused being above the age of eighteen years, suffers death or takes the risk of death with his own consent." Whereas section 87[1] of the Indian Penal Code provides that " an act not intended and not known to be likely to cause death or grievous hurt is not an offence by reason of any harm which it may cause, or be intended by the doer to cause, to any person, above eighteen years of age, who has given consent, whether express or implied, to suffer that harm; or by reason of any harm which it may be known by the doer to be likely to cause to any such person who has consented to take the risk of that harm."

Abetment of suicide under section 306[1] of the Indian Penal Code is punishable " with imprisonment of either description for a term which may extend to ten years and with fine," while abetment of suicide of " a person under eighteen years of age, a delirious person, an idiot, or a person in a state of intoxication " is punishable under section 305[1] of the Indian Penal Code with " death or transportation for life, or imprisonment for a term not exceeding ten years, and with fine."

Testamentary Capacity or Capacity to make a Valid Will.—A civil Court may invalidate a will if it is proved that the testator, at the time of making his will, was not of a " sound and disposing mind " and had not sufficient mental capacity to understand the nature and consequences of his act, and if it is satisfied that he disposed of his property in a way which he would not have done under normal conditions.

1. Vide *Appendix VII.*

If a medical practitioner has to examine a person as to his fitness to make a valid will, he should, before testifying, make the testator enumerate the amount of his property, the names of the relatives and others to whom he has left his legacies, and should make him repeat the main provisions of the will, enquiring reasons for any disposal of property which seems unjust or out of the common, or for any legal heirs being omitted. He should also find out if he knows the nature of the will and realises its consequences, and if he is not influenced by any insane delusions in disposing of his property.

If a medical practitioner has reason to suspect that he is under the influence of some person who prevents him from exercising his own discretion in making his will, it is better that he should see him alone and encourage him to speak out freely. It must be noted that a will is invalid if it is executed under the undue influence of any person.

Persons can make valid wills during lucid intervals. Persons affected by an insane delusion can make a valid will, if the delusion is not related in any way to the disposal of the property or to the persons affected by the will.

Wills made in a fit of drunkenness are considered valid unless the individual was so drunk as not to know the nature of what he was doing and unless they were repudiated in sober moments. Wills may be contested but cannot be declared invalid on the mere ground of the eccentricity, slovenliness, neglect of person and clothing, and offensive and disgusting habits of the testator, for these do not constitute unsoundness of mind.

In the case of *Katrak and another* v. *Khorshedbai and others* before the High Court of Bombay the will of a Parsi priest[1] was contested on the following grounds :—

/1. That the deceased was suffering from a delusion that his brother and sister had been instrumental in causing his son's death with a view to inheriting his property. This delusion so operated on his mind that he had lost his testamentary capacity.

2. That the deceased was not in a sound mind as he moved about in dirty clothes, kept food in cupboards for days and then ate the same in that condition, took away sandalwood offered at the agiary (fire temple) and sold the same for his benefit and sold sacred water of the sea to non-zorostrians and so on.

Dealing with the alleged delusion, His Lordship said that the evidence led in the case did not justify this conclusion. Even if there was this delusion, it did not prevent the deceased from making a valid will, inasmuch as it had not influenced him in not considering the claims of his relatives. The other allegations only showed that the deceased was a miser and did not at all prove that he had lost his testamentary capacity or was of unsound mind.

Having regard to the life led by the deceased and the fact that he had ceased to live with his brother and sister for over thirty years his Lordship found nothing unusual in his leaving his whole fortune amassed by leading a very frugal life, to the agiary to which he devoted his whole life. The evidence of the alleged delusion and unsoundness of mind was meagre, unsatisfactory and unreliable and did not justify his Lordship in coming to the conclusion that he was incapable of making a testamentary disposition. His habits of life might be eccentric, but the deceased was able to look after his affairs and showed clear-headedness.

1. *Times of India, Dec.* 2, 1936.

The will having been proved to have been properly executed by the deceased his Lordship granted probate thereof to the plaintiffs and dismissed the caveat making the defendants pay their own costs.

Wills made by persons *in extremis* are regarded as suspicious and may be set aside, for the mental condition in such cases is seldom normal.

Persons of extreme age and feeble health with defective memory and mental sluggishness are capable of making a will, unless their mind has become so impaired that they are incapable of understanding the business in which they are engaged when in the act of making their will.

Persons suffering from motor aphasia, agraphia, or any other nervous disease not affecting the brain, may be able to make perfectly valid wills.

CRIMINAL RESPONSIBILITY

The plea of insanity is generally brought forward in charges of murder in order to escape capital punishment. If insanity is established, the accused person is found "not guilty," and is ordered to be kept in a mental hospital, jail or other suitable place of safe custody.[1] An insane person is not punished for his crime, as he is devoid of free will, intelligence and knowledge of the act, but society must be protected against the attacks of an insane person.

The law presumes every individual at the age of discretion to be sane and to possess a sufficient degree of reason to be responsible for his criminal acts unless the contrary is proved to the satisfaction of the Court. In criminal cases where insanity is raised as a plea of irresponsibility the burden of proving it lies on the defence. Insanity may be proved from facts alleged or proved by the prosecution or independently by the defence. When a person accused of murder is alleged to be insane, the presiding officer of the Court generally asks the medical officer to keep the accused under observation and to certify whether he is insane or not. The medical officer takes the following points into consideration before deciding whether the murder was the result of insanity :—

1. *The Personal History of the Murderer.*—The murderer may be eccentric, melancholic, degenerate, neurasthenic, etc.

2. *The Absence of Motive.*—Not only does an insane person commit murder without any motive but he often kills his nearest and dearest relations, *e.g.*, his wife and children. It must, however, be remembered that in cases of homicide by sane persons it may, at times, be difficult to trace a motive, though there may be one. On the other hand, insane persons are known to have committed murders with a motive, however trifling it may be. Again, a sane person may commit murder on a very trivial excuse. I know of a case in which a young Passi murdered his sister-in-law with a *gandasa* (chopper) lying near on the mere ground that she asked him in joke to drink urine in place of water.

In an appeal at the High Court of Allahabad of one Lokmani, who had been convicted of murdering his wife and sentenced to death under section 302, I.P.C., by the Sessions Judge of Kamaun, their lordships set aside the conviction and the

1. *For procedure of trial of insane persons see Secs.* 464-75, *Cr. P. C., Appendix VI.*

sentence, as there was no motive for committing the murder. The accused admitted the crime before the Magistrate, and when asked why he did it, he said it was the will of God. Their lordships came to the conclusion that, by reason of unsoundness of mind, he was incapable of knowing the nature of the act.[1] On the other hand, in a case where one Inayat picked up a carpenter's adze that was lying near and killed his nephew, 9 years old, by giving with it two blows on the neck, it was held that the circumstance of an act being apparently motiveless is not a ground from which the existence of a powerful and irresistible influence or homicidal tendency can be safely inferred; he was convicted of murder, and was sentenced to suffer the penalty of death.[2] In another case where one Jalal killed a young woman of 26 years with a *toka,* it was held that the mere want of motive and the fact that the accused showed some sign that he suffered from a certain hallucination are not sufficient to attract the application of section 84, I.P.C. He was convicted of murder under section 302, I.P.C., and was sentenced to transportation for life.[3]

3. *The Absence of Secrecy.*—The murderer, if he happens to be insane, does not try to conceal the body of his victim, nor does he attempt to evade law by destroying evidence of his crime or by running away from the scene of the murder.

Cases.—1. On the 10th January, 1918, a girl, aged twelve years, murdered a child, three years old, in the District of Agra by inflicting about twenty-six wounds on the body with a *gandasa,* out of which one on the neck was fatal, the rest being more or less simple. The motive as alleged by the police was theft of brass wristlets worn by the child and worth four or six annas, but the girl did not make any attempt to conceal the wristlets or to run away from the spot where the murder was committed. It was argued in the Sessions Court that the girl probably had homicidal tendency, and at my suggestion the learned Sessions Judge ordered her to be kept under observation in the lunatic asylum of Agra for a period of six months.

2. In the case of *King-Emperor* v. *Bhagwati Parshad,* the accused, a Hindu male, about 24 years old, was convicted by the Sessions Judge of Lucknow of two offences under section 302, I.P.C., of causing the deaths of two old women, by hitting them on their heads with a piece of wood and causing fractures of the skull bones. On an appeal being preferred the Judicial Commissioner set aside the conviction on the ground of his unsoundness of mind and directed him to be kept in safe custody in the lunatic asylum at Bareilly. It came out in evidence that after committing the murders on the night of the 10th February, 1922, he made no attempt to run away or conceal himself. Medical evidence also proved that the accused was insane and had fixed delusions. He complained of the visit of a black man every night at 1 a.m., who stayed with him and beat him. He wore a garland of animal bones and had amulets of red cloth tied round his arm. He had a delusion of such an amulet being placed in his mouth rendering him invisible.

4. *Multiple Murders.*—A sane person usually murders only one person with whom he is at enmity or against whom he has a grievance, and does not shed more blood unnecessarily. On the other hand, an insane person may kill several persons, mostly his friends and relatives for whom he has great regard and affection. It is, however, possible for an insane person to have only one as his victim.

5. *Want of Preparedness or Prearrangement.*—An insane person does not make any prearranged plan to kill anybody, but a sane person, as a rule, makes all the necessary preparations prior to committing a crime.

Overbeck-Wright, however, cites the following exceptional case in which an insane person exhibited elaborate premeditation and contrivance in committing a murder[4]:—

1. *Leader, Sep.* 17, 1925.
2. *Crim. Law Jour., Nov.,* 1928, *p.* 1006.
3. *Crim. Law Jour., Nov.,* 1929, *p.* 1024.
4. *Lunacy in India, p.* 32.

Bertha Peterson, aged 45, daughter of the Rector of Biddenden, was indicted for the murder of John Whibley. The deceased, a shoemaker, had been a teacher in the Sunday-school of Biddenden, and there had been rumours, eighteen months before the murder of his having behaved indecently towards a little girl of eleven. The prisoner was much interested in the rumour, was a disciple of Mr. Stead, took a great interest in the Criminal Law Amendment Act, and appears to have allowed her attention to be absorbed by these subjects until she became even more crazy than the general run of the nasty-minded apostles of purity. She purchased a revolver and practised with it. She wrote to the deceased expressing her regret for the mistaken attitude she had adopted towards him, and asking him to meet her in the parish school-room in the presence of witnesses, and shake hands as a token of forgiveness. The meeting took place, and then, asking deceased to take a good look at a picture on the wall she placed a revolver to the back of his head and shot him dead. Evidence was given of various eccentricities in the previous conduct of the prisoner, and Dr. Davies, Superintendent of the Kent County Asylum, and Dr. Hoare, Surgeon to the Maidstone Gaol, in which the prisoner had been detained pending her trial, stated that in their opinion the prisoner was under the hallucination that she was ordered to shoot the man. The jury returned a verdict of "Guilty but insane."

6. *Want of Accomplices.*—An insane person has no accomplice in the criminal act. Lunatics in mental hospitals never conspire to escape or kill the Superintendent or his assistant.

The English law of the criminal responsibility of the insane is based on the answers given by fourteen Judges in 1843 to the following hypothetical questions put to them by the House of Lords in connection with the celebrated case of Mc Naughten who, labouring under persecutional delusions, shot Mr. Drummond, the Private Secretary of Sir Robert Peel, at Charing Cross :—

Question I.—"What is the law respecting alleged crimes committed by persons afflicted with insane delusions in respect of one or more particular subjects or persons, as, for instance, where, at the time of the commission of the alleged crime, the accused knew he was acting contrary to law, but did the act complained of, with a view, under the influence of insane delusion, of redressing or revenging some supposed grievance or injury, or of producing some supposed public benefit ?"

Answer I.—"Assuming that your lordships' inquiries are confined to those persons who labour under such partial delusions only, and are not in other respects insane, we are of opinion that (notwithstanding the accused did the act complained of with a view, under the influence of insane delusion, of redressing or revenging some supposed grievance or injury, or of producing some public benefit) he is nevertheless punishable, according to the nature of the crime committed, if he knew at the time of committing such crime that he was acting contrary to law, by which expression we understand your lordships to mean the law of the land."

Question II.—"What are the proper questions to be submitted to the jury when a person, alleged to be afflicted with insane delusions respecting one or more particular subjects or persons, is charged with the commission of a crime (murder, for example) and insanity is set up as a defence ?"

Question III.—"In what terms ought the question to be left to the jury as to the prisoner's state of mind at the time when the act was committed ?"

Answers II and III.—"As these two questions appear to us to be more conveniently answered together, we submit our opinion to be that the jury ought to be told in all cases that every man is presumed to be sane, and to possess a sufficient degree of reason to be responsible for his crimes, until the contrary be proved to their satisfaction ; that to establish a defence on the ground of insanity, it must be clearly proved that, at the time of committing the act, the accused was labouring under such a defect of reason, from disease of the mind, as not to know the nature and quality of the act he was doing, or, if he did know it, that he did not know that he was doing what was wrong. The mode of putting the latter part of the question to the jury on these occasions has generally been, whether the accused at the time of doing the act knew

the difference between right and wrong; which mode, though rarely, if ever, leading to any mistake with the jury, is not, we conceive, so accurate when put generally and in the abstract as when put with reference to the party's knowledge of right and wrong in respect to the very act with which he is charged. If the question were to be put as to the knowledge of the accused, solely and exclusively with reference to the law of the land, it might tend to confound the jury by inducing them to believe that an actual knowledge of the law of the land was essential in order to lead to a conviction; whereas the law is administered on the principle that every one must be taken conclusively to know it without proof that he does know it. If the accused was conscious that the act was one which he ought not to do, and if that act was at the same time contrary to the law of the land, he is punishable. The usual course, therefore, has been to leave the question to the jury, whether the accused had a sufficient degree of reason to know that he was doing an act that was wrong; and this course we think is correct, accompanied with such observations and corrections as the circumstances of each particular case may require."

Question IV.—"If a person under an insane delusion as to existing facts commits an offence in consequence thereof, is he thereby excused?"

Answer IV.—"The answer must of course depend upon the nature of the delusion; but making the same assumption as we did before, namely, that he labours under such partial delusion only, and is not in other respects insane, we think he must be considered in the same situation as to responsibility as if the facts with respect to which the delusions exist were real. For example, if, under the influence of his delusion, he supposes another man to be in the act of attempting to take away his life, and he kills that man, as he supposes, in self-defence, he would be exempt from punishment. If his delusion was that the deceased had inflicted a serious injury to his character and fortune, and he killed him in revenge for such supposed injury, he would be liable to punishment."

Question V.—"Can a medical man, conversant with the disease of insanity who never saw the prisoner previously to the trial, but who was present during the whole trial and the examination of all the witnesses, be asked his opinion as to the state of the prisoner's mind at the time of the commission of the alleged crime, or his opinion whether the prisoner was conscious at the time of doing the act that he was acting contrary to law, or whether he was labouring under any, and what, delusions at the time?"

Answer V.—"We think that the medical man, in the circumstances supposed, cannot in strictness be asked his opinion in the terms above stated, because each of those questions involves the determination of the truth of the facts deposed to, which it is for the jury to decide, and the questions are not mere questions upon a matter of science, in which case such evidence is admissible. But where the facts are admitted, or not disputed, and the question becomes substantially one of science only, it may be convenient to allow the question to be put in the general form, although the same cannot be insisted on as a matter of right."

The crux of these answers is known as "the Mc Naughten rule" or "the legal test", which is as follows:—

"That to establish a defence on the ground of insanity, it must be clearly proved that at the time of commiting the act, the party accused was labouring under such a defect of reason from disease of the mind, as not to know the nature and quality of the act he was doing or, if he did know it, that he did not know he was doing what was wrong."

This legal test has also been accepted in India as the law of criminal responsibility, and is embodied in section 84 of the Indian Penal Code, which runs as follows:—

"Nothing is an offence which is done by a person who at the time of doing it, is, by reason of unsoundness of mind, incapable of knowing the nature of the act, or that he is doing what is either wrong or contrary to law."

In order to exempt a person from criminal responsibility under this section it must be proved that the unsoundness of mind existed at the time of committing the offence. Subsequent insanity does not affect the crime though it affects the trial.[1] It may be necessary to enquire into the previous mental condition to prove the state of mind at the time of committing the offence. Further, unsoundness of mind should be of such an extent as would render the sufferer incapable of knowing the nature and character of the act or would render him incapable of understanding that the act he was doing was morally wrong or was an offence against the law of his country. A person can thus be exonerated from criminal responsibility if his cognitive faculties have been affected by unsoundness of mind. Hence idiots, imbeciles, and persons who are deprived of all understanding and memory are not responsible for criminal offences, and do not present any difficulty in Courts of law. Difficulty, however, arises in those cases where persons labour under partial delusion only and are otherwise quite sane. In such cases these individuals should be placed as regards criminal responsibility in the same situation as if the facts with which the delusion existed were real.[2] For instance, if, in consequence of an insane delusion, a person thinks another man to be a wild beast or a jar made of clay, and kills him, he is exempted from criminal responsibility, as he does not know the physical nature of the act. If he kills a child under an insane delusion that by doing so he is saving him from sin and sending him to heaven, he knows the nature of the act that it will result in death, but he is not capable of understanding that what he is doing is morally wrong. In a criminal case[3] Martin, B. cited before the jury as an instance of delusion the case of a man who fancied himself to be a king dispensing justice to his subjects. "If such a man were to kill another under the supposition that he was exercising his prerogative as a king, and that he was called upon to execute the other as a criminal he would not be responsible." Again, if a person kills another man under the influence of a delusion that he is attempting to take his life, he would be exempt from punishment, inasmuch as he, by reason of insanity, is incapable of knowing that his act is contrary to the law of his country. He is justified in killing that man in self-defence if his delusion were true.[4] Similarly, a person who kills another man under the belief arising from an insane delusion, that the man had committed adultery with the prisoner's wife would be entitled to have his offence reduced under exception I of section 300, I. P. C., as having been committed under grave provocation.[5] On the other hand, if a person kills another man under the influence of an insane delusion that he had inflicted a serious injury to his character and fortune, he is criminally responsible for his offence, seeing that no one is entitled by law to kill a person in revenge for such injury, even if his delusion were true.[6]

One Karma Urang, accused, had a dream in which the goddess *Kali* appeared before him and told him that his father was a descendant of *Kali* and that if he (the accused) did not kill his father, his father would kill him. The accused honestly

1. *Cr. P. C., Sec. 466, Appendix VI.*
2. *Gour, Penal Law of India, Ed. II, Vol. I, p. 487.*
3. *R. v. Townby, 3 F. and F. 839.*
4. *Mac Naughten's Case I. O. and K. 130.*
5. *Mayne, Cr. Law of India, Part II, p. 175.*
6. *Mac Naughten's Case, Loc. Cit.*

believed this and cut off his father's head the next day and was coolly proceeding with it to the Court with the object of producing the head before the Court, when he was arrested. The medical evidence showed that he was under a definite delusion. It was ruled in an appeal in Calcutta High Court that the accused must under the circumstances be held to have been incapable at the time of the doing of the act by reason of unsoundness of mind of knowing the nature of the act or that he was doing what was either wrong or contrary to law within the meaning of section 84, I.P.C., and that he could not be convicted of murder.[1]

In a case where one Mani Ram, after murdering four persons (his own relatives) in rapid succession with a *gandasa* dropped it and began to run away and subsequently volunteered the information that he had murdered his elder nephew, one of the deceased, their lordships held that the mere presence of the five circumstances, *viz.*, absence of any motive, accomplices, secrecy, etc., did not fulfil the requirements of section 84, I.P.C. A man might be suffering from insanity in the sense in which the words would be used by an alienist, but might not be suffering from unsoundness of mind as defined in this section. The law recognised nothing but incapacity to realise the nature of the act, and presumed that where a man's mind or his faculties of ratiocination were sufficiently clear to apprehend what he was doing, he must always be presumed to intend the consequences of the action he took. It was perfectly clear from the conduct of the accused that he knew what he was doing and that what he was doing was wrong. Their lordsrips had no option in the matter but to find that the accused had wholly failed to establish the unsoundness of mind. They, therefore, dismissed his appeal and confirmed the sentence of death.[2]

In a murder appeal,[3] in which the appellant, Muhammad Hashim, aged 40 years, had been sentenced to transportation for life by the Sessions Judge of Bulandshahr for having killed Khalil, a boy of seven or eight years, by stabbing him in the stomach with a butcher's knife, it was held by their lordships that the mere incident of the murder itself seemed to them to indicate the act of a mad man. The accused was not said to have had the slightest enmity or grudge either against the little boy, Khalil, or his father or any relative of his. There was no motive whatever for committing such a crime and this in itself suggested that the accused must have been of unsound mind. In addition to this there were statements of a number of witnesses and even some prosecution witnesses, which tended to show that the accused was of unsound mind at the time. The civil surgeon who was superintendent of the jail never expressed any decided opinion that the accused was of sound mind at the time of committing the offence or not of sound mind but merely stated that he found no signs of insanity from the period that the accused was under his observation. Technically the superintendent's report was not admissible in evidence because he was never called as a witness. Their lordships, however, considered that no great importance could be attached to the superintendent's report in view of the positive evidence referred to above showing that the accused must have been deranged in mind to the extent of being incapable of knowing the nature of the act that he was doing. In their lordships' opinion his act was not an offence by reason of unsoundness of mind under section 84 of the Indian Penal Code.

Their lordships, therefore, found that the accused did stab Khalil with a knife and caused his death but acquitted him upon the ground that at the time when he committed the act he was, by reason of unsoundness of mind, incapable of knowing the nature of the act. Under section 471 of the Criminal Procedure Code their lordships directed that the accused be detained in safe custody in such place and manner as the sessions judge might think fit and the sessions judge should report the action taken to the local government.

In another murder appeal[4] before the Oudh Chief Court, in which the appellant, Onkar Datt Nigam, made murderous assaults by firing five shots into a second class compartment in which were travelling an Anglo-Indian gentleman (Mr. Ralph) and Miss Edna Doran, who were quite strangers to him, at Unao Railway Station, on May

1. *Crim. Law Jour., March*, 1929, *p.* 247.
2. *Crim. Law Jour., Jan.*, 1927, *p.* 121.
3. *Leader, Sept.* 23, 1933.
4. *Cr. Law J., Vol.* 36, 1935, *p.* 393 ; vide *also Lahore H. C., Cr. App. No.* 704 *of* 1936, *Cr. Law Jour.*, 1937, *Vol.* 38, *p.* 893.

9, 1933, it was held that the accused was suffering from unsoundness of mind to such an extent as to make him incapable of knowing the nature and character of his act, which he had committed and which made him incapable of understanding that the act he was doing was more or less wrong, or was an offence against the law of the country, that his cognitive faculties had been affected by unsoundness of mind and that he was entitled to an acquittal. It was also held that as the accused was proved to be a criminal lunatic, and as in the opinion of the medical expert on mental diseases, there was no likelihood of his ever regaining his mental capacity, he might be directed, under section 471, Criminal Procedure Code, to be detained in safe custody in a mental hospital.

It was proved that the appellant committed the offence without any attempt at concealment of the crime. There was no accomplice with him, and not only did he shoot one person, but he tried to kill two and there was also no premeditation in the commission of the offence. The medical evidence as to the mental condition of the accused given by the mental expert (Col. Overbeck Wright) was that he was insane at the time of the commission of the offence, and that he was suffering from hebephrenia.

Loss of Control.—A person accused of crime in India is not entitled to exemption from criminal responsibility on the mere ground of loss of the power of self-control at the time of perpetrating the offence, unless it is attributable to insanity satisfying the usual legal tests; *viz.*, inability to distinguish right from wrong or to know the nature and consequences of the act. This view was taken into consideration by the learned Judges when they convicted Lakshman[1] who, being vexed with the cries of his two small children, had killed them. A similar view was also taken in the case of Venkata Sami[2] who had murdered his brother's child wife. The application of these legal tests in all such cases is not very sound; inasmuch as there is a form of insanity, known as impulsive insanity, which affects the will and emotions and not the cognitive faculties. The patient is able to realise the difference between right and wrong and the nature and consequences of the act, and yet he commits the crime being impelled by an irresistible impulse induced by a diseased mind. Such a condition should be recognized in Courts as a sufficient ground for exemption from criminal liability. "Criminal responsibility should, however, not be extended to one who with no mental disorder acts from overmastering anger, jealousy or revenge. There must be insanity first."[3]

The plea of irresistible impulse was brought forward in a murder trial at Manchester, but was overruled as there was evidence of premeditation, that the prisoner bought the knife with which he committed the murder and sharpened it on both edges.[4] In a case where one Shersing attempted to kill his wife and his mother-in-law, and did kill his brother-in-law, aged ten years, and subsequently set fire to the hut belonging to his mother-in-law, it was ruled that at the time of the commission of the offences the accused was in a highly excited and unbalanced condition, he was, nevertheless, conscious that what he was doing was wrong and a crime. His appeal was, therefore, not allowed.[5] In another case where one Tolaram had murdered his father on the 11th November, 1926, it had been proved that the accused was melancholic and had been subject to fits of epilepsy and was at the time of murder suffering from vertigo. It was held that the fact that the physical and mental ailments from which a man suffered had rendered his intellect weak and had affected his emotions and will was not sufficient to bring his case within section 84, I.P.C. The question is

1. *Lakshman Dagdu*, 10 *Bombay*, 512.
2. *Venkatasami*, 12 *Madras*, 459.
3. *Shamsul Huda, Principles of Law of Crimes*, *p.* 294.
4. *Jour. Amer. Med. Assoc., April* 16, 1921, *p.* 1116.
5. *Lahore High Court Cr. Appeal No.* 1046 *of* 1922; *Criminal Law Jour., May* 1924, *p.* 395.

whether his cognitive faculties had been impaired to the degree described in the last para of section 84. On the occasion in question the loss of the power of self-control was not due to the want of consciousness of the nature and quality of his act, brought about by a diseased state of mind, but was obviously the result of the sudden rousing of passions which he was unable to subdue at the time. It was clear that he struck the deceased not in a paroxysm of insanity but in a fit of anger.[1]

One Matin Ali,[2] son of a retired extra-assistant commissioner, with a friend of his engaged a taxi in August, 1932, from Nagpur for Chindwara, and while returning shot the owner and the cleaner of the car at night near Silvani Ghat. He was absconding and arrested on the fourth day of the occurrence. He was sentenced to transportation for life by the Sessions Judge of Chindwara for this double murder. On an appeal preferred by him, the Judicial Commissioner in the course of his judgment observed that the case did not fall within section 84 of the Indian Penal Code because the mental faculties of Matin distinguishing right from wrong from a moral point of view were absolutely clear. The applicant fully believed that taking life of another was not only illegal but immoral. The appellant divided himself into two parts, *viz.*, Matin Ali and Rumi Safa (free lance). According to him there resided in his physical body both good and evil spirits and in spite of his control the evil spirit forced him to kill useless persons like himself to make the world better. Matin did not commit suicide as the world would have taken him as a coward. The present crimes were committed in a fit of impulsive insanity without any motive or premeditation, nevertheless they did come under purview of section 302, I.P.C., but necessitated indulgent consideration. Having regard to the fact that the appellant belonged to a respectable family and had received higher education, the Judicial Commissioner directed that the case be laid before the local Government for such indulgent consideration as they may be pleased to show to the appellant under section 401 of the Criminal Procedure Code.

In the case of *Rex* v. *Ronald True*, who was tried at the Central Criminal Court of London on a charge of murdering a prostitute by strangulation after spending the night at her flat on March 6, 1922, Mr. Justice Mc Cardie, in his charge to the jury, said, " even if the prisoner knew the physical nature and that it was morally wrong and punishable by law, yet was by mental disease deprived of the power to control his actions, then the verdict should be guilty but insane." His Lordship definitely conceded the doctrine of criminal irresponsibility on the ground of impairment of the power of self-control, but pointed out that this exemption should be applied with great care. The jury found the accused guilty of " wilful murder ", and he was sentenced to death. The sentence was subsequently upheld by the Court of Criminal Appeal. Shortly afterwards a representation was made to the Home Secretary that True was insane, whereupon he appointed a committee of three mental specialists to examine True and to certify if he was sane or insane. On their having certified that he was insane the Home Secretary ordered True to be removed to a criminal asylum.

In July, 1922, the Lord Chancellor appointed a committee consisting of the well-known lawyers under the chairmanship of Lord Justice Atkin to consider and report upon what changes, if any, were desirable in the existing law, practice and procedure relating to criminal trials in which the plea of insanity is raised as a defence. This committee recommended that the rules formulated in the Mc Naughten case be maintained, and further recommended an additional rule that it should be recognized that a person charged criminally with an offence is irresponsible for his act

1. *Lahore High Court Cr. Appeal No.* 228 *of* 1927; *Crimin. Law Jour., July,* 1927. *p.* 598.
2. *Leader, Sept.* 25, 1933.

when the act is committed under an impulse which the prisoner was, by mental disease, in substance deprived of any power to resist. These recommendations were incorporated in a bill, called the Criminal Responsibility (Trials) Bill, which was moved in the House of Lords in 1924, but was negatived.

Somnambulism.—This is an abnormal mental condition, and means walking during sleep. In this condition the mental faculties are partially active and are so concentrated on one particular train of ideas that a somnambulist is capable of performing most remarkable and incredible pieces of work, which would have baffled his intelligence during his waking hours. A somnambulist may thus solve a very difficult problem or may commit theft or murder. A person who is the victim of a somnambulisitc habit has generally no recollection of the events occurring during the fit after he awakes. In some cases he remembers the events of one fit in subsequent fits and follows them with exact precision, though he forgets them in the normal state.

Somnambulism forms a very good plea of defence for exemption from criminal liability, if it can be proved that the accused committed the offence during the fit. For instance, a man named Fraser[1] who killed his child by dashing its head against the wall was acquitted of murder on the ground that he was in a fit of somnambulism at the time of committing the murder and was, therefore, unconscious of the nature of the act. In another case where Maggie Alexander[2] was charged with having murdered her child with a razor the jury returned a verdict of "guilty but insane", as it was definitely proved in evidence that the accused was a somnambulist, that she committed this act in a state of somnambulism, and therefore did not know what she was doing, that she did not appreciate the nature and quality of the act, and that she did not know she was doing it at all because of this somnambulistic state from which she unquestionably suffered.

Semisomnolence or Somnolentia.—This is half way between sleep and waking and is very often called sleep drunkenness. This condition is mostly allied to a mental condition occurring in some cases immediately after an epileptic fit. If a person is suddenly aroused from deep sleep, he may unconsciously commit some horrible and illegal deed owing to his mind being in a state of confusion, especially if he is having a dream or a nightmare at the time. He is not criminally responsible for such a deed.

A woman in Hungary attacked her daughter in the middle of the night with an axe, inflicting serious injuries. At her trial she pleaded nightmare as a defence. She alleged that she had heard gossip that her daughter had been seen walking at night in remote places with a young man, and had been filled with suspicion. On the night of the assault she dreamt of her daughter's disgrace and saw her arrested for an offence against public morals. Under the influence of the dream she gave way to an overpowering impulse to kill her. She had been brought to her senses only by the girl's screams. The Medical Advisory Board of Criminal Jurisdiction, to which the court referred the matter, advised that the woman had told the truth and was not responsible for the assault. She was accordingly acquitted.[3]

1. *Brit. Med. Jour.*, 1878, *Vol. I, p.* 108.
2. *Lancet*, Dec. 14, 1929, *p.* 1265.
3. *Times, Jan.* 2, 1937, *p.* 9 ; *F. H. C., Med.-Leg. and Criminolog. Rev., Vol. V, Part I, Jan.*, 1937, *p.* 94.

Hypnotism or Mesmerism.—This is a sleep-like condition brought on by artificial means or by suggestion and is allied to somnambulism. During a hypnotic trance, though unconscious of surrounding objects, a person may perform acts suggested by the hypnotiser, but does not remember them afterwards. Sometimes, the suggestive influence may last beyond the period of the hypnotic trance. Difference of opinion exists as to whether a hypnotised person can be made to commit a criminal act but the best authority would seem to indicate that while persons under hypnotic control can be influenced to commit acts in line with defects in character or weakness of morals which they might otherwise not commit, the fundamental principle holds true that no one can be compelled by hypnotic influences to commit any deed of which he was not capable in the normal state. A person cannot be hypnotised against his will, hence if he volunteers to be hypnotised, he is expected to have anticipated all the consequences of the act and agreed to become responsible for them and the well-settled principle of law that a person cannot take an advantage of his own misconduct would govern in case he violated the law in the state of hypnotism.[1]

Hypnotism as a defence to a criminal act is not generally recognized in Courts. In a case where Gouffe was murdered by hanging, Bompard, one of the murderers, unsuccessfully pleaded that she had been hypnotised by Eyraud, the other murderer, and while under his influence was induced to take part in the murder.

Delirium.—According to Tuke[2] delirium is "a perversion of the mental processes, the perversion being manifested in speech or action. The disturbance is characterized by incoherent speech, hallucinations, illusions and delusions, restlessness, watchfulness, apparently purposeless actions, inability to fix the attention." Owing to hallucinations and delusions being present in a state of delirium the patient may commit violent fatal acts. Such a person is not legally responsible for acts committed during an access of delirium if, in the words of section 84, I.P.C., he lost consciousness to such an extent as would prevent him from knowing the nature of the act or distinguishing between right and wrong.

Drunkenness.—The law relating to drunkenness and criminal responsibility is laid down in the following two sections of the Indian Penal Code :—

Section 85.—Nothing is an offence which is done by a person, who, at the time of doing it, is, by reason of intoxication, incapable of knowing the nature of the act, or that he is doing what is either wrong, or contrary to law : provided that the thing which intoxicated him was administered to him without his knowledge or against his will.

Section 86.—In cases where an act done is not an offence unless done with a particular knowledge or intent, a person who does the act in a state of intoxication shall be liable to be dealt with as if he had the same knowledge as he would have if he had not been intoxicated, unless the

1. *Medico-Legal Jour., Vol. XLI, March-April,* 1924, *No.* 2, *p.* 48.
2. *Dict. of Psych. Medicine.*

thing which intoxicated him was administered to him without his knowledge or against his will.

It is evident from these sections that drunkenness caused by the voluntary use of alcohol or some other intoxicating drug is no excuse for the commission of a crime, but insanity produced by drunkenness, voluntary or otherwise, absolves one from criminal responsibility, if it can stand the usual legal tests applied in the other forms of insanity. In the case of *Reg.* v. *Davis*[1] where the prisoner was charged with wounding with intent to murder, the defence was that the accused was of unsound mind at the time of the commission of the act, and the evidence established the fact that he was suffering from delirium tremens resulting from overindulgence in drink. Stephen J., in summing up to the jury said " but drunkenness is one thing and the diseases to which drunkenness leads are different things ; and if a man by drunkenness brings on a state of disease which causes such a degree of madness even for a time, which would have relieved him from responsibility if it had been caused in any other way, then he would not be criminally responsible. In my opinion, in such a case the man is a mad man, and is to be treated as such, although his madness is temporary. If you think there was a distinct disease caused by drinking, but differing from drunkenness and that by reason thereof he did not know that the act was wrong, you will find a verdict of not guilty on the ground of insanity."

In cases where the intention of a person committing a crime is an ingredient of the crime itself, the fact of his being drunk at the time when the act was committed may be taken into consideration in considering whether he formed the intention necessary to constitute the crime. In the case of *Rex* v. *Beard*,[2] where the accused was indicted for wilful murder, it was proved by the prosecution that he had ravished a young girl, and in aid of the act of rape had placed his hand on her mouth to prevent her from screaming, at the same time pressing her throat with his thumb, causing death by suffocation. The chief defence was drunkenness, it being contended that when the crime was committed his mind was so affected by drink that the charge ought to be reduced to manslaughter. It was held by the House of Lords that the drunkenness was no defence unless it could be established that Beard at the time of committing the rape was so drunk as to be incapable of forming the intent to commit it. Death resulted from a succession of acts—the rape and the act of violence causing suffocation. These acts could not be regarded independent of each other, and as it was not alleged that the prisoner was too drunk to form the intent of committing the rape, the defence of drunkenness to the charge of murder must fail. In the course of their judgment the House of Lords have laid down " that evidence of drunkenness which renders the accused incapable of forming the specific intent essential to constitute the crime should be taken into consideration with the other facts proved in order to determine whether or not he had this intent. Evidence of drunkenness falling short of a proved incapacity in the accused to form the intent necessary to constitute the crime, and merely establishing that his mind was affected by drink so that he more

1. 1881, 14 *Cox C. C.* 563.
2. 1920 *A. C.* 479 ; *Llewellyn Jones, Trans. Med.-Leg. Society, Vol. XXII, p.* 33.

readily gave way to some violent passion, does not rebut the presumption that a man intends the natural consequences of his acts." This observation of their Lordships has been followed in the cases in Indian Courts and is deemed as a final statement of the law on this point. In the case of *King-Emperor* v. *Bishan Singh*,[1] where the accused was charged with having murdered three persons by firing a gun in a state of intoxication, it was held that the accused was not in such an advanced state of intoxication as not to be fully aware of what he was doing and not to be perfectly cognizant of the probable consequences of his act. When firing at the persons in question he must at least be deemed to have intended to cause such injuries as he knew were likely to result in death, and accordingly he must be held guilty of murder within the terms of section 302, Indian Penal Code. In the case of *King-Emperor* v. *Judagi Mallah*[2] where the accused caused the death of his cousin, Deonarain, by stabbing him on the throat with a knife in the course of a drunken brawl, it was held that the accused was incapable by reason of drunkenness to form the intent necessary to constitute the crime. It was brought out in evidence that after he committed the crime he ran about saying that he had killed this man and was going to be hanged. He said that he had done a wrongful act. The accused had the knowledge and the intention which would make him liable under section 302, Indian Penal Code, and, therefore, he was guilty of murder.

1. *Lahore High Court, Cr. App. No.* 201 *of* 1929; *Crim. Law Jour., July,* 1929, *p.* 662.
2. *Patna High Court, Death Reference Case No.* 8, 1929; *Criminal Law Journal, March,* 1930, *p.* 243; *see also Rangoon H. Court Cr. App. No.* 1992 *of* 1933, *K. E.* v. *Nga Sein Gule, Cr. Law Jour.,* 1935, *Vol.* 36, *p.* 228.

CHAPTER XX

LIFE ASSURANCE

Definition.—By *Life Assurance* or *Insurance* is meant a contract by which a person, whose life is insured, agrees to pay to an insurance company, a certain sum of money, called a premium, annually or by periodical instalments, for a specified number of years or for life, so that he or his heirs may get a stipulated sum from the company at a certain age or after his death. The written document or contract specifying the terms which the company undertakes to fulfil is called a policy.

FORMS OF POLICY

Various companies issue prospectuses showing the tables of rates payable at different ages according to the form of policy taken out by the insurer. The chief forms of policy are—

1. **A life policy**, according to which an insured person has to pay an annual premium until death.

2. **A terminable policy**, by which an individual pays the premium for a limited number of years, the policy being payable at death.

3. **An endowment policy**, by which a person agrees to pay for a number of years, say up to fifty-five, when he receives the amount of money for which his life was insured.

4. **An investment policy**.—According to this policy a limited number of premiums is paid, and every paid premium secures a fixed and definite benefit according to the table given in the prospectus, so that the payments may be discontinued at any time without forfeiture of the policy.

THE MEDICAL EXAMINER

In order that they may not run the risk of insuring bad or undesirable lives, insurance companies usually appoint a medical man as their officer to examine the proposer medically in towns and cities, wherever their offices or agencies are situated.

MEDICAL EXAMINATTION OF A PROPOSER

A person who proposes to have his life insured against death, accident or sickness. is introduced by the agent of the company to their medical officer, at his consulting room, who is supplied with a printed form of queries which he must fill up in his own handwriting after a careful medical examination of the proposer. He is required to send this form with his opinion and recommendation regarding the life of the person direct to the general agents of the company without showing it to any one else.

The medical examiner is paid by the company for the examination, and is bound to look to their interests, but he must not show any partiality. He should try to be absolutely fair to the company as well as to the applicant. If he happens to be an ordinary medical attendant of the proposer and if he knows that the latter suffers from a disease, which renders him incapable of being insured, he should refuse to examine him, and inform the company accordingly.

The examination must be made in private where no other person should be permitted to be present except in the case of a female, when her relative or friend may be requested to be present. The examination should always be made in the day time, and the applicant should be stripped to the waist.

During the examination the inquiries which the medical examiner is expected to make relate to—

A. The Personal History.
B. The Family History.
C. The Personal Examination.

A. THE PERSONAL HISTORY

Under this heading careful inquiry should be made about the applicant's occupation, residence, personal habits and past diseases, since all these points are very important to determine whether the life can be regarded as worth insuring or not.

The applicant must be cautioned to tell the whole truth and nothing but the truth, as concealment of any material fact may subsequently render the policy null and void, and the premiums paid may be forfeited.

Occupation.—Certain occupations involve special risk, *e.g.,* those of engine drivers, mechanical engineers, spinning and weaving masters, miners, makers of explosives, soldiers, sailors, and medical men who are always exposed to the dangers of infection. Farmers and clergymen are the most long lived among the professional men.

Residence.—Residence in a tropical climate is more conducive to ill-health than in a temperate climate; hence some companies always put an extra premium on the rate which an applicant would have to pay if he were to remain all his life in a temperate region.

Personal Habits.—The question of the proposer's habits is one of great importance, though it is very difficult for the medical man to get the correct information as he has to depend on the truthfulness of the applicant himself unless the medical man happens to be personally acquainted with him. However, an experienced medical man can find out if the applicant is addicted to alcohol by observing the condition of the tongue and conjunctivæ, the presence of tremors of the hands, the dilated capillaries of the cheeks and the rosy tip of the nose. The application should be declined if there is the least suspicion of intemperance. Similarly, excessive smoking can be inferred from tremors of the hands, tachycardia, indigestion and constipation. Cancer of the lip, tongue or throat is more common among smokers than among non-smokers..

It should be enquired whether the applicant is habituated to the use of opium or *bhang,* and the quantity which he uses. It should also be ascertained if the applicant is taking any exercise or leading a sedentary life. In the case of exercise, whether it is in excess or not. A professional athlete is not assurable, as he is always open to risks and his heart is usually hypertrophied.

Past Diseases.—While recording past illnesses the examiner should try to obtain as complete a statement of each of the diseases from which the applicant has suffered with a view to determine if any of those diseases has left any permanent ill-effects on his constitution or to discover any tendency to disease in the future. An applicant should never be recommended for assurance if he is suffering at the time from any ailment however slight it may be, or if he has recently recovered from an acute disease.

The diseases which are chiefly required to be inquired from a life assurance standpoint are—

Phthisis and Haemoptysis.—The applicant should be rejected if he gives a history of having suffered from phthisis or hæmoptysis in the past, and at the same time if he comes from a stock of consumptives and has feeble physique and light weight. He may be accepted with an addition of ten years or so to his actual age if he has not shown any symptoms of phthisis, or if he has had no attack of hæmoptysis within the last ten years and if he is at least thirty-five years of age and living in healthy surroundings. However, he should, under no circumstances, be insured if he is addicted to the use of alcohol, for rightly has it been said that " alcoholism makes the bed for tuberculosis."

Pneumonia and Pleurisy.—No person can be regarded as insurable for six months after an attack of pneumonia, as it may lead to tuberculosis. An attack of pleurisy should always be looked upon with suspicion as it has a tendency to lead to tuberculosis. A person who has suffered from empyema can be accepted at the ordinary rate if six months have elapsed since his complete convalescence.

Asthma.—The applicant should be rejected if he gives a history of recent asthma. However, he may be accepted with an addition of a few years to his age if he had a last attack some years ago, and if his lungs were not particularly emphysematous.

Discharge from the Ears.—Persons suffering from chronic otorrhœa may die from intracranial complications, but many are known to have lived to an old age. Hence an applicant having a slight chronic discharge escaping freely from the ears may be accepted with a comparatively small extra. On the other hand, an applicant who shows any signs or symptoms of activity or extension, or a copious or sanious discharge from his ears, or complains of headache, local pain or giddiness is not insurable and should be rejected.[1]

Blindness.—If the applicant is otherwise all right, ten years may be added to his actual age for calculation of the premium.

Dysentery.—If the attacks have been frequent, the possibility of a liver abscess must not be lost sight of.

Hernia.—The applicant must be rejected if no truss is worn. An addition of one or two years is quite sufficient in the case of a reducible inguinal hernia, provided a well fitting truss is used. A person with a partially reducible hernia should be rejected, especially if he is engaged in hard laborious work. A femoral hernia is more dangerous than an inguinal one and should, therefore, be rated very highly. An applicant with an umbilical hernia should, as a rule, be rejected. In all cases of hernia the risk is enhanced very much if the applicant lives in an outlandish place where he is not likely to get surgical aid, when necessary.

Stricture of the Urethra.—If the stricture is of a slight degree it requires an increased rating, but the more severe forms should be declined.

Varicose Veins.—In the case of varicose veins the proposal may be accepted after an operation is performed, but it should be rejected if an operation is not agreed to, or if there is any danger of their rupture and fatal hæmorrhage, or of embolism if they are very large. However, the life may be accepted with an addition of some years if the varicose veins are not very large.

Skin Diseases.—Chronic skin diseases, such as psoriasis and eczema, as a rule, leave no adverse influence on the average expectation of life; but the existence of chronic eczema may sometimes lead to an attack of nephritis, and some years should be added before the proposal is accepted.

Gout.—The death rate of persons suffering from gout is usually heavy between fifty-five and sixty-five. Hence cases of this class should be insured for an endowment policy with an addition of at least five years. There is a greater risk of suffering from diseases of the heart and kidneys if an individual gets an attack of gout in the earlier age.

Acute Rheumatism.—If the attacks have been severe, compelling the patient to be in bed each time for two or three weeks and leading to heart complications, the applicant should be rejected altogether; otherwise five to ten years may be added to the actual age for rating the premium.

Syphilis.—If a man is suffering from syphilis, the date and character of the primary chancre must be mentioned. It should also be mentioned, if he had had an attack of secondary or tertiary syphilis, as also the nature and the duration of the treatment adopted by him and when it was finally discontinued. If any signs of the active disease are visible on the body, the proposal should be postponed. On the contrary, it can be accepted with an addition of about 5 years, if the applicant is above thirty years, has been carefully treated for the disease and has had no symptoms for the last two years.

Fevers.—If the applicant gives a history of having suffered from malarial fever, he should be thoroughly examined for the presence of an enlarged liver or spleen and for the existence of a cachectic appearance. An attack of ordinary fever should not affect the proposal, provided some months have passed since the last attack.

Epilepsy.—An applicant suffering from epilepsy should not be insured if there is a history of the disease in the family, or if he has acquired it after thirty years of

1. *Cecil Bosanquet, Practitioner, Nov.*, 1930, *p.* 594.

age when it is very often due to syphilis. However, in the absence of a family history, the proposal may be accepted with an addition of some five to ten years, if there has been no attack within the last ten years.

Nervous Diseases.—Hemiplegia, paraplegia, locomotor ataxy and many other nervous diseases are barriers against assurance.

Insanity.—Persons who have suffered from insanity are usually not accepted by insurance companies.

B. THE FAMILY HISTORY

An inquiry about the family history is necessary to determine if there is any hereditary influence which is likely to interfere with the longevity of the applicant. In addition to an inquiry about the general health of the living relatives, such as the father, mother, brothers, sisters, or any other near relatives, or the age at which, and the disease from which, any of them died, questions should be asked of the applicant to determine if any of them ever suffered from one of the following diseases, as they are liable to hereditary tendency :—

1. Phthisis.
2. Gout.
3. Cancer.
4. Diabetes.
5. Insanity.
6. Rheumatism.

The insurance company usually rejects all those cases which give a history of a hereditary disease but, in some cases, may accept them after loading them with a higher rate of premium by adding five to ten years to their actual age.

C. THE PERSONAL EXAMINATION

The personal examination must be carried out with minuteness and great care. The following is the order in which it has generally to be done :—

Identification.—It is very essential to describe some physical peculiarity, scar, or any other mark, which would serve the purpose for the future identification of the applicant, if necessary.

Age.—In connection with age, mention should be made as to whether the applicant looks younger or older than the avowed age. In addition, the determination of age is necessary for the calculation of *the expectation of life, i.e.,* the average number of years which he may be expected to live at that particular age.

The Rule for Calculating the Expectation of Life.—The expectation of life is calculated from life tables made up from census returns, but the rough and practical methods for the purpose of calculating this are two formulæ as given by Walford and Willich.

Walford's Formula.—The formula given by Walford is tolerably accurate for deducing the expectation of life. It is obtained by subtracting the actual age of the proposer from ninety-six, the fixed number between the ages of twenty and forty-five, and dividing the remainder by two. Between twenty and thirty the result hardly comes up to the average and over forty it is slightly above the average. For ages above forty-five, ninety should be taken as a fixed number instead of ninety-six.

Willich's Formula.—An approximate result between the ages of twenty-five and seventy-five may be obtained from Willich's formula which is $\frac{2\ (80—A)}{3}$, where "A" represents the age of the proposer. For example, at the age of forty-four, the expectation of life would be, according to this formula, $\frac{2\ (80—44)}{3} = \frac{72}{3} = 24$ years.

General Appearance.—Under this heading the figure and personal formation of the applicant should be described together with the height, weight and circumference of the chest and abdomen. All these dimensions should be carefully measured and not guessed. In taking the height and weight the applicant should be asked to take off his boots or shoes and necessary clothes. However, he may be allowed to keep his clothes on if his height is measured with his boots on, as the heels of the boots will almost counterbalance the clothes in calculating the proportion between the height and weight. To estimate this the following table for the age of 30 should be taken into consideration :—

HEIGHT.	STANDARD WEIGHT	CIRCUMFERENCE OF CHEST.
Ft.-in.	Lbs.	Inches.
5-0	112	33½
5-1	116	34
5-2	126	35
5-3	133	35½
5-4	139	36
5-5	142	37
5-6	145	37½
5-7	148	38
5-8	155	38½
5-9	162	39
5-10	169	39½
5-11	174	40
6-0	178	40½
6-1	182	41

A margin of fifteen to twenty per cent. is allowed either way under ordinary circumstances. For men over or under thirty, half a pound for every year may be added or deducted as the case may be. After fifty it is not necessary to make any addition as normally there should be very little change in build after this age.

The standard weight of Indians can be calculated from the height by Buchanan's formula by taking 100 lbs. as the average weight for a height of five feet and adding 3 lbs. in weight for every inch above that, *e.g.*, 5 feet 1 inch=103 lbs., 5 feet 2 inches=106 lbs., and so on. Twenty per cent. has to be allowed either way if there is a good family history, and all the organs are quite sound and healthy. But more allowance can be made if the weight happens to be in excess over the standard weight, provided everything else is all right as the well-to-do Indians have the habit of consuming rich food containing too much fatty and starchy substances.

The circumference of the chest should be taken by a tape measure at the level of the nipples, when the applicant is breathing normally, when he has taken forced expiration, and full inspiration. The chest measurement should not be less than half the height and a variable proportion of more than fifteen per cent. should be regarded as suspicious. The difference between forced expiration and full inspiration should not be less than two inches for a height of five feet, and three inches for six feet height.

The girth of the abdomen at the level of the umbilicus must not be more than the chest measurement.

Internal Organs.—A careful examination of the internal organs should be made by palpation, percussion and auscultation, where necessary. Any deviation from the normal should at once be described without any reservation. Before examining the heart a thorough investigation should be made as to the symptoms of cardiac disease, such as faintness, giddiness or shortness of breath, especially when there is a history of syphilis, or some specific fever, such as acute rheumatic fever, diphtheria or influenza. It is not necessary to take the blood pressure in all cases, but it should always be taken when the proposer is above forty years of age, or when there is a

suspicion of a cardiovascular or renal disease. It should also be taken in cases of gout or syphilis. Some insurance companies insist that the blood pressure should be taken in the case of individuals who propose to have their lives insured for twenty thousand rupees or more.

The normal maximal systolic blood pressure is considered roughly to be equivalent to 100 plus the age of the individual, in millimetres of mercury.

Cases having a blood pressure below 110 should be postponed for at least twelve months, especially if there is the slightest suspicion of dormant tubercle. A proposer having a blood pressure much higher than the normal for his age should be required to pay an extra premium, while a person having a blood pressure of 200 or more at any age should always be rejected.

The urine should be passed in the presence of the examiner to avoid fraud, and to obtain a fresh specimen to examine for the presence of albumen, sugar, blood or any other abnormal constituents. Diabetic applicants should always be rejected.

OPINION

Lastly the medical examiner has to give his opinion as to whether the life should be accepted at ordinary rates (first class), at enhanced rates with a certain amount of loading (2nd class), or should be rejected altogether (third class) after he has fully considered the general state of the health, the condition of all the different organs, the constitution and personal habits of the applicant and his family history.

THE MEDICAL REFEREE

In addition to the medical examiner, the insurance companies appoint a medical man as their chief medical officer or referee, who examines candidates for life assurance at the head office, reads and criticises the reports sent in by the medical examiners from all the branch offices and advises the board of directors to accept the proposed lives or to refuse them. He may also decide, after consulting the actuary, to add any extra premium, if necessary. In certain cases he may send for further information from the medical examiners, if he finds that any point is ambiguous, or not clear.

THE PROOF OF DEATH

On the death of an assured person, the insurance company, before paying the amount of money in terms of the policy to the heirs of the deceased, requires the proof of his death from the physician who attended him during his last illness, because fraudulent cases of death have been recorded to practise deception on the companies. Thus, some time ago, a case occurred in Benares, where an assured person disappeared to defraud an insurance company after having first fabricated evidence of his own death. He as well as his associates were convicted and sentenced to imprisonment by a Court of law. Another case occurred in which one Hakam Singh got the life of one Diwan Chand, a poor man, insured for Rs. 32,000, within a short period of four months, and after he got all the policies assigned in his favour he began to devise plans to bring about the death of Diwan Chand so as to enable him to claim the money due on the policies. Having failed in the various attempts in killing Diwan Chand by administering him poisons, such as hyoscine, arsenic, and poisoned meat bitten by a poisonous snake, he (Hakam Singh) rendered him unconscious by giving him liquor mixed with chloral hydrate, and induced him to sleep in a stable, where he was given a few whiffs of chloroform, petrol was sprinkled on his body, quilt and other clothes, and fire was set to them so that Diwan Chand was burned to death.[1] The companies usually value the certificates of registered medical practitioners only.

Sometimes, the question may occur as to whether death was due to an accident or not, especially if the life was insured against an accident. Death is said to be due to an accident when caused by external and violent means owing to unexpected and unforeseen occurrences.

---

1. *King-Emperor* v. *Hakam Singh, Cr. Law Jour., May,* 1930, *p.* 517.

CHAPTER XXI

LAW IN RELATION TO MEDICAL MEN

THE MEDICAL ACT

In order that persons requiring medical aid should be enabled to discriminate between qualified and unqualified practitioners, an Act called the Medical Act of 1858, was passed by the Parliament in 1858, which came into force from the first day of October, one thousand eight hundred and fifty-eight. This act created the General Medical Council, which, according to the Act amended in 1886, consists of five members nominated by the Crown with the advice of the Privy Council, eighteen members appointed by the universities in the United Kingdom having medical faculties, nine members appointed by the medical corporations, such as the Royal Colleges of Physicians and Surgeons and six members directly elected by the members of the profession as a whole. To these are added three dentists who are members of the Dental Board and are appointed for dental business.

This Council maintains the register of medical men practising in Great Britain and Ireland, has the controlling power over the discipline of the profession and over the curricula and examinations of medical schools and colleges. It also publishes the British Pharmacopœia.

By part II of the Medical Act of 1886 medical graduates of the Indian Universities recognised by the General Medical Council are entitled to be registered on the payment of a fee of five pounds as colonial practitioners in the medical register, and their names are entered in a separate list, known as the Colonial List. The General Medical Council withdrew their recognition of the degrees granted by the Indian universities from March 1, 1930, on the ground that they had no direct means to ascertain if the universities in India were maintaining a suitable standard of medical education, when in 1929 the Legislative Assembly did not sanction the financial grant necessary for maintaining a medical inspector for carrying out the annual inspection of the medical examinations. It appears that after carefully considering the reports of medical inspectors to the Indian Medical Council on Bombay, Lucknow, Patna and Madras the executive committee of the General Medical Council resolved at their meeting held on May 25, 1936, that the degrees of M.B., B.S. granted by the universities of Bombay, Lucknow and Madras (together with other qualifications granted by the universities of Bombay and Madras which were previously registrable) should again be recognised for registration if granted on or after February 25, 1930 ; and that the degrees of M.B., B.S. granted by the university of Patna should be recognised for registration if granted on or after May 11, 1935. In February, 1937, the Executive Committee of the General Medical Council also passed a resolution that holders of diplomas granted by the Punjab University on or after the 25th February, 1930, should be entitled to registration in the Colonial List. The Committee passed a similar resolution on the 24th May, 1937, recognising the diplomas of the University of Calcutta granted on or after the 16th October, 1936.

PRIVILEGES OF REGISTERED PRACTITIONERS

It is incumbent on every medical practitioner in the British Isles to have his name registered, as by the act of registration, he is entitled to practise medicine in all its branches in the United Kingdom and in any other part of His Majesty's dominions overseas, to get certain official appointments, to grant medical certificates required by an Act of Parliament, to be exempted from serving on juries and inquests or in the militia, to use certain professional titles and to recover professional fees by legal procedure, unless he happens to be a fellow of the Royal College of Physicians, since its fellows are prohibited from taking legal proceedings for the recovery of their professional fees.

In India, owing to the want of uniformity of standard in preliminary education and the medical courses in schools and colleges, and owing to the prevalent Ayurvedic and Unani systems, no Medical Act had been passed till recently to control or to restrict the medical practices. In 1916 the Government of India passed the Indian Medical Degrees Act, known as Act No. VII of 1916, to regulate the grant of titles implying qualifications in Western Medical Science, and the assumption and use by unqualified persons of such titles.

By section 6 of this Act whoever voluntarily and falsely assumes or uses any title or description or any addition to his name implying that he holds a degree, diploma, licence or certificate conferred, granted or issued by any authority recognized by the Governor-General in Council, or recognized by the General Council of Medical Education of the United Kingdom, or that he is qualified to practise Western medical science, shall be punishable with fine which may extend to two hundred and fifty rupees, or, if he subsequently commits, and is convicted of, an offence punishable under this section, with fine which may extend to five hundred rupees:

Provided that nothing in this section shall apply to the use of any person of any title, description, or addition which, prior to the commencement of this Act, he used in virtue of any degree, licence or certificate conferred upon, or granted or issued to him. Section 7 provides that no Court shall take cognizance of an offence punishable under this Act, except upon complaint made by order of the Local Government, or upon complaint made, with the previous sanction of the Local Government, by a Council of Medical Registration established by any enactment for the time being in force in the province.

Within the last few years the Provincial Governments have created Medical Councils in Bombay, Madras, Bengal, Bihar, Assam, Punjab, and United Provinces by passing the Medical Act for the registration of certain medical practitioners and supervision of medical education in their own provinces. Each of these Councils consists of members elected by the registered medical practitioners and those nominated by the Provincial Government. The administrative head of the medical department is generally the President of the Council and the Registrar or Secretary of the Council maintains a medical register for the province. Persons possessing medical qualifications included in the schedule maintained by the Provincial Medical Councils are eligible for registration on payment of the prescribed fees and on furnishing proof of the qualifications possessed by them.

It was found desirable that the Government of India should pass an Act on some such lines as the Medical Act of 1858 so as to have uniformity of qualifications and to confer almost the same rights and privileges on the registered practitioners as in Great Britain and Ireland. At the Simla session of the Legislative Assembly in 1929 the Government of India

proposed to introduce a bill for the creation of an All-India Medical Council, but had to give up the idea at the time, as it was not approved at a meeting of the ministers and the administrative heads of the medical departments of the different provinces. In June, 1930, the Government of India again convened a meeting of the ministers, administrative heads of the medical departments and the representatives of the medical faculties of various universities, and formulated a new bill for the creation of the All-India Medical Council which was introduced in the Legislative Assembly in the winter session of 1933 at Delhi. It was passed into an Act, known as the Indian Medical Council Act, 1933 (Act No. XXVII of 1933), in the autumn session at Simla and received the assent of the Governor-General on the 23rd day of September, 1933. The Act has since been modified upto the 1st December, 1937. The object of the Act is to constitute a Medical Council in India in order to establish a uniform minimum standard of higher qualifications in medicine for all provinces. The Medical Council constituted under this Act shall consist of (*a*) one member from each Governor's province, to be nominated by the Central Government, (*b*) one member from each British Indian University, to be elected by the members of the Senate of the University (or, in the case of the University of Lucknow, the Court), from amongst the members of the medical faculty of the University, (*c*) one member from each province where a Provincial Medical Register is maintained, to be elected from amongst themselves by persons enrolled on the Register who possess recognised medical qualifications or medical qualifications granted by a British Indian University, and (*d*) four members to be nominated by the Central Government. The President of the Council shall be elected by the members of the Council from amongst themselves provided that for four years from the first constitution of the Council the President shall be a person nominated by the Central Government who shall hold office during the pleasure of the Central Government and, where he is not already a member, shall be a member of the Council in addition to the members prescribed in this Act. The members of the Council shall hold office for five years.

The Medical Council recognises for the purpose of this Act those medical qualifications which are granted by medical institutions in British India and which are included in the first schedule.[1] The Medical Council also recognises the medical qualifications granted by medical institutions outside British India which are included in the second schedule,[2] and are considered sufficient qualification for enrolment on any Provincial Medical Register. The Medical Council is also empowered to complete or enter into negotiations with the authority in any state or country outside British India which by the law of such state or country is entrusted with the maintenance of a register of medical practitioners, for the settling of a scheme of reciprocity for the recognition of medical qualifications, and in pursuance of any such scheme the Central Government may, by notification in the Official Gazette, amend the second schedule so as to include therein any medical qualification which the Council has decided should be recognised.

1. Vide *the Indian Medical Council Act* 1933 *as modified upto* 1st *Dec.*, 1937, *p.* 11.
2. *Ibid., pp.* 12, 13 *and* 14.

DISCIPLINARY CONTROL

The General Medical Council exercises disciplinary control over registered medical practitioners in virtue of the 29th section of the Medical Act, 1858, which provides that—

"If any registered medical practitioner shall be convicted in England or Ireland of any felony or misdemeanour, or in Scotland of any crime or offence, or shall after due inquiry be judged by the General Council to have been guilty of infamous conduct in any professional respect, the General Council may, if they see fit, direct the Registrar to erase the name of such medical practitioner from the Register."

Infamous conduct is a technical legal expression, and means "disgraceful or dishonourable" conduct in a qualified professional person acting as such. What constitutes the disgraceful or dishonourable conduct has often been a controversial point in a Court of law. Sir Donald Mac Alister in 1892 defined it as follows :—

"If it is shown that a medical man, in the pursuit of his profession, has done something with regard to which it would be reasonably regarded as disgraceful or dishonourable by his professional brethren of good repute and competency, then it is open to the Council to say that he has been guilty of infamous conduct in a professional respect."

The Indian Medical Council is almost analogous to the General Medical Council, but it does not maintain a register of medical practitioners and does not exercise any disciplinary jurisdiction over them. These functions are carried out by the Provincial Medical Councils, which have the power to remove the names of medical practitioners permanently or for a specified period from their registers when they are judged after due inquiry to have been guilty of infamous conduct in a professional respect. They have also the power to direct the restoration of any name so removed. It must be borne in mind that the Provincial Medical Councils like the General Medical Council do not take cognizance of any offence of misconduct committed by a registered medical practitioner unless someone lodges a complaint in writing accompanied by one or more statutory declarations as to the facts alleged, when they assume the functions of professional Courts of Justice.

The General Medical Council have brought to the notice of registered medical practitioners the following statement, which summarises the resolutions and decisions of the Council upon forms of professional misconduct that have from time to time been brought before the Council in the exercise of their disciplinary jurisdiction over the members of the medical profession :

It must be clearly understood that the instances of professional misconduct which are given below do not constitute, and are not intended to constitute, a complete list of the offences which may be punished by erasure from the medical register and that by issuing this notice the Council are in no way precluded from considering and dealing with any form of professional misconduct (as, for example, immorality involving abuse of professional relationship) which may be brought before them, although it may not appear to come within the scope or precise wording

of any of the categories herein set forth. Circumstances may and do arise from time to time in relation to which there may occur questions of professional conduct which do not come within any of these categories. In such instances, as in all others, the Council have to consider and decide upon the facts brought before them.

It may be mentioned that some of the Provincial Medical Councils have issued a similar warning notice for the information of medical practitioners registered in their provinces with a view to apply the principles therein laid down so far as they are relevant under Indian Enactments.

1. *Certificates, Notifications, Reports, etc.*—Registered medical practitioners are in certain cases bound by law to give, or may be from time to time called upon or requested to give, certificates, notifications, reports and other documents of a kindred character, signed by them in their professional capacity, for subsequent use either in courts of justice or for administrative purposes.

Such documents include, among others, certificates, notifications, reports, etc.—

(*a*) Under the Acts relating to births, deaths, or disposal of the dead ;

(*b*) Under the Acts relating to lunacy and mental deficiency and the rules made thereunder ;

(*c*) Under the Vaccination Acts and the Orders made thereunder ;

(*d*) Under the Factory Acts and the Regulations made thereunder ;

(*e*) Under the Education Acts ;

(*f*) Under the Public Health Acts and Orders made thereunder ;

(*g*) Under the Workmen's Compensation Acts ;

(*h*) Under the Acts and Orders relating to the notification of infectious diseases ;

(*i*) Under the National Insurance Acts and the Regulations made thereunder ;

(*j*) Under the Old Age Pensions Acts and the Regulations made thereunder ;

(*k*) Under the Merchant Shipping Acts ;

(*l*) In connection with sick benefit, insurance and friendly societies ;

(*m*) For procuring the issue of Foreign Office Passports ;

(*n*) For excusing attendance in Courts of Justice, in the public services, in public offices, or in ordinary employments ;

(*o*) In connection with naval and military matters ;

(*p*) In connection with matters under the control of the Ministry of Pensions.

Any registered practitioner who shall be shown to have signed or given under his name and authority any such certificate, notification, report, or document of a kindred character which is untrue, misleading, or improper, whether relating to the several matters above specified or otherwise, is liable to have his name erased from the Medical Register.

2. *Unqualified Assistants and Covering.*—The employment by any registered medical practitioner in connection with his professional practice of an assistant who is not duly qualified or registered, and the permitting of such unqualified person to attend, treat or perform operations upon persons in respect of matters requiring professional discretion or skill, is in the opinion of the Council in its nature fraudulent and dangerous to the public health, and any registered medical practitioner who shall be shown to have so employed an unqualified assistant is liable to have his name erased from the Medical Register.

Any registered medical practitioner who by his presence, countenance, advice, assistance, co-operation, knowingly enables an unqualified or unregistered person, whether described as an assistant or otherwise, to attend, treat, or perform any operation in respect of any matter requiring professional discretion or skill, to issue or procure the issue of any certificate, notification, report or other document of a kindred character (as more particularly specified in division 1 hereof), or otherwise to engage in professional practice as if the said person were duly qualified and registered, is liable, on proof of the facts, to have his name erased from the Medical Register.

The foregoing do not apply so as to restrict the proper training and instruction of *bona fide* students, or the legitimate employment of dressers, midwives, dispensers, surgery attendants, and skilled mechanics, under the immediate personal supervision of a registered medical practitioner.

3. *Sale of Poisons.*—The employment, for his own profit and under cover of his own qualifications, by any registered medical practitioner who keeps a medical hall, open shop, or other place in which scheduled poisons or preparations containing scheduled poisons are sold to the public, of assistants who are left in charge but are not legally qualified to sell scheduled poisons to the public, is in the opinion of the Council a practice professionally discreditable and fraught with danger to the public, and any registered practitioner who is proved to have so offended will be liable to have his name erased from the Medical Register.

4. *Dangerous Drugs.*—The contravention by a registered medical practitioner of the provisions of the *Dangerous Drugs Acts* and the regulations made thereunder may be the subject of criminal proceedings, and any conviction resulting therefrom may be dealt with as such by the Council under the powers given them by section 29 of the Medical Act, 1858. But any contravention of the Acts or the Regulations, involving an abuse of the privileges conferred thereunder upon registered medical practitioners whether such contravention has been the subject of criminal proceedings or not, will, if proved to the satisfaction of the Council, render a registered medical practitioner liable to have his name erased from the Medical Register.

5. *Association with Unqualified Persons.*—Any registered medical practitioner who either by administering anæsthetics or otherwise, assists an unqualified or unregistered person to attend, treat, or perform an operation upon any other person, in respect of matters requiring professional discretion or skill, will be liable on proof of the facts to have his name erased from the Medical Register. But under section 16 of the U. P.

Medical Act of 1917 the adoption by a registered medical practitioner of a theory of medicine or surgery not in accordance with the accepted theory for the time being or his association with a *vaid, hakim,* or homœopath or an unregistered practitioner holding one of the qualifications recognised under the Act is not regarded as constituting infamous conduct.

6. *Advertising and Canvassing.*—The practices by a registered medical practitioner—

(*a*) of advertising, whether directly or indirectly, for the purpose of obtaining patients or promoting his own professional advantage ; or, for any such purpose, of procuring or sanctioning or acquiescing in the publication of notices commending or directing attention to the practitioner's professional skill, knowledge, services, or qualifications, or deprecating those of others ; or of being associated with or employed by those who procure or sanction such advertising or publication ; and

(*b*) of canvassing or employing any agent or canvasser for the purpose of obtaining patients ; or of sanctioning, or of being associated with or employed by those who sanction, such employment ;

are in the opinion of the Council contrary to the public interest and discreditable to the profession of medicine, and any registered medical practitioner who resorts to any such practice renders himself liable on proof of the facts to have his name erased from the Medical Register.

A registered medical practitioner is, however, justified in advertising to the public by putting up a door-plate of a modest size and containing his name and qualifications with only one speciality on his consulting room or on his residential house, but it is not proper for him to affix it on a chemist's shop or on a house where he does not reside or on the corner of a street with an arrow mark pointing the direction of his residence.

A registered medical practitioner can deliver public lectures or publish in the lay press signed articles on medical subjects, but in doing so he should not try to attract patients to himself by praising directly or indirectly his own line of treatment in a particular disease affirming that it is superior to the method of treatment adopted by other practitioners.

7. *Association with Uncertified Women practising as Midwives.*—Whereas it has been made to appear to the Council that certain registered medical practitioners have, from time to time, by their countenance or assistance or by issuing certificates, notifications, or other documents of a kindred character, enabled uncertified persons to attend women in child-birth otherwise than under the direct and personal supervision of a duly qualified medical practitioner, contrary to law ;

And whereas such conduct is in the opinion of the Council discreditable to the profession of medicine, and calculated to defeat the purpose of the Statutes made in the public interest for the protetcion of mothers and infants ;

Notice is hereby given that any registered practitioner who is proved to have so offended will be liable to have his name erased from the Medical Register.

It may be pointed out that in the United Kingdom a woman is not allowed to attend women in childbirth unless she is certified under the Midwives Act. In India, however, there is no such restriction.

The decision of the General Medical Council when given after due inquiry and without malice is final but, according to section 27 of the United Provinces Medical Act, 1917, an appeal shall lie to the Local Government from every decision of the Council under section 24 or 26.

DUTIES OF A PHYSICIAN

When a medical man is registered to engage in the practice of medicine and surgery after he has obtained a necessary degree or diploma from a university or a medical corporation, he is presumed by law—

1. To use the necessary skill, care and attention in the treatment of his patients.

2. To continue to treat his patients and to pay them visits as long as it is necessary, unless he has given due notice for discontinuing his treatment or visits, so as to enable them to obtain the services of another medical attendant, or the patients themselves have signified their intention of changing the doctor or where he is convinced that the illness is an imposture and he is being made a party to a false pretence.

There is a common belief among the public that a medical practitioner is at the beck and call of anyone who chooses to send for him, but it must be remembered that there is no law to compel a medical practitioner to attend a patient except in a case where he has previously bound himself by contractual obligations or has already undertaken the treatment.[1] Neither a police nor any other official has the right to force or commandeer a physician's services without his consent under any circumstances, except during military necessity.[2] The Coroner of Aldershot also observed in an inquest that a doctor is not obliged to attend a case if he does not want to ; he can be criticized if he promises to attend and then fails to do so, but he is perfectly entitled—like any other professional man—to say he cannot attend a case.[3] Nevertheless, it is necessary to remember that a medical practitioner should not hesitate to render medical or surgical assistance in an emergency, especially in a locality where there is no other suitable medical aid ; refusal in such a case would be considered a deriliction of moral and professional duty. A medical practitioner serving on the staff of a charitable hospital is bound to render professional services to every patient attending the institution.

3. To use clean and proper instruments and appliances.

4. To furnish his patients with proper and suitable medicines, if he is in the habit of dispensing his own medicines. If he has no dispensary of his own he should legibly write prescriptions, using such abbreviations as are usually employed and mentioning full and detailed instructions in language which the chemist or pharmacist dispensing prescriptions can

1. *Conduct of Medical Practice*, 1927, *p.* 69.
2. *Carl Scheffel, Med. Juris.*, 1931, *p.* 84.
3. *Lancet, March*, 15, 1930, *p.* 602.

readily understand. He is held responsible for any damage in health, temporary or permanent, caused to the patient as a result of his wrong or ununderstandable instructions mentioned in the prescription.

5. To give in simple language full directions to his patients or their attendants concerning the administration of remedial measures including the articles of diet. Exact quantities and precise times for the administration of medicines should be specifically mentioned.

6. To keep inviolate the secrets of his patients communicated to him by them or discovered by him at the time of the medical examination (*Vide* Appendix IX for Hyppocratic and Vedic oaths). Professional secrecy is an implied term of contract between the medical practitioner and his patient, and its disclosure would be a breach of trust and confidence and would render the medical attendant liable to damages. In France and Germany disclosure of medical secrets is regarded as a criminal offence. It must, however, be remembered that a medical witness is bound to reveal them in a judicial proceeding, if ordered by the Court.

A medical man should not answer inquiries addressed to him by an insurance company respecting a person who may have consulted him without having obtained the patient's consent which, if possible, should be in writing. But, if he is a medical examiner, he must furnish all the information acquired by him without reservation, even though it may be detrimental to the interests of his client.

It must be borne in mind that a medical man cannot keep professional secrets in those cases where he is required by law to furnish certain information to the public authorities, *e.g.*, notification of infectious diseases, mentioning the cause of death in certificates of death, etc. If he thinks that the divulging of a professional secret is in the best interests of a community or the public, he can do so, and will be absolved from legal liabilities. For instance, a medican man can inform the warden of a hostel, if any boarder suffers from a venereal disease. Such information is treated as a privileged communication.

A case[1] occurred at a turkish bath at Debretzin, where a venereal diseases specialist recognized in a young man who was about to enter the water one of his own patients who had consulted him two weeks ago for a syphilitic sore on the penis. He went up to the young man, and whispered to him not to enter the bath on account of his chancre, but the young man persisted in entering the bath ; hence the specialist sent for the manager and explained matters to him. The latter asked the man to leave at once. He left the bath, and sued the specialist for trespassing the medical secrecy laws, but lost his case, the Court decreeing that the specialist only carried out his professional duty, and acted in the interests of the community.

PHYSICIAN'S RESPONSIBILITY IN CRIMINAL MATTERS

Ordinarily it may be presumed that a medical practitioner should at once communicate to the police any information about a criminal act that might have come to his knowledge in his professional work, but this is not always the case. He should not play the part of a detective, but use his own discretion. For instance, he should hand over to the police a man, whom, from the nature of his injury he may suspect to be an assailant in

1. *Lancet, April* 16, 1921, *p.* 822.

a murder case. If he happens to treat a person who has attempted to commit suicide, he is not bound by law to report him to the proper authorities, but he has to inform the police if he happens to die. If the friends or relatives of the suicide undertake to carry the information to the police, he must see that they do.

What is the duty of a medical practitioner if he is called in to treat a case of criminal abortion ? This is a question which often arises. In the case of *Kitson* v. *Playfair and wife* in 1896 Mr. Justice Hawkins in charging a grand jury said : " he doubted very much whether a doctor called in to assist a poor, wretched woman, not in procuring abortion, for that in itself was a crime, but for the purpose of attending her and giving her medical advice how she might be cured so as to go forth about her business—would be justified in reporting the facts to the public prosecutor. To his mind a thing like that would be a monstrous cruelty. There might be cases when it was the obvious duty of a medical man to speak out. In a case of murder, for instance, a man might come with a wound which might be supposed to have been inflicted in the course of a deadly scuffle. It would be a monstrous thing if the medical man might screen him and try to hide the wound which might be the means of connecting the man with a serious crime."

The Royal College of Physicians of London took the opinion of Sir Edward Clarke, and Mr. Horace Avory on these remarks of Mr. Justice Hawkins. Their joint opinion was that a medical practitioner was not liable to be indicted for not communicating to the police the information about criminal abortion obtained by him in his professional capacity. This question again came to the forefront during the trial of a case of a criminal abortion which was held at the Birmingham Assizes in December, 1914. In this case it was alleged that an illegal operation had been performed upon a woman with a view to procure abortion. The woman subsequently died from septicæmia. She had been attended by three medical men, but none of them had given information to the police and in consequence there was no evidence upon which a jury could convict the prisoner who was charged with having performed the illegal operation. In charging the grand jury Mr. Justice Avory made the following observations[1] : —

" Under circumstances like those in the present case, I cannot doubt that it is the duty of the medical man to communicate with the police or with the authorities in order that one or other of those steps may be taken for the purpose of assisting the administration of justice. No one would wish to see disturbed the confidential relation which exists, and which must exist, between the medical man and his patient, in order that the medical man may properly discharge his duty towards his patient ; but there are cases, and it appears to me that this is one where the desire to preserve that confidence must be subordinated to the duty which is cast upon every good citizen to assist in the investigation of a serious crime such as is here imputed to this woman. In consequence of no information having been given, it appears to me, that there is no evidence whatever upon which the woman can properly be put upon her trial. I have been moved to make these observations because it has been brought to my notice that an opinion, to which I was a party some twenty years ago, when I was at the Bar, has been either misunderstood or misrepresented in a text-book of medical ethics, and I am anxious to remove any such misunderstanding if it exists. It may be the moral duty of a medical man, even in cases where the patient is not

1. *Brit. Med. Jour., Feb.* 5, 1916, *p.* 206.

dying, or not likely to recover, to communicate with the authorities when he sees good reason to believe that a criminal offence has been committed. However that may be, I cannot doubt that in such a case as the present, where the woman was, in the opinion of the medical man, likely to die, and therefore her evidence was likely to be lost, that it was his duty, and some one of those gentlemen ought to have done it in this case."

This matter was also taken up by the Royal College of Physicians of London, who after discussing it fully and obtaining Counsel's opinion on some legal points passed the following resolutions for publication to the medical profession at their meeting held on January 27, 1916 :—

The college is of opinion—

1. That a moral obligation rests upon every medical practitioner to respect the confidence of his patient, and that without her consent he is not justified in disclosing information obtained in the course of his professional attendance on her.

2. That every medical practitioner who is convinced that criminal abortion has been practised on his patient should urge her, especially when she is likely to die, to make a statement which may be taken as evidence against the person who has performed the operation, provided always that her chances of recovery are not thereby prejudiced.

3. That in the event of her refusal to make such a statement, he is under no legal obligation (so the college is advised) to take further action, but he should continue to attend the patient to the best of his ability.

4. That before taking any action which may lead to legal proceedings, a medical practitioner will be wise to obtain the best medical and legal advice available, both to ensure that the patient's statement may have value as legal evidence, and to safeguard his own interests, since in the present state of law there is no certainty that he will be protected against subsequent litigation.

5. That if the patient should die, he should refuse to give a certificate of the cause of death, and should communicate with the coroner.

The college has been advised to the following effect :

1. That the medical practitioner is under no legal obligation either to urge the patient to make a statement, or, if she refuses to do so, to take any further action.

2. That when a patient who is dangerously ill consents to give evidence, her statement may be taken in one of the following ways :—

(a) A magistrate may visit her to receive her deposition on oath or affirmation. Even if criminal proceedings have not already been instituted, her deposition will be admissible in evidence in the event of her death, provided that reasonable written notice of the intention to take her statement was served on the accused person, and he or his legal adviser had full opportunity of cross-examining her.

(b) If the patient has an unqualified belief that she will shortly die, and only in these circumstances her dying declaration will be admissible. Such a declaration may be made to the medical practitioner, or to any other person. It need not be in writing, and if reduced into writing it need not be signed by the patient nor witnessed by any other person, though it is desirable that both should be done, or that, if the patient is unable to sign, she should make her mark. If possible, the declaration should be in the actual words of the patient, and if questions are put, the questions and answers should both be given, but this is not essential. If the declaration cannot then and there be reduced into writing, it is desirable that the person to whom it is made should make a complete note of it as soon as possible.

It must be remembered that this is the view held by the Royal College of Physicians, but it has not been accepted as the law on this subject. If followed, it may land a medical practitioner into conflict and difficulty

with the legal authorities. Hence, in view of the important observations of the two eminent judges as mentioned above a medical practitioner is advised to adopt the following course of action in cases of criminal abortion :—

A medical practitioner must give the best attention in treating a case of attempted abortion or in which an illegal operation has been performed to procure abortion, but he must immediately call in another practitioner for consultation just to save his own skin in the event of a police or magisterial inquiry. He must also make a careful record of the woman's general condition and the signs present in her genital organs. It is not incumbent on him to give the information to the police, especially when the abortion has been procured by the woman herself or with the assistance of a relative or friend. If he happens to know that the abortion was induced by a professional abortionist, he must send at once a report to the police, so as to lead to the arrest of such a person. If the woman's condition is so serious that she is about to die, he must arrange to record her dying declaration as to the cause of her condition. If death occurs, he must give information to the police or magistrate for necessary action before the body has been disposed of.

DUTIES OF A PATIENT

When a patient employs a medical practitioner for the treatment of his ailment, he may reasonably be expected to supply his doctor with complete information concerning the facts and circumstances of the case, to allow him full opportunity for his own treatment, to obey his instructions and carry out his directions to the very letter as regards his diet, medicine, mode of life, and to pay him a reasonable fee for his services.

MALPRAXIS

Malpraxis, malapraxis or malapractice is defined as want of reasonable care and skill, or wilful negligence on the part of a medical man in the treatment of a patient such as to lead to his bodily injury or to the loss of his life. The law relating to malpraxis is contained in sections 52, 80, 81, 88, 89, 90, 91, 92 and 304-A of the Indian Penal Code (*vide* Appendix VII).

The question of malpraxis arises in civil Courts, when a patient raises it as a plea for not giving fees to his physician who files a suit against him for their recovery, or when a patient brings an action for damages against his medical attendant if he has suffered injury in consequence of negligent or unskilled treatment. A criminal charge is brought against him, if gross carelessness or negligence has been displayed in the treatment and has led to the death of the patient.

The question of malpraxis may also arise in criminal Courts, when the defence counsel may attribute the death of the assulted person to the negligence or undue interference of the medical attendant in the treatment of the deceased.

In such cases the medical man should be able to prove that he used reasonable and ordinary care and skill in the treatment of his patient to the best of his judgment. He is, however, not liable for an error of

judgment. The Court expects a general medical practitioner to use only the average degree of skill and knowledge which other general practitioners of his qualifications use, but does not expect him to perform a cure or bring the highest possible degree of skill and knowledge in the treatment of his patients. On the other hand, the Court expects a specialist to possess and exercise the higher degree of skill and learning in his special line than the general practitioner, and judges him by comparing him with other specialists engaged in the same line. A medical practitioner will be exonerated from a charge of malpraxis if it is proved that he exercised reasonably sound judgment in applying his medical knowledge and skill for the benefit of his patient and that he consulted, or suggested the consultation of, a brother practitioner in the diagnosis of, or treatment of, a case or that he examined, or suggested the examination of an alleged fractured or dislocated limb by X-rays. But no new treatment in the form of an experiment can be adopted without the consent of the patient or his guardian if he happens to be a minor. Again, a medical practitioner is guilty of malpraxis if he has a sore or infection, and dresses his patient, and consequently infects him or does not warn a dresser with infection. It is also advisable to inform the patient if he is suffering from an infectious disease, such as plague, small-pox, etc.

A medical practitioner, whether licensed or unlicensed, may, under the English law, be charged with manslaughter by negligence, and may, under the Indian law, be charged with having caused death by doing a rash or negligent act not amounting to culpable homicide under section 304-A, I. P. C., when a patient dies from the effects of an anæsthetic, operation, or some other treatment, if it can be proved that the death was the result of gross negligence or criminal inattention on the part of the medical attendant. In such a case there is a presumption of the absence of intention to cause death, and of the want of knowledge that the act done will most probably result in death. In a case where the accused cut out the piles of a person with an ordinary knife and, from the profuse bleeding, the person died, it was held that the accused was guilty of a rash and negligent act.[1] An unqualified person who was in charge of a dispensary had to make up a quantity of quinine mixture for cases of fever. He went to a cupboard where non-poisonous medicines were supposed to be kept and took therefrom a bottle with an outside wrapper marked 'pioson'. This wrapper he tore off and threw away. The bottle was itself labelled 'strychnine hydrochloride'; but, without regarding this and apparently because there was a resemblance between this bottle and another in which quinine hydrochloride was kept, he made up the entire contents of the bottle as if it had been quinine. The result was that seven persons died. It was held that he was guilty under section 304-A, even though he had no intention of doing any bodily harm to the deceased, and had made up the mixture with an intent to prevent or cure the fever.[2]

In order to prove that reasonable care and diligence and necessary professional skill had been exercised in the course of the treatment a

1. *Sukaroo Kobiraj,* (1887) 14 *Cal.* 566; *Ratanlal and Thakore, The Law of Crimes, Ed. XIV, p.* 765.

2. *DeSouza,* (1920) 42 *All.* 272: *Ratanlal and Thakore, The Law of Crimes, Ed. XIV, p.* 766.

medical practitioner should take the following precautions before he undertakes to administer an anæsthetic or perform an operation :—

1. The administration of an anæsthetic or the performance of an operation should not be undertaken without the consent of the patient, or his guardian, if he is minor or unconscious, after the nature and consequences of the operation have been explained to him or to his guardian. But in cases of accident or other emergencies where delay is dangerous an operation may be performed without the consent of the patient or his relative or guardian, if the medical attendant thinks that the operation is absolutely necessary to save the life of the patient.

In a case where the surgeon is not sure what he would have to do during the operation owing to some obscure signs, he should obtain a written authority to use his discretion in doing what appears to him to be in the best interests of the patient.

2. In a case of criminal wounding an operation ought not to be performed unless it is absolutely necessary. In such a case care should be taken to keep an accurate record of the state of the patient before it is performed. It is also better, before performing any operation, to get the opinion and help of another surgeon, if possible.

3. An anæsthetist should be a duly qualified man, and he should always administer a generally accepted anæsthetic, after he has examined the heart, urine, etc., of the patient to prove that he had used reasonable care and skill in administering it.

4. In the case of death from anæsthesia the surgeon or anæsthetist should at once report the matter to the police for holding a public inquiry.

RESPONSIBILITY OF A MEDICAL MAN FOR NEGLIGENT ACTS OF NURSES OR STUDENTS

A medical practitioner may be held responsible civilly, but not criminally, for a negligent act of his nurse, student or assistant employed to carry out nursing and medical duties to his patients, if the act was committed in his presence and to which he acquiesced. The principle is that "as a general rule a man is responsible for any wrongful act done by his agent or subordinate provided such act is within the reasonable scope of their employment." But he is not held responsible if the negligent act was done in his absence and the nurse, student or assistant was considered quite competent to perform the act and had traversed beyond his instructions in committing it.

The question whether a surgeon can be held liable for the negligence of one of the nurses or other attendants at an operation does not seem to have been definitely decided by Courts of law. In a case where a sponge was found to have been left in the abdomen after an operation at a general hospital in 1904, the jury found the surgeon responsible for the negligent act of the nurse whose duty was to count the sponges as she was employed by the surgeon and was under his control so long as the operation lasted in the operating theatre. The surgeon was, therefore,

ordered to pay £25 damages to the plaintiff.[1] In another case where a pair of forceps was left in the abdomen, the pronouncement of the Judge seemed to leave it doubtful whether a surgeon can leave it to the theatre nursing sister to count the swabs, sponges and instruments in an abdominal operation and thus be relieved of all responsibility. On the other hand, in the case[2] of *Van Wyk* v. *Lewis* tried in South Africa in 1924 where the plaintiff sued the surgeon claiming £2,000 damages for leaving a swab in the abdomen after an operation on the gall-bladder, the Judge of the lower Court decided against her. An appeal was lodged and the three Judges of the Appeal Court affirmed the judgment of the Court below. One of the Judges (Innes, C.J.) laid down the following proposition :—

" Assuming that the sister was negligent in her check it does not follow that the surgeon is liable for the consequences. The contention is, in fact, disposed of by the opinion already expressed as to the independent part in the operation played by the surgeon and to the reasonableness of relying upon her count. She was not the servant of the surgeon. She was under the general control during the operation, but she was also a collaborator to whom as already pointed out, it was reasonable to entrust the work of counting and checking the swabs. It was urged that the surgeon contracted to do work which required special skill and that he could not, without the consent of the patient, devolve that work on any other person. That argument would make the surgeon liable for every fault of the anæsthetist also. But the real position is that the respondent undertook an operation in the performance of which he was bound to exercise all reasonable care and skill. If it was consistent with the exercise of such care to rely upon the sister to check the swabs, thus setting himself free to devote all his energies to the surgical details of the operation, then he is not liable for her negligence."

In the case[1] of *Mahon* v. *Osborne* tried at the Manchester Assizes where a swab had been left behind in the patient's body after an operation, it was held by the jury that the surgeon was not to delegate the task of checking the swabs to the sister who was trained to undertake it, but that he must somehow keep count of every swab while his mind was intent upon the all engrossing complexities of the operation. Mrs. Mahon, the plaintiff, was awarded £616 3s. 6d. and costs against Mr. R. P. Osborne, the surgeon who had relied on the counting and checking of the swabs by an experienced theatre staff. The surgeon appealed against this decision, and the Court of Appeal in delivering judgment directed that there should be a new trial of this case and laid down that the leaving of a swab in the patient's body was not *ipso facto* negligence on the part of the surgeon and that some positive evidence of neglect was necessary before the plaintiff could succeed.[2]

The managers of a charitable hospital cannot be held responsible for the negligent acts of the members of the hospital staff in matters relating to the professional treatment of the patients in their charge if it can be

1. *Byrne* v. *Thorne ; Dixonmann, Forens. Med. and Toxic., Ed. VI, p.* 264.
2. *The Med.-Leg. and Criminolog. Rev., Vol. I, Part III,* 1933, *p.* 195.
3. *Lancet, May* 7, 1938, *p.* 1075.
4. *Lancet, Feb.* 18, 1939, *p.* 399.

proved that the managers exercised "the due care and skill" in selecting the properly qualified and experienced medical staff. The physicians and surgeons employed in the hospital are not the servants of the managers, as the latter cannot interfere with the professional treatment of the patients, nor can they issue any orders to the medical staff in this connection. In respect of the nursing staff, the managers are liable for the negligence connected with their "administrative or ministerial" duties but not with their professional duties.[1] The ministerial or administrative duties consist in the provision of proper and adequate food, bedding, shelter, the summoning of medical aid in cases of emergency, etc. The professional duties refer to the careful and skilful nursing carried out by the nurses.

Cases.—1. *Southern* v. *Skyrme and Thomas.*—In this case Mr. T. A. Southern, a consulting mining engineer, brought an action for alleged negligence against Dr. Skyrme and Mr. Lynn Thomas of Cardiff in connection with the treatment of an injury sustained on July 19, 1904.

The plaintiff, while riding his bicycle in Cardiff, came into collision with a trap, and was knocked down, bruising his face, wrist, hand, knee and ankle on the right side, and sustaining a fracture of the surgical neck of the humerus, complicated by subglenoid dislocation of the head of the bone, and much bruising of the soft parts about the shoulder. The dislocation was reduced by the defendants but the head of the bone remained rotated with the articular surface inwards, and the fractured surface outwards. The fractured end of the shaft was drawn upwards and inwards into the axilla, and the fractured surfaces could not be brought into end-to-end apposition. The best that could be done in the circumstances was done, the bones were brought into lateral apposition and union took place; the plaintiff recovered with an arm, the movements of which were limited. Skiagrams were taken which showed the displacement of the head, and the fact that the bones had united not by end-to-end but by lateral apposition. Upon these skiagrams the plaintiff founded his action for damages, claiming £2,000. At the first trial heard at the Cardiff Assizes in December, 1905, the jury disagreed and were discharged. At the second trial heard before Mr. Justice Bigham and a special jury in the King's Bench Division in November, 1906, several eminent surgical specialists gave evidence. The jury gave a verdict against both defendants, damages £100.—*Brit. Med. Jour.*, *Dec.* 16, 1905, *p.* 1620; *Ibid.*, *Dec.* 1, 1906, *p.* 1607; *Ibid.*, *Dec.* 8, 1906, *pp.* 1658, 1673.

2. *Tughan* v. *Darnell.*—In this case damages were recovered against a medical practitioner for negligence in prescribing belladonna liniment without warning those who were nursing the patient of the dangerous character of the drug and the consequent need for caution in using it. It was established that the defendant acted in accordance with the ordinary usage of the profession, giving the directions usual in the circumstances, and it might be supposed that, having done this he would be held to have exercised the care which a prudent and reasonable man must use in order to be exonerated from a charge of negligence. The learned Judge, however, ruled that this standard was not sufficient and that the law requires that a physician should use the highest degree of care, prudence and foresight known to the law; this he referred to as having been well defined by another Judge as "consummate care, prudence and foresight." The jury found for the plaintiff, damages £65, and judgment was entered accordingly.—*Brit. Med. Jour.*, *April* 7, 1906, *pp.* 815, 833.

3. *Operation in a State of Drunkenness.*—A physician had been convicted at the Durham Assizes of the manslaughter of a miner's wife on whom he operated for eclampsia while he was in a state of drunkenness. It was proved that the woman died two days later from the injuries received during the operation which were due to want of reasonable skill and care owing to intoxication. He was sentenced by the Judge to twelve months' imprisonment.—*Jour. Amer. Med. Assoc.*, *April* 15, 1922, *p.* 1139.

1. *Brit. Med. Jour.*, *April* 29, 1933, *p.* 767.

4. *Hillyer* v. *Bartholomew's Hospital.*—In this case the plaintiff, a medical man, sued the defendant institution for damages on account of injuries alleged to have been caused to him while being examined under an anæsthetic through the negligence of the defendant's staff. The examination was conducted by a surgeon, chosen by the plaintiff himself. While upon the operating table the plaintiff's left arm was permitted to come in contact with a hot water tin projecting from beneath the table; in addition the upper part of his right arm was bruised by some one pressing against it during the examination. As a result traumatic neuritis and paralysis of both arms followed, which had prevented the plaintiff exercising his profession. The case came on for hearing before Mr. Justice Grantham who refused to leave the question of negligence to the jury and gave judgment for the hospital. This decision was left undisturbed by the Court of Appeal, who held that the action was not maintainable. The only duty that the hospital owed to its patients was that it had used due care and skill in the selection of its staff. The Court also held in connection with the members of a hospital staff engaged in an operating theatre that as soon as the door of the theatre or operating room had been closed on them for the purpose of an operation they ceased to be under the orders of the hospital authorities and were at the disposal and under the sole orders of the operating surgeon until the whole operation had been completely finished, the surgeon was, for the time being, supreme, and the hospital authorities could not interfere with or gainsay his orders.—1909, 2 *K. B.* 820.

5. In the case of *Nance* v. *Beatie* (*Kansas*) the evidence tended to show that the defendant, a dentist, treated the plaintiff's jaw several weeks after he made an unsuccessful attempt to remove an impacted wisdom tooth. He then dismissed the case advising the plaintiff that she needed no further professional attention. A few days later, another dentist extracted the tooth apparently with little difficulty, and found the jaw infected. Pus flowed freely from the wound. The Court thought that the negligence was sufficient to uphold the verdict of the jury in favour of the plaintiff.—*Jour. Amer. Med. Assoc., Nov.* 9, 1929, *p.* 1500.

6. A case occurred in Paris where while a dentist was treating a young woman, a small sharp instrument, termed a "nerve puller", slipped from his hand and fell into her throat. Later it necessitated a serious surgical operation on the stomach. The client brought a suit against the dentist, but the Court before rendering a decision heard the testimony of expert witnesses who declared that the dentist was not guilty of any tort, as the dropping of the nerve puller was due to the imprudence of the client, who had seized his hand. However, the Civil tribunal did not take the view of the experts, and decided that in not foreseeing the reactions of his patients, and in not taking all precautions to prevent such an accident, the dentist had been guilty of negligence, and imposed on him a fine of 20,000 francs in addition to the cost of the operation that the patient had been obliged to undergo.—*Jour. Amer. Med. Assoc., Dec.* 28, 1929, *p.* 2041.

7. A hot water bottle used to warm a bed was negligently left in it. An unconscious patient, after an operation, was placed in the bed in such a position that the hot water bottle lay between her shoulders, and as a result she was severely burnt. While she was recovering from the anæsthetic the operating surgeon came into the room. She complained to him bitterly of the pain between her shoulders, but he paid no attention to her complaint and saying that she "had a fine jag on," left the room. Thereafter she sued the surgeon for damages, and judgment was rendered in her favour. It was held that when his patient complained, the surgeon owed her the duty of making an examination, hence he showed carelessness to that extent.—*Haring et al* v. *Banks* (*N. J.*), 146 *A.* 67; *Jour., Amer. Med. Assoc., April* 12, 1930, *p.* 1170.

8. *Babu Benarsidas Kankan* v. *Major Shyam Behari Lal.*—In August, 1925, Babu Benarsidas Kankan who was a munsif at Tilhar consulted Major Shyam Behari Lal, Civil Surgeon of Shahjahanpur, as he was suffering from dyspepsia, sleeplessness and palpitation of the heart. According to the plaintiff's case the doctor made a very superficial examination and said that there could not be anything wrong with the plaintiff's heart, but some medicine should be prescribed for his bad digestion.

When the civil surgeon was writing out a prescription the plaintiff told him that occasionally he felt dryness in his ear and used to find an accumulation of white matter like dry wax in his ear. The doctor prescribed a mixture of carbolic acid and paraffin for the ear trouble also telling him to use three or four drops of the mixture whenever he felt dryness or irritation in the ear. The plaintiff got the prescriptions dispensed by a chemist at Bareilly but did not use the prescription for the ear drops as he did not feel any further trouble in his ear. On October 10, 1925, the doctor gave the plaintiff a certificate recommending him for three months' leave on account of the plaintiff's suffering from neurasthenia. The doctor in giving history of the case did not mention anything about the ear trouble as it appeared that the plaintiff made no complaint of such trouble after August 10, 1925.

The plaintiff had no recurrence of the irritation in his ear until the end of July, 1926. On August 1, 1926, he had the doctor's prescription for ear drops dispensed by a chemist at Aligarh where he was stationed. On August 3, the plaintiff had three or four drops of the medicine dropped into his ear by his brother, Hirday Narain, at 8 or 9 p.m. As soon as the drops were applied the plaintiff felt an acute burning sensation in his ear which caused considerable pain, but after a time the pain subsided and the plaintiff was able to go to sleep. Next morning he felt pain in his right ear and sent for Dr. Chand Behari Lal of Aligarh who said that the ear appeared to have been burnt by some medicine. The plaintiff consulted several doctors about his ear and adopted proper medical treatment, but after the inflammation had subsided it was found that the drum of the right ear had been practically destroyed. The hearing of the ear had become very defective and he suffered from troublesome noises in the ear. The plaintiff sued Major Shyam Behari Lal who had prescribed the ear drops and Amulya Charan De, the chemist who had dispensed the prescription, in the Additional Judge's Court at Aligarh, claiming Rs. 15,000 as damages for the permanent injury to his ear and for the mental and physical pain and for the expenses for consulting a number of doctors.

The Additional Judge dismissed the plaintiff's suit, and so the plaintiff appealed to the division bench consisting of Justices Bannerji and King at the High Court, Allahabad. The doctor denied that he was guilty of negligence or want of medical skill in giving the prescription and maintained that the prescription was medically correct and could not have caused the injury if properly dispensed and used. He also contended that the plaintiff was not justified in using the prescription about a year after it had been given without obtaining fresh medical advice. The chemist asserted that he had dispensed the prescription correctly and that he was not guilty of any negligence.

On consideration of all the evidence their lordships agreed with the Court below in finding that the doctor was not negligent in his examination and diagnosis, but was negligent in prescribing a novel prescription for which no authority could be found, although the complaint for which it was prescribed was not uncommon. They found also that the mixture was dangerous in the sense that it was likely to cause harm to the plaintiff's ear unless it was applied after a vigorous shaking and that the doctor had no justification for prescribing such a novel and dangerous mixture for a petty complaint. They found also that the doctor did not give any clear warning as to the necessity of shaking the mixture before use. If such directions were essential to avoid the risk of harm he should have entered the necessary directions in the prescription itself.

As regards the chemist their lordships agreed with the Court below that he was not guilty of negligence or breach of duty. He had admittedly dispensed the prescription correctly. He had labelled the bottle as 'poison' and as there was no direction in the prescription about shaking the mixture, their lordships held that he was not to blame in failing to label the bottle 'shake the bottle'. The same prescription had been dispensed in precisely the same manner by two firms of Calcutta chemists and also by a chemist in Bareilly. Their lordships found no reason, therefore, for holding that the chemist was guilty of negligence or lack of skill. Their lordships accordingly dismissed the appeal as against the chemist with costs, and allowed the

appeal as against the doctor to the extent of granting the plaintiff a decree for Rs. 4,000 against the doctor with proportionate costs in both Courts.—*Leader, May* 29, 1932, *p.* 157.

9. *Manslaughter by Negligence.*—An unqualified practitioner treated a patient who complained of pain in the chest, and dissuaded him from consulting a medical man. Hæmoptysis and high fever developed, but he continued to treat the patient on his own responsibility. On the fourth day the family introduced a medical man who diagnosed severe pneumonia and pleurisy and two days later the patient died. The unqualified practitioner was prosecuted for manslaughter by gross negligence and sentenced by the Court to three months' imprisonment. According to the verdict his negligence consisted in having undertaken the case without any medical training. It was further considered negligence that the patient was dissuaded from consulting a medical practitioner.—*Lancet, Jan.* 24, 1931, *p.* 213.

THE WORKMEN'S COMPENSATION ACT.

Under the Workmen's Compensation Act, 1923 (Act VII of 1923) as modified up to the first August, 1938, an employer is liable to pay compensation to a workman employed on monthly wages not exceeding three hundred rupees, if personal injury is caused to him by accident arising out of and in the course of his employment provided that the employer is not so liable—

(*a*) in respect of any injury which does not result in the total or partial disablement of the workman for a period exceeding seven days ;

(*b*) in respect of any injury, not resulting in death, caused by an accident which is directly attributable to—

(i) the workman having been at the time thereof under the influences of drink or drugs, or

(ii) the wilful disobedience of the workman to an order expressly given, or to a rule expressly framed, for the purpose of securing the safety of workmen, or

(iii) the wilful removal or disregard by the workman of any safety guard or other device which he knew to have been provided for the purpose of securing the safety of workmen.

If the workman is killed from an accident arising out of and in the course of his employment, his dependants will be entitled to compensation for his death. The Act further provides that if a workman employed in any employment specified in the following Part A of Schedule III contracts any disease peculiar to that employment or if a workman, whilst in the service of an employer in whose service he has been employed for a continuous period of not less than six months in any employment specified in the following Part B of Schedule III, contracts, any disease specified therein as occupational disease peculiar to that employment, the contracting of the disease shall be deemed to be an injury by accident for purposes of compensation and, unless the employer proves the contrary, the accident shall be deemed to have arisen out of and in the course of the employment :—

Schedule III. List of Occupational Diseases.

Occupational diseases.	Employment.
Part A	
Anthrax.	Any employment— (*a*) involving the handling of wool, hair, bristles or animal carcases or parts of such carcases, including hides, hoofs and horns ; or (*b*) in connection with animals infected with anthrax ; or (*c*) involving the loading or unloading or transport of any merchandise.
Compressed air illness or its sequelæ.	Any process carried on in compressed air.
Poisoning by lead tetra-ethyl.	Any process involving the use of lead tetra-ethyl.
Poisoning by nitrous fumes.	Any process involving exposure to nitrous fumes.
Part B	
Lead poisoning or its sequelæ excluding poisoning by lead tetra-ethyl.	Any process involving the use of lead or any of its preparations or compounds except lead tetra-ethyl.
Phosphorus poisoning or its sequelæ.	Any process involving the use of phosphorus or its preparations or compounds.
Mercury poisoning or its sequelæ.	Any process involving the use of mercury or its preparations or compounds.
Poisoning by benzene and its homologues, or the sequelæ of such poisoning.	Handling benzene or any of its homologues and any process in the manufacture or involving the use of benzene or any of its homologues.
Chrome ulceration or its sequelæ.	Any process involving the use of chromic acid or bichromate of ammonium, potassium or sodium, or their preparations.
Arsenical poisoning or its sequelæ.	Any process involving the production, liberation or utilisation of arsenic or its compounds.

Occupational diseases.	Employment.
Part B—Contd.	
Pathological manifestations due to— (*a*) radium and other radio-active substances ; (*b*) X-rays.	Any process involving exposure to the action of radium, radio-active substances, or X-rays.
Primary epitheliomatous cancer of skin.	Any process involving the handling or use of tar, pitch, bitumen, mineral oil, paraffin, or the compounds, products or residues of these substances.

A commissioner appointed by the Provincial Government will not entertain any claim for compensation unless notice of the accident has been given to him as soon as practicable after the occurrence of the accident and unless the claim is preferred before him within one year of the occurrence of the accident or, in the case of death, within one year from the date of death. The commissioner may, for the purpose of deciding any matter referred to him for decision in connection with any claim for compensation, choose one or more persons possessing special knowledge of any matter relevent to the matter under inquiry to assist him holding the inquiry. He has all the powers of a Civil Court under the Code of Civil Procedure, 1908, for the purpose of taking evidence on oath and of enforcing the attendance of witnesses and compelling the production of documents and material objects, and he is also deemed to be a Civil Court for all the purposes of section 195 and of Chapter XXXV of the Code of Criminal Procedure, 1898. The commissioner is required to take down the evidence of a medical witness word for word as far as possible, although he is allowed to make a brief memorandum of the substance of the evidence of every other witness in the proceeding. If he thinks fit, he may submit any question of law for the decision of the High Court and, if he does so, he is required to decide the question in conformity with such decision. An appeal lies to the High Court from certain orders of the Commissioners provided a substantial question of law is involved and the amount in dispute in the appeal is not less than three hundred rupees.

A qualified medical practitioner is usually asked to examine a workman either on his own behalf or on the behalf of the employer, and to give his opinion as to whether the workman is partially or totally disabled from an accident or occupational disease. In such cases the medical practitioner must be very careful in making a thorough examination of the injured workman before he pronounces his opinion, inasmuch as he is apt to exaggerate the symptoms or to practise deliberate fraud and to delay the recovery. The medical practitioner should not, however, approach every case of accident with a suspicious mind, as owing to

financial anxiety from insecurity of compensation the injured workman may develop anxiety neurosis which is likely to prejudice his recovery to a great extent.

MALINGERING OR FEIGNED DISEASES

Malingering or shamming a disease or injury or exaggerating its effects is very common in India and is usually practised by soldiers or policemen to evade their duties, by prisoners to avoid hard work, by persons to evade legal responsibility for their criminal conduct, by workmen to claim compensation under the Workmen's Compensation Act, or by beggars to excite the sympathy of charitable people. Similarly, an assaulted person tries to aggravate the effects of injuries or simulates them when he has none, so as to mislead the medical jurist. A medical man who has any experience of medico-legal or police work in India must have come across such cases of feigned diseases and injuries.

The number of diseases shammed by a malingerer is legion. Ophthalmia, dyspepsia, intestinal colic, diabetes, spitting of blood, ulcers, burns, feigned abortion, rheumatism, lumbago, neurasthenia, nervous diseases, such as aphasia, sciatica, vertigo, headache, epilepsy, insanity and paralysis of the limbs, and feigned bruises and injuries of the internal organs, are very common. In some cases it is quite easy to find out the deception, but in others it is difficult to find out whether an individual is shamming or not. In such cases the medical man should bear in mind the following hints before he decides the question of malingering :—

1. Keep the patient under observation and have him carefully watched without his knowledge.

2. Pay him several unexpected visits before you decide on the case.

3. Hear patiently the history of the case and compare the symptoms, if they refer to a particular disease or a group of diseases, and find out if there are any discrepancies in his description of the symptoms of the disease which he simulates.

4. Have all the bandages and dressings removed. An injured person often comes to a medical man with the application of turmeric (*haldi*) on the body. It should be thoroughly washed and wiped out to ascertain if there are any abrasions or bruises on the body.

5. Try to find out the motive of deception in each case.

6. Be chary in giving credence to the story of the bystanders or relatives of the malingerer.

7. Examine each and every organ carefully and thoroughly.

8. Suggest in the presence of the patient some heroic method of treatment, such as the application of an actual cautery or some severe operation. In one case, where an assaulted man pretended aphasia, he started speaking when he was laid down on the operating table and a big amputation knife was shown to him to open his skull to find out the injury on his brain. I have often succeeded in making the malingerers

admit their deception by applying strong currents of electricity or Liston's long splint, or by administering some nasty drug, such as castor oil, etc.

9. Administer an anæsthetic, if necessary.

FITNESS FOR GOVERNMENT SERVICE

The medical man, especially the civil surgeon, has to give a certificate of fitness before a person is recruited into Government service. The age limit, under ordinary circumstances, is twenty-five, but in some cases it is twenty-eight and in exceptional cases the Local Government may waive the question of age in favour of their employee if he happens to be over age. The diseases which may render an individual unfit for Government service, civil or military, are syphilis, phthisis, hernia, hydrocele, varicocele, varicose veins, diseases of the eyes constituting colour blindness, or short sight beyond — 4 or some organic disease, such as optic atrophy, etc.

WILLS

Ordinarily a medical man has got nothing to do with the drawing up of a will, as the proper person to write a will is the legal adviser of the patient but, on occasions, the medical man may be consulted about the making of a will when the patient is lying at the point of death (*in extremis*), or when the patient wishes to make his medical attendant his legatee, a witness or an executor. In the latter case the question of using undue influence on the patient by the medical man may be brought up in Court to invalidate the will.

The following hints in connection with the making of a will are necessary to be followed : —

1. Any person who has attained the age of majority and is of sound mind can make a will to dispose of his property.

2. The medical man should find out that the testator, at the time of making his will, is in full possession of his intellect to understand what he is doing.

3. If the testator bequeaths his property to the medical man or his wife he should at once inform the natural heirs of the fact, and consult his legal advisers if he has the least suspicion that the question of " undue influence " will be raised later in Court.

4. In drawing up a will it is not necessary to use legal technicalities, but a plain and simple language must be used.

5. A will should be begun with " This is the last Will and Testament of me——of——", and should be ended thus " and hereby I revoke and make void all former or other Wills and codicils."

6. The name of the place and the date, month and year should be entered at the end before the testator signs the will or directs someone else to sign it in his presence. In the latter case the testator must acknowledge to the witnesses that the signature was made in his presence and by his direction.

7. Any alterations, if made in the will, should be initialled by the testator and witnesses.

8. No scratches, erasions, or alterations must be made after the will is executed.

9. A clause appointing an executor should be inserted thus "And I do hereby nominate, constitute, and appoint A. B. an executor/executrix of this my Will."

10. Two witnesses, present at the time, must sign the following attestation at the end of the will after the testator has affixed his signature "signed by the said testatrix/testator and acknowledged by him/her to be his/her last Will and Testament in the joint presence of us, and subscribed by us as witnesses in the presence of the said testator/testatrix and of each other." It should be borne in mind that a witness to a will may not be a beneficiary. If the patient left a sum of money to his doctor and the doctor had signed as a witness, this part of the will would be invalid.

11. The occupation and address of the witnesses should be given after their signatures.

SECTION II

TOXICOLOGY

CHAPTER XXII

POISONS AND THEIR MEDICO-LEGAL ASPECT

Definition.—It is difficult to give an exact definition of the term "poison," for substances which are harmless to the body in certain conditions may become dangerous in other conditions. For instance, the salts of potassium are not only not poisonous in small doses, but are essential for the maintenance of a healthy condition of the body. In large quantities, however, they act as acute poisons, capable of destroying life. Broadly speaking, a poison may be defined as a substance of the nature of a drug which, if administered in a way and in an amount in which it is likely to be administered, will produce deleterious effects of a serious nature. This, however, only applies to the term as usually employed. It does not cover the poisonous gases, which are not substances of the nature of a drug. But they are not often used criminally, except during warfare.[1]

Law relating to Poisons.—In cases of criminal poisoning in India the law does not insist on the precise definition of a poison, since sections[2] of the Indian Penal Code dealing with the offences relating to the administration of a poison make use of such self-explanatory terms as "any poison or any stupefying, intoxicating, or unwholesome drug, or other thing", or "any corrosive substance or any substance which it is deleterious to the human body to inhale, to swallow, or to receive into the blood". With regard to "any poisonous substance" used in section 284 of the Indian Penal Code[3] all that the law requires is that the substance is such as, if taken, will be dangerous to life, or likely to cause hurt or injury to any person. Again, the law takes cognizance of malicious intention of the individual who administers a drug or other substance with a view to cause injury or death, irrespective of the quantity or quality of the substance.

Sale of Poisons.—In England the laws restricting the sale of poisons are strictly enforced. Hence it is very difficult to obtain poisons. Legally qualified druggists alone are allowed to sell or dispense poisons. According to the Pharmacy Act passed in 1868, amended in 1908 and consolidated

1. *The Geneva Protocol of* 1925 *prohibits the use of poisonous or asphyxiating gases in warfare, but it is alleged that the Italians used them in the Italo-Ethiopian war.*
2. Vide *Appendix VII, Sections* 328, 324 *and* 326, *I.P.C., as also sections* 299 *and* 304 *A, Indian Penal Code.*
3. Vide *Appendix VII.*

into the Pharmacy and Poisons Act, 1933, the druggist can sell certain poisons only to a person known to him, or introduced by some one known to him, and has to make an entry in the register regarding the name and quantity of the poison sold, the name and address of the person to whom it is sold, the purpose for which it is sold, and the date of sale. The entry has to be signed also by the purchaser, as well as by the introducer. But in India the laws are not strict enough. Poisons can be bought at any chemist's or druggist's. In fact there was no law in the whole of British India, restricting the sale of poisons, until the Poisons Act was passed in 1904 by the Governor-General in Council providing for regulating the possession and sale of all poisons in certain local areas and the importation, possession and sale of white arsenic without a licence throughout the whole of British India. This Act was repealed, and another Poisons Act[1] (Act No. XII of 1919) was passed in 1919, which extends to the whole of British India, including British Baluchistan and the Sonthal Parganas. Under this Act the Governor-General in Council may, by notification in the Gazette of India, prohibit except under and in accordance with the conditions of a licence, the importation into British India of any specified poison, and may by rule regulate the grant of licences. Subject to the control of the Governor-General in Council, the Local Government may by rule regulate within the whole or any part of the territories under its administration the possession for sale and the sale, whether wholesale or retail, of any specified poison. The Local Government may also by rule regulate the possession of any specified poison in any local area in which the use of such poison for the purpose of committing murder or mischief by poisoning cattle appears to it to be of such frequent occurrence as to render restrictions on the possession thereof desirable.

Under the rules[2] made by the Government of the United Provinces in exercise of these powers a medical practitioner who does not possess qualifications registrable under the United Provinces Medical Act, 1917, is not to be granted a licence for the sale of any of the poisonous preparations of the British Pharmacopœia enumerated in item No. 6 of the schedule.[3]

It is also provided that a licence-holder shall not sell powdered white arsenic to any person unless the same is, before the sale thereof, mixed with soot in the proportion of an ounce of soot at least to one pound of the white arsenic, or with indigo or Prussian blue in the proportion of half an ounce of indigo or Prussian blue to one pound of arsenic, and so on in proportion for any greater or less quantity :

Provided that the licensing authority may, after full investigation and reference, if necessary, to higher authorities, permit on such conditions and with such restrictions as it thinks necessary any licence-holder to sell white arsenic without any admixture.

With a view to regulate the cultivation, manufacture, importation, exportation, possession, sale and use of Dangerous Drugs, especially those

1. Vide *Appendix X.*
2. Vide *Appendix X for other rules by the U. P. Government under section 2 of the Act.*
3. Vide *Section 2 of the rules made by the U. P. Government under section 2 of the Act in Appendix X.*

derived from opium, Indian hemp and coca leaf in accordance with the Geneva Convention or in pursuance of any international convention, the Indian Legislature passed in 1930 the Dangerous Drugs Act (Act No. II of 1930), which extends to the whole of British India, including British Baluchistan and the Sonthal Parganas, and provides uniform penalties for offences relating to the dangerous drugs.[1] This Act was amended in 1933 and 1938 and the amended acts are known as the Dangerous Drugs Amendment Act, 1933 (Act No. XXVI of 1933) and the Dangerous Drugs Amendment Act, 1938 (Act No. III of 1938).

The object of the Dangerous Drugs Acts of Great Britain, 1920-32, and of the statutory regulations made thereunder is to prevent traffic in dangerous drugs, and to endeavour to keep the use of such drugs entirely under the control of the medical profession. Persons authorised to keep these drugs include qualified medical practitioners, registered dentists, registered veterinary surgeons and pharmacists, who are employed or engaged in dispensing medicines at a public hospital or other public institution. According to these Acts a medical practitioner who prescribes any of these dangerous drugs must date and sign the prescription with his usual signature, giving his full name and address except in the case of a health insurance prescription, must state the name and address of the patient in full and must specify the total amount of the drug to be supplied. The pharmacist or dispenser must retain possession of the prescription, and keep it on the premises where it was dispensed, unless it is a health insurance prescription. In no circumstances must the patient be allowed to have possession of it. The prescription must not be dispensed more than once, unless the prescription authorises repetition at repeated intervals, but in all cases the total number of dispensings must not exceed three. The medical practitioner is required to keep and preserve for two years prescriptions, records, and registers, etc., of all such drugs purchased and used in his practice. All these must at all times be available for inspection. The medical practitioner who administers any of these drugs or of their salts to his patient in his presence or under his direct supervision is under no obligation to keep the records required by the regulations.

The Therapeutic Substances Act, 1925, makes provision for regulating the manufacture, sale, and importation into Great Britain and Northern Ireland of certain therapeutic substances, such as vaccines, sera, toxins, antitoxins, antigens, salvarsan and analogous substances, insulin, and preparations of the posterior lobe of the pituitary body. Under this Act no person shall manufacture for sale or import into Great Britain or Ireland any of these substances unless he holds a licence from the Ministry of Health. This Act does not apply to the preparation by a registered medical practitioner of a therapeutic substance for any of his own patients or for an individual patient of another such practitioner. The Therapeutic Substances Regulations, 1931, prescribe the detailed standards of sera, etc.

The rules regarding the custody and dispensing of poisonous drugs in all hospitals and dispensaries in India provide that all poisonous drugs shall be issued by the Government Medical Storekeeper with labels

1. Vide *Appendix XI.*

printed on orange-coloured paper with the word "Poison" in large English and Vernacular characters affixed to all bottles, vessels, etc., containing such articles, that they shall be kept separate from all others in an almirah, box or drawer to which the word "Poisons" shall be affixed, that written prescriptions containing poisons shall be dispensed by a Provincial Subordinate Medical Service or Provincial Medical Service officer attached to a dispensary and not by a compounder unless he (or she) has put in at least four years service, and that a copy of these rules pasted on stiff paper or board shall be suspended in every apartment where medicines are dispensed.[1]

Errors in the Dosage of Poisons in Prescriptions.—The dispenser or druggist is liable to punishment for any harm accruing to a patient from a prescription containing an unusually large dose of a poison, if it is dispensed by him. It is, therefore, necessary that he should always bring the errors of such over-doses to the notice of the prescriber by writing a private letter to him before the medicine is dispensed.

Poisoning in India.—Human poisoning, as well as cattle poisoning, are both prevalent in India.

Human Poisoning.—Both suicidal and homicidal cases of poisoning are much more common in India than in England owing to the facility with which poisons can be had in any *bazaar*. Again, accidental cases of poisoning are not unfrequently met with on account of the carelessness with which the earthen pots containing innocuous and poisonous roots and drugs are indiscriminately mixed up in a so-called grocer's shop. Accidental cases of bites by venomous snakes frequently occur in India.

The poisons that are chiefly used for suicidal purposes are opium and arsenic. Sometimes, potassium cyanide, hydrocyanic acid, oxalic acid, or one of the corrosive acids is used.

The poisons that are usually selected for the purposes of homicide are arsenic, pounded glass, mercury, copper, antimony, aconite, oleander, nux vomica, and *madar*. Opium is, sometimes, used to kill children or intoxicated persons.

Datura is used, not as a rule, with homicidal intent, but for the purpose of stupefying persons to facilitate theft or robbery. In rare cases cannabis indica, chloral hydrate and chloroform are also used for the same purpose.

Cattle Poisoning.—This is resorted to by *Chamars* who deal in hides. The poisons employed to destroy cattle are often arsenic, abrus precatorius, yellow oleander and sometimes aconite. A common mode in which arsenic is administered to an animal is to make a small quantity of white arsenic into a paste with some flour dough, and then to wrap it up in some fresh grass or stems of the grain plant. Sometimes, a bamboo *sui* is armed with arsenic paste and thrust into the tongue of an animal, especially in the Punjab. Other poisons that are also used, though rarely, are mercuric chloride, copper sulphate, lead oxide, croton, nux vomica, *madar* juice and snake venom.

1. *The U. P. Medical Manual*, 1934, *pp.* 61, 62.

CLASSIFICATION OF POISONS

Poisons are classified according to the chief symptoms which they produce on the body, as follows :—

I. Corrosives.—Strong acids and alkalies.

II. Irritants.—A. Inorganic.—

Non-metallic.—Phosphorus, Chlorine, Bromine, Iodine.

Metallic.—Arsenic, Antimony, Mercury, Copper, Lead, Zinc, Silver, etc.

B.—Organic.—

Vegetable.—Castor-oil seeds, Croton oil, *Madar,* Aloes, etc.

Animal.—Cantharides, snake and insect bites, etc.

C. Mechanical.—Diamond dust, Powdered glass, Hair, etc.

III.—Neurotics.—

1. Affecting the brain (Cerebral).—
   (*a*) Somniferous.—Opium and its alkaloids.
   (*b*) Inebriant.—Alcohol, Ether, Chloroform.
   (*c*) Deliriant.—Datura, Belladonna, Hyoscyamus, Cannabis indica.
2. Affecting the spinal cord (Spinal).—Nux vomica, Gelsemium.
3. Affecting the heart (Cardiac).—Aconite, Digitalis, Oleander, Tobacco, Hydrocyanic Acid.
4. Affecting the lungs (Asphyxiants).—Poisonous irrespirable gases, *e.g.*, Carbon dioxide, Carbon monoxide, Coal gas, etc.
5. Affecting the peripheral nerves (Peripheral).—Conium, Curara, etc.

The Methods of Administering Poisons.—The following are the methods by which poisons may be administered into the system :—

1. By the mouth.
2. By inhalation through the air passages.
3. By absorption through the skin and serous membrane.
4. By hypodermic injection. The dose that acts as poison by this method may be taken as one-third less than that which is required when administered by the mouth.
5. By introduction within the spinal membranes.
6. By injection into a blood vessel.
7. By introduction into an open wound.
8. By introduction into the natural orifices, such as the rectum, vagina, urethra, ears, etc. The dose to act as poison through the rectum is probably double that required by the mouth.

The Channels of Elimination.—The channels of elimination by which poisons are excreted from the body are the urine, bile, milk, saliva, mucous and serous secretions and perspiration.

ACTION OF POISONS

The action of poisons on the animal system is—

1. Local.
2. Remote.
3. Both, *i.e.*, combined.

1. **Local.**—The local action of a poison by coming into direct contact with the part may consist in—

(*a*) Chemical destruction by corrosives, such as, strong acids and alkalies.

(*b*) Congestion and inflammation by irritants, such as weak acids, arsenic, tartar emetic, or cantharides.

(*c*) An effect on the nerves of sensation or motion, *e.g.*, tingling of the skin and tongue by aconite, and dilatation of the pupils by belladonna or datura.

2. **Remote.**—Remote action is produced either by shock acting reflexly through severe pain caused by corrosives, or by poisons being first absorbed into the system through the blood and then exerting a specific action on certain organs and tissues. For instance, cantharides acting on the kidneys produces nephritis, nux vomica acting on the spinal cord causes tetanic convulsions, opium acting on the brain produces narcosis, and potassium chlorate acting on the blood converts oxyhæmoglobin into methæmoglobin.

3. **Both**, i.e., **Combined.**—Certain drugs, such as carbolic acid, oxalic acid, phosphorus, etc., act locally by producing irritation and inflammation of the parts with which they come into contact, and then produce remotely serious symptoms, after they have been absorbed through circulation.

CAUSES MODIFYING THE ACTION OF POISONS

The causes which modify the action of poisons are four in number—

1. The quantity.
2. The form.
3. The mode of administration.
4. The condition of the body.

1. **The Quantity.**—The natural presumption is that a large dose of a poison will produce death more rapidly by causing severe symptoms than a smaller one, but, in some cases, the evil effects are mitigated by vomiting excited by a large dose of a poison, such as copper sulphate. Moreover, the action of a poison varies with the quantity of its dose. For instance, a very large dose of arsenic may produce death by shock without causing irritant symptoms, while a smaller dose than a lethal one may produce its therapeutic action, as happened in the case of the late Mr.

Fulham of Agra, when he was being poisoned by small doses of arsenic. Similarly, oxalic acid, when administered in a large dose, produces a local corrosive action, and may result in instantaneous death from shock, but in a smaller dose it may prove fatal by acting on the heart, while in still smaller doses it acts on the spinal nervous system and the brain.

2. **The Form.**—Under this head will have to be considered—(*a*) Physical state ; (*b*) Chemical combination ; (*c*) Mechanical combination.

(*a*) *Physical State.*—Poisons administered in the form of gases or vapours act at once and most energetically. Poisons in the form of solutions act much more rapidly than powders. Poisons in the form of solids act very slowly, because they are difficult to be absorbed and, in some cases, may prove quite harmless.

(*b*) *Chemical Combination.*—The action of a poison depends upon the solubility or insolubility resulting from a chemical combination. Thus, silver nitrate and hydrochloric acid are both strong poisons when taken separately but, when combined, form an insoluble salt of silver chloride which is almost innocuous. Similarly, baryta (barium dioxide) and sulphuric acid act as poisons if administered separately but, in combination, form an insoluble salt, barium sulphate, which has no poisonous effects on the system. In the same way strong acids and alkalies, when administered together, are rendered inert by their neutralizing effect.

It should be borne in mind that certain poisons which are almost insoluble in water may become dissolved in the acid secretion of the stomach, and are then readily absorbed into the blood. For instance, lead carbonate, white precipitate and copper arsenite, which are insoluble in water, are thus rendered sufficiently soluble for absorption through the mucous membrane of the stomach.

(*c*) *Mechanical Combination.*—The action of a poison may be altered very much if combined mechanically with inert substances. For instance, a small dose of a concentrated mineral acid produces a corrosive action, but the same dose, largely diluted with water, may be taken internally with impunity. A heavy poisonous powder, when mixed with water, will settle down at the bottom of a vessel, and the victim fails to take it ; while it would have been swallowed had it been taken with a fluid of nearly the same specific gravity as that of the powder. For this reason arsenic is usually mixed with milk, tea, coffee or cocoa, when administered for homicidal purposes. Again alkaloids, when taken with animal charcoal, are rendered more or less inert.

3. **The Mode of Administration.**—The rapidity of the action of a poison depends upon the mode in which it is introduced into the system. Thus, a poison acts most rapidly when inhaled in a gaseous or vaporous form or introduced into the blood current by injection into a vein, by subcutaneous injection, or by application to an open wound. Next in rapidity is the action of a poison which is applied to a serous surface, next when introduced into a cellular tissue, and next when applied to a mucous membrane. The least rapid is the action of a poison applied to the unbroken skin. In this case a drug dissolved in oil acts more rapidly than a watery solution.

A poison ingested into the stomach acts more rapidly than when injected into the rectum, since the absorptive power of the stomach and small intestine is greater than that of the large intestine and rectum. Again, if a poison is eliminated as rapidly as it is absorbed, no poisonous symptoms are likely to occur. On the other hand, if the rate of absorption is greater than that of elimination the poison tends to accumulate in the system, and has a cumulative action. For example, mercury, lead, etc., are cumulative poisons.

Absorption by the stomach occurs more rapidly when the stomach is empty than when it is full of food at the time of taking the poison. In some cases, however, absorption may be hastened if the nature of the stomach contents is such as will dissolve the poison. Thus, the action of phosphorus will be hastened if oil is taken immediately it is swallowed, as it dissolves in all oils except turpentine.

Finally it must be remembered that some poisons, when administered by the mouth, are quite harmless, although they are highly dangerous when given subcutaneously. Thus, snake poisons, when swallowed into the stomach, have no poisonous effect on the body. Curare, when taken by the mouth, is practically inert, but it is highly toxic if administered hypodermically. Hydrogen sulphide is more poisonous when inhaled into the lungs than when given in solution either by the mouth or as an enema by the rectum.

4. **The Condition of the Body**.—Under this head will have to be considered—(*a*) Age; (*b*) Idiosyncrasy; (*c*) Habit; (*d*) The state of health; (*e*) Sleep and intoxication.

(*a*) *Age*.—Ordinarily, poisons have a greater effect at the two extremes of age. Certain drugs, such as belladonna and calomel, are, however, better tolerated by children than by adults.

(*b*) *Idiosyncrasy*.—This means natural susceptibility or tolerance of an individual towards certain drugs, such as arsenic, mercury, potassium iodide, tartar emetic, opium, strychnine, etc., as also towards various articles of diet, such as shell-fish, pork, pulses, vegetables, etc., which may be harmful to others. Thus, a medicinal dose of arsenic or mercury may produce alarming symptoms in susceptible persons, while even a very large dose of the same drug may be tolerated by other individuals without any deleterious effects. I have known half a grain of calomel to produce in an adult acute symptoms of mercurial poisoning. The same is the case with certain kinds of food. Hence the proverb "one man's meat is another man's poison."

(*c*) *Habit*.—By a long continued use of such drugs as opium, tobacco, alcohol, strychnine, and arsenic, people establish the habit of tolerating very large doses which, under ordinary circumstances, are liable to prove fatal. Even infants and children who cannot bear very small doses of certain drugs, such as opium, etc., may, by the influence of habit, be made to bear considerably large doses of these drugs with comparative impunity. It should, however, be borne in mind that the habit cannot altogether counteract the evil effects of these poisons and that their habitual use is apt to impair the constitution or give rise to organic disease.

(*d*) *The State of Health.*—Broadly speaking, a healthy and vigorous person is less likely to succumb to the effects of poison than one who is enfeebled by disease. But in some diseases larger doses of certain drugs may be given with impunity without causing any harmful effects, for example, opium in tetanus, delirium tremens and mania, and strychnine in paralysis ; while in other diseases certain drugs cannot be given even in small doses without producing deleterious effects, *e.g.*, opium in granular kidney and apoplexy, and mercury in chronic Bright's disease. Similarly, digitalis, tobacco or tartar emetic even in a small dose may produce symptoms of syncope when given to a person having a weak or a fatty heart.

(*e*) *Sleep and Intoxication.*—During sleep all the bodily functions are languid. Hence the action of a poison is delayed if a person goes to sleep soon after taking it. The action is also retarded if one takes a poison when in an intoxicated condition.

DIAGNOSIS OF POISONING

This has to be made in the living, as well as in the dead.

I. **In the Living.**—The medical man's task becomes very difficult in diagnosing a case of poisoning as, in order to avoid police investigation, nobody is willing to supply him with a true and correct history of the case. However, he can, to a certain extent, diagnose a case of poisoning from the following characters of the symptoms exhibited by the patient : —

1. The onset is usually sudden in a previously healthy individual except in chronic poisoning, where the symptoms develop gradually, and may be easily mistaken for a disease. At the same time it must be remembered that in some diseases, such as cholera, apoplexy, gastro-enteritis, etc., the symptoms may appear suddenly.

2. The symptoms usually commence within about an hour after the poison has been taken in a particular kind of food, drink or medicine ; but the poison will have no connection with the food, drink or medicine if it is not administered by the mouth but by some other channel.

Again, the symptoms of some diseases, such as cholera, apoplexy and rupture of the stomach, may appear all of a sudden soon after taking a meal or drink. In this connection it may be mentioned that a criminal may take the advantage of some epidemic disease occurring at the time, and may administer a poison producing the symptoms almost similar to those of the epidemic, so that the death may be attributed to it. I have seen cases in which arsenic was administered, and the death was attributed to cholera raging in the locality at the time. But the post-mortem examination revealed the signs of irritant poisoning, and the Chemical Examiner detected arsenic in the viscera.

3. The symptoms are uniform in character, and rapidly increase in severity followed either by death or early recovery. Sometimes, remissions may occur as in opium poisoning, and certain poisons may leave sequellæ of long duration.

4. Persons partaking at the same time of the same kind of food or drink containing poison suffer from similar symptoms of poisoning at or about the same time.

5. The detection of poison in food, medicine, vomit, urine or fæces is strong proof of poisoning. Hence, in suspicious cases, these articles must be preserved in clean glass stoppered bottles for chemical analysis.

Table showing instances of similarities of Symptoms produced by Poisons and Diseases

Symptoms.	Poison.	Disease.
1. Colic.	Lead, copper, arsenic.	Volvulus, obstruction.
2. Collapse.	Corrosives, arsenic, antimony, aconite, tobacco, lobelia, antipyrine, exalgin, etc.	Diphtheria, cholera, fever.
3. Coma.	Opium, morphine, chloral hydrate, veronal, trional, sulphonal, paraldehyde, alcohol, camphor, chloroform, carbolic acid, atropine, hyoscine, cyanides, carbon monoxide, carbon dioxide.	Uræmia, diabetes, eclampsia, epilepsy, brain injury, apoplexy, and other brain diseases.
4. Contracted Pupils.	Opium, morphine, chloral hydrate, carbolic acid, pilocarpine, muscarine.	Irritation of 3rd nerve, paralysis of sympathetic, and certain nervous diseases, such as tabes dorsalis.
5. Convulsions.	Nux vomica and its alkaloids, camphor, cyanides, santonin, arsenic, antimony and opium in rare cases.	Tetanus, hysteria, epilepsy, meningitis, eclampsia, uræmia, dentition in children.
6. Cramp.	Arsenic, antimony, lead.	Cholera, diarrhœa.
7. Cyanosis.	Aniline, antifebrin, exalgin, opium, nitrobenzene.	Valvular heart disease and diseases of the respiratory system.
8. Delirium.	Datura, belladonna, hyoscyamus, cannabis, alcohol, camphor, cocaine.	Pneumonia, phthisis, meningitis, nephritis, fevers, epilepsy, insanity and delirium tremens.
9. Diarrhœa.	Irritant poisons, digitalis, colchicum.	Dysentery, cholera, typhoid, tubercle.
10. Dilated Pupils.	Belladonna, hyoscyamus, stramonium, datura and their alkaloids, aconite (altornate dilation and contraction), gelsemium, alcohol, chloroform, conium, cocaine, nicotine.	Paralysis of 3rd nerve, Irritation of sympathetic. Certain nervous diseases causing optic atrophy.
11. Dry Skin.	Belladonna, hyoscyamus, datura and their alkaloids.	Fever, pneumonia.
12. Moist Skin.	Opium, aconite, antimony, tobacco, lobelia, alcohol.	Acute rheumatism.
13. Paralysis.	Conium, aconite, gelsemium, physostigmine, arsenic, lead.	Injury to cord or brain, apoplexy, hysteria.
14. Vomiting.	Corrosive and irritant poisons generally.	Gastric ulcer, acute gastritis, brain tumour, cholera, acidosis, etc.

II. **In the Dead.**—Diagnosis in the dead has to be made from—

A. Post-mortem appearances.

B. Chemical analysis.

C. Experiments on animals.

D. Moral and circumstantial evidence.

A. POST-MORTEM APPEARANCES

In order to make a probable guess of the poison and to look for its characteristic post-mortem appearances, it is advisable that a medical officer, before commencing a post-mortem examination on the body of a suspected case of poisoning, should read the police report and endeavour to get as much information as possible from the relatives of the deceased regarding the quality and quantity of the poison administered, the character of the symptoms with reference to their onset and the time that elapsed between the taking of the poison and the development of its first symptoms, the duration of the illness, nature of the treatment adopted, and the time of death. He will find that in most cases the account supplied by the police and the relatives is very meagre, or incorrect and misleading. His task is, therefore, very difficult, especially when many of the poisons except corrosives and irritants do not show any characteristic post-mortem signs, and when bodies are in an advanced state of decomposition. In cases where positive signs of poisoning are not manifest, the medical officer should not give a definite opinion regarding the cause of death, but should suggest that the viscera be forwarded to the Chemical Examiner for analysis. He must carry out, in all the cases of suspected poisoning, a thorough examination of the body, both external and internal, as far as possible.

External Examination.—Some poisons, such as hydrocyanic acid, carbolic acid or opium, give off a peculiar smell on opening the body. Hence no odorous disinfectant that is likely to mar such smell should be used. The surface of the body and the clothes may show stains or marks of vomit, fæces or of the poison itself. The skin may be jaundiced in phosphorus poisoning, or yellow in acute copper poisoning.

The natural orifices, such as the mouth, nostrils, rectum and vagina, may show the presence of poisonous material, or the signs of its having been ued.

It must be borne in mind that the presence of wounds or disease sufficient to account for death does not contraindicate the use of a poison. It is, therefore, necessary to preserve the viscera in all cases of suspected poisoning, even if there are no positive post-mortem signs of poisoning.

Internal Examination.—The alimentary system should be chiefly examined, as the signs of irritant and corrosive poisons are likely to be found in the œsophagus, stomach and intestines.

The changes produced by irritant and corrosive poisons in the digestive tract, especially the stomach, are—

1. Hyperæmia.
2. Softening.

3. Ulceration of the mucous membrane.
4. Perforation.

These have to be differentiated from similar appearances caused by disease and putrefaction.

1. **Hyperaemia.**—Hyperæmia (redness) of the mucous membrane caused by an irritant poison is generally marked at the cardiac end and greater curvature of the stomach, but rarely at the pyloric end. It is usually of a deep crimson colour, and may be found either in patches or so diffused over its whole surface as to give it a velvety appearance as in arsenical poisoning. The mucous membrane is often covered with a viscid secretion which may be blood-stained.

Instead of redness some other discoloration may be found due to poison or fruit juice. For instance, yellow colour may be due to nitric acid, blue or green colouration to copper, and blackening may be due to sulphuric acid poisoning. Discoloration produced by staining of fruit juice is uniform, and is not marked by signs of inflammation.

It should be noted that the appearance of the mucous membrane of the stomach in the healthy state is pale and white or nearly so, except during the act of digestion, when it becomes reddened. A slight redness is often visible in the stomach, if death has occurred during the process of digestion. Redness is also found in the stomach as a result of general venous congestion in cases where death has occurred from asphyxia. It is, sometimes, so intense that it leads one to suspect poisoning.

On the 3rd August, 1929, a Brahmin male died all of a sudden in a street while returning from a dispensary where he had gone for some medicine. Owing to a good deal of redness of the mucous membranes of the stomach and the upper part of the small intestine and general congestion of the other abdominal organs it was suspected that death might be due to some irritant poison, but the microscopic examination of a lung tissue showed that death was due to lung apoplexy, and the Chemical Examiner did not find any poison in the viscera.

Hyperæmia caused by disease is uniformly spread over the whole surface, and not in patches; besides the ridges of the mucous membrane are more likely to be involved in poisoning than in disease. Redness produced by post-mortem hypostasis is limited to the posterior wall, the most dependent part. In this case there is no thickening of the mucous membrane, nor is there any glairy mucus on its surface.

It is right to bear in mind that the redness caused by poisoning is rapidly altered by putrefaction, but it is difficult to give the exact time when such a change occurs. It generally depends upon the nature of the poison and the degree of decomposition. In a case of arsenical poisoning the redness of the gastric mucous membrane was perceptible nineteen months after interment,[1] and in the other case the hyperæmic condition of the stomach and intestines was evident, when the body was exhumed after twenty-one months' burial.[2]

2. **Softening.**—Softening of the mucous membrane of the stomach, especially at its cardiac end and greater curvature, is usually caused by

1. *Taylor, Poisons, Ed. III, p.* 119.
2. *Ibid., Reg.* v. *Bacon, Lincoln Summer Assizes,* 1857.

the action of corrosive poisons, chiefly the alkalies. It is also observed in the mouth, throat and œsophagus. But when caused by disease it is confined to the stomach alone and commonly found at its cardiac end.

Some corrosive poisons, such as carbolic acid, produce hardening and shrinking of the mucous membrane instead of softening.

Softening caused by putrefaction commences at the most dependent parts, and affects all the coats of the stomach without the detachment of its mucosa, and the softened patch is not surrounded by an inflamed area as is the case in corrosive poisoning.

3. **Ulceration.**—Ulceration caused by corrosive or irritant poisons is generally found at the greater curvature of the stomach, and presents the appearance of an erosion with thin, friable margins and surrounded by the softened mucosa due to intense inflammation. An idiopathic gastric ulcer is situated frequently on the lesser curvature with sharply defined, but thickened and indurated edges. The mucous membrane is commonly reddened only in the neighbourhood of the ulcer; while the redness is generally diffused over other parts of the stomach and extends up to the duodenum and small intestine, when the ulcer is due to a corrosive or irritant poison.

4. **Perforation.**—Perforation of the wall of the stomach or small intestine resulting from corrosive poisoning is rare, though it may be met with in cases of sulphuric acid poisoning. Perforation caused by poisoning must be distinguished from one caused by disease or by the post-mortem action of the gastric juice.

In a perforation caused by poisoning the aperture is large, the edges are ragged and irregular and the coats are easily lacerated. The tissues round the margins are disintegrated beyond the edges of the aperture. The stomach in such a case is charred owing to the severe corrosive action.

If a perforation has been the result of a chronic ulcer due to disease, the aperture is commonly oval or rounded, the margins are more or less punched out, and the stomach does not show the signs of charring, but it shows chronic adhesions to the neighbouring organs. Very rarely perforation may follow an ulcer caused by irritant poisoning, when its appearance will be similar to that produced by the idiopathic ulcer.

In a perforation produced after death by auto-digestion of the stomach by the gastric juice the aperture is very large and irregular with rough and pulpy edges; there is no inflammation or charring of the stomach, but the surrounding mucous membrane is often softened and gelatinous.

B. CHEMICAL ANALYSIS

The most important proof of poisoning is the detection of poison in the excreta, (vomit, urine, etc.,) during life, and in the contents of the stomach and bowels, and in the tissues of the body after death. The finding of poison in food, medicine or any other suspected substance is a corroborative, but not a conclusive proof; for the poison may have been added to any of these substances just to substantiate a false charge against

an enemy. In cases of *feigned* poisoning it is advisable to elicit from the patient the poison he suspects to have been administered to him, so as to note if the symptoms complained of are referable to the same poison. The medical man should also preserve for chemical analysis only the portions of the vomit and urine ejected in his presence.

When poison has been detected in the stomach contents, the defence pleader may argue that it may have been introduced after death, or the contents may have been preserved in an unclean vessel. But these arguments are quite futile and worthless if the poison has also been detected in one or more of the solid viscera, such as the liver, spleen, kidneys, etc., and if clean china plates and glass bottles, free from contamination, have been used for examining and preserving the stomach and other viscera.

It is not necessary to lay any stress on the amount of poison actually recovered except in those cases where it is alleged that the poison may have been administered as a medicine, that it may have been present owing to the deceased being habituated to its use, that it may have been a natural constituent of the body or a normal constituent of some article of food, or that it may have been produced in the body during the process of decomposition, *e.g.*, leucomaines and ptomaines.

It is quite possible that a person may die from the effects of a poison, and yet none may be found in the body after death, if the whole of the poison has disappeared from the lungs by evaporation, or has been removed from the stomach and intestines by vomiting and purging, and after absorption has been eliminated from the system by the kidneys and other channels. Certain vegetable poisons may not be detected in the viscera, as they have no reliable tests, while some organic poisons, especially the alkaloids and glucosides, may, by oxidation during life or by putrefaction after death, be split up into other substances which have no characteristic reactions sufficient for their identification.

I have seen cases in which there were definite signs of death from poisoning, although the Chemical Examiner failed to detect the poison in the viscera preserved for chemical analysis. In his annual report for the year 1927, the Chemical Examiner of Bengal also mentions that of the cases in which medical officers gave definite opinions that death was due to poisoning, poison was detected only in 60.37 per cent cases. It has, therefore, been wisely held by Christison that, in cases where a poison has not been detected on chemical analysis, the judge, in deciding a charge of poisoning, should weigh in evidence the symptoms, post-mortem appearances and moral evidence.

Examination of the Viscera and their Contents.—A medical officer who has no experience of chemical analysis should never undertake the analysis, nor should he ever make any guess from the nature of the stomach contents, etc.; but, after obtaining necessary orders from the District Magistrate he should forward the viscera to the Chemical Examiner for analysis. The Magistrate conducting the proceedings should furnish the Chemical Examiner with a copy of the medical officer's post-mortem report and with every fact and detail either from deponents or from the

police investigation, which may indicate the direction in which analytical inquiry may yield a positive result.[1]

The Chemical Examiner has got the most responsible work, as his findings are final, because he is not, as a rule, liable to cross-examination (Vide Sec. 510, Cr. P. C., Appendix VI).

The Chemical Examiner or his assistant who receives the articles for analysis from medical officers first verifies the seals, and compares the labels with the invoice list of materials sent, and then opens the bottles, etc. He then places the contents in separate shallow porcelain basins after weighing and measuring them according to the nature of the material.

A careful inspection of the contents of the stomach and its mucous membrane is now made both with the naked eye and with a hand magnifying lens, making a note of the colour and reaction of the contents. Any foreign substances, such as particles of undissolved poisons and fragments of seeds, leaves, roots, etc., of poisonous plants, are next picked up and examined on a slide under the microscope. The inner wall of the stomach is then washed with distilled water, and the washings added to the contents. A little of the stomach contents may be taken on a slide, rubbed up with a drop or two of glycerine, and when examined under the microscope, may show fragments of *datura* seeds or *bhang* leaves.

For chemical analysis the contents of the stomach are diluted with water, and the solid viscera are finely chopped up and macerated in water. If the Chemical Examiner has any clue or indication of the nature of the poison, he begins by searching for it. If not, he usually divides the mixtures into three parts for the examination of volatile, vegetable and mineral poisons.

1. **Volatile Poisons.**—Volatile poisons, such as alcohol, ether, hydrocyanic acid, benzene, nitro-benzene, aniline, carbolic acid, bromine, iodine and phosphorus, are separated by distilling the first portion of the mixture acidulated with tartaric acid, but to separate ammonia, nicotine and volatile bases the mixture has to be rendered alkaline by the addition of magnesia. The distillate is then examined for the presence of these poisons by applying distinctive tests for each.

2. **Vegetable Poisons.**—The detection of vegetable poisons depends on the isolation of their alkaloids and glucosides from the stomach contents or organs of the body and the suspected articles of food, and their identification by the application of chemical and physiological tests.

These alkaloids may be grouped under three heads : (1) those derived from pyridine, *e.g.*, atropine, coniine, (2) those derived from quinoline, *e.g.*, cinchonine, narcotine, and (3) substituted amines and amides. Most of the vegetable alkaloids belong to the first two groups. They are mostly solid, crystalline and colourless, except a few, such as coniine, nicotine and pilocarpine, which are liquid. They are insoluble in water, but soluble in ether, while with acids they form salts, which dissolve in water, but not in ether. This fact of solubility is made use of in separating them from organic mixtures for which the following processes are adopted :—

1. *G. O. No. 3072/VI—1092, dated 25th July, 1917 ; I. G. C. H. U.P.'s Circular No. 57 dated 6th August, 1917 ; U. P. Med. Manual, 1934, p. 220.*

(*a*) **Stas's Process.**—The second part of the original mixture is acidulated with tartaric or citric acid, and digested with alcohol in a glass flask, allowed to stand on a water bath for half an hour and then to stand for twenty-four hours. The alcoholic extract is then strained off and evaporated at a low temperature to a syrupy consistence. The extract may now be examined for the presence of opium, but, in the absence of opium, the extract is treated with cold alcohol, and again evaporated, treated with water acidulated with acetic acid and filtered. The filtrate is neutralised with sodium carbonate, shaken up with ether, chloroform or amyl alcohol, and separated in a separating funnel; the extract is then evaporated to dryness in glass dishes. The dry residue is now ready for the detection of alkaloids.

(*b*) **Otto's Process.**—This is a modification of Stas's process, and has the reputation of being simpler, and at the same time equally accurate. In this method the alkaloid is converted into a salt, such as a sulphate, by the addition of an acid. The salt is then formed into a solution, dissolving it in water. The solution is shaken up several times with ether which removes all foreign fatty matters. The solution, which is now comparatively pure, is rendered alkaline by adding caustic soda. Ether is now added to the alkaline solution so as to dissolve the alkaloid, which may be separated from it by evaporation.

(*c*) **Dragendorff's Process.**—Dragendorff has elaborated a modified form of Stas-Otto process for the separation of alkaloids, glucosides and vegetable principles from each other.

The mixture is digested with water slightly acidulated with sulphuric acid at a temperature of 40° to 50° C. for some hours, and is then filtered, the filtrate being collected. The process of extraction and filtration is repeated two or three times, and the filtrates thus collected are combined, evaporated to a syrupy consistence, and are heated for about twenty-four hours with alcohol. The alcoholic extract thus formed is filtered and evaporated to dryness. The residue is dissolved in a half per cent solution of acetic acid and filtered. This acid filtrate is shaken with the following solvents to dissolve out the undermentioned substances:—

1. Petroleum ether to dissolve out carbolic acid, picric acid, camphor, capsicin and piperine.

2. Benzene to dissolve out cantharidin, colchicine, colocynthin, digitalin, absinthin, elaterin, santonin and thiene.

3. Chloroform to separate papaverine, picrotoxin, digitalein, cinchonine, helleborin, saponin and jervine.

The acid solution is then rendered alkaline, by the addition of ammonia, or a solution of caustic potash or soda, and the following solvents are added with shaking to separate the undermentioned substances:—

1. Petroleum ether to dissolve out volatile alkaloids and aniline, as also strychnine, brucine, coniine, nicotine, etc.

2. Benzene to dissolve out strychnine, brucine, aconitine, quinine, atropine, veratrine, codeine, narcotine, thebaine, physostigmine, etc.

3. Chloroform to dissolve out narceine, morphine, papaverine, etc.

4. Amyl alcohol to dissolve out morphine, solanine, salicin, and saponin.

5. Chloroform after the residue is evaporated to dryness, when curarine will separate out.

(*d*) **Hankin's Process.**—Dragendorff's process is far too elaborate to suit the requirements of an Indian Chemical Examiner. Hence Dr. E. H. Hankin, M.A., Sc. D., Chemical Examiner of Agra, has modified it in the following manner :—

a. The viscera are chopped into small pieces with a pair of scissors, and placed in a large beaker or flask with the rectified spirit in which they have been preserved.

b. After adding a few drops of acetic acid the beaker is boiled by standing it in a water-bath. In cases of suspected aconite poisoning it is advisable not to heat the viscera above 65° C.

c. After this digestion the material in the beaker is filtered, and the solid portion left in the filter should be again extracted with alcohol. The alcoholic solution should be filtered off and added to the first portion of the filtrate.

d. These mixed filtrates contain most of all the alkaloids present, though with impurities. They are to be evaporated almost dry over a water-bath in a current of air.

e. When the filtrate has thus been converted to syrupy consistency, 20 c. c. of water should be added slowly while stirring it. It should then be filtered by using a funnel and an ordinary thin filter paper. It may be necessary to centrifuge it in case the liquid does not filter easily.

f. This watery extract is placed in a separating funnel and tested with a litmus paper. The reaction should be acid. If not, acetic acid should be added. 50 c. c. of ether are then added and shaken violently for one minute.

g. The separating funnel is fixed in a stand and allowed to rest for at least an hour, when the ether separates out and floats in the form of a layer on the surface of the watery liquid. If the two liquids have not separated by this time the addition of a further quantity of ether and shaking will cause separation. In rare cases it may be necessary to submit the mixed liquids to a strong and powerful centrifuging machine. In about five minutes a dense layer of glutinous matter forms, separating the two layers of ether and the watery liquid.

h. After the liquids are separated, the separating funnel should be held in the mouth of a second separating funnel to allow the watery liquid to flow into the latter. The ethereal layer (the acid ether extract) contains impurities, and may contain some rare poisons which are not ordinarily required to be looked for in India. Hence it may be discarded. If the ethereal layer is strongly coloured, the watery liquid has to

be treated with chloroform. In testing for certain poisons, such as strychnia, it is desirable to submit the liquid to further alternate and repeated washings with ether and chloroform. Chloroform should be shaken with the acid liquid with the help of a shaking machine. It is easy for chloroform to separate out if the glutinous layer does not exist; otherwise it may be necessary to use a centrifuge.

i. The acid watery liquid in the lower funnel contains the commoner alkaloids. A few c. c. of chloroform, a piece of litmus paper and 50 c. c. of ether should be added to the liquid, to which a sufficient quantity of ammonia should be added to make the whole mixture alkaline. It should then be shaken up immediately for at least half a minute; because the alkaloids present are capable of passing readily into the chloroform ether mixture as soon as they are liberated by the addition of ammonia. They become changed after the lapse of a few minutes, and then pass with difficulty into the ether solution.

j. The separating funnel should be placed in a stand, and should not be disturbed till the liquids have separated. The separation may be hastened by the addition of a few c.c. of alcohol.

k. The liquid now separates into two layers. The upper ethereal layer, called the *alkaline ether extract* contains most of the alkaloids; and the lower watery layer contains impurities. In cases of opium poisoning this portion of the watery extract contains the substance giving the *meconic reaction.*

l. The watery liquid should be tapped off and discarded.

m. The ethereal liquid should be received into a porcelain basin.

n. A few drops of a half per cent solution of acetic acid in water should be added to the ethereal solution in the basin.

o. This should be evaporated on a water-bath, until two or three drops of the dilute acid are left. In the case of aconite poisoning it should be evaporated at a temperature below the boiling point. The evaporated dilute acid should now be tested for different alkaloids.

General Tests for Alkaloids.—1. *Wagner's Reagent.*—Iodine dissolved in a solution of iodide of potassium gives a reddish-brown precipitate, if added to most alkaloids.

2. *Mayer's Reagent.*—Biniodide of mercury gives a yellowish-white crystalline precipitate with an alkaloid solution. Biniodide of mercury is prepared by adding a solution of iodide of potassium to one of mercuric chloride, when a scarlet precipitate is formed, which is just dissolved by a further addition of either of the two.

3. *Sonnenschein's Reagent.*—Phosphomolybdic acid gives a yellow amorphous precipitate with most alkaloids.

4. *Scheibler's Reagent.*—Phosphotungstic acid has the same reaction as No. 3.

5. *Platinic Chloride.*—A solution of platinic chloride gives a brown precipitate with alkaloids.

6. *Tannin, Picric Acid, or Mercuric Chloride.*—Each of these, when added to alkaloids, precipitates them.

Mineral Poisons.—Two methods, *viz.*, wet and dry, are employed for extracting mineral poisons from organic mixtures.

Wet Method.—To oxidise the organic matter add strong hydrochloric acid to the third portion of the original mixture, and heat it carefully with potassium chlorate added in small portions at a time. The resulting solution will be a chloride of a metal, which can be obtained clearer by filtering it. The solution may then be tested for the presence of metals by the grouping reagents and confirmatory tests.

Dry Method.—The organic matter in the mixture is destroyed by heat so as to incinerate it completely. To the ashes thus obtained add strong nitric acid. The excess of the free acid should be removed by heat, and the nitrate should be dissolved in water and tested in the usual way. If the mixture is strongly acid in reaction, caustic potash may be added to neutralise it.

C. EXPERIMENTS ON ANIMALS

Domestic animals may be fed with the suspected food, or with the poison after it is separated from the viscera and the symptoms exhibited by them should be noted. However, the evidence derived in this manner cannot be relied on in all cases, as some symptoms, such as vomiting, etc., may be produced without any poison, and some animals may not be affected even by poisons. For example, rabbits are insusceptible to the leaves of belladonna, hyoscyamus and stramonium; so are pigeons to opium. But the cat and the dog are affected by poisons almost in the same way as man.

D. MORAL AND CIRCUMSTANTIAL EVIDENCE

In a case of criminal poisoning the fact whether the accused was the person who administered the poison can be proved only from moral and circumstantial evidence. This is furnished by common witnesses, who testify to the recent purchase of the poison by the accused, etc. The medical witness should not hazard an opinion on moral and circumstantial proof. He should certify to the cause of death from medical facts only. He should not, however, omit to note the surroundings of the patient, and the nervousness and anxieties of the relatives or some other persons regarding the haste with which they want the body to be disposed of by burial or cremation.

THE DUTY OF A MEDICAL PRACTITIONER IN A CASE OF SUSPECTED POISONING

A medical practitioner must be very cautious in giving his opinion about poisoning. On mere suspicion he should never give a verbal or

written opinion lest he be the victim of an action for damages brought against him. In a suspicious case of acute poisoning the medical practitioner must try to find out the nature of the suspected poison so that he can at once administer the appropriate treatment and save the patient's life. In a case where he suspects slow poisoning by the administration of small doses at varying intervals he should make a very careful note of all the symptoms exhibited by the patient. He should also collect the vomited matter and twenty-four hours' urine, if possible, and get them analysed for the presence of poison. It is always advisable to call in one or two brother-practitioners in consultation, and to have the patient removed to a hospital, where the doctor in charge should be informed of the suspicion, so that he would not allow any one except the hospital nurses to administer medicine and nourishment. If the patient cannot be removed to the hospital and if he can afford the expenses, the employment of two trained and trustworthy nurses to take charge of the patient in his house and also of the preparation and administration of his food and medicine for day and night will be a safeguard against further administration of poison. If that arrangement is not possible, the only alternative left for the medical practitioner is to take some near relative or friend in his confidence and inform him of his suspicion. The patient may also be warned against the danger, if he happens to be an adult and in full possession of his senses.

In every case of suspected poisoning the medical practitioner, whether in private practice or in Government service, must preserve the vomited matter or stomach wash and samples of urine and fæces passed in his presence and likely to contain poison and suspected articles of food, drink or medicine in separate wide-mouthed glass bottles or jars with tightly fitting glass stoppers. These bottles or jars should be properly labelled with the name of the patient, the material preserved and the date of the examination, and should be kept under lock and key in his own custody till required for transmission to the Chemical Examiner for chemical analysis. The medical practitioner must also preserve any other evidence of the suspected poisoning. If he fails in his duty in this connection, he may render himself liable to be charged with causing disappearance of evidence under section 201, I. P. C.[1] It must, however, be proved that the medical practitioner did it with the intention of screening the accused; otherwise it is merely an error of judgment for which he cannot be held responsible.

If the medical practitioner in private practice is convinced that the patient upon whom he is attending is suffering from homicidal poisoning, he is bound, under section 44, Criminal Procedure Code,[2] to communicate the fact to the nearest police officer or magistrate. Non-compliance is punishable under section 176, Indian Penal Code.[3] He is not liable for giving notice, if the case has already been reported to the police by the village headman, village watchman or any other officer required under law to give such information under section 45, Criminal Procedure Code.[2]

1. Vide *Appendix VII.*
2. *Vide Appendix VI.*
3. Vide *Appendix VII.*

The medical practitioner is not bound to supply information of his own accord to the police or magistrate, if he is sure that his patient is suffering from suicidal poisoning, since section 309 of the Indian Penal Code which refers to the offence of an attempt to commit suicide is not included in the sections of the Indian Penal Code for which information has to be given under section 44, Criminal Procedure Code. The medical practitioner is, however, bound to divulge all the information regarding the case that has come to his notice, if he is summoned by the investigating police officer to give such information under section 175, Criminal Procedure Code.[1] If he conceals any information, he is liable to be prosecuted under section 202, Indian Penal Code.[2] If he gives false information, he is liable to be charged with the offence of giving false information under section 193, Indian Penal Code.[2] To avoid these difficulties the Inspector-General of Police, Bengal, suggests that every case of suspected poisoning should be treated as homicidal and the question of suicide must be decided by the police after investigation.[3] The medical officer in charge of a government (public) hospital is required to report to the police all cases of suspected poisoning, whether accidental, suicidal or homicidal, admitted into his hospital.

If a case of suspected poisoning proves fatal, the medical practitioner should never grant a death certificate, but must communicate the fact of the death to the nearest police officer for necessary investigation.

TREATMENT IN CASES OF POISONING

A medical practitioner should always have an emergency case ready for cases of poisoning, so that he may be able to adopt immediate treatment without loss of time.

The treatment should be based on the following principles :—

1. Removal of unabsorbed poison from the body.
2. Use of antidotes.
3. Elimination of poison absorbed into the system.
4. Treatment of general symptoms.

1. **Removal of Unabsorbed Poison.**—If a poison has been introduced into a wound subcutaneously as a result of bites or stings, a ligature should immediately be applied above the wound, which should then be excised or sucked provided there is no abrasion or ulcer in the mouth. If a poison has been inhaled into the respiratory passages, the patient must be made to inhale pure air or oxygen. Oxygen should be used with a mask similar to one which is used for gas administration. It is better to use a mixture of 95 to 93 per cent of oxygen and 5 to 7 per cent of carbon dioxide, for the carbon dioxide in this excess stimulates the respiratory centre to act more vigorously.

If a poison has been taken into the stomach, it should be removed by washing out the stomach with a stomach tube (syphon tube) or stomach

1. Vide *Appendix VI.*
2. Vide *Appendix VII.*
3. *Directions for forwarding cases to the Chemical Examiner, Bengal for medico-legal examination,* 1937 *p.* 12.

pump. An ordinary rubber tube of about half an inch in diameter and about five feet in length with a glass funnel attached at one end will serve the purpose of a stomach tube. A mark should be made at a distance of twenty inches from the blunt end (stomach end). The tube should be warmed and covered with olive or sweet oil, or some other lubricant, and should be passed into the stomach by depressing the tongue with the finger well back into the pharynx, and thus passing it downwards. When the mark on the tube is reached it has entered the stomach. The funnel should now be held high above the patient's head and about a pint or two of warm water should be poured down it into the stomach. When the funnel is almost empty, the tube below it should be compressed between the finger and thumb and lowered below the level of the stomach. On removing the pressure the contents of the stomach which will run out of the funnel owing to the tube acting as a syphon should be received in a perfectly clean bowl or bucket. This process should be repeated until a clear, odourless fluid comes out. A part or all of the material ejected by the first washing of the stomach must always be preserved for chemical analysis. Instead of warm water it is preferable to use a solution of a suitable antidote, such as a solution of potassium permanganate in poisoning by opium, datura and other organic substances. If the stomach contains large pieces of food or poison, it may be necessary to produce vomiting by an emetic before using the tube, in order to prevent the blocking of the narrow opening at its blunt end. In such an event it is also advisable to enlarge the opening with a pair of scissors.

It is advisable to use a mouth gag in those cases where it is difficult to keep the mouth open for the passage of the tube.

A stomach wash out on a comatose or unconscious patient, whose cough reflex is absent, may be fraught with danger, as the stomach contents which are regurgitated around the tube may flow into the trachea and cause either immediate suffocation or later broncho-pneumonia. As a safeguard against such an accident it is necessary that the mouth and pharynx should be lower than the larynx. This is usually achieved either by putting pillows under the shoulders and bending the head right back, or by hanging the head and shoulders over the end of the bed. Both these methods are not satisfactory, inasmuch as the former method does not get the mouth quite low enough if there should be a copious regurgitation of fluid, and the latter method is arduous with a heavy patient. Marriott,[1] therefore, suggests that the patient be taken to the operating theatre and placed in the Trendelenburg position. In this position the gravitation of fluid from the mouth into the trachea is impossible.

The stomach tube should never be used in cases of poisoning by corrosives except carbolic acid, as there is danger of causing perforation of the œsophagus or stomach owing to the softening and ulceration produced by them. In cases of irritant poisoning the stomach tube should be passed with caution.

When the stomach tube or pump is not available, or when a patient is conscious, and does not wish to have it passed into the stomach, free emesis should be produced by tickling the fauces with the fingers, a

1. *Lancet, May* 6, 1933, *p.* 962.

feather, or a leafy twig of a tree. The vomited matter must be preserved for chemical analysis. The following emetics may also be administered :—

1. Copious draughts of warm water.

2. A table-spoonful of ground mustard or two table-spoonfuls of common salt in half a pint of warm water.

3. Half a drachm of sulphate of zinc in a tumblerful of warm water, to be repeated in a quarter of an hour, if necessary.

4. Twenty to thirty grains of ipecacuanha powder, or two to six drachms of ipecacuanha wine. In the case of a child syrup of ipecacuanha, from half a tea-spoonful to two tea-spoonfuls, according to the age, is to be preferred, as it is easy of administration.

5. Fifteen to thirty grains of ammonium carbonate dissolved in water.

6. Five to ten grains of copper sulphate dissolved in water, but it should not be used except in cases of poisoning by phosphorus.

7. 1/10 grain of apomorphine hydrochloride hypodermically. This acts promptly and produces vomiting within three or four minutes, but it causes great prostration and its effects are occasionally greatly prolonged. Hence it must be used with caution.

2. **Use of Antidotes.**—Antidotes are remedies which counteract the effects of poisons. They are divided into *mechanical, chemical* and *physiological.*

Mechanical antidotes are those which render poisons inert by mechanical action. For instance, finely powdered charcoal acts mechanically by absorbing and retaining within its pores organic and also, to a less degree, mineral poisons. Fats, oils and egg albumen prevent the action of the poison by forming a coating on the mucous membrane of the stomach. Bulky food acts as a mechanical antidote to glass, as it prevents its action by imprisoning its particles within its meshes.

Chemical antidotes are those which counteract the action of the poison by forming harmless or insoluble compounds when brought into contact with it. The examples are acids for alkalies, alkaline carbonates and magnesia for mineral acids, lime for oxalic acid, sodium sulphate for lead and tannin and albumen for alkaloids. It must be remembered that only those substances should be selected as chemical antidotes which are by themselves almost harmless, so that if an excess is given they will not produce any ill-effects. Thus, vinegar or lemon-juice should be used as an antidote to a caustic alkali, but nor a mineral acid, such as hydrochloric or sulphuric acid which, if given in excess, might prove as harmful as the original poison.

From his experiments on animals Jona has proved that the administration of adrenalin delays the absorption of rapidly acting poisons, such as cyanides, strychnine and aconite, by its constricting action on the vessels of the gastric mucosa.[1]

1. *Brit. Med. Jour., Feb.* 3, 1913, *p.* 271.

A very important chemical antidote for organic poisons is potassium permanganate owing to its oxidising properties. A solution of potassium permanganate in the proportion of 10 to 15 grains in the pint is commonly used in opium poisoning, but should be used in all cases of organic poisons. The patient should drink as much as he can of it both before and after vomiting or it should be introduced by means of the stomach tube when the patient is unconscious. If this remedy is used, the magistrate should be informed of the fact as its use greatly decreases the chance of detection by the Chemical Examiner. This, however, should not deter the medical man from using the drug, as his duty is to save life. If in doing so he destroys evidence that might be useful to the police, that is sad for the police, but is no concern of the doctor. If without harming the patient he can obtain material of evidential value, then by all means he should do so but not otherwise. Hence, before trying the permanganate, he may wash out the stomach with water and preserve this washing for the Chemical Examiner if it is possible to do so without causing the patient to undergo any extra risk.

The following formula is a useful chemical antidote which is recommended in cases where the nature of the poison swallowed is not definitely known, or in cases where it is suspected that a combination of two or more poisonous substances had been taken :—

Powdered charcoal	2 parts
Tannic acid	1 part
Magnesia (Magnesium Oxide)	1 part

These drugs are mixed together, and the mixture is administered in the doses of a tea-spoonful stirred up in a tumblerful of water, to be repeated frequently. Charcoal has the property of absorbing alkaloids. Tannic acid precipitates alkaloids, glucosides and many of the metals. Magnesia neutralises acids, and is used as an antidote to arsenic, if hydrated ferric oxide is not at hand.

Physiological antidotes or antagonists are those which act on the tissues of the body and produce symptoms exactly opposite to those caused by the poison acting on the same tissues. Thus, a perfect physiological antidote is one which exactly counteracts each evil effect produced by the poison but most of the known antidotes are only partial in their action, and when pushed to their physiological action are liable to prove dangerous to life. Atropine is an example which, though it is regarded and used as a physiological antidote of morphine, is liable to cause death by paralysing the motor and sensory nerves just like morphine. Hence caution must be observed while using it. Atropine and physostigmine are two real physiological antagonists, as both of them affect nerve-endings and produce opposite effects. Atropine paralyses the vagus nerve-endings, accelerating the heart's action, while physostigmine stimulates these nerve-endings, producing slowing of the heart. Atropine dilates the pupil by paralysing the third nerve-endings, while physostigmine contracts the pupil by directly stimulating the terminals of the third nerve. Atropine diminishes glandular secretion by paralysing the secretory nerve-endings in the body, while physostigmine increases glandular secretion by stimulating the secretory nerve terminals. Atropine stimulates the central nervous system, while physostigmine depresses it.

Atropine and pilocarpine, strychnine and bromides with chloral hydrate, digitalis and aconite, and chloroform and amyl nitrite are the other examples of physiological antidotes.

3. **Elimination of Absorbed Poison.**—The poison which has been absorbed into the system should be eliminated by the natural emunctory channels by giving hot baths, warm packs, diuretics and purgatives (when not contra-indicated).

4. **Treatment of General Symptoms.**—Pain in the stomach due to the irritation of the gastric mucous membrane should be relieved by the administration of anodynes and demulcents. If it is very severe, one-fourth grain of morphine should be administered hypodermically.

Shock and collapse should be combated by warmth to the surface in the form of friction, a hot bath, or hot bottles, or by the hypodermic injection of stimulants in the form of sulphuric ether, strychnine, digitalis, or caffeine.

Water containing 40 grains of sodium chloride to the pint and sweetened with glucose should be given frequently by the mouth, if there is dehydration owing to intense vomiting or diarrhœa. If the water cannot be retained by the mouth, an enema of two pints of warm normal saline should be administered. In severe cases it is advisable to administer intravenously normal saline particularly by the drip method. It also dilutes the poison in the blood, and promotes its elimination from the system, especially by the kidneys.

Respiratory failure should be treated by the hypodermic injection of atropine or strychnine, by artificial respiration and by the inhalation of oxygen or oxygen and carbon dioxide.

Coma should be treated by the hypodermic injection of 1/8 grain of strychnine, by the intravenous or intramuscular injection of 5 to 15 c. c. of a 25 per cent solution of coramine and by lumbar puncture. In a case where coma is prolonged, an intravenous injection of normal saline may be administered continuously by the drip method.[1]

Convulsions should be controlled by chloroform or by a drug belonging to the barbituric acid group.

Appropriate treatment will be necessary for remote effects of poisons, such as ulceration or contracting cicatrices after corrosive poisoning and neuritis after chronic arsenic poisoning.

1. *Marriott, The Treatment of Acute Poisoning,* 1935, *p.* 11.

CHAPTER XXIII

CORROSIVE POISONS

I. MINERAL ACIDS

Mineral acids have a local chemical action of corroding and destroying the tissues they come into contact with, and may produce fatal consequences, if extensive. They have no remote effects on the system.

They act as irritants when slightly diluted, but as stimulants when well diluted and given in the pharmacopœial doses.

Cases of poisoning by corrosive, mineral acids are rare in India, but are more frequent in Europe. They are very rarely used for homicidal purposes though, sometimes, cases are met with in which corrosives are thrown on the face out of jealousy or in fits of rage. Accidental cases of swallowing acids in mistake for some harmless medicine do occur, especially amongst children. Occasionally a cooly while carrying a jar containing some concentrated mineral acid may accidentally fall down, and break the jar so as to spill the acid which may affect him, as also the passers-by. A few suicidal cases also occur.

General Symptoms.—The symptoms supervene in the act of swallowing the concentrated acid or immediately after taking it. There is intense burning pain in the mouth, throat, and œsophagus, extending down to the stomach. The pain is attended with frothy eructations, retching and vomiting of a brownish or blackish matter containing blood, mucus and shreds of mucous membrane. The ejected matter has an intensely acid reaction, stains the clothes on which it falls, and effervesces when it comes into contact with the alkaline ground. Sometimes, when the quantity ingested is very large the whole surface of the stomach becomes corroded. In such a case no vomiting occurs, as the stomach is unable to expel its contents.

Thirst is intense, but it cannot be appeased owing to great pain and difficulty in swallowing, and each attempt to drink is followed by renewed retching and vomiting.

The lips and angles of the mouth are shrivelled and excoriated with a continuous flow of saliva containing mucus, blood, and detached pieces of the corroded mucous membrane, unless the acid has been poured down into the back of the throat by means of a spoon or tube. Sometimes, the mucous membrane becomes loose, and falls out of the mouth.

The voice becomes hoarse and husky from the inflammation of the epiglottis and larynx, and articulation becomes painful and difficult. There is also difficulty in breathing.

The bowels are constipated, though usually there is tenesmus. Rarely, there may be loose motions containing altered blood, and shreds of mucous

membrane. The urine is scanty or suppressed. There may be difficulty and pain in micturition.

The pupils are frequently dilated, the eyes looking wild and sunk.

There is a general condition of collapse. The skin is cold and clammy, and the pulse is slow and feeble, but the mind remains clear till death.

Death occurs within a few hours from shock or from spasm or œdema of the glottis, and within twenty-four hours from collapse due to perforation of the stomach and peritonitis.

If death does not occur within twenty-four hours, reaction may set in, when the pulse becomes full, with a rise of temperature. The process of separation of the sloughs and reparation will follow. Usually death occurs towards the end of the first week from septic absorption, or it may occur after months or years from exhaustion and malnutrition owing to starvation resulting from cicatrisation and stenosis of the œsophagus or pylorus, and incurable dyspepsia due to destruction of the coats of the stomach.

Treatment.—The stomach tube or emetics must never be used. The poison should be immediately diluted and neutralized *in situ* by administering a pint of water or milk to which 4 table-spoonfuls of calcium or magnesium oxide or calcined magnesia are added. But as these are not likely to be at hand, oil, soap solution, lime water, or powdered white wall plaster suspended in water, should be administered without delay, and should be followed by demulcent drinks, such as barley water, linseed, tea, etc.

The use of alkaline carbonates or bicarbonates should be avoided as far as possible, as they evolve carbon dioxide gas, which will increase distress, and may even cause perforation by suddenly distending the stomach.

Intense thirst should be relieved by giving pieces of ice to suck, and pain should be relieved by hypodermic injections of morphine. Nutrient enemata should be given to keep up the strength of the patient. Excoriations on the surface must be treated as burns. Tracheotomy must be resorted to if suffocation is threatened from an affection of the larynx.

Post-mortem Appearances.—The conditions found after death depend on the quantity and strength of the poison used, and the time that the patient survives after taking the poison. If death has occurred in a short time there will be signs of corrosion and destruction of the mouth, throat, œsophagus and stomach, varying from a few localised patches to extensive destruction. There may be perforation of the stomach with the escape of its contents into the peritoneal cavity, and consequent destruction of the peritoneum and abdominal organs. The tissues beyond the corroded area show the signs of inflammation.

If the patient lived for some days, the signs of repair due to separation of the sloughs will be evident, and the cicatrised tissue will be noticeable if death did not occur for a very long time.

The marks of corrosion may also be noticed on the skin and clothes.

SULPHURIC ACID (OIL OF VITRIOL), H_2SO_4.

Properties.—Pure sulphuric acid is a colourless, heavy, oily liquid, which emits no fumes when exposed to the air. When mixed with water it evolves much heat, and is reduced in volume. It chars and blackens the skin, cloth and any other organic matter. The portion of the cloth or paper which comes into contact with the acid is destroyed, leaving a reddish-brown stain which is usually moist. Similarly, the stain on wood is damp black owing to its charring effect.

The acid of the British Pharmacopœia has a specific gravity of 1.84 and contains 95 per cent of sulphuric acid by weight in water. *Acidum sulphuricum dilutum* is a pharmacopœial preparation, the dose being 5 to 60 minims. It contains 10 per cent of the acid. *Acidum sulphuricum aromaticum*, which is known as elixir of vitriol, is a non-official preparation. The dose is 5 to 20 minims.

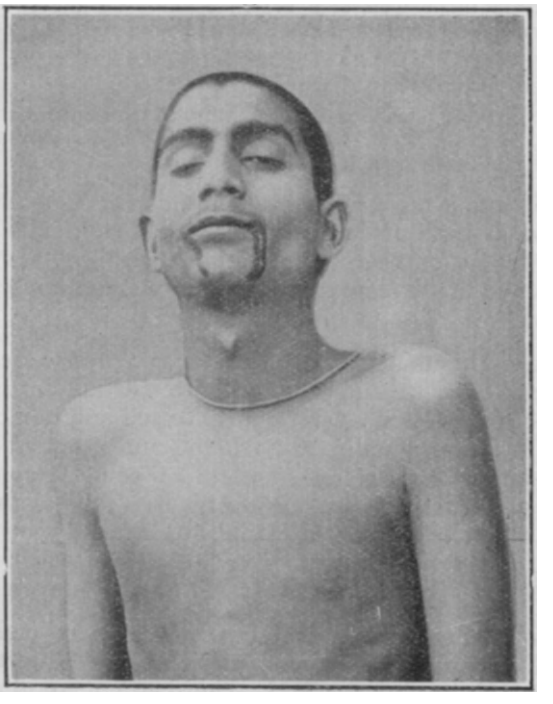


Fig. 122.—Sulphuric Acid Poisoning: Stains on angles of mouth and chin due to corrosive action of Sulphuric Acid.

The sulphuric acid of commerce is usually brown or dark in colour, and often contains impurities, such as lead sulphate, arsenic, nitric acid and the lower oxides of nitrogen. A stronger form of the acid is known as Nordhausen acid which is a brown, oily, fuming liquid and is represented by the formula $H_2S_2O_7$. It is also called pyrosulphuric acid, and is used in the manufacture of indigo.

Special Symptoms.—In addition to the general symptoms of corrosive poisoning, the following symptoms are observed :—

The tongue is swollen, and is covered with a white coating, resembling soaked parchment, which subsequently becomes darker or brown in colour. It may become a corroded and shapeless mass, if the acid is highly concentrated. The teeth are of a chalky-white colour, and are deprived of their polish. The lips are usually swollen and excoriated, and brown or even black streaks resulting from the action of the acid flowing from the mouth may be found extending from its angles to the sides of the chin and sometimes to the front of the neck. Occasionally salivation has been observed on the second or third day. In rare cases delay has been caused in the appearance of the symptoms.

In October, 1856, a man of 56 years swallowed by mistake a dessert-spoonful of oil of vitriol and was admitted into Guy's Hospital. He was able to walk upstairs to his bed and did not appear very ill, although dejected. The lining membrane of his mouth was of a brown colour. He vomited slightly at first, and there was one fluid evacuation from the bowels of a brown colour. On the two following days he appeared depressed, but there were no urgent symptoms. The case was considered slight, and there was every expectation that he would recover. However he died suddenly on the fourth day.[1]

A girl after having swallowed a quantity of concentrated sulphuric acid sat quietly down to tea with some friends, although the quantity of acid taken was sufficient to cause her death in a few hours.[2]

Sulphate of indigo is a dark blue liquid, and consists of one part of indigo dissolved in nine or ten parts of sulphuric acid. It is much used in dyeing, and may give rise to accidental poisoning. It produces almost the identical symptoms except that the mouth, vomited matter and urine are coloured blue.

Fatal Dose.—The dangerous effects of sulphuric acid depend more upon its degree of concentration than upon the absolute quantity taken. It is quite possible for a few drops of concentrated sulphuric acid to produce death from suffocation by directly coming into contact with the glottis resulting in œdema. Half a tea-spoonful of concentrated sulphuric acid administered by mistake for castor oil caused the death of a child, one year old.[3] The smallest fatal dose for an adult is one drachm, though recovery has followed four ounces of the strong acid.[4] An ounce of sulphate of indigo killed a young woman in about eleven hours.[5]

Fatal Period.—The average fatal period is from eighteen to twenty-four hours. The smallest recorded period is three-quarters of an hour,[6] the longest being forty-five weeks.[7] There is yet another case in which death took place from stricture of the œsophagus two years after the poison had been taken.[8] In children death may ensue instantaneously

1. *Taylor, On Poisons, Ed.* III, *p.* 183.
2. *Ibid., p.* 184.
3. *Med. Gaz., Vol. XXIX, p.* 147.
4. *Guy and Ferrier, Forens. Med., Ed. VI, p.* 365.
5. *Taylor, On Poisons, Ed. III, p.* 199.
6. *Gazette Medicale de Paris,* Dec. 28, 1850 ; *Ibid., p.* 192.
7. *Taylor, On Poisons, Ed. III, p.* 192.
8. *Ibid.; Beck, Med. Juris., Vol.* 2 *p.* 426.

from suffocation due to the spasmodic closure of the glottis by the acid getting into the larynx.

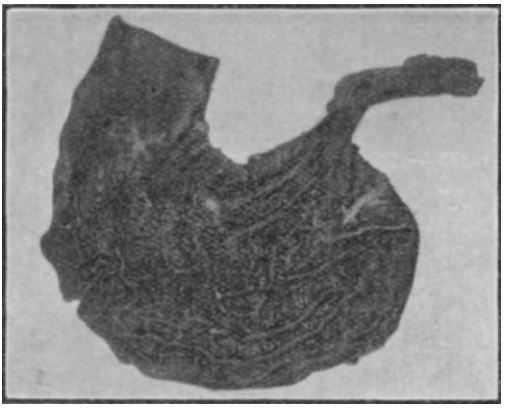


Fig. 123.—Stomach in poisoning by Sulphuric Acid.

Post-mortem Appearances.—These are the usual appearances of corrosive poisoning. The mouth, lips and sometimes the surrounding skin show brown or brownish-black corroded spots. The mucous membrane is dark-brown or black. There is great disorganization and blackening of the stomach, and its perforation is more frequent. When there is no perforation, the stomach is collapsed and contracted, the contents being a dark-brown and grumous liquid, consisting chiefly of mucus and altered blood. The mucous membrane may be of a dark-brown or black colour, and is often corrugated and detached in shreds or patches. The folds are large and deep from swelling, and are sometimes so softened as to tear even under gentle manipulation. On removing the mucous membrane the underlying coats of the stomach are red and intensely inflamed. The small intestine, especially the duodenum, may show patches of corrosion and inflammation if death has occurred after eighteen or twenty hours. Fatty changes are observed in the liver and kidneys. Coagulated blood is found in the blood vessels.

In February, 1927, F., a Mahomedan male, aged 30 years, swallowed a quantity of a mixture of sulphuric and nitric acids after murdering his wife and child, and died in eighteen hours. At the post-mortem examination held five hours after death the tongue was found yellowish-brown and corroded, and the lips were also found corroded and yellowish-brown. Yellowish-brown streaks were noted running from the middle of the lower lip down to the chin and to the right side of the front of the neck. Similar stains were found on the fingers of both the hands. The mucous membrane of the mouth and pharynx was detached in places and yellowish-brown in colour. The œsophagus was corrugated, was deprived of its mucous membrane at several places and was yellowish-brown in colour. The stomach contained a pint of a brown grumous liquid, and was corroded and almost charred. The fundus was so much thinned that it gave way on removal from the abdominal cavity. The duodenum presented the same appearance as that of the stomach. The remaining portion of the small intestine contained a sanious dirty liquid, and was congested and inflamed with hæmorrhagic patches, especially in its upper part. The large intestine was normal and contained fæcal matter. The other viscera were normal.

In his annual report of 1928, the Chemical Examiner, Punjab, reports the case of a young female child who was given some sulphuric acid by

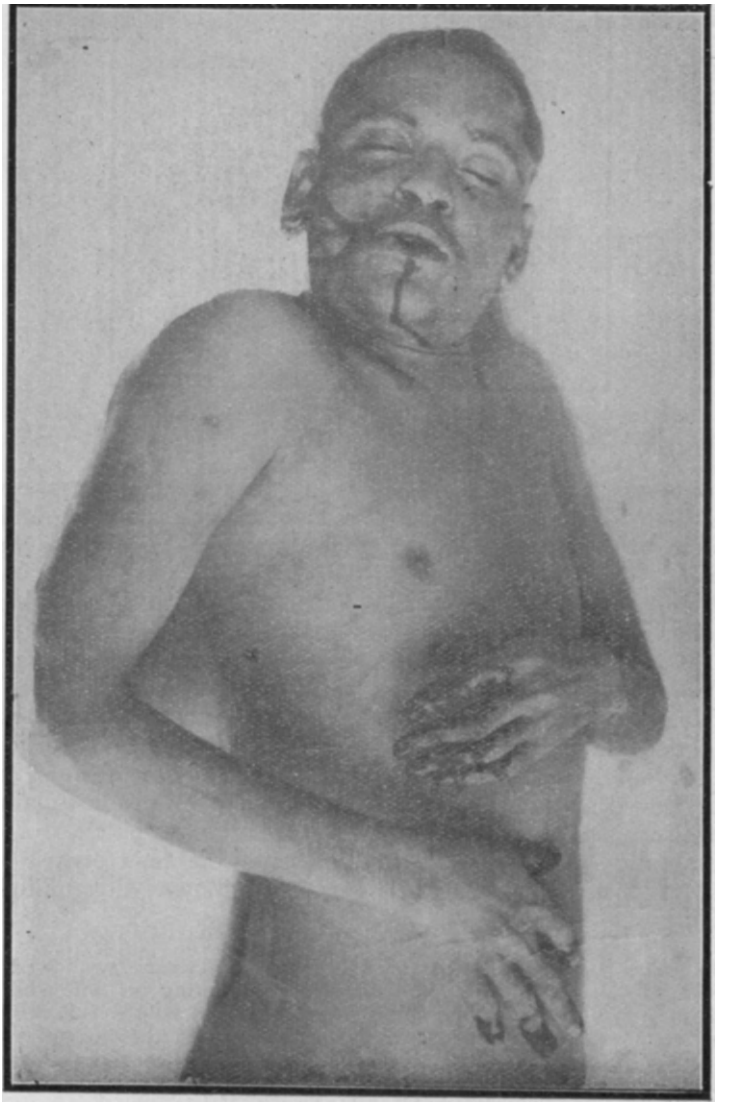


Fig. 124.—Poisoning by a mixture of sulphuric and nitric acids: Stains on lips, right angle of mouth, chin and fingers due to corrosive action of these acids.

mistake, and died rapidly. The mucous membrane of the mouth and stomach was corroded and congested. The stomach was perforated at the greater curvature by a hole about the size of a four anna piece.

Chemical Analysis.—The acid is at first separated from the organic mixture by filtration or dialysis, and then the following tests are applied for its identification :—

Tests.—1. The strong acid chars wood, while the dilute acid chars a blotting paper especially when heated. 2. Barium nitrate or chloride solution produces a white precipitate, insoluble in hydrochloric acid. 3. Heated with copper filings, mercury or chips of wood, sulphurous acid

Fig. 125.—Stomach in poisoning by a mixture of sulphuric and nitric acids.

gas is evolved ; this is known by its sulphur-like odour and by first rendering blue, and then bleaching, starch paper dipped in a solution of iodic acid or potassium iodide. 4. On heating and evaporating with veratrine on a porcelain dish, a crimson deposit is obtained.

Stains on Clothing.—The stained cloth should be soaked in alcohol and the tests applied to the alcoholic solution.

Medico-Legal Points.—Sulphuric acid is very largely used commercially in several trades. Hence it is easily obtainable and may be taken for suicidal purposes.

Owing to its acid taste and physical changes brought about in the food it is not possible to use it for homicidal purposes unless the victim happens to be a child or an adult who is drunk or helpless.

A baby,[1] aged 6 months, died at Bhandara within five hours as a result of sulphuric acid being administered to her by the step-mother during the mother's absence, in consequence of a quarrel between the two. The lining membrane of the mouth, pharynx and œsophagus was dark-brown and corroded, and the stomach was blackish with a big perforation. A case[2] occurred at Ahmedabad where a man was caught by some persons and held by them while his wife poured some sulphuric acid into his mouth. He died on the third day. A case[3] is also recorded where a young man was taken by his friend to witness a hockey match. Feeling thirsty he asked his friend for a drink of water. On drinking the water he felt burning pain in his lips, tongue and throat and vomited. The vomited matter was found to contain sulphuric acid.

Accidental cases have occurred from its having been mistaken for glycerine, syrup or castor oil.

1. *U. P. Chemical Examiner's Annual Report*, 1930 ; *Leader, June* 14, 1931, *p.* 14.
2. *Bombay Chemical Analyser's Annual Report*, 1929, *p.* 5.
3. *Madras Chemical Examiner's Annual Report*, 1929, *p.* 3.

On the 11th February, 1923, a Mahomedan woman, aged 25, swallowed about an ounce of commercial sulphuric acid by mistake for a dose of cough mixture, and immediately suffered from severe symptoms of acute poisoning, and ultimately died after six months.

The acid has been injected into the vagina as an abortifacient and administered accidentally as an enema.[1]

Vitriol Throwing (Vitriolage).—Malicious persons occasionally resort to strong sulphuric acid to disfigure the face or ruin the clothes by throwing a quantity of it at the hated person. The local effects of the acid are severe burning pain and corrosion of the tissues with the formation of brownish-black eschars, which leave permanent scars. Death may occur from the severe burns inflicted on the skin. Blindness may result, if the eyes are involved. It is necessary to wash the parts immediately with plenty of water, and the burns may be treated afterwards with linimentum calcis.

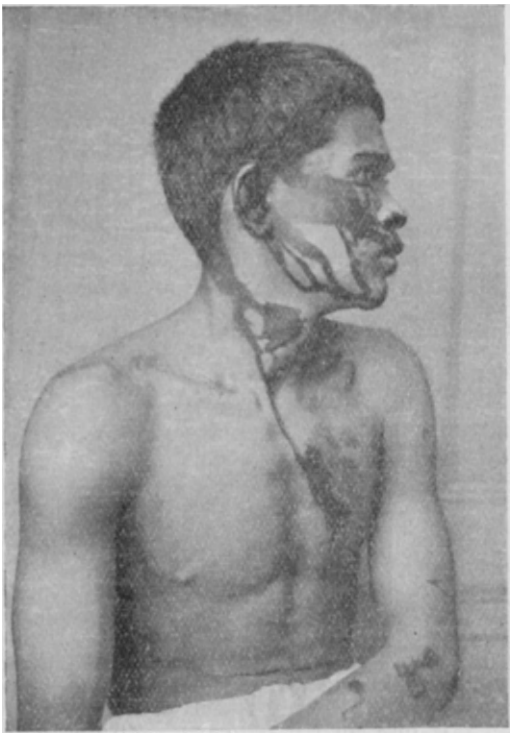


Fig. 126.—Effects of sulphuric acid thrown over the face and body. (*From a photograph kindly lent by Dr. H. S. Mehta*).

The face, hands and other parts of the body may be burnt accidentally in chemical laboratories and in manufacturing establishments, where the acid is used.

1. *Dixonmann, Forens. Med., Ed. VI, p.* 353.

NITRIC ACID (AQUA FORTIS, RED SPIRIT OF NITRE), HNO_3.

Properties.—Pure nitric acid is a clear, colourless liquid, giving off colourless fumes when exposed to the air and having a peculiar and choking odour. It is a powerful oxidising agent, and dissolves all the metals except gold and platinum. Commercial nitric acid varies in colour from yellow to deep red from the presence of lower oxides of nitrogen. Saturated with red oxides of nitrogen it is generally known as fuming nitric acid.

The pharmacopœial acid contains 70 per cent of nitric acid in water, and has a specific gravity of 1.42. The following two non-official preparations are made from it :—

1. *Acidum nitricum dilutum.*—It contains 10 per cent by weight of nitric acid. The dose is 5 to 20 minims.

2. *Acidum nitro-hydrochloricum dilutum.*—One drachm contains about 12.5 per cent by weight of nitric acid and 13.5 per cent by weight of hydrochloric acid. The dose is 5 to 20 minims.

Special Symptoms.—The lips, tongue and mucous membrane of the mouth are softened and white at first, and later become intensely yellow from the formation of xanthoproteic acid. The teeth also become yellow, and the enamel is partially destroyed by the action of the acid. The skin and cloth which come into direct contact with the acid are coloured yellow. These yellow stains turn to orange on the addition of ammonia water. The colour of the blood contained in the vomited matter is yellowish-brown. Owing to the development of a larger quantity of gas by the direct action of the acid on organic matter in the stomach the abdomen is more distended and tender than in poisoning by sulphuric acid. Gaseous eructations are also more frequent and distressing with this acid. Lock-jaw and insensibility are known to have occurred as special symptoms. The pouring of nitric acid into the ear has caused death.[1]

The fumes of nitric acid produce irritation of the eyes, cough and dyspnœa, and may cause death immediately from suffocation, or later from pneumonia. Several deaths from inhalation of the fumes have been recorded.

Mr. Stewart and one of the janitors of an educational institute were carrying a jar of nitric acid when it fell on the floor, and broke, and in attempting to save some of the spilt acid, they inhaled the fumes which were immediately diffused. Mr. Stewart returned home unconscious of the mischief which had been done. After an hour or two difficulty of breathing came on, and he died in ten hours after the accident. The janitor suffered from similar symptoms and died the following day.[2]

In a factory near Bucharest three firemen were called to put out a fire due to the breakage of a case containing bottles of nitric acid. The acid was concentrated and contained no undue proportion of nitrous acid and sulphur impurities. The sawdust in the packing became soaked with the acid and burst into flames in many places. When the firemen arrived they found a thick pungent smoke which caused an intense but transient sensation of irritation in the respiratory passages. They

1. *Morrison, Arch. gen. de Med.*, 1826, *XI*, 104 ; *Witthaus, Med. Juris. and Toxic., Vol. IV, p.* 295.
2. *Chemical News, Mar.* 14, 1863, *p.* 132.

were taken to hospital, but were discharged at their own request after a few hours. All the three, however, were brought back to the hospital on the same day ; they vomited, complained of sharp pain and quickly developed pleurisy and broncho-pneumonia. One of the three died and the other two recovered.[1]

Fatal Dose.—The smallest quantity on record is two drachms which killed a boy, aged 13 years ; a similar dose killed an adult woman in 14 days. But a smaller quantity—even a drachm—would suffice to kill a child, and under certain conditions, an adult ; for the fatal result depends on the extent of the mischief produced by its corrosive action on the throat, windpipe and stomach.[2] Recovery has taken place after half an ounce or more.

Fatal Period.—The average fatal period is from twelve to twenty-four hours. A Hindu silver-smith took one ounce of the pure acid and died in ten hours.[3] A goldsmith's wife drank the concentrated acid with the intention of committing suicide, and died after twenty days.[4] The shortest recorded period in an adult is one hour and forty-five minutes and a few minutes in an infant while the longest recorded periods are eight months and two years.[5]

Post-mortem Appearances.—The skin and the mucous membranes are yellow in colour, but the colour of the mucous membrane of the stomach is greenish if bile is present. The stomach wall is soft, friable and ulcerated, but perforation is not so common as in sulphuric acid poisoning. There may be corrosion of the duodenum. In his annual report for 1929, the Chemical Analyser to the Government of Bombay reports a case of death by nitric acid poisoning, in which the post-mortem examination showed that the lips and fingers were stained yellow and corroded. The alimentary canal from the lips to the duodenum was stained yellow and there was necrosis of the coats of the stomach with perforation.

In death from inhalation of the nitric acid fumes the larynx, trachea and bronchial tubes are usually congested, and the lungs are, sometimes, œdematous or show an effusion of blood. Inflammatory changes in the lining membrane of the right auricle of the heart may be found in some cases.[6]

Chemical Tests.—1. If a strong ferrous sulphate solution and sulphuric acid are added to a solution containing nitric acid, a brown ring is formed at the junction of the two fluids.

2. Nitric acid forms a blood red colour with brucine and a rich orange colour with morphine.

3. When heated with strong sulphuric acid and copper, reddish-brown fumes of nitric oxide are given off, and the solution becomes green.

4. If caustic potash or ammonia is added to a nitric acid stain on cloth, the yellow colour changes to orange. The colour disappears if caused by iodine, but no change occurs on a stain caused by bile.

1. *Lancet, Dec.* 7, 1929, *p.* 1218.
2. *Taylor, On Poisons, Ed. III, p.* 208.
3. *Ind. Med. Gaz., June,* 1902, *p.* 211.
4. *Madras Chemical Examiner's Annual Report,* 1929, *p.* 3.
5. *Taylor, On Poisons, Ed. III, pp.* 208, 209.
6. *Holland, Med. Chem. and Toxic., Ed. V, p.* 173.

Medico-Legal Points.—Nitric acid is very largely employed in the arts and manufactures. It is used for cleansing nickel ornaments and separating gold from other metals. It is also used in the preparation of gun cotton, nitroglycerine, picric acid, sulphuric acid and colouring matters.

Cases of poisoning by this acid are not very common. The cases that have been recorded are chiefly suicidal or accidental. The Punjab Chemical Examiner[1] reports a case in which a young student in one of the Lahore Colleges finding that he had failed in one of the subjects shut himself in his room, and committed suicide by taking nitric acid. A remarkable accidental case occurred, in which a woman, while trying to pour strong nitric acid from a bottle into the cavity of a carious tooth, swallowed some of it, and died from its effects.[2] A few homicidal cases have occurred, the victims being either infants and children, or drunken, helpless adults. Taylor[3] mentions a case, in which a woman killed her infant shortly after its birth by pouring nitric acid down its throat. A case occurred in France, where a woman died from the effects of nitric acid poured into her ear by her husband while she was intoxicated.[4] The acid has also been used as an abortifacient. Strong nitric acid has occassionally been thrown in the face to destroy or disfigure the features.

Cases..—1. On the 3rd July, 1923, Dr. Monmath Basu, a medical practitioner attached to Messrs. Mackintosh Burn Company's brick-field at Jogernathnagore Akra, was playing cards with some friends in a house close to his dispensary when he was disturbed and startled by some shouts of "thief, thief", and forthwith ran to his quarters. On a search being made, one Askhoy Kumar Nascar was found standing at a place close to the outer side compound wall. The doctor took him to be a thief, dragged him into the dispensary, placed him upon a chair and emptied a bottle of strong nitric acid over his head. Then the contents of a second bottle of the acid were similarly poured on his back and other parts of his body. The poor man fell down groaning in agony, and was removed, on an improvised stretcher, to a remote part of the brick-field. Unable to bear his great agony the man cut his own throat with a fish knife. The next morning some neighbours removed him to the Alipore Police Hospital, where he succumbed to his injuries. The accused was found guilty under Sec. 304, I. P. C., and sentenced to one year's rigorous imprisonment.—*Leader, Oct.* 15, 1923.

2. At about 6 a.m. on July 21, 1932, Baijnath, the complainant, was proceeding along Beni Bandh when the two accused, Haridas and Ramparsad, assaulted him with *lathis*. Bajinath fell down and accused Ramparsad sat upon his chest, while Haridas took a phial from his pocket and poured out the contents, presumably nitric acid, into Baijnath's right eye, and when an attempt was made to pour the same into the left eye as well, they fell on the eyebrows instead. The result was the permanent loss of the vision of the right eye. They were found guilty under section 326, I. P. C., and were each sentenced to rigorous imprisonment for two years including solitary confinement for one month. They were further ordered to pay a fine of Rs. 200 each, in default of which each should undergo a further term of six months' imprisonment. Out of the fine, if realised, Rs. 300 were ordered to be paid to Baijnath.—*Leader, Oct.* 5, 1932, *p.* 6.

---

1. *Annual Report,* 1928, *p.* 9.
2. *Brit. Med. Jour., Vol. I,* 1812, *p.* 235.
3. *On Poisons, Ed. III, p.* 201.
4. *Witthaus, Loc. Cit.*

HYDROCHLORIC ACID (MURIATIC ACID, SPIRITS OF SALTS), HCl.

Properties.—Pure hydrochloric acid is a colourless gas, having a specific gravity of 1.259 and an intensely irritating odour. It is extremely soluble in water, one volume of this liquid dissolving 480 volumes of the gas at 0° C. (32°F.). The acid of commerce, which is generally known as muriatic acid or spirits of salts, is a solution of this gas in water, having a yellow colour, fuming strongly in damp air, and yielding dense white vapours with ammonia. It not infrequently contains a trace of arsenic, derived from sulphuric acid used in generating it. The acid of the British Pharmacopœia is a colourless fuming liquid, having a specific gravity of 1.161 and contains 32% of hydrochloric acid by weight (limits 31 to 33). *Acidum hydrochloricum dilutum* is an official preparation which contains 10 per cent of hydrochloric acid by weight. The dose is 5 to 60 minims.

Special Symptoms.—It is less active than the other two acids. Hence the symptoms produced by it are much milder. It does not stain the skin or mucous membrane, but stains dark cloth reddish-brown. Salivation, convulsions, delirium and paralysis of the limbs have occurred as special symptoms in some cases.

The fumes of the acid cause great irritation of the air passages. Those who are constantly exposed to the fumes of this gas suffer from chronic poisoning. It is characterised by coryza, conjuctivitis, pharyngitis, laryngitis and bronchitis.

Fatal Dose.—The usual fatal dose is four drachms of the concentrated acid. The smallest dose that has proved fatal to a girl, 15 years old, is one drachm.[1] Recovery has taken place after swallowing one ounce and a half in one case and two ounces in another.[2]

Fatal Period.—The usual fatal period is from eighteen to thirty hours. In a case recorded by Christensen[3] death occurred in one and a half hours after a dose of about 200 c. c. hydrochloric acid. Death has also occurred in two hours, and has been delayed for several days.

Post-mortem Appearances.—The mucus membranes acted on by the acid are usually ash-grey, or black in colour interspersed with erosions. The stomach wall is red owing to acute gastritis. Perforation is very rare indeed.

Chemical Analysis.—It should be remembered that this acid is found in a free state to an extent of 0.2 per cent or more in the gastric juice. Hence the detection of a minute quantity in the stomach contents is no proof of poisoning by this acid, unless distinct marks of its chemical action

1. *Johnson, Brit. Med. Jour., March,* 1871, *p.* 271.
2. *Dixonmann, Forens. Med., Ed. VI, p.* 359.
3. *Ugeskrift for Læger, Copenhagen, Jan.* 26, 1928, *p.* 86; *Jour. Amer. Medical Assoc., March* 31, 1928, *p.* 1088.

are seen on the throat and stomach. It may be recovered from vomit by distillation, and should then be tested by the following tests :—

1. A solution of silver nitrate produces a heavy, curdy, white precipitate of silver chloride, insoluble in excess or in nitric acid, but soluble in ammonium hydroxide. The white precipitate becomes grey on exposure to sunlight.

2. If heated with manganese dioxide, chlorine gas is evolved, known by its greenish-yellow colour, irritating smell and bleaching action on vegetable colouring matter.

3. When brought near ammonia, white fumes of ammonium chloride are given off.

Medico-Legal Points.—Hydrochloric acid is chiefly used for preparing chlorine, for dissolving metals and for medicinal purposes. It has been, sometimes, used for erasing writing in attempts at forgery. Accidental and suicidal cases of poisoning by this acid have occurred in Great Britain as also in India. A case[1] occurred in Bombay where a Parsi lady, aged 22, died as a result of having accidentally swallowed a quantity of hydrochloric acid, mistaking it for a dose of some medicine prescribed for a cold.

Homicidal cases are very rare indeed. Hydrochloric acid was introduced into the vagina of a pregnant girl with a view to procure abortion but without success. Atresia vaginæ was, however, produced to such an extent that it was necessary to perforate the child at term.[2]

Hydrochloric acid was thrown in the face of a young Parsi. As a result of this criminal assault he suffered from fulminating conjunctivitis of both eyes. Hydrochloric acid was detected on the coat of the victim and in the glass which was used.[3] A bottle of hydrochloric acid was also thrown on the Head Ticket Inspector at Victoria Terminus, Bombay, while he was standing near the Crawford Market. As a result of this he received grievous burns.[4]

HYDROFLUORIC ACID, HF.

This is a colourless gas, which becomes a fuming liquid when dissolved in water. On account of its etching property on glass it is kept in gutta-percha bottles.

Acute Poisoning—Symptoms.—The fumes of the gas, when inhaled, produce inflammation and ulceration of the conjuctiva, nostrils and gums, and severe cough due to laryngitis and bronchitis. There may be intense vomiting and collapse.

The liquid acid produces on the skin severe and painful burns and ulcers which are difficult to heal. When taken internally, it immediately produces retching, vomiting, agonizing pain in the abdomen and diarrhœa. Collapse sets in and death occurs usually from closure of the glottis with shreds of mucous membrane.

Sodium flouride is a white powder, crystallising in colourless cubes, having an acrid, bitter taste and dissolving in 25 parts of water. It is widely used as a wood preservative and as an insecticide. It is the constituent of most "roach powders"

1. *Times of India, Jan.* 17, 1930.
2. *Jhrbt. u. d. Fortschr. d. Geb.*, 1893, 858; *Witthaus, Med. Juris. and Toxic., Vol. IV, p.* 279.
3. *Bombay Chem. Analyser's Annual Report,* 1931.
4. *Times of India. Jan.* 30, 1937.

It is also used for the etching of glass. It has, sometimes, been mistaken for baking powder, magnesium sulphate and powdered sugar, and has produced poisonous symptoms. It is a general protoplasmic poison and exerts a strong local irritant action on the mucous membranes.

The symptoms of poisoning by this salt are pain in the stomach, nausea, vomiting, diarrhœa, muscular cramps, spasmodic contraction of the extremities and tetany.

Fatal Dose and Fatal Period.—In one case half an ounce of the acid solution caused death in thirty-five minutes.[1] In another case about a table-spoonful of 9 per cent acid, diluted with water proved fatal in about an hour.[2] In 1885, a man in New York swallowed 118 c.c. of the acid used in glass etching and died in about two hours.[3] A woman, 39 years old, took a heaping table-spoonful of sodium fluoride in water by mistake for magnesium sulphate, and died in seven hours.[4] Half a tea-spoonful of silico-fluoride has proved fatal.[5]

Treatment.—Ammonia vapour is the antidote, when the fumes are inhaled. Weak alkalies should be administered to neutralize the liquid acid, when taken internally. Milk and demulcent drinks should be administered and castor oil should be given as a purgative. Gastric lavage with lime water or a weak solution of calcium chloride and the intravenous injection of calcium chloride or intramascular injection of calcium gluconate have been recommended in acute poisoning by sodium fluoride.[4]

Post-mortem Appearances.—The lips, tongue and mouth may show white patches or may be charred. The œsophagus may show shreds of the denuded epithelium. The mucous membrane of the stomach may be ecchymosed or blackened, but not eroded. The trachea, lungs and other organs are hyperæmic.

In the case in which a woman died after taking a heaping table-spoonful of sodium fluoride, the post-mortem examination showed that the skin had deep greyish-blue cyanosis, most marked over the scalp, face, neck, upper thorax, and upper extremities. The buccal and pharyngeal mucous membrane and the mucosa of the vagina, labia minora and the inner aspect of the labia majora showed similar bluish discoloration. The gastric mucosa and sub-mucosa showed extreme acute passive congestion and œdema with multiple petechiæ; the mucosa was covered with a thick layer of blood-tinged mucin. The lower third of the œsophagus showed similar changes. The duodenal mucosa showed more extensive petechial hæmorrhages. The liver was of deep slate-purple with mottled greyish-yellow areas, presenting acute passive congestion with advanced cloudy swelling and patchy fatty degenerative infiltration. The kidneys showed passive congestion and cloudy swelling. The lungs were congested.

Chronic Poisoning.—This occurs among those who are exposed to the fumes of hydrofluoric acid or who ingest small quantities of its salts for a prolonged period. The symptoms as described by Holland[6] are neuralgia, weak heart, dropsies, phlebitis, painful urination and loss of calcium salts from the system, impairing nutrition of the bones. Death occurs from respiratory failure.

II. ORGANIC ACIDS

OXALIC ACID (ACID OF SUGAR), $H_2 C_2 O_4$, $2 H_2 O$

Oxalic acid is prepared from sugar by oxidation with nitric acid, but it is manufactured on a large scale from pine sawdust which is oxidised by fusion with caustic alkalies. It can also be prepared by heating sodium or potassium formate.

1. *King, Trans. Path. Soc., London,* 1873, *XXIV,* 98.
2. *Stevenson, Brit. Med. Jour.,* 1899, *Vol. II, pp.* 1145, 1376.
3. *Witthaus, Med. Juris. and Toxic., Vol. IV, p.* 309.
4. *Sharkey and Simpson, Jour. Amer. Med. Assoc., Jan.* 14, 1933, *p.* 97.
5. *Chem. Zeitsch.,* 1925, 49, *p.* 805.
6. *Med. Chem. and Toxic., Ed. V, p.* 149.

Properties.—Oxalic acid occurs in the form of colourless, transparent prismatic crystals, and resembles in appearance the crystals of magnesium sulphate and zinc sulphate for which it is sometimes mistaken. The following are the distinguishing tests by which they can be recognised :—

—	Oxalic Acid.	Magnesium sulphate.	Zinc sulphate.
Taste.	Sour and acid.	Nauseating bitter.	Metallic bitter
Reaction.	Strongly acid.	Neutral.	Slightly acid.
Heat.	Sublimes.	Fixed.	Fixed.
Sodium Carbonate.	Effervescence, but no ppt.	No effervescence, but a white ppt.	No effervescence, but a white ppt.
Stains of ink, or iron moulds.	Disappear.	No action.	No action.

Oxalic acid is soluble in ten parts of cold water and in two and a half parts of cold alcohol, but very sparingly in ether. It volatilises completely at 150° without leaving any residue. Heated with strong sulphuric acid it splits up into carbon dioxide, carbon monoxide and water.

Symptoms.—Oxalic acid has both a local and a remote action on the system. It acts locally as corrosive when administered in a large quantity and in a solid or concentrated form but, when taken in weaker solutions or in combination, acts locally as irritant and the nervous symptoms are more evident. It also acts as a poison when applied to a wound.

The symptoms begin immediately or soon after taking a large dose of the concentrated acid. These are a very sour acid taste, thirst, pain and burning in the mouth, throat and stomach, extending over the whole abdomen. Vomiting soon sets in. It very often persists till death. The ejected matter contains altered blood and mucus, and appears greenish-brown or black, resembling coffee grounds. In some cases vomiting may not occur or may be delayed for some time. Thus, in one instance, in which the acid was much diluted, vomiting did not occur for seven hours.[1] Tenesmus is present, but purging is rare unless the case is prolonged for some time. The urine is diminished in quantity and may be suppressed for two or three days. Later, it increases in quantity and contains albumin in a large quantity. The sediment after a few hours shows hyaline casts and octahedral crystals of calcium oxalate under the microscope. Great prostration occurs with cold, clammy sweats, a feeling of numbness of the limbs, feeble, irregular and rapid pulse, and shallow gasping hurried respirations. The condition of collapse passes into coma, which ultimately ends in death. Sometimes cramps, convulsions, lock jaw and delirium precede death.

In his treatise on *Poisons* Christison[2] has remarked : "If a person immediately after swallowing a solution of a crystalline salt which tasted purely and strongly acid is attacked with burning in the throat, then with burning in the stomach, vomiting, particularly of bloody matter,

1. *Christison, Poisons, p.* 211.
2. *Amer. Ed. I, p.* 179.

imperceptible pulse and excessive languor, and dies in half an hour, or still more in twenty, fifteen or ten minutes, I do not know any fallacy which can interfere with the conclusion that oxalic acid was the cause of death. No parallel disease begins so abruptly and terminates so soon; and no other crystalline poison has the same effect."

Fatal Dose.—The average fatal dose is four drachms. The smallest recorded fatal dose is one drachm of the solid acid which proved fatal to a boy, sixteen years old.[1] Recoveries have taken place, on prompt administration of remedies, after an ounce[2] or even two ounces[3] had been swallowed.

Fatal Period.—The shortest recorded periods are three minutes in one case[4] and ten minutes in another.[5] The usual period is one to two hours. Death may be delayed for several days, the longest period known being twenty-one days.[6]

Treatment.—Give chalk, calcined magnesia, whiting or plaster from a wall in a small quantity of water or milk with a view to neutralise the acid and to form insoluble calcium oxalate. A saccharated solution of lime is considered the best form of treatment. Alkalies or their carbonates must not be administered as they unite with oxalic acid, and form soluble poisonous salts. Large draughts of water should also be avoided as they dissolve the poison, and thus increase its rapid absorption.

After the acid is neutralised in the stomach, vomiting may be promoted by emetics, or the stomach may be washed out very cautiously, and the bowels may be relieved by an enema or by a purgative, preferably castor oil. The usual symptomatic treatment must then be followed. Infusion[7] of an isotonic or hypertonic solution of glucose has been recommended, as retention of urea seems to be the principal disturbance. Coutt ascribes to this the cramps and convulsions often observed in such cases.

Post-mortem Appearances.—If the acid has been taken in a concentrated form, the marked signs of corrosion are found in the mouth, throat, œsophagus and stomach. Their mucous membranes are white and shrivelled, and are easily detached from the underlying tissues. They may, sometimes, be found black in colour from altered blood. The inner surface of the œsophagus is corrugated and shows longitudinal erosions. The stomach contains a dark-brown, grumous liquid, acid in reaction. The blood vessels are seen as dark brown or black streaks over its internal surface. Perforation of the stomach is rare, though the walls are often softened and easily torn. The stomach may be pale and not corroded if death has occurred immediately after taking the poison. The intestines generally escape, but the upper part of the duodenum may be affected.

1. *Barker, Lancet, Dec.* 1, 1855, *p.* 521.
2. *Taylor, On Poisons, Ed. III, pp.* 232, 233.
3. *Tapson, London Med. Gaz.,* 1842, *Vol. II, p.* 491.
4. *Ogilvie, Lancet, Aug.* 23, 1845, *p.* 205.
5. *Med. Times and Gazette, April* 25, 1868, *p.* 456.
6. *Dixonmann, Forensic Medicine, Ed. VI, p.* 360.
7. *Revista Medica Latino-Americana, Buenos Aires, July,* 1925, *p.* 1118; *Jour. Amer. Med. Assoc., Oct.* 10, 1925, *p.* 1170.

The kidneys are congested and loaded with oxalates.

If the acid is very diluted there will be the signs of local irritation, *viz.*, redness, congestion and inflammation of the mucous membrane.

If the effects are only narcotic there will be congestion of the lungs, liver, kidneys and brain, without any local appearances.

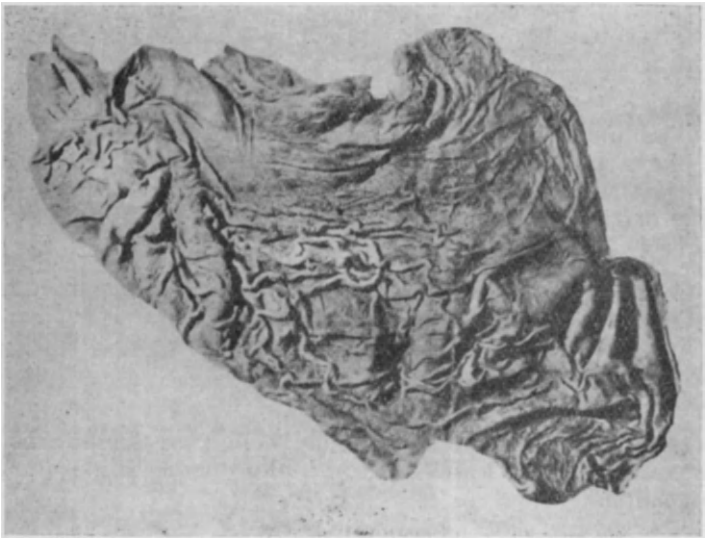


Fig. 127.—Stomach in poisoning by Oxalic Acid.
(*From Pathology Museum, Grant Medical College, Bombay*).

In the case[1] of a Parsi who committed suicide with oxalic acid, the mucous membranes of the mouth and œsophagus were whitened, soft and easily stripped off. The mucous membrane of the stomach was corroded, and its whole thickness was perforated in one place. The interior of the stomach and its contents were blackened. The intestines were grey and gangrenous looking.

Chemical Analysis.—To separate the acid the organic mixture may be dialysed, or it may be boiled and filtered. To the filtrate is added acetate of lead, when a precipitate of lead oxalate is formed. The precipitate is washed with water and in the watery solution a current of hydrogen sulphide is passed for about half an hour, so that a black precipitate of lead sulphide is thrown down. The black precipitate is now filtered, the filtrate is heated to remove any excess of hydrogen sulphide and is evaporated to dryness, when the crystals of oxalic acid are found which can be tested by dissolving them in water. There is yet a third method by which the organic mixture is evaporated and extracted with alcohol acidified with a little hydrochloric acid. The alcoholic solution is then evaporated to dryness, and the residue is dissolved in water to apply the tests for oxalic acid.

1. *Bombay Chemical Analyser's Annual Report*, 1923, *p.* 3.

Tests.—1. A solution of barium nitrate gives a white precipitate of barium oxalate, soluble in hydrochloric acid or nitric acid without effervescence.

2. A solution of silver nitrate gives a copious white precipitate, soluble in ammonia and nitric acid.

3. Calcium chloride or sulphate gives a white precipitate, insoluble in acetic acid, but soluble in strong hydrochloric acid.

4. Lead acetate gives a white precipitate, soluble in nitric acid, but insoluble in acetic acid.

Medico-Legal Points.—In the form of oxalate of ammonium, sodium, potassium or calcium, oxalic acid exists as a natural constituent of several plants and vegetables, such as sorrel, rhubarb, cabbages, lichens and guano. Hence it may gain access to the body through food and drugs of vegetable origin. It often occurs as a constituent of the human urine, 0.02 gramme (0.3 grain) being excreted in 24 hours.

Oxalic acid is largely used in calico printing, in the manufacture of straw hats, and in cleaning brass and copper articles, and wooden surfaces. It is used for removing writing and signatures from paper and parchment documents. It is a common household remedy for removing ink-stains and iron-moulds from linen.

Cases of accidental poisoning by oxalic acid have, sometimes, occurred from it having been swallowed in mistake for a saline purgative of magnesium sulphate.

A young woman took 2 drachms of oxalic acid by mistake for magnesium sulphate at about 8 a.m. on the 29th October, 1930. Immediately she complained of burning sensation in the mouth, throat and abdomen, and induced vomiting by tickling her fauces. She vomited many times and two hours later she brought up a good quantity of blood. She was removed to the King George's Hospital, Lucknow, where she was found restless with a rapid pulse (132 per minute) and hurried respirations (52 per minute). She complained of very severe epigastric pain. She had no difficulty in swallowing, but experienced burning pain in the abdomen after swallowing liquids. Excoriations were present on the tongue and the post-pharyngeal wall, but not on the lips and gums. She vomited occasionally, and brought up a few streaks of blood with the detached pieces of the mucous membrane. She was given lime water and morphine hypodermically. She was discharged cured on the third day.

A case[1] occurred at Amritsar where 30 grains of oxalic acid were used instead of 40 grains of tartaric acid in the manufacture of "darling Seidlitz powders". Due to the prompt action of the Police Department, all the tins containing these powders were confiscated from different areas in the Punjab, and no accidents occurred.

During recent years cases of suicide by oxalic acid poisoning, although very few, have occurred in India due to its increased use as a remover of stains on clothes and the ease with which it can be obtained at a druggist's shop. Owing to its taste, it is rarely used for homicidal purposes.

Oxalic acid is eliminated chiefly by the kidneys and, in non-fatal cases, is found in the urine for about two weeks.[2]

1. *Punjab Chemical Examiner's Annual Report,* 1929 *p.* 10.
2. *Hood, Lancet, Vol. I,* 1886, *p.* 347.

When applied externally, oxalic acid does not produce corrosion of the skin, nor does it produce any deleterious effects on the system. Workmen engaged in trades requiring the constant handling of the acid are not known to have suffered from ill-health except that their finger nails are white, opaque and brittle, but they may, in rare cases, suffer from the symptoms of chronic poisoning, especially when exposed to its vapour.

A man,[1] aged 53, was employed in America in cleansing radiators by means of boiling them over a fire with a strong solution of oxalic acid. During the operation, he scooped crystals of the acid with his fingers into the radiator filler. As the boiling progressed the concentration increased and vapour was emitted which was extremely irritating to breathe. In due course the man was compelled to leave his work and later became disabled and confined to bed. The initial symptoms consisted in epistaxis, severe headaches, spells of vomiting, constant pain in the back and rapid loss of weight. Extreme nervousness developed and the man stated that he was scarcely able to move, as he seemed to be paralysed. An ulcer was noticed on the nasal septum with marked congestion of the mucous membrane of both nostrils and down the back of the throat. Anæmia and severe albuminuria were present.

Binoxalate (Acid oxalate) of Potassium, KHC_2O_4, $2H_2O$.—This is commercially known as " salt of sorrel " or " essential salts of lemon ", and is used for the same purpose as oxalic acid. It is acid in reaction and sour in taste, and dissolves in 40 parts of cold, and in 6 parts of boiling, water. It is likely to be mistaken for acid tartrate of potassium (cream of tartar).

This salt is practically as poisonous as oxalic acid, producing similar symptoms and post-mortem appearances, and requiring similar treatment. Four drachms may be regarded as a fatal dose.

A lady, recently confined, took half an ounce of the salt by mistake for cream of tartar; she was immediately seized with violent pain in the abdomen and convulsions, and died in eight minutes. On inspection the lining membrane of the stomach and small intestine was found inflamed.[2] Braithwaite[3] records the case of a young woman, aged 24 years, who swallowed three-quarters of an ounce of salts of lemon at about 6-45 p.m. and died at 7-10 p.m., *i.e.*, in 25 minutes. On post-mortem examination white corrosions were seen at the left corner of the mouth, on the tongue and on the inside of the cheeks. The stomach showed at the cardiac end two circular perforations about $1\frac{1}{2}$ inches apart. The stomach wall around these was found to be extremely thin and quite denuded of the mucous membrane for a radius of several inches.

Cases of recovery have been recorded. For instance, a young lady, aged 20 years, swallowed an ounce of acid oxalate of potassium dissolved in warm water, but recovered in two days under proper medical treatment.[4]

Chronic Poisoning.—A fatal case[5] of chronic poisoning by this salt has been recorded, in which a girl was charged with the murder of her father. He began to be ill about December 5, and died on January 26

1. *C. D. Howard, Jour. Ind. Hyg.*, 1932, *XIV, pp.* 283-290; *The Med.-Leg. and Criminological Review, Apr.*, 1933, *p.* 145.
2. *J. de Chim. Med.*, 1842, *p.* 211.
3. *Brit. Med. Jour., Vol. I*, 1905, *p.* 183.
4. *Med. Times and Gaz., Vol.* 27, *p.* 480.
5. *Edin. Month. Jour., July*, 1862, *p.* 92.

following. He suffered from vomiting, heat and irritation in the mouth and throat, prostration of strength and constant pains in the chest and abdomen. The post-mortem examination showed inflammation of the mucous membrane of the stomach and part of the bowels. They contained a dark coloured fluid. The mucous membrane of the gullet was destroyed. The coats of the stomach, which were thickened and injected had a gangrenous appearance.

CARBOLIC ACID (PHENOL, PHENYL ALCOHOL OR PHENIC ACID), C_6H_5OH

This is hydroxybenzene obtained from coal tar oil by fractional distillation, and is commonly prepared from acetylene by synthesis.

Pure carbolic acid occurs as long, colourless, prismatic, needle-like crystals, which turn pink on exposure to light, and are deliquescent in moist air. It has no acid reaction, but forms carbolates when acted upon by strong bases. It has a characteristic odour and has a burning, sweetish taste. It is slightly soluble in cold water (1 in 13), but freely in boiling water), alcohol (90%), ether, chloroform, glycerine and fixed and volatile oils. To all these it communicates its characteristic odour. The pharmacopœial dose of carbolic acid (phenol, B.P.) is 1 to 3 grains. The other preparations prepared from it are—

1. *Phenol Liquefactum* (Acidum Carbolicum Liquefactum).—dose, 1 to 3 minims.

2. *Glycerinum Phenolis.*—dose, 5 to 15 minims.

3. *Suppositorium Phenolis.*—1 grain in each.

4. *Trochiscus Phenolis.*—Each contains approximately ½ grain in each.

5. *Unguentum Phenolis.*—Phenol made up with paraffin, lard and bees wax.

The crude carbolic acid of commerce is a dark-brown liquid containing several impurities, chiefly cresol.

Poisoning by carbolic acid is known as *carbolism.* The acid in a concentrated form acts locally as a corrosive, and remotely as a narcotic poison. It coagulates albumin, but does not enter into chemical combination with it, and thus it has a great penetrating power. Applied to the skin, it causes a burning sensation, followed by tingling, numbness and anæsthesia, and produces a white, opaque eschar which, falling off in a few days, leaves a brown stain, which may persist for several weeks. When applied for some time and prevented from evaporating by the application of India rubber tissue, carbolic acid may cause necrosis of the part even in weak solutions. It causes irritation and necrosis of the mucous membranes and, if applied in sufficient quantity, may lead to sloughing and inflammation.

Symptoms.—Immediately after swallowing the concentrated acid, there is a hot burning sensation in the mouth, throat and stomach, with occasional vomiting of frothy mucus. The mucous membranes of the lips and mouth become hard and white. Owing to the rapid absorption of the

acid these symptoms are soon followed by giddiness and insensibility, which soon deepens into coma. The lips are cyanosed, the pupils are contracted, the skin is cold and clammy, the pulse is small and thready, and the respirations are slow, laboured or stertorous. There is a strong odour of carbolic acid in the breath. Convulsions and lock-jaw may be present. The urine is suppressed or scanty. When voided, it is normal in colour, or of a greenish hue, which becomes dark or olive green on exposure to the air, and stains the linen as well. This change of colour is due to the formation of hydroquinone and pyrocatechin, oxidation products of carbolic acid, and serves as a warning of the toxic properties of the acid when used as an antiseptic dressing for some time. This symptom is known as carboluria.

Death results from paralysis of the respiratory and cardiac centres.

Fatal Dose.—Four drachms is the average fatal dose, though an oily solution of one drachm has proved fatal.[1] A quarter of a tea-spoonful dissolved in glycerine killed a child, six months old.[2] Doses of 6 or 7 grains have caused symptoms of poisoning. On the other hand, recoveries have ensued after large doses, as much as six ounces.[3]

Fatal Period.—Death usually occurs in three to four hours, although it has occurred in a few minutes. An aged male took an ounce of the liquid acid, and died in three minutes.[4] A male, 60 years old, died in ten minutes after swallowing half an ounce of the acid.[5] A girl of 17 years died also in ten minutes after taking about one ounce.[6] In a case where carbolic acid was applied to the back of an adult, death occurred in twenty minutes.[7] On the other hand, death has been delayed for six hours[8] and even for several days.

Treatment.—Ordinary emetics often fail to produce vomiting owing to the local anæsthesia. Pass a soft stomach tube with caution, and wash out the stomach with lukewarm water containing syrup calcis or sodium sulphate, until the contents of the stomach lose their peculiar odour. Sodium sulphate in strong solution is one of the best known antidotes. It forms the harmless sodium sulphocarbolate. In the adbsence of sodium sulphate magnesium sulphate may be used. Alcohol is not an antidote, but a 10 per cent solution is used in the belief that it will neutralise the action of carbolic acid and thus prevent extensive sloughing of the tissues. No doubt the effect of alcohol is simply that of diluting and washing away the carbolic acid. Experiments carried out in the United States Hygiene Laboratory show that the toxicity of carbolic acid is increased by alcohol.[9] Give demulcents, such as white of egg and milk. Give atropine sulphate hypodermically before administering stimulants, such as caffeine,

1. *Lancet, Jan.* 30, 1869, *Vol. I, p.* 179.
2. *Brit. Med. Jour., May* 20, 1882.
3. *Hind, Lancet, April* 12, 1884, *p.* 659.
4. *Phil. Med. Times, Vol. II, p.* 214; *Collis Barry, Leg. Med., Vol. II, Ed. II, p.* 506.
5. *Barlow, Lancet, Sept.* 18, 1869, *p.* 404.
6. *Marwood, Austral. Med. Gaz.,* 1893, *XII, p.* 78.
7. *Collis Barry, Leg. Med., Vol. II, Ed. II, p.* 507.
8. *Virchow and Hirsch, Jahresbericht., Vol. I,* 1871, *p.* 337.
9. *Jour. Amer. Med. Assoc., July* 15, 1916, *p.* 233.

strophanthine and strychnine. Administer intravenously normal saline containing 3 grains of sodium bicarbonate to the ounce to combat the circulatory depression, as also to dilute the carbolic acid content of the blood and to encourage excretion by promoting a flow of urine. Use oxygen inhalation or carry on artificial respiration, if necessary.

Apply castor oil to the burns caused by carbolic acid on the skin after washing the surface with alcohol or soap and water.

Post-mortem Appearances.—White or brownish stains may be seen on the angles of the mouth and on the chin. The mucous membranes of the lips, mouth and throat are corrugated, sodden, whitened or ash-grey, and partially detached, marked by numerous small submucous hæmorrhages.

The mucous membrane of the œsophagus is tough, white or grey, corrugated and arranged in longitudinal folds.

The stomach is brown and leathery with hæmorrhagic spots and prominent rugæ, or it is, sometimes, soft and greyish-white in colour. It may contain a reddish fluid mixed with mucus and shreds of epithelium, emitting an odour of carbolic acid. The same changes are observed in the duodenum. The kidneys show hæmorrhagic nephritis in the cases of delayed death. The lungs are congested and œdematous. The brain is congested. The blood is dark and semifluid, or only partially coagulated.

Chemical Analysis.—Carbolic acid may be separated from organic matter by washing it with ether, decanting the ethereal liquid, and allowing the ether to evaporate. It may also be readily separated from organic matter by distillation with dilute sulphuric acid. The following tests may then be applied to the distillate :—

1. *Landolt's Test.*—Bromine water produces a whitish-yellow precipitate of tribromo-phenol which, when treated with an alcoholic solution of sodium amalgam, yields free carbolic acid.

2. *Millon's Test.*—Heated with Millon's reagent, a solution of carbolic acid produces a red colour. This test is very delicate, as it will give a red colour to a solution containing only 1 part of carbolic acid in 2,000,000 parts of water. It is, however, not characteristic of carbolic acid, as it produces the same reaction with many substances, especially monophenols and proteins.

Millon's reagent is prepared by dissolving 1 part of mercury in 1 part of strong nitric acid (sp. gr. 1.4) by diluting with twice its volume of water. The solution is allowed to stand overnight and a clear liquid is then decanted off for use as a reagent.

3. A few drops of a very dilute ferric chloride solution added to a solution of carbolic acid yields a purple or violet blue colour which disappears on the addition of mineral acids, but is not discharged by acetic acid (to be distinguished from salicylic acid).

4. Heated with ammonia and calcium hypochlorite an aqueous solution of carbolic acid yields a blue colouration, which becomes red or yellow on the addition of an acid. This is known as Lex's test.

5. *Libermann's Test.*—This is a very delicate and characteristic test, which is performed as follows :—

Dissolve a crystal or drop of carbolic acid in concentrated sulphuric acid, cool if necessary, then add a very small crystal of sodium nitrite, a deep blue or green colouration will be produced ; when poured on a beaker of water, it is turned red which is again turned green or blue on adding alkali.

Medico-Legal Points.—Carbolic acid is largely used as an antiseptic and as a disinfectant. It is used in the preparation of many disinfecting powders. For instance, Macdougall's disinfecting powder consists of crude carbolic acid and calcium sulphite. Calvert's carbolic acid powder is made by adding carbolic acid to the siliceous residue obtained from the manufacture of aluminic sulphate from shale.

Being easily procurable several cases of accidental and suicidal poisoning by carbolic acid have occurred. On account of its powerful odour and taste carbolic acid is very rarely used for homicidal purposes, though it has been, sometimes, used for the murder of infants. It has also caused death when used as an abortifacient by injection into the vagina or uterus. Edmunds[1] mentions a case in which a woman injected a drachm of carbolic acid to a quart of water into the uterus as a douche, and died in one hour and forty minutes.

Poisonous symptoms, followed by fatal results in some cases, have occurred from swallowing carbolic acid, from its application to a wound or an unabraded skin, from injection into an abscess cavity, rectum or uterus, as also from inhalation of its vapour.

Cases.—1. On October 20th, 1921, a man, aged 30 years, in robust health and sober habits, broke accidentally a bottle of crude carbolic acid he was carrying home in his trouser pocket, and became unconscious in twenty minutes. About an hour later he was removed to Whipps Cross Hospital, where he was found unconscious with stertorous breathing and extensive carbolic acid staining and burning of the left hip, left thigh and scrotum. His pupils were contracted. Later in the evening he became irritable and vomited. The next day he regained consciousness, but complained of severe abdominal pain, and passed blood in the urine. On the 22nd, he appeared better, but did not pass urine. and on the 23rd it was recognised that there was complete suppression of urine. He remained mentally clear till the 28th. At 6-20 p.m. there was a sudden change ; he felt cold and collapsed. The pulse went, and the extremities became cold and clammy. He died at 6-25 p.m. At the autopsy there was no erosion of the mucous membrane of the stomach. The liver and spleen were congested. and the kidneys showed acute hæmorrhagic nephritis.—*Turtle and Dolan, Lancet, Dec.* 16, 1922, *p.* 1273.

2. A youth employed at a chemists' shop dropped a winchester of crude carbolic acid. He immediately got a cloth and went down to mop up the fluid. Within a few minutes he fell to the ground unconscious and was at once dispatched to the hospital. Here he was examined in less than thirty minutes from the accident. He was absolutely comatose, cyanosed, stertorous, with a subnormal temperature and a thready, rapid pulse. The breath smelt strongly of carbolic acid, and the mouth and nose were covered with froth. He was cold, but not clammy, and generally livid. He was given intravenous saline injections to which two drachms of sodium bicarbonate per pint were added. The effect was certainly marked. The breathing assisted by the use of oxygen improved almost at once, and the patient recovered fully in two days. The urine showed the presence of carbolic acid.—*Smith, Lancet, Dec.* 23, 1923, *p.* 1359.

1. *Medical and Surgical Reporter,* 1887, 57, *p.* 345.

3. On September 18, 1930, a nurse had the misfortune to slip on a wet floor, upsetting in her fall a vessel containing a moderate quantity of "pure carbolic", *i.e.*, acidum carbolicum liquefactum. The drug was spilt over a considerable area of her clothing, and affected the skin of the face and neck, the whole length of both upper limbs, the chest and upper abdomen and small areas of the back and both lower limbs. The clothes were immediately removed and large quantities of methylated spirit were applied to the burns. In a very short time she was unable to sit up and her consciousness rapidly became clouded, and before three minutes from the time of the accident she was completely unconscious and became comatose. Her face and her upper and lower limbs were continuously twitching, her pupils were semi-dilated and fixed, her colour was greyish-blue and was visibly deepening, her respirations were laboured and bubbling, her mouth and nasal cavities were full of frothy mucus and no pulse could be felt at the cardiac apex or wrist. Thirty ounces of normal saline containing ninety grains of sodium bicarbonate were infused into a vein. In about two hours' time she was completely conscious and reasonable. During the following twenty-four hours she vomited incessantly and continued to do so during the three subsequent days. Her urine was at first green and contained albumin; it remained green for two days and abluminous for three days. It was never diminished in quantity and the microscopic examination did not show any casts. The burns which were superficial were treated by the tannic acid method.—*J. Taylor, Proceedings of the Royal Society of Medicine, Nov.*, 1930, *p.* 63.

Carbolic acid is excreted chiefly in the urine in combination with sulphuric and glycuronic acids and also as hydroquinone and pyrocatechin. It is also eliminated from the system by the lungs, salivary glands, skin, liver and stomach.

Carbolic acid may normally occur in traces in the urine in the form of phenolsulphonate of potassium, derived from the digestion of albuminous substances or of their putrefaction. From his experiment Engel[1] has estimated that the quantity of carbolic acid excreted by a healthy man living on mixed diet is 15 milligrammes in twenty-four hours.

Cresol, Creolin (a constituent of Jeye's disinfecting fluid), lysol and izal are all similar in action to carbolic acid, but they are believed to be less toxic. Stapelmohr[2] reports a case in which the terminal phalanx of the thumb sloughed off, and had to be amputated after application of a 5 per cent dilution of a compound solution of cresol.

Smith[3] describes the case of a man, aged 32, who, with intent to commit suicide, swallowed 2 ounces of a weed-killer of emulsified tar acids consisting of 35 per cent orthocresol, 40 per cent metacresol and 25 per cent paracresol. He had no pain or vomiting, but became comatose in about an hour and died in about one hour and forty minutes. The necropsy did not reveal any signs of corrosion of the lips, tongue, mouth, pharynx, œsophagus, stomach or intestines. In a case[4] where a Hindu male, 17 years old, died after swallowing a quantity of lysol, the post-mortem examination showed that the mouth, pharynx and œsophagus were ulcerated. The tongue was white and the stomach was perforated. The liver and kidneys were congested.

Death has occurred after swallowing one to two tea-spoonfuls of lysol. On the other hand, recovery has followed much larger doses. A woman, 25 years old, took about two ounces of a preparation labelled "Lysol pure" at 7-18 p.m. After swallowing she felt no pain, but only a slight burning in the throat, and then she went off to sleep. At 7-45 p.m. she was comatose, the pupils were contracted, the face was cyanosed, and the lips and skin of the face were burnt as though by some

1. *Annal. de. Chimie et de Physique*, 5 *ser., and XX*, *p.* 230, 1880; *Blyth, Poisons, their Effects and Detection, Ed. V*, *p.* 188.
2. *Hygeia Stockholm, May* 16, 1917, *p.* 438; *Jour. Amer. Med. Assoc., July* 21, 1917, *p.* 248.
3. *Brit. Med. Jour., April* 28, 1928. *p.* 714.
4. *Punjab Chem. Examiner's Annual Report*, 1934, *p.* 10.

corrosive fluid. The breathing was rattling and stertorous, and the breath smelt strongly of carbolic acid. There was foam on the lips, mouth and nose, but the pulse was fairly good. The urine passed was very dark and smoky, but did not contain albumin or blood. The stomach was washed out with warm water, and a pint of warm water containing an ounce of magnesium sulphate was left inside it. Brandy was given per rectum, and oxygen inhalation was administered. The patient recovered in a couple of days.[1] Several cases of suicide by lysol occur every year, chiefly in Germany. In February, 1923, a case occurred at Bangalore, where a woman was charged with attempting to commit suicide on Christmas Eve by drinking lysol. She was found guilty, and fined fifty rupees.

Accidental cases of poisoning have, sometimes, occurred. A European boy, 14 years old, died in four hours and forty-five minutes after he had taken an enema of one and a half ounces of lysol in a pint of water.[2] Shore[3] describes a case of accidental poisoning by absorption of lysol through the unbroken skin. A seaman purchased a bottle of lysol, and put it into his hip-pocket. Afterwards he met with an accident, as a result of which the bottle was broken, and the lysol saturated his clothes, so that he was burned from the hip to the heel on that side; there was a certain amount of burning on the other leg as well. The man died in about three-quarters of an hour. The post-mortem examination showed that the kidneys were red and enlarged.

CREOSOTE

This is obtained from wood tar by destructive distillation, and consists chiefly of a mixture of guaiacol, creosol, and other phenols. It is an oily liquid, having a burning taste and a very strong peculiar odour. It is colourless when fresh, but brown on exposure to light. It is slightly soluble in water, but freely in alcohol, ether and glacial acetic acid. It is an official preparation, the dose being 2 to 10 minims.

Creosote is used externally as an application in toothache, and internally in tuberculosis. Poisoning has, therefore, occurred from such uses although toleration for it is established by gradually increasing the dose. It acts much in the same way as carbolic acid, but it does not impart a dark colour to the urine.

Symptoms.—Local corrosive action on the mucous membranes of the lips, tongue and mouth, burning pain in the stomach, nausea, vomiting, diarrhœa, cyanotic lips, contracted pupils, coma and stertorous breathing. Convulsions may occur in infants. It is eliminated by the kidneys, and its odour may be perceived in the urine.

In a case[4] in which an infant, two months old, was given a gramme of creosote by mistake for a laxative, there was little indication of a local caustic action, only a little vomiting at first and no bowel symptoms at any time. Hæmolytic jaundice, hæmoglobinuria and leucocytosis were the main symptoms. Death occurred in sixty hours.

Fatal Dose and Fatal Period.—Two drachms killed an aged woman in thirty-six hours.[5] Twenty-four to thirty drops killed an infant, ten days old, in sixteen hours.[6] A woman, 52 years old, died in five days after taking three six-drop doses of creosote in milk.[7] Recovery has, however, occurred after one ounce of creosote.[8]

Treatment.—This is the same as that for carbolic acid poisoning.

1. *Semple, Brit. Med. Jour., Oct.* 24, 1925, *p.* 774.
2. *William Hartigan, Brit. Med. Jour., Nov.* 24, 1900, *p.* 1498.
3. *Trans. Med.-Leg. Society, Vol. VII, p.* 92.
4. *I. Thorling, Upsala Lakare forenings Forhandlingar, Upsala, Sept.* 1, 1921, 26, *No.* 5-6; *Jour. Amer. Med. Assoc., Nov.* 12, 1921, *p.* 1614.
5. *Pereira, Mat. Med.*, 1842.
6. *Purchhauer, Freidreich's Blatter f. ger. Med.*, 1886; *Dixonmann, Forens. Med. and Toxic., Ed. VI, p.* 484.
7. *Zawadzki, Centralbl. f. innere Med.*, 1894, *XV, p.* 401; *Dixonmann, Ibid.*
8. *Schultze, Munch. Med. Wchnschr*, 1894, *XLI, p.* 219; *Witthaus, Manual of Toxic.*, 1911, *p.* 1194.

Post-mortem Appearances.—The mucous membranes of the lips, tongue, mouth, œsophagus and stomach are grey or red in colour, inflamed and eroded in patches. The brain and lungs are congested. The kidneys are usually congested, but they may be inflamed. In the abovementioned case where death took place in five days two large erosions were found in the upper part of the œsophagus and others near the pylorus. The stomach was red and injected, and the kidneys were acutely inflamed.

In a case[1] where a woman died of creosote poisoning, the post-mortem appearances were almost negative. No excoriations were found on the mouth, lips or œsophagus, the stomach was uniformly redder than normal, and parts were slightly hæmorrhagic. There was no sign of ulcer or of gross hæmorrhage. The intestines were normal. The liver, spleen, and kidneys were normal. The brain was hyperæmic and softer than usual but there was no lesion and no suspicion of meningitis. The heart was normal, and the lungs were hyperæmic. The odour of creosote was present in the brain as well as in the stomach contents.

Chemical Test.—Ferric chloride gives a dirty green or brown colour, discharged by water.

Pyrogallic Acid (Pyrogallol), $C_6H_3(OH)_3$.—This is a white, odourless, crystalline powder, which melts at 132° C., and is very soluble in water. In alkaline solution it rapidly absorbs oxygen and darkens in colour. It is used as a reducing agent in the dye and photographic industries.

Pyrogallic acid acts as a poison when it is swallowed or when it is applied to the skin. When absorbed into the system, it destroys the red blood corpuscles, and forms methæmoglobin in the blood. The chief symptoms are dyspnœa, vomiting, diarrhœa, dark coloured urine containing hæmoglobin and methæmoglobin, low temperature, paralysis, collapse and death.

Treatment.—This consists in the washing out of the stomach, administration of stimulants and oxygen by inhalation and maintenance of external warmth.

Chemical Tests.—Pyrogallic acid produces a red colour with ferric chloride and a bluish-black colour with ferrous sulphate.

PICRIC ACID (CARBAZOTIC ACID, TRINITRO-PHENOL, TRINITROPHEN), $C_6H_2(NO_2)_3OH$

This is obtained by the action of nitric and sulphiruc acids on phenol. It exists as yellow crystalline prisms or plates, and explodes under the action of heat or percussion. It is soluble in about 90 parts of water, and in 10 parts of alcohol. It has no odour, but has an intensely bitter taste, and consequently has been used as a substitute for hops in beer. It is used as a yellow dye for silk and wool. It has produced toxic effects, when swallowed in the form of a solution, applied externally and also when inhaled in the form of dust.[2] Cheron relates a case of poisoning which occurred from the local application of about six grains of the acid to the vagina.[3] It is now largely used as a dressing for burns, and one death[4] has been recorded from poisonous effects thus produced. Death has also occurred from the inhalation of nitrous acid fumes and volatilized picric acid.[5]

Picric acid is, sometimes, used by malingerers to simulate icterus and to escape military service.[6]

Picric acid precipitates albumin, and causes local necrosis. It decomposes the red blood corpuscles, and produces methæmoglobin. It also irritates the central nervous system, causing convulsions.

1. *Jour. of State Medicine, Vol. XXXI, No.* 1, *Jan.*, 1930, *p.* 41.
2. *Cheron, Jour. de Therap.*, 1880, 7, *p.* 132.
3. *Ibid.*
4. *Alexander, Med. Press and Cir.*, 1912, *p.* 112.
5. *Lancet, Aug.* 15, 1891.
6. *Mende, Deut. Med. Woch.*, 1918, 44, *p.* 1440; *Peterson, Haines and Webster, Leg. Med. and Toxic., Ed. III, Vol. II, p.* 719.

Symptoms.—Pain in the stomach; severe vomiting of yellow matter; diarrhœa with yellow stools; the conjunctivæ and the skin assume a bright yellow colour, which is known as "picric jaundice"; the pupils are dilated, there may be itching and eczema; the urine is at first dark yellow in colour, and later becomes ruby red, owing to the formation of picramic acid, but it does not contain bile or albumin; there may be anuria and strangury; rapid pulse; muscular cramps; convulsions; drowsiness; delirium; stupor and collapse.

Picric acid is eliminated in the urine, though the elimination is slow. In one case its presence was detected in the urine for six days after the administration of a single dose of one gramme of picric acid.[1] It is also eliminated in the fæces.

Fatal Dose and Fatal Period.—The pharmacopœial dose of picric acid is 1 to 5 grains. The fatal dose is uncertain. Poisoning has followed thirty grains, but recovery has ensued after swallowing about 300 grains.[2]

Treatment.—Wash out the stomach. Give diuretics and purgatives. Administer morphine to relieve pain. The antidotes are proteins as found in raw eggs and milk. The administration of large doses of dextrose has been recommended as this substance was believed to aid the reduction of picric acid to the less poisonous picramic acid.[3]

Post-mortem Appearances.—All the viscera are stained yellow.

Chronic Poisoning.—Men who handle picric acid in munition plants and get dusted over with it suffer from dermatitis which may be extremely irritating. Workman engaged in the manufacture of the explosive, melinite, which chiefly consists of picric acid, suffer from a form of chronic poisoning, the chief symptoms being abdominal cramps, vomiting, diarrhœa, loss of appetite and loss of weight.[4]

A case[5] is recorded in which a youth, aged 17, was unpacking "Explosive D," ammonium picrate, which covered his face and hands and got into his eyes. He suffered from conjunctivitis and tubular nephritis with heavy albumin and casts in urine.

Chemical Tests.—An aqueous solution is intensely yellow, and is acid to litmus. Ammonio-sulphate of copper produces a green precipitate.

When an aqueous solution of picric acid is warmed with potassium cyanide, a blood-red colouration is produced owing to the formation of potassium isopurpurate.

SALICYLIC ACID, $HC_7 H_5 O_3$

This is prepared by the interaction of sodium carbolate (phenate) and carbon-dioxide. It may also be obtained from natural salicylates contained in *gaultheria* and sweet *birch*. It is an odourless crystalline solid, sweetish and acrid in taste, sparingly soluble in cold water (1 in 500), but readily in hot water, alcohol, ether and chloroform. The official dose is 5 to 10 grains.

Accidental cases of poisoning occur from an over-dose when given in medicine, and from its widespread use in preserving food and liquors. In his annual report of 1910 Dr. Choonilal Bose, Chemical Examiner of Bengal, mentions a fatal case of poisoning by salicylic acid which occurred in Cooch Behar. A patient was administered an ounce and a half of sodium salicylate (dose 10 to 30 grains) in mistake for sodium sulphate. He suffered from symptoms of irritant poisoning.[6]

Symptoms.—These are burning pain in the throat and stomach, difficulty of swallowing, thirst, nausea, vomiting, diarrhœa, headache, noises in the ears, flushing

1. *Warren, Autenrieth's Detection of Poisons, Ed. VI, p.* 120.
2. *Adler, Weiner, Med. Woch.*, 1880, 30, *p.* 819; *Dixonmann, Forens. Med. and Toxic., Ed. VI, p.* 483.
3. *Rymsza, Ein Beitrag z. Toxicol. der Pikrinsaure. Dorpat*, 1889; *Peterson, Haines and Webster, Leg. Med. and Toxic., Vol. II, Ed. III, p.* 720.
4. *Alice Hamilton, Jour. Amer. Med. Assoc., May* 19, 1917, *p.* 1445.
5. *Jour. Amer. Med. Assoc., Oct.* 19, 1929, *p.* 1243.
6. *Deutsche Med. Woch.*, 1881; *Dixonmann, Forens. Med. and Toxic., Ed. VI, p.* 478.

of the face, profuse perspiration, cold, moist skin, slow, weak, and irregular pulse, confused mind, delirium, insensibility and coma. Hæmorrhages occur from the mucous membranes, *e.g.*, epistaxis, bleeding from the gums, retinal hæmorrhages causing amblyopia, and bleeding from the kidneys giving rise to hæmaturia. There may be bleeding from the uterus, leading to abortion. Death occurs from the stoppage of the heart or respiration.

Salicylic acid is eliminated chiefly by the kidneys as salicyluric acid. Its elimination in the urine begins within fifteen minutes of its administration by the mouth and ends as a rule within forty-eight hours. It is also excreted in perspiration, bile and milk.

Chronic Poisoning.—This occurs when salicylic acid and its salts are administered for a prolonged period or when articles of diet preserved by salicylic acid are taken daily for a long time. The chief symptoms are loss of appetite, impaired digestion, diarrhœa alternating with constipation, eczematous eruptions on the skin and mental depression. The urine may be albuminous.

Fatal Dose and Fatal Period.—One ounce of salicylic acid has caused death after four days.[1] A less quantity would prove fatal if the heart or kidneys were diseased. An infant died from the application of a ten per cent ointment of salicylic acid to the head and neck.[2] Thirty-four grammes[3] of sodium salicylate proved fatal to a patient, 17 years old, and 100 grammes[4] of the same drug caused the death of a child, 5 years old.

Treatment.—Emetics, lavage, sodium bicarbonate, magnesium oxide, raw eggs, milk, warmth and stimulants.

Post-mortem Appearances.—Signs of gastritis, enteritis and nephritis may be found. The organs are usually found hyperæmic. The internal organs were found congested in the case of a European, aged about 45 years, who died from salicylic acid poisoning.[5]

Tests.—Ferric chloride gives a violet colour, which disappears on the addition of acetic acid, but the colour yielded by carbolic acid to ferric chloride is permanent.

Methyl Salicylate.—This is also known as artificial oil of wintergreen, and is obtained by the interaction of methyl alcohol and salicylic acid. It is a colourless liquid, having a characteristic, aromatic odour and a sweetish, warm, aromatic taste. It is slightly soluble in water, and freely soluble in glacial acetic acid and in carbon bisulphide. The medicinal dose is 5 to 15 minims.

Methyl salicylate has caused symptoms of poisoning resembling those of poisoning by salicylic acid, followed occasionally by death. A woman took 60 grammes of methyl salicylate with the intention of committing suicide. Her stomach was immediately washed out, and she was also given an emetic. In two hours she suffered from nausea, vomiting and diarrhœa. Later on, she developed convulsions and cyanosis of the extremities with cold perspiration. She became semi-comatose and died in 17 hours and 15 minutes.[6] A woman died in 15 hours after she had taken about an ounce of methyl salicylate to procure abortion.[7] Doses of 10 c.c. to 12 c.c. of methyl salicylate have proved fatal to children.[8] On the other hand, a child, aged 2, recovered after swallowing an ounce of methyl salicylate from a bottle. Olive oil was administered soon after the poison was taken, and sodium bicarbonate was afterwards given by mouth and rectum. Artificial respiration was also performed.[9]

1. *Holland, Med. Chemistry and Toxic., Ed. IV, p.* 473.
2. *Zumbroich, Monateschr. f. Kinderh.*, 1918, 15, *p.* 167; *Peterson, Haines and Webster, Leg. Med. and Toxic., Vol. II, Ed. III, p.* 716.
3. *Quincke, Berl. Klin. Wchmschr.*, 1882, *XIX, p.* 709; *Ralph Webster, Leg. Med. and Toxic.*, 1930, *p.* 811.
4. *Langmead, Lancet, Vol. I*, 1906, *p.* 1822.
5. *Bengal Chem. Exam. Rep.*, 1922, *p.* 7.
6. *Legrain and Mlle. Badonnel, Jour. Amer. Med. Assoc., April* 15, 1922, *p.* 1140.
7. *Pinkham, Boston, Med. and Surg. Jour., Vol.* 117, *p.* 548.
8. *Meyerhoff, Jour. Amer. Med. Assoc., May* 31, 1930, *p.* 1751.
9. *Myers, Jour. Amer. Med. Assoc., Dec.* 25, 1920, *p.* 1783.

Acetylsalicylic Acid (Aspirin).—This is obtained by the action of acetic anhydride or acetyl chloride on salicylic acid. It occurs as a white, inodorous, crystalline powder, having a slightly acid taste. It is sparingly soluble in water, but dissolves in 5 parts of alcohol and freely in ether. It is a pharmacopœial preparation, the dose being 5 to 15 grains. Its action is antipyretic and analgesic.

Cases of poisoning by aspirin have occurred from its use as a medicine or from its use as a suicidal poison, especially in Hungary. Poisoning occurs in sensitive people after taking medicinal doses of aspirin.

Symptoms.—These are headache, dizziness, buzzing in the ears, thirst, nausea, vomiting, red and swollen face, weak and rapid pulse, quick breathing, and general perspiration. In severe cases there are clinical signs of acidosis with Cheyne-Stokes respiration. Death occurs from heart failure.

Fatal Dose and Fatal Period.—Four hundred and fifty to six hundred grains[1] of aspirin is the minimum fatal dose, although five[2] and ten[3] grains have caused an enormous swelling of the face, especially the eyelids, lips, nose and tongue, while two hundred[4] and three hundred[5] grains have proved fatal. A Mahomedan woman, aged 18 years, recovered after she had swallowed about an ounce of aspirin in mistake for magnesium sulphate. A man, aged 48, recovered after he had taken 120 five-grain tablets (600 grains) of aspirin with a view to commit suicide.[6] A rare case is reported in which a woman, aged 45 years, died in about ten minutes after a dose of five grains.[7] A man, aged 72, died in twelve hours after a dose of 150 five-grain tablets.[8] A man, 50 years old, died in about twelve hours after he had taken one thousand grains for the purpose of committing suicide.[9]

Treatment.—This consists in the washing out of the stomach and the administration of saline purgatives. Intravenous injection of a 4 per cent solution of sodium bicarbonate is very beneficial. This may also be administered by mouth or by rectum after vomiting has stopped. Large quantities of water may be given to hasten the elimination of the poison. Cardiac stimulants may be administered if necessary.

Post-mortem Appearances.—These are not characteristic, but there may be hæmorrhages in the viscera.

Chemical Analysis.—Aspirin can be easily extracted with water. The aqueous solution is shaken out with ether, and the other extract is then evaporated. The residue contains aspirin. If the residue is heated wih sodium or potassium hydroxide or even with water, aspirin is hydrolyzed into salicylic acid and acetic acid.

ACETIC ACID CH_3 COOH

This acid occurs in nature in combination with alcohols in the essences of many plants, and is formed during the decay of certain organic substances. It is prepared on a large scale from pyroligneous acid obtained in the distillation of wood. It is a clear, colourless acid liquid, having a pungent odour.

It acts as a corrosive poison in the concentrated form, known as glacial acetic acid, but acts merely as an irritant poison when diluted. Vinegar (*Sirka*), which contains four to five per cent of acetic acid may cause poisonous symptoms when taken in large quantities. Vinegar and acetic acid contain traces of sulphuric acid as an impurity. Acidum aceticum dilutum is a pharmacopœial preparation, the dose being 30 to 60 minims. It contains 6 per cent of absolute acetic acid.

1. *Balazs, Medizinische Klinik, Berlin, Nov.* 7, 1930, *p.* 1664 : *Jour. Amer. Med. Assoc., Feb.* 7, 1931, *p.* 477.
2. *Morgan, Brit. Med. Jour., Feb.* 11, 1911, *p.* 307.
3. *Killen, Brit. Med. Jour., Feb.* 25, 1911, *p.* 476.
4. *Neale, Brit. Med. Jour., Jan.* 18. 1936, *p.* 110.
5. *Balazs, Loc. Cit.*
6. *Lipetz, Brit. Med. Jour., April* 7, 1934, *p.* 652.
7. *Dysart, Jour. Amer. Med. Assoc., Aug.* 5, 1933, *p.* 446.
8. *Neale, Brit. Med. Jour., Jan.* 18. 1936, *p.* 110.
9. *Ibid.*

Symptoms.—The mucous membrane of the mouth, tongue and other parts of the body, with which the acid comes in contact, are softened and present the appearance of a yellowish-white colour. There is intense pain extending from the mouth to the stomach. The other symptoms are vomiting, difficulty in swallowing, convulsions, irritable cough and collapse. The symptoms of suffocation are usually more marked, as the acid being volatile affects the larynx and lungs during the act of swallowing. According to Sklodawski[1] hæmoglobinuria is a constant feature in this poisoning. It appears within the first twelve hours, and is evident even in the benign form. This sign may be helpful in differential diagnosis from other poisons.

Fatal Dose.—One drachm of the concentrated acid has caused the death of a child, but recovery has been recorded in an adult after taking six fluid ounces.[2]

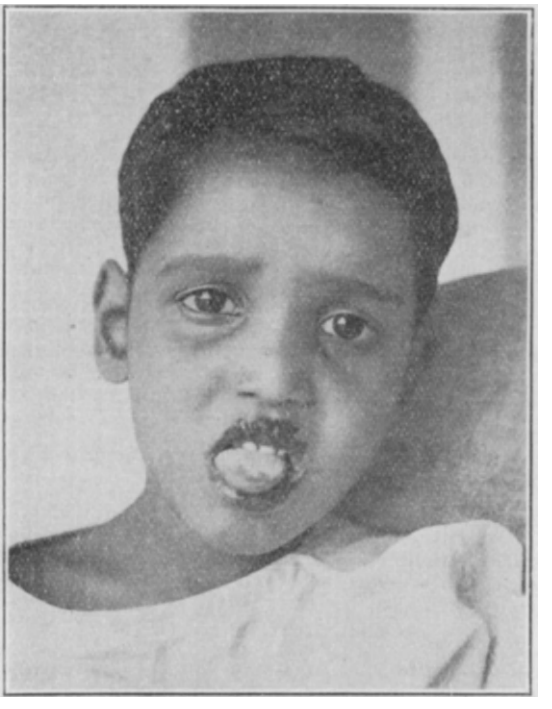


Fig. 128.—Acetic Acid Poisoning: Stains on the lips and tongue caused by glacial acetic acid.

Fatal Period.—Rapid though variable. Death occurred from shock in two hours after the ingestion of 60 to 70 c.c. of glacial acetic acid.[3] A child, aged 2 years, died in about thirty-six hours after a dose of glacial acetic acid administered by mistake.[4]

Treatment.—Neutralise the acid by giving magnesia, and then produce emesis by giving emetics. Give demulcents, and allay pain by hypodermic injections of morphine. Laryngeal symptoms may be treated by the application of cold compresses to the throat and by giving the patient pieces of ice to suck. Tracheotomy may be performed if necessary.

1. *Presse Medicale, Paris, Nov.* 28, 1925, *p.* 1573; *Jour. Amer. Med. Assoc., Jan.* 9, 1926, *p.* 156.
2. *Dixonmann, Forens. Med. and Toxic., Ed. VI, p.* 362.
3. *Thomas A. Gonzales, Morgan Vance and Milton Helpern, Leg. Med. and Toxicology, p.* 477.
4. *Taylor, On Poisons, Ed. III, p.* 242.

Post-mortem Appearances.—Erosion or corrosion of the mucous membrane of the mouth, œsophagus, stomach and intestines with ecchymosed patches.

Chemical Analysis.—Acetic acid may be separated from organic mixtures by distillation. If combined it should be liberated by adding phosphoric acid.

Tests.—It is recognised by its characteristic odour. When heated with alcohol and sulphuric acid acetic ether (ethylacetate) is formed, which is known by its peculiar aromatic smell.

Ferric chloride added to its solution, after it is neutralised with ammonia, produces a deep red colour which, when boiled, changes to a red-brown precipitate of ferric subacetate.

TARTARIC ACID, $C_4\ H_6\ O_6$

This acid is a constituent of a large number of plants, and occurs in many fruits, especially grapes. It may be prepared from potassium acid tartrate. It occurs as colourless crystals or as a white powder, is odourless and strongly acid in taste. It is soluble in less than 1 part of water, in about 2.5 parts of alcohol and slightly soluble in ether. It is a pharmacopœial preparation, the dose being 5 to 30 grains. It is also a constituent of Seidlitz powder (Pulvis effervescens compositus).

Ordinarily this acid is not regarded as a poison, but in large doses it may act as a poison. A few severe and fatal cases of poisoning by it have been recorded.

Symptoms.—These are more of a strongly irritant nature than corrosive. There is a burning sensation in the throat and stomach, followed by vomiting and diarrhœa. Death may occur from exhaustion.

Fatal Dose and Fatal Period.—Trevithick[1] records the case of a woman, aged 67 years, who swallowed a strong solution containing at least 140 to 180 grains of tartaric acid and died on the seventh day. Gill[2] reports the case of a man of 24 years who was given an ounce of tartaric acid in mistake for aperient salts and died on the ninth day. A man[3] in Delhi was given 4 *tolas* (720 grains) of tartaric acid in place of some "salts" for his constipation from an Indian medicine shop. After taking it with a bottle of soda he was seized with vomiting and purging containing blood. He eventually recovered. Four ounces[4] of acid tartrate of potash (cream of tartar) caused death in 48 hours, and about six ounces and a half caused death in twelve hours.[5]

Treatment.—Neutralise the acid by giving soda and magnesia. Give astringents and opium.

Post-mortem Appearances.—Erosions of the mucous membrane of the œsophagus, and inflammation of the greater part of the alimentary canal. According to Tardieu the blood remains persistently fluid and acquires the colour of red currant juice.

Chemical Analysis.—Tartaric acid forms large transparent crystals, and is readily soluble in water and alcohol, but with difficulty in ether. Calcium chloride yields a white precipitate soluble in acetic acid (Distinction from oxalic acid). Boiling darkens tartrates, and potassium permanganate decolourises them.

Silver Mirror Test.—With a neutral solution silver nitrate produces a white precipitate of silver tartrate, soluble in excess of the tartrate, also in nitric acid and ammonium hydroxide. Take the original precipitate of silver tartrate in a test tube (which has been cleaned carefully with caustic soda and distilled water) and add to it ammonium hydroxide until it is almost (but not quite) dissolved. Then drop in a crystal of silver nitrate to the bottom of the test-tube and allow it to stand in a

1. *Brit. Med. Jour.*, 1893, *Vol. I, p.* 1321.
2. *Lancet*, 1845, *Vol. I, p.* 18 ; *R. v. Watkins, C.C.C., Jan.*, 1845.
3. *Punjab Chemical Examiner's Annual Rep.*, 1926.
4. *Lancet, Oct.* 28, 1837.
5. *Roger, Friedreich's Bl. f. ger., Med.*, 1887, *XXXVIII*, 196 ; *Witthaus, Med. Juris. and Toxic., Vol. IV, p.* 843.

beaker of boiling water; in a short time, owing to the reduction of the silver salt, a beautiful mirror of metallic silver forms on the sides of the tube.[1]

CITRIC ACID, $C_6H_8O_7$

This acid is found free in the juice of the lemon, orange and many other sour fruits, and is stated to occur to the extent of from 0.05 to 0.1 per cent in human and cow's milk. It is prepared by boiling lemon juice and neutralising with calcium carbonate. It occurs as large, colourless prismatic crystals or as a white powder. It is odourless and strongly acid in taste. It dissolves in less than 1 part of water, in about 1.5 parts of alcohol and is slightly soluble in ether. The pharmacopœial dose is 5 to 30 grains.

As shown by experiments on animals, citric acid is more poisonous than tartaric acid. Fatal cases[2] of poisoning by this acid have occurred. A young girl[3] died after she had taken 25 grammes of citric acid as an abortificient. The treatment is the same as in poisoning by tartaric acid.

Tests.—Calcium chloride yields a white precipitate on boiling but not in the cold. Boiling has no effect on citrates, but potassium permanganate turns them green. It gives no mirror test with silver nitrate.

III. ALKALIES

Like acids, alkalies act as corrosive poisons when administered in a concentrated form, but act as irritant poisons when dilute.

The hydroxides or hydrates and carbonates of alkalies which act as corrosives are the following :—

1. **Ammonia (Hartshorn)**, NH_3—Gaseous ammonia, when dissolved in water, forms a strong solution of ammonia (Liquor Ammoniæ Fortis), known as spirits of Hartshorn. It is a colourless liquid, having a very pungent characteristic odour, and a strong alkaline reaction. The solution is largely employed for domestic purposes, such as removing paint, oil, and dirt generally from clothing. When freshly prepared the gas is freely given off and serious poisonous symptoms have occurred from its inhalation when large ammonia jars or ammonia refrigerators in factories have burst. The phamacopœial preparation, liquor ammoniæ dilutus (liquor ammoniæ) is an aqueous solution containing 10 parts of ammonia by weight. The dose is 10 to 20 minims.

2. **Postassium Hydroxide (Potassium Hydrate, Caustic Potash)**, KHO.—This is usually met with as hard, deliquescent, white pencils or cakes. It is soapy to the touch, acrid to the taste, rapidly absorbs carbon dioxide from the air, and is very soluble in water. Its solution is known as liquor potassæ (liquor potassii hydroxide), which has also a soapy feel, and a strong alkaline reaction, and can be given internally in 10 to 30 minim doses, freely diluted.

3. **Sodium Hydroxide (Sodium Hydrate, Caustic Soda)**, NaHO.—This occurs as white, solid masses or as cylindrical sticks, closely resembling potassium hydroxide. It is strongly caustic and when dissolved in

1. *Perkin, Quantitative Chem. Analysis,* 1919, *p.* 218.
2. *Witthaus, Med. Juris. and Toxic., Vol. IV, p.* 843.
3. *Zangger quoted in Erich Leschke. Clinical Toxicology. Eng. Trans., by Stewart and Dorrer,* 1934, *p.* 270.

water forms a solution known as liquor sodiæ. It is largely employed in manufactures, but cases of poisoning are rarely met with.

4. **Ammonium Carbonate (Sal Volatile),** $(NH_4)_2\ CO_3$.—This occurs as translucent, hard crystalline masses. It has a strongly ammoniacal odour and a pungent, ammoniacal taste. It is soluble in 4 parts of water. Exposed to air it partially dissociates, and becomes converted into porous lumps or a white powder. The pharmacopœial dose is 5 to 10 grains as a stimulant or expectorant and 30 grains as an emetic. Commercial ammonium carbonate is a mixture of hydrogen ammonium carbonate and ammonium carbonate, and possesses a strongly ammoniacal odour.

5. **Potassium Carbonate (Pearl Ash,** *Javakhar*), $K_2\ CO_3$.—This salt occurs as a white, crystalline powder, having a caustic and alkaline taste, the dose being 2 to 5 grains. It is highly deliquescent, and very soluble in water but insoluble in alcohol. It is used for washing and other cleansing purposes.

6. **Sodium Carbonate (Soda, Washing Soda,** *Sajjikhara*), $Na_2\ CO_3$.—This occurs as large, transparent, monosymmetric crystals. When exposed to the air the crystals soon effloresce, and become white on the surface. They are soluble in water but insoluble in alcohol. The dose is 5 to 15 grains. Exsiccated sodium carbonate (Sodii carbonas exsiccatus) is obtained by the action of heat on sodium carbonate. It occurs as a dry, inodorous, white powder, with a strongly alkaline taste, and dissolves readily in water. The dose is 2 to 5 grains. The impure combined carbonates of sodium and potassium are sold in the *bazaar* as *papad khara.*

A mixture of caustic soda and sodium carbonate, known as soap lye, is used for washing purposes. Casper[1] reports two cases of poisoning by soap lye. In one case, an unmarried woman attempted to murder her child, aged three years, and in the other an old man accidentally swallowed six to eight ounces of this instead of beer and died on the fifth day.

Symptoms.—The usual symptoms of corrosive poisons are present, with the following exceptions :—

1. The taste is acrid and soapy.

2. The vomited matter is strongly alkaline, and does not effervesce on coming in contact with the earth. It is at first thick and slimy, and later contains dark altered blood, and shreds of the mucous membrane from the gullet and stomach.

3. Purging, which is rare in poisoning by corrosive acids, is a frequent symptom, accompanied with severe pain and straining. The motions consist of stringy mucus mixed with blood.

It should be noted here that the sense of heat and burning pain in the throat and stomach are much greater when a strong solution of ammonia is swallowed than when a solution of caustic soda or potash is taken.

The ammoniacal vapour is very irritating to the respiratory organs. When inhaled, it produces congestion and watering of the eyes, running of

1. *Forens. Med., Eng. Trans., Vol. II, pp.* 111, 112.

the nose, and a feeling of suffocation with a sense of great heat in the throat. Death may occur immediately from suffocation due to inflammation of the glottis, or later from pneumonia or broncho-pneumonia. A Bengali druggist of Agra was seriously affected by the gas escaping from a suddenly opened bottle containing a strong solution of ammonia, and suffered from conjuctivitis, corneal ulcers and irido-cyclitis and had almost lost his vision.

Fatal Dose.—The average fatal dose of ammonia, caustic potash or caustic soda is half an ounce. The smallest fatal dose of liquor ammoniæ fortis is one fluid drachm, and that of caustic potash is forty grains. A dose of 8.5 grammes of caustic soda taken with a view to commit suicide killed a Turkish woman, aged 20 years, in eleven days, and a dose of 60 grammes killed another woman, 35 years old, in twenty-nine hours and thirty minutes.[1] Half an ounce of carbonate of potassium is regarded as a fatal dose. The fatal dose of carbonate of sodium is not certain. It is much less poisonous than potassium carbonate. Recovery has occurred even after a dose of twelve ounces of sodium carbonate.[2]

Fatal Period.—Usually within twenty-four hours. Inhalation of ammonia vapour has caused death in four minutes,[3] and three ounces of a strong solution of potassium carbonate taken internally killed a boy in three hours.[4] Death may occur after weeks or months, or even after two or three years from inaniation and starvation due to the œsophageal or pyloric stricture.

Treatment.—Neutralise the poison by acids, chiefly vegetable, *viz.*, acetic (vinegar), citric (lemon juice) or tartaric acid mixed with a large quantity of water. These should be followed by olive oil, white of egg, milk, butter and acidulated demulcent drinks. Pieces of ice should be given to suck. Cold should be applied to the abdomen. Morphine may be given hypodermically to relieve pain, and ether to counteract the effects of collapse.

The œsophageal stricture should be dilated by means of a bougie, or it may be necessary to perform œsophagostomy or gastrostomy.

In poisoning by ammonia vapour give oxygen inhalation, or keep the patient in an atmosphere rendered moist with steam. Anodynes may be given for pain.

Post-mortem Appearances.—These indicate marks of corrosion, but not so well-marked as in poisoning by the mineral acids. The mucous membrane of the mouth, throat, gullet, stomach and duodenum is softened, exfoliated and inflamed in patches of chocolate or black colour. The contents of the stomach are turbid, usually blood-stained, but frequently coffee-coloured. Perforation of the stomach is rare, but may occur in ammonia poisoning. The deeper tissues are inflamed and congested.

The mucous membrane of the larynx and trachea shows the same appearances as are found in the mouth, throat, etc. In protracted cases of

1. *Willimot and Gosden, Brit. Med. Jour., June* 9, 1934, *p.* 1022.
2. *Med. T. and Gaz., Aug.* 13, 1834.
3. *Christison, "Poisons," p.* 194.
4. *Taylor, On Poisons, Ed. III, p.* 251.

poisoning stenosis is found more often at the lower end of the œsophagus than at the pylorus.

In the case[1] of a man who died from poisoning by a solution of ammonia, the viscera were found in a highly congested state, including the œsophagus, the lungs and the pancreas, which latter was adherent to the duodenum, and the contents of the stomach smelled strongly of ammonia and had a soapy feel. The Chemical Analyser detected both free and combined ammonia in the viscera.

Chemical Analysis.—The contents of the stomach are alkaline in reaction and soapy to the feel. Ammonia may be separated from organic mixtures by distillation, and other alkalies may be separated by dialysis or by incinerating them in a porcelain capsule to drive off animal and vegetable matter. The residual ash is then dissolved in acidulated water, and tested for the presence of sodium and potassium as given in the following table :—

Reagents.	Ammonium.	Potassium.	Sodium.
1. Caustic potash and heat.	Ammonia gas is given off known by its odour, by its turning red litmus paper blue and by giving rise to white fumes of ammonium chloride when a glass rod wet with hydrochloric acid is brought into contact with it.	Nil.	Nil.
2. Nessler's reagent.	Brown precipitate.	Nil.	Nil.
3. Tartaric acid (strong) and alcohol.	Nil.	White crystalline precipitate.	Nil.
4. Platinic chloride.	Nil.	Yellow crystalline precipitate.	Nil.
5. Flame test.	Nil.	Violet.	Yellow.

The caustic alkalies give a brown precipitate with silver nitrate ; while their carbonates give a whitish-yellow precipitate and effervesce on the addition of an acid.

Ammonia is formed during putrefaction. Hence its detection is of no consequence unless analysis is undertaken immediately after death when the body is still fresh.

Medico-Legal Points.—Poisoning by alkalies is much less frequent than poisoning by mineral acids. A few suicidal and accidental cases have, however, occurred. In most of the accidental cases the alkalies were taken by adults or children in mistake for beer, medicine, etc. Homicidal cases are very rare indeed. A case[2] of attempted murder by the administration

1. *Bombay Chemical Analyser's Annual Report*, 1929, *p.* 5.
2. *Fazekas, Deuts. Zeit. f. d. ges. gericht. Med.*, 1934, *XXIII*, 194 ; *Med.-Leg. and Criminol. Review, Oct.*, 1934, *p.* 372.

of caustic soda is recorded. A man, aged 78, and his wife, aged 76, were given soup containing 7.99 grammes of caustic soda. After the first swallow the couple noticed that the soup had an unpleasant taste and smell and their mouths began to burn. The man spat out the liquid but the woman swallowed a teaspoonful. A dark red painful swelling on the tip and sides of the tongue was found ; the man recovered in 8, the woman after 20, days.

Cases have occurred, where a solution of caustic soda has been thrown maliciously on the face and body of an enemy. In one case[1] a cloth soaked in caustic soda solution was rubbed on the eyes of one Rajaram, a *Mukhtar-i-am* of a lady zamindar, when he was struck with acute pain, and sat down and began to cry. As a result of throwing this corrosive substance he lost his power of sight in one eye completely. His other eye was saved, though it was not in a normal condition at the time. The motive for the assault was the grudge and enmity existing between the complainant and the accused, Gajadhar. In another case[2] a man visited a brothel in Bombay City early one morning and threw some corrosive liquid over the face and bodice of a prostitute who was sleeping there. The liquid on analysis proved to be a concentrated solution of caustic soda.

Caustic soda is also applied externally to the neck of an ox or a buffalo so as to render it unfit for bearing the yoke of a plough owing to its local corrosive action and thus causing a serious loss to an agriculturist.[3]

Poisoning by ammonia is more common than poisoning by fixed alkalies. Owing to the strong smell ammonia is not generally used for homicidal purpose, though a case is recorded in which a man was tried for the murder of a child by administering to it spirits of hartshorn.[4] In another case a man was convicted of throwing maliciously a liniment containing a strong solution of ammonia into a woman's face, with intent to injure her. A portion reached the eyes but she recovered from its effects.[5] Cases have also been recorded in which ammonia was taken with a view to procure abortion. In one case a woman swallowed 90 grammes of aqua ammoniæ, was delivered of a dead fœtus on the second day and died on the eighth day.[6] In the other case ammonia was injected into the vagina, causing atresia vaginæ.[7]

POTASSIUM PERMANGANATE, $KMnO_4$

This is a dark, purplish-red, crystalline salt, and a powerful oxidising agent for almost all organic substances, and is destructive to the low organisms of infectious diseases. Condy's fluid contains 2 per cent of potassium permanganate and Darby's fluid also contains it. Poisoning by potassium permanganate is very rare, but a few accidental and suicidal cases have lately been recorded.

1. *Leader, May* 28, 1925, *p.* 10.
2. *Bombay Chemical Analyser's Annual Report*, 1928, *p.* 6.
3. *Beng. Chem. Exam. Ann. Rep.*, 1937, *p.* 16.
4. *Regina* v. *Haydon, Somerset Spring Assizes*, 1845 ; *Taylor, On Poisons, Ed. III, p.* 259.
5. *Reg.* v. *Gavan, Stafford Summer Assizes*, 1873 ; *Ibid., p.* 258.
6. *Francais, Ann. d'hyg.*, 1872, *s., XLVII*, 456 ; *Witthaus, Med. Juris. and Toxic., Vol. IV, p.* 329.
7. *Neugebauer, Verwachs. u.* "*Vereng. d. Scheide,*" *Berlin*, 1895 ; *Ibid.*

Symptoms.—These are burning pain in the mouth, throat and stomach, spreading over the whole abdomen, intense thirst, difficulty in swallowing, almost continuous vomiting and difficult breathing. The salt corrodes the tongue and pharynx, and stains the parts black or dark brown. Death occurs from paralysis of the heart.

Fatal Dose and Fatal Period.—The official dose of potassium permanganate is 1 to 3 grains, though 1 and 2 grains have respectively caused alarming symptoms.[1] A case is recorded in which a married woman, aged 36 years, who took pills containing 22 grains of potassium permanganate during a period of four days with a view to cure amenorrhœa, suffered from poisonous symptoms but recovered in a week.[2] A handful of the crystals of potassium permanganate taken in beer caused the death of a woman of forty-seven years in thirty-five minutes.[3] Thompson reports the case of a woman who swallowed fifteen to twenty grammes of the salt and died in five hours.[4] A single woman, aged about 67 years, swallowed about an ounce and a half of a strong solution of potassium permanganate, and died in about five hours.[5] A female child, aged 2 years, who swallowed about half a teaspoonful of potassium permanganate and was treated in the King George's Hospital, Lucknow, died in twenty-seven hours from suffocation due to œdema glottidis. A mechanic died four days after he had irrigated his urethra with 25 grammes of potassium permanganate dissolved in a teacupful of water for two days.[6] A woman, 37 years old, took ten grammes of the solid salt with intent to commit suicide, and died on the fourth day from pneumonia.[7] On the other hand, a youth, 17 years old, who, with a view to commit suicide, swallowed the mercury from a thermometer and then a tablespoonful of potassium permanganate in some water, recovered under prompt treatment.[8]

Treatment.—Usually there is not much corrosion of the stomach, hence it may be lavaged with charcoal. Give white of egg and milk. Administer intravenous injections of calcium bromide or intramuscular injections of calcium gluconate.[9] Treat the symptoms on the general lines.

Post-mortem Appearances.—Signs of corrosion if a strongly concentrated solution or the solid form of the salt is taken. The mouth, pharynx and œsophagus are often corroded and blackened. The parts that escape corrosion are usually congested and inflamed. Œdema of the glottis and inflammation of the larynx and trachea have been observed.

Chemical Tests.—1. Oxalic acid decolourises a solution of potassium permanganate. 2. Fused with caustic potash or soda or mica foil, a green residue will be left. 3. A solution of cocaine hydrochlorate throws down a precipitate of characteristic appearance under the microscope.

1. *Bidwell, Boston Med. and Surg. Jour.*, 1886.
2. *Hawthorne, Lancet. Nov.* 25, 1899, *p.* 1467.
3. *Box, The Lancet, Aug.* 12, 1899, *p.* 411.
4. *Petersb. Med. Wochenschr.*, 1895; *Dixonmann, Forens. Med. and Toxic., Ed. VI, p.* 414.
5. *A Douglas Cowburn, Transactions, Medico-Legal Society, Vol. XIV, p.* 17.
6. *Willimott and Freiman, Brit. Med. Jour., Jan.* 11, 1936, *p.* 58.
7. *Adler, Med. Klinik, Berlin, Aug.* 16, 1914, *p.* 33; *Peterson, Haines and Webster, Leg. Med. and Toxic., Vol. II, Ed. II, p.* 160.
8. *Hoke and Wachter, Med. Klin.*, 1932, *XXVIII, p.* 1558; *The Med.-Leg. and Criminol. Rev., April*, 1933, *p.* 158.
9. *Leschke, Clinical Toxicology, Eng. Trans. by Stewart and Dorrer*, 1934, *p.* 82.

CHAPTER XXIV

IRRITANT POISONS

Irritant poisons are those which, by their specific action, set up inflammation in the gastro-intestinal canal.

General Symptoms.—The symptoms are delayed from half an hour to an hour or more. These are burning pain, difficulty in swallowing, feeling of constriction in the throat and œsophagus, severe pain in the stomach, intense thirst, nausea and violent, persistent vomiting. The vomited matter at first contains food, then becomes bilious and lastly contains altered blood. There is purging accompanied by tenesmus and pain, and tenderness over the abdomen ; the stools may contain mucus and blood. There is dysuria. Collapse sets in, when the skin is cold and clammy, and the pulse is quick, feeble and intermittent. Cramps also occur in the legs. Sometimes, convulsions occur before death, which may take place at once from shock, or from exhaustion in one to four days.

If the patient survives for some time reaction sets in, consequently the skin becomes hot and dry with a rise of temperature, but death may occur later from stricture of the œsophagus.

Diagnosis.—Irritant poisoning has to be diagnosed from certain diseases, such as cholera, acute gastritis, acute gastro-intestinal catarrh, peritonitis, colic, and rupture of the stomach.

A. INORGANIC

I. NON-METALLIC POISONS

PHOSPHORUS

There are two varieties of phosphorus ; *viz.*, *yellow* or *crystalline* and *red* or *amorphous*. The yellow variety usually occurs in the form of waxy, semi-transparent sticks. It is insoluble in water, somewhat soluble in alcohol and ether, and also to a slight extent in fatty and ethereal oils, but readily dissolves in carbon bisulphide. When exposed to the air it slowly oxidises and emits white fumes, which have a garlic-like odour, and are luminous in the dark. At 34° C. it ignites in the air, burning with a very white flame. On account of the ease with which phosphorus undergoes oxidation it is always preserved under water.

Yellow phosphorous is very poisonous, and is used in preparing vermin paste for the destruction of rats and other vermin. This paste contains one to four per cent of phosphorus mixed with oil, flour, sugar and some pigment, probably indigo. It is also used in the manufacture of lucifer matches, and enters into the composition with which these matches are tipped.

Yellow phosphorus (dose, 1/100 to 1/25 grain), oleum phosphoratum (dose 1 to 5 minims) and pilula phosphori (dose, 1 to 4 grains), the former official preparations, are not included in the British Pharmacopœia of 1932. Elixir phosphori (B.P.C.) is the best fluid preparation, the dose being 15 to 60 minims.

Red phosphorus is a reddish-brown, amorphous powder, and is prepared by heating the yellow variety at a temperature of 240° C. to 250° C. in an atmosphere of nitrogen or carbon dioxide. It is insoluble in carbon bisulphide, is not luminous in the dark, and has no taste or odour. It does not oxidise in air at ordinary temperatures, and is not, therefore, preserved under water. Unlike the yellow variety, it is not poisonous, but the commercial red phosphorus may be poisonous, as it sometimes contains as much as 0.6 per cent of the yellow variety. It is used in the manufacture of "safety" matches, but the matches do not contain phosphorus, being tipped with a mixture of potassium chlorate and antimony sulphide. They are ignited by being rubbed upon the side of the containing box, which is covered with a thin layer of red phosphorus and powdered glass.

Symptoms.—In acute poisoning the symptoms may appear in a few minutes after swallowing a poisonous dose, but usually they are delayed from one to six hours. In a case where a girl swallowed a quantity of phosphorus paste the symptoms did not set in till the fifth day.[1]

The symptoms complained of by the patient are a garlic-like taste in the mouth, and burning pain in the throat, gullet and stomach followed by intense thirst, nausea and vomiting. The ejected matter has a garlicky odour, is luminous in the dark, and is coloured with bile, but later contains almost pure blood. The breath is also garlicky in odour and may be luminous in the dark. Doarrhœa is not a constant symptom but, when present, the motions are dark, offensive and, sometimes, phosphorescent just like the vomited matter. In rapidly fatal cases these symptoms become severe, collapse sets in, and the patient passes into a state of delirium or convulsions and coma.

In most cases, however, the symptoms abate, and there is a semblance of recovery. After a period of intermission lasting from two to six days jaundice makes its appearance, and becomes well-marked. The pain in the stomach increases in severity, and the abdomen becomes distended. The liver is greatly enlarged and tender to touch, and so is the spleen. Vomiting is much more distressing. Diarrhœa is more severe. Both the vomited matters and the motions contain blood. There are also hæmorrhages from the nose and other mucous membranes, such as the urethra, vagina and uterus. Abortion occurs in a pregnant woman with alarming flooding. Subcutaneous hæmorrhages or purpuric spots may be present. The urine becomes very scanty, highly coloured and strongly acid in reaction, containing albumin, blood, bile-pigments and tube casts, and occasionally leucin, tyrosin and cystin.

Nervous symptoms develop; *viz.*, frontal pains, restlessness, insomnia, singing in the ears, deafness, impaired vision, formication, cramps, tremors

1. *Taylor, On Poisons, Ed. III, p.* 277.

and paralysis. There is frequently priapism. The pulse becomes feeble, quick and irregular. Fever sets in, and a condition of stupor or coma supervenes ending in death. Sometimes, delirium or convulsions precede death.

West[1] reports the case of a woman, 52 years old, who swallowed phosphorus rat-poison. Within fifteen minutes she complained of burning in the mouth and pain in the abdomen. In twelve days she was considered well. Four weeks later, she developed jaundice with great depression, black vomit, and pains in the head, back and legs. Death occurred in six days.

Fatal Dose.—The smallest fatal dose for an adult is one-eighth of a grain,[2] but one to two grains may be regarded as an ordinary fatal dose. An infant of five weeks is reported to have died from sucking the head of a single lucifer match containing about one-fiftieth of a grain of phosphorus.[3] Recovery has followed the doses of four and six grains.

Fatal Period.—The shortest recorded fatal period is half an hour.[4] Death occurs in four to ten hours if it is due to collapse; otherwise it usually takes place in from two to seven days, but may be delayed for two to three weeks.

Treatment.—The stomach should be washed out with a solution of potassium permanganate of the strength of about 10 to 15 grains to one pint of water. Potassium permanganate acts as a chemical antidote, oxidising phosphorus, forming harmless compounds, phosphoric acid and phosphates, and itself changing to manganese dioxide. Large doses of charcoal should afterwards be administered.

The stomach can also be washed out with warm water until the smell of phosphorus disappears, and then with magnesia suspended in water or with milk of magnesia. Two to three grain-doses of copper sulphate may be given every five minutes until free emesis is produced. It acts as an antidote, as it combines with phosphorus and forms an insoluble harmless salt, phosphide of copper. Oils and fats must never be given, for they dissolve phosphorus, but old unrectified turpentine (French turpentine) is recommended as an antidote. It combines with phosphorus and produces a solid non-poisonous substance, terebinthino-phosphoric acid. It is given in half-drachm does in mucilage every hour. If it cannot be obtained, the oxidising agents, such as hydrogen peroxide and sanitas, may be administered. Liquid petrolatum is also recommended as an antidote in phosphorus poisoning. It is physiologically inert and acts entirely by reason of its physical properties. Administered one hour after the poison is taken it affords complete protection against the onset of harmful effects.[5] Purgatives, especially magnesium sulphate, should be given to evacuate the bowels. Morphine may be given hypodermically to relieve pain, and to combat shock. Sodium bicarbonate may be given to

1. *Lancet, Vol. I,* 1893, *p.* 245.
2. *Lobel, Ann. d. Hyg.,* 1857, *VI, p.* 422.
3. *Luff, Forens. Med., Vol. I, p.* 141.
4. *Med. Chir. Trans.,* 1867, *Vol.* 50.
5. *Atkinson, Jour. of Laboratory and Clinical Medicine, St. Louis, Dec.,* 1921, *Vol. VII, No.* 3, *p.* 148.

increase the alkalinity of the blood which is generally lowered by the action of phosphorus.

Post-mortem Appearances.—Petechial hæmorrhages are commonly found under the skin, which is usually yellow. On opening the cavities of the body the smell of garlic may be observed, but this is not possible in India owing to the rapid occurrence of putrefactive changes. Casper[1] describes the post-mortem examination of a case in which he noticed a greyish-white vapour smelling strongly of phosphorus streaming from the vagina and the relaxed open anus. The mucous membranes of the stomach and intestines are yellowish- or greyish-white in colour, and are softened, thickened, inflamed and corroded, or completely destroyed in patches exhibiting even perforations. Their contents may be garlicky in odour, and luminous in the dark.

The liver presents the most characteristic appearances. It is very much enlarged, but may be of normal size or contracted. It is doughy in consistence, uniformly yellow, easily friable, and contains many hæmorrhagic spots in its substance. There is fatty degeneration of the liver cells. In *acute yellow atrophy* the liver is smaller in size, greasy, leathery and of a dirty yellow colour. Its capsule is wrinkled. The liver-cells are mostly necrosed, and contain crystals of leucin and tyrosin.

The heart and kidneys show signs of fatty degeneration. The blood is fluid and disorganized, the colouring matter of the hæmoglobin being dissolved in the liquor sanguines.

Cases.—1. A married woman,[2] 37 years old, was admitted to hospital on September 7th, 1927, with a history of having swallowed a tea-spoonful of phosphorous mice paste two days previously. Within 15 minutes of taking the paste her mouth had been washed out and sulphur administered. The patient was said to have vomited several times after the washing out of the stomach. On admission to hospital she appeared rather nervous, but answered questions quite sensibly. She said she had no pain anywhere, but had belched wind several times. She asked for food, but was only allowed water to drink since it was thought that milk might aid absorption of the phosphorus. There was no ulceration of the lips, tongue, buccal mucous membrane, or throat. The pulse was 80 and of good volume and tension; there was no hepatic enlargement, jaundice, or abdominal tenderness. Next day her physical and mental condition was apparently better, and she was allowed tea and sugar and a piece of dry toast. There was no sign of enlargement of the liver and no jaundice. The pulse remained the same, and she had no pain or sickness; the food was retained. On September 10th, she was drowsy, but not comatose. She resisted strongly attempts at palpation and percussion over the lower right costal margin. Albumin and bile were found in the urine, but no leucin or tyrosin. A few granular and cellular casts with occasional polymorphonuclear leucocytes were seen, and the urea percentage was 2. On the next day the patient was troubled with retching, but did not vomit. The jaundice was more pronounced, especially in the conjuctivæ, and the lower edge of the liver was felt easily about an inch below the right costal margin. The patient appeared to be tender over this area and became restless on palpation, though she lay quietly and dozed, except when being examined. The tongue was dry, and there was sordes of the mouth, but no petechial hæmorrhages of the skin were observed. She became comatose and died on the morning of September 12th.

On post-mortem examination there was marked jaundice of the skin and conjuctivæ, some petechial hæmorrhages were seen on the surfaces of both lungs, and there were fluid effusions (10 ozs. and $8\frac{1}{2}$ ozs.) into both pleural cavities. The

1. *Forens. Med., Eng. Trans., Vol. II, p.* 100.
2. *Swinton, Brit. Med. Jour., Decr.* 10, 1927, *p.* 1080.

heart was normal in size, but showed some fatty degeneration of the wall, with well-marked petechial hæmorrhages in the papillary muscles. The pericardium was normal, but the cellular tissue in front of it showed obvious hæmorrhages in the papillary muscles. Petechiæ covered the upper surface of the diaphragm, particularly over the left dome. The liver was enlarged and there was evident fatty degeneration. The right lobe extended to the level of the third rib above, and below to about an inch below the right costal margin, and $2\frac{1}{2}$ inches below the ensiform cartilage. The abdominal viscera smelt very strongly of garlic. The spleen, which weighed $6\frac{1}{2}$ ounces, was deeply congested, but there was no peritonitis. The left kidney weighed $6\frac{1}{2}$ ounces, and there were well-marked hæmorrhages beneath the capsule and into the lower half of each pyramid; the capsule stripped readily. The right kidney weighed $5\frac{3}{4}$ ounces and both organs showed fatty degeneration. There was no inflammation of the outer coats of the stomach. The viscus contained a blackish-brown glairy fluid resembling altered blood which did not smell of phosphorus. Black specks, looking like altered blood, were studded over the inner coat, but these were not firmly adherent to the stomach wall. Hæmorrhages were seen on the surface of the pancreas. The uterus was non-gravid and appeared to have undergone fatty degeneration. Hæmorrhages, each about the size of a split pea, studded the mesentery surface. The bladder was distended, but the urine was not luminous in the dark. No naked-eye pathological changes were found in the brain.

2. On February 8, a male child, 18 months old, ate a piece of bread over which a thick layer of roach paste containing 1.19 per cent phosphorus was spread for the destruction of roaches, and became drowsy and vomited. On the following day the vomiting was repeated, the eyes became somewhat yellow, and the patient was slightly drowsy and passed urine only once. On February 10, he became irritable and was given castor oil; the resulting stools were yellowish-brown and fluid in character, but without blood. In the evening he vomited five or six times. On the next day the abdomen was distended and the wrists and ankles began to swell. The urine was "blood red" and the patient vomited a black material and the drowsiness and jaundice increased. He no longer recognised the parents, and had taken no food and passed no urine during the last sixteen hours. The child was removed to the hospital at 11 a.m., when he was in a moribund comatose condition with icteroid skin, slight cyanosis of the lips and finger nails and œdema of the hands and feet. Blood was noted in the nostrils, oozing from the mouth and about the anus. There were two small ecchymotic patches in the skin, about 1 c.m. in diameter, one over the hypochondrium and another over the inner aspect of the left thigh. The pupils were dilated and irregular and reacted sluggishly to the light. The sclera were jaundiced. The mucous membrane of the mouth was normal; the throat was injected and the tonsils were enlarged. The cervical glands were moderately large. The pulse was 90 per minute and the respirations were 16 per minute with periods of apnœa. The knee reflexes were exaggerated. The spleen was slightly enlarged and the liver was hard and enlarged and almost reached the crest of the ileum. The temperature was 98.6° F., and four hours later it had risen to 103° F. The urine was acid, with a specific gravity of 1020 and albumin. Hyaline and granular casts were seen. 20 per cent dextrose was given intravenously, and transfusion of whole blood by direct method, but the child died nine hours after admission to the hospital.

The post-mortem examination showed that the lungs were congested and œdematous. The spleen was red, enlarged and firm. The liver was markedly enlarged, firm and yellowish green, the capsule was smooth and the substance was markedly fatty with small areas of congestion. The stomach showed post-mortem digestion at the cardiac end. Its contents were dark brown mucus. The walls were œdematous, the mucosa specially so. The rugæ were yellowish and the rest of the mucosa was red, with small capillary hæmorrhages. The lymph nodes along the upper border of the stomach and in the fissure of the liver were slightly enlarged, pale and moist. The duodenum was normal. The kidneys were enlarged, the cortex was swollen and yellow. The medullary pyramids were deep red. The capsule was free. The bladder was normal. Phosphorus was detected in the liver, kidney, spleen and intestines.[1]

1. *Mc Lean, Mc Donald and Sullivan, Jour. Amer. Med. Assoc., Dec.* 7, 1929, *p.* 1789.

Chronic Poisoning.—This form of poisoning, although rare in these days, may occur among persons exposed to the white fumes resulting from the oxidation of yellow phosphorus in factories.

The symptoms are a sallow complexion, lassitude, pain in the abdomen, vomiting, diarrhœa and emaciation ; but the chief characteristic symptoms are caries of the teeth and necrosis of the jaws, especially the lower jaw. It is supposed that the vapour of phosphorus gains access to the jaw (phossy jaw) through a carious tooth or an interspace caused by a missing tooth. Death occurs from debility, blood poisoning or pyogenic infection.

Pregnant women usually abort.

Treatment.—Advise thorough cleanliness and ventilation by the use of extraction fans in the match factories. The air of the work rooms may be saturated with turpentine. The teeth of workmen should be occasionally examined, and the carious teeth, if found, should be filled in or extracted. The workmen should also be persuaded to use systematically mouth washes of sodium bicarbonate.

Chemical Analysis.—Although phosphorus is very readily oxidised in air, it may be detected in the unoxidised form in a dead body several days after death even when it has reached an advanced state of decomposition. This is probably due to the fact that the reducing gases which are developed during decomposition protect phosphorus from oxidation. Hoffmann[1] detected phosphorus in the putrid intestinal contents after three months in a case which proved fatal in eight hours, and after five months in another case which proved fatal in three days. Poleck[2] found free phosphorus in a cadaver three months after poisoning had occurred. Alpers[3] found phosphorus in the gastro-intestinal contents of a woman who had died as a result of acute phosphorus poisoning and whose body was not exhumed until four weeks after death. Felletar[4] proved the presence of free phosphorus in bodies which had been buried for twelve and thirteen months. It must, however, be remembered that such instances are very rare, and it is advisable that in cases of suspected phosphorus poisoning the chemical analysis should be made as early as possible. It must also be borne in mind that phosphorus occurs in combination mainly as phosphates in various articles of food and in the tissues and fluids of the human body ; hence its detection in these forms has no value for medico-legal purposes, but its presence in the body in the elementary form is sufficient to prove phosphorus poisoning, as it does not occur free in nature.

Phosphorus may be separated by distillation from organic mixtures, and may be detected by its smell and luminosity in the dark. Its phosphorescence is diminished by the presence of alcohol. Hence, in cases of suspected phosphorus poisoning a saturated solution of common salt should be used as a preservative instead of alcohol. It can also be separated by

1. *Lehrbuch der ger. Med.*, 1887, *p.* 679 ; *Witthaus, Manual of Toxic., Ed. II, p.* 671.
2. *Archiv. d. Pharmazie*, 225 (1887), 189 ; *Autenreith, Detection of Poisons (Warren's Translation), Ed. VI, p.* 21.
3. *Pharm. Ztg.* 58 (1913), 127 ; *Autenreith, Ibid., p.* 21.
4. *Pest. Med.-Chir. Presse*, 1890, *XXVI, p.* 148 ; *Witthaus, Loc. Cit.*

shaking the contents of the stomach, etc., with carbon bisulphide, which dissolves phosphorus.

Tests.—1. *Mitscherlich's Test.*—If the mixture is acidulated with sulphuric acid and distilled, the luminous vapour of phosphorus will be seen in the cool condenser in the dark. This is a very delicate test, and will reveal 1 in 200,000.

2. *Lipowitz's Test.*—If pieces of sulphur are added to the mixture acidulated with sulphuric acid and boiled, they become phosphorescent in the dark.

3. *Scherer's Test.*—If a piece of blotting paper soaked in a solution of silver nitrate is suspended in a flask containing the suspected material mixed with lead acetate and ether, the paper will become black from the formation of silver phosphide after keeping it in the dark for some hours.

4. *Phosphine Test.*—If hydrogen is passed through the warmed suspected fluid material, phosphuretted hydrogen is evolved, which will burn with a green flame. If the gas is passed into a solution of silver nitrate, a black precipitate is formed. The green flame, when examined with a spectroscope, shows one band in the orange and yellow between C and D, but very close to D, and several bands in the green. This test is also known as Dussart-Blondlot's test.

Medico-Legal Points.—The poisonous effects are more powerful if phosphorus is dissolved or well triturated than when used in solid lumps.

In England phosphorus poisoning is usually suicidal. Pregnant women have often been accidentally poisoned by phosphorus as they take it to induce criminal abortion. It is seldom used for homicidal purposes. The odour and taste as also the luminosity in the dark reveal its presence. Casper[1] describes a case in which the luminous appearance of the poisoned food led to a suspicion of poisoning with phosphorus and this was subsequently proved. A woman mixed a preparation of phosphorus into soup, and gave it to her husband. He ate it in a dark room in the presence of a few friends who noticed that the warm liquid, as he stirred it, was luminous. At the Norwich Autumn Assizes, 1871, a girl, aged 18, was convicted of an attempt to poison a family with a vermin compound of phosphorus. She put the substance into a tea pot containing tea. When hot water was poured on it, the smell at once led to suspicion. The girl was sentenced to penal servitude for life.[2]

Poisoning by phosphorus is rare in India, but a few accidental and suicidal cases have occurred. A Hindu student took phosphorus for three days to improve his memory, and died on the seventh day after the first dose.[3] A Hindu male child[4] died after swallowing accidentally 4 or 5 "cracker caps", little reddish pellets containing phosphorus and enclosed in circular discs of paper. They are intended to be rubbed against any hard or rough surface when they will ignite spontaneously and continue

1. *Vierteljahrsschrift. July,* 1864; *Taylor, On Poisons, Ed. III, p.* 275.
2. *Reg. v. Fisher; Taylor, On Poisons, Ed. III, p.* 275.
3. *Ind. Med. Gaz., June,* 1887, *p.* 171.
4. *Bombay Chemical Analyser's Annual Report,* 1930, *p.* 6.

burning in a succession of small explosions accompanied by evolution of bright sparks and clouds of irritating vapour. A Parsi lad,[1] not seeing his name in the first published list of the successful candidates at the matriculation examination declared on May 31, 1933, took a dose of phosphorus with fatal consequences, as he thought that he had failed. His name appeared in the second list of successful candidates.

A case[2] of attempted homicidal poisoning by phosphorus is recorded. A woman administered tips of matches in a betel to her husband with the intention of poisoning him. The man, on chewing the betel, detected a peculiar taste and smell, and immediately spat it out. The chewed betel was found to contain tips of lucifer matches in which phosphorus was detected.

Phosphorus is occasionally used to set fire, and is frequently suspected of being the cause of the so-called spontaneous combustion occurring in cotton bales. In his annual report for 1928 the Bombay Chemical Analyser mentions that he received two small tin pill-boxes from Ahmedabad where they had been seized in connection with a case of suspected arson. One contained a piece of charred cotton wool waste and the other a few fragments of a dark, fuming, semi-solid substance. The fuming matter proved to be yellow phosphorus, and the same was detected in the charred cotton. Yellow phosphorus, rolled up in a wet cloth,[3] or dissolved in carbon bisulphide,[4] was also employed to set fire to postal letter boxes during the civil disobedience movement in 1932.

Phosphuretted Hydrogen (Phosphorus Trihydride, Phosphine, PH_3). This is obtained by boiling phosphorus in a flask with a solution of potassium or sodium hydroxide. It is also produced, when calcium phosphide is brought into contact with water. It is a colourless gas, very slightly soluble in water and having a penetrating, garlic-like odour. It is not inflammable in air at ordinary temperatures, but it ignites at a temperature below 100° C.

This gas, when inhaled, is highly poisonous, reducing the oxyhæmoglobin of the blood, and proves rapidly fatal, when 2000 parts of it are contained in one million parts of air. Four hundred to six hundred parts of the gas per million parts of air produce dangerous symptoms, if inhaled for half to one hour, while 100 to 200 parts per million of air is the maximum amount that can be inhaled for an hour without serious results.[5] The chief symptoms are marked dyspnœa, diarrhœa, weakness, tremors, convulsions and death from respiratory failure. Several cases of fatal poisoning by this gas have occurred on board ships carrying ferro-silicon as part of their cargo. Ferro-silicon is an alloy of iron and silicon used in the manufacture of steel. It often contains as an impurity calcium phosphide, which undergoes decomposition in the presence of moisture and evolves phosphuretted hydrogen.

1. *Free Press Journal, June* 4, 1933.
2. *Ind. Med. Gaz.*, Oct. 1907, *p.* 394 ; *Beng. Chem. Exam. Rep.*, 1906.
3. *Madras Chem. Examiner's Annual Rep.*, 1932, *p.* 8.
4. *Punjab Chem. Examiner's Annual Rep.*, 1932, *p.* 7.
5. *Henderson and Haggard, Noxious Gases,* 1927, *p.* 188.

CHLORINE

Chlorine is a greenish-yellow gas, having an unpleasant irritating odour even when diluted, and is largely used as a disinfectant and bleaching agent. Poisoning by chlorine is very rare except accidentally in chemical laboratories and factories where chlorine and its compounds, especially bleaching powder (calcium hypochlorite) is used or manufactured. It was largely ured as a lethal gas by the Germans during the last Great War.[1]

Symptoms.—The chief effects after inhalation of the gas are an intense feeling of tightness in the chest, and a sense of being totally unable to take an inspiration. The cases may be of three degrees, mild, severe or extreme.

Mild.—In these cases cough is frequent and painful. Cough may be dry and harsh, or may be accompanied by greenish, viscid sputum. Other signs are headache, pain in the eyes, and abdominal pain with hurried respiration. These symptoms soon subside.

Severe.—The patient is cyanotic, with frequent panting and painful respirations, headache, a little pyrexia and drowsiness.

Extreme.—Dry, red throat, cracked and furred tongue, intense cyanosis, œdema in the lungs, pulse 80 and respirations 30 per minute, coma and death.

Inhaled in a concentrated form, *i.e.*, when not freely diluted with air, it causes death by cardiac paralysis or by asphyxia.

People exposed to the vapours of chlorine suffer from its chronic effects. They become anæmic, suffer from dyspeptic complaints and acidity, and lose flesh. Their teeth soon become carious. Lung troubles then set in resulting in chronic bronchitis and emphysema.

Compounds of Chlorine.—The compounds of chlorine, such as bleaching powder (calx chlorinata), Dakin's solution (liquor sodæ chlorinatæ chirurgicalis) and Labarraque's disinfecting fluid (liquor sodæ chlorinatæ), act as irritant poisons, and produce acute gastritis, and suffocative bronchitis.

Fatal Dose.—Uncertain. Air containing about 1 part of chlorine in 3,000 may prove fatal immediately by causing acute œdema of the lungs. Three to four drachms of a bleaching fluid consisting of a solution of potassium or sodium hypochlorite caused the death of an infant. Recovery has, however, been recorded after a dose of twenty ounces.[2]

Fatal Period.—Forty-eight hours in the case of a patient who took two breaths of pure chlorine.[3]

Treatment.—Removal of the patient in fresh air, and steam or oxygen inhalation. In severe cases of chlorine poisoning it is very necessary to get rid of the exudation collecting in the air tube which is asphyxiating the patient. This can be done by squeezing the thorax, inverting the posture and resorting to artificial respiration, especially the mouth to mouth method. It is suggested that atropine should be administered hypodermically, as it lessens secretion of fluid and dilates the bronchioles, but is of no use in severe cases. As a prophylaxis against the gas the soldiers in the late war were provided with respirators (masks) soaked in a solution of sodium bicarbonate and hyposulphite of soda, also known as thiosulphate of soda, and goggles for the eyes. When the bleaching powder has been swallowed, evacuate the stomach contents, and treat the symptoms by giving demulcent drinks and hypodermic injections of morphine to relieve pain.

Post-mortem Appearances.—Intense inflammation of the air-passages, emphysema and œdema of the lungs, which, on section exude tenacious, frothy and slightly

1. *For detailed description* vide *an address on Gas Poisoning by Leonard Hill, Brit. Med. Jour., Dec.* 4, 1915, *p.* 801.
2. *Dixonmann, Forens. Med. and Toxic., Ed. VI, p.* 430.
3. *Sury-Bientz, Viertelijahr, f. ger. Med.*, 1888.

blood-stained secretion. The same kind of secretion fills the trachea and bronchi. Acute catarrhal condition of the stomach and duodenum. Congestion of the abdominal organs. The odour of chlorine in the ventricles of the brain. The heart is enlarged.

Tests.—The gas can be recognised by its odour and its bleaching action on moist litmus paper. Chlorine water dissolves gold-foil. Chlorine water (or gas) added to a mixture of potassium iodide and starch paste turns it blue, which is discharged on heating.

BROMINE

Bromine is a dark, reddish-brown liquid, volatilising at ordinary temperatures in red and intensely irritating fumes of an unpleasant odour. In a free state it is found only in laboratories and chemical works. Cases of poisoning by it are rare. Fatal results, however, occur when it is taken internally, or its fumes are inhaled.

Symptoms.—When taken internally in a liquid form, it acts as a corrosive poison and causes intense burning pain in the mouth, throat, stomach and abdomen, dysphagia, vomiting and eructations of a peculiar offensive vapour. The toxic action is so rapid and powerful that unconsciousness and collapse soon supervene without even producing the initial symptoms of thirst, vomiting, purging, etc. It produces a severe wound if it drops on the hand or any other part of the body.

Its fumes, when inhaled, cause symptoms of violent catarrhal inflammation of the air-passages, producing cough, constriction of the chest, and hæmoptysis.

Compounds of Bromine.—Bromides of ammonium, sodium and potassium act as sedatives to the nervous system when taken in medicinal doses (5 to 30 grains), but produce poisonous symptoms, known as "bromism", when administered in large doses, or when continued for too long a period. The symptoms are skin eruptions in the form of red papules on the face and back, salivation, foul odour from the breath, delirium and death. In some cases the patient loses sexual power, becomes dull and is unable to work. He often becomes demented or melancholic.

A case[1] is recorded where a saturated solution of potassium bromide was administered by a man to his wife with intention to cause harm.

Bromine is eliminated in the milk. An infant, 6 months old, got a painful pustular eruption due to bromine secreted in the milk of the mother, who had been addicted to the use of a proprietary remedy, "Miles restorative nervine", a solution of bromides in syrup.[2]

During the late war the Germans used certain organic compounds of bromine in asphyxiating and lachrymating shells. The vapours of these substances in concentration as little as one part in several millions of air are said to cause watering of the eyes and inability to open them, so specifically irritating are they to the conjuctivæ. In greater concentrations they are said to cause irritation of the mucous membrane of the respiratory tract.[3]

Fatal Dose and Fatal Period.—Uncertain. One ounce of undiluted bromine has caused death in seven hours and a half.[4] About 2 grains of free bromine caused the death of a girl, aged ten years, in 12 hours. She was given a mixture containing potassium bromide and chlorine water in three doses.[5] A woman suffering from epilepsy took two drachms of potassium bromide continuously for weeks. At last she got poisonous symptoms and died in 5 days.[6] One ounce and a half of potassium bromide have caused alarming symptoms without any fatal result.[7] A dose of one

1. *Beng. Chem. Examiner's Annual Report*, 1934, *p.* 13.
2. *Jour. Amer. Med. Assoc., April* 9, 1921, *p.* 1012; *Amer. Jour. Dis. Children*, 1921, 21, *p.* 167.
3. *Leonard Hill, Brit. Med. Jour., Dec.* 4, 1915, *p.* 801.
4. *Snell, New York Med. Jour., Nov.* 1850, *V, p.* 179.
5. *Hering, Zeitschr. f. Medicinalbeamte*, 1889, *II, p.* 217.
6. *Eigner, Weiner Med. Presse*, 1886.
7. *Dougall, Glasgow Med. Jour.*, 1893.

hundred grammes of sodium bromide caused death in six days from a bilateral pneumonia of the inferior lobe.[1]

Treatment.—Administer apomorphine hypodermically or other emetics, and give starch or albumen. Give ammonia vapour and steam for inhalation when the bromine fumes are inhaled.

Post-mortem Appearances.—When liquid bromine is administered, there is inflammation of the œsophagus and stomach with dark brown stains on the mucous membrane, which presents a leathery, parchment-like appearance. Occasionally there is perforation of the stomach, or the stomach wall is destroyed altogether.

When bromine fumes are inhaled, there is irritation of the air-passages with the signs of bronchitis or pneumonia.

Chemical Analysis.—Free bromine may be separated from organic mixtures by distillation. If combined, the mixture should be saturated with potassium bichromate, and acidulated with sulphuric acid before it is distilled.

Tests.—Bromine can be recognised by its colour and odour, as well as by its colouring starch paper yellow. It forms an orange or yellow coloured solution in chloroform or carbon bisulphide, and with phenol forms a crystalline white precipitate due to the formation of tribromophenol, insoluble in water. Compounds of bromine give a whitish-yellow precipitate with silver nitrate, which is not readily soluble in ammonium hydrate, but soluble in potassium cyanide.

IODINE

This is a solid, having blue-black, soft and scaly crystals with a metallic lustre and an unpleasant taste. At all temperatures it gives off a violet coloured vapour possessing a characteristic odour. It is only slightly soluble in water, but is freely soluble in alcohol, ether, carbon bisulphide and an aqueous solution of potassium iodide. The following are the pharmacopœial preparations of iodine :—

1. *Liquor Iodi Fortis* (Tinctura Iodi Fortis, or Liniment Iodine).—Contains 10 per cent of iodine by weight and 6 per cent of potassium iodide by weight.

2. *Liquor Iodi Mitis* (Tinctura Iodi Mitis or Tincture Iodine).—Contains 2.5 per cent of iodine and 1.5 per cent of potassium iodide. Dose, 5 to 30 minims.

3. *Liquor Iodi Simplex.*—Contains approximately 10 per cent of iodine by weight. Dose, 3 to 15 minims.

4. *Syrupus Ferri Iodidi.*—Contains 7½ grains of ferrous iodide in 120 minims. Dose, 30 to 120 minims.

Acute poisoning by free iodine is a very rare occurrence. Accidental cases of poisoning by drinking carelessly the tincture or liniment of iodine have occurred, and a few suicidal cases have also been reported. In his annual report for 1930 the Chemical Examiner of the United Provinces of Agra and Oudh reports the case of a woman who attempted to commit suicide by taking tincture of iodine. On analysing the vomited matter he found potassium iodide but no free iodine. The preparations of iodine cannot be used for homicidal purposes, as they colour farinaceous foods blue.

Iodine, if swallowed in the solid form, acts as a corrosive poison, while its vapours are strongly irritant to the respiratory passages. A strong solution of iodine (liquor iodi fortis) has produced irritant symptoms when injected into a cyst or a body cavity or applied to the skin. During its elimination by the kidneys it causes their inflammation, giving rise to suppression of urine.

Symptoms.—Acute Poisoning.—Soon after swallowing a large dose of iodine, there is a burning pain in the mouth, œsophagus and stomach, followed by intense

---

8. *Vilen, quoted by Erich Leschke in his Clinical Toxicology, Eng. Trans. by Stewart and Dorrer*, 1934, *p.* 95.

thirst, salivation, vomiting and purging. The vomited matters and stools are dark or blue in colour, contain blood, and have the peculiar odour of iodine. The urine is suppressed or scanty, dark, red-brown in colour and has a strong smell of iodine. The pulse is small and compressible, the skin is cold and clammy and the patient passes into a state of collapse. Consciousness is retained till death. Severe symptoms from poisoning by potassium iodide are more frequently seen in patients suffering from goitre. Some people are particularly susceptible to the poisonous symptoms of this salt even from medicinal doses (5 to 30 grains).

Chronic Poisoning.—The symptoms of chronic poisoning, sometimes. occur from a continued use of large doses of potassium iodide medicinally, and are known as *iodism*. The symtpoms are heavy pain over the frontal sinus, running of the nose, salivation, nausea, vomiting, purging, emaciation, wasting of the breasts, testicles, and other glands and erythematous patches on the skin.

Eller and Fox[1] report a fatal case of iododerma in a man, aged 31 years, with macules, papules, tubercles, rupioid lesions and fungating and granulomatous ulcerations on the trunk and extremities. The eruptions commenced a few weeks after the administration of three doses daily of 5 grains of potassium iodide and 1/60 grain of arsenic. This was continued for four months, and the patient died from profound iodide intoxication four months later. Large quantities of iodides were found in the urine during the month preceding the death, and at the post-mortem examination, in the skin, liver and kidneys.

Fatal Dose.—Twenty grains of iodine proved fatal to a child of four years,[2] and one drachm of the tincture containing less than 2 grains of iodine killed an adult,[3] but recovery has followed the doses of one fluid ounce and a half-glass[4] of the tincture.

The fatal dose of potassium iodide is uncertain. Six grains taken four times a day proved fatal in 8 days to a patient suffering from renal and cardiac diseases.[5] An elderly woman who was being treated for chronic bronchitis died suddenly after taking three doses of a mixture containing 10 grains of potassium iodide per dose. There were acute œdema and ulceration of the glottis and acute œdema of the lungs.[6]

Fatal Period.—The average fatal period is twenty-four hours, but in cases of poisoning by local application death may be delayed for some days. Culpepper[7] reports the case of a boy, 11 years old, who was poisoned by absorption of iodine from a raw surface extending on both legs from the knees to the feet and died on the sixth day. A case is, however, recorded in which a young woman died in 24 hours from gangrene of the left tonsil caused by two applications of old tincture of iodine to the throat.[8]

Teatment.—Evacuate the stomach by emetics, or wash it out with water containing starch and albumen or a 5 per cent solution of sodium thiosulphate. Give alkalies, arrowroot and barley water, and treat the symptoms. Tracheotomy may have to be performed if death is threatening from œdema of the glottis. A case of a woman, 62 years old, is recorded in which tracheotomy was performed owing to œdema glottidis brought on after a total of 25 grains of iodine taken in two days. She had also œdema of the pharyngeal mucous membrane and swelling of both submaxillary glands.[9]

In poisoning by potassium iodide stop its administration, and give large doses of bicarbonate of sodium or sulphanilic acid, or lessen the dose or double it.

1. *Arch. Derm. and Syph., Nov.*, 1931, *p.* 745 ; *Brit. Med. Jour., Epit., Jan.* 16, 1932, *p.* 12.
2. *Gairdner, "Essay on Effects of Iodine"*, 1824, *p.* 20.
3. *Jackson, Prov. Med. Jour., June* 10, 1847, *p.* 356.
4. *Bellot, La Med. Moderne, Paris*, 1893 ; *Peterson, Haines and Webster, Leg. Med. and Toxic., Ed. II, Vol. II, p.* 167.
5. *Wolf, Berlin Klin. Wchnschr.*, 1886, *XXIII, p.* 578 ; *Webster, Legal Med. and Toxic.*, 1930, *p.* 386.
6. *Sydney Smith, Forens. Med., Ed. VI, p.* 481.
7. *Therap. Gazette*, 1888, 3 *S., IV, p.* 225.
8. *Jour. Amer. Med. Assoc., March* 11, 1922, *p.* 746.
9. *Snell and Suvin, Lancet, April* 9, 1927, *p.* 759.

Post-mortem Appearances.—The gastro-intestinal mucous membrane is inflamed, excoriated and may be coloured brown. The stomach contents may be coloured blue owing to the presence of starchy food. The heart, liver and kidneys may show fatty degeneration.

Chemical Analysis.—If iodine is present in a free state in organic mixtures, it may be extracted by agitating it with chloroform or carbon bisulphide, and then obtained by evaporation and sublimation. If in combination, nitric acid may be added and then iodine may be extracted as above.

Tests.—Free iodine is recognised by its peculiar odour, the violet colour of its vapour, and by its turning starch paper blue.

Iodides produce a flocculent, whitish-yellow precipitate with a solution of silver nitrate, insoluble in ammonia, but soluble in potassium cyanide. A solution of mercuric chloride produces a scarlet precipitate, soluble in excess of either. Mixed with chlorine water and starch, a blue colouration is formed, which disappears on heating but reappears on cooling.

BORON

Boracic Acid or Boric Acid (Acidum Boricum, B.P.), H_3BO_3.—This occurs in powder or in white, pearly lamellar crystals. It is feebly acid and soapy or greasy to the touch, and is slightly acid and bitter in taste. It is soluble in 25 parts of cold water, in 3 parts of boiling water, in 4 parts of glycerine and in 30 parts of alcohol. The dose is 5 to 15 grains. It is largely used as an antiseptic in surgical practice, and commercially as a preservative of milk and other articles of food.

Boric acid is a natural constituent of vegetable food products. Mr. A. Scott Dodd has found an amount of boric acid varying from 0.010 to 0.022 per cent in currants, raisins, muscatels, and sultanas. The amounts varying from 0.003 to 0.025 per cent are present in such dried fruits as apricots, dates, cherries, figs, peaches, prunes, pears and apple-rinds. A. Hebrebrand has found minute quantities of boric acid in the juice of oranges and lemons. J. T. Dunn and H. C. L. Bloxham have found it in quantities varying from 0.005 to 0.003 per cent in the peel, and from 0.002 to 0.008 per cent in the pulp, of oranges from California, West Indies and South Africa. Wines have been found to contain less than 0.01 per cent acid boric.[1]

Borax ($Na_2B_4O_7$, $10H_2O$).—This salt is also known as sodium pyroborate, sodium biborate or sodium borate, and is known in the vernacular as *shohaga* or *tankankhar*. It is used as a preservative of food, and also as an abortifacient. The pharmacopœial preparation is called borax purificatus, which occurs as transparent, colourless crystals, having a sweetish taste. It is soluble in 25 parts of cold water, in equal parts of glycerine, but insoluble in alcohol. The dose is 5 to 15 grains.

The adulteration of milk and other food articles with these drugs is regarded as noxious and injurious to health, though there are no ill-effects from swallowing them in small doses.

Accidental cases of poisoning have occurred from the application of boracic acid on raw and abraded surfaces, or from washing out the abscess cavities or the stomach, rectum or bladder with the acid. John Birch[2] cites the case of an infant, 18 days old, who died after the application to the mouth of at least 2 ounces of borax and honey to prevent thrush.

Symptoms.—The chief symptoms are loss of appetite, nausea, vomiting, diarrhœa and suppressed or scanty urine. There are erythematous eruptions on the skin, and the symptoms of collapse are soon evident. Death occurs from paralysis of the heart. Sometimes, delirium and hallucinations appear.

Boracic acid and borax, when taken internally, are easily absorbed and are rapidly excreted by the kidneys, over half the quantity appearing in the urine within

1. *Lancet*, *Feb.* 23, 1929, *p.* 405.
2. *Lancet*, *Feb.* 11, 1928, *p.* 287.

the first twelve hours, but afterwards the excretion is slow, and the remaining quantity is not completely eliminated for five days or more. They are also excreted to a slight extent in the saliva and milk.

Fatal Dose.—Uncertain. Five grain-doses of boric acid administered internally for a chronic bladder ailment have produced poisonous symptoms.[1] A teaspoonful of boric acid caused the death of a woman, aged 70, in 46 hours.[2] Fifteen grammes proved fatal in one case.[3] Thirty grammes of boric acid dusted over a chronic ulcer from a severe burn of the abdomen and thigh killed a girl, aged 4 years, in 5 days.[4]

Fatal Period.—The usual fatal period is three to four days. Death, however, occurred in twenty-four hours in the case of a young pregnant woman, who swallowed boric acid with intent to procure abortion.[5] A painter, aged 66, took about an ounce of borax in mistake for a proprietary saline cathartic, and died in three hours.[6]

Treatment.—Wash out the stomach, give saline purgatives, treat the symptoms and combat the collapse.

Post-mortem Appearances.—Congestion of the stomach with several spots of erosions on its mucous membrane. The abdominal organs are congested and inflamed. There may be ecchymosis on the inner surface of the pericardium. In the fatal case of borax mentioned above the post-mortem examination revealed nothing, but the chemical analysis of the stomach contents demonstrated its presence.

Chemical Analysis.—Borax or boracic acid can be separated from organic mixtures by evaporating them with sulphuric acid, extracting them with alcohol, or by drying the material, fusing the residue with sodium carbonate and nitrate, and testing the resultant for borates.

Tests.—1. Barium nitrate solution yields a white precipitate, soluble in dilute hydrochloric acid or nitric acid.

2. Silver nitrate gives a white precipitate in strong solutions but brown in dilute ones.

3. If alcohol is added to the solution to which concentrated sulphuric acid has been added and ignited, it will burn with a green flame.

A solution in dilute hydrochloric acid imparts a rosy colour to a turmeric paper dipped into it. The colour changes to bluish-green on addition of caustic potash or ammonia.

1. *Glaister, Med. Juris. and Toxic., Ed. VI, p.* 598.
2. *Sinigar, Lancet, Aug.* 4, 1917, *p.* 162.
3. *Schwyzer, New Yorker Med. Monatschr.*, 1895, *VIII*, 264 ; *Witthaus, Med. Juris. and Toxic., Vol. IV, p.* 11.
4. *Gissel, Zentralbl. f. Chir.*, 1933, *LX, pp.* 1635-1638 ; *Med.-Leg. and Criminological Rev., July*, 1934, *p.* 275.
5. *Brit. Med. Jour., Dec.* 7, 1907, *p.* 1695.
6. *Caryl Potter, Jour. Amer. Med. Assoc., Feb.* 5, 1921, *p.* 378.

CHAPTER XXV

IRRITANT POISONS—(*Contd.*)

II. METALLIC POISONS

ARSENIC

Metallic arsenic is not poisonous, as it is insoluble in water and therefore incapable of absorption from the alimentary canal, but it oxidises by exposure to the air, and then becomes poisonous. It is believed that some portion of elementary arsenic may undergo oxidation in the alimentary canal under some conditions and may produce poisonous symptoms. When rubbed on the skin in a finely powdered state it acts as a poison, as it is capable of being absorbed in the form of an oxide.

When volatilised by heat, it readily unites with oxygen of the air, forming the poisonous vapour of arsenic trioxide. The vapours emanating during smelting of arsenic ores are destructive to vegetation and animal life, and cause chronic injurious effects to smelters.

COMPOUNDS OF ARSENIC

Arsenious Acid (Arsenious Oxide, Arsenic Trioxide or Arsenious Anhydride), As_4O_6.—This is commonly known as *white arsenic* or merely as *arsenic*. It is called in the vernacular *Sankhya* or *Somalkhar*. It is sold as a white, gritty, crystalline powder, or in the form of a solid mass or cake. The mass first appears transparent and crystalline, but after some time becomes white and opaque, having a porcelain-like appearance.

Arsenious acid is odourless and tasteless, but it is, sometimes, described as having a roughish taste due to mechanical irritation of the tongue caused by the gritty character of the powder. If heated on charcoal it is reduced to metallic arsenic, which, in a vaporous form, has an odour of garlic, and a very faint sweet taste. It is a pharmacopœial preparation, and is called arseni trioxidum, the dose being 1/60 to 1/12 grain. It is almost insoluble in water, one-half to one grain dissolving in one ounce of cold water, and twelve to sixteen grains in one ounce of water kept boilig for an hour.

Arsenious acid is a heavy substance, its specific gravity being 3.699. A tea-spoonful containing finely-powdered arsenious acid weighs 150 grains, a table-spoonful weighs 350 grains and a pinch or the quantity taken up between the finger and the thumb of an adult weighs 17 grains.[1] In spite of this heavy weight powdered arsenic has the curious property of floating on water as a white film. If stirred up a good deal the film disappears, but reappears on standing. It is soluble in spirits and wines in the same proportion as in water, but is much more soluble in acids and alkalies. It is also soluble in about 8 parts of glycerine.

1. *Taylor, On Poisons, Ed. III, p.* 291.

Arsenious acid is largely used in the arts, in calico-printing, in the preparation of wall papers, artificial flowers and taxidermy, and as a mordant in dyeing. It constitutes a principal ingredient of fly papers, and many powders and pastes used for killing rats and vermin, and is an adulterant of "complexion or violet powders." In India, it is used for preserving timber and skins against white ants. It is not unfrequently used by *hakims* and *vaids* in the treatment of certain diseases, such as fevers, rheumatism, skin diseases, syphilis, and impotence.

Arsenites.—These are formed when arsenious acid combines with alkalies and their carbonates or with other metals. The alkaline arsenites thus formed are soluble salts. The arsenites that are commonly used as poisons are—

1. **Potassium Arsenite** (K_3AsO_3), **and Sodium Arsenite** (Na_3AsO_3). —These are both poisonous, and are used in manufacturing fly papers, sheep-dips and weed-killers. Potassium arsenite is also used in medicine, and is known in the British Pharmacopœia as Liquor Arsenicalis or Fowler's solution, the dose being 2 to 8 minims. It is prepared by heating arsenious anhydride and potassium carbonate in water and is coloured with compound tincture of lavender.

2. **Copper Arsenite (Scheele's Green)**, $CuHAsO_3$ **and Copper Aceto-arsenite (Paris Green, Schweinfurt Green, or Emerald Green)**, (*Hirwa*), $3\,Cu\,(AsO_2)_2, Cu\,(C_2H_3O_2)_2$.—These are used for colouring artificial flowers, wall papers, articles of dress, toys and sweetmeats. They are insoluble in water, but soluble in acid juices of the stomach. Chevers[1] reports two cases of chronic poisoning caused by sleeping in rooms papered with aceto-arsenite of copper. M. Elzas describes a case of chronic arsenic poisoning in which a man of 30, healthy until 1918, suddenly developed polyneuritis, œdema and eruption, and 0.0005 gramme of arsenic per thousand was detected in the urine. He died some months after he had married and had his house repapered. Three milligrammes of arsenic per square meter were found in one of the wall papers.[2] In his annual report for 1927 the Chemical Analyser, Bombay, cites the case of a man who committed suicide by taking Scheele's green, and no less than 69 grains were found in the contents of the stomach.

Arsenic Acid, H_3AsO_4.—This is less poisonous than arsenious acid. When deprived of water by heating it changes into a white amorphous powder, known as arsenic anhydride or arsenic pentoxide. It is used in manufacturing dyes and fly papers.

Sodium or Potassium Arsenate, Na_3AsO_4 or K_3AsO_4.—This is formed by the action of arsenic acid on sodium or potassium. It is used as a homicidal and cattle poison.

A case is recorded in which potassium arsenate was maliciously put into a bottle of wine. A mouthful of wine was swallowed and it produced poisonous symptoms.[3] Two cases of poisoning by arsenate of sodium are reported. It was given by a druggist

1. *Med. Juris., p.* 125.
2. *Nederlandsch Tijdschrift* v. *Ganeeskunde Amsterdam, April* 16, 1921, *p.* 2145; *Jour. Amer. Med. Assoc., July* 16, 1921, *p.* 241.
3. *Jour. de Chimie Medicale,* 1854, *p.* 254.

to two young men in mistake for tartrate of sodium. In five minutes after the substance had been taken, they were attacked with cramps in the stomach. One died and the other recovered after he remained for some time in a critical condition.[1]

Anhydrous sodium arsenate (*Sodium arsenas anhydrosus*) is a white powder, soluble in water. It is a non-official preparation, the dose being 1/40 to 1/10 grain.

Arsenic Sulphides.—These are found naturally as ores of arsenic, the chief being *realgar* or *red arsenic* (*mansil*), As_2S_2, and *orpiment, yellow arsenic* or *king's yellow* (*hartal*), As_2S_3. Both these varieties are used as pigments in the arts. Mixed in two parts of quicklime the yellow variety is commonly used as a depliatory by Indian women.

Both the sulphides in the pure form, being insoluble, are said to be non-poisonous but, in the commercial form are invariably found to contain a large proportion of arsenious acid, which renders them poisonous.

Arsenic Trichloride, $AsCl_3$.—This is formed by burning arsenic in chlorine or by the action of hydrochloric acid on arsenious acid. It is a highly poisonous, colourless, fuming liquid, and is used in the treatment of cancerous tumours.

Arsenic Tri-iodide (Arsenious Iodide), AsI_3.—This is obtained by heating a mixture of iodine and arsenic. It occurs in small organe-coloured crystals or crystalline masses, and is used for skin affections in 1/16 to $\frac{1}{4}$ grain-doses. It is soluble in water, in alcohol, in chloroform, in ether and in carbon bisulphide. It is contained to the extent of 1 per cent in the official preparation, Liquor *Arseni et Hydrargyri Iodidi* (*Donovan's solution*), the dose being 5 to 15 minims.

Arseniuretted Hydrogen (Arsenic Hydride, Arsine), AsH_3.—This is formed by the action of nascent hydrogen on soluble arsenic compounds. It is a colourless gas, having a fœtid odour of garlic. It burns with a bluish-white flame, forming water and white fumes of arsenious oxide. It acts as a deadly poison, its discoverer Gehlen having been killed on the ninth day after inhaling a small quantity of the pure gas.

ORGANIC COMPOUNDS OF ARSENIC

The most important organic compounds of arsenic which are used in medicine are cacodylic acid, atoxyl, salvarsan, neosalvarsan, silver salvarsan and sulpharseno-benzene.

Cacodylic Acid (Dimethyl-arsenic Acid), $As(CH_3)_2O_2H$.—This is a white, crystalline substance, readily soluble in water and alcohol and forms salts known as cacodylates, when it unites with metals and organic substances. These salts are supposed to be non-toxic, though Murrell[2] affirms that the symptoms resulting from the administration of sodium cacodylate (dose, $\frac{1}{2}$ to 1 grain) are far more severe than those which follow the exihibition of arsenic in its ordinary forms. Professor Fraser of Edinburgh,[3] on the other hand, assumes from his clinical observations and

1. *Amer. Jour. Med. Sci., Oct.*, 1852, *p.* 553.
2. *Brit. Med. Jour., Jan.* 12, 1901, *p.* 120.
3. *Ibid., March* 22, 1902, *p.* 713.

chemical analyses that " when a salt of cacodylic acid is administered to a patient, it is absorbed and eliminated, but with the other constituents that it does not become dissociated, and is therefore incapable of forming any compound in the body which can produce the well-known pharmacological activities of the compounds of arsenic usually employed for therapeutic purposes."

Atoxyl (Sodium para-aminophenylarsonate).—It is also known as soamin or arsamin, and is a white, crystalline, inodorous powder with a slightly saline taste. It is soluble in about six parts of water and dissolves freely in hot water with neutral reaction. It is soluble in 125 parts of alcohol (90 per cent), and is easily soluble, when anhydrous, in methyl alcohol. It usually contains about 24 per cent of arsenic. It is an official preparation, the dose being 1 to 3 grains by mouth or hypodermically dissolved in water. It must be used with caution, as it may cause blindness due to optic atrophy. It has even caused death. A man received 2.4 grammes in four hypodermic injections within eight days and died from pulmonary œdema on the second day after the last injection.[1]

Sodium acetarsenate (arsacetin) is synthetised from atoxyl by the introduction of an acetyl radicle, and may be used in the same doses as atoxyl, but it is less poisonous.

Salvarsan (Dioxy-diamino-arseno-benzol Di-hydrochloride, Arseno-benzol B.P., " 606 ", Kharsivan or Arsphenamine).—It is a pale yellow, crystalline, odourless powder, slowly dissolving in water with acid reaction. It is hygroscopic, and decomposes readily by exposure to the air. It is soluble in glycerine, and dissolves in three parts of methyl alcohol, but is insoluble in ether. It contains not less than 30 per cent or more than 34 per cent of arsenic.

Neosalvarsan (Dioxy-diamino-arseno-bezene-sodium mono-methane-sulphinate, Novarsenobenzol, Novarsenobillon, " 914 ", Neokharsivan, Neoarsphenamine or Neoarsphenamina (B.P.)).—It is a yellow powder, readily dissolving in water with neutral reaction. It readily changes in the air, becoming highly poisonous. It is, therefore, supplied in sealed glass ampoules. It contains 20 per cent of arsenic.

Silver Arsphenamine (Silver Salvarsan).—It is a dark brown powder, soluble in water and of alkaline reaction. It contains about 20 per cent of arsenic and 15 per cent of silver. The dose is 0.1 to 0.3 gramme intravenously.

Sulpharsphenamina (Sulpharsenobenzene).—It is a yellow powder, dissolving readily in water. It contains from 19 to 22 per cent of arsenic. It is administered subcutaneously or intramuscularly in doses of from 0.1 to 0.6 gramme.

PROPRIETARY ARTICLES CONTAINING ARSENIC

1. **Rough on Rats.**—A greyish powder consisting of white arsenic and barium carbonate. Strength, 98.89 per cent of arsenious oxide.

1. *Munch. Med. Wchnschr.*, 1909, 56, *p.* 972.

2. **Fly Papers.**—Strength varying from half a grain to one grain of arsenious acid per each paper. Fly papers were soaked in tea and administered by Mrs. Maybrick to her husband who died from slow arsenical poisoning in about a month.[1]

3. **Weed-Killer.**—This consists of a strong solution of caustic soda and arsenite of sodium. Strength, 14 to 40 per cent of arsenious acid. In 1891, a man died and four members of his family were made seriously ill by drinking gooseberry wine stored in a cask in which a weed-killer had been sent out.[2] Each fluid ounce was found to contain nearly 6 grains of arsenious acid. At the Carmarthan Assizes in November, 1920, Mr. H. Greenwood was charged with having murdered his wife by administering Eureka weed-killer, a pink powder containing 60 per cent of arsenic, in red wine but was acquitted by the jury.

4. **Fly-Water.**—This consists of one part of arsenite of sodium or potassium, two parts of sugar and twenty parts of water. It is used for killing flies. Paper dipped in this solution, and dried, is also used for this purpose.

5. **Fly-Powder.**—This is a mixture of metallic arsenic and arsenious acid. It contains from 4 to 11 per cent of arsenious acid. A case is reported in which a man administered it to his wife with homicidal intent. The woman died in six days.[3]

6. **Sheep-Dip.**—This is used to destroy parasites in wool. It is sold in packets in the form of a yellow granular powder containing about 20 per cent of arsenious acid. It is prepared by mixing arsenious acid and potassium or sodium carbonate with soft soap and ground sulphur. It is made into a solution by mixing it with tar water. A case is recorded in which a woman took a quantity of sheep-dipping liquid and died in 24 hours.[4]

Symptoms.—In cases of acute poisoning the symptoms usually appear within half an hour, but they may be delayed for several hours, especially in those cases where arsenic enters the system by channels other than the mouth, *e.g.*, by its application to the skin or to ulcerated or diseased surfaces. The patient first of all complains of a feeling of faintness, depression and nausea, and then severe burning pain in the throat and stomach which increases on pressure. Intense thirst and severe vomiting are constant symptoms. The vomited matter at first contains the ordinary contents of the stomach, but later on contains mucus and blood in streaks or in spots. The colour is dark brown, yellow, green, or bluish, on account of yellow sulphide of arsenic, or indigo of arsenic, being mixed with bile. Rarely, vomiting may be absent. Robertson Milne describes the case of a Mahomedan male who after a meal took in mistake for chalk a tola of white arsenic. He had marked salivation and burning pain in the stomach but no vomiting. He passed two or three motions, became rapidly unconscious and died in 2 hours and 35 minutes.[5]

1. *R.* v. *Maybrick, Liverpool Summer Assizes,* 1889.
2. *Taylor, Princ. and Pract. of Med. Juris., Ed. IX, Vol. II, p.* 481.
3. *Taylor, On Poisons, Ed. III, p.* 350.
4. *Ibid., p.* 342.
5. *Ind. Med. Gaz.,* 1902, *p.* 209.

Purging is usually accompanied by tenesmus, pain, and irritation about the anus. The stools are expelled frequently and involuntarily, and are dark coloured, fœtid and bloody, but later become colourless, odourless and watery, resembling the "rice water stools of cholera." The urine is suppressed or scanty and contains blood. There is pain in micturition. There may be severe cramps in the calf muscles, as well as other muscles, which usually commence with purging. The patient becomes restless, greatly prostrated and passes into a state of collapse. The surface becomes cold and clammy, and the face is pale and anxious but later becomes cyanosed. The eyes are sunken. The pulse is feeble, irregular and frequent. The respirations become laboured. Lastly, convulsions and coma precede death. The intellect generally remains clear to the end.

When a very large dose is taken death may occur rapidly from shock without producing any symptoms. On the other hand, a large quantity often causes intense vomiting which expels the arsenic from the stomach before it is absorbed and thus the patient's life is saved. Several such cases[1] had occurred in the Punjab during 1925.

Narcotic Form.—In this form the gastro-intestinal symptoms, if present at all, are very slight. The patient complains of giddiness, formication and tenderness of the muscles, and becomes delirious, but soon passes into a state of coma, and dies without regaining consciousness. The pupils are dilated. Sometimes, there is complete paralysis of the extremeties.

At about 8 p.m. on the 24th July, 1906, a convict compounder in Port Blair was found to be groaning and having violent spasms, being quite unconscious, just after he went to bed after taking some milk. This condition lasted only a few minutes after which he died. There was no vomiting or purging. The post-mortem examination showed inflammation of the mucous membrane of the stomach and small intestine with large patches of punctiform hæmorrhages. The large intestine was healthy and contained semisolid fæces. Arsenic was detected in the viscera.—*Bengal Chemical Examiner's Report,* 1906; *Ind. Med. Gaz., Oct.,* 1907, *p.* 393.

Sub-Acute Form.—This is the condition which usually results when the poison is administered in small doses at repeated intervals with the object of causing death by gradual prostration. The symptoms are first dyspepsia, cough and tingling in the throat, then vomiting, purging with abdominal pain and tenesmus, foul tongue, dry and congested throat, and a feeling of depression and languor. The motions are bloody. The symptoms of neuritis are more pronounced. The patient complains of severe cramps in the muscles, which are extremely tender on pressure. He is very restless and cannot sleep. Ultimately collapse sets in, and results in death. In cases which end in recovery, chronic peripheral neuritis may persist, ending in paralysis from degeneration of the nerves extending up to the nerve centres.

Unusual Symptoms.—These are convulsions, lock-jaw, delirium of a maniacal character, rise of temperature, salivation, loss of speech, ringing in the ears, and disordered vision with intolerance to light. Death occurs from asphyxia.

In poisoning by arseniuretted hydrogen the symptoms are nausea, vomiting, burning pain in the abdomen, jaundice, intense headache, feeling

1. *Punjab Chemical Examiner's Annual Report,* 1925, *p.* 7.

of faintness and giddiness, pain in the kidneys with the passage of hæmoglobin in the urine, cyanosis and collapse. Death occurs from œdema of the lungs or sudden failure of the heart.

Fatal Dose.—Three grains of arsenious oxide is the average fatal dose. Two grains is the smallest amount known to have caused death. Half-a-fluid ounce of Fowler's solution, equal to two grains of arsenic, taken in divided doses during a period of five days has proved fatal to a woman.[1] Two ounces of fly-water containing two grains and a half of arsenic have caused the death of a strong, healthy girl, aged nineteen years.[2] Recovery has taken place after much larger doses, varying from sixty grains[3] to two ounces,[4] but these are exceptional cases.

The fatal dose of arseniuretted hydrogen is uncertain. An exposure to a concentration of 50 parts of this gas in a million parts of air for one hour is dangerous to adults,[5] while an exposure to a concentration of 250 to a million parts of air for half an hour is fatal.[6]

Fatal Period.—The average fatal period is twelve to forty-eight hours, though death has frequently occurred within two to three hours. The shortest period is twenty minutes[7] in one case, forty-five minutes[8] in another case and one hour[9] in a third case. In these cases it appears that death occurred from shock before the poison was absorbed into the system. In mild or sub-acute cases life may be prolonged for several weeks. In one case after a dose of 180 grains of white arsenic death did not occur until three months and seventeen days.[10] In such cases the symptoms of gastro-intestinal irritation subside, and are usually followed by nervous affections.

Diagnosis.—Arsenic poisoning has to be diagnosed from—

1. **Acute Gastritis, Enteritis, and Gastro-enteritis.**—In these cases the history and presence of some disease or some cause to account for the symptoms will help the diagnosis. There will be no pain in the throat.

2. **Asiatic Cholera.**—The usual excuse given to account for the symptoms of arsenic poisoning is an attack of Asiatic cholera.

The differentiating points between arsenic poisoning and Asiatic cholera are given below in a tabulated form :—

1. *Castle, Prov. Med. Jour., June* 28, 1848, *p.* 347.
2. *Letheby, Lancet, Jan.* 2, 1847, *p.* 44.
3. *Med. Gaz., Vol. II, p.* 771, *and Vol. XIX, p.* 258.
4. *Lancet, Oct.* 21, 1852, *p.* 299.
5. *Kohn and Abrest, Ann. des Falsifications,* 1915, *VIII, p.* 215 ; *Ralph W. Webster, Leg. Med. and Toxic.,* 1930, *p.* 585.
6. *U. S. Bureau of Mines, Technical Paper, No.* 248, 1921, *p.* 67 ; *Ibid.*
7. *Thompson's case quoted by Taylor, On Poisons, Ed. III, p.* 308, *but this is a doubtful case.*
8. *Madras Chemical Examiner's Annual Report,* 1929, *p.* 2.
9. *Finlay, Lancet,* 1883, *Vol. II, p.* 643.
10. *G. St. George, Brit. Med. Jour., Feb.* 5, 1921, *p.* 192.

Symptoms.	Arsenic poisoning.	Cholera.
1. Pain in the throat.	Before vomiting.	Not so.
2. Purging.	Follows vomiting.	Usually precedes vomiting.
3. Stools.	High coloured, bloody, fæculent, fœtid, and discharged with straining and tenesmus. Very rarely "rice-water."	"Rice-water," liquid, whitish, and discharged in an almost continuous and involuntary jet.
4. Voice.	Not affected.	Peculiar, rough and whistling.
5. Conjunctivæ.	Inflamed.	Not so.
6. Post-mortem venous congestion.	Not present.	Usually present.
7. Blood.	Thicker and contains clots.	Liquid.

3. **Cholera Morbus.**—This occurs in summer and autumn, and is followed by the indiscreet use of some dish, but does not necessarily attack all or several persons partaking of the dish, which excites the outbreak. There are no throat symptoms. It is fatal only among very young and very old individuals, death occurring in three or four days from exhaustion.

4. **Peritonitis.**—The initial pain in the abdomen is not localised in the stomach, but elsewhere. Constipation is a constant symptom. The throat symptoms are absent and tympanites is present obscuring the liver and spleen dullness.

Treatment.—1. It should be remembered that when taken in a finely powdered state on an empty stomach, arsenic sticks to the mucous membrane of the stomach, excites violent inflammation and the formation of a tenacious, which glues it to the surface, and protects it from the action of both emetics and antidotes.

The first step in the treatment is to remove the poison as promptly as possible from the stomach. If the stomach is full, *i.e.*, if no vomiting has occurred, empty it by giving emetics, but do not use tartar emetic, or copper sulphate. If not, wash out the stomach by passing the stomach tube, preferably with large draughts of warm milk and water, and then pour into it through the tube a freshly prepared solution of hydrated ferric oxide, which will convert the arsenious acid into ferric arsenite, a harmless and insoluble salt. It is prepared by adding an alkali (half an ounce of strong ammonia or potassium carbonate dissolved in about half a tumblerful of water) to the tincture of ferric chloride (one and a half ounces of the tincture mixed with a wineglassful of water). The precipitate should be separated from the excess of ammonia by straining through a muslin cloth, and should be given in table-spoonful doses at short intervals, until the symptoms are relieved. Calcined magnesia mixed with an equal quantity of animal charcoal may be administered, if hydrated ferric oxide cannot be obtained.

Dialysed iron in one-ounce doses has been recommended by some as an antidote, but it is not so efficacious as the hydrated ferric oxide.

George B. Lawson, W. P. Jackson and George S. Cattanach[1] recommend the intravenous injection of 7½ grains of sodium thiosulphate in 10 per cent solution. They tried it in 28 cases of poisoning by arsenic after the stomach was washed out with warm water. Of these 15 recovered and 13 died from 6 hours to 13 days.

2. Give demulcents, such as *ghee,* milk, albumen water, or barley water.

3. Give pieces of ice to suck to relieve thirst.

4. Give castor oil to clear the bowels.

5. Give hypodermic injections of morphine to relieve pain.

6. Administer subcutaneous or intravenous injections of normal saline or 5 to 10 per cent solution of glucose in cases of severe diarrhœa.

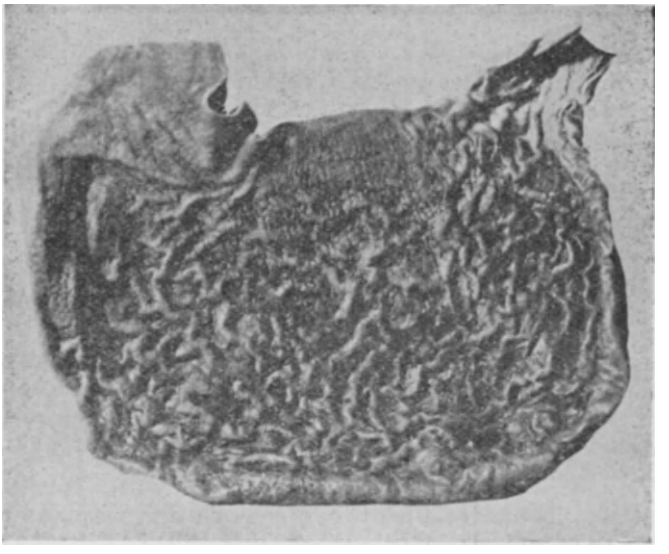


Fig. 129.—The stomach in acute poisoning by Arsenic. (*From the Pathological Museum, Grant Medical College, Bombay*).

7. Use massage to relieve cramps and hot-water bottles to combat collapse.

8. Administer hypodermically strychine or other heart stimulants, if necessary.

The treatment of arseniuretted hydrogen consists in the supply of fresh air, oxygen inhalations, blood transfusion, infusion of salt solution and administration of alkaline drinks to aid its elimination from the blood and tissues.

1. *Jour. Amer. Med. Assoc., July* 4, 1925, *p.* 21.

Post-mortem Appearances—External Appearances.—Rigor mortis lasts longer than usual. The body, sometimes, presents a shrunken appearance. The eyeballs are sunken, and the skin chiefly of the hands and feet is cyanosed, but not so much as in death from Asiatic cholera. The skin may be found jaundiced as happened in a case described by Von Hoffmann.

Inernal Appearances.—The mucous membrane of the mouth, pharynx, and œsophagus is not generally affected, but may occasionally be found inflamed or ulcerated. In the case of Soufflard, who committed suicide with 12 grammes of arsenic, the gums, inner surface of the cheeks, palate, fauces and uvulva were bright red, and the pharynx and œsophagus were found injected.[1] Taylor reports two fatal cases in which the œsophagus was found inflamed.[2] In the case of a man who died after taking two gulps of a weed-killer, the post-mortem examination revealed slight blistering of the lips, and the mucous membrane which was covered with slimy mucus presented patches of injection ("crimson plush") at the lower end of the œsophagus.[3] I found the œsophagus congested and inflamed in a case in which a Mahomedan male committed suicide with arsenic.

The stomach is the chief organ that exhibits characteristic post-mortem changes even if the poison has been administered by means other than the mouth. These changes, however, depend on the quantity of the poison taken, and the time that has elapsed since its administration.

On opening, the stomach contains articles of food in a process of digestion mixed with gritty sandy particles of arsenic, or a dark brown, odourless, turbid and unctuous liquid with crystals of arsenic embedded in large masses of mucus. The inner wall of the stomach, which is swollen, softened and congested, is generally tinged with streaks of blood and white particles of arsenic are embedded in the tough mucus or lymph covering it. On scraping this mucus, the mucous membrane is found highly congested and inflamed wholly or in many small patches, its colour varying from brownish-red or bright scarlet to vermilion. The congestion is due to petechial hæmorrhages from the minute vessels most marked along the crests of the rugæ. Inflammation is more marked at the greater curvature, posterior part and the cardiac end of the stomach. Ulceration of its mucous membrane has been noticed if arsenic is given in a very crude form. Gangrene and perforation have also been observed in rare instances.

The small intestine appears flabby and contains large flakes of mucus with very little fæcal matter. On opening the intestine, the mucous membrane is found finely injected and pale violet coloured, and presents the signs of inflammation with submucous hæmorrhages along its whole length, but more marked in the duodenum and jejunum. These changes are similar to those in the stomach, but less intense. The epithelium is flabby, œdematous and sheds freely.

The large intestine contains a small quantity of seromucus, but more often is empty and contracted. The cæcum and rectum are inflamed, and

1. *Bull, Ac. Med.*, 1838-39, *III, p.* 664.
2. *Guy's Hosp. Rep.*, 1837, *II, p.* 77; 1850-51, 2 *S. VII, p.* 190.
3. *Wilfrid, A. Aldred, Brit. Med. Jour., Sep.* 14, 1907, *p.* 626.

their mucous membrane is flabby. The intestinal glands are often enlarged and swollen, but not inflamed. The peritoneum is congested and pink in colour.

Sometimes, in fatal cases the stomach and intestines may not show any signs of inflammation. Taylor records several cases in which death was caused by arsenic, but no inflammatory changes were visible in the stomach or intestine.[1] Rai Chuni Lal Bose Bahadur reports a case in which a child of eight years died within six hours after taking some molasses mixed with arsenic. On post-mortem examination the stomach was found congested, but the intestines were healthy, and contained semi-solid healthy fæcal matter.[2] A woman died in Agra from the symptoms of irritant poisoning. The post-mortem examination did not show any signs of irritant poisoning, but arsenic was detected in the viscera.[3] In Moradabad a man, aged 50 years, died with the symptoms of irritant poisoning after 6 hours of the onset of the symptoms. The post-mortem examination failed to reveal any definite signs of poisoning but suggested early pneumonia. On chemical analysis the viscera were found to contain arsenic.[4] In an Etawah case two ladies, one aged 22 and the other 70, were found dead in their house at about midnight under suspicious circumstances. On inquiry it was found that some rapidly-acting poison was responsible for the deaths. On analysis the poison found in the viscera of both the ladies was arsenic, the quantity in the case of the young lady being 9.31 grains (of which 9.26 grains were in the stomach), and in that of the other only 0.0076 grain. The post-mortem appearances were not, however, indicative of acute arsenical poisoning. The intestines of both the ladies contained fæcal matter, and the stomach of both contained digested food, it being about one seer (two pounds) in the case of the young lady.[5]

The liver, spleen and kidneys are highly congested, enlarged, and may show signs of fatty infiltration and degeneration.

Arsenic has been known to penetrate through the walls of the stomach, and has appeared on the liver, omentum and endocardium. Rai Chuni Lal Bose Bahadur reports a case of arsenical poisoning in which a deposit of yellow arsenic was found on the internal surfaces of both the ventricles.[6]

The lungs are congested with subpleural ecchymoses.

Both sides of the heart contain loosely coagulated blood, and ecchymoses are often present under the endocardium, and in the muscle of the left ventricle. In a large number of fatal cases of arsenical poisoning I have found petechial hæmorrhages on the internal surface of the pericardium and ecchymosed patches in the endocardium and muscle of the left ventricle. These signs are typical of poisoning by arsenic, although they are, sometimes, found in poisoning by phosphorus and barium and also in deaths from acute infectious diseases, *e.g.*, influenza.

1. *On Poisons, Ed. III, pp.* 302, 303.
2. *Ind. Med. Gaz., Oct.,* 1907, *p.* 393.
3. *United Provinces Chemical Examiner's Annual Report,* 1924, *p.* 5.
4. *Ibid.,* 1925, *p.* 3.
5. *Ibid.,* 1930 ; *Leader, June* 14, 1931, *p.* 14.
6. *Ind. Med. Gaz., May,* 1892, *p.* 142.

In cases where life is prolonged for some time, cloudy swelling and fatty degeneration of the myocardium, liver and kidneys are seen.

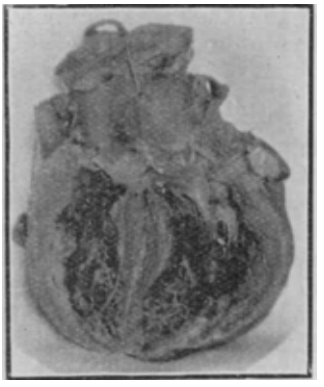


Fig 130.—Heart showing ecchymoses in the left ventricle in acute poisoning by arsenic.

The membranes of the brain are hyperæmic, and the ventricles are full of serum.

In poisoning by arseniuretted hydrogen the post-mortem examination shows a dirty yellow colour of the skin. The mucous membrane of the stomach and small intestine is yellow in colour and may show signs of inflammation. The liver is normal in size or somewhat enlarged, and may show some fatty degeneration. The spleen shows evidence of blood destruction in the deposits of blood pigment throughout the organ. The kidneys are enlarged and congested. The lungs may be œdematous.

Chronic Poisoning.—Chronic arsenical poisoning occurs among persons engaged in works and factories where arsenic in one form or other is used, among persons inhabiting rooms, the walls of which have been painted with arsenical pigments, or papered with coloured papers, or among persons who have been taking the drug for a prolonged period or in too large a quantity.

Symptoms.—The symptoms of chronic poisoning are exhibited in four stages.

First Stage.—The symptoms in the first stage are those of gastric troubles, *viz.*, malaise, loss of appetite, salivation, colicky pain, constipation, or sometimes diarrhœa and vomiting of glairy mucus tinged with bile. The gums are red and soft, and the tongue is coated with a thin, white, silvery fur. The temperature is raised to 102° or 103° F. with a frequent pulse.

Second Stage.—This is marked by cutaneous eruptions and catarrh of the larynx and bronchial tubes. There is a feeling of dryness and itching in the fauces and larynx. Hence the voice becomes hoarse and husky. The eyes are suffused and the conjunctivæ are greatly congested. There is running from the nose with intense coryza. The patient gets spasmodic cough with expectoration tinged with blood on account of inflammation of the bronchial tubes.

Erythematous, eczematous, urticarial or pustular eruptions manifest themselves on the skin, chiefly on the folds of the armpits and groins, as well as on the scrotum. After a certain time the skin becomes pigmented, and the epidermis comes off in desquamations. The nails become brittle and loose. The hair becomes dry and may fall out.

Third Stage.—In this the sensory troubles are more prominent. They resemble those met with in alcoholic poisoning more than in lead poisoning.

The first symptom, which appears from a week to three or four weeks, is headache, followed by numbness, tingling, formication and cutaneous anæsthesia. Perspiration is well-marked. There is extensive tenderness of the muscles of the extremities on pressure and the knee jerk is usually lost. The loss of sexual power is a constant symptom, but the special senses are not deranged.

Fourth Stage.—This is the state of paralysis. In this stage the muscles become weak and feeble, so that the patient gets easily fatigued while walking or ascending a stair-case. He also adopts an ataxic gait when he walks. The extensor muscles of the extremities atrophy ; hence the patient is unable to use his limbs, and becomes bed-ridden ; but the sphincters are rarely affected. Tremors are noticed in the muscles which become markedly paralysed. The interosseous and intercostal muscles are more often affected. These are followed by general emaciation, dysuria, mental hebetude or delusions and death occurs from failure of the heart muscle.

Treatment.—Remove the patient from the source of poison, administer potassium iodide in 5-grain doses and treat the symptoms as they arise. Also inject sodium thiosulphate intravenously every day.

Post-mortem Appearances.—The stomach and intestines present a chronic inflammatory condition but, more often, there may not be any characteristic changes. The liver shows the signs of fatty degeneration, and the kidneys show parenchymatous nephritis. The muscles are greasy and atrophied.

Chemical Tests.—1. Ammonio-nitrate of silver produces a yellow precipitate of arsenite of silver in an arsenious acid solution.

2. Ammonio-sulphate of copper gives a bright green precipitate of arsenite of copper (Scheele's green.)

3. *Reinsch's Test.*—This is a very delicate test, and arsenic may be readily detected to the extent of 1 : 1,000,000 and 1 : 7,000,000 if the solution is concentrated. The method of procedure is as follows :—

Drop one or two strips of bright copper foil into the suspected solution previously acidulated with pure hydrochloric acid and boil it for five to ten minutes, when the copper foil is coated steel-grey with a deposit of arsenic, if present. The foil is then removed, washed successively in distilled water, alcohol and ether, dried on filter paper and then heated by placing it in a small test-tube. The deposit, if due to arsenic, volatilizes and forms a white deposit further up in the cooler portions of the tube. This deposit, when seen under the microscope, shows octahedral crystals of arsenious acid with their apices chopped off. If the coating is very thick it may be scraped off, dissolved in acid and the liquid tests may be applied. Before proceeding with Reinsch's test a control test should be tried to prove the purity of hydrochloric acid and copper foils.

Sometimes even with sufficient amount of arsenic present for its successful detection by Reinsch's test in the normal course, very faint blackening or only a faint dulling of copper is obtained specially in the case of solid tissues, such as the liver, or in the case of earthly matter or ashes containing vomit or stool. In such cases by heating the faintly

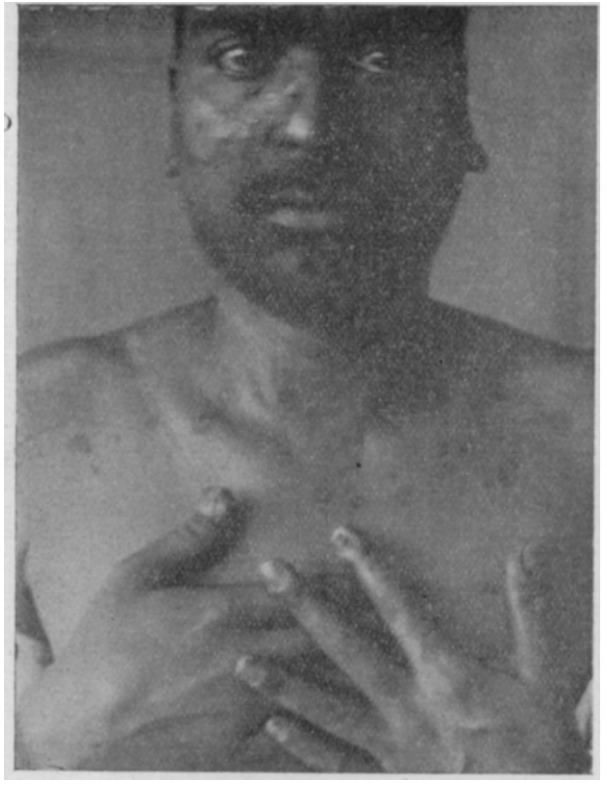


Fig. 131.—Chronic Arsenical Poisoning: Front view.
(*From a photograph kindly lent by Dr. H. S. Mehta*).

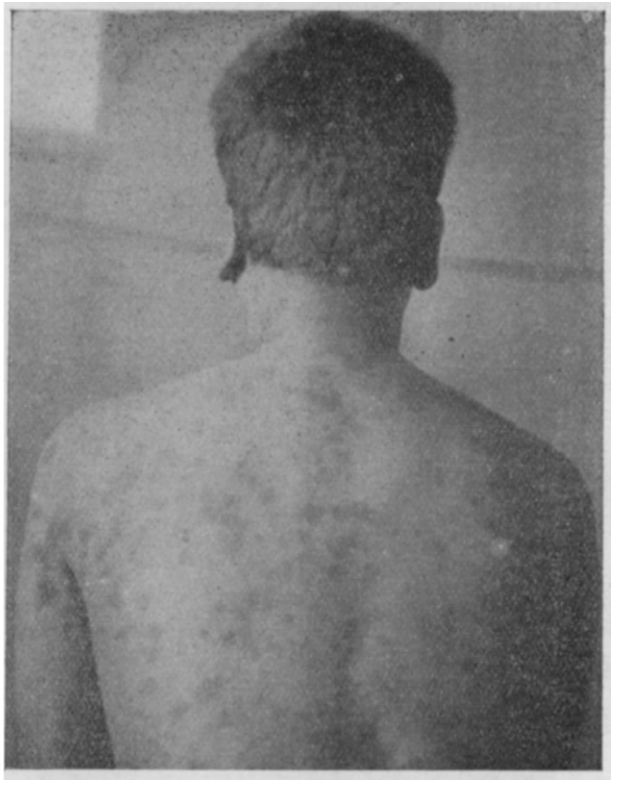


Fig. 132.—Chronic Arsenical Poisoning: Back.
(*From a photograph kindly lent by Dr. H. S. Mehta*).

blackened or dulled copper strips, crystals of arsenious acid are not detected. As the arsenic present in such cases is in a higher state of oxidation or the alkali present in earthy matter, etc., partly or wholly neutralises the acid in the Reinsch's bath the deposition of arsenic is retarded. In such cases the following modification, evolved by Mr. D. N. Chatterji, F.I.C., Chemical Examiner to Governments, United Provinces and Central Provinces, should be adopted:

> Put more hydrochloric acid in the bath and boil with a little sulphurous acid solution (SO_2 water), till sulphurous acid is removed. Then introduce the copper foil. A black deposit on copper is easily obtained. On now heating the copper in the usual way characteristic crystals of arsenious acid in the sublimate are detected.

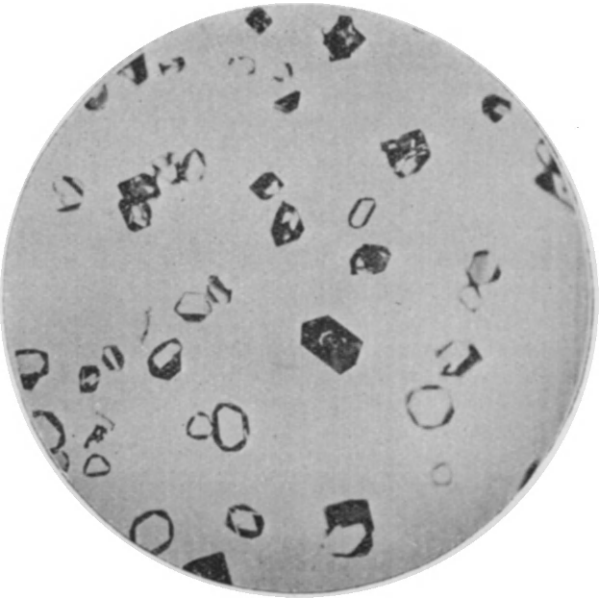


Fig. 133.—Microphotograph of Arsenious Acid Crystals × 300.
(*R. B. Dr. K. N. Bagchi*).

4. *Marsh's Test.*—This forms such a delicate test for the presence of arsenic that exceedingly small quantities even up to one thousandth of a milligramme may be detected.

The test is based on the formation of arseniuretted hydrogen, when the compounds of arsenic except the metal and its sulphides are brought into contact with nascent hydrogen. It is carried out by means of a Woulffe's bottle (hydrogen generating bottle) to which is connected a long glass tube ending in a jet. Granulated zinc and dilute sulphuric acid are dropped into this bottle, when hydrogen will be evolved, and will burn with a pale blue flame on applying a light to it. It must be remembered that the hydrogen is not ignited at once but after about ten minutes, when all the air in the bottle is chased out, otherwise the mixture of hydrogen and oxygen will ignite with a loud explosion and break the apparatus thus

injuring those round about. The other reason for expelling all the air out is that even if traces of air are left behind, when the flame is applied to the tube, water will be formed to the detriment of the arsenical deposit which will appear as a greyish-white cloud.

On adding the suspected mixture of arsenic into the bottle, the flame begins to burn with a bluish- or greenish-violet or purple tint due to the formation of AsH_3 which also emits a garlic-like odour. If a cold porcelain dish be depressed into the flame, a blackish brown stain of metallic lustre is produced. This stain is readily soluble in a solution of calcium hypochlorite ; while the addition of ammonium sulphide does not dissolve, but detaches it from the porcelain, and on heating turns it yellow.

If the deposit be heated with a few drops of strong nitric acid, and if silver nitrate be then added, a brick red (reddish-brown) precipitate of silver arsenate is formed, which is soluble in ammonia.

If the flame be extinguished, and the central part of the tube conveying AsH_3 be heated to redness by means of a spirit lamp for some time, a brilliant arsenical mirror of a darker and less silvery white colour appears immediately beyond the heated spot. If the portion be cut off and heated in a dry test-tube a white deposit is formed on its inside, which shows octahedral crystals under the microscope. Very low mirrors, such as obtained with 0.006, 0.008 and 0.001 mgms. of arsenic trioxide do not give crystals of arsenious acid when heated in dry test tubes. In order to determine whether such mirrors are due to arsenic, the following technique evolved by Mr. K. R. Ganguly, Assistant Chemical Examiner, United Provinces, Agra, may be adopted :

> The two ends of the narrower portions of the Marsh's tubes (used for determination of arsenic by deposition of mirrors) containing the mirror, should be sealed with air instead of hydrogen inside it. The mirror should then be passed gently over a flame several times, until the mirror is visible ; the broader portion of the sealed tube should then be heated to drive the crystals of arsenious acid in the narrower part. After cooling, the characteristic crystals of arsenious acid may be detected under the high power of a microscope.

Commercial zinc and sulphuric acid are often contaminated with arsenic ; hence a control experiment must be made to prove the purity of these reagents, or the exit tube may be heated for at least thirty minutes before any of the suspected fluid is introduced into the hydrogen generating bottle. If the tube remains free from deposit, the purity of the reagents is established.

To obtain pure hydrogen without any trace of arsenic it is better to use hydrogen generated by the electrolysis of water.

5. *Gutzeit's Test.*—One c.c. of the suspected solution is placed in a large test-tube with a piece of chemically pure zinc and a few c.c. of dilute sulphuric or hydrochloric acid containing enough solution of iodine in potassium iodide to colour it yellow so as to remove sulphur dioxide and hydrogen sulphide if formed. A plug of absorbent cotton is inserted in the upper part of the tube, and the mouth is covered with a piece of filter paper moistened with a concentrated solution of silver nitrate (1 : 1). If

arsenic is present the paper is turned yellow owing to the formation of a double compound of silver arsenide and silver nitrate ($AsAg_3$. $3AgNO_3$). On the addition of water the yellow colour becomes black by the separation of silver.

The colour produced by antimony is not yellow, but brown or black. On the other hand, phosphuretted hydrogen produces the same colour as that of arsenic. In order to avoid this fallacy absorbent cotton is moistened with a solution of lead acetate.

6. *Fleitmann's Test.*—If the suspected solution is heated with arsenic-free zinc pieces and strong caustic potash, arseniuretted hydrogen is evolved which blackens a piece of white filter paper moistened with silver nitrate if held over the mouth of the tube.

7. *Biological Test.*—This is also a delicate test, and will detect arsenic to the extent of one thousandth of a milligramme. It is based on the fact that certain moulds, such as penicillium brevicaule, have the property of developing volatile products with arsenic which are known by a garlic-like odour. The test is carried out by putting the suspected substance into a glass flask with some small pieces of bread or biscuit, and by sterilising for half an hour at 120° C. When cold, the mixture is inoculated with a culture of penicillium brevicaule and kept at a temperature of 37° C. If arsenic is present, a garlic-like odour will emanate owing to the formation of arseniuretted hydrogen gas or an organic combination of arsenic.

8. *Dry Test.*—If a mixture containing arsenious acid be mixed with sodium carbonate, and heated on a charcoal support in the inner flame of a blow pipe, a characteristic garlicky odour is given off.

A small quantity of the powder, if heated on a platinum foil, volatilises completely as a white vapour.

Medico-Legal Points.—1. Arsenic is used homicidally much more frequently in India than in any other country, as it is cheap, is easily obtained in every town and is easily concealed in the food in consequence of its freedom from smell and taste. A very small quantity of arsenic is necessary to produce fatal effects, although cases have occurred, where much larger quantities were given for homicidal purposes. In a homicidal case that came under my observation in 1931, 101.6 grains of arsenic were detected in the stomach contents.

Instead of administering a single fatal dose of arsenic at once, the murderer in Western countries usually administers small doses over a long period in order to produce the symptoms simulating gastro-enteritis and thus to hide the crime.

Arsenic is often employed to produce abortion, especially in the form of ointment or paste on abortion sticks. It is also used to poison cattle. Wells are known to have been poisoned by arsenic not only during war, but also in peace times.[1]

1. *Chevers, Med. Juris., p.* 119.

A case occurred at Nagpur, where the accused was stated to have added poison to water as it was being drawn from a well. A quantity of arsenic was found in the water.[1] A bundle of cloth was recovered from a well in the district of Pubna. The cloth contained some dark grey coloured pasty substance which, on analysis, was found to be arsenic.[2]

Arsenic is used occasionally for suicidal purpose, but owing to much pain caused by its ingestion suicides resort to this poison much less frequently than to opium.

Accidental cases, sometimes, occur from its admixture with drink or articles of food, or from its improper medicinal use. White arsenic has been mistaken for baking soda, cream of tartar, sugar, salt and flour. Accidental cases may also occur from drinking water from streams containing arsenical mineral deposits. Accidental deaths occur from an overdose, when it is given by women to their husbands as a love-philter.[3]

Accidental or homicidal poisoning by orpiment is not possible owing to its bright yellow colour, which can be easily recognised. But suicidal poisoning, though rare, is recorded.

In January, 1921, a case came under my observation, in which a Hindu male, 50 years old, committed suicide by taking orpiment. The stomach contained a lot of mucus in which were entangled particles of yellow sulphide of arsenic. The mucus was adherent to the inner wall of the stomach, which was inflamed with bloody patches and ulcerations spread all over the surface, especially at the greater curvature and posterior aspect.

A Mahomedan male, aged about 20, committed suicide by swallowing yellow sulphide of arsenic. About 37 grains of it were found in the stomach after death.[4]

A woman, aged about 25, died in about 6 hours after taking yellow arsenic with a view to commit suicide. About 81 grains of the sulphide were detected in the stomach.[5]

Chronic arsenical poisoning with the symptoms of peripheral neuritis broke up among beer drinkers in an epidemic form in the county of Lancashire in 1900. Beer was found contaminated with arsenic, varying from 0.01 to 0.3 grain or even 1.4 grains per gallon, and derived from impure sulphuric acid used in the manufacture of glucose and cane sugar required for brewing it.[6] A case[7] is also recorded where an outbreak of arsenic poisoning occurred among more than three hundred French officers and sailors by the drinking of wine in February, 1932. The wine on chemical analysis was found to contain sulphurous acid and 3 to 12 mg. of arsenic per litre. It appeared that sulphurous acid might have got into the wine from applying sulphur to the wine casks in the cleansing process. Arsenic appeared to have got into the wine through the grapes being contaminated with arsenic by the spraying of the vines with copper or other arsenic-containing solutions to protect them against insects. It was also possible that arsenic-containing sugar might have been added to the wine.

1. *U. P. Chem. Exam. Annual Rep.*, 1907.
2. *Beng. Chem. Exam. Annual Rep., Ind. Med. Gaz., Aug.* 1915, *p.* 303.
3. *Chevers, Loc. Cit., p.* 118.
4. *Bombay Chem. Analyser's Annual Rep.*, 1935, *p.* 5.
5. *Beng. Chem. Exam.'s Annual Rep.*, 1936, *p.* 14.
6. *Brit. Med. Jour., Feb.* 16, 1901, *p.* 397.
7. *Jour. Amer. Med. Assoc., July* 23, 1932, *p.* 319.

2. **Method of Introduction.**—In most of the homicidal cases arsenic is administered by the mouth after disguising it with articles of food, such as sweetmeat, bread, *dal*, cooked vegetables, and drinks, such as milk, tea, coffee, port wine, or with medicine. It has, sometimes, been given with *prepared pan.* It has occasionally been injected into the rectum after mixing it with a liquid to be used as an enema.

Arsenic has also been introduced into the vagina either for the purpose of committing suicide or for procuring abortion. It has produced poisonous symptoms, when used as an urethral injection.

Cases of poisoning have also occurred from the application of arsenic paste to a cancerous growth, or of its ointment or solution to a blistered or abraded surface, or even to the uninjured skin.

Sometimes, fly-papers or weed-killers are soaked in water, tea or wine and the solution is then administered with homicidal intent.

Salvarsan.—This is used intramuscularly or intravenously to destroy the microbes of syphilis. It forms an acid solution in water, and is, therefore, neutralised by the cautious administration of caustic soda. To obviate this difficulty neosalvarsan is used, which makes a neutral solution in water convenient for intravenous injections. There is no difference in toxic or therapeutic effects between the two drugs. Arsenic is found in the blood soon after the injection of either of them and is rapidly eliminated by the kidneys and bowels, but may be retained for a longer time in the tissues after an intramuscular injection. Thus, arsenic was not detected in the viscera of a female patient who died fourteen days after an intravenous injection, but a large quantity was detected in the gluteus muscle when a post-mortem examination was held on the body of a woman who died thirty-six days after an intramuscular injection.[1]

A slight rise of temperature, headache, giddiness, nausea and slight diarrhœa lasting for a day or two are the only symptoms usually experienced by patients after an intravenous injection of salvarsan or neosalvarsan. More severe poisoning ending in death has, however, ensued after the first injection or after two or three injections repeated at short intervals. The symptoms in such cases are abdominal pain, vomiting, profuse diarrhœa with bloody stools, headache, jaundice, hyperpyrexia, dilated pupils, anuria, cramps, convulsions, coma, collapse and death. Extensive sloughing, abscesses and necrosis of the surrounding tissues may occur at the site of the intramuscular injection. Optic atrophy which is so common in poisoning by atoxyl is not produced by salvarsan.[2] Death occurs among those suffering from visceral and degenerative nerve lesions. It may also occur from faulty technique or from auto-intoxication.

Sometimes, the symptoms may not appear immediately after the intravenous administration of the drug, but may be delayed for some weeks and death may occur from acute yellow atrophy of the liver.

1. *W. Wechselmann, Treat. of Syphilis with Salv., Eng. Trans., p.* 86.
2. *Pract., July,* 1912, *p.* 94.

Policard and Pinard[1] cite a case of syphilis in a man, aged 28 years, who, fifty days after three doses equivalent to 1.05 gramme of neoarsphenamine (neosalvarsan), developed acute yellow atrophy of the liver and died in six days.

Fatal Dose.—The official dose of salvarsan is 0.3 to 0.6 gramme and that of neosalvarsan is 0.15 to 0.9 gramme. The toxic dose for an average rabbit weighing about 2,000 grammes is about 0.3 gramme. The toxic dose for a man weighing, on an average, 12 stone would be about 10 to 15 times the highest therapeutic dose.[2] According to Holland 10.5 grammes might cause death.[3] From his experiments Ehrlich[4] has shown that the *dosis maxima bene tolerata* is six times as great as the *dosis curativa*. Medicinal doses have, however, occasionally caused deaths as mentioned above. 0.4 gramme of salvarsan injected intravenously caused the death of a man, *æt.* 25 years, in twenty-five hours.[5] Neve[6] records the case of a Kashmiri Mahomedan, aged 20 years, who died in 25 hours after the first intravenous injection of 0.5 gramme of salvarsan. A case is recorded in which death occurred in about 2 hours after an intravenous injection of one of the salvarsan derivatives with a 10 c.c. syringe.[7] A woman, 42 years old, suffering from Addison's disease, died in twelve hours after an intravenous injection of 0.15 gramme of neosalvarsan.[8]

Treatment.—Adrenaline injected hypodermically before or after the salvarsan injection has warded off the serious symptoms.[9] Give intravenous or rectal injections of normal saline as well as hypertonic solutions of sodium chloride and sodium bicarbonate to render the blood alkaline and to eliminate arsenic from the system. Inject intravenously sodium thiosulphate in doses of 0.45, 0.6 and 0.9 gramme dissolved in 5 c.c. of water. Administer strychnine hypodermically to combat collapse and use oxygen inhalation.

Bernard Appel[10] states that sodium dehydrocholate in doses of 10 c.c. of a 5% solution administered intravenously combats the toxic action of salvarsan and its derivatives on the liver. In a number of cases in which nausea and vomiting followed intravenous injections of neosalvarsan, the patients were treated by mixing the solution of the drug with a solution of sodium dehydrocholate and injecting the mixture intravenously.

Post-mortem Appearances.—Nothing[11] definite beyond cloudy swelling or fatty degeneration of the liver, kidneys and heart. In some cases hæmorrhage from a cerebral vessel may be found. In other cases there

1. *Paris Medical, Jan.* 8, 1921, *p.* 42 ; *Peterson, Haines and Webster, Leg. Med. and Toxic., Vol. II, Ed. II, p.* 258.
2. *Martindale and Westcott, Extra Pharm., Vol. I, Ed. XVI, p.* 177.
3. *Med. Chem. and Toxic., p.* 288.
4. *Martindale and Westcott, Salv., p.* 18 ; *M. and W. Loc. Cit.*
5. *Nesbitt, Brit. Med. Jour., Feb.* 21, 1914, *p.* 429.
6. *Ind. Med. Gaz., Jan.*, 1915, *p.* 20.
7. *Ibid., Aug.*, 1923, *p.* 386.
8. *Hellfors, Medizinische Klinik, Berlin, Jan.* 20, 1933, *p.* 117 ; *Jour. Amer. Med. Assoc., March* 18, 1933, *p.* 860.
9. *Milian, Brit. Med. Jour., May* 9, 1914, *p.* 1044.
10. *Archives of Dermatology and Syphilology, Chicago, March*, 1933, *p.* 401 ; *Jour. Amer. Med. Assoc., July* 15, 1933, *p.* 242.
11. *Willcox and Webster, Brit. Med. Jour., April* 1, 1916, *p.* 475.

may be the signs of acute encephalitis with hæmorrhagic spots in the brain.

3. **Tolerance.**—Individuals who are in the habit of taking arsenic acquire a certain amount of toleration to bear it up to four grains or more in one dose. They use it daily with the idea of improving their looks, and becoming more hardy to carry weights and to climb mountains. This habit is common among the peasants of Styria and Hungary. The people using this drug as a habit are called *arsenophagists,* and suffer from the symptoms of mild arsenical poisoning if the drug is withheld from them.

In India, some people are in the habit of taking arsenic daily as an aphrodisiac. Sometimes, it is given in small quantities with a view to produce death from slow poisoning, but instead it makes a man plumper and stronger as happened in the case of the late Fulham of Agra who was being poisoned with arsenic by Clark.

Arsenic is largely given by grooms to improve the coats of horses. If it is withheld, the animals become dull and lose flesh.

4. **Solubility of Arsenic.**—When administered in a soluble form by the mouth, arsenic gets absorbed into the blood almost in a few minutes, but when taken in solid lumps it may not be absorbed by the stomach and, sometimes, passes out with the fæces without producing any poisonous symptoms. For instance, in 1872, a Parsee in Bombay had swallowed two masses of arsenious oxide without any serious effects. Within forty-five hours after swallowing the poison he passed per rectum two lumps, one weighing eighty grains and the other weighing twenty-five grains.[1]

Arsenious acid is converted into yellow sulphide of arsenic in the stomach and intestines, but sulphide of arsenic is not converted into white arsenic.

5. **Elimination.**—Arsenic, when taken for some time in medicinal doses, does not accumulate in the system, so that it may give rise to sudden poisonous symptoms. It is, therefore, not regarded as a *cumulative* poison.

Arsenic is eliminated through the urine, fæces, skin, hair and nails, and to some extent through the sweat, saliva, bile, bronchial secretion and milk. After its administration arsenic appears in the urine and fæces usually from two to eight hours, but it may be detected within half an hour after a single dose of five drops of liquor arsenicalis (Fowler's solution).[2] The elimination by these channels continues for a period of two to three weeks, after which arsenic is not found in the urine and fæces, although it may be found in the hair and nails. A case is, however, recorded in which arsenic was detected in the urine ninety-three hours after the administration of a single large dose, which produced the symptoms of acute poisoning followed by paralysis.[3] Willcox[4] reports

1. *Ind. Med. Gaz.,* 1872, *p.* 183.
2. *Dixonmann, Forens. Med. and Toxic., Ed. VI, p.* 379.
3. *Wood, Boston Med. and Surg. Jour.,* 1893, *CXXVII, p.* 414.
4. *Lancet, Jan.* 27, 1923, *p.* 168.

a case in which a Government official of a tropical country was administered arsenic on October 6, 1922, and the chemical analysis of his hair revealed the presence of arsenic on December 19, 1922, when he was suffering from the symptoms of chronic poisoning.

By dividing hair in small successive lengths from the root upwards and analysing them separately one may obtain important information regarding the time that has elapsed since the administration of arsenic. For instance, if arsenic is administered to a patient daily for a few days and then discontinued, the portion of the hair growing during this period yields a much larger amount of arsenic than the portion growing during the non-arsenic period. The time depends upon the rate of the growth of hair which is generally about half an inch per month. Bagchi describes a case in which he was able to show on analysing the distal and proximal ends of the hair that a patient suffering from suspected arsenic paralysis had been given arsenic two to three months before his admission into the hospital in Patna.[1]

In the fatal cases of acute arsenical poisoning where the patient has survived for ten to fourteen days, it is hardly possible to find the poison in the viscera usually preserved for chemical analysis, although arsenic was detected in the viscera of a woman who survived fifty-two days after taking the last dose.[2] On the other hand, a case is reported in which arsenic was found in the vomit and fæcal matter, but was not found in the viscera when death occurred after six days.[3]

Arsenic is excreted into the stomach and intestines after absorption, even when administered by channels other than the mouth. Hence its detection in these organs does not prove that it had necessarily been administered by the mouth. In a case reported by Stich arsenic was detected in the stomach contents of a woman who had been poisoned by the introduction of a large amount in the vagina. In another case of poisoning per vaginam reported by him it was found in the stomach contents and fæces of a woman, and also in the organs of three months' fœtus.[4]

Arsenic becomes fixed in the cancellous tissue of the bones, chiefly the long ones, owing to the conversion of their phosphates into arsenates. Its elimination being much slower, its presence can be detected in the bones long after every trace has disappeared from the other organs, such as the liver, kidneys, etc. Hence it is essential to preserve the long bones for chemical analysis in suspected cases of arsenical poisoning when a body is exhumed, or when it is very much decomposed. Traces of arsenic were found by Dr. Hankin, Chemical Examiner, U.P., in the femurs removed from the body of the late Fulham, which was exhumed in Agra about fourteen months after death. In a case where death occurred within forty hours of the onset of the symptoms of acute poisoning, arsenic was detected in the pieces of femur and the viscera which were sent for chemical analysis.[5]

1. *Patna Jour. of Med., Jan.*, 1936, *p.* 7.
2. *Dixonmann, Forens. Med. and Toxic., Ed. VI, p.* 379.
3. *Sind Chemical Analyser's Annual Report*, 1923, *p.* 23.
4. *Munch. Med. Wochenschr., March* 12, 1901, *p.* 425; *Peterson, Haines and Webster, Leg. Med. and Toxic., Vol. II, Ed. II, p.* 237.
5. *U. P. Chemical Examiner's Annual Report*, 1924, *p.* 5.

6. **Deposit of Arsenic.**—In acute poisoning arsenic, after it is absorbed, gets deposited more in the liver than in the kidneys and spleen, and in chronic poisoning it is also found deposited in the brain, the spinal cord and the muscular and bony tissues. In fatal results occurring from salvarsan poisoning it is interesting to note that arsenic is not found in the brain or nervous system.[1]

7. **Power of Preservation.**—Not only does arsenic not disappear by putrefaction, but it has the power of retarding decomposition to a certain extent, especially in cases of prolonged administration, and the stomach and other tissues are often well preserved some months after death, though this is not always the case. Thus, the body of the late Fulham of Agra was well preserved when it was exhumed some fourteen months after death, even though the grave was a *katcha* one, and the lid of the coffin had already given way.

8. **Is Arsenic a Normal Constituent of the Body ?**—Arsenic is physiologically not a normal constituent of the body, but it is widely distributed in nature. It has been found in minute quantities in several varieties of vegetables[2] and on apples[3] as the result of spraying the fruit trees with arsenical preparations. It has also been shown that arsenic is present in the form of some organic compound in some kinds of fish, such as sole, crustaceans, oysters and other shell-fish.[4] From investigations carried out on Indian food stuffs Bagchi and Bose have been able to show the presence of arsenic in amounts varying from a trace to 6.4 mgms. per kilo in animal foods, such as chicken, goat flesh, beef, beef liver and various kinds of fish and in traces only in vegetable foods, such as rice, wheat flour, *dals* (pulses), potato and all green vegetables, *e.g.*, cauliflower, cabbage, spinach, *patal*, brinjals and lady's fingers.[5] Hence arsenic may be taken into the human economy in very minute quantities along with the articles of food. In some cases it may be absorbed into the system from medicines, water or even air. Recent researches have shown that arsenic derived from the food ingested is found normally in human tissues and excretions, and Billeter and E. Marfurt[6] claim to have found appreciable quantities of arsenic in all the organs examined, and affirm that the body of an adult person contains probably about 0.1 mgm. of arsenic.

From investigations carried out in Calcutta, Bagchi and Ganguli[7] have found that arsenic is present on an average from 0.02 to 0.03 mgm. per litre in the urine and about ten times this quantity in the fæces of persons belonging to different communities who do not expose themselves to any trade or industry connected with arsenic nor take any medicine containing any arsenic preparation. They have also found arsenic in human tissues.

1. *Brit. Med. Jour., April* 1, 1916, *p.* 475.
2. *Jour. Pharm. Chim.*, 1912 ; *Blyth, Poisons, their Effects and Detection, Ed. V, p.* 586.
3. *Willcox, Trans. Med.-Leg. Society, Vol. XXIII, p.* 153.
4. *Swedish Commission, Lancet, Sep.* 8, 1923, *p.* 531 ; *Willcox, Trans. Med.-Leg. Society, Vol. XXIII, p.* 152 ; *Chapman, Analyst, Vol. LI,* 1926. *p.* 548.
5. *Ind. Science Congress Proceedings,* 1935, *p.* 411 ; *Patna Jour. of Med., Jan.,* 1936, *pp.* 7-8.
6. *Helv. Chim. Acta,* 1923, 6, *pp.* 780-784 ; *Jour. of State Med., Jan.,* 1924, *p.* 41.
7. *Ind. Med. Gaz., Aug.,* 1937, *p.* 477.

The liver contains the largest amount (2 mgm. per kilo). Bone and tooth come next. The amount found in the blood is negligible. Fœtal tissues contain no arsenic, while the placenta is fairly rich in arsenic. It is, therefore, necessary that a medical man ought to be very cautious in affirming that death was due to arsenic poisoning in a case where a very small amount, a minute fraction of a grain, is detected in the viscera, unless some of the characteristic symptoms and post-mortem appearances of arsenic poisoning were present. In a murder case where about 1/5th gain of arsenic was found in the viscera of the victim, Justice Young of the Allahabad High Court acquitted the accused on the ground that this amount might be due to the food that the deceased took or that might be the normal arsenic-content of the viscera.

Arsenic is, sometimes, found as an accidental impurity in some medicines, such as bismuth nitrate, sodium sulphate, magnesium sulphate and glycerine. A firm of chemists at Bradford was fined for selling glycerine which was found to contain arsenic to the amount of 1/13 grain to the pound. The Medical Officer of Health for Bradford stated in his evidence that a Royal Commission had recommended local authorities to take action in cases where arsenic was found to exist in glycerine to a greater amount than 1/100 grain to the pound.[1]

9. **Post-mortem Imbibition of Arsenic.**—In a criminal charge of arsenical poisoning the plea is, sometimes, raised by the defence that the poison was introduced into the stomach after death, and the post-mortem imbibition occurred in the tissues. Such a presumption is certainly possible, but the transudation of poison through the organs in such cases seeks an anatomical course; hence the organs of the left side are affected sometime before those of the right. Besides, the fact of ante- or post-mortem inbibition of arsenic can be ascertained by examining the condition of the mucous membranes of the stomach and duodenum. The signs of inflammation and ulceration, being the result of vital processes, will be absent in post-mortem imbibition of the poison.

When arsenic has been found in exhumed bodies a further question may arise as to whether arsenic found in the body has been absorbed from the earth which surrounded the coffin or the body. In this connection it must be remembered that arsenic met within the soil is usually an insoluble salt mixed with lime or iron, hence it is impossible that an insoluble salt should percolate into the cadaver buried in such soil, especially if the body is laid in a coffin. However, to avoid the possibility of any doubt, it is safest, if the body has to be disinterred, to preserve for chemical analysis samples of the earth surrounding the coffin or the body.

Illustrative Cases.—1. A lady had an illegitimate child whom she placed with a nurse. When the child was some weeks old, it was, at various times, attacked with sickness, pain and purging. The child died and a small quantity of arsenic was found in the viscera. At the inquest it was found out that the child became worse after each of the visits made by the mother and at each visit she was observed to rub its gums with the ends of her fingers. She was carrying arsenic in the hollows of her finger nails, and had thus secretly conveyed it into the child's mouth.—*Taylor, On Poisons, Ed. III, p.* 160.

1. *Brit. Med. Jour., July* 15, 1906, *p.* 158.

2. A woman was charged with having caused the death of one Azimullah of nearly 70 years, by giving him arsenic mixed with two loaves of bread and potato curry on the evening of the 16th January, 1923. The deceased took one bread with that vegetable and he had broken two morsels from the other bread when he began to feel uneasy and so he left it as it was. Shortly afterwards vomiting and purging commenced. Some *ghee* was administered to relieve the burning pain, but early next morning he expired. The Chemical Examiner, U. P., detected arsenic in the viscera as well as in the bread. The viscera were found to contain 1 grain of arsenic. The amount of arsenious oxide in the bread was 2.6 grains per ounce. Assuming that one of the *chapatis* weighed not less than two ounces, Azimullah must have taken at least 5 grains of arsenic.—*K. E.* v. *Mt. Sharifan, All. H. Court, Cr. App. No.* 449, 1923.

3. A Hindu female, aged 20 years, of Cuttack, introduced a plug of cotton wool smeared with arsenic into the vagina to procure abortion, but on finding that it had no desired effect, she took some arsenic internally and died from its effect. On post-mortem examination the stomach and small intestine were found congested, and there was ecchymosis near the cardiac end of the stomach. The uterus was enlarged and uniformly congested. It contained a fœtus of about four months with its membranes and liquor amnii intact. Arsenic was detected in the viscera as well as in the plug of cotton wool removed from the vagina.—*Bengal Chemical Examiner's Report, Ind. Med. Gaz., Aug.*, 1915, *p.* 304.

4. Abdul Majid, aged 35 years, was given by Ibrahim arsenic mixed with milk on the evening of the 12th May, 1926. Within half an hour he suffered from burning pain in the stomach and began to vomit and had purging. At about 2 a.m., while he was suffering from the acute symptoms of poisoning, he was assaulted by Ibrahim with a *gandasa* and he received several extensive incised wounds on the face and left shoulder. He died at 8 a.m. on the 13th May, 1926. Arsenic was detected in the vomit as well as in the viscera.—*King-Emperor* v. *Ibrahim, Allahabad High Court, Criminal Appeal No.* 518 *of* 1926.

5. In the beginning of 1931, a Mahomedan male became ill soon after taking his night meal in the Police Lines, Lucknow. He complained of severe burning pain in the stomach, had persistent vomiting and purging and was in a state of collapse. He was removed to the Police Hospital, where he was diagnosed as a case of cholera, and was transferred to the King George's Hospital for more efficient treatment. Soon after admission to this hospital he died. The Police suspecting foul play forwarded the body to me for post-mortem examination. The examination was held six hours after death, and showed characteristic appearance of acute poisoning by arsenic. The Chemical Examiner detected arsenic in the stomach contents and in the viscera.

6. A Hindu male, aged about 45, survived for seven days after he took some arsenic in bananas sent by his neighbour who owed some money to him. He had frequent vomiting, blood-stained stools, extreme thirst, pain in the throat and abdomen, cramps in the legs and headache for two days and nights. He was removed to hospital where except headache other symptoms subsided. He was in hospital for five days, and during this period he had vomiting only once and had yellowish-green watery stools. About ten hours before death he passed a large quantity of a dark, tarry stool, gradually collapsed and died.—*Bengal Chemical Examiner's Annual Report*, 1932, *p.* 14.

7. A Mahomedan woman mixed arsenic in some *halwa*, and got it distributed by a servant of her relative to a large number of families residing in Lahore. The *halwa* was tasted by about 33 persons including children. All developed symptoms of arsenic poisoning and while some recovered after treatment at home, a large number was removed to the Mayo Hospital where all except an old woman and her eight-year-old grandson revived.—*Leader*, *October* 23, 1935, *p.* 10.

8. A case occurred at Gaya where arsenic was used as an intoxicant. A Hindu male, aged 24, who was in the habit of taking intoxicants took one early morning about half a pound of *bhang sherbat*. As this did not produce any intoxicating effect on him, he took about 20 grains of arsenic. Immediately afterwards symptoms of poisoning appeared and death occurred within an hour and a half after his admission to hospital.—*Bengal Chemical Examiner's Annual Report*, 1932, *p.* 16.

ANTIMONY

The following are the compounds of antimony of which antimony tartaratum and antimony trichloride are important from a medico-legal point of view :—

1. **Antimony Tartaratum**, $[K\ (SbO)\ C_4H_4O_6]_2,\ H_2O$.—This is also called tartarated antimony, potassio-tartrate of antimony or tartar emetic. It is a pharmacopœial preparation, known as *antimonii potassii tartras,* and occurs in colourless, transparent crystals or in a white, granular powder, containing about 35 per cent of metallic antimony. It is insoluble in alcohol (90 p.c.), but is soluble in seventeen parts of cold water and in three parts of boiling water, the solution having a faintly acid and nauseating metallic taste. The dose is 1/32 to $\frac{1}{8}$ grain as a diaphoretic, $\frac{1}{2}$ to 1 grain as an emetic and $\frac{1}{2}$ to 2 grains by intravenous injection (in 2% solution). It has been occasionally mistaken for tartaric acid, Epsom salts, sodium bicarbonate and, sometimes, for cream of tartar. It constitutes an ingredient of many quack pills, such as Dixon's pills, etc. It is largely used in veterinary practice for improving the condition of the horse's skin.

Vinum antimoniale, a non-official preparation, is a solution of tartar emetic in sherry wine, the strength being 2 grains to an ounce. The dose is 10 to 30 minims as an expectorant and 2 to 4 drachms as an emetic. It is, sometimes, employed for criminal purposes.

Sodium antimonyl tartrate is an official preparation, known as *antimonii et sodii tartras,* and occurs as a white crystalline powder, freely soluble in water, and insoluble in alcohol (90 p.c.). The dose is the same as that of antimonii et potassii tartras.

2. **Antimony Trioxide (Antimonious Oxide)**, Sb_2O_3.—This is a non-official preparation, known as *antimonii oxidum,* and occurs as a greyish-white powder, having no taste, or odour. The dose is 1 to 2 grains. It is an ingredient of *Pulvis antimonialis* (James's powder, dose 3 to 6 grains), and gives rise to an important series of salts. When volatilised it condenses into two distinct forms, prismatic and octahedral crystals. It is almost insoluble in water, but soluble in hydrochloric acid and in the gastric juice forming antimony trichloride. It is readily soluble in tartaric acid, and in a boiling solution of hydrogen potassium tartrate (cream of tartar), forming potassium antimony tartrate or tartar emetic.

3. **Antimony Trichloride (Butter of Antimony)**, $SbCl_3$.—This is a colourless, deliquescent, crystalline substance, fusing to a yellow, oily liquid at the temperature of 73.2° C. It dissolves unchanged in a small quantity of water, but a white powder of oxychloride (SbOCl) is formed if an excess of water is added. When dissolved in hydrochloric acid, it is known as a *bronzing liquid,* and is employed in the arts and in farriery. It is often employed by quacks as an escharotic.

When taken internally, antimony trichloride acts chiefly as a strong corrosive. The corrosive symptoms may, however, be slightly pronounced and the narcotic symptoms may be more evident as in a case recorded by

Taylor[1] in which an army surgeon swallowed, for the purpose of suicide, two to three ounces. When seen about an hour later there was entire prostration of strength, with coldness of the skin, and incessant attempts to vomit. Severe griping pains were felt in the abdomen, and there was a frequent desire to evacuate the bowels, but nothing was passed. In the course of a few hours reaction took place, the pain subsided, and the pulse rose to 120. There was a strong disposition to sleep, so that he appeared as if labouring under the effects of a narcotic poison. In this state he continued until he died ten hours and a half after he had swallowed the poison.

4. **Antimony Trisulphide (Black Antimony)**, Sb_2S_3.—This is known as *Surma* in the vernacular. It occurs native as the steel grey ore, and is also formed as an orange red or brick red powder when sulphuretted hydrogen is passed through a solution of antimony trichloride or tartar emetic. The orange variety is an ingredient of Plummer's pill and antimony sulphuratum. The mineral often contains arsenic as an impurity.

Antimony Hydride (Antimoniuretted Hydrogen or Stibin), SbH_3.—This is a colourless, offensive, poisonous gas, which closely corresponds to arseniuretted hydrogen; but it differs from the latter in being less poisonous.

Organic Preparations.—Organic preparations, such as Stibenyl, Stibamine, Urea Stibamine, Stibosan (Von Heyden '471') and Neostibosan (Von Heyden '693b') have been introduced in medicine in recent years for the treatment of kala-azar and other protozoal diseases. Most of these preparations are used intravenously or intramuscularly.

Proprietary Medicines.—Dixon's pills contain 0.06 grain of tartar emetic in each pill, while Johnson's pills and Mitchell's pills contain 0.002 to 0.003 grain of tartar emetic per pill.

Acute Poisoning.—Symptoms.—The symptoms usually appear from a quarter to half an hour after taking a poisonous dose of tartar emetic. The first symptom is a strong metallic taste followed by a burning sensation in the mouth and œsophagus with a feeling of constriction in the throat. This is immediately followed by nausea and incessant vomiting with pain in the stomach and abdomen. The ejected matter at first consists of the stomach contents and later becomes fluid, tinged with bile and blood. The patient complains of intense thirst and difficulty of swallowing, as the lips, mouth and throat become swollen and sore. In some cases there is salivation. These symptoms are followed by profuse diarrhœa with bloody stools and suppressed urine. The pulse is small, rapid and imperceptible, and the respirations become laboured and painful. There are cramps in the lower extremities, sometimes accompanied by tetanic spasms. The skin is cold and clammy. The patient then faints away, is greatly prostrated, becomes unconscious and lastly dies from heart failure. In some cases the patient becomes delirious and comatose before death occurs.

1. *On Poisons, Ed. III, p.* 474.

In rare cases, vomiting and purging may be absent, and the symptoms affecting the nervous system may be more pronounced. Taylor[1] mentions the case of a veterinary surgeon, who swallowed by mistake 200 grains of tartar emetic for sodium carbonate. Vomiting came on in fifteen minutes, but only after tickling his throat. In a case[2] reported by Mr. Freer a man, aged 28, vomited only once about half an hour after he had taken 240 grains of tartar emetic by mistake for Epsom salts. He had violent pain in the stomach and abdomen with spasmodic contractions of the muscles of the abdomen and arms. The fingers were clenched, the muscles quite rigid, and there was involuntary aqueous purging. He recovered after six hours, but suffered from profuse night sweats. Dobie[3] cites a case in which the prominent symptom after a drachm of tartar emetic was coma, followed by death on the sixth day.

When antimony salts are injected intravenously, the poisonous symptoms which are commonly met with are fits of coughing and retching, giddiness, nausea, vomiting, diarrhœa and pains in the joints. A metallic taste is frequently observed. The pulse is feeble, rapid and irregular. Collapse occasionally sets in. In rare cases, unconsciousness and cyanosis occur, followed by death. Fakhry[4] reports the case of a woman who suffered from asphyxia after an intravenous injection of one grain of tartar emetic solution (6 %) as a treatment of schistosomiasis. She was unconscious and cyanosed with slow, laboured and superficial respirations, insensitive cornea and open eyes. The pulse could not be felt either in the radial or temporal artery, and the extremities were cold. Intracardiac and intravenous injections of 0.2 c.c. of adrenaline solution (1 in 1000) restored her to life.

When antimony salts are applied externally, pustular eruptions are produced on the skin. In some cases such eruptions are produced even when these salts are administered internally or intravenously.

Acute poisoning by antimony is similar to that by arsenic, but there are no remissions of the symptoms as in arsenical poisoning.

Fatal Dose.—The minimum fatal dose for an adult is probably five to ten grains of tartar emetic, although two grains have killed an adult, and three-quarters of a grain have killed a child.[5] A healthy woman, 25 years old, took 1½ grains without ill-effects, but took a similar dose twenty-four hours later, when she suffered from violent vomiting and purging and died in about thirty-six hours.[6] On the other hand, recoveries have occurred from large doses of 170,[7] 200,[8] 240[9] and even 478[10] grains.

1. *On Poisons, Ed. III, p.* 459.
2. *Lancet, May* 22, 1847, *p.* 535.
3. *Lancet,* 1887, *Vol. I, p.* 773.
4. *Lancet, Dec.* 12, 1931, *p.* 1325.
5. *Guy's Hosp. Rep., Oct.* 1857, 3 *S., III,* 369 ; *Taylor, On Poisons, Ed. III, pp.* 457, 464.
6. *Beu. Bulletin gem. de. Therapeutique,* 1856, *Vol. II, p.* 231.
7. *Carpenter, New York Med. Record,* 1883, *Vol. XXIV, p.* 401.
8. *Taylor, Loc. Cit.*
9. *Lancet, Loc. Cit.*
10. *Gleaves, West. Jour. Med. and Surg.,* 1848, 3 *S., I, p.* 23.

Six drachms of antimony trichloride have proved fatal to a man, aged 46 years, in about eight hours.[1] Recoveries from four or five drachms taken by a boy, aged 12 years,[2] and from one ounce[3] have been recorded.

Fatal Period.—Death usually occurs within twenty-four hours. It occurred in six hours in one case, and in ten hours in another.[4] It may be prolonged for several days or weeks. It should be remembered that death occurs much more rapidly in young children who are very susceptible to antimony salts. Charier[5] reports the case of a child who was given three-quarters of a grain of tartar emetic in an enema, and died within an hour. The shortest recorded period in cases of poisoning by antimony trichloride is less than two hours[6] and the longest is twenty-four hours.[7]

Treatment.—Use the stomach tube for tartar emetic, but use only emetics in the case of antimony trichloride. Give a drachm of tannic acid as an antidote to form an insoluble salt of antimony tannate, or give liquids containing tannin or tannic acid, such as strong and hot tea, coffee, or infusion of gallnuts. Demulcent drinks, such as milk, oils, mucilage, albumen water, linseed tea, etc., should then be given. Morphine may be given to relieve pain, and ice to control vomiting. Stimulants, such as caffeine, strychnine, camphor, alcohol and ether, may be given to combat heart failure.

Post-mortem Appearances.—Redness and inflammation of the mucous membranes of the stomach and duodenum with patches of submucous hæmorrhages. Small ulcers and hæmorrhagic extravasations are occasionally found in the cæcum and rectum. Pustular exudations and aphthous spots are, sometimes, found on the mucous membrane of the mouth, œsophagus and stomach. The stomach is corrugated and contracted, and its wall may be pale or yellow. The contents of the stomach are dark brown in colour, slightly acid in reaction and consist chiefly of a grumous, bloody fluid mixed with mucus which adheres to its inner wall. The liver, spleen and kidneys are congested. The brain is congested with effusion into the ventricles. The lungs are usually congested, and are dark in colour.

In exceptional cases the post-mortem appearances of poisoning by antimony may be absent. For instance, in the case of Mrs. Taylor, one of the victims of Dr. Pritchard, where death occurred from acute poisoning by tartar emetic, the post-mortem examination revealed nothing, although the poison was detected in the viscera, urine, blood and intestinal contents.[8]

In poisoning by antimony trichloride the post-mortem appearances will be charring and corrosion of those parts with which it has come into

1. *W. Bell, Brit. Med. Jour., June* 10, 1922, *p.* 917.
2. *Taylor, On Poisons, Ed. III, p.* 473.
3. *Ibid, p.* 474; *Lancet, Feb.* 26, 1848, *p.* 230.
4. *Med. Gaz., Vol. XLV, p.* 801.
5. *Jour. de. Chim. Med.*, 1847, 3 *S., III,* 472; *Witthaus, Med. Juris. and Toxic., Vol. IV, p.* 860.
6. *Cooke, Lancet, May* 19, 1883, *p.* 860.
7. *Med. T. and Gaz., Oct.* 22, 1864.
8. *Peterson, Haines and Webster, Leg. Med. and Toxic., Vol. II, Ed. II, p.* 267.

contact. In the case recorded by Cook[1] the mucous membrane of the stomach was almost black from intense congestion, but the corrosion and blackening of the lips, tongue, mouth, pharynx and œsophagus were not found.

Chronic Poisoning.—This occurs from the administration of repeated small doses of tartar emetic. The symptoms are nervous irritability, giddiness, headache, nausea, persistent vomiting of bile and mucus, and watery purging sometimes alternating with constipation. The tongue becomes foul; there is loss of voice, and the pulse is weak and rapid. The skin is cold and clammy. There is great prostration and the patient is very much emaciated. He abhors the sight of food, as he cannot retain it in the stomach. Death results from exhaustion, or from the effects of a larger dose than usually administered. Sometimes, cramps occur instead of relaxation of the muscles.

Treatment.—Remove the patient from the source of poisoning, and eliminate the poison from the system by giving potassium iodide.

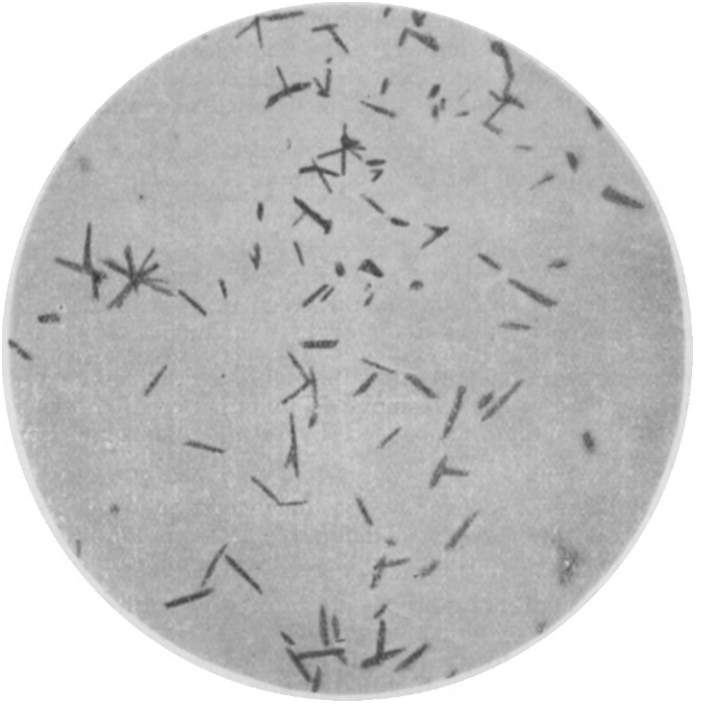


Fig. 134.—Microphotograph of Antimony Trioxide Crystals × 400 (*R.B. Dr. K. N. Bagchi*).

Post-mortem Appearances.—The post-mortem appearances in chronic poisoning are not so characteristic as in acute poisoning. The body is emaciated. The tongue and the interior of the mouth are covered with fur or marked with aphthous spots. There may be ulcerations in the

1. *Lancet, May* 19, 1883, *p.* 860.

stomach and intestines. The heart, liver and kidneys show fatty degeneration.

Chemical Tests.—1. The addition of hydrochloric acid to a liquid solution gives a white precipitate, soluble in excess.

2. Sulphuretted hydrogen forms an orange precipitate of sulphide of antimony, soluble in caustic potash or ammonia.

3. If the fluid containing some free hydrochloric acid be put in a platinum capsule, and a fragment of zinc be introduced, a black deposit of metallic antimony is formed on the inside of the capsule ; this will be turned yellow on adding ammonium sulphide.

4. *Reinsch's Test.*—Same as in arsenic, but on heating, the sublimate of antimony trioxide appears amorphous or in needle-shaped crystals.

5. *Marsh's Test.*—The process is the same as in arsenic, but the flame produced by burning stibine (SbH_3) has a bluish-green tint, and the stain formed by the deposit of antimony on the porcelain dish is black and lustreless, insoluble in hypochlorite of lime, but soluble in stannous chloride. On heating the delivery tube the metallic and silvery mirror of antimony is formed on both the sides in the vicinity of the heated part ; the mirror does not sublimate, yielding octahedral crystals as in arsenic.

Medico-Legal Points.—Antimony as a metal is not considered poisonous but when inhaled in the form of vapour it is said to have produced dangerous symptoms.

Poisoning by antimony salts is rare in India. In his annual report for 1922, the Chemical Analyser of Sind reports the case of a person who died from the effects of antimony tartar given in 24-grain doses thrice with a purgative. The poison was detected in the viscera.

In Europe, a few homicidal and still fewer suicidal cases have occurred. For homicidal purposes tartar emetic is given in small doses for several days, so that the symptoms caused by it may simulate some gastro-intestinal disease.

Accidental cases of poisoning by tartar emetic have been recorded from an overdose when given medicinally, or from its administration in mistake for cream of tartar, Epsom salts, bicarbonate of sodium, etc.

Outbreaks of acute accidental poisoning by antimony have, sometimes, occurred from drinking lemonade prepared in cheap enamelled utensils. They are due to the tartaric acid in the " lemonade crystals " or the citric acid of fresh lemons dissolving some of the antimony oxide which is used instead of the non-poisonous tin oxide in the manufacture of the white enamel coating.[1] About seventy workmen of a firm at New Castle on Tyne suffered from the symptoms of acute antimony poisoning after they had taken lemonade prepared from tartaric acid crystals which were dissolved in boiling water overnight in enamelled buckets. They all recovered. The enamel of the bucket contained antimony trioxide equivalent to 5 per cent of metallic antimony. Dr. Dunn found on analysis

1. *Brit. Med. Jour., June* 16, 1934, *p.* 1085.

that an ordinary tumbler of ten ounces contained 0.57 grain of antimony or 1.52 grains of tartar emetic.[1] In a school at Folkeston lemonade from fresh sliced lemons was prepared in white enamelled jugs. Half an hour after it was served, twenty-five persons were sick.[2]

Acid vegetables and fruits may extract antimony from cheap enamelled vessels ; hence they should not be cooked in such vessels.[3] Hellen Lukis reports the cases of three families in which all the members were stricken down with sickness and diarrhœa ; investigation showed that the symptoms came on shortly after eating rhubarb pie baked in a cheap new enamelled pie dish.[4]

Tartar emetic is given to confirmed drunkards as a cure for the habit, and accidental poisoning has occurred from an overdose thus given.

Tartar emetic acts as a depressant to the heart muscle ; hence even if given in medicinal doses it may prove fatal to the persons who are aged, infirm and debilitated from disease, while these doses would not have any deleterious effect on strong, healthy individuals.

Cases of accidental poisoning, sometimes, occur from chloride of antimony, as it is used in arts as a *bronzing liquid.*

Method of Administration.—Symptoms of poisoning have occurred not only from its administration by the mouth, but from its external application in the form of a powder or ointment to the unbroken skin, from its use as an enema and from its absorption into the system by wearing a cloth to colour which tartar emetic was used as a mordant.

Elimination of Antimony.—By the vomit and purging it promotes, antimony is largely expelled immediately after swallowing it, and is eliminated rapidly by the kidneys after it is absorbed into the system. It is also eliminated by the mucous membrane of the stomach even if administered by any other channel than the mouth. Before it is eliminated it is deposited into the liver, spleen, kidneys and long bones. It is also excreted in the bile and milk.

A woman who was given tartar emetic as a medicine for pleurisy suckled her infant, and the child got an attack of vomiting soon after every attempt to suck the breast.[5]

Antimony, like arsenic, has a preservative effect on the bodies of persons poisoned by it. For example, in two cases of exhumation the bodies were found in a remarkable state of preservation after a burial of twenty-one months and five years.[6] On analysis antimony was found to be present in the internal organs, such as the stomach, liver, kidneys, intestine and even the brain.

Antimony is not a normal constituent of the body, nor is it met with in any of the food articles. Hence any attempt based on these grounds to

1. *Lancet, Aug.* 18, 1928, *p.* 337.
2. *Brit. Med. Jour., March* 11, 1933, *p.* 423.
3. *Miller, Jour., Home Econ.*, 1916, *VIII, p.* 361.
4. *Brit. Med. Jour., April* 1, 1933, *p.* 581.
5. *Sir R. Christison, Poisons, p.* 483.
6. *Stevenson, Brit. Med. Jour., April* 11, 1903, *p.* 873. *See Chapman Case.*

explain its presence in the tissues must necessarily fail. The poison, if present in the body, must have been administered—there is no other possible explanation.

The Chapman Case.—Severino Klosowski *alias* George Chapman was a Pole, who in his youth had received some sort of medical training as a dispenser and surgical dresser in a hospital at Prague in Poland. In 1889, he came to England, and acted as a barber's assistant. He assumed the name of Chapman and lived with a woman, Mrs. Isabella Spink, who passed as Mrs. Chapman. The couple went to live at Hastings, where Chapman managed to procure about an ounce of tartar emetic from a chemist. Leaving Hastings he became a proprietor of the "Prince of Wales" public-house, Bartholomew Square, Finsbury. Mrs. Chapman now became ill, the chief symptom being frequent vomiting. Chapman ascribed her illness to excessive drinking. On Christmas day, 1897, Mrs. Chapman was extremely ill and died about midday. The cause of death was certified to be consumption. He then met Bessie Taylor whom he engaged as a barmaid. In the spring of 1899 he persuaded her to go with him through some form of marriage. In March they moved into the "Monument" public-house in Southwark, where the woman became ill. On the 1st January, 1901, Dr. Stoker saw her, when she was suffering from vomiting, pain in the stomach, thirst, loss of appetite, tenderness of the abdomen, tenesmus and occasional diarrhœa. During the course of her illness three other doctors had been called in consultation at different times, and at least four different diagnoses were made, namely, uterine mischief, hysteria, cancer of the stomach or intestines and obstruction of bowels. She died on the 13th February, 1901, when Dr. Stoker filled in the death certificate "intestinal obstruction, vomiting and diarrhœa" as the cause of death.

In September, 1901, Maud Marsh, 19 years old, entered his service as a barmaid. Shortly afterwards it was given out that they were married. Subsequently there was a suspicious fire at the "Monument", and at the end of 1901, the couple moved to the "Crown" public-house close by in the borough. In 1902, Maud began to feel unwell, the symptoms being sickness, diarrhœa and abdominal pains. On the 28th July, she was admitted into Guy's Hospital, where she remained as an in-patient until the 20th August. A provisional diagnosis of peritonitis was made by the attending physician, but no cause was found for that particular condition. She left the hospital considerably improved, but on returning to the Crown she at once became ill again, and on October 10, she was seen by Dr. Stoker, who continued attending upon her until her death on Wednesday, the 22nd October. On the 20th October, the mother of Maud Marsh came to stay at the house. On the following day a surgeon practising at Croydon and the medical attendant of the Marsh family was called in by the father in consultation with Dr. Stoker. They both arrived at the conclusion that the deceased was suffering from some form of irritant poisoning, probably ptomaine. After death Dr. Stoker refused to grant a certificate of death and suggested that either a post-mortem examination or an inquest should be held. After a good deal of hesitation Chapman allowed a private post-mortem examination at the Southwark mortuary on the 23rd October. Nothing was found in the viscera to account for the death, but a chemical analysis detected traces of arsenic together with antimony in a far larger quantity in the stomach contents and other viscera. Subsequently Sir Thomas Stevenson, at the request of the Coroner, made a second analysis and found antimony in nearly all parts of the body, from which he recovered $7\frac{1}{4}$ grains of metallic antimony, equivalent to $20\frac{1}{8}$ grains of tartar emetic. He concluded that death was due to antimony administered in a soluble form either as tartar emetic or as metallic antimony. From the fact that some of the poison was found in the intestine, he arrived at the further important conclusion that a large dose must have been administered shortly before death, as otherwise it would have been absorbed. Chapman was arrested and prosecuted for having caused the death of Maud Marsh by the administration of tartar emetic.

It was proved during the trial that Chapman prepared food at times for the deceased with his own hands, and also administered, as a rule, during her illness, both food and medicine. Sickness often followed immediately after taking food, drink or medicine. On the night before the death the mother of the deceased took a small quantity of the brandy and water that had been brought by the prisoner for the use of the deceased. Shortly after taking the brandy the lady was attacked by violent sickness and diarrhœa, lasting several hours.

Suspicion being raised about the deaths of Bessie Taylor and Isabella Spink, their bodies were exhumed. The body of Bessie Taylor was found in a remarkable state of preservation after a burial of twenty-one months, doubtless owing to the preservative qualities of the large quantity of antimony shown to be present in the tissues. Sir Stevenson recovered 29.12 grains of tartar emetic from the body, but found no evidence of uterine or intestinal disease. The body of Isabella Spink, who had been buried for about five years, was also found preserved in a most remarkable manner. Sir Stevenson stated that on opening the coffin the head and face were those of a woman, who might have been coffined the day before. The eyeballs were found intact. Antimony was found in the various organs and tissues in such a quantity as to suggest that she had taken more antimony than Maud Marsh. No signs of tuberculosis were found in the body.

Chapman was found guilty and executed at Wandsworth Gaol on the 7th April, 1903.—*Brit. Med. Jour., April* 11, 1903, *p.* 873 ; *Ibid., Sept.* 24, 1904, *p.* 752.

MERCURY (*PARA*)

Mercury or quicksilver is a liquid metal having a bright silvery lustre. It is easily converted into the form of a dull grey powder when shaken up with oil or triturated with sugar, chalk or lard. The process is known as deadening, and is used in preparing mercurial ointment and emplastrum. The metal is not acted upon by hydrochloric acid. It is slightly dissolved by dilute cold sulphuric acid but is completely dissolved by strong sulphuric and nitric acids. It is a pharmacopœial preparation, and is called *Hydrargyrum*, the dose being ½ to 3 grains by the mouth and ½ to 1 grain by intramuscular injection. The other official preparations of the metal are—

1. *Hydrargyrum cum creta* (*Grey powder*).—It is a greyish-blue powder and contains 33 per cent of mercury. The dose is 1 to 5 grains. If kept long and exposed to light, a portion of the mercury is converted into mercuric oxide which produces a poisonous action on the system.

2. *Injectio hydrargyri* (*Mercurial cream*).—It contains 1 grain of mercury in 10 minims. The dose is 5 to 10 minims by intramuscular injection.

3. *Pilula hydrargyri* (*Blue pill*).—It contains 33 per cent of mercury. The dose is 4 to 8 grains.

4. *Unguentum hydrargyri* (*Blue ointment*).—It contains 30 per cent of mercury.

5. *Unguentum hydrargyri compositum* (*Scott's ointment or dressing*). —It contains 12 per cent of mercury.

6. *Unguentum hydragyri nitratis forte* (*Citrine ointment*).—It contains 6.7 per cent of mercury.

7. *Unguentum hydrargyri nitratis dilutum.*—It contains 20 per cent of the strong ointment of mercuric nitrate.

COMPOUNDS OF MERCURY

1. **Mercuric Oxide**, HgO.—This is known in the vernacular as *Sipichand.* It is a brick-red crystalline powder but it forms an amorphous yellow powder when a mercuric salt is acted upon by caustic soda or potash. Both the red and yellow varieties are insoluble in water.

Red mercuric oxide is used for preparing a non-official ointment, *Unguentum hydrargyri oxidi rubri* (red precipitate ointment).

Yellow mercuric oxide is a pharmacopœial preparation, and is known as *Hydrargyri oxidum flavum*. It is contained in the official preparations of *Oculentum hydrargyri oxidi* and *Oculentum Atropinæ cum hydrargyri oxido* and enters into the composition of *Hydrargyrum oleatum* (mercuric oleate) and *Unguentum hydrargyri oleati.*

2. **Mercuric Chloride (Perchloride of Mercury, Corrosive Sublimate)**, $HgCl_2$.—It exists in the form of heavy, colourless masses of prismatic crystals or as a white, crystalline powder. It has a styptic, nauseous, metallic taste. It is soluble in eighteen parts of cold water and three parts of boiling water. It is readily soluble in alcohol (90%), ether and glycerine and is very soluble in solutions of the alkaline chlorides. On account of its antiseptic properties it is largely used in medicine as well as in taxidermy. It is a violent poison, and is obtained in the *bazaar*, often mixed with impure subchloride. The official dose of mercuric chloride (*Hydrargyri perchloridum*) is 1/32 to 1/16 grain. The pharmacopœial solution, *Liquor hydrargyri perchloridi,* contains 1/10 grain of mercuric chloride in 110 minims, the dose being 30 to 60 minims.

When ammonia is added to a watery solution of mercuric chloride, ammonio-chloride of mercury is formed. It is also known as ammoniated mercury or white precipitate (*Hydrargyrum ammoniatum*, B.P.). It is a white, heavy tasteless powder, insoluble in water, alcohol (90%) and ether. It is used in preparing an official ointment, *Unguentum hydrargyri ammoniati* (white precipitate ointment).

3. **Mercuric Iodide**, HgI_2.—This is also called red iodide of mercury or biniodide of mercury. It is a scarlet red powder, obtained by the action of a watery solution of mercuric chloride on one of potassium iodide. It is almost insoluble in water, but soluble in about 130 parts of alcohol, and freely in ether, in nitric acid and in a solution of potassium iodide or mercuric chloride. The official dose is 1/32 to 1/16 grain. It forms one of the constituents of Donovan's solution (*Liquor arsenii et hydrargyri iodidi*), the dose of which is 5 to 15 minims.

4. **Mercuric Cyanide**, $Hg(CN)_2$.—This is nearly as poisonous as corrosive sublimate, but has no corrosive action. It exists as white, prismatic crystals, having a bitter, metallic taste but no odour. It is soluble in 12 parts of water and in 15 parts of alcohol.

Mercuric oxycyanide, $HgO\ 3\ [Hg(CN)_2]$, is a white crystalline powder, soluble in 18 parts of water. The official dose is 1/12 to 1/6 grain by intramuscular injection and 1/6 grain by intravenous injection.

Mercuric thiocyanate (sulphocyanide), $Hg(CNS)_2$, is an insoluble powder, which, when ignited, gives off abnoxious fumes of the metal and forms an exceedingly voluminous ash. It is moulded into pellets, which are known as "Pharaoh's serpents", as these, when burnt, produce long snake-like tubes of ash. Chuni Lal Bose gives an account of a non-fatal

poisoning of a girl, 14 months old, by a piece of Pharaoh's serpent swallowed by her accidentally with parched rice.[1]

5. **Mercuric Nitrate**, $Hg(NO_3)_2$.—This is crystalline, but deliquescent. It is used for painting on porcelain, and is used by hatters and furriers, as well as in veterinary medicine. It acts as a corrosive poison, and is similar in action to mercuric chloride. Symptoms of chronic poisoning occur among hatters and furriers.

6. **Mercuric Sulphide (Cinnabar)**, HgS.—This is known in the vernacular as *hingul, ras sindoor, cheena sindoor* or *shingarf*. It occurs as a chief ore of mercury, and is artificially prepared as a red crystalline powder, which is then known as the pigment vermilion. It is regarded as non-poisonous, but its vapours are poisonous. Cases of acute poisoning have occurred from its use as a fumigant. Chronic poisoning has also occurred from it having been used to colour vulcanised rubber meant for artificial teeth.

Mercuric Sulphate, $HgSO_4$.—This is a white, crystalline powder, and acts as a corrosive poison. It has been administered in mistake for sulphocarbolate of sodium, and has caused death.[2] It has also been taken with suicidal intent.[3]

Mercuric Methide (Mercury Dimethyl), $Hg(CH_3)_2$.—This is a highly poisonous liquid, and has produced death by the inhalation of its noxious vapour.[4] It has also produced insanity.

Mercurous Chloride (Subchloride of Mercury, Calomel), Hg_2Cl_2.—This is sold in the *bazaar* as *raskapoor* in fibrous, heavy, dirty white masses, often mixed with mercuric chloride. The pharmacopœial preparation, *Hydrargyri subchloridum*, is a heavy, amorphous, white and tasteless powder, insoluble in water, alcohol (90 per cent) or ether but soluble in a mixture of nitric and hydrochloric acids. The dose is $\frac{1}{2}$ to 3 grains and $\frac{1}{2}$ to 1 grain intramuscularly. When heated, it sublimes without fusing. It is converted into mercuric chloride by chlorine water, nitrohydrochloric acid, alkaline chlorides and common salt; hence it should never be prescribed with any of these substances. Exposure to sunlight decomposes it into mercury and mercuric chloride. It enters into the composition of the following preparations:—

1. *Injectio hydrargyri subchloridi* (*Calomel injection or calomel cream*).—It contains 1 grain of calomel in 20 minims. Dose, 10 to 20 minims intramuscularly.

2. *Lotio hydrargyri nigra* (*Black wash*).—The strength of calomel is nearly 3 grains to 1 fluid ounce.

3. *Unguentum hydrargyri subchloridi* (*Calomel ointment*).—It contains 20 per cent of calomel.

1. *Ind. Med. Gaz., March*, 1905, *p.* 99.
2. *Witthaus, Manual of Toxicology, Ed. II, p.* 738.
3. *Derobert, Annales de Medecine Legale, March*, 1937, *p.* 219; *Med.-Leg. and Criminol. Rev., April*, 1937, *p.* 227.
4. *Edwards, St. Barth. Hosp. Rep.*, 1865, *Vol. I, p.* 141.

Calomel is one of the ingredients of a non-official preparation, *Pilula hydrargyri subchloridi* (*Plummer's pill*), the dose being 4 to 8 grains.

Calomel is generally regarded as a safe medicine, though medicinal doses have produced toxic effects. R. F. Bolt[1] cites the case of a man, aged 65 years, who, owing to marked idiosyncrasy to calomel, had an attack of acute poisoning after taking a 1-grain pill. The symptoms were intense abdominal pain, vomiting, urticarial rash, œdema of the fore-arms, legs, neck, eyelids and lobes of the ears, severe pain in the right loin, scanty urine, dry skin, and a rise of temperature to 99.8° F. Recovery occurred in four or five weeks. In some cases death may occur indirectly from septic poisoning from extensive ulceration and gangrene of the mouth and throat.

Calomel, administered hypodermically or intramuscularly, may cause fatal poisoning. Runeberg[2] reports the case of a woman, 34 years old, who received three hypodermic injections of calomel of 1½ grains each in one month, developed the symptoms of mercurial poisoning and died on the twenty-third day after the last injection. Backer[3] reports a case of fatal poisoning following the intramuscular injection of 1 c.c. of a 10 per cent suspension of calomel, death resulting one week after the third injection.

Subsulphate of Mercury (Turpeth Mineral), $HgSO_4$, $2HgO$.—This is a lemon-yellow powder, sparingly soluble in water. It is used as an emetic in three to five-grain doses, especially on the continent and in the United States. It has occasionally caused death by acting as an irritant poison.

Mercurous Nitrate, $Hg_2(NO_3)_2$.—This is colourless and crystalline. It is soluble in water acidulated with nitric acid, and is as poisonous as mercuric nitrate.

Novasurol (Merbaphen).—This is a double salt of sodium mercuri-chlorphenyl oxylacetate with diethyl-barbituric acid. It is a white, crystalline powder, soluble in water, and contains 33.9 per cent of mercury. It is a powerful diuretic, the dose being ½ to 2 c.c. of a ten per cent solution by intravenous or intramuscular injection. A case of homicidal poisoning by the injection of 30 c.c. of novasurol occurred at Cologne in 1926. The patient died from mercury poisoning with bleeding diarrhœa, inflammation of the mucous membrane of the mouth, and anuria.[4]

Mercurochrome-220 (Dibromo-hydroxy-mercury Fluorescein), $C_{20}H_7O_5Br_2$. HgOH, Na_2.—This is also known as mercurome, and occurs as iridescent green scales, and dissolves readily in water. It contains approximately 23 to 24 per cent of non-ionised mercury. It has been used intravenously in cystitis, gonorrhœa, articular rheumatism, endocarditis, and in septicæmic conditions. The dose for a man weighing 63½ kilogrammes (10 stone) is 0.13 to 0.32 gramme intravenously in 0.5 per cent or even greater dilution. Ten milligrammes per kilogramme of body weight given intravenously kill rabbits. Five milligrammes per kilogramme

1. *Brit. Med. Jour., Aug.* 14, 1920, *p.* 245.
2. *Deutsch. Med. Wchnschr.*, 1889, *XV, p.* 4; *Peterson, Haines and Webster, Leg. Med. and Toxic., Vol. II, Ed. II, p.* 187.
3. *Hospitalstid.*, 1921, 64, *p.* 737; *Ibid.*
4. *Erich Leschke, Clin. Toxic., Engl. Trans. by Stewart and Dorrer*, 1934, *p.* 35.

of patients' weight in a 1 per cent solution have been injected intravenously twice a week without trouble in several cases.[1] G. P. B. Huddy[2] treated 29 adult patients with mercurochrome after operation with a view to preventing the onset of post-operative pneumonia. He injected intravenously 20 c.c. of a 1 per cent solution immediately following an operation, and 10 c.c. of a similar solution two days later. Reaction started in 10 patients after the second injection. This suggests that the drug may be cumulative. The most common reaction consisted of a rigor with a rise of temperature to about 102° F. In one case there was blood in the urine and stools, and in another case there was a severe rigor with headche, cyanosis and collapse.

Toxic effects have occurred mostly after the prolonged use of the drug in fairly large doses. A. V. St. George[3] reports that death followed the intravenous injection of a 1 per cent. solution in five cases of sepsis. The post-mortem examination showed that it induced nephritic and intestinal lesions which resulted in death.

Acute Poisoning—Symptoms.—The symptoms are mostly due to corrosive sublimate, and commence immediately after swallowing the poison. They are rarely delayed beyond half an hour, although in a case reported by Wood[4] the symptoms were delayed one hour and a half. These are an acrid, metallic taste and a feeling of constriction or choking sensation in the throat, hoarse voice and difficult breathing. The mouth, tongue and fauces become corroded, swollen and coated with a greyish-white coating. Hot burning pain is felt in the mouth, extending down to the stomach and abdomen, followed by nausea, retching and vomiting. The vomited matter is a greyish, slimy mucoid material containing blood and shreds of mucous membrane. This is followed by diarrhœa with bloody stools and accompanied by tenesmus. The urine is suppressed or scanty, containing blood and albumin. The pulse becomes quick, small and irregular, and collapse soon supervenes. In some cases spasms, tremors, convulsions and unconsciousness are observed before death occurs. Gangrenous colitis[5] may be observed, if the patient has survived six or more days.

It should be noted that the symptoms are liable to great variation in different cases although the doses have been the same.

Salivation, gingivitis and loosening of the teeth with fœtid breath are usually common when mercurial vapours are inhaled.

Diagnosis.—This has to be diagnosed from arsenical poisoning. The symptoms of mercurial poisoning commence sooner, and the acridity and the constriction of the throat are more marked. The vomited matters and stools more often contain blood. The irritation of the kidneys is also more pronounced.

1. *Joseph O'Carroll, Lancet, Dec.* 31, 1927, *p.* 1416.
2. *Lancet, Ibid.*
3. *Jour. Amer. Med. Assoc., Dec.* 26, 1925, *p.* 2005.
4. *Jour. Amer. Med. Assoc.*, 1915, *LXIV, p.* 507.
5. *Berger, Applebaum and Young, Jour. Amer. Med. Assoc., Feb.* 27, 1932, *p.* 700.

Fatal Dose.—An intravenous injection of 0.06 gramme of metallic mercury as a 40 per cent oil emulsion has proved fatal. On the contrary, recovery has followed 27.2 grammes (2 c.c.) of metallic mercury injected intravenously with a view to commit suicide.[1] Thirty grains of red oxide of mercury taken with an ounce of acetic acid proved fatal to a girl of 17 years within 30 hours.[2] The average fatal dose of mercuric chloride for an adult is three to five grains. Its smallest recorded dose is two grains which killed a child.[3] Recovery[4] has resulted after the administration of ninety or one hundred grains, or even much larger doses under prompt treatment by milk, eggs, and emetics. The average fatal dose of mercuric cyanide is ten to twenty grains. That of mercuric nitrate is one drachm, and of turpeth mineral is forty to sixty grains, though three to six grains have caused death in from 3 to 15 hours, when administered to young children.[5] Six grains is the smallest quantity of calomel which has caused the death of a boy, aged fourteen years, in three weeks from ulceration and gangrene of the face.[6]

Fatal Period.—The usual fatal period is 3 to 5 days, but death may take place much sooner or later than this. The shortest recorded period from mercuric chloride poisoning is half an hour,[7] and the longest is twenty-three days.[8]

Treatment.—If vomiting has not already commenced, give emetics or pass the stomach tube cautiously and wash out the stomach with warm water to which carbonate of magnesium has been added. Albumen in the form of raw white of egg, or vegetable gluten, mixed with a large quantity of skim milk should then be administered; the albuminate of mercury thus formed, although insoluble in water. is soluble in excess of albumen, and is liable to be digested and absorbed if left in the stomach. It must, therefore, be removed by the administration of emetics or lavage of the stomach.

Three to four tablespoonfuls of animal charcoal suspended in about a pint of water should be administered as soon as possible, as it has the great power of abosrbing mercury salts. The addition of about five drachms of magnesium sulphate increases the absorptive power of the charcoal and hastens the removal of the ingested poison.[9]

Imaz and Martinez[10] advise lavage of the stomach with 500 c.c. of water containing 30 grammes of sodium thiosulphate in cases of acute poisoning by corrosive sublimate, when the drug has been swallowed. A ten per cent solution of sodium thiosulphate may also be administered

1. *Leschke, Clin. Toxic.. Eng. Trans. by Stewart and Dorrer,* 1934, *p.* 33.
2. *Brit. Med. Jour., Vol. I,* 1896, *p.* 19.
3. *Taylor, Princ. and Pract. of Med. Juris., Ed. IX, Vol. II, p.* 437.
4. *Witthaus, Med. Juris., and Toxic., Vol. IV, p.* 735.
5. *Mc. Phedron, Med. News, Phila.,* 1833, *XLIII, p.* 682; *Witthaus, Ibid., p.* 739.
6. *London Med. Gaz., Vol.* 18, 1836, *p.* 484.
7. *Taylor, On Poisons, Ed. III, p.* 378.
8. *Scott Sugden, Brit. Med. Jour., April* 8, 1905, *p.* 767.
9. *Leschke, Clin. Toxic., Engl. Trans. by Stewart and Dorrer,* 1934, *p.* 43.
10. *Revista Especialidades, Buenos Aires, Oct.,* 1929, *p.* 1145; *Jour. Amer. Med. Assoc., May* 31, 1930, *p.* 1806.

intravenously. A woman,[1] who took ten 7½-grain tablets of mercuric chloride with suicidal intent, recovered after five intravenous injections of 10 c.c. of sodium thiosulphate administered every eight hours.

Rosenthal[2] recommends the use of sodium formaldehyde sulphoxylate as an antidote for mercury poisoning. His method of treatment consists of gastric lavage with a 5 per cent solution of sodium formaldehyde sulphoxylate, 200 c.c. being left in the stomach and intravenous injection of 10 g. of the substance in 5 to 10 per cent solution, the dose being repeated in 4 to 6 hours. He also gives high colonic lavage with a 1 in 1000 solution.

Intravenous injections of 20 to 40 c.c. of a 25 per cent solution of glucose have been recommended. The solution of glucose in the form of a drop by drop enema has also a beneficial effect. Later on, the symptoms should be treated as they arise. It is said that sodium chloride is to be avoided, as it favours the absorption of mercury, but Professor Michaud[3] recommends a liberal supply of sodium chloride (about 15 grammes daily) with the food in order to combat the dangerous acidosis and, in addition, a 0.9 per cent solution of sodium chloride and a 40 per cent solution of sodium bicarbonate hypodermically and intravenously. Since the introduction of this method he has saved two cases of serious poisoning by corrosive sublimate.

Post-mortem Appearances.—The appearances of corrosive poisoning will be present if the poison is taken in a concentrated form. Otherwise the signs of irritant poisoning will be observed.

The mucous membrane of the lips, mouth and pharynx presents a diffuse greyish-white escharotic appearance. The same appearance is noticeable in the œsophagus; its mucous membrane appears also corrugated and eroded. The stomach contents are masses of coagulated albumen mixed with mucus and liquid blood. Its mucous membrane is corroded, inflamed and covered with a greyish deposit of mercury, or a black deposit of its sulphide.

During the post-mortem examination great care should be taken in removing the stomach from the abdominal cavity, lest it might be ruptured owing to the great softening of its walls. Perforation of the stomach is very rare.

The intestines, chiefly the cæcum and rectum, are found inflamed. The liver and spleen are congested. The kidneys are often acutely inflamed.

It must be remembered that the post-mortem lesions are found in the alimentary canal even if death has occurred from absorption of corrosive sublimate as a result of the external application to the skin or irrigation of wounds or abscess cavities, or of the uterus and vagina.

1. *Merchbanks, Smith and Church, Jour. Amer. Med. Assoc., Feb.* 21, 1931, *p.* 611.
2. *Jour. Amer. Med. Assoc.*, 1934, *Vol.* 102, *p.* 1273; see also *Josephine Barnes, Lancet, Jan.* 14, 1939, *p.* 89.
3. *Fortschritte der Therapie, No.* 16, 1930; *Ars Medici, Nov.*, 1930, *p.* 502.

Chronic Poisoning.—This form of poisoning occurs among those who are exposed to the vapours of mercury in factories where mercury and its salts are largely used. It also occurs among those who have taken internally for a prolonged period excessive doses of mercury compounds, or used the mercurial ointment in the form of an external application.

Symptoms.—These are nausea, digestive disturbances, colicky pain and vomiting. Ptyalism or salivation is a constant symptom which is accompanied by foul breath, and inflamed and ulcerated gums, which usually present a blue line at their junction with the teeth. Later, the teeth become loose and carious ; necrosis of the jaws occurs, and diarrhœa, general wasting and anæmia result.

The skin eruptions of an erythematous, eczematous or pustular type may be noticed. The nervous symptoms known as *mercurial tremors* supervene. These first of all affect the muscles of the tongue producing stammering and hesitation of speech, and then affect the muscles of the face ; these later extend to the muscles of the arms and legs. They are excited by voluntary movements, and are absent during sleep. The tremors are followed by paralysis of the limbs. The patient complains of cough with bloody expectoration, and dies from exhaustion. Sometimes, he is affected by mental disturbances and hallucinations, which may result in insanity.

The lung and kidney affections, as well as nervous affections are likely to be aggravated by the toxic effects of mercury.

Treatment.—The patient should be removed from the surroundings where he was exposed to the poison. He should be directed to drink milk freely and to gargle his mouth with potassium chlorate or borax, to keep his bowels open by saline purgatives, and to take warm baths to promote the action of the skin. Intravenous injections of sodium thiosulphate in doses of 0.45 to 0.6 gramme in 5 c.c. of water on alternate days are considered efficacious for the treatment of salivation.[1]

It is advisable to give potassium iodide in small doses, so that the poison may be converted into mercuric iodide, which is soluble in excess of the potassium salt. Massage and electricity should be advised for paralysis. Narcotics should be given for severe tremors.

Chemical Tests for Mercuric Salts.—1. Hydrochloric acid and sulphuretted hydrogen give a yellow precipitate which canges to orange, brown and lastly black, insoluble in alkalies or dilute acids.

2. Caustic potash gives a yellow precipitate.

3. Potassium iodide gives a scarlet precipitate, soluble in excess.

4. Stannous chloride gives a white precipitate, changing to black.

5. If a piece of a bright wire of copper be introduced into the solution acidulated with a few drops of hydrochloric acid, a silver coating of mercury will be formed on the wire.

1. *H. C. Semon, Brit. Med. Jour., April* 12, 1924, *p.* 662.

Chemical Tests for Mercurous Salts.—1. Hydrochloric acid gives a white precipitate blackened by ammonia.

2. Potassium iodide gives a greenish precipitate, which becomes black if the reagent is added in excess.

3. Caustic potash yields a black precipitate, insoluble in excess.

4. Potassium bichromate gives a brick-red precipitate.

5. Stannous chloride gives a white precipitate, changing to grey.

Reinsch's Test.—This is used to detect mercury in organic mixtures. A grey coating of mercury forms on the copper foil. If the copper foil is dried and heated in a dry test tube, mercury will volatilise and deposit as round globules of the metal on the part of the cooler tube, which can be seen under the microscope.

Medico-Legal Points.—Metallic mercury, when perfectly pure, can hardly be considered to be poisonous. Cases are recorded where individuals have swallowed a pound or two of the liquid metal as a treatment of chronic constipation without any harmful effects. During the trial of a murder case at Armagh in June, 1905, it was proved in evidence that the accused first tried to kill the old woman by repeated administrations of metallic mercury, but eventually put strychnine into the meal which caused her death. The analyst who made an examination of the organs said that he discovered two hundred and ninety-six grains of pure metallic mercury in the body. The mercury, however, was not the cause of death and did not act as a poison. He found one-seventh of a grain of strychnine in the stomach, liver and kidneys and there was little doubt that strychnine had been the cause of death.[1] In exceptional cases, however, mercury may undergo chemical changes in the body and operate as a poison. A girl, who took four and a half ounces by weight of mercury as an abortifacient, did not abort, but in a few days suffered from mercurial tremors and loss of muscular power. These symptoms continued for two months, but there was no salivation and no blue line on the gums.[2]

In India, mercury is, sometimes, given in food to cause injury. A case is recorded in which liquid mercury was administered to a woman in her food. The woman vomited twice after taking mercury and had redness and swelling of the gums which bled on pressure with the finger.[3] Metallic mercury was introduced into a plantain which was given to a person to eat, but the metal was seen by the intended victim in the portion of the fruit before he ate it.[4] A Mahomedan male of Karachi, in his afternoon meal, was given *dall* and *chapati* for eating by his wife. He suspected *para* in these and reported the matter to the police. All these articles were examined and found to contain metallic mercury and a *kowri* (shell), which were given to the woman by her paramour.[5]

1. *C. J. S. Thompson, Poison Mysteries, p.* 345.
2. *Lancet, Vol. II,* 1873, *p.* 329 ; *Taylor, On Poisons, Ed. III, p.* 360.
3. *Brown, Med.-Leg. Rep., Beng. Pres.,* 1869, *p.* 152 ; *Ibid.*
4. *Bombay Chemical Analyser's Annual Report,* 1921.
5. *Ibid.,* 1927, *p.* 24.

Mercurial vapours are certainly poisonous, and accidents have occurred from their inhalation. A case is recorded by Seidel[1] in which a woman inhaled for some affection or other 2.5 grammes of mercury poured on red-hot coals, and died in ten days with all the symptoms of mercurial poisoning.

Mercury in a finely divided state, when rubbed into the skin as an ointment, is readily absorbed, and produces salivation and other effects of mercurial poisoning. It has also caused death in a few instances when its application was too liberal. Thus, three persons were found dead in bed ; the previous day they had rubbed into the body, for the purpose of curing the itch, an ointment containing 270 grammes of finely divided mercury.[2]

Amalgams which are the alloys of mercury ast as poisons. Stock[3] has drawn attention to a special danger of chronic mercury poisoning by copper amalgam used for stopping carious teeth.

Poisoning by mercuric oxide is very rare. In his annual report for 1929 the Chemical Analyser of Bombay reports the case of a young Christian woman who had taken some red powder given her by a friend as a cure for a headache from which she had been suffering. Within a quarter of an hour she had felt pains in the abdomen and had vomited blood-stained matter. Her stomach was washed out at the J. J. Hospital, and she recovered the next day. About nine grains of red oxide of mercury were separated from the stomach washings, in which it had been plainly visible as a deposit.

A case[4] is also recorded in which red oxide of mercury was given by a woman to her female infant, 6 days old, with intent to kill her, who had some deformity in her legs. The infant became suddenly ill, was unable to suck and was salivating profusely, but she was saved by prompt treatment.

Of all the salts of mercury the chloride and nitrates are responsible for most of the cases of acute poisoning. It should be noted that mercuric salts are more poisonous than mercurous salts. Children bear mercury well, and some persons have idiosyncrasy for mercury salts.

Mercuric chloride is extensively used as a disinfectant and as an antiseptic. Hence accidental cases of poisoning by this salt are likely to occur from the use of too strong a solution used in washing abscess cavities or in irrigating the vagina, uterus or rectum. Cases of poisoning have also occurred from its introduction into the vagina in tablet form as a contraceptive, antisyphilitic or abortifacient measure. C. Haltermann[5] has found records of ten cases of poisoning, where mercuric chloride was inserted into the vagina in tablet form in amounts, varying from 0.25 to

1. *Maschka's Handbuch, II, p.* 295 ; *Blyth, Poisons, Ed. V, p.* 686.
2. *Leiblinger quoted by Blyth, Ibid.*
3. *Med. Klin.,* 1928 ; *Nos.* 29 *and* 30 ; *Leschke, Clin. Toxic., Engl. Trans. by Stewart and* Dorrer, 1934, *p.* 51.
4. *Bombay Chem. Analyser's Annual Rep.,* 1935, *p.* 5.
5. *Zentralbl. f. Gynakol., Sep.* 19, 1925, *p.* 2133 ; *Brit. Med. Jour., Oct.* 24, 1925, *Ep., p.* 61.

3 grammes. Local nercrosis and ulcers occurred especially in the posterior wall. Poisoning was due to absorption of mercury albuminate from the necrotic patches. Seven of these ended in death in one to three weeks. A case is also recorded in which a woman committed suicide by introducing three tablets of corrosive sublimate into the vagina. The whole of the vagina sloughed thus facilitating absorption of the poison from the wound. In such cases it is possible for deposits of mercury albuminate to be formed in the periproctal tissue, hence it is advisable to inject milk at once as a neutraliser into the tissue lying between the vagina and rectum. Remoter lesions are severe parenchymatous nephritis and fatty degeneration of the heart.[1]

Mercuric chloride is often administered internally and an accidental case of poisoning may occur from an overdose. The soloid preparations have been swallowed accidentally, and have given rise to poisoning in some cases.[2] Sometimes, the salt is selected for suicidal poisoning, as also for homicidal purposes.

Zamboni[3] reports the case of a pregnant woman, aged 25, who took, with suicidal intent, six one-gramme tablets of mercuric chloride dissolved in a glass of water. Forty-eight hours later, he saw the patient, who, bent on suicide, refused all aid (gastric lavage was impossible), and her condition was becoming serious every hour. The doctor administered in generous quantities a phleboclyster consisting of an isotonic solution of dextrose. The abdomen had become meteoric. The pain was greatly increased by the condition of pregnancy which was of five months' duration. To stimulate per cutaneous elimination, the patient was given six or seven prolonged hot baths, from which considerable benefit was derived. Oliguria and later anuria which dominated the situation rather than gastro-intestinal symptoms did not change during the first three days in spite of copious endovenous injections of the isotonic dextrose. After the hot baths manifestations in the zone of the uterus announced the pending spontaneous elimination of the fœtus. After five or six hours, with a few pains and weak contractions the fœtus was discharged, and this was followed by profuse hæmorrhage. The fœtus was in a condition of complete maceration. Twenty hours later, the condition of the patient improved, and a few cubic centimetres of dense reddish urine were voided. This was succeeded by an abundant elimination of blood from the urethral tract. After several such eliminations, urine mixed with blood and in more abundant quantities was passed. To stimulate diuresis, the injections of dextrose solution were replaced by intravenous injections of a 40 per cent solution of methenamine. The effect was good, for the amount of urine voided in twenty-four hours increased during the succeeding days to from 500 to 700 c.c. Large doses of potassium iodide were also administered. The patient left the hospital on the twentieth day well on the way to recovery.

In his annual report for the year 1934, the Chemical Examiner, Madras, describes the following cases of poisoning by mercuric chloride. Of these the first two are homicidal and the last suicidal.

1. A man was suspicious of his wife's conduct and there had been frequent quarrels between them. One evening on returning from work he found his wife absent from home. He went in search of her, found her and asked her to return home to serve him food but she refused. As he was hungry he went home and began to eat the food that had been prepared by his wife early in the evening. The food had a queer taste and suspecting that his wife might have poisoned the food,

1. *Rosenthal, Zentralbl. f. Gynak., Jan.* 9, 1926, *p.* 122; *Brit. Med. Jour., Feb.* 20, 1926, *Ep., p.* 36.
2. *Leonard Fuller, Brit. Med. Jour., Jan.* 18, 1913, *p.* 116; *Ferguson Floyd, Ibid., Feb.* 1, 1913, *p.* 220.
3. *Reforma Medica, Naples,* 45, *March* 9, 1929, *p.* 327; *Jour. Amer. Med. Assoc., July* 27, 1929, *p.* 339.

he reported the matter to the village magistrate. The food was forwarded to the Chemical Examiner, who found in it about 17¼ grains of corrosive sublimate.

2. A man was given milk poisoned with corrosive sublimate and he died four days later. In the visceral matters only very small quantities of mercury were found, whereas in the vomits that had been collected there were 25 grains of corrosive sublimate.

3. A man, aged 40, was arrested and escorted by the Police from Palni to Melur. On the way his escort allowed him to drink coffee at a hotel after which he had severe abdominal cramps and vomiting. He was admitted in hospital where he died about a fortnight later. Before his death he confessed to having swallowed perchloride of mercury at the coffee hotel. Extremely minute quantities of mercury were detected in the visceral matters but one of the vomits was found to contain about 1-1/6th grains of perchloride of mercury.

After it is absorbed into the system mercury is eliminated in the saliva, urine and fæces, and in the milk and perspiration, if the quantity is large. It also passes rapidly to the fœtus in utero through the placental circulation. In some cases the elimination is so rapid, that mercury may not be detected in the solid organs, even though death has occurred from its poisoning. Taylor[1] says that it is thus completely eliminated in fifteen days from the system without leaving any trace in the organs ; while according to Witthaus[2] the elimination is rapid and complete in from one to four days if a single dose is given, but it is slow if the poison is given in repeated small doses, and may be detected in the tissues even after thirteen years.

Mercury may be detected in the bones even in acute poisoning. In a case in which a person died in Patiala very suddenly and the body was cremated, the ashes and pieces of bones were forwarded to the Chemical Examiner for analysis. Mercury was detected in the spongy parts of the bones.[3]

Mercury is often used as a medicine ; hence the detection of a small quantity in the viscera does not contraindicate death from some other cause.

Mercury is not a constituent of the human body ; hence its detection in the tissues proves that it must have been introduced into the system from outside.

COPPER (*TAMBA*)

The salts of copper which are important from a toxicological point of view are—

1. **Copper Sulphate (Blue Vitriol, Blue Stone)**, $CuSO_4$, $5H_2O$.— The vernacular name of this salt is *Nila tutia*. It occurs in large, blue, slightly efflorescent crystals, freely soluble in water and having a styptic taste. It is converted into a bluish-white salt, $CuSO_4$, H_2O, when heated to 100° C. It becomes anhydrous at 220° to 240° C. The anhydrous salt is white and extremely hygroscopic. Copper sulphate is given as an astringent in ¼ to 2-grain doses and as an emetic in 5 to 10-grain doses.

1. *On Poisons, Ed. III, p.* 388 ; *Med. Gaz.*, 1850, *Vol.* 46, *p.* 255.
2. *Med. Juris. and Toxic., Vol. IV, pp.* 767-68.
3. *Punjab Chemical Examiner's Annual Report*, 1925, *p.* 3.

In large doses it acts as an irritant poison. It is also probable that small doses of the coarsely powdered salt, repeated frequently, would produce gastric and intestinal irritation and cause death, especially if prescribed when the mucous membrane of the intestinal canal is in a congested state.[1]

2. **Copper Carbonate.**—The normal carbonate has not been obtained, but the basic carbonate, $CuCO_3$, Cu (HO), occurs native as malachite, and is obtained when carbonate of sodium is added to a solution of copper sulphate. Natural verdigris, the green deposit, which appears on copper when exposed to atmospheric moisture and carbon dioxide, is the same compound.

3. **Copper Subacetate (Artificial Verdigris, Aerugo),** $Cu_2C_2H_3O_2$, CuO.—This is known in the vernacular as *zangal.* It occurs in powder, or in bluish-green masses of very minute crystals. It is frequently employed in the arts. It is used externally in medicine.

Acute Poisoning—Symptoms.—These commence from a quarter to half-an-hour after swallowing the poison with a metallic taste in the mouth, burning pain in the stomach, thirst, nausea, eructations and repeated vomiting. The vomited matter is blue or green in colour, and can be distinguished from bile by its turning deep blue on the addition of ammonium hydroxide. The colour does not change in the case of bile. There is diarrhœa with much straining, the motions being liquid and brown, but not bloody. The urine is suppressed or diminished in quantity, and may contain blood. The skin becomes jaundiced, and cramps of the legs or spasms and convulsions occur. There is frontal headache, and the symptoms of collapse set in if the dose is large.

In some cases there is complete paralysis of the limbs, followed by insensibility and coma ending in death.

Fatal Dose.—Uncertain. Half-an-ounce of verdigris[2] has killed a woman, aged 24 years. About half-an-ounce of copper sulphate has proved fatal to a woman, aged 20 years.[3] One ounce of copper sulphate has also proved fatal to an adult.[4] Recovery has followed a dose of 120 grammes or nearly four ounces of copper sulphate.[5] It should be remembered that copper sulphate taken in small doses for some time is supposed to be more dangerous than when a large quantity is swallowed at a time.

Fatal Period..—The usual fatal period is one to three days, but may be prolonged for several days. On the contrary, a woman suicide died from spasm of the glottis soon after she had swallowed a strong solution of copper sulphate.[6] A young lady[7] died after about an hour after the onset of symptoms of poisoning. On analysis copper sulphate equivalent to 38 grains and methylene blue were detected in the stomach and its

1. *Chevers, Med. Juris., p.* 263.
2. *Niemann, Taschenbuch, p.* 458; *Taylor, On Poisons, Ed. III, p.* 442.
3. *Madras Chemical Examiner's Annual Report,* 1931, *p.* 2.
4. *Starr, Med. Record, May* 27, 1882, *p.* 564.
5. *Bernatzic, Encycl. d. Ges. Heilkunde, XI, S.* 433; *Collis Barry, Leg. Med., Vol. II, p.* 411.
6. *Wachholz Ztchr. f. Med. Beamte,* 1893, *VI, p.* 397; *Witthaus, Manual of Toxicology, Ed. II, p.* 708.
7. *U. P. Chemical Examiner's Annual Report,* 1930, *p.* 5.

contents. A Mahomedan female,[1] 24 years old, died within 2 or 2½ hours after she had taken copper sulphate with intent to commit suicide. A child[2] died in 4 hours from an unknown quantity of copper sulphate. A man died in six hours after he had taken an unknown quantity of copper carbonate.[3]

Treatment.—There is no need to use emetics, as vomiting occurs in five or ten minutes after taking the poison. Wash out the stomach with water containing potassium ferrocyanide, which forms insoluble cupric ferrocyanide. Administer white of egg or milk as an antidote. The albumen contained in them will form an insoluble salt, albuminate of copper. Give demulcent drinks. Relieve pain by opium, and use diuretics if the urine is suppressed. Give castor oil to remove the poison from the intestines. Support the patient by giving nutrient enemata, and by using stimulants hypodermically.

Post-mortem Appearances.—The skin may be yellow owing to jaundice. The mucous membrane of the alimentary canal may be congested, swollen, inflamed and excoriated. The contents of the stomach are greenish, and so is the colour of its mucous membrane. The colon, sometimes, shows large ulcerations, and the rectum may be perforated. The liver may be soft and fatty. The kidneys may show the signs of parenchymatous inflammation. In the case reported by Starr[4] the blood of the entire body was found coagulated in the vessels, and changed to a chocolate colour.

Chronic Poisoning.—This may occur among the workers in copper and its salts or its alloys owing to the inhalation of copper dust. It may also occur from food being contaminated with verdigris obtained from dirty copper vessels.

Symptoms.—These are a green or purple line on the gums, a constant coppery taste in the mouth, giddiness, headache, dyspepsia, vomiting, diarrhœa with colicky pain, anæmia, and paralysis; but paralysis is not so common as in lead poisoning.

The skin becomes jaundiced; the hair, urine and perspiration become green.

Mallory[5] of Boston points out that chronic copper poisoning causes the symptom-complex known, under the different names, as hæmochromatosis, bronzed diabetes and pigment cirrhosis.

Treatment.—Remove the cause, and use massage and warm baths. Keep the patient in fresh air, and attend to his diet and dyspepsia. Copper vessels used for cooking purposes should be tinned, and kept scrupulously clean.

Post-mortem Appearances.—The chief post-mortem appearances are fatty degeneration of the liver and degeneration of the epithelial cells of the kidneys.

1. *Bengal Chemical Examiner's Annual Report*, 1931, *p.* 7.
2. *Med. Gazette*, 1836, *Vol. XVIII, p.* 742; *Taylor, On Poisons, Ed. III, p.* 448.
3. *Med. Gaz., Vol. XXXI, p.* 495; *Taylor, On Poisons, Ed. III, p.* 443.
4. *Med. Record, May* 27, 1882, *p.* 564.
5. *Archives of Internal Medicine, Chicago, March* 15, 1926, *p.* 336.

Chemical Tests.—1. Hydrochloric acid and hydrogen sulphide give a brownish-black precipitate, insoluble in ammonium sulphide, but soluble in potassium cyanide, and freely soluble in warm nitric acid.

2. Ammonium hydroxide gives a greenish-blue precipitate, soluble in excess forming a blue solution.

3. Potassium ferrocyanide gives a reddish-brown precipitate of copper ferrocyanide, which is insoluble in acetic and hydrochloric acids, but forms a greenish-blue liquid with ammonium hydroxide.

4. An iron wire or a piece of zinc, if introduced into a solution of a copper salt acidulated with a few drops of hydrochloric acid, soon becomes covered with a red coating of metallic copper. This test may be used in the case of an organic mixture containing copper.

Medico-Legal Points.—1. Copper as a metal is not poisonous. Copper coins, when swallowed, may remain in the stomach or in the intestines for days without producing any poisonous symptoms. However, when alloyed with other metals and reduced to a fine powdery state, copper may act as a poison. All the copper salts are poisonous.

2. The strong metallic taste of copper salts prevents their use for homicidal purposes though, in India, copper sulphate is known to have been used homicidally either mixed with powdered glass, sweetmeat or some other article of food. In his annual report for the year 1935, the Chemical Examiner, Madras, records a case in which a woman of immoral character put copper sulphate in the food intended for her husband. The husband tasted the food and noticed a peculiar burning sensation in the mouth as well as the peculiar colour of the food. The matter was reported to the police, and the woman was prosecuted and sentenced to undergo eight months' rigorous imprisonment.

Copper sulphate has been used, though rarely, as a cattle poison. In his annual report for the year 1907, the Chemical Examiner of the United Provinces of Agra and Oudh mentions a case in which copper sulphate was found in a piece of rag stated to have been inserted into the rectum of a buffalo. In his annual report for the year 1919, the Chemical Analyser of Bombay also mentions some cases of cattle poisoning by copper sulphate.

Suicidal cases are occasionally met with. Sometimes, copper sulphate is taken internally with a view to procure abortion.

Accidental cases occur from contamination of food due to the formation of verdigris resulting from the action of vegetable acids on copper cooking vessels which are dirty and have not been properly tinned.

The author has seen two cases of accidental poisoning. In one case a child playfully swallowed a big crystal of copper sulphate. In the other case an adult woman took it by mistake for a condiment. Both recovered after having suffered from pain in the stomach, vomiting and purging.

3. Poisonous symptoms may occur from the application of the salt to an abraded or raw surface and from its introduction into the vagina.

4. Copper sulphate is added to impart a rich green colouration to preserved and tinned peas, other vegetable substances and pickles, but

the quantity is so small (probably one grain to one pound), that toxic effects are not usually produced and the salt, when taken into the stomach, is very likely converted into harmless albuminate of copper.

5. Copper is a normal constituent of the body, and is found in the liver. It is taken into the system along with food, as it exists in minute traces in almost all the varieties of food, such as cereals, potatoes, beans, spinach, different varieties of fruits, and even in mineral water. Hence the detection of copper in the viscera is of no value unless the quantity found is excessive; however, on account of free vomiting provoked by its salt, a very small quantity may be left in the organs. It is, therefore, essential to examine chemically the vomited matter, whenever available.

6. Copper is eliminated from the system more by the bowels than by the kidneys. It is also excreted in traces in the saliva, bile and milk, and is said to pass to the fœtus *in utero* through the blood of the mother. It is possible that a portion may accumulate very slowly in the body.

Cases.—1. **Acute Poisoning.**—A Hindu lady, aged about 16, took sulphate of copper to commit suicide and died of its effects. The post-mortem examination showed that the stomach was somewhat contracted; there were patches of greenish-blue colour on the peritoneal coat, and the mucous membrane presented a corrugated appearance, stained blue throughout. The stomach contained about eight ounces of a greenish-blue fluid. The whole of the small intestine presented a bluish appearance, most marked in the duodenum. The mucous membrane of the rectum was congested, and it contained greyish-blue coloured fæcal matter. The internal organs were also congested. Sulphate of copper was detected in the vomit as well as in the viscera.—*Bengal Chemical Examiner's Rep.*, 1915; *Ind. Med. Gaz., Aug.*, 1915, *p.* 304.

2. **Chronic Poisoning.**—A. B., aged 20 years, who had been engaged for two and a half years in the brush grinding department of a crucible works, suffered from chronic poisoning by inhalation of dust produced in the grinding of brass. At the time when he came under observation, he was flabby and emaciated, the skin and conjunctivæ were slightly jaundiced and a green line on the gums was present. He complained of colicky pain in the abdomen, dryness in the mouth and throat, and pain in the epigastrium after food. There were loss of appetite and obstinate constipation with considerable straining at stool. There were frequent attacks of faintness and marked shortness of breath on the least exertion. There was progressive emaciation, and tremors on the least excitement lasted for two months.—*H. Roberts, Brit. Med. Jour., Sept.* 11, 1909, *Vol. II, p.* 702.

LEAD (*SHISHA*)

The following are the preparations of lead, which are used in medicine or the arts:—

1. **Lead Acetate,** $Pb\ (C_2H_3O_2)_2,\ 3H_2O$.—This is commonly called sugar of lead or salt of Saturn. It occurs in white masses of acicular crystals, slightly efflorescent and having a sweetish taste. It dissolves in water, forming an acid solution. It looks very much like loaf sugar. It is an official preparation, the dose being ½ to 2 grains. It occurs in the composition of an official preparation, *Suppositorium plumbi cum opio* and a non-official preparation, *Pilula plumbi cum opio* (dose 2 to 4 grains).

2. **Lead Subacetate,** $Pb_2O\ (C_2\ H_3O_2)_2$.—This is the chief constituent of Goulard's extract (*Liquor plumbi subacetatis fortis*), which is a

colourless liquid with a sweet, astringent taste, and alkaline reaction. The extract contains 25% of lead subacetate. Goulard water or Goulard's lotion (*Liquor plumbi subacetatis dilutus*) is prepared by adding 12.5 parts of Goulard's extract to 1000 parts of water.

3. **Lead Carbonate,** $PbCO_3$.—This is a white, crystalline powder, almost insoluble in water, but soluble in dilute acids. In the form of a basic carbonate or white lead (*Safeda*), $(PbCO_3)_2$, PbH_2O_2, it is extensively used as a pigment in oil painting. It is also used as an ointment. Chronic cases of poisoning occur from the use of this salt.

4. **Lead Nitrate,** $Pb(NO_3)_2$.—This is a crystalline, poisonous salt, soluble in water, and is used in calico printing.

5. **Lead Sulphate,** $PbSO_4$.—This is a heavy white powder, insoluble in water and is, therefore, supposed to be non-poisonous, but cases of poisoning have occurred from sucking yarn coloured white with this salt.[1]

6. **Lead Chromate,** $PbCrO_4$.—This is a bright yellow, insoluble powder, known as chrome yellow, and is used as a pigment. Fatal cases of poisoning have occurred from the use of sweetmeats coloured with this salt. Joseph Uttal[2] also reports three cases of chronic poisoning from the use of tobacco snuff adulterated with lead chromate as a colouring agent.

7. **Lead Chloride,** $PbCl_2$.—This occurs as white needleshaped crystals, sparingly soluble in cold water, but more so in boiling water. When heated in contact with air it is converted into an oxychloride, which is employed as a white pigment, known as Pattinson's white lead. The yellow oxychloride obtained by heating lead oxide and ammonium chloride is known as Cassel yellow, and is used as a pigment.

8. **Lead Iodide,** PbI_2.—This is a tasteless, odourless and bright yellow powder, slightly soluble in cold water, but readily soluble in boiling water. It is used in preparing a non-official ointment, *unguentum plumbi iodidi.*

9. **Lead Sulphide (Galena),** PbS.—This is naturally found in the form of cubic crystals, but is sold in the *bazaar* in a powder form as *Surma* in place of sulphide of antimony which is used as a collyrium for the eyes.

10. **Lead Monoxide (Litharge, Massicot).**—PbO.—This is called *Murdasang* in the vernacular. It is a pale brick-red or pale orange scaly mass, very slightly soluble in water, but readily soluble in nitric and acetic acids. It is a constituent of *emplastrum plumbi* (diachylon plaster). Quacks use lead monoxide as a remedy for syphilis. It is also commonly used by painters and glaziers, and is a constituent of certain hair dyes.

11. **Lead Tetroxide (Red lead, Minium),** Pb_3O_4.—This is a scarlet crystalline powder, varying in colour, according to its mode of preparation. It is insoluble in water but partially soluble in nitric acid. It is called *Sindur* or *metia sindur* in the vernacular, and is employed as a pigment. It is used to adulterate snuff to improve its colour. It is frequently used

1. *Brit. Med. Jour., Jan.* 1867.
2. *Jour. Amer. Med. Assoc., Jan.* 28, 1928, *p.* 288.

as an abortifacient and occasionally used as a cattle poison either alone or mixed with white arsenic. A case[1] is recorded in which a young woman gave it to her husband in food but without any ill effects.

Tetra-Ethyl Lead.—This is a clear, heavy oily liquid, somewhat volatile at ordinary temperatures. It has a specific gravity of 1.62, and has a peculiar sweetish odour. It is insoluble in ether and hot or cold water, but soluble in alcohol and acetone, and miscible in all proportions with fats and oils. It decomposes in sunlight with the formation of crystalline triethyl lead oxide which, in the presence of a halogen, forms the triethyl lead salt.

This substance is added to petrol, and is used as a motor fuel. It is possible that it may find access to the organism through the inhalation of dust and fumes, and act as an active and dangerous poison. This possibility aroused alarm in the United States, and a special advisory committee was appointed by Surgeon-General Hugh S. Cumming of the United States Public Health Service to determine whether or not there was a public health hazard in its manufacture and use as a motor fuel. The Committee[2] came to the following conclusions :—

"1. Drivers of cars using ethyl gasoline (petrol) as a fuel, and in which the concentration of tetra-ethyl lead was not greater than 1 part to 1,500 parts by volume of gasoline (petrol), showed no definite signs of lead absorption after exposures approximating to two years.

2. Employees of garages engaged in the handling and repairing of automobiles and employees of automobile service stations may show evidence of lead absorption and storage, as indicated by the lead content of the fæces and the appearance of stippled cells in the blood. In garages and stations in which ethyl gasoline (petrol) was used, the amount of apparent absorption and storage was increased, but the effect was slight.

3. In regions in which ethyl petrol had been used to the greatest extent as a motor fuel for a period of between two and three years, no definite cases have been discovered of recognisable lead poisoning or other disease resulting from the use of ethyl petrol.

4. There are at present no good grounds for prohibiting the use of ethyl gasoline (petrol) as a motor fuel, provided that its distribution and use are controlled by proper regulations."

In conclusion the committee stated that their findings were derived from a relatively small number of individuals, who were exposed to the effects of ethyl gasoline for a period of time comparatively brief, and urged the necessity of further investigations. Hence it is quite natural that the report of the committee cannot be regarded as the last word on the subject. In April. 1928, the Minister of Health,[3] England, set up a Departmental Committee, which in their final report state in the most conclusive terms that the general use of ethyl petrol would involve no danger to public health whatever. Their experiments have proved that the particles of particulate lead in the atmosphere would not be great enough to endanger the health of even drivers and policemen on duty. Moreover, the hazard from exhaust gases and spilling in a well-ventilated garage is

1. *U. P. Chem. Examiner's Annual Rep.*, 1928, *p.* 4.
2. *Jour. Amer. Med. Assoc., Jan.* 30, 1926, *p.* 370 ; Vide *Brit. Med. Jour., Jan.* 14, 1928, *p.* 61, *and March* 3, 1928, *pp.* 363, 366.
3. *Lancet, April* 12, 1930, *p.* 820.

equally minute, and even in badly ventilated garages is not serious. Even those whose skin would be constantly exposed to the liquid would undergo no danger, for it caused no ill-effects whatever even when applied continuously to the shaved skins of rabbits for four months. The committee recommend that the fuel shall continue to be dyed, that all cans and pumps containing it shall be labelled, and that the amount of lead tetraethyl should not exceed 1 part in 1300 by volume or 650 by weight.

Acute Poisoning.—This occurs mostly from lead acetate.

Symptoms.—A sweet metallic taste, dryness of the throat, and intense thirst immediately after swallowing the poison. Vomit occurs within half-an-hour, the vomited matter being white or tinged with blood. Colicky pain comes in paroxysms, but is relieved by pressure. The abdominal walls are tender and contracted. Constipation is a constant feature, though purging has occurred in some exceptional cases, when the stools are offensive and dark or black from the formation of lead sulphide. The urine is scanty. The tongue is coated and the breath is very foul and offensive. Great prostration occurs with cold clammy skin and quick feeble pulse. The nervous symptoms develop; *viz.*, drowsiness, insomnia, headache, vertigo, muscular cramps, convulsions, numbness, and paralysis of the lower limbs. Wasting follows, and death occurs generally from exhaustion.

Sub-acute Form.—The sub-acute form of poisoning results from the administration of repeated small doses of a soluble salt, such as lead acetate. A black line is marked on the gums, and the gastro-intestinal symptoms are usually present. The face is livid and sunken and the look is anxious. The secretions are mostly arrested. The urine is scanty and deep red. The nervous symptoms are more prominent, such as numbness, vertigo, dragging pain in the loins, cramps and paralysis of the lower limbs. Death, though rare, may occur from convulsions and coma within three days.

After apparent recovery the symptoms, sometimes, return probably in an aggravated form, and the illness lasts for a long time.

Fatal Dose.—Uncertain. Lead is not an active poison, though alarming symptoms have been produced even from the medicinal doses of acetate of lead. Recovery has followed one ounce of sugar of lead, or of lead carbonate. One and-a-half ounces of carbonate of lead have proved fatal, and a "knife-pointful" of litharge taken with a view to procure abortion has caused death.[1]

Fatal Period.—Uncertain. A man, aged 26 years, died in about 34 hours after taking red lead.[2] Two children who swallowed Goulard's extract died within 36 hours.[3] An adult who took 1½ ounces of white lead died on the 3rd day.

Treatment.—Administer sodium or magnesium sulphate to form insoluble lead sulphate, and then wash out the stomach with plain water.

1. *Lesser, Vierteljahraschr. f. ger. Med.*, 1898; *Dixonmann, Forens. Med. and Toxic., Ed. VI, p.* 397.
2. *Bengal Chem. Examiner's Annual Rep.*, 1936, *p.* 13.
3. *Collis Barry, Leg. Med. and Toxic., Vol. I, p.* 342.

Dilute sulphuric acid may be substituted for sodium or magnesium sulphate. In the absence of the stomach tube vomiting should be excited by giving simple emetics. Give demulcent drinks, such as barley water, milk or white of egg. Hypodermic injections of morphine and atropine may be given to relieve colic. Hegler[1] recommends the administration of calcium chloride or slow intravenous injection of from 8 to 10 c.c. of a ten per cent solution of calcium bromide for the treatment of colic. Give alkaline purgatives or wash out the bowels by high enemata. Give calcium salts and an alkaline diet consisting of milk, vegetables and potatoes to favour the deposit of lead in the bone marrow. Leschke[2] recommends the administration of calcium gluconate in doses of 0.5 gramme with milk five to six times a day.

Intravenous injections of sodium thiosulphate are considered beneficial in the treatment of acute and subacute poisoning. In mild cases it is advisable to begin with the intravenous injection of 0.6 gramme of sodium thiosulphate dissolved in from 10 to 20 c.c. of distilled water. This dose may be increased gradually to 1 gramme. In severe cases the initial dose may be 1 gramme. The injections have to be continued for about a week.[3]

Post-mortem Appearances.—The signs of acute gastro-enteritis are present. The mucous membrane of the stomach may be thickened and softened with eroded patches, and may be covered with a whitish-grey deposit. The same appearances may be observed in the duodenum.

Chronic Poisoning (Plumbism, Saturnine Poisoning).—This occurs among persons employed in factories and industries in which lead and its salts are used; thus, it occurs among painters, compositors, plumbers, pewters, enamel workers, glass blowers, electric light workers, glaziers, lace workers, lead smelters, card players, etc. It may also result from tinned foods contaminated with lead, from drinking water or cider stored in leaden cisterns and from the constant use of hair dyes and cosmetics containing lead. M. Bodron,[4] Public Health Commissioner, Brest, describes in the *Press Medicale* an epidemic of lead poisoning in which thirty-three persons were affected after eating bread baked in an oven, which was heated with wood that had been obtained by breaking old boats. The wood was found covered with paint that was rich in lead salts. Clayton[5] describes an outbreak of chronic lead poisoning amongst yarn workers at Accrington involving nine women, thus proving greater susceptibility of women to lead poisoning. Children are less susceptible.

Chronic lead poisoning occurs in India from the use of *ghee* (clarified butter) stored in brass or copper vessels lined inside with tin. *Ghee* bcomes impregnated with lead derived from the tin which often contains it as an impurity, and forms a poisonous salt, oleate of lead. The poison may also be conveyed into the system by taking the food cooked in tinned

1. *Deutsche, Medizinische Wochenschrift, Leipzig, April* 14, 1933, *p.* 570; *Jour. Amer. Med. Assoc., July* 1, 1933, *p.* 92.
2. *Clin. Toxic., Eng. Trans. by Stewart and Dorrer,* 1934, *p.* 24.
3. *Hegler, Loc. Cit.*
4. *Jour. Amer. Med. Assoc., Nov.* 25, 1925, *p.* 1981.
5. *Brit. Med. Jour., Feb.* 10, 1906, *p.* 311.

vessels—the common practice in India. Candy[1] quotes the cases of chronic lead poisoning observed by Mankad and Fozdar in Ahmedabad.

Chronic lead poisoning may occur from absorption of lead through the raw or intact skin. Gottheil[2] reports that a patient suffered from chronic lead poisoning after local applications to extensive burns of dilute Burow's solution of aluminium acetate holding in suspension lead sulphate, and died after eight weeks.

Symptoms.—A metallic taste in the mouth ; a black line on the gums, especially of the upper jaw, but it is absent if there are no teeth or if they are kept clean. This is due to the decomposed food in the mouth forming hydrogen sulphide, which forms black sulphide of lead. The patient complains of dyspepsia, becomes emaciated and anæmic, and has a sallow earthy complexion. The pulse is slow and of high tension, the blood pressure being greatly increased. The blood shows the presence of punctate basophilia among the red corpuscles. Interstitial nephritis and general arterio-sclerosis are often present. The urine contains albumin, lead and hæmatoporphyrin.

The chief prominent symptoms are colic and constipation, arthralgia, encephalopathy and paralysis.

1. **Colic and Constipation (Dry Belly-ache).**—Colicky pain felt round the umbilicus is very intense, but is relieved by pressure. The abdominal muscles are retracted, though hard and tense. There is obstinate constipation. Tenesmus is usually present, but diarrhœa is very rare.

2. **Arthralgia.**—The patient complains of rheumatic pain of a shooting nature in the bones and large joints, such as the knees, elbows and shoulders, but the small joints are not affected. Contractions and twitchings of the muscles may be present.

3. **Encephalopathy.**—This involves cerebral and psychical affections, such as intense headache, dizziness, insomnia, anæsthesia, optic neuritis, amaurosis, convulsions, hallucinations, delirium, insanity, eclampsia and coma. Besides these there are other symptoms, *viz.*, vaginismus, abortion in pregnant women and loss of sexual power in men.

4. **Paralysis.**—Paralysis first affects the extensor muscles of the forearm and fingers except the supinator longus and causes " wrist drop " and " claw shaped hand." It then spreads to the extensors of the foot, resulting in " dropped foot ". The tibialis anticus is generally not affected. The muscles begin to waste, and the condition resembles that of acute anterior poliomyelitis.

Tremors, which are increased by movements, are observed in the muscles before paralysis sets in.

Treatment.—This consists in the removal of the patient from the influence of the poison. Medicinal doses of potassium or sodium iodide, parathyroid and parathormone should be administered to assist the

1. *Ind. Med. Gazette, March,* 1933, *p.* 136 ; *see also Ind. Med. Gaz., Oct.,* 1937, *p.* 595.
2. *Jour. Amer. Med. Assoc.,* 1910, 54, *p.* 1056.

elimination of lead through the kidneys. Sodium bicarbonate should be given in large doses of 20 to 30 grammes a day divided in four or five portions, as it increases the output of lead owing to the transformation of the insoluble tribasic lead phosphate to the soluble dibasic phosphate through the liberated carbonic acid.[1] Saline purgatives, such as magnesium sulphate and sodium sulphate, should be given to remove lead from the bowels. An acid diet deficient in calcium, hot baths, sulphur baths, galvanism and massage should also be tried for removing the poison from the system. Hypodermic injections of strychnine hydrochloride may be administered for paralysis.

Adequate exhaust ventilation in lead manufactories, scrupulous personal cleanliness and periodic medical examination of the workers by a factory surgeon to detect the earliest signs of lead poisoning are the chief measures which are recommended to prevent chronic lead poisoning. Every day the workers should take a diet rich in calcium together with a lot of milk and should drink water containing minute doses of sulphuric acid. They should be given four drachms of magnesium or sodium sulphate as a saline purgative once a week.

Post-mortem Appearances.—Not constant. A black line along the margin of the gums. The paralysed muscles are flaccid, and show fatty degeneration. The intestines are contracted and thickened. The liver and kidneys are found hard and contracted, the seat of granular degeneration. The heart may be hypertrophied, and there may be atheroma of the aorta and aortic valves.

Detection of Lead in Urine.—In impending or doubtful cases of plumbism it is necessary to analyse urine and fæces for the presence of lead. Mere detection of lead is not sufficient for a positive diagnosis of lead poisoning, but the actual quantity should be determined, inasmuch as traces of lead may be found in the urine and fæces of healthy people owing to the fact that small quantities of lead are ingested with such articles of food, as sausages, meat, beans, cherries, apples and other fruits. It has been estimated that the average American excretes from 0.02 to 0.08 mgm. of lead per litre of urine and from 0.03 to 0.1 mgm. per gramme ash of fæces.[2] From investigations carried out in Calcutta, Bagchi and Ganguli[3] have shown that the average lead content per litre of normal urine is 0.008 mgm. in Hindus, 0.014 mgm. in Mahomedans and 0.031 mgm. in Anglo-Indians, while the average lead content per litre of normal fæces is about ten times the amount eliminated in the urine of Hindus and Mahomedans and about five times the amount excreted in the urine of Anglo-Indians. The difference in the lead content of the excreta appears to be due to the difference in the lead content of the common food stuffs taken by different communities.

1. *Aub and his collaborators, Lead Poisoning, Baltimore,* 1926; *Leschke, Clinic. Toxic. Engl. Trans., by Stewart and Dorrer,* 1934, *p.* 25.
2. *Kehoe and his colleagues, Jour. of Industr. Hygiene, Sept.,* 1933; *Brit. Med. Jour., April* 28, 1934, *p.* 766.
3. *Ind. Jour. of Med. Res., Vol. XXV, No.* 1, *July,* 1937, *p.* 147; *see also Boyd and Ganguli, Ind. Jour. of Med. Res., Vol. XX,* 1932, *p.* 75.

The quantitative method of determining lead in urine devised by Francis, Harvey and Buchan and modified by Lynch, Slater and Osler[2] is as follows :—

Five hundred cubic centimetres of urine are measured out of a 24-hour sample and evaporated to about 25 c.c. and transferred to a silica flask, all the residue being washed out from the evaporating basin with about 20 c.c. of concentrated nitric acid and dissolved by gentle heating. After cooling, about 6 c.c. of concentrated sulphuric acid are added and heated with further addition of nitric acid, drop by drop, until complete oxidation takes place which is indicated by absence of charring on further heating. The free nitric acid present in the solution is driven off by boiling strongly after diluting with about 40 c.c. of water and adding about 25 c.c. of a saturated solution of ammonium oxalate. The heating is continued for some time more till it is reduced to a small bulk by the decomposition of excess of sulphuric acid indicated by white fumes of sulphur trioxide.

To the oxidation product 5 c.c. of 10 per cent. ammonium acetate and ammonium citrate solutions are added and the mixture is rendered alkaline with ammonia. It is then treated with 2 c.c. of 5 per cent. sodium cyanide solution and transferred to a 150 c.c. separating funnel and extracted with these portions of 0.1 per cent. solution of diphenylthiocarbazone in chloroform (about 20 c.c. in all) and three or four times again with pure chloroform (about 35 c.c. in all) until the last traces of the dye are completely removed from the aqueous mixture. The combined extracts are washed with water and distilled to drive off chloroform. The residue is oxidised by heating with 1 c.c. of concentrated nitric acid and a small crystal of potassium sulphate in a boiling water bath for about thirty minutes after which 0.5 c.c. of concentrated sulphuric acid is added and the heating is continued over a low flame adding nitric acid, drop by drop, as required. When oxidation is complete, the free nitric acid is driven off by boiling strongly with water. After cooling, it is diluted with 20 c.c. of water and transferred to a 50 c.c. Nessler cylinder. An exactly similar cylinder is selected for the standard lead solution, a known amount of which (0.01 mgm. of lead per c.c. of the solution) is carefully measured from a burette and run in the cylinder. Five cubic centimetres of acetate solution are measured in both the cylinders. To each cylinder are then added 2 c.c. of 5 per cent sodium cyanide, 5 c.c. of 6N (approx.) ammonia, water to the 50 c.c. mark and finally 2 drops of 4 per cent sodium sulphide with constant stirring. The brown colour developed in the first cylinder is matched against the standard with the known amount of lead in the second cylinder. By repeating the process of final matching with different amounts of standard solutions an accurate comparison can be made to a limit of 0.005 mgm. of lead.

In these experiments the glassware, reagents, and distilled water must be free from lead or contain such small amounts of lead that they may be neglected. Blank determinations should always be made to see if lead is taken up from the apparatus or any other source during the course of the experiments.

Chemical Tests.—1. Hydrochloric acid produces a white precipitate, soluble in boiling water, but insoluble in ammonia.

2. Hydrogen sulphide produces a black precipitate, insoluble in dilute acids, and caustic potash or ammonia.

3. Potassium iodide gives a yellow precipitate, soluble in boiling water, but reappears on cooling.

4. Potassium chromate or dichromate gives a yellow amorphous precipitate, soluble in potassium hydroxide and strong hydrochloric acid, but insoluble in acetic acid. A yellow precipitate produced by potassium chromate with a copper salt is soluble in acetic acid.

1. *Analyst*, Dec., 1929, *p.* 725.
2. *Analyst*, Dec., 1934, *p.* 787.

5. Sulphuric acid gives a white crystalline or granular precipitate, insoluble in nitric acid, but soluble in hydrochloric acid and ammonium acetate.

Medico-Legal Points.—1. Lead in the metallic form is not poisonous, but it is probably acted upon by the secretion of the intestine, and may act as a poison after it is absorbed into the system as a salt. Lead missiles remaining embedded in the tissues owing to gun-shot injuries have produced poisonous symptoms within a few weeks or even after years.

The compounds of lead are poisonous, provided that they are in a condition fit for absorption, either by the skin, gastric mucous membrane or lungs.

In the absence of air, pure water has no action upon lead, but in the presence of air slightly soluble lead hydroxide is formed. Again, the solvent action of water upon lead is greatly influenced by the presence of chlorides, nitrates, and carbon dioxide dissolved under pressure. Water containing carbonates, sulphates and phosphates has no action on lead.

2. Acute lead poisoning is very rare, and usually terminates in recovery. Hence it has very little toxicological importance, but chronic poisoning is more common, and is very interesting from a hygienic point of view, as it is regarded as an industrial disease.

Cases of chronic lead poisoning may be referred to a medical man under the Workmen's Compensation Act, 1923, for the workmen who contract the disease in the course of and by reason of their employment are entitled to compensation from their employer during such time as they are incapacitated from earning their living, or if death occurs from the disease, the dependants of the deceased are entitled to compensation.

The chief compounds of lead which produce poisonous symptoms are acetate, carbonate, chromate and oxides of lead. The chloride and nitrate do not figure so much in medico-legal work, as they are not easily obtainable by the public.

Homicidal poisoning by lead salts is a rare occurrence.

In Landberg, a town in Bradenburg, a woman gave her husband a powder containing white lead in a glass of beer. Soon after taking it the husband was taken ill with severe colic. As the symptoms recurred repeatedly, a doctor was consulted, who found the patient with a livid complexion and suffering from spasmodic contraction of the intestine, severe constipation which could, in no way, be overcome, and retention of urine. His condition grew worse, and he died about a month later. On the examination of the body the stomach and the intestines were shown to form a dark mass containing sulphide of lead, which was detected in nearly every organ of the body, the total quantity being 19 grains.[1]

Lead is used criminally as an abortifacient. It acts by producing tonic contractions of the uterus and by causing degeneration of embryonic cells and the chorional epithelium. A woman is reported to have successfully aborted after having taken half an ounce of a solution of lead acetate (70 grains to a pint of boiling water) three times a day for about a month. She also suffered from symptoms of chronic lead poisoning.[2]

1. *Lancet, Jan.* 7, 1928, *p.* 48.
2. *J. N. Marshall Chalmers and Sidney Lionel Thompsett, Lancet, April* 30, 1938, *p.* 994.

The paste used for anointing "abortion sticks" often contains red lead as a chief ingredient. The use of diachylon or lead paste as an abortifacient had been so common that on the recommendation of the Pharmaceutical Society the Privy Council of England ordained in May, 1917, that the substance should be included in the first part of the schedule of poisons.

Mixed with arsenious acid red lead is used as a cattle poison.

Most of the accidental cases have occurred from administering a large dose of lead acetate in mistake.

Accidental chronic poisoning has occurred from the use of litharge or lead monoxide (*Murdasang*) as a remedy for syphilis by quacks.

Chevers[1] reports three cases of chronic poisoning from the use of litharge as a remedy for syphilis. In one case a man took twenty-five grains of litharge mixed with white sugar continuously for five days and died on the forty-ninth day. In the other two cases the patients were *sowars* (cavalry men), who swallowed, on three successive days, a powder containing two drachms of litharge and one drachm of "bans lochan". Both of them suffered from lead colic, but recovered. In September, 1923, a young Mahomedan male was admitted into the King George's Hospital, Lucknow, with distention of the abdomen, persistent constipation, severe abdominal colic and muscular weakness as a result of litharge having been administered to him by a quack for the treatment of a syphilitic sore on the penis. He recovered after twenty days' stay in the hospital.

A woman in the third month of pregnancy ingested 50 grammes of lead monoxide, a little at a time, with the aim of inducing abortion. Four days after the first dose she had the symptoms of bilateral pyelitis and neuritis. Abortion took place spontaneously twenty-three days later. She recovered from the abortion and from the lead poisoning.[2]

3. Lead is normally present in almost all human tissues. Recent researches carried out by Bagchi, Ganguli and Sardar[3] have shown that the amount of lead present in individual cases varies according to the difference in the lead content of the food ingested. Lead is retained in large quantities in bone, tooth, hair and nails. The maximum amount of lead is found in hair, especially the black hair of Indian women. The skin is very poor in lead. The ovary is free from lead, while the testicle contains quite an appreciable amount. The fœtal tissues do not show any affinity for lead although it is believed otherwise.

4. Lead enters the system by the skin and by the respiratory tract, but chiefly by the gastro-intestinal tract. It is eliminated largely in the fæces, and to a small extent in the urine. It is also slowly excreted in the bile, saliva and milk. However, being a cumulative poison it tends to accumulate in the system. Chunilal Bose[4] reports that lead was detected in the urine of a man about six weeks after he was poisoned by white lead taken in mistake for betel-lime.

5. Idiosyncrasy plays a great part in the effects of the poison. Some people, even though exposed to the action of lead salts, may not

1. *Med. Juris., Ed. III, pp.* 204, 205.
2. *A. Saturski, Zentralblatt fur Gynakologie, Leipzig, Jan.* 8, 1927, *p.* 102; *Jour. Amer. Med. Assoc., June* 11, 1927, *p.* 1941.
3. *Ind. Jour. Med. Res., XXVI,* 4, *April,* 1939, *p.* 935.
4. *Calcutta Med. Jour., Feb.* 1916.

be affected. Persons addicted to alcohol are more prone to the attack of chronic poisoning. Gouty persons are soon affected; it should, however, be remembered that chronic poisoning develops gout and granular kidneys.

6. Not only does abortion occur in a pregnant woman suffering from chronic lead poisoning usually between the 3rd and 6th months, but a healthy woman, if impregnated by a man suffering from chronic lead poisoning, is likely to abort.

7. Blair Bell, Williams and Cunningham[1] have carried out investigations on the toxic effects in the human subject of lead administered intravenously. The lead used was metallic lead in colloidal form for the treatment of malignant neoplasms. They have shown that there is great difference in individual tolerance to lead, and that the male is more tolerant than the female to the toxic effects of lead, as the following figures tend to demonstrate:—

Maximum and Minimum amount of Lead required to produce Toxic Symptoms: Average for all ages

Maximum.		Minimum.	
Males	Females	Males	Females.
0.34 g.	0.29 g.	0.1 g.	0.04 g.

THALLIUM

Thallium was discovered by William Crookes in 1861 in the seleniferous deposit from a sulphuric acid manufactory. It is a soft, heavy metal, having a tin-white lustrous colour, but, on exposure to the air, tarnishes upon its surface, owing to the formation of black thallous oxide. It is chiefly used in the dye and glass industries.

The chief salts of thallium which are of value from a toxicological point of view are thallium acetate and thallium sulphate.

Thallium acetate was used as a remedy for the night-sweats of phthisis, but is now used only as a depilatory in the treatment of ring-worm of the scalp. It is administered to children under ten years of age in the dose of 8 to 8.5 mgm. per kilogramme of body weight. The hair of the head begins to loosen about the seventh day, and falls out from the fourteenth to the nineteenth day. Thallium acetate is also used for removing the superfluous hair, and is a constituent of some proprietary depilatory creams.

Thallium sulphate is used for killing rats. It is a constituent of rat-poison pastes, known as Zelio-paste and Zelio-grains (corn), which are used in Germany and other countries.

Thallium is a highly poisonous substance, resembling lead in all its characters. Taken in a large dose, it acts as an irritant to the stomach

1. *Lancet*, Oct. 17, 1925, p. 793.

and has a selective action on highly specialised cells of the body, causing marked fatty degeneration in the heart and liver and necrosis in the kidneys.

Acute Poisoning.—The symptoms of acute poisoning occur from a few hours to fourteen days after the administration of a therapeutic dose of thallium acetate due to a personal idiosyncrasy or an overdose through an error of the dispenser. In mild cases the symptoms are joint pains in the legs and feet, loss of appetite, drowsiness and hypochlorhydria. These generally pass off in a few days.

In severe cases the symptoms are dryness in the mouth, difficulty in swallowing, colic, vomiting, diarrhœa, pains in the muscles, joints and nerves, albuminuria, delirium, convulsions, collapse and death. There may be drowsiness followed by coma. After recovery, the patient may suffer from peripheral neuritis, optic atrophy, loss of sight and hearing, and mental disorders.

Chronic Poisoning.—This occurs among workmen employed in a chemical factory where thallium is isolated from pyrites residues. Chronic poisoning also occurs among the persons who use a depilatory cream containing thallium acetate for a prolonged period.

The symptoms consist of restlessness, insomnia, fatigue, loss of appetite, abdominal colic, pains in the lower limbs, tachycardia, epilation, marked eosinophilia, lymphocytosis, sometimes optic atrophy, loss of knee jerks and injury to the endocrine glands.

The falling out of the hair of the head is the most striking and important clinical diagnostic symptom of poisoning by thallium.

Fatal Dose.—Uncertain.

Fatal Period.—The average fatal period is twenty-four to thirty hours, although death occurred in twenty-four hours after ten times the normal dose of thallium acetate.[1] Death has also occurred from the second to the sixteenth day after the administration of therapeutic doses.[2] Two boys, each aged 9 years, died in five days from the effects of 5 grammes of thallium acetate wrongly prescribed for 0.5 gramme.[3]

Treatment.—Wash out the stomach and give large quantities of milk. Administer intravenously 20 c.c. of a 3 per cent solution of sodium thiosulphate per day. Administer sodium iodide and purgatives to aid the elimination of thallium from the system. Give hydrochloric acid.

Post-mortem Appearances.—In the case of three children, aged 10 years, 7 years and 5 years, who died after they had been given by mistake ten times the normal doses of thallium acetate, *viz.*, 39, 36, and 28 grains respectively, the post-mortem examination showed that the stomach contained a little mucus and showed submucous petechial hæmorrhages. The heart showed a remarkable degree of fatty degeneration. The spleen was congested. The kidneys were congested, the glomeruli were swollen,

1. *Roche Lynch and Scovell, Lancet, Dec.* 20, 1930, *p.* 1340.
2. *Brit. Med. Jour., Jan.* 6, 1934, *p.* 26.
3. *Jour. Amer. Med. Assoc., June* 22, 1935, *p.* 2280.

and severe cloudy swelling and necrosis of the cells were also seen in the convoluted tubules. The degenerative changes were more marked in the body of the youngest child who lived for three days.[1]

Chemical Analysis.—The detection of thallium in viscera or urine is carried out as follows[2] :—

A weighed quantity of the material is broken up with hydrochloric acid and potassium chlorate by Fresenius and Babo method and, when solution of all the material except fat has been achieved, the liquid is filtered (well washing the precipitate), and the filtrate boiled until practically all the excess of chlorine has been driven off or removed by the sulphur dioxide method. If necessary, it is filtered again, discarding the precipitate. To the filtrate ammonium chloride and ammonia are added and it is then boiled. The precipitate which consists of iron, calcium and magnesium chiefly in the form of phosphates, is filtered off and discarded. It may be advisable to add a small amount of calcium chloride solution to the liquid before the ammonia to ensure complete removal of phosphate.

The filtrate is then saturated with sulphuretted hydrogen or an excess of freshly prepared ammonium sulphide is added ; a black precipitate is then formed. The precipitate is filtered off, well washed with dilute ammonium sulphide, and finally with distilled water. It consists of thallium sulphide together with traces of other metals which precipitate with sulphuretted hydrogen in alkaline solution, but so far as toxicological analyses are concerned, the only likely metal to be present—and then only in traces—is copper. When the precipitate is completely washed it is dissolved off the paper with hot dilute hydrochloric acid. The thallium is converted into thallous chloride and any trace of the copper which was present as the sulphide remains behind, for this sulphide is insoluble in dilute hydrochloric acid. To the solution ammonia is added in slight excess and the liquid is boiled. If the process of separation has been completed no precipitate should occur at this stage. Any precipitate should be filtered off and discarded. This filtrate is then made very faintly acid, and excess of potassium iodide solution is added. An immediate yellow precipitate of thallous iodide forms. As there is some tendency for thallous iodide to come down in colloidal form, the liquid should be boiled and allowed to stand for twelve hours.

The precipitate is then collected in a weighed Gooch crucible and is well washed, first with potassium iodide solution and subsequently with alcohol, until the washings give no reaction for an iodide. The crucible is then dried at 120° C. until constant weight is obtained. Although thallous iodide is very slightly soluble in water (1 : 17,000), it is almost completely insoluble in potassium iodide solution and in alcohol, so that filtration in the cold and washing with these reagents will give an accurate estimate of the amount present. The iodide after weighing may be confirmed as thallium by dissolving it in a solution of sodium thiosulphate, in which it is only dissolved with difficulty, whereas lead iodide is readily soluble.

1. *Roche Lynch and Scovell, Lancet, Dec.* 20, 1930, *p.* 1342.
2. *Ibid., p.* 1342.

Finally, some of the iodide may be heated in a Bunsen flame on a platinum wire and the characteristic green line in the spectrum obtained.

Medico-Legal Points.—Poisoning by thallium is rare as contrasted with that of lead or mercury, owing probably to the relatively infrequent use of the former in medicine and industry. A few accidental cases of poisoning have occurred from the internal administration of thallium acetate or from the external application of depilatory creams containing thallium acetate. Ramond[1] reports the case of a young girl who suffered from abdominal pain, paralysis of the lower limbs and alopecia after she had used on her face a depilatory cream containing 2.5 per cent of thallium acetate for three months. Mahoney[2] also describes three cases in which three young women suffered from retro-bulbar neuritis from the application of the proprietary depilatory, Koremlu Cream, containing 7.18 per cent of thallium acetate, over their faces, arms and legs for a period of one year and a half.

Suicidal and homicidal cases of poisoning are reported to have occurred from the internal use of rat-poison paste containing thallium sulphate. Greving and Gagel[3] describe a case in which a woman, aged 30 years, who attempted suicide by eating half a tube of Zelio-paste, suffered from great pain, albuminuria, achlorhydria, alopecia, peripheral neuritis, rapid loss of weight, angina pectoris, tachycardia, incontinence of urine and fæces, and amenorrhœa.

Two interesting cases[4] of murder by thallium are recorded. Zelio-paste, a rat poison, was administered in both cases in the liquid drunk by the victims. In the first case a woman, aged 48, drank about 0.909 to 2.728 grammes of thallium sulphate in three months. The symptoms were partly gastric and partly of a nervous nature, which simulated typhoid fever and later progressive paralysis of the insane. Eight months after burial the body was exhumed and was found to be well preserved. On analysis 1.6215 grammes of thallium sulphate were detected in the body. In the second case, a man, aged 40 years, drank 1 to 3 tubes of zelio-paste in his wine and coffee. Polyarthritis was surmised, as the patient complained of pains in the feet, but later gastro-intestinal symptoms supervened, the hair fell out, and the patient died. At the post-mortem examination 1.332 grammes of thallium sulphate were detected in the body. From these cases it is evident that gastro-intestinal and poly-neuritic symptoms together with trophic disturbances of the hair should lead to a suspicion of thallium poisoning.

ZINC (*JASAT*)

The salts of zinc, which are important from a toxicological point of view are—

1. *Presse Medicale,* 1929, *XXXVII, p.* 691 ; *also see Brit. Med. Jour., Feb.* 21, 1931, *p.* 321.
2. *Jour. Amer. Med. Assoc., Feb.* 20, 1932, *p.* 618 ; *also see Jour. Amer. Med. Assoc.,* 1931, *pp.* 1866-1868.
3. *Klin. Woch, VII,* 1928, 1323 ; *Leschke, Clinic. Toxic., Eng. Trans. by Stewart and Dorrer,* 1934, *p.* 31.
4. *Krsek H., Cas. lek. cesk.,* 1934, 40 *(Czech)* ; *Med.-Leg. and Criminol. Rev., Oct.,* 1934, *Vol. II, Part IV, p.* 372.

1. **Zinc Chloride**, $ZnCl_2$.—It occurs as colourless, opaque, deliquescent rods or masses, freely soluble in alcohol, ether and water. It is used in medicine as a caustic. It is contained in the proportion of about 350 grains to the ounce of water in Burnett's fluid, which is used as a disinfectant. It is also used to load textile fibres. Clothes made with these fibres, when worn, produce ulcers and sloughs of the skin, with which they come into contact.

2. **Zinc Sulphate** (White Vitriol, White Copperas, *Safed tutia*), $ZnSO_4$, $7H_2O$.—This is a white, crystalline solid, closely resembling magnesium sulphate and oxalic acid, but having a metallic taste. It is extremely soluble in water, but slightly soluble in alcohol. The pharmacopœial dose is 1 to 3 grains as a tonic and 10 to 30 grains as an emetic. It occurs in *Unguentum zinci oleatis* (Zinc ointment).

3. **Zinc Oxide** (*Jasat bhashm*), ZnO.—This is a soft, white, tasteless, odourless powder, commercially known as *Zinc White ;* it becomes yellow on heating. The oxide is insoluble in water, but dissolves in acids, forming different salts. It is a pharmacopœial preparation, the dose being 5 to 10 grains. It is largely used as a pigment in place of " white lead," which becomes blackened by hydrogen sulphide present in the atmosphere.

Zinc oxide is present in the official preparations of *Gelatinum zinci* (Unna's paste), *Pasta zinci oxidi composita* (Compound paste of zinc oxide) and *Unguentum zinci oxidi.* Mixed with zinc chloride in the form of a paste, zinc oxide is used for filling or stopping carious teeth.

Zinc oxide is not, as a rule, poisonous. An epileptic took as much as one pound in seven months without any ill-effects, the largest quantity taken in one day being seventy grains.[1] A case is, however, recorded[2] where a woman suffered from acute gastro-intestinal catarrh with general weakness and stomach pains after accidentally swallowing 10 grammes of zinc oxide triturated in lemon juice.

The fumes of zinc oxide are highly poisonous.

Zinc Stearate.—This is prepared by precipitating a curd soap solution with zinc sulphate. It is a white, amorphous powder, insoluble in water, and yields 13 to 15 per cent of zinc oxide. It is used as a dusting powder, and may produce poisonous effects from its inhalation.

Acute Poisoning—Symptoms.—A metallic styptic taste, salivation, vomiting, pain in the stomach and abdomen, severe purging, collapse, convulsions and death.

Guilbert and Tardieu[3] report the following rare case of perforation of the stomach resulting from the ingestion of zinc sulphate :

A man, aged 53, swallowed 15 grammes of zinc sulphate in mistake for sodium sulphate, and suffered immediately from severe burning in his mouth, tongue, œsophagus and stomach, followed by salivation, nausea and abdominal pains with vomiting. In an hour hæmatemesis occurred and frequent diarrhœa, with ultimately

1. *Lancet, Mar.* 1, 1862, *p.* 224.
2. *Leschke, Clinic. Toxic., Eng. Trans. by Stewart and Dorrer,* 1934, *p.* 83.
3. *Rev. de Med.,* 1932, *XLIX, pp.* 245-250 ; *The Med.-Leg. and Criminol. Review, April,* 1933, *p.* 164.

profuse sweats and collapse. Twelve hours later, he suffered from painful micturition, polyuria, but no hæmaturia. About two months after the accident he complained of severe pain in the pyloric and duodenal region on palpation. The X-ray examination revealed perforation of the stomach which had led to a diverticulum around the pylorus and the duodenum and produced chronic duodenitis.

If zinc chloride is taken, the corrosive symptoms are more prominent and aggravated. These are burning pain in the mouth, throat, gullet and stomach immediately after swallowing the poison, profuse salivation, dysphagia, metallic taste in the mouth, persistent vomiting tinged with blood and traces of mucous membrane, profuse diarrhœa with blood and tenesmus, great prostration, collapse and death. In prolonged cases aphonia, perversions of the special senses, tetanic spasms of groups of muscles and muscular weakness are usually observed. The local action may lead to severe contraction of the internal organs with which is comes into contact, and may cause stricture of the œsophagus or pylorus.

A girl, 2 years old, drank a spoonful (5 grammes) of a 50 per cent solution of zinc chloride ordered for her mother's endometritis just after a meal when the stomach was full. The child vomited at once. Gastro-enterostomy was performed, when the stomach was found shrunken, shrivelled and its walls were like leather.[1]

A female,[2] 23 years old, drank 500 c.c. of a 30 per cent solution of zinc chloride intended for a vaginal douche. Soon afterwards she suffered from pain on swallowing and also in the stomach, and vomiting, and for many days she vomited all her food. The pharynx was red but not corroded, and the stomach was painful on pressure. A tube could at first be passed into the stomach, but later could not pass beyond the cardiac opening. Three weeks later, the stomach was removed and found to be only 10 cm. long and two fingers in breadth, and was completely occluded at the cardiac and pyloric ends with signs of necrosis and with enormous secondary cicatricial contraction. The patient died after three days of purulent peritonitis.

Fatal Dose.—The smallest fatal dose of zinc sulphate is half-an-ounce,[3] though recovery has occurred after a dose of two ounces. The smallest fatal dose of the solid zinc chloride that has been recorded is six grains, but recovery has followed a dose of two hundred grains.[4] Two drachms of Burnett's fluid is the smallest quantity known that has caused the death of an adult.[5] On the other hand, recovery has followed a dose of three ounces.[6]

Fatal Period.—Death from zinc sulphate poisoning, though rare, has occurred in 2 hours after taking 3 ounces of zinc sulphate,[7] and on the 5th day after taking half an ounce as mentioned in the above case.

Death occurs within a few hours from primary shock and collapse caused by the chloride. Thus, a girl, aged 17 years, died in less than two hours after having swallowed half a wineglassful of Burnett's fluid, and a woman, aged 28 years, died in four hours after she had taken an ounce of a strong solution of zinc chloride.[8] A man of 62 years died in 4½ hours

1. *T. V. Cosanky, Jahrbuch, fur. Kinderheilkunde, Berlin,* 1921, 95, *No.* 5-6, *p.* 339 ; *Jour. Amer. Med. Assoc., Nov.* 5, 1921, *p.* 1532.
2. *Pfeiffer, Deuts. Zeits. f. d. ges. gerichtl. Med.,* 1932, *XIX, pp.* 1-23 ; *The Med.-Leg. and Criminol. Rev., Jan.,* 1933, *p.* 80.
3. *Marsh, Med. Times and Gaz., Sep.* 6, 1862, *p.* 252.
4. *Holland, Med. Chem. and Toxic., Ed. V, p.* 357.
5. *Cousins, Med. Times and Gaz.,* 1862, *Vol. II, p.* 404.
6. *Hossall, Lancet, Vol. II,* 1853, *p.* 259.
7. *Buchner Friendreich's Bl. J. ger. Med.,* 1882, *XXXIII, p.* 255 ; *Witthaus, Med. Juris. and Toxic., Vol. IV, p.* 785.
8. *Taylor, On Poisons, Ed. III, p.* 480.

after having taken 2 ounces of " soldering liquid " made by adding zinc to hydrochloric acid.[1]

In some cases the primary effects may be recovered from and the patient may die weeks or months afterwards from inanition or perforation. Tuckwell[2] reports a case in which death occurred from the secondary effects of disorganisation of the stomach and stricture of the œsophagus one hundred and sixteen days after a dose of 4 ounces of Burnett's fluid.

Treatment.—Emetics need not be given as zinc sulphate produces vomiting, but it should be promoted by giving warm water or warm milk and by tickling the fauces. Wash out the stomach with water containing sodium carbonate except when zinc chloride has been taken. The antidotes are eggs, milk and vegetable artringents containing tannin, such as strong decoctions of green tea. Treat the symptoms as they arise. For instance, give opium to relieve pain, and warmth and stimulants to combat collapse.

Post-mortem Appearances.—The usual consequences of irritant poisoning, *viz.*, redness and congestion in the mouth, gullet, stomach and intestines, are to be seen if zinc sulphate has been taken ; but their mucous membrane will be whitened, detached and corroded if zinc chloride has been used. There may be ulceration and even perforation of the stomach.

In the case of a widow, aged 53, who died in twenty-four hours after taking at least an ounce of zinc sulphate the mucous membrane of the stomach showed patches of intense inflammation, but this was more marked in the small intestine which was inflamed throughout. So vivid was the congestion of the small intestine that it was plainly visible from the outside before it was slit up. The large intestine was also inflamed in patches.[3]

Chronic Poisoning.—This occurs among zinc smelters who inhale the fumes. It has also resulted from drinking water or milk stored in zinc vessels. Gimlette[4] describes an epidemic of zinc poisoning through drinking contaminated water among Sikh and Pathan soldiers stationed at Pahang in the Malay States. The water-supply was obtained from the rainfall collected from the galvanised iron roofs of the barracks by means of zinc gutters and down spouts leading into galvanised iron tanks.

Symptoms.—Digestive disturbances ; dyspepsia ; colic with constipation, but more often diarrhœa ; anæmia ; peripheral neuritis leading to paralysis.

Chemical Tests.—1. Ammonium sulphide in an alkaline solution gives a white precipitate, insoluble in caustic potash or ammonia, but soluble in mineral acids.

2. Caustic potash, soda or ammonia throws down a white precipitate, soluble in excess of the reagent.

3. Potassium ferrocyanide gives a white gelatinous precipitate, insoluble in hydrochloric acid. Magnesium sulphate and oxalic acid which resemble zinc sulphate, are not precipitated by potassium ferrocyanide.

1. *Crosse, Brit. Med. Jour.*, 1883, *Vol. II*, *p.* 820.
2. *Brit. Med. Jour.*, 1874, *Vol. II*, *p.* 297.
3. *Mackintosh, Brit. Med. Jour.*, *Vol. II*, 1900, *p.* 1706.
4. *Brit. Med. Jour.*, *Sep.* 7, 1901, *p.* 615.

4. Potassium ferricyanide gives a brownish-orange, yellow or fawn coloured precipitate.

Medico-Legal Points.—Zinc is soluble in the weak acids of food; hence acute poisoning may occur accidentally from eating food cooked in zinc-lined vessels.

A sudden outbreak[1] of zinc poisoning occurred amongst the inmates of a large institution near London. About 400 persons were served at tea with stewed apples cooked in galvanised iron vessels. Within a few minutes more than 200 of those who partook of the stew complained of dizziness, colic and tightness in the throat. There was some diarrhœa. Only ten persons were at all seriously ill, and all of them were able to carry out their ordinary work next day, so that obviously the effects of the poisoning soon passed off. A chemical examination of some of the stewed apples remaining from the meal showed that they contained 7 grains of zinc, expressed as zinc oxide, in the pound; this is equivalent to 25 grains of hydrated zinc sulphate to the pound.

Poisoning by zinc salts is very rare indeed. Accidental poisoning has occurred from zinc sulphate having been taken in mistake for magnesium sulphate. Cases are recorded in which it was taken with intent to commit suicide or to procure abortion. It was administered homicidally in about four instances.[2] Zinc chloride has been used suicidally, but rarely for homicidal purposes. Poisoning by this salt has occurred from its application to a wound or to a raw cancerous surface, from injection of a 50 per cent solution into the rectum in mistake for glycerol,[3] as also from vaginal douching with a solution of 1 drachm to 1 litre of water.[4] Burnett's fluid has caused poisonous symptoms through being mistaken for fluid magnesia. Chevers[5] mentions three cases of poisoning by Burnett's fluid occurring in the Calcutta Medical College Hospital. In one of these a student swallowed about two ounces, mistaking it for syrup and died in about a week. In two other cases the patients recovered as the fluid was accidentally taken on a full stomach soon after dinner.

A painter is reported to have died from shock caused by caustic burns from molten zinc chloride splashed on his body from a boiling drum.[6]

Zinc stearate causes poisonous symptoms in children from accidental inhalation of the powder, and produces interstitial pneumonia and peribronchial inflammation. Schlaepfer[7] reports the case of a child, aged $7\frac{1}{2}$ months, who died thirty-four hours after inhaling zinc stearate. Cyanosis and dyspnœa were the principal symptoms, and a state of "acidosis" was noted twelve hours before death. At the necropsy the lungs were found voluminous. The bronchioles contained plugs of zinc stearate and mucus, which appeared as wormlike masses. Extensive areas of emphysema were separated from each other by small scattered, atelectatic zones.

The salts of zinc are eliminated from the system chiefly by the bowels and to a slight extent by the kidneys. Zinc may be found in a small amount in the body after death owing to its absorption by food kept in zinc or galvanised iron vessels.

1. *Brit. Med. Jour., Feb.* 3, 1923, *p.* 201.
2. *Witthaus, Med. Juris. and Toxic., Vol. IV, p.* 713.
3. *Ibid.*
4. *Brit. Med. Jour., Dec.* 2, 1922, *Ep., p.* 79.
5. *Med. Juris., Ed. III, p.* 297.
6. *Times of India, Aug.* 19, 1936, *p.* 10.
7. *Amer. Jour. of Diseases of Children, Chicago, April,* 1926, *p.* 474.

BISMUTH

The salts of bismuth which are commonly used in medicine are—

1. **Bismuth Carbonate (Bismuth Oxycarbonate** or **Bismuth Subcarbonate)**, $2(Bi_2O_2CO_3)$, H_2O.—It is a heavy powder, insoluble in water, but soluble with effervescence in nitric and hydrochloric acids. It is a pharmacopœial preparation, the dose being 10 to 30 grains. It occurs in the composition of *Trochiscus bismuthi compositus*, $2\frac{1}{4}$ grains being contained in each.

2. **Bismuth Subnitrate (Bismuth Oxynitrate)**, $BiONO_3$, H_2O.—It is a heavy, white powder in minute crystalline scales. It is insoluble in water and alcohol, but soluble in dilute nitric acid. It is known as *magistery of bismuth*, and is, sometimes, used as a cosmetic under the name of *pearl white*. It is a non-official preparation, the dose being 5 to 20 grains.

3. **Bismuth Salicylate (Bismuth Oxysalicylate)**, $BiOC_7H_5O_4$.—It is a heavy white, amorphous powder, having no taste or odour. It is insoluble in water, alcohol and glycerine. It is an official preparation, the dose being 10 to 30 grains, and 1 to 2 grains intramuscularly. It is a constituent of *Injectio bismuthi salicylas* in the proportion of 2 grains in 20 minims. The dose is 10 to 20 minims intramuscularly.

4. **Precipitated Bismuth.**—It is prepared by the reduction of a solution of bismuth trichloride in hydrochloric acid by means of hypophosphorous acid. It is a grey, insoluble powder, easily diffusible in water. It is an official preparation, the dose being $1\frac{1}{2}$ to 3 grains intramuscularly. It is contained in *Injectio bismuthi* (Bismostab), the dose of which is 8 to 15 minims intramuscularly.

The organic salts of bismuth which have recently come into use in medicine are bismuth subgallate (dermatol), bismuth oxyiodogallate (airol), bismuth arsphenamine (bismarsen), bismuth stovarsol (bistoval) and certain other preparations, popularly known by the trade names of trepol, neotrepol, muthanol, etc.

Symptoms.—A metallic taste; salivation; pain in the throat and abdomen; sore mouth; vomiting; purging, the stools being greyish-black; a violet black line is formed on the gums which may be inflamed, ulcerated or even gangrenous; the garlic-like odour (bismuth breath) probably due to the presence of tellurium as an impurity; the weak and feeble pulse; pain over the præcordial region; suppressed or scanty urine which is dark and contains albumin and casts; collapse and lastly death.

W. H. Reznik[1] reports a case of bismuth poisoning in a woman suffering from diabetes after she had taken internally 5 to 7 ounces of bismuth subnitrate in a fortnight. The symptoms comprised a bluish-black discoloration of the gums, which were swollen and inflamed; a similar discoloration of the tongue, most noticeable at the apex of the papillæ and arranged in vertical striations along the lateral margins; a patchy diffuse discoloration of the buccal mucosa; swelling and tenderness of the parotid glands; moderate anæmia and basophilic stippling of the red cells. Bismuth was detected in the urine. Recovery followed the withdrawal of the salt.

Paul Blum[2] observes that in cases of syphilis treated by the intravenous injections of bismuth salts stomatitis appears to have been most frequently observed, although gastro-intestinal, renal and hepatic lesions have been described. Cases have been reported in which patients under treatment of intravenous injections of bismuth salts developed quite suddenly severe albuminuria, followed by the passage of epithelial, granular and hyaline casts. He emphasises the point that the lesions in the mouth and intestine are the first signs of bismuth poisoning—they are the danger signals which indicate the necessity for a systematic examination of the urine.

1. *Bull. John Hopkins Hospital, May*, 1926, *p.* 323; *Brit. Med. Jour., June* 26, 1926, *Ep., p.* 107.
2. *Paris Medical, July* 29, 1922, *p.* 105; *Brit. Med. Jour., Oct.* 28, 1922, *Ep., p.* 58.

Fatal Dose.—A dose of two drachms of bismuth subnitrate has caused the death of an adult.[1] Recovery[2] has, however, occurred after a dose of one ounce given in milk for X-ray examination of the stomach.

Fatal Period.—Nine days in the above case. A man died within less than five minutes after an intravenous injection of 15 mg. of bismuth tartrate suspended in 5 c.c. of sterile distilled water.[3] A Hindu male died within two hours after an intramuscular injection of neotrepol into the gluteal region.[4] A case is cited in which the injection of bismuth paste into the left knee joint was followed by death in about six weeks.[5]

Treatment.—Use the stomach tube or emetics. Give intravenous injections of sodium thiosulphate. Administer demulcents. Give ice to relieve vomiting and opium to relieve pain. Give purgatives and clear the bowel by high enemata.

Post-mortem Appearances.—Those of acute gastritis. In the case mentioned above the throat, larynx and œsophagus were inflamed and there was inflammatory redness in the stomach and throughout the intestinal canal.

In the case of a male infant, 1 month old, who died within 44 hours after having been administered 190 grains of bismuth subnitrate as a remedy for diarrhœa, the skin was grey and cyanotic as also the viscera. The bowels were moderately distended with gas. A very small quantity of green watery fæcal matter was found in the large bowel but no blood or pus was seen and no inflammation or ulcerations were present. The blood was of a dark chocolate brown colour, typical of methæmoglobin, and spectroscopically it gave a very positive reaction for methæmoglobin.[6]

Tests.—1. Hydrogen sulphide in a weak acid solution gives a black precipitate, insoluble in ammonia, but soluble in strong nitric acid.

2. Potassium chromate or bichromate yields a yellow precipitate, soluble in nitric acid, but insoluble in potassium hydrate.

3. Water Test.—Hydrochloric acid gives a white precipitate, soluble in excess. To the solution thus obtained if a large quantity of water is added, a white precipitate, insoluble in tartaric acid, is obtained. The same test is applicable in the case of antimony, but the white precipitate is soluble in tartaric acid.

Medico-Legal Points.—The salts of bismuth are ordinarily non-poisonous. Large quantities (1 to 4 ounces), especially of the carbonate and subnitrate, mixed with gruel or bread and milk, are used as a bismuth meal for X-ray examination of the œsophagus, stomach and intestines, as they obstruct the passage of the X-rays. These salts are more readily absorbed by abraded surfaces, and poisonous cases have resulted from the use of bismuth paste for the treatment of sinuses, abscess cavities and burns.

Taken internally, bismuth carbonate has produced poisoning in some instances owing to its conversion into soluble chloride.[7] Bismuth subnitrate has produced poisonous symptoms possibly owing to the presence of the nitrite. It has produced fatal poisoning in children from its reduction to nitrite by the action of putrefactive fæcal bacteria in the large intestine. The symptoms exhibited in such cases are cyanosis, diarrhœa, methæmoglobinæmia, dyspnœa, collapse and death from failure of respiration. Bœhme[8] reports two fatal cases of poisoning in infants caused by the reduction of the subnitrate to nitrite, and also suggests that infant but not adult fæces reduce bismuth subnitrate to nitrite.

1. *Taylor, On Poisons, Ed. III, p.* 487.
2. *Phillips, Cleveland Med. Jour., June,* 1917, *p.* 419.
3. *Stephen H. Curtis, Jour. Amer. Med. Assoc., Nov.* 22, 1930, *p.* 1588.
4. *Chenoy, Ind. Med. Gaz., May,* 1926, *p.* 234.
5. *Phillips, Cleveland Med. Jour., June,* 1917, *p.* 419.
6. *Harold F. Roe, Med. Jour. Amer. Med. Assoc., July* 29, 1933, *p.* 352.
7. *Brit. Med. Jour., Oct.* 28, 1922, *Ep., p.* 58.
8. *Arch. f. Exp. Path.*, 1907, *LVII, p.* 441 ; *Witthaus, Manual of Toxic., Ed. II, p.* 687.

Bismuth subnitrate is more soluble in the stomach of a dyspeptic patient owing to the presence of butyric and lactic acids. It should, therefore, be prescribed with caution in such cases, lest toxic effects be produced.

Bismuth is eliminated from the system in the fæces, urine and saliva. Like lead, the greater portion of it passes out either unaltered from the bowels, or becomes converted into bismuth sulphide imparting a black or dark-brown colour to the fæces.

SILVER (*CHANDI*)

The only salt that has any toxicological value is *silver nitrate* ($AgNO_3$), also known as *lunar caustic* or *lapis infernalis*. It is administered internally in pill form in $\frac{1}{8}$ to $\frac{1}{4}$-grain doses. It occurs as large colourless, rhombic crystals. Mixed with potassium nitrate it is moulded into white or greyish-white cylindrical rods or cones, and is known as *toughened caustic* or *argenti nitras induratus*. It is freely soluble in distilled water. Its solution has a styptic metallic taste and acid reaction.

Silver nitrate is used externally as a styptic and as a destroyer of exuberant granulations and warts. It is also used in photography, and constitutes a chief ingredient of indelible ink and hair-dyes.

Argyrol and Protargol, which are the organic preparations of silver, are largely used in ophthalmic practice. Collargol (silver in a colloid state) is opaque to the X-rays, and is, therefore, used in a 20 per cent solution for injecting into the ureter and renal pelvis for the purposes of diagnosis.

Acute Poisoning.—Cases of acute poisoning have occurred from the accidental slipping of lunar caustic while applying it to the throat. When thus swallowed, it acts as a corrosive poison.

Symptoms.—Severe pain in the throat and stomach and vomiting. The vomited matter is at first flaky white, but becomes black on exposure to light, and may contain blood. These are followed by frequent motions, the stools, sometimes, containing blood. Cramps, convulsions and collapse precede death.

Fatal Dose and Fatal Period.—Uncertain. Thirty grains of silver nitrate have caused the death of an adult. A woman, aged 51 years, died in three days, after she took, in divided doses, 50 grains contained in a mixture of 6 ounces.[1] A child, 15 months old, died in violent convulsions in 6 hours, when a piece of lunar caustic slipped down his throat.[2] A case of recovery is recorded in which an old man swallowed 45 grains of silver nitrate in three doses of 15 grains each within two hours.[3] Recovery has also occurred after swallowing one ounce with the intent of committing suicide.[4]

Treatment.—Give sodium chloride as an antidote to form insoluble silver chloride. Produce vomiting by administering ipecacuanha powder, or hypodermic injection of apomorphine hydrochloride. Give demulcent drinks, eggs and milk. Give opium and stimulants.

Post-mortem Appearances.—The local action of the caustic will be evident by stains, at first white, but becoming black on exposure to light. These stains are noticed on the mouth, on the mucous membrane of the alimentary canal touched by the poison, as also on the white clothing. The signs of gastro-intestinal inflammation are present.

In the case[5] of an insane patient, aged 31, who died on the fourth day after swallowing one stick of silver nitrate with suicidal intent, the stomach and duodenum showed inflammation of the mucous membrane, and the internal organs showed slight parenchymatous changes.

1. *Taylor, On Poisons, Ed. III, p.* 493.
2. *Scattergood, Brit. Med. Jour., May* 27, 1871, *p.* 527.
3. *Chatterjee, Ind. Med. Gaz., March,* 1868, *p.* 61.
4. *Orfila, Toxic., Vol. II, p.* 22; *Witthaus, Med. Juris., Vol. IV, p.* 305.
5. *Jour. Amer. Med. Assoc., Jan.* 21, 1928, *p.* 219.

Chronic Poisoning.—This results from the long continued use of an organic or inorganic silver salt as a medicine or from its long application to the granulations of wounds and ulcers. It also affects those who constantly come in contact with silver salts owing to their occupation.

Symptoms.—These are a black line on the gums and a general discoloration of the skin (*argyria*) due to the deposition of minute silver particles in the cutaneous tissues. This discoloration is permanent, greyish-blue or dark-grey in colour and first affects the lips, inside of the cheeks, gums, nostrils, eyelids and lastly the chin. It also affects the viscera, chiefly along the walls of the smaller blood vessels. Albuminuria and paralysis of the extensor muscles common in lead poisoning are also met with.

Smith records the case of a patient who took silver nitrate in $\frac{1}{4}$-grain doses three times daily for three months for nasal catarrh, when the skin of the face and hands—the exposed surfaces—assumed a darker colour.[1]

Olson[2] cites a case of argyria following the local use of argyrol. A woman sustained a fracture of the nose, the laceration of the soft parts extending to the right lower eyelid. In addition to other measures argyrol was dropped into her right eye. The argyrol made its way to the lacerated tissue of the right lower eyelid, nose and cheek, and caused bluish-green and slate-grey pigmentation. Goldstein[3] reports a case in which the face, lips and hands became of a peculiar slate-blue colour from the local application of argyrol to the throat twice daily for a year.

Treatment.—No treatment of any kind is available for the removal of argyria, although the intradermal injection of equal parts of 12 per cent sodium thiosulphate solution and 2 per cent potassium ferricyanide solution has been recommended,[4] but Dr. Acharya, Professor of Ophthalmology, has not found this method successful in the King George's Hospital, Lucknow.

In the case cited by Oslon five grains of hexamethylamine were given three times a day, when argyria improved to some extent. The dose was then increased to ten grains, when after six weeks the signs of irritation in the stomach and kidneys were evident.

Post-mortem Appearances.—Pigmentation in the corium, liver and kidneys, Dark colouration of serous and mucous membranes.

Chemical Tests.—1. Hydrochloric acid gives a white, curdy precipitate, insoluble in nitric acid, but readily soluble in ammonia and potassium cyanide.

2. Potassium iodide gives a pale yellow precipitate, insoluble in ammonia.

3. Potassium bichromate gives a brick-red precipitate, soluble in ammonia.

4. Hydrogen sulphide or ammonium sulphide gives a dark brown precipitate of silver sulphide.

Medico-Legal Points.—Cases of silver poisoning are mostly accidental. Of the suicidal cases one was that of an adult male who recovered after swallowing about an ounce of silver nitrate, and the other was that of an insane person who died after swallowing a stick of silver nitrate. A case is also recorded in which a soldier applied lunar caustic to his cornea to evade military duty.[5]

Silver is partly eliminated in the urine and fæces, but a great deal is retained in the system, and deposited in the tissues.

IRON (*LOHA*)

The pharmacopœial preparations of iron salts, which are largely used in medicine, are mostly prepared from sulphate and perchloride of iron. These two salts produce poisonous symptoms when administered in large doses.

1. *Illinois, Med. Jour., Dec.*, 1920, *p.* 517.
2. *Jour. Amer. Med. Assoc., July* 14, 1917, *p.* 87.
3. *Ibid., Nov.* 5, 1921, *p.* 1514.
4. *Weymann, Jour. Amer. Med. Assoc., Nov.* 18, 1929, *p.* 1367.
5. *Witthaus, Med. Juris. and Toxic., Vol. IV, p.* 305; *Berl. kl. Wchnschr.*, 1891, *XXVIII, p.* 466.

Iron Sulphate (Ferrous Sulphate), $FeSO_4$, $7H_2O$.—This is commercially known as green vitriol or copperas, and is called *Kasis* in Hindustani and *Hirakashi* in Gujarati. It forms green monosymmetric crystals effloresceing on exposure to the atmosphere. It is freely soluble in water. The official dose is 1 to 5 grains. It is used in making blue black ink and dyes.

Perchloride of Iron (Ferric Chloride), Fe_2Cl_6, $12H_2O$.—This is an extremely deliquescent salt, rapidly soluble in water. When its watery solution is slowly evaporated, yellow crystals are formed. When dissolved in alcohol, it forms a non-official preparation, called *Tinctura ferri perchloridi* (tincture of iron). The watery solution is a pharmacopœial preparation, known as *Liquor ferri perchloridi*. The doses of both these preparations are five to fifteen minims.

The following are the other pharmacopœial preparations derived from iron :—

1. *Syrupus Ferri Phosphatis Compositus*.—This is called Parish's food. Dose, 30 to 120 minims.

2. *Syrupus Ferri Phosphatis cum Quinina et Strychnina*.—This is called Easton's syrup. Sixty minims contain $\frac{1}{2}$ grain of iron, 4/5 grain of quinine sulphate and 1/60 grain of strychnine hydrochloride. Dose, 30 to 60 minims.

3. *Syrupus Ferri Iodidi*.—Two fluid drachms contain $7\frac{1}{2}$ grains of ferrous iodide. Dose, 30 to 120 minims.

4. *Ferrum Redactum*.—It is a fine, greyish-black powder, insoluble in water and in alcohol, but freely soluble in dilute hydrochloric acid. It is strongly attracted by the magnet. Dose, 1 to 10 grains.

5. *Ferri Sulphas Exsiccatus*.—This is a greyish-white powder, slowly but entirely soluble in water. Dose, $\frac{1}{2}$ to 3 grains. It enters into the composition of *Pilula ferri carbonatis* (Blaud's pills), dose, 5 to 30 grains and *Pilula aloes et ferri*, dose, 4 to 8 grains.

6. *Ferri Carbonas Saccharatus*.—It is a greenish-brown powder, having a sweet, feebly chalybeate taste. Dose, 10 to 30 grains.

7. *Ferri et Ammonii Citras*.—It occurs as deep-red, transparent scales, freely soluble in water, but almost insoluble in alcohol (90 per cent). Dose, 5 to 15 grains. It occurs in *Injectio ferri*, dose being 15 to 30 minims intramuscularly.

8. *Ferri et Quininæ Citras*.—It occurs as greenish-yellow scales, with a bitter and chalybeate taste. It freely dissolves in water. Dose, 5 to 15 grains.

Symptoms.—An inky, metallic taste in the mouth ; violent pain in the stomach and abdomen ; vomiting ; purging with black motions ; suppression of urine ; collapse and death. Sometimes, there are convulsions and paralysis of the extremities.

Fatal Dose.—The fatal dose of ferrous sulphate is not known. A girl, who swallowed an ounce of the salt recovered, though she suffered for some hours from violent pain, vomiting and purging.[1] One ounce and-a-half of the ferric chloride tincture has proved fatal,[2] though recovery has followed a dose of 3 ounces.[3]

Fatal Period.—Uncertain. Five weeks after a dose of one ounce and-a-half of the tincture of ferric chloride was taken. Three minims of the tincture injected into the nævus of a child, 9 months old, caused death in 5 minutes.[4]

Treatment.—Wash out the stomach with the stomach tube, or administer emetics. Give sodium carbonate or bicarbonate dissolved in a large amount of water or milk, demulcent drinks, opium and stimulants, if necessary.

1. *Christison, On Poisons, p.* 506 ; *Hall, New York Med. Jour.,* 1883, *XXXVIII, p.* 401.
2. *Ibid.*
3. *Dubin, Med. Press, Feb.* 21, 1849.
4. *Lancet, Feb.* 7, 1874.

Post-mortem Appearances.—Appearances as those of acute gastro-enteritis. The mucous membrane of the stomach is inflamed, and thickened towards its pyloric end.

Chemical Tests.—1. Alkaline solutions of ferrous and ferric salts yield, on the addition of ammonium sulphide, a black precipitate, soluble in hydrochloric and nitric acids.

The reactions which distinguish the ferrous from ferric salts are given below in a tabulated form :—

Reagents.	Ferrous Salts.	Ferric Salts.
1. Hydrogen sulphide in acid solution.	No precipitate.	White precipitate and reduces to ferrous.
2. Ammonium sulphide.	Black precipitate.	Brownish-black precipitate.
3. Potassium Ferrocyanide.	Prussian-blue precipitate.	Reddish solution but no precipitate.
4. Potassium ferricyanide.	White precipitate turning blue on exposure.	Blue precipitate. (Turnbull's blue).
5. Potassium sulphocyanide.	No precipitate.	Intense blood-red colour.
5. Acid Tannic.	Black precipitate.	Greenish-black, inky precipitate.

Medico-Legal Points.—1. Iron is a normal constituent of the body, it being present in the colouring matter of the blood. It is also present in food and is often a constituent of tonic medicines.

2. Ferrous sulphate has been administered with criminal intent in coffee.[1] It has been used as a cattle poison, especially to kill sheep.

Perchloride of iron has been given for homicidal purposes to persons in an intoxicated condition. Poisonous, irritant symptoms have followed the use of iron chloride as an injection into the uterus. Both the sulphate and the chloride have been used in poisonous doses to procure abortion.

3. Iron is eliminated in the fæces and urine.

TIN (*KALAI*)

The only salts that are of any toxicological interest are stannous and stannic chloride. They occur as whitish-yellow crystals, but, being deliquescent, are met with in acid watery solutions. A mixture of these two chlorides in solution is known as *Dyers' Spirit* and is used as a mordant in calico-printing.

Symptoms.—A metallic taste in the mouth ; nausea accompanied by vomiting ; pain in the abdomen ; purging ; feeble, irregular pulse ; cyanosis ; headache ; great depression ; collapse ; unconsciousness or drowsiness.

Fatal Dose.—Not known. Half a drachm of tin chloride solution has caused death. Four to ten grains of mallate of tin have proved fatal in children.

Fatal Period.—Not known.

Treatment.—Emetics or the stomach tube should be used. Eggs, bland demulcent drinks, stimulants and anodynes should be next administered.

Post-mortem Appearances.—Not known ; probably those of gastro-enteritis.

1. *Ann. de Therap.*, 1872, *p.* 146.

Chemical Tests.—1. Sulphuretted hydrogen yields with stannous solutions a dark brown precipitate, and with stannic solutions a yellow precipitate. Both precipitates are soluble in ammonium sulphide.

2. Mercuric chloride gives a white precipitate with a stannous salt, which turns grey and lastly black on boiling with excess of the reagent.

3. Gold chloride produces a purple precipitate with a stannous salt, but none with a stannic salt.

Medico-Legal Points.—1. Poisoning by tin salts is very rare indeed. Accidental cases occur from the use of tinned fruits owing to the mallic acid of fruits acting on tin and forming mallate of tin.

Poisonous symptoms may arise from wearing silk articles of clothing, such as silk stockings, which are, sometimes, impregnated with tin chloride. A. Jolles[1] reports the case of a young woman who developed poisonous symptoms from wearing yellow silk stockings heavily impregnated with tin chloride. She complained of motor and sensory disturbances in the lower extremities which were stained yellow. The urine was albuminous, and marked nervous symptoms like ataxia were noted a few weeks later. She became anæmic, but recovered in a few months after the stockings had been discarded.

A fatal case[2] of poisoning has occurred from the accidental use of "putty powder", a higher oxide of tin, which is used for polishing silver vessels.

2. Tin is eliminated in the urine and fæces.

CHROMIUM

The following preparations of chromium are important from a toxicological point of view:—

Chromic Acid (Chromic Anhydride, Chromium Trioxide), CrO_3.—This occurs as crimson, needle-shaped crystals. It is deliquescent and readily soluble in water and alcohol. It is a powerful oxidising agent, and is used in preparing *Liquor acidi chromici*. It is prepared by the action of strong sulphuric acid on a cold saturated solution of potassium bichromate and, therefore, exists in "battery fluids" used in bichromate cells. It is a powerful corrosive, and is used as a caustic in medicine.

Potassium Chromate, K_2CrO_4.—This is a yellow, crystalline salt with a disagreeable bitter taste, and readily soluble in water, the solution being alkaline in reaction. It is chiefly used in manufacturing chrome yellow (lead chromate), a very poisonous salt.

Potassium Dichromate (Red Chromate), $K_2Cr_2O_7$.—This is also known as potassium bichromate. It is an orange-red, crystalline salt, having a bitter and metallic taste. It is soluble in ten parts of water, forming an acid solution, which is highly poisonous, having a special action on the nervous system. It is insoluble in alcohol. It is used by the dyers, furniture stainers and photographers.

Acute Poisoning.—The toxic effects appear within a few minutes, say 5 minutes or less, after swallowing the poison, usually potassium dichromate or chromic acid. The symptoms are a bitter metallic taste, intense pain in the stomach, vomiting and diarrhœa. The vomited matter is yellow, and sometimes tinged with bile and blood. The stools are yellow owing to the reduction of the salt, and may contain blood. The pupils are dilated, and do not react to light. The respirations are very slow and gasping. The pulse is feeble and almost imperceptible. These are followed by muscular cramps, collapse, unconsciousness and death. Convulsions may occur in some cases.

1. *Munch. Med. Wchnschr.*, 1902, 48, *p.* 327; *Wein. Med. Presse*, 1901, 42, *p.* 496.
2. *Med. Press and Circ.*, 1894, *N. S.*, 57, *p.* 450.

Fatal Dose.—Two drachms of potassium bichromate have proved fatal to an adult woman,[1] but the smallest dose is 30 to 45 grains (2 to 3 grammes) which killed a man, aged 43 years, in ten days,[2] while 10 grains have killed a child of twenty months.[3] Recovery has followed a dose of 273 grains taken in solution in mistake for tea,[4] and a dose of half-an-ounce in the case of two individuals who attempted to commit suicide.[5] In the third case a man recovered at the end of a week after he had swallowed, with suicidal intent, about two ounces of the bichromate in solution, mixed with pearl ash.[6] Death has occurred in 12 hours after swallowing a table-spoonful of chromate of potassium in place of Glauber's salt,[7] as also from a table-spoonful of a 50 per cent solution of chromic acid.[8] Six grammes of chromic acid have also proved fatal.[9]

Fatal Period.—The shortest period is 40 minutes from a dose of one ounce of bichromate of potassium.[10] A woman died in four hours after she had taken two drachms of this salt.[11] The average is $8\frac{1}{4}$ hours. A woman,[12] 21 years old, died on the tenth day after swallowing 15 grammes of potassium bichromate with intent to commit suicide.

Treatment.—Empty the stomach by emetics, or wash it out with warm water. The stomach may then be washed out with a weak solution of silver nitrate. Give solutions of magnesium or calcium carbonate in water or in milk, and administer demulcents as well as stimulants.

Post-mortem Appearances.—The mucous membrane of the stomach is inflamed and corroded in patches, and coloured olive-green or purple due to the conversion of the salt into oxide. The duodenum also shows the same appearances. The blood is chocolate coloured and shows the spectrum of methæmoglobin. Fatty degeneration of the liver and heart, and acute inflammation of the kidneys. In a case of death from potassium bichromate reported by Dr. Willcox there were no changes in the viscera except slight brown discoloration of the stomach wall. The chemical analysis revealed the presence of chromium in the viscera.[13]

Chronic Poisoning.—This is apt to occur among those who are employed in the manufacture of chromic acid and its salts, and are thus constantly handling them, or are exposed to their dust.

Symptoms.—Irritation and inflammation of the mucous membrane of the nose causing sneezing, salivation and conjunctivitis. The nasal membrane then becomes ulcerated, and perforation occurs in the lower part of the septum.

Deep ulcerated sores, known as *chrome holes* occur on the hands, face and other parts of the body, resembling hard chancres in appearance. At the same time eczematous and psoriatic rashes may appear on the skin, and the periosteum may be inflamed and painful.

Treatment.—The use of white petrolatum to the nasal septum causes all unpleasant symptoms to disappear.[14]

---

1. *Taylor, On Poisons, Ed. III, p.* 489.
2. *Klimesh, Wien. Kl. Wchnschr.*, 1889, *II, p.* 732; *Witthaus, Med. Juris. and Toxic., Vol. IV, p.* 703.
3. *Mc. Crorie, Glas. Med. Jour.*, 1881, *XV, p.* 378.
4. *Philipson, Lancet, Jan.* 16, 1892, *p.* 138.
5. *Warwick, Lancet,* 1880, *Vol. I, p.* 167; *Waugh, Ibid.*, 1885, *Vol. II, p.* 1135.
6. *Taylor, On Poisons, Ed. III, p.* 489.
7. *Neese, Pharm. Ztschr. f. Russl.*, 1862, *No.* 7; *Witthaus, Loc. Cit., p.* 702.
8. *Burghart, Charite Ann.*, 1896-7, *XXIII*, 196; *Ibid., p.* 313.
9. *Gadamer, Lehrbuch der Chemischen Toxikologie*, 1924, *p.* 244; *Webster, Leg. Med. and Toxicol.*, 1930, *p.* 425.
10. *Stewart, Brit. Med. Jour.*, 1898, *Vol. II, p.* 420.
11. *Taylor, On Poisons, Ed. III, p.* 489.
12. *Leschke, Clin. Toxic., Eng. Trans. by Stewart and Dorrer*, 1934, *p.* 85.
13. *Trans. Med.-Leg. Society*, 1909-10, *p.* 69.
14. *Jour. Amer. Med. Assoc., Sep.* 14, 1929, *p.* 837.

Chemical Tests.—An alkaline solution of a chromium salt yields a green precipitate, soluble in excess on the addition of ammonium sulphide. With a solution of nitrate or acetate of lead, chromates or bichromates give a bright yellow precipitate, soluble in boiling water. This solution, on cooling, deposits golden yellow spangles of lead chromate. A solution of chromic acid gives a yellow precipitate with barium nitrate or chloride, soluble in hydrochloric and nitric acids. With silver nitrate it gives a brick-red precipitate, soluble in ammonia. When boiled with dilute sulphuric acid and alcohol it acquires a green colouration.

Medico-Legal Points.—Poisoning by chromates is extremely rare, though they are very poisonous. Chromic acid has produced fatal symptoms from an external application as well as from accidentally swallowing it while applying it to the throat with a throat brush. A case[1] is reported in which a young woman died in 27 hours after an external application of about 50 grains of chromic acid in half-an-ounce of water to a mass of papillary growth on the external genitals.

Accidental and suicidal cases, though rare, have occurred from swallowing a bichromate or a chromate solution. A man,[2] aged 75 years, swallowed inadvertently and on a fasting stomach a quantity of a 5 per cent solution of potassium bichromate containing between 1 and $1\frac{1}{2}$ grammes of the salt. After a short time vomiting and diarrhœa set in. The fæces were green in colour and did not contain blood. Between six and seven hours later, the patient developed painful convulsions of the arms and legs. Dyspnœa and retention of urine were present. Two days later, the patient was catheterized and 125 c.c. of red brown urine were obtained. The urine contained sugar, albumin, erythrocytes and leucocytes. Camphor and olive oil were administered with intestinal lavage, and venesection was performed twice. The patient was discharged cured after about six weeks, but with cardiac arrhythmia and low blood pressure. A young woman[3] in Dera Ghazi Khan was suffering from a bad cough, and took some medicine from a travelling *hakim*. She developed gastro-intestinal symptoms immediately after swallowing the medicine, and died in eleven hours. Potassium bichromate was detected in the viscera submitted to the Chemical Examiner. A man who was heavily involved in debts committed suicide by swallowing potassium bichromate. The post-mortem examination revealed marked corrosion of the lower half of the œsophagus. The mucous membrane of the stomach and intestine was corroded and dark in colour.[4]

Potassium bichromate has been given in a few instances for homicidal purposes, as also for procuring abortion. Dr. Michael Verzar[5] describes a case of attempted murder in which an electrician, 46 years old, was given altogether 120 grains of potassium bichromate in red wine and also in a mixture of soda and lemonade. The man recovered after a fortnight.

The salts of chromium are eliminated mainly by the kidneys, and to some extent by the liver and bowels.

A case[6] is recorded in which several persons were poisoned through the application of an ointment in which potassium chromate was used by mistake instead of the sulphur ordered for the treatment of scabies. Of these twelve died.

POTASSIUM

The following salts of potassium have caused poisonous symptoms:—

Potassium Nitrate (Saltpetre, Nitre, Sal Prunelle), KNO_3.—In the vernacular, the salt is called *Sorakhar* or *Kalmi Sora*. It exists as colourless, rhombic crystals. It has a cool, saline taste, and is soluble in water. Its solubility increases with the rise of temperature. It is chiefly used in the manufacture of gunpowder and in pyrotechny. The dose is 5 to 15 grains.

1. *White, University Med. Magazine*, 1889, 2, *p.* 54.
2. *Hygiea, Stockholm, Jan.* 31, 1927, *p.* 84; *Jour. Amer. Med. Assoc., May* 27, 1927, *p.* 1530.
3. *Punjab Chemical Examiner's Annual Report*, 1925, *p.* 3.
4. *Madras Chemical Examiner's Annual Report*, 1937, *p.* 3.
5. *Jour. Amer. Med. Assoc., July* 23, 1927, *p.* 307.
6. *Ibid., Nov.* 21, 1919, *p.* 1590.

Acute Poisoning.—Accidental cases of poisoning, though rare, have occurred from its use in mistake for magnesium sulphate (Epsom salts) or sodium sulphate (Glauber's salt). In the annual report of the Chemical Examiner of the United Provinces for 1921 a case is reported in which potassium nitrate was given as a purge by mistake for magnesium sulphate, with fatal results. It was once used to commit suicide and once to cause abortion.[1] Used as an enema containing 124 grains it has caused death.[2]

Symptoms.—Nausea, pain in the stomach and epigastrium, vomiting and purging. The vomited matters and stools may contain blood. The urine may also contain blood. Dyspnœa, weak irregular pulse, collapse, convulsions and death. Coma may precede death. Recovery from large doses is slow, and gastric disturbances, parasthesia, cramps and muscular twitchings or paralysis may persist for two or three months.

Fatal Dose.—The smallest is 2 drachms.[3] The usual fatal dose is an ounce, though recovery has occurred even after 4 ounces taken in mistake for magnesium sulphate.[4]

Fatal Period.—The shortest recorded fatal period is 45 minutes and the longest is 60 hours, the average being 13 hours.[5]

Treatment.—Wash out the stomach with the stomach tube. Give stimulants by hypodermic injection. Apply mustard plaster on the epigastrium and warmth to the body. Administer mucilaginous drinks and treat the prominent symptoms.

Post-mortem Appearances.—The mucous membrane of the stomach is stained bright red or brownish-red, inflamed and detached in various parts. A small perforation at the fundus of the stomach has been observed in one case.[6] The small intestine is acutely inflamed. The blood is liquid.

Elimination.—Potassium nitrate is eliminated largely by the kidneys and to a slight extent by the intestines. A small portion may be reduced to nitrite.

Cases.—1. **Acute Poisoning.**—One night a woman[7] took a portion of a powder containing saltpetre sold to her child by a grocer by mistake for magnesium sulphate, and almost fainted afterwards. Next forenoon she took the remainder, the greater portion of the powder, and shortly afterwards was seized with pain in the stomach and back, and vomited more than two pints of bright red coloured fluid. Shortly after 2 p.m. she was in a state of collapse, the pulse being feeble, irregular and about 56 per minute and the skin cold. Up to 6 p.m. she had vomited several times, and the washhand basin was half filled with dark coloured fluid like coffee grounds, but in larger clots. The tongue was dry and the pupils dilated. She had not passed urine till then. She slowly began to recover, and was able to leave bed in about a week.

2. **Chronic Poisoning.**—A strong and healthy farmer,[8] aged 57, who was suffering from chronic sacro-iliac arthritis, took equal parts of potassium nitrate and sulphur in spoonful doses four times a day and continued this treatment for twenty-six days. He came under observation on the 20th February, 1921, when he looked very ill; his eyes were sunken, he had lost much weight, was very nervous and complained of intense pain, aggravated by touch or motion. The pulse varied from 85 to 95 per minute and the temperature was normal or subnormal at all times. The stools were loose. The deep reflexes were normal and there was no paralysis. The quantity of urine was diminished, 21 ounces being the total quantity voided in twenty-four

1. *Orfila, Toxic., Ed. V, Vol. I, pp.* 354, 356; *Witthaus, Med. Juris. and Toxic., Vol. IV, p.* 305.
2. *Ibid.*
3. *Dixonmann, Forens. Med. and Toxic., Ed. VI, p.* 366.
4. *Bailey, Phila. Med. and Surg. Reporter,* 1872, *Vol.* 26, *p.* 76.
5. *Witthaus, Loc. Cit., p.* 306.
6. *Souville : Orfila, Toxic., Vol. I, p.* 354.
7. *William A. Caskie, Brit. Med. Jour., May* 6, 1911, *p.* 1052.
8. *Emil Windmeuller, Jour. Amer. Med. Assoc., Sept.* 10, 1901, *p.* 858.

hours. The urine contained albumin, a few hyaline and waxy casts and red blood cells. The muscular pains gradually subsided, but restlessness persisted. He was sleepless for days at a time, and the loss of flesh was very rapid. He died on the 7th March—17 days after he came under observation.

Potassium Chlorate, $KClO_3$.—This is a colourless, crystalline salt with a cool saline taste, soluble in 16 parts of cold water and in 3 parts of boiling water, but almost insoluble in alcohol. It is largely used in the manufacture of matches and in pyrotechny, and in calico-printing and dyeing. It must be handled carefully, as it explodes when rubbed with many substances, especially sulphur, sulphides, sugar, charcoal, tannic acid and glycerine. Potassium chlorate is a pharmacopœial preparation, the dose being 5 to 10 grains.

Accidental cases of poisoning occur chiefly from an overdose of potassium chlorate or from it having been swallowed in mistake when prescribed as a gargle. Suicidal cases are rare. A homicidal case is recorded where potassium chlorate was given to a child by his mother and step-mother. The child died in about 3 hours.[1]

Symptoms.—When swallowed in large doses, it causes pain in the stomach and abdomen, severe vomiting and diarrhœa. When absorbed, it breaks up the red blood corpuscles, converting the hæmoglobin into methæmoglobin and setting up secondary symptoms, such as pain in the loin, hæmoglobinuria, suppression of urine, bloody tube-casts, cyanosis of the skin, jaundice, drowsiness, delirium, coma and death.

Fatal Dose.—According to Witthaus[2] the smallest fatal dose is 3 drachms for an adult, 75 grains for a child and 15 grains for an infant. A man, aged 41, took 80 lozenges (each containing 5 grains) in two doses morning and evening for sore throat and influenza and died from the symptoms of caustic poisoning.[3] Fountain, experimenting upon himself, took about 9 drachms and died in seven days from nephritis.[4] It should, however, be remembered that a quantity taken in divided doses is more apt to cause death than when taken in a single dose.

Fatal Period.—The shortest recorded fatal period is $2\frac{1}{2}$ hours in the case of a child, three weeks old, and the longest period for an adult is 12 days.[5]

Treatment.—Administer emetics, or wash out the stomach, and give alkaline drinks to increase the flow of urine and pilocarpine hypodermically to stimulate its excretion by the saliva. Use oxygen inhalation, stimulants, transfusion of defibrinated blood, or normal saline solution.

Post-mortem Appearances.—Submucous hæmorrhages in the mucous membrane of the stomach and duodenum, which is swollen, reddened and easily detached. The liver and spleen are enlarged and dark brown in colour. Th kidneys are enlarged and inflamed. The lungs are marked with subpleural ecchymoses. The heart is dilated. The brain and its membranes are congested. The blood is chocolate coloured with degenerated red blood corpuscles.

Elimination.—After administration by the mouth potassium chlorate appears in the saliva in five minutes, but it is chiefly eliminated in the urine.

Chemical Tests.—Acidify the suspected solution with dilute sulphuric acid and add a few drops of indigo solution until the colour is blue. The addition of sulphurous acid will discharge the blue colour, if potassium chlorate be present.

Potassium Sulphate (Sal Polychreest, Sal de Duobus), K_2SO_4.—This forms colourless, rhombic crystals, having a bitter, salty taste. It is soluble in ten parts of water. It is extensively used for agricultural purposes. Accidental cases of poisoning occur from its use. It has also been employed in France for procuring abortion.

---

1. *Wagner, Samml. von Vergiftsfalle*, 1934, 47, B. 48 ; *Med.-Leg. and Criminological Rev., April*, 1935, *p.* 135.
2. *Manual of Toxic., Ed. II, p.* 691.
3. *Brit. Med. Jour., Jan.* 12, 1907, *p.* 116.
4. *Amer. Med. Times*, 1860.
5. *Witthaus, Manual of Toxic., Ed. II, pp.* 692, 693.

Symptoms.—Pain in the abdomen, vomiting, diarrhœa, exhaustion and collapse ending in death.

Fatal Dose.—The pharmacopœial dose is 15 to 45 grains. The smallest fatal dose is 2 drachms.[1] The usual fatal dose is 2 ounces.

Fatal Period.—$1\frac{1}{2}$ hours when 2 drachms were taken and 2 hours in another case when 10 drachms were given in divided doses to a woman within a week of her confinement.[2]

Treatment.—Empty the stomach by emetics or wash it out by the stomach tube, and treat the symptoms of irritation and depression as they arise.

Post-mortem Appearances.—The mucous membrane of the stomach is congested and inflamed.

Potassium Sulphide (Liver of Sulphur), K_2S.—This occurs in dull green, solid masses, and is used as potassa sulphurata in the ointment of skin diseases.

Symptoms.—It acts as an irritant poison, but at the same time exhibits narcotic symptoms owing to its rapid decomposition into sulphuretted hydrogen. Death may occur in 15 minutes.[3]

Treatment.—Give dilute solutions of chloride of soda or lime and then treat the symptoms.

Post-mortem Appearances.—The body surface is livid. Redness of the stomach and duodenum with deposit of sulphur. The lungs are gorged with dark blood.

Chemical Tests.—1. A solution of silver nitrate gives a black precipitate with sulphides.

2. If the solution be heated after adding an acid, hydrogen sulphide will be evolved, known from its turning white paper black, when moistened with lead acetate solution.

ALUMINIUM

Alum (*Phitkari*).—This is a double salt of sulphate of aluminium and potassium (potash alum), $Al_2(SO_4)_3\ K_2SO_4,\ 24\ H_2O$, or sulphate of aluminium and ammonium (ammonia alum), $Al_2(SO_4)_3\ (NH_4)_2(SO_4)_3,\ 24\ H_2O$. It occurs as transparent, colourless and octahedral crystals, having a sweetish astringent taste. It is soluble in water and glycerine but insoluble in alcohol. It is largely used as a mordant for dyeing, as a constituent of certain baking powders to whiten bread, and for purifying water before filtering it.

Alum is also a pharmacopœial preparation, and is known as *alumen*, the dose being 5 to 10 grains. It enters into the composition of *Glycerinum aluminis*, the dose being 30 to 60 minims.

Symptoms.—Burning pain in the mouth, throat and stomach; vomiting mixed with blood; dyspnœa; frequent pulse; subnormal temperature; loss of co-ordination; convulsions of a clonic nature; death. In a solid form it acts as a corrosive in the mouth and throat.

Fatal Dose.—Half-an-ounce to an ounce of alum. One drachm given in syrup killed a child, aged 3 years, who was suffering from diphtheria.[4] Recovery has occurred after much larger doses.

Fatal Period.—Twenty-four hours.

Treatment.—Emetics; lime water; sodium carbonate in large quantities of milk.

1. *Med. Times and Gaz.*, Dec. 30, 1843.
2. *Ann. d' Hyg.*, 1842; *Guy and Ferrier, Forens. Med., Ed. VI, p.* 377.
3. *Ibid., p.* 378.
4. *Taylor, Princ. and Pract. of Med. Juris., Vol. II, Ed. IX, p.* 419.

Post-mortem Appearances.—The tongue, mouth and œsophagus are œdematous and corroded. The mucous membrane of the stomach is corrugated, loosened or hardened, and stained red or velvety. The intestines are inflamed.

Chemical Tests.—1. An alkaline solution with ammonia and ammonium sulphide gives a gelatinous white precipitate, soluble in caustic potash.

2. Ammonia gives a white gelatinous precipitate, insoluble in excess of the reagent (Distinction from zinc).

3. Caustic potash gives a white precipitate, soluble in excess, which reappears on adding ammonium chloride, but not on adding hydrogen sulphide.

4. Ammonium carbonate gives a white flocculent precipitate.

5. A blue incrustation is formed on charcoal when heated with a solution of cobalt nitrate.

Medico-Legal Points.—Aluminium is present in many vegetables, in many fruits, in milk, in eggs, and in sea food and probably in the tissues of the human and animal bodies. It is also suggested that some may be absorbed from the metal dissolved out of the aluminium vessels by the acids and alkalies used for cooking purposes, but Finn and Inouye[1] state that "there is no scientific evidence of any chronic poisoning taking place from food cooked in aluminium utensils."

It is possible that slow poisoning may occur among aluminium workers. A case[2] is recorded in which a man working with the metal suffered from loss of memory, tremors, jerky movements, impaired co-ordination, chronic constipation and incontinence of urine.

MAGNESIUM

Magnesium Sulphate (Epsom Salts), $MgSO_4$, $7H_2O$.—This forms colourless, rhombic prisms, and dissolves readily in water. Its solution has a cool, bitter taste, and acts as a purgative. The pharmacopœial dose is 30 to 240 grains. When taken in excess, it acts as an irritant poison. It is contained in the official preparations of *Mistura magnesii hydroxidi* (Cream of magnesia), dose, 60 to 240 minims and *Mistura sennæ composita* (Black draught), dose, 1 to 2 fluid ounces.

Magnesium sulphate closely resembles oxalic acid and zinc sulphate, hence the latter salts have been frequently mistaken for magnesium sulphate.

Symptoms.—These commence in less than half-an-hour after swallowing a poisonous dose. Burning pain in the stomach and intestines, nausea; vomiting; purging; dilated pupils; paralysis of the lower limbs; tetanic spasms; suppression of urine; collapse and death from respiratory failure.

Sometimes, after swallowing a large dose, the patient becomes pale, feels giddy, falls down and dies from syncope. A Christian boy,[3] 7 years old, was given in the early morning 2 ounces of magnesium sulphate as an aperient and had vomited it up. He had again been given another dose of 2 ounces about 2 hours later and had again vomited, but one hour later he became unconscious and was removed to J. J. Hospital, Bombay, where he was found unconscious and cyanosed with dilated pupils reacting sluggishly, shallow respirations, and a feeble pulse. He died in less than an hour.

Fatal Dose.—One ounce has caused death,[4] though the same quantity may be given as a purgative. Two ounces have caused the death of a boy, ten years old.[5]

Fatal Period.—Forty minutes after a fatal dose of 2 ounces as mentioned above. Eighty minutes after swallowing 4 ounces.[6] Death occurred in 60 hours in a case

1. *Jour. Amer. Med. Assoc.*, 1928, *Vol.* 90, *p.* 1010.
2. *J. Spofforth, Lancet*, 1921, *Vol. I, p.* 1301.
3. *Bombay Chem. Analyser's Annual Report*, 1930, *p.* 4.
4. *Luff, Fornes. Med., Vol. I, p.* 151.
5. *Christison quoted in Dixonmann's Forens. Med. and Toxic., Ed. VI, p.* 370.
6. *Sang, Lancet*, 1891, *Vol. II, p.* 1037.

where 310 c.c. of a 4 per cent solution of magnesium sulphate had been injected subcutaneously.[1]

Treatment.—Empty the stomach; give stimulants and treat the symptoms. Subcutaneous or intravenous administration of calcium salts has been recommended, as the calcium salts have an antagonistic action on the inhibitory effect of magnesium sulphate.

Post-mortem Appearances.—Signs of irritation of the gastro-intestinal tract may be present. In a case[2] in which a young farmer died in one hour and ten minutes after taking a solution of magnesium sulphate, the post-mortem examination showed that the stomach contained approximately a litre of yellowish-brown liquid, and its lining membrane had a dark red hæmorrhagic appearance. There were recent hæmorrhages throughout the small intestine and considerable blood was mixed with the contents. The hæmorrhages averaged 5 mm. in their largest dimensions. There were a few hæmorrhages in the region of the cæcum. There was marked congestion of the lungs, trachea and main bronchi, and of the heart, liver and kidneys. There was also acute splenic hyperplasia. The chemist reported 883½ grains of magnesium sulphate in the contents of the stomach.

Chemical Tests.—1. Caustic potash gives a white precipitate.

2. Carbonate (but not bicarbonate) of sodium throws down a white precipitate of basic carbonate of magnesia.

3. Rosy pink incrustation on charcoal, if heated with cobalt nitrate.

BARIUM

Barium Chloride, $BaCl_2$.—This forms colourless, rhombic crystals, having an acrid taste, and soluble in water. It is chiefly used as a chemical reagent. It is highly poisonous, and has been taken in mistake for Carlsbad salt or Glauber's salt. It is a non-official preparation, the dose being ½ to 1½ grains.

Barium Nitrate, $Ba(NO_3)_2$.—This crystallizes in large, colourless octahedra. It is soluble in water. It is used in pyrotechny to make green fire.

Barium Carbonate, $BaCO_3$.—This occurs as a mineral witherite. It is a fine white powder, slightly soluble in water, but soluble with effervescence in dilute acids, and is readily decomposed by the free acids of the stomach. It is largely used as a poison for rats and mice.

Barium Sulphate, $BaSO_4$.—This occurs native as heavy spar. It is a heavy white, tasteless, odourless powder. It is insoluble in water, and only very slightly soluble in dilute acids. It is largely used as a white pigment, known as permanent white. It is not poisonous and has recently come into very large use for X-ray examination of the œsophagus, stomach and intestines.

Barium Sulphide (*Baryta Sulphurata, B.P.C.*), BaS.—This occurs as a greyish-black powder, and dissolves readily in water giving off an offensive odour of hydrogen sulphide. It is a deadly poison and is chiefly used as a depilatory.

Symptoms.—The symptoms appear within half-an-hour after swallowing the poison. These are severe pain, nausea, salivation, vomiting, intense thirst, persistent purging, dilatation of the pupils, dimness of vision, singing in the ears, violent cramps in the legs, convulsions, paralysis, collapse or coma and death.

Fatal Dose.—Two and a quarter grains of barium chloride given to a woman in 1/12-grain doses produced severe symptoms of poisoning.[3] Two and a quarter grammes of barium chloride taken on two consecutive nights in mistake for chloral hydrate killed a 55-year-old man suffering from asthma on the third day.[4] A mouthful

1. *Curtis, Jour. Amer. Med. Assoc.*, 1921, *Vol.* 77, *p.* 1492.
2. *H. S. Thatcher, Jour. Amer. Med. Assoc., Oct.* 20, 1928, *p.* 1185.
3. *Ferguson, Med. Times, March* 28, 1845, *p.* 508.
4. *Fuhner, Sammlung von Vergiftungsfallen*, 1930, *Vol. I; Leschke, Clin. Toxic., Engl. Trans. by Stewart and Dorrer*, 1934, *p.* 88.

of a solution containing 130 grains of the chloride proved fatal to a man.[1] Less than a teaspoonful (100 grains) of the chloride caused the death of a woman, aged 23 years.[2] On the other hand, recovery has followed a dose of 370 grains of the chloride.[3] Four drachms of barium nitrate have killed a man, aged 46 years.[4] Sixty grains of barium carbonate have destroyed life in two cases.[5] A young woman recovered after having swallowed half a tea-cupful of the powdered carbonate mixed with water on an empty stomach.[6] Thirty-eight grammes of barium sulphide have caused death.[7]

Fatal Period.—Uncertain. The shortest period is 10 minutes in a case where 40 grammes of barium sulphide had been taken by mistake for barium sulphate.[8] One hour[9] in another case. A man who was given a powder containing barium carbonate and barium sulphide in soup by his wife died on the third day.[10] The longest period is 7 days.[11]

Treatment.—Give one-ounce doses of sodium or magnesium sulphate as an antidote, and then give emetics or wash out the stomach with milk and water. Use morphine to relieve pain, and stimulants to combat collapse.

Post-mortem Appearances.—Reddening, congestion and inflammation of the mucous membrane of the stomach and duodenum ; sometimes erosions of the mucous membrane. In the case of a woman who died in about 2 hours after taking half-an-ounce of powdered barium chloride the stomach was found perforated posteriorly in the lesser curvature near the cardiac end, but the perforation was due to disease and not to the poison taken.[12] The heart is large and flabby. The lungs and brain are congested.

Chemical Tests.—1. Dilute sulphuric acid gives a white precipitate, insoluble in hydrochloric or nitric acid.

2. Potassium bichromate yields a yellow precipitate.

3. *Flame Test.*—The flame assumes a green colouration when a barium salt is held in it by a loop of platinum wire moistened with hydrochloric acid.

Medico-Legal Points.—The soluble salts of barium are highly poisonous. They have locally an irritant action and remotely have a depressant action on the heart.

Most of the cases of poisoning by barium salts are accidental, taken in mistake for Epsom or other salts. A few are suicidal.

A family in Hissar District ate *chapatis* made with *atta* (wheat flour) mixed with pills of barium carbonate used for destroying rats. Soon afterwards all of them began to vomit and purge, exhibiting the symptoms of an irritant poison[13]

An accidental fatal case[14] of poisoning by barium sulphide occurred under tragic circumstances in the Sassoon Hospital at Poona on April 3, 1923. His Highness the Rajasahib of Akalkota had some stomach trouble and went to the hospital by appointment to consult the X-ray specialist. It was arranged to X-ray the stomach and in

1. *Sterne, Zeitchrift. f. Med. Beamte*, 1896, *IX*, p. 381.
2. *Walsh, Lancet, Feb.* 26, 1859, p. 211.
3. *Wolf, Wchnschr. f. d. ges. Heilk*, 1850, No. 37, *R ; Witthaus, Med. Juris. and Toxic., Vol. IV*, p. 683.
4. *Pharm. Jour., June*, 1872, p. 1021.
5. *Taylor, On Poisons, Ed. III*, p. 274.
6. *Wilson, Med. Gaz., Vol.* 14, p. 448.
7. *Webster, Leg. Med. and Toxic.*, 1930, p. 416.
8. *Bensaude and Antoine, Bull. Soc. med. des. Hop., Paris*, 1919, *XLIII*, p. 15 ; *Peterson, Haines and Webster, Leg. Med. and Toxic., Ed. II, Vol. II*, p. 285.
9. *Ann. d'Hyg.*, 1841, *Pt.* 2, p. 217.
10. *Madras Chem. Examiner's Annual Rep.*, 1933, p. 4.
11. *Dixonmann, Forens. Med. and Toxic., Ed. VI*, p. 369.
12. *Wildbery quoted by Taylor, On Poisons, Ed. III*, p. 273.
13. *Punjab Chemical Examiner's Annual Report*, 1928, p. 9.
14. *The Med.-Leg. Jour., July and August*, 1923, *Vol. XI*, p. 106.

order to note the process of food digestion a meal of barium sulphate mixed with porridge had to be given. There being no barium sulphate in the hospital, an order for the drug was sent to the Poona Drug Stores which unfortunately supplied barium sulphide instead of barium sulphate. About two table-spoonfuls of this were mixed up with a bowlful of porridge and about two or three mouthfuls of the mixture of barium sulphide were taken which probably contained about a tea-spoonful of the salt. The Raja could not take any more on account of the offensive odour of the mixture and started vomiting immediately afterwards. He then complained of a burning pain at the pit of the stomach. In spite of prompt treatment the patient collapsed and died within two hours after having taken the drug. There was no post-mortem examination. The chemist who dispensed the drug and the X-ray specialist who administered it were both convicted by the Assistant Collector for causing death by a rash and negligent act.

A Hindu male,[1] aged 55, took about $1\frac{1}{2}$ drachms of a depilatory powder containing 1 part barium sulphide and 8 parts washing earth or *botni mitti* in mistake for a laxative powder at 4 a.m. on April 14, 1929. Vomiting commenced soon afterwards and was persistent ; he had 3 motions in the course of the next 4 hours. At 10 a.m. he noticed difficulty in lifting the arms and extending the legs and could not close the fist tightly. At 5 p.m. his tongue was found coated and dry, and the pulse slow, full and intermittent. The heart sounds were booming, the second aortic sound being markedly accentuated and intermittent with a beat missing after every five or six beats. There was paresis of the arms and legs, and the grip was very weak. The deep reflexes were absent. There was no sensory disturbance. The brain was absolutely clear. He was given one drachm of magnesium sulphate in solution every two hours, and he recovered after he had taken altogether six doses. He had 5 thin watery motions in the night.

A Mahomedan woman, aged 25 years, took a quantity of a depilatory powder containing barium sulphide with intent to commit suicide at 7 p.m. on the 18th July, 1931. Soon afterwards she had vomiting which contained blood and emitted the odour of hydrogen sulphide. She could not swallow anything, as there was excoriation of the throat. She was at once removed in a collapsed condition to the King George's Hospital, Lucknow, where she died at 10-40 p.m.

Barium is eliminated chiefly in the fæces, though slightly in the urine.

SODIUM

Sodium Chloride (Common Salt or Table Salt), NaCl.—This is called *Namak* in the vernacular. It occurs in colourless, cubical crystals or small white, crystalline powder, and is largely used in the alkali industry. It is a necessary article of food for man and other animals. It is soluble in 3 parts of cold water. It occurs in official preparations of *Liquor sodii chloridi physiologicus* (Physiological or normal saline solution) and *Injectio sodii chloridi et acaciæ*.

Symptoms.—In large doses it causes irritant symptoms, followed by paralysis.

Fatal Dose.—Half a pound.[2]

Treatment.—Emetics or stomach tube.

Post-mortem Appearances.—Not characteristic.

CADMIUM

Cadmium Chloride, $CdCl_2$, $2H_2O$.—This is an efflorescent salt, having white, silky crystals. When heated, it gives up its water of crystallization, and becomes anhydrous.

Cadmium sulphide is used as a pigment in oil and water colour.

1. *Bhupal Sing, Ind. Med. Gazette, Sep.*, 1929, *p.* 506.
2. *Med. Times, Jan.* 4, 1840.

Symptoms.—Pain in the stomach, nausea, vomiting, diarrhœa, giddiness, collapse, loss of consciousness and death.

Prcdan[1] reports a series of experiments on cats exposed to cadmium oxide fumes and the dusts of cadmium oxide and sulphide. It was found that the inhalation of cadmium oxide fumes and dust produced an increase in the rate of respiration and abundant salivation which appeared during the exposure or immediately afterwards. Later, the respiration became more dyspnœic and noisy. The animals refused to eat and drink. In high concentrations cadmium oxide gave rise to pulmonary œdema and death; in smaller amounts there ensued generalised pneumonia and broncho-pneumonia, emphysema and atelectasis. There was fatty cellular infiltration in the liver and kidneys. Cadmium is found mainly in the lungs, liver and kidneys.

The sulphide dust produces vomiting, diarrhœa, occasionally salivation and an increased rate of respiration which is dyspnœic and noisy. The excretion is effected very slowly through the kidneys and gastro-intestinal tract.

Fatal Dose.—From his experiments on animals Blyth[2] considers 4 grammes to be a dangerous dose of a soluble salt of cadmium for adults. A Mahodeman lad, about 14 years of age, died after swallowing 2 drachms and 10 grains of cadmium chloride in a decoction of senna leaves.[3]

Fatal Period.—One and-a-half hours in the above recorded case.

Treatment.—Evacuate the stomach contents by emetics, or wash out the stomach with the stomach tube. Treat the symptoms.

Post-mortem Appearances.—Not characteristic. Congestion of the stomach, lungs and brain may be found.

In a case[4] where an English engineer died four days after inhaling the vapour of molten cadmium for three hours, the post-mortem appearances were inflammation of the pharynx, trachea, bronchi, and gastro-intestinal canal, with fatty degeneration of the heart and liver.

Chemical Tests.—1. Hydrogen sulphide gives a yellow precipitate in caustic potash.

2. Cadmium salt forms a brownish incrustation if heated in the reducing flame of a blow-pipe on a piece of charcoal.

Medico-Legal Points.—Poisoning by cadmium is extremely rare, but may occur as an industrial disease. In India an accidental case of poisoning has occurred from the use of cadmium chloride, given by a quack in mistake for magnesium sulphate, as mentioned above.

Cadmium resembles zinc very closely in its effects, but is more toxic.

GOLD (*SONA*)

Gold Chloride (Auric Chloride), $AuCl_3$.—This occurs as soluble, deliquescent, brown crystals, and is used in photography.

Gold and Sodium Chloride.—This is a non-official preparation, consisting of equal parts of anhydrous gold chloride and anhydrous sodium chloride. It is an orange-yellow, odourless powder, soluble in water and having a saline metallic taste. The dose is 1/30 to 1/12 grain, increased to ½ grain in a pill with kaolin ointment.

Sodium Aurothiosulphate (Sanocrysin).—This is a double thiosulphate of gold and sodium containing 37.4 per cent of gold. It is a non-official preparation, and has been recommended in phthisis in doses of 0.1 to 1 gramme in 10 c.c. of water to be given intravenously.

1. *Brit. Med. Jour., Aug.* 27, 1932, *Ep., p.* 42.
2. *Poisons, Ed. V, p.* 630.
3. *Hinder, Ind. Med. Gaz., June,* 1866, *p.* 156.
4. *Erich Leschke, Clin. Toxic., Engl. Trans. by Stewart and Dorrer,* 1934, *p.* 89.

Other proprietary preparations of gold salts are crisalbine; krysolgan, lopion, myochysin, solganal, etc.

Symptoms.—These are due to its local corrosive action. The lips, tongue, teeth, and the inside of the cheeks are purple coloured, followed by tenderness of the epigastrium, salivation, persistent vomiting, diarrhœa, fever, albuminuria, and collapse.

When given in an initial large dose or too frequently, sanocrysin may produce poisoning, the symptoms being skin rashes, unpleasant taste, stomatitis, nausea, vomiting, abdominal pain, diarrhœa, albuminuria, hæmoglobinuria, jaundice, fatty degeneration of the liver, myocardial failure and pulmonary œdema. Exposure of the face or neck to bright sunlight after a course of sanocrysin may cause a permanent purple discoloration of the skin due to the gold being deposited in the skin.[1]

A case[2] is recorded where a patient suffering from tuberculosis was treated by gold salts without any ill-effects, but during the second course the blood picture of severe anæmia with marked decrease of the granular white corpuscles (agranulocytosis) occurred and the patient died.

Fatal Dose and Fatal Period.—Not certain. A boy of six years who playfully swallowed a solution of about 12 grains of gold chloride buried in a dust heap, suffered from poisonous symptoms, but recovered after the symptomatic treatment had been adopted.[3] An intramuscular injection of 0.05 gm. of sanocrysin proved fatal to a male phthisical patient, 37 years old, after 8 days. A man was treated with sanocrysin in April, 1934. The first injection of 0.05 gm. was followed by ulticaria of the trunk and arms, so that the doses were decreased to 0.025 gm. and given once a week. The last injection which was given on November 2, was followed by a papular eruption over the right shoulder and upper arm, as well as by diffuse polyneuritis which increased until complete paraplegia was developed. Death occurred on November 17, with symptoms of asphyxia of bulbar origin. The total quantity of sanocrysin injected was in all 5.4 gm.[4] Six grains of gold fulminate caused death, the prominent symptoms being salivation, vomiting and convulsions.[5]

Treatment.—This consists in the administration of eggs and other albuminous substances.

Chemical Tests.—1. Hydrogen sulphide produces a black precipitate, soluble in ammonium sulphide.

2. Ammonia yields a reddish-yellow precipitate.

3. Stannous chloride gives a purple precipitate.

PLATINUM

The soluble salts of platinum are poisonous and act as irritant poisons, the chief symptoms being burning pain in the mouth, salivation, nausea, vomiting, pain in the abdomen, diarrhœa with bloody motions, headache and slight jaundice.

A double chloride salt of platinum and potassium is used in photography. Hence it is liable to cause poisonous symptoms from its accidental internal use. Hardman and Wright[6] report a case in which a woman gave to her infant 8 grains of potassium chloroplatinite in mistake for a teething powder. The infant suffered from the symptoms of gastro-intestinal irritation and died from cardiac failure in 5 hours. On post-mortem examination there was a brownish-yellow patch on the posterior wall of the stomach, otherwise the mucous membrane was pale. The spleen was enlarged. The kidneys were congested and showed punctiform hæmorrhages. Platinum was found in the stomach and intestines. A chronic intussusception was also present.

1. *Beaumont, Brit. Med. Jour.*, 1928, *Vol. II*, p. 815.
2. *Jacquetin and Alenie, Bull. Soc. Med. Hop. Par.*, 1932, *S.* 3, *XLVIII*, pp. 539-47 ; *Med.-Leg. and Criminol. Rev.*, 1934, *Part I, Vol. II*, p. 92.
3. *Stevenson, Guy's Hosp. Report*, 1893.
4. *Jour. Amer. Med. Assoc., April* 13, 1935, p. 1350.
5. *Woodman and Tidy, Forens. Med.*, p. 185.
6. *Brit. Med. Jour.*, 1896 ; *Dixonmann, Forens. Med. and Toxic., Ed. VI*, p. 418.

Chemical Tests.—Hydrogen sulphide gives a dark brown precipitate, insoluble in hydrochloric acid. Concentrated solution of potassium or ammonium salts in presence of hydrochloric acid gives a yellow crystalline precipitate of the double chloride.

NICKEL AND COBALT

Poisoning by the salts of these metals is exceedingly rare. The chief salts that are likely to produce poisonous symptoms are carbonyl of nickel and cobalt. Nickel carbonyl, $Ni(CO)_4$, which is a colourless, mobile, highly refractive liquid, has produced fatal symptoms among workmen employed in nickel work. Toxic symptoms are also produced by the inhalation of air charged with the vapours of nickel carbonyl, which is converted into a gaseous condition at 104° F.

In 1903, two of the men employed at Mond's Chemical Works, Clydach, who had been exposed to the vapours of nickel carbonyl, died and their deaths were found to be due to the inhalation of this substance. Dr. Jones reported that there had been twenty-six cases of slight poisoning under his care last summer in chemical workers. The post-mortem examination of both the bodies showed that the lungs were œdematous and intensely congested, and the heart was in a state of degeneration and loaded with fat. The other organs including the brain were congested. Dr. Mott further examined the brain of one of the deceased and found capillary hæmorrhages in its substance and chromolytic changes in the nerve cells of the medulla and pons, especially of the cardio-respiratory centres.[1]

Workmen employed in the cobalt mines of Schneeberg and Joachimsthal often suffer from malignant lymphosarcoma of the lungs, known as the *Schneeberg lung cancer*. This condition is probably due to the inhalation of the dust of arsenic and radium present in cobalt ores. A case of acute poisoning has, however, occurred from the inhalation of cobalt dust free from arsenic. A young man who was working at the breaking of cobalt in a narrow, unventilated room was taken ill with stomach pains, eructation and very violent vomiting. He suffered from hæmaturia which lasted for three months.[2]

Symptoms.—Nausea, vomiting, headache, giddiness, fever, dyspepsia, convulsions, coma and death.

Post-mortem Appearances.—The stomach and intestines are ecchymosed and inflamed. The brain is congested. The lungs are congested and œdematous. The heart is flabby and dilated.

In the case of a man, aged 49 years, who died in seven days from nickel carbonyl poisoning, the post-mortem examination showed that the lungs were œdematous, with hyperæmia and hæmorrhages. The brain showed haemorrhages with degeneration of small areas and thrombi of some of the vessels.[3]

Chemical Tests.—1. Ammonium chloride, ammonium hydrate and ammonium sulphide yield a black precipitate with nickel and cobalt salts.

2. Caustic potash, soda or ammonium hydroxide gives a green precipitate with a nickel salt, and a blue precipitate with a cobalt salt, soluble in excess.

3. Cobalt gives a blue borax bead and nickel, a reddish-yellow or grey bead.

OSMIUM

Osmium Tetroxide (Osmic Acid), OsO_4.—This is a crystalline salt, melting at 40° C., and boiling at 100° C. It has a caustic burning taste and a penetrating odour. Its vapours are most irritating and poisonous, the chief injurious effects being the inflammation of the eyes and respiratory passages, and the painful eruptions on the skin.

---

1. *Brit. Med. Jour., Jan.* 24, 1903, *p.* 214 *and Feb.* 21, 1903, *p.* 416.
2. *Remond and Favre quoted by Leschke, Clin. Toxic., Eng. Trans. by Stewart and Dorrer*, 1934, *p.* 87.
3. *Brandes, Jour. Amer. Med. Assoc.*, 1934, *Vol. CII, p.* 1204.

CHAPTER XXVI

IRRITANT POISONS—(*Contd.*)

B. ORGANIC POISONS

1. VEGETABLE POISONS

Vegetable purgatives, when given in large doses, act as irritant poisons, and their action is due to an active principle, acrid oil or resin residing in them. When applied externally to the skin, they produce inflammation, pustular eruptions or vesications, and unhealthy callous sores or ulcers. When taken internally, the symptoms of gastro-intestinal irritation are more marked, while the nervous and cerebral symptoms are mostly absent. Death generally results from exhaustion.

The post-mortem appearances are the signs of irritation and inflammation of the alimentary canal.

RECINUS COMMUNIS (CASTOR-OIL PLANT, *ARANDI*)

The castor-oil plant belongs to N. O. *Euphorbiaceæ*. Its seeds contain an active principle, *ricin*, a toxalbumin, which is highly poisonous and produces severe inflammation of the gastro-intestinal tract, affecting primarily the small intestine. It exercises a remarkable power of coagulation so that the blood coming into contact with a minute quantity that has been absorbed is coagulated, blocks the lumina of the intestinal capillaries, and causes thrombosis and ecchymosis.[1]

The fixed oil expressed from the seeds is a pharmacopœial preparation, known as *Oleum ricini*, which is a viscid, nearly colourless or pale yellow liquid with a faint, characteristic odour and a slight but unpleasant taste. It is soluble in $3\frac{1}{2}$ parts of alcohol (90%) and in an equal quantity of absolute alcohol. It is largely used in medicine as a purgative, the dose being 60 to 240 minims.

Symptoms.—Pain in the throat; vomiting; colicky pain in the abdomen; purging with blood or rice water stools; cold skin; prostration; collapse and death.

Fatal Dose and Fatal Period.—The lethal dose of ricin for a man weighing 60 kilogrammes is calculated by Stillmark[2] to be 6 milligrammes, which is generally equal to ten castor oil seeds, although a single seed[3] has produced alarming symptoms, and two seeds[4] have caused the death of a man, 26 years old, in six days. Three seeds[5] have also proved fatal in

1. *H. Stillmark, Arbeit d. Pharmakol. Inst. Dorpat, Pt. III, p.* 59; *Dymmock, Pharmac. Ind., Vol. III, pp.* 304, 310.
2. *Ibid., p.* 304.
3. *Gullan, Brit. Med. Jour., May* 6, 1905, *p.* 988.
4. *Meldrum, Brit. Med. Jour., Feb.* 10, 1900, *p.* 317.
5. *Med. Times and Gaz., May* 25, 1851, *p.* 555.

forty-six hours to a man, 32 years old. On the other hand, recovery has occurred aftery twenty-four seeds,[1] as also after a handful of the seeds (probably twenty-five to thirty or more).[2]

Treatment.—Evacuate by washing out the stomach, administer stimulants and hypodermic injections of morphine, and apply warmth externally.

Post-mortem Appearances.—Congestion, softening and inflammation of the mucous membrane of the alimentary canal, with occasional erosions. Fragments of the seeds may be found in the stomach and intestines. The blood is usually seen in the serous cavities.

Detection of the Seeds.—There are two varieties of the seeds, *viz.*, a large red seed with brown blotches yielding 40 per cent of oil which is largely used for illumination, and a small grey seed having bright, polished, brown spots and yielding 37 per cent of oil, the better quality of which is used for medicinal purposes.

Medico-Legal Points.—Accidental cases occur among children from eating the seeds in mistake. The seeds have been criminally administered in food.

A case is recorded in which a *khidmatgar* (servant), out of spite, gave castor oil in some tea to his master and his wife. Both of them were taken ill. Castor oil was detected in the vomit.[3]

Ricin acts much more powerfully when injected into the blood, than when taken by the mouth, as it is partly destroyed in the stomach by the gastric ferments. When small non-toxic doses are injected subcutaneously for some time, imunity is produced, antiricin being formed.

Ricin is excreted by the intestinal epithelium.

Case.—On March 13, 1905, a farmer labourer in robust health, aged 28, who was on a visit to Liverpool, was watching some bags of castor oil seeds being loaded on one of the Atlantic liners; one of the bags burst, and he picked up a seed and ate it. He noticed that it burnt his mouth and throat a little, and made his eyes water, and then almost immediately he felt weak and collapsed, his knees giving way. He staggered to a policeman on duty to whom he complained of "feeling choked and as if he could not breathe." He was at once taken to the Stanley Hospital, where he was in a state of great collapse; his face was swollen and blue, the pupils a little dilated, the body cold and the hands and feet cold and cyanosed. The respirations were very shallow and the pulse imperceptible at the wrist. He was in a semi-conscious condition though he would reply sensibly when roused. The stomach having been washed out with a warm saline solution, he improved under the treatment of stimulants and hypodermic injections of atropine and strychnine and was able to leave the hospital on March 18.—*Gullan, Brit. Med. Jour., May* 6, 1905, *p.* 988.

CROTON TIGLIUM (CROTON, *JAMALGOTA* OR *NAEPALA*)

This plant belongs to N. O. *Euphorbiaceæ.* Its seeds are very poisonous, and contain *crotin*, a toxalbumin, similar to ricin, but less poisonous. The fixed oil (croton oil) extracted from the seeds contains a powerful vesicating resin composed chiefly of crotonoleic acid, tiglic or

1. *Park, Glas. Med. Jour.*, 1880.
2. *L. T. R. Hutchinson, Ind. Med. Gaz., May,* 1900, *p.* 196.
3. *Sind Chemical Analyser's Annual Report,* 1925, *p.* 22.

methyl crotonic acid, crotonol and several volatile and fatty acids. It is brownish-yellow to dark reddish-brown in colour, and has a disagreeable odour and an acrid burning taste. It dissolves freely in alcohol, ether, chloroform or olive oil. It is a non-official preparation, known as *Oleum crotonis* (*Oleum tiglis*), and is given as a drastic purgative in ½ to 1-minim doses.

When dropped on the skin, croton oil produces burning, redness and vesication; the vesicles may later suppurate and cause scarring. When swallowed, it acts on the stomach and intestines and produces gastro-intestinal irritation.

Symptoms.—Hot burning pain in the mouth and throat extending to the abdomen; salivation; vomiting; purging with severe griping pain and bloody stools; great prostration; collapse and death.

Fatal Dose.—A single seed is said to have produced severe symptoms of poisoning, and four seeds have caused death. Three minims of the oil[2] proved fatal to a child, 13 months old. Twenty and thirty minims respectively have proved fatal to adults, while recovery has followed half-an-ounce of the impure oil.[3]

Fatal Period.—A man died in four hours after swallowing two drachms and-a-half of croton oil.[4] A child who was given three minims of the oil by mistake died in six hours. An aged woman died in convulsions in three days from a tea-spoonful of croton oil embrocation.[5]

Treatment.—Wash out the stomach; administer demulcent drinks and morphine to allay pain, and stimulants to combat collapse.

Post-mortem Appearances.—Inflammation of the alimentary canal is usually found, but in the case of a man who died from a dose of two drachms and-a-half of croton oil no marked change was found in the mucous membrane of the stomach.

Tests.—The oil should be extracted from the contents of the stomach or other substances by means of ether after they have been slightly acidulated with tartaric acid. After evaporation of the ether, the residue will produce irritation or vesication, if a drop is rubbed on the inside of the arm.

The following chemical test is at times done in the Chemical Examiner's Laboratory at Agra:—

Treat the oil with twice its volume of absolute alcohol; pour the clear alcoholic solution into a concentrated solution of caustic soda or caustic potash (up to 40 per cent). A brilliant brownish-red or reddish-violet ring according to the age or origin of the oil indicates the presence of croton oil.

1. *Dixonmann, Forens. Med. and Toxic., Ed. VI, p.* 537.
2. *Med. Times and Gaz.,* 1870, *Vol. II, p.* 466.
3. *Dixonmann, Loc. Cit.*
4. *Orfila, Toxic., Vol. I, p.* 108; *Taylor, On Poisons, Ed. III, p.* 501.
5. *Med. Gaz., Vol. XLIII, p.* 41.

Detection of the Seeds.—The croton seeds are ½ inch long, 1/3 inch broad, oval or oval-oblong, odourless and about the size of a grain of coffee. They are covered with a dark brown or brownish-grey shell, which on scraping becomes black. The kernel is white and oily. One seed weighs about four grains. The seeds resemble very much the smaller variety of castor oil seeds, but the latter are bright, polished and mottled.

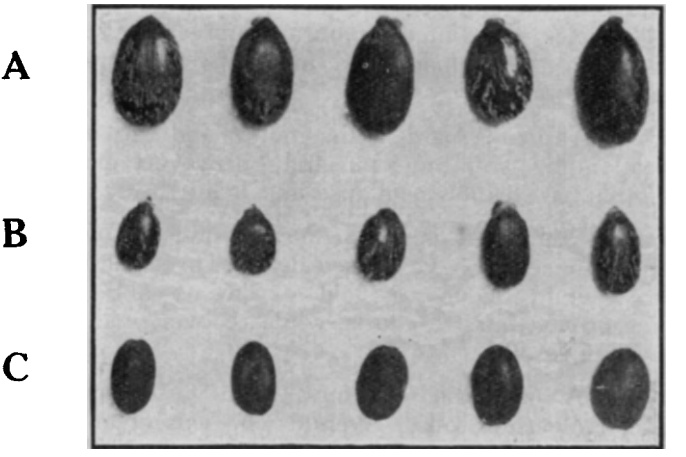


Fig. 135.—A. Castor oil seeds (large variety)
B. Castor oil seeds (small variety)
C. Croton seeds.

Medico-Legal Points.—Poisonous symptoms have been produced by eating the seeds or by inhaling their dust. Accidental poisoning has resulted from swallowing croton oil by mistake, from its administration as a purgative by quacks in too large medicinal doses or from taking internally its preparations meant for external use.

A case[1] is recorded where several people suffered from great stomach disorder and vomiting after eating ice-cream accidentally mixed up with croton oil by mistake at a marriage party in Delhi.

On November 18, 1928, a Hindu male, 45 to 50 years old, was admitted into the King George's Hospital, Lucknow, with a history that about 14 days ago he had been given croton oil by a *hakim*. At the time he was very much emaciated with hollow cheeks and sunken eyes, and was passing involuntarily frequent motions, which were watery and dark brown in colour. The pupils were normal, the eyes being injected. The pulse was feeble. He died at 3-35 p.m. on November 28—twenty-four days after he took croton oil. On post-mortem examination the stomach contained one pound of muddy coloured liquid with mucus adherent to the mucous membrane. This was inflamed and was excoriated at the cardiac end and at the first half of the greater curvature. There were small patches of submucous hæmorrhages at the cardiac and pyloric ends. The vessels of the stomach were engorged with blood. The small intestine was empty, and inflamed with extensive hæmorrhages along its wall. The large intestine contained watery greenish-yellow fæcal matter. It was congested and was marked with deep ulcers along its lower part. The spleen was congested and enlarged, and the liver and kidneys were congested. Microscopically the stomach showed extensive necrosis of its mucous membrane,

1. *Leader, June* 29, 1924.

acute congestion and a certain amount of inflammation of the submucous coat. There was very marked congestion of the small intestine with necrosis of the superficial layer of the mucous membrane. There was marked acute congestion of the large intestine, with the mucous membrane and submucous coat filled with acute inflammatory catarrhal cells and with deep ulcers with markedly inflamed margins. There were cloudy swelling and intense congestion of the kidneys.

Croton oil has been taken as an abortifacient and has been administered in food with homicidal intent. In his annual report for 1923, the Chemical Examiner of the United Provinces reports a case from Bareilly, where two persons suffered from irritant poisoning after taking some *gulgulas* (a kind of sweetmeat) in which croton oil was detected.

A case[1] of ordeal by croton seeds is recorded. A man and his brother were suspected of having stolen two bales of yarn and Rs. 200|- from their co-tenant. The owner of the property decided to discover the thief through black magic. He enlisted the services of a quack, who held a *puja* before an idol and distributed black pills, one pill to each of the assembled villagers. The two suspected brothers also were each given a pill. These two were white and quite different from the black pills. One of the brothers protested at what appeared to him to be an obviously invidious distinction but the quack explained that the whiteness of the pills was due to accidental coating with sacred ash. As the explanation was apparently satisfactory, this brother gulped down the pill. The other brother who was more cautious ate only a part of the pill and kept the other portion. The first victim who swallowed the whole pill developed purging, vomited blood and died in 20 to 24 hours. The second victim who had swallowed only part of a pill recovered under proper treatment. The Chemical Examiner detected, by microscopic examination, in the stomach and in the intestines of the deceased, tissue similar to that found in the outer covering of croton seeds. The suspected poison and a grinding stone also showed under the microscope tissue resembling that found in the outer covering of croton seeds. The quack was arrested and sentenced to undergo rigorous imprisonment for two years.

Croton seeds are poisonous to fish and a case is recorded where croton oil was used for poisoning fish in a tank in Contai, Midnapur.[2]

The root of the plant is used as an abortifacient in Malay Peninsula and the fruit is, sometimes, boiled in water and added to food with homicidal intent.[3]

Croton oil is, sometimes, employed by wild tribes to poison their arrows, but Windsor found that the arrow poison used by the Abor tribe of the North-East Frontier of India was a paste made by pounding the soft parts of croton tiglium, and not obtained from the seeds.[4]

When applied to the skin, croton oil may produce watery and bloody stools owing to the excretion of the crotonoleic acid into the intestines.

COLOCYNTH (BITTER APPLE, *INDRAYAN*)

This is the dried pulp of the fruit of *Citrullus Colocynthis* (*N. O. Cucurbitaciœ*), which grows widely throughout India. The pulp freed from its seeds is a pharmacopœial drug, called *Colocynthis,* the dose being 2 to 5 grains. It occurs as white, spongy, light fragments, having an intensely bitter taste. Its official preparations, *Extractum colocynthidis*

1. *Madras Chem. Examiner's Annual Rep.,* 1936, *p.* 9.
2. *Beng. Chem. Exam. Rep.,* 1916 ; *Ind. Med. Gaz., Aug.,* 1917.
3. *Gimlette, Med. Pois. and Char. Cures, p.* 146.
4. *Ind. Med. Gaz., Jan.,* 1912, *p.* 11.

compositum and *Pilula colocynthidis et hyoscyami*, are largely used as purgatives in doses of 2 to 8 grains and 4 to 8 grains respectively.

The root and the fruit of the plant contain a glucoside, colocynthin, which is amorphous or crystalline, bitter in taste and readily soluble in water and alcohol. It is a drastic purgative and acts as a powerful irritant to the ailmentary canal, when taken in large doses.

Symptoms.—Severe abdominal pain ; vomiting of a yellow colour containing mucus but no blood ; frequency of watery, yellow coloured stools, often stained with blood ; irregular pulse ; collapse and occasionally death.

Fatal Dose and Fatal Period.—Fifteen to thirty grains have caused death in a few cases.[1] According to Roques[2] a decoction of less than a drachm of the powder has proved fatal. Christison[3] records the case of a young woman who died in twenty-four hours after swallowing a tea-spoonful and-a-half of the powdered pulp. One to two drachms have proved fatal to a woman in forty hours.[4] Recovery has, however, followed much larger doses, even as much as 3 ounces.[5]

Treatment.—Empty the stomach, give morphine to allay pain and administer demulcents, astringents and stimulants.

Post-mortem Appearances.—Redness, inflammation and occasionally ulceration of the stomach and intestines. The liver and kidneys may be inflamed.

Medico-Legal Points.—Colocynth is occasionally taken for the purpose of committing suicide or for procuring abortion. Borton[6] mentions the case of a young woman, aged 18 years, recently married, who under the impression that she had become pregnant took two tea-spoonfuls of the powdered pulp and suffered from poisonous symptoms though she recovered under prompt treatment. Butler[7] records another case in which a married woman, 26 years old, took about four tea-spoonfuls of the drug with a view to procure abortion. She also suffered from the poisonous symptoms but did not abort.

ERGOT

This is the sclerotium (compact mycelium or spawn) of the parasitic fungus, *Claviceps purpurea*, attacking the grains of several plants, such as rye, oats, wheat, barley and *bajra*, in wet seasons and in ill-drained soils. The ear of the plant is occupied wholly, or in part, by the diseased grains, each of which is of a deep purple colour, tapering at both ends, curved and 1/3 to 1½ inches long. These diseased grains collected, dried and powdered form the ergot of the shops.

1. *Querleux, Arch. de Med. et. Pharm. milit.*, 1909, 53, *p.* 276 ; *Peterson, Haines and Webster, Leg. Med. and Toxic., Vol. II, Ed. II, p.* 756.
2. *Taylor, Princ. and Pract. of Med. Juris., Vol. II, Ed. IX, p.* 779.
3. *Poisons, p.* 595.
4. *Tidy, Lancet, Feb.* 1, 1868, *p.* 158.
5. *Orfila, Toxic., Vol. I, p.* 695.
6. *Brit. Med. Jour., June* 8, 1907, *p.* 1364.
7. *Ibid., June* 29, 1907, *p.* 1537.

Ergot is lighter than water, and has a disagreeable odour and a mawkish rancid taste. It contains three principal alkaloids, ergotoxine, ergotamine and ergometrine, together with tyramine, histamine and acetyl choline which are formed by the breaking down of the proteins of rye during the growth of the fungus.

Ergometrine, recently isolated by Dudley and Moir,[1] differs from the other alkaloids in the fact that it produces its effects much more rapidly, that it is less active in producing gangrene when administered for a prolonged period and that its use is not followed by nausea, headache and depression.

Ergot is contained in *Extractum ergotæ liquidum*, the dose of which is 10 to 20 minims. When powdered and deprived of its fat, ergot forms *Ergota preparata* (Prepared ergot), the dose being 5 to 15 grains. *Ergotoxinæ æthanosulphonas* is an official preparation derived from ergot. It occurs as colourless, odourless crystals, sparingly soluble in water, but freely in methyl alcohol. The dose is 1/120 to 1/60 grain by subcutaneous or intramuscular injection.

Acute Poisoning—Symptoms.—Dryness and irritation of the throat, thirst, nausea, vomiting, burning pain in the stomach, colic, slight diarrhœa, giddiness, dilatation of the pupils, depressed action of the heart, suppression of the urine, cramps, stupor, convulsions, abortion in pregnant women, delirium, and coma. Death occurs usually from asphyxia.

Chronic Poisoning (Ergotism).—This occurs among those who take ergot as a medicine for a long, continued period or among people who eat bread made of rye flour infested with the ergot fungus.

Symptoms.—The symptoms are those of gastro-intestinal catarrh, followed by a convulsive or gangrenous form. In the convulsive form the patient complains of itching, tingling and numbness of the hands and feet, which soon spread over the whole body. He then gets tonic contractions of various muscles, especially those of the extremities. Dimness of vision, loss of hearing, epileptiform convulsions and dementia are the next symptoms from which the patient suffers. Death occurs from asphyxia due to spasm and weakness of the respiratory muscles.

In the gangrenous form the fingers and toes become painful, swollen, and become dark owing to dry gangrene setting in. Gangrene may extend up to the elbow or knee.

Stewart McKay[2] reports the case of a married woman, aged 30 years, who suffered from gangrene of the fingers following the administration of liquid ergot. She purchased from a chemist a twelve-ounce bottle containing ergot and finished it in one week with the idea of inducing abortion. But having had no desired effect in three days she obtained a second bottle containing the stronger medicine, which she finished in seven days. However, before she had finished the mixture she noticed that her arms began to ache, her skin was itching and her fingers were swollen, which slowly became gangrenous, though she did not abort.

Drs. Robertson and Ashby[3] describe an outbreak of chronic ergot poisoning among the Jewish population of Manchester which used black bread made from rye

1. *Brit. Med. Jour., March* 16, 1935, *p.* 522.
1. *Brit. Med. Jour., August* 18, 1906, *p.* 365.
2. *Ibid., Feb.* 25, 1928, *p.* 302.

flour as an article of diet. The general symptoms complained of were coldness in the extremities, numbness and lack of sensation in the fingers—a sensation like an insect creeping over the skin—headaches, depression, twitchings in the limbs, and staggering gait. One of the affected had a definite dry gangrene of both hands. From investigations it was found that the average Jewish person consumed about half a pound of rye bread per diem, the flour of which contained one per cent of ergot. Half a pound of bread contained about five to six ounces of flour, the rest being the water which was added before baking. Five ounces of flour equal 2285 grains, of which one per cent was ergotised. Each person thus consumed 22.85 grains of ergot daily.

Fatal Dose and Fatal Period.—Thirty grains of ergot have caused severe symptoms of poisoning.[1] Two handfuls of powdered ergot have caused the death of a pregnant woman.[2] On the other hand, recovery has occurred after taking 150 grains. One fluid drachm of the liquid extract of ergot has produced toxic effects,[3] while ounce-doses have often been administered without any deleterious effects.

Treatment.—Give emetics or wash out the stomach with warm water, and empty the bowels by purgatives or enemata. Keep up the body heat; use stimulants and amyl nitrite for inhalation. In chronic poisoning the treatment should be directed to remove the cause.

Post-mortem Appearances.—Jaundice of the skin and ecchymoses of the blood in the abdominal organs. The lungs, kidneys and uterus may be hyperæmic.

Chemical Analysis.—Ergot may be separated from an organic mixture, suspected bread or flour by treating it with alcohol acidulated with sulphuric acid. The extract thus obtained is red in colour, and shows two bands—one in the green and the other in the blue—in the spectroscope. If heated after adding caustic potash, ergot assumes a lake-red tint, and emits a fishy odour, which is due to the evolution of trimethylamine.

The following colour tests may also be applied for detecting the alkaloids of ergot:—

1. If a small amount of the alkaloidal residue be dissolved in about 1 c.c. of concentrated sulphuric acid and a trace of ferric chloride solution be added, the solution acquires an orange-red colour changing to deep red, while the margin appears bluish or greenish-blue.

2. To a small amount of the alkaloidal residue dissolved in a few cubic centimetres of glacial acetic acid add a trace of ferric chloride solution. If this solution is allowed to float cautiously on concentrated sulphuric acid contained in a test tube without shaking it, a brilliant violet or intense blue colour is formed at the zone of contact.

3. About 2 grammes of finely powdered ergot are freed from oil with 10 to 15 c.c. of petroleum ether in a small separation funnel closed with a plug of cotton wool. An infusion is prepared from 1 gramme of the ergot thus treated in 20 grammes of water and 1 drop of hydrochloric acid. Four grammes of this corresponding to 0.2 gramme

1. *Meadows, Med. Times and Gaz.*, 1879, *Vol. II, p.* 397.
2. *Davidson, Lancet*, 1882, *Vol. II, p.* 526.
3. *Faulkner, New York Med. Jour.*, 1844, *XXXIX, p.* 668.

of ergot are filtered off, and after the addition of 1 drop of ammonium hydroxide are vigorously shaken with 10 c.c. of ether. Five c.c. of the clear ether are withdrawn and layered on about 2 c.c. of pure sulphuric acid in a test tube; within a few minutes a corn-flower blue zone must form about 0.5 m.m. below the interface of the two liquids. After standing for one-and-a-half to two hours it becomes wider and less distinct, until it gradually fades away. It can best be observed in dispersed light by holding the test tube against a window fitted with frosted glass.[1]

Medico-Legal points.—Ergot is largely used as an abortifacient. Its action is more effective on the uterus, which is already contracting. It fails in the early pregnancies. Fatal cases do not seem to occur from a single large dose, but from small or medicinal doses administered for a long time.

CAPSICUM ANNUUM AND CAPSICUM FRUTESCENS (CHILLIES, RED PEPPER, CAYENNE PEPPER, *LALMIRCH*)

These plants belong to N. O. *Solanaceæ*. Capsicum fruits are powdered and are then universally employed in India as a principal condiment in preparing various *chutneys* and *curries*. The chief constituents to which capsicum fruits owe their pungency and acridity are capsaicin, capsicin (a crystallisable substance), a volatile alkaloid smelling like coniine, a volatile oil, a resin and fatty matter. The dried ripe fruit of *capsicum minimum* is known as capsicum and is used in medicine as a pungent stomachic and carminative in doses of ½ to 2 grains. Capsicum also occurs in the official preparations of *Tinctura capsici* (dose, 5 to 15 minims) and *Unguentum capsici.*

In large doses capsicum acts as an irritant poison and causes difficulty of swallowing, pain in the stomach and inflammation of the œsophagus and stomach. Locally applied, it produces redness and even vesication of the skin.

Chillies are used in India for the purpose of torture, when money or confession of some guilt has to be extorted. They are either introduced into the vagina, rectum or urethra, or rubbed on the breasts of females. The "Pindaris" used to torture their victims by covering their heads with nose-bags containing chillies. Well-pounded chillies are, sometimes, thrown into the eyes to facilitate robbery. A peon in Calcutta cashed a cheque for four thousand rupees, and while he was passing through Dalhousie Square a man threw a quantity of well-pounded chillies into his eyes and blinded him for the time being. When the peon was in agony the man relieved him of his money and tried to make good his escape, but was arrested.[2]

The fumes arising from burning chillies are very irritating, and are used by superstitious people to scare away devils and ghosts.

A boy, 15 years old, who was suffering from hip joint disease, died after swallowing medicine containing cayenne pepper prescribed by a

1. *K. Hering, Ap. Ztg.*, 43, 91, 1381; *Jour. State Med., June,* 1929, *p.* 369.
2. *Leader, Aug.* 27, 1926.

medical botanist. On post-mortem examination the stomach was found red and inflamed in patches. The botanist was charged with having caused the death of the boy, but was acquitted.[1]

The seeds, which are contained in a capsule, resemble *datura* seeds.

ABRUS PRECATORIUS (JEQUIRITY, INDIAN LIQUORICE, *GUMCHI* OR *RATI*)

This is a beautiful climbing plant, belonging to N. O. *Leguminosœ* and found all over India. Its seeds are egg-shaped and scarlet in colour, with a black spot at one end, and are each about 1/3rd inch long and $\frac{1}{4}$th inch broad, having an average weight of $1\frac{3}{4}$ grains. They are used by Indian goldsmiths for weighing silver and gold. White seeds are also met with.

The seeds contain an active principle, *abrin*, a tox-albumin, similar in action to ricin extracted from castor oil seeds. In addition to this, the seeds[2] contain poisonous proteins, a fat-splitting enzyme, abrussic acid, hæmagglutinin and a quantity of *urease*. The shell of the seeds contains a red colouring matter.

Abrin is a tasteless, amorphous solid, having a pale grey colour. It dissolves readily in cold water with the exception of a few flocks, and the solution, which is of a faintly yellow colour, froths on agitation. It is also soluble in glycerine.[3] The root and the stems also contain an active principle, glycyrrhizin. Abrin loses its activity when boiled and, therefore, the seeds, when cooked, may be used without any harmful effects. The seeds are powdered, boiled with milk, and are then used as a nervine tonic in 1 to 3-grain doses. If administered uncooked, they produce vomiting and diarrhœa. A decoction of the decorticated seeds, if instilled into the eyes, will produce purulent ophthalmia and may cause fatal poisoning due to its absorption through the conjuctivæ.

Symptoms.—In a few hours after an extract of the seeds is injected under the skin of an animal, an œdematous swelling with ecchymosis appears, and slowly extends over a portion of the body. The animal is disinclined to take food, and three or four days later it drops down and is unable to move. It then gets tetanic convulsions, or becomes cold, drowsy and comatose, and dies in twenty-four to forty-eight hours.

The symptoms are very much like those of snake-poisoning. Hence the peasants think that the animal died from the effects of a snake-bite.

In human poisoning a swelling with ecchymosis occurs near the seat of injection which becomes painful. The swelling rapidly increases and erysipelas supervenes. Death occurs from 3 to 5 days.

1. *R.* v. *Stevens, C. C. C., May,* 1864 ; *Taylor, On Poisons, Ed. III, p.* 505.
2. *Chopra, Indigenous Drugs of India,* 1933, *p.* 263.
3. *Warden and Waddell, The Non-bacillary Nature of Abrus Poison, Calcutta,* 1884 ; *Ind. Med. Gaz., July,* 1884, *p.* 184.

In a case[1] of attempted suicide where the powdered seeds of abrus precatorius were taken with ground nut oil, the symptoms were vomiting, feeble pulse, cold, clammy skin, and sunken eyes with normal pupils. No deep sleep, no tingling of the skin or throat, no convulsions or twitchings or no delirium was noticed.

Fatal Dose.—One and-a-half to two grains. Half a grain of the prowdered seed rubbed up with ten minims of distilled water and injected subcutaneously into cats killed them in 19½ to 40 hours.[2]

Doses[3] of about 0.0005 mgm. to 0.001 mgm. of abrin per kilogramme of body weight injected subcutaneously are said to be poisonous.

Fatal Period.—The average fatal period is 3 to 5 days. The shortest is 24 hours.

Treatment.—Anti-abrin can be produced by repeated, small and gradually increasing doses which can be used curatively in abrus poisoning.

Post-mortem Appearances.—Fragments of a " sui " containing ground-up seeds of abrus precatorius are usually found in the wound, which may, sometimes, be so small as not to be easily observed. Œdema is found at the seat of injection and patches of ecchymoses like purpura are seen under the skin, pleura, and pericardium, and peritoneum. The mucous membrane of the stomach and intestines is highly congested with numerous hæmorrhagic patches on its surface as well as in the interior of the organs, such as the lungs, liver and spleen.

Test—Physiological.—A watery infusion of abrin or a decoction of the seeds, if dropped into the eye, causes purulent ophthalmia.

A thin emulsion of the bruised seeds in distilled water, when injected hypodermically into a fowl, kills the bird in twelve to twenty-four hours.

Medico-Legal Points.—The seeds of Abrus precatorius are usually employed criminally for destroying cattle, and occasionally for homicidal purposes. The seeds alone, or mixed with *datura*, opium and onion, are worked with a small quantity of spirits into a paste, which is made into spikes or " suis ", and then hardened in the sun. These spikes which weigh, on an average, 1½ to 2 grains, are then placed in a wooden handle, and thrust with great force into the skin of the animal intended to be killed. For homicidal purposes the spike is kept between two fingers, and is pushed into the skin while slapping a person.

The spikes thus prepared are less active than the freshly powdered seeds. One spike weighing two grains on being rubbed up with water and injected subcutaneously into a chicken does not usually produce a fatal result till after the lapse of thirty-six hours ; whilst half a grain of the freshly powdered seed produces death in about eighteen hours.[4]

1. *Madras Chemical Examiner's Annual Report*, 1924.
2. *Warden and Waddell, Loc. Cit., Ind. Med. Gaz., June*, 1884, *pp.* 155, 156.
3. *Chopra, Indigenous Drugs of India*, 1933, *p.* 263.
4. *Warden and Waddel, Loc. Cit., Ind. Med. Gaz., July*, 1884, *p.* 189.

Cases of human poisoning by " sui " pricking, though very few, have occurred especially in the district of Drug, C.P., and in the districts of Bareilly, Pilibhit and Shahajahanpur, U.P. In his annual report for 1908, the Chemical Examiner of the United Provinces mentions the following case of human " sui " poisoning, which occurred in the district of Drug : —

" The deceased was sleeping on a *charpoy*. Some one came into the room and gave him a slap on his right cheek. A wound was found in this position, in which were pieces of the foreign substances. More pieces of the foreign body were found on the *charpoy*. These pieces were found to be fragments of a " sui " as used in " sui " poisoning of cattle, and contained ground-up seeds of Abrus precatorius. Death occurred in two days and thirteen hours later after the symptoms of inflammation in the chest, eyes, neck and mouth."

The Chemical Examiner of Bengal also describes the following two homicidal cases of " sui " poisoning : —

A Santalin widow had some property and her husband's younger brother who was likely to inherit the property after her death, was not on good terms with her. So she had executed a deed adopting her brother's son. While asleep one night, an unknown person came at midnight and gave a *chati* or slap of *bish* (poison) on her chest. She extricated the thorn-like substance from her chest and kept it. Next morning she went to her brother's house in another village. On the fifth day after the injury she felt serious pain in the chest and a local doctor was called in. She could then speak with great difficulty. She died on the seventh day after the injury. Abrus precatorius was detected in the thorn-like substance.[1]

A Hindu woman was attacked by her nephews one night while sleeping and severely handled by them. They then ran away, and she felt a burning sensation over her body and found some broken pieces of a conical shaped substance stuck near her breast and other similar fragments in her bed. The woman did not die. Abrus precatorius was detected in the fragments.[2]

SEMECARPUS ANACARDIUM (MARKING NUT TREE)

This tree belongs to N. O. *Anacardiaceœ*. Its fruit, called marking nut (*Bhilawan*), weighs 25 to 55 grains, and has a hard, black rind within which is a thick pericarp. The pericarp or fleshy pulp of the fruit or seed abounds in a brownish, oily, acrid juice, which turns black on exposure to the air, and is used by *dhobis* (washermen) as " marking ink " in admixture with lime for linen and cotton clothes. It was thought that this oily juice was closely allied to the vesicating oil obtained from the pericarp of cashew-nut (*Anacardium occidentale, Kaju*), and that it consisted of about 90 per cent of an oxyacid, named anacardic acid and 10 per cent of a higher, non-volatile alcohol, called cardol. Naidu[3] claims to have isolated from it catechol and a monohydroxyphenol, called anacardol, besides two acids, and a fixed oil from the kernel of the nut. As a result of their investigations Pillay and Siddiqui[4] have succeeded in establishing that neither anacardic acid and cardol nor catechol and anacardol are present in *Bhilawan*, and have isolated the following from the juice of the pericarp : —

1. A monohydroxyphenol, named *semecarpol*, which boils at 185-90°/2.5 mm., congeals below 25° to a fatty mass and forms 0.1 per cent of the extract.

1. *Annual Report*, 1929, *p.* 14.
2. *Annual Report*, 1930, *p.* 10.
3. *Jour. Indian Inst. of Science*, 1925, *VIIIA, p.* 129.
4. *Jour. Ind. Chem. Soc.*, 1931, *Vol. VIII, p.* 517.

2. An o-dihydroxy-compound, named *bhilawanol,* which distils constantly at 225-26°/3 mm., congeals below 5°, and forms 46 per cent of the juice (15 to 17 per cent of the nut).

3. A tarry corrosive residue, out of which no further chemical individual could be isolated even after repeated purification by means of solution in dilute alcohol and precipitation with alcoholic lead acetate.

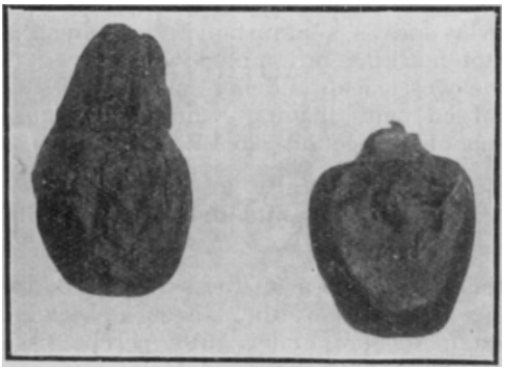


Fig. 136.—Marking nuts.

The alcoholic extract of the pericarp was found to contain an ether-soluble and an ether-insoluble acid besides tannic acid, and the ethereal extract of the kernel contained 32.3 per cent of a fatty oil.

The juice is used internally in 1 to 2-minim doses as a remedy for nervous and scrofulous affections and syphilis. Mahomedan writers consider 2 *dirhems* (about 144 grains) of the juice to be a fatal dose. When applied externally, it produces irritation and a painful blister containing an acrid serum, which produces eczematous eruptions of the neighbouring skin, with which it comes into contact. These eruptions may develop into deep ulcers and cause sloughing and even death, if the juice is applied to the scrotum.[1] They are usually accompanied by constitutional symptoms, such as fever, reddish-brown and bloody urine and pain in passing urine as well as stools.[2]

During the process of chemical examination of marking nut juice a little of the liquid was rubbed on the skin of the dorsum of the left hand of S. R. Nayudu. After an interval of two days it produced very severe irritation and blistering. The blisters tended to spread along the margin till the whole dorsum of the hand was swollen and blistered. There was very intense itching and oozing of serum. The hand took about a fortnight to heal and the dorsum of the hand was stained black for some weeks at the spot of the application of the juice. Some time after the healing of the blisters on the hand, Nayudu developed suppurative lymphadenitis of the axilla which required operative treatment, and kept him in bed for two months.[3]

Accidental poisoning may result from the administration of the juice by Hakims or Vaids. In Bombay an oily substance was applied by a Hakim to the paralysed limbs of a child of 12 years, from the corrosive action of which the child died in the G. T. Hospital. The substance proved on analysis to be a preparation of marking nut.[4] A Hindu male of Angul took some milk boiled with marking nuts for relief of pain in the chest and had vomiting and purging and died after a few hours.[5]

1. *Chevers, Med. Juris., p.* 286.
2. *Dymmock, Loc. Cit., p.* 391 ; *Am. Jour. Pharm., March,* 1882, *p.* 131.
3. *Madras Chem. Examiner's Annual Rep.,* 1933, *p.* 10.
4. *Bombay Chem. Analyser's Annual Report,* 1925, *p.* 6.
5. *Bengal Chem. Examiner's Annual Report,* 1929, *p.* 13.

Criminally the juice is introduced into the vagina as a punishment for infidelity, is applied to the skin to produce a bruise to support a false charge, or is thrown over the body of an enemy out of revenge. Some twigs imbued with marking nut juice were thrown into the bed of a man, and when his feet touched them, they produced severe vesication. The juice of marking nut was detected on the twigs.[1] During his wife's absence a man had been carrying on with a woman, but on his wife's return he stopped visiting the woman. The woman was very much annoyed with the wife of her lover and as a punishment for alienating his love, she poured some juice of marking nut mixed with oil on the private parts of the wife when she was asleep and her husband was not at home. The woman was charged with having voluntarily caused grievous hurt by means of a poison under section 326 I.P.C.[2]

The bruised nut is, sometimes, applied locally to the os uteri for inducing criminal abortion.[3] It is also instilled into the eyes by malingerers to produce ophthalmia.

Detection.—To find out whether a vesication on the skin is produced by marking-nut juice, remove the epidermis of the blister and extract with alcohol, or apply lint soaked in alcohol under gutta percha tissue over the vesicle. The alcoholic extract with liquor potash assumes a bright greenish colour turning to reddish-brown.

The Chemical Examiner,[4] Madras, employs the following tests for the detection of the stains on a cloth caused by marking-nut juice :—

The stains are extracted with alcohol. To the alcoholic solution lead acetate is added to precipitate albuminoids and organic acids and the solution is filtered. To the filtrate ammonium oxalate is added to de-lead it. The solution is again filtered, diluted with water and extracted with petroleum ether. After evaporation, this petroleum ether extract yields a thick brownish liquid which turns bluish-green with a solution of caustic potash. When a little of the liquid is rubbed on the skin, it produces after an interval of about two days a painful and irritating blister which spreads over the surrounding area.

CALOTROPIS GIGANTEA AND PROCERA (*MADAR, AKDO*)

These plants belong to N. O. *Asclepiadeœ*, and grow wild on waste ground throughout India. The fresh leaves and stalks of these plants on crushing exude a thick, acrid, milky juice, which, according to Rajagopal Naidu,[5] is acid in reaction and forms into a white clot or coagulum leaving a clear straw coloured serum after it is left for some time. The coagulum yields a yellowsh-brown resin and a snow white, crystalline substance, having the formula $C_{27}H_{46}O_2$. The resin is slightly poisonous, about eight milligrammes being necessary to kill a frog, weighing about 20 grammes, while the white crystalline substance is insoluble in water and is

1. *Madras Chemical Examiner's Annual Report*, 1924.
2. *Beng. Chem. Exam. Ann. Rep.*, 1937, *p.* 11.
3. *Chevers, Med. Juris.*, *p.* 267.
4. *Annual Report*, 1933, *p.* 10.
5. *Madras Chemical Examiner's Annual Report*, 1936, *p.* 13 ; *see also Ibid.*, 1932, *p.* 3 ; *Ibid.*, 1933, *p.* 11.

non-poisonous, but it is soluble in most of the organic solvents, such as alcohol, acetone, ether and petroleum ether, and still more soluble in chloroform and carbon tetrachloride.

The serum yields a water soluble extract which is highly poisonous, half a milligramme causing convulsions, paralysis and death in a frog and two hundred milligrammes proving fatal to a rabbit, weighing about 34 ounces.

Fig. 137.—Calotropis Gigantea.

When applied to the skin, *madar* juice acts as an irritant poison, producing redness, inflammation and vesication. It irritates the eyes and may cause inflammation involving eyesight when dropped into them. When administered internally, it acts as a gastro-intestinal irritant and also as a cerebro-spinal poison.

Symptoms.—Acrid bitter taste, burning pain in the throat and stomach, salivation, stomatitis, vomiting, diarrhœa, dilated pupils, tetanic convulsions, collapse and death.

Fatal Dose.—Not determined.

Fatal Period.—A boy, aged about 8 years, who was given some *madar* juice with milk for treatment of dog bite, died in about half an hour.[1] A woman[2] died in one hour. She drank *madar* juice at about 7 a.m. The symptoms began at 7-30 a.m. with vomitings and purgings, followed by convulsions in which she rolled on the ground. She collapsed and died at about 8 a.m. A man[3] in Jhang drank *madar* juice with intent to commit suicide, and died within two hours. On analysis the typical cauliflower crystals of *madar* juice were detected in the stomach and intestines.

Treatment.—Lavage the stomach cautiously. Administer demulcent drinks. Give morphine hypodermically to relieve pain and to prevent convulsions. Administer diffusible stimulants to combat collapse.

Post-mortem Appearances.—Signs of irritation in the stomach and intestines. In a case from Tonk where *madar* juice was found in the viscera of a baby, three months old, the post-mortem appearances were the signs of stomatitis in the mouth ; the stomach was perforated in a few places, and milk was found on the surface of the intestines.[4] In the case of a woman who died within one hour after *madar* juce had been swallowed, the post-mortem examination showed bloody discharges in the nostrils and mouth. The stomach was congested and contained about 2

1. *Bengal Chem. Exam.'s Annual Rep.*, 1936, *p.* 12.
2. *Madras Chem. Examiner's Annual Report*, 1933, *p.* 6.
3. *Punjab Chemical Examiner's Annual Report*, 1926, *p.* ii.
4. *U. P. Chemical Examiner's Annual Report*, 1922, *p.* 3.

ounces of chime-like fluid. The small intestines were congested. The liver, spleen and kidneys were congested. The trachea was injected. The heart was empty. The brain and its membranes were congested. The reactions of *madar* juice were obtained from the stomach and its contents.

Tests.—Col. Black, late Chemical Examiner for the Punjab, recommended to Mr. Chatterji, late Chemical Examiner for the Central and the United Provinces, the following test as successfully employed by him :—

The material under examination is heated for a sufficiently long time with absolute alcohol under a reflux condenser. If now the alcoholic extract is allowed to evaporate spontaneously, characteristic cauliflower-like masses separate out and are readily identified. But Mr. Chatterji has found the masses which separate out as "nodular", and he relies on the following tests for the identification of *madar* juice :—

1. The suspected material is digested with absolute alcohol for about an hour under a reflux condenser. The extract is distilled with the addition of a little 50 per cent (by volume) sulphuric acid in the presence of alcohol.. The distillate has a characteristic fruity odour. This should be compared with the odour obtained from *madar* juice under similar conditions.

2. Treated with strong hydrochloric acid, the residue from an alcoholic extract gives a greenish-blue colour which disappears on keeping or heating. With strong sulphuric acid it gives a green colour, changing to brown and violet.[1]

In his annual report for the year 1936, the Chemical Examiner, Madras, describes the following scheme of examination which is used in his laboratory in suspected cases of *madar* juice poisoning :—

"The alcoholic extract of the viscus or other suspected material is divided into two portions (*a*) and (*b*)—

(A) Portion (*a*) is saponified with alcoholic potash and extracted with petroleum ether. The petroleum ether extract is evaporated to dryness, taken up with a little chloroform, treated with a slight excess of a solution of digitonin in rectified spirit, evaporated again to dryness and extracted with ordinary ether. This ether solution on evaporation gives a crystalline residue in the presence of *madar* juice. A little taken on a watch glass placed over a porcelain slab and treated with concentrated sulphuric acid gives a red colour. Addition of a few drops of chloroform and a few drops of acetic anhydride to this red colour changes it to a beautiful purple.

The alkaline alcoholic solution after extraction with petroleum ether as above is evaporated nearly to dryness, taken up with absolute alcohol and filtered. The filtrate on treatment with dry hydrochloric acid in excess and keeping for some time, shows on dilution with warm water a characteristic pleasant ester odour in the presence of *madar* juice.

1. *Chatterji, The Analyst, Nov.*, 1930.

(B) The other portion (*b*) is evaporated to dryness, taken up with water acidulated with acetic acid, filtered, treated with excess of lead acetate and again filtered. The filtrate is treated with excess of hydrogen sulphide, filtered free from lead and evaporated to dryness over a water bath. The residue is extracted with absolute alcohol and the alcoholic solution evaporated to dryness. A little of this extract on injection into a frog produces, in the presence of *madar* juice, convulsions ending in paralysis, death and bloating."

Medico-Legal Points.—The flowers, leaves, root-bark and milky juice of *madar* plants are used in Indian medicine. The powdered root-bark in 3 to 10-grain doses is used as an alterative and in 30 to 60-grain doses as an emetic in place of ipecacuanha. The tincture prepared from it is used in dysentery. The milky juice is used as a vesicant, as a depilatory and as a remedy for chronic skin affections.

Madar juice is used by tanners for removing hair from the skins. It also imparts a yellow colour to the skin and destroys the offensive odour of the fresh leather.

Madar juice is often used for procuring criminal abortion. It is either administered by the mouth or introduced into the uterus on an " abortion stick ". A case[1] is described where death occurred from the internal administration of *madar* juice with intent to procure criminal abortion. The juice was detected in the viscera. A case[2] is reported where two pieces of sticks with some brownish sticky substance adhering at their ends were removed from the uterus of a female, 32 years old, alleged to have died as a result of criminal abortion. The sticks were found to be of *madar*. A case[3] is also recorded where a woman introduced into her uterus pieces of cloth smeared with *madar* juice with a view to procuring abortion in the sixth or seventh month of pregnancy. As she could not bear the pain caused by the insertion she committed suicide by falling into a well.

Madar juice is occasionally used for purposes of suicide, infanticide and homicide. A case of infanticide is reported from Etawah in which *madar* juice was found in the organs of a new-born female child.[4] In the district of Manbhum a young woman was killed by the administration of *madar* juice and her body was hanged with a rope loosely tied round her neck. At the autopsy a faint ligature mark round the neck was found to be post-mortem, but, on the other hand, there were patches of inflammation in the mucous membrane of the stomach. The viscera, on analysis, showed the presence of *madar* juice.[5]

Smeared on a rag, *madar* juice is, sometimes, used as a cattle poison. It is either given with fodder or introduced into the rectum of the animal intended to be killed. A case occurred at Ghazipur where a she-goat after return from grazing died with symptoms of pain and convulsions.

1. *U. P. Chem. Examiner's Annual Report*, 1923.
2. *Bengal Chem. Examiner's Annual Report*, 1931, *p.* 8.
3. *Madras Chem. Examiner's Annual Report*, 1933, *p.* 7.
4. *U. P. Chem. Examiner's Annual Report*, 1929, *p.* 5; *see also Madras Chem. Exam. Annual Rep.*, 1936, *for a case of infanticide.*
5. *Bengal Chem. Exam.'s Annual Rep.*, 1936, *p.* 12.

A cloth ball found in the rectum of the animal and the viscera removed from the body revealed the presence of *madar* juice.[1]

PLUMBAGO ROSEA (*LAL CHITRA*) AND PLUMBAGO ZEYLANICA (*CHITRA*)

The roots of these plants, which belong to N. O. *Plumbagineœ* contain, as an active principle, *plumbagin*, a crystalline glucoside, which exists as fine glistening needles of a golden yellow colour. It is almost insoluble in cold water, moderately soluble in hot water, and freely soluble in ether, chloroform, alcohol, benzene, acetone, acetic acid, etc. Externally, plumbagin[2] is a powerful irritant and has a well-marked germicidal action on bacteria and unicellular organisms. In small doses it acts as a sudorific and stimulates the contraction of the muscular tissue of the heart, intestine and uterus. In large doses it causes death from respiratory failure. The minimum lethal dose has been found to be 0.5 mgm. per gramme of frogs, 0.1 mgm. per gramme of mice and 10 mgm. per kilogramme of rabbits.[3]

When applied externally, the roots produce painful irritation and blisters; while administered internally they act as narcotico-irritant poisons, producing pain in the stomach, thirst, vomiting and diarrhœa.

The crushed roots are largely used for procuring criminal abortion. They are either taken internally or, in the form of a paste, are applied to the os uteri, or painted on the "abortion sticks". Deaths have ensued from this use.

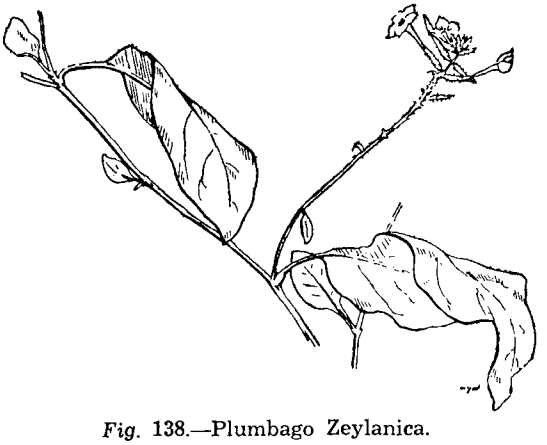


Fig. 138.—Plumbago Zeylanica.

A woman[4] was given a quack medicine containing plumbago root by her paramour to cause miscarriage. She died after having suffered from severe gastro-intestinal irritation with vomiting and purging for ten days. At the post-mortem examination severe congestions of the lungs, heart, liver, kidneys and the genital canal were found with the expulsion of the fœtus from the gravid uterus.

Plumbago roots are rarely used with homicidal intent. Chevers[5] mentions a case in which a woman mixed a small quantity of the powdered root (*Lal Chitra*) with milk and gave it to her husband. After two hours vomiting and purging occurred and in a short time the man

1. *U. P. Chem. Examiner's Annual Report*, 1929.
2. *Bhatia and Lal, Ind. Jour. of Med. Research, Jan.*, 1933, *p.* 777.
3. *Keien Ko, Japanese Jour. Med. Sciences*, 1931; *Chopra, Indigenous Drugs of India*, 1933, *p.* 365.
4. *Beng. Chem. Exam. Annual Report*, 1933, *p.* 13.
5. *Med. Juris., p.* 252.

died. On post-mortem examination the surface of the stomach was corrugated and covered with small inflamed patches, and the mucous membrane of the intestine was injected. Plumbagin was detected in the stomach contents, the vomited matter and the remnants of the food.

The root of plumbago rosea is also applied to the skin to simulate a bruise.

Walsh[1] records a case where one Jitan Ali Mir of Murshidabad reported to the police on the morning of August 22nd, 1898, that some eighteen or nineteen men armed with *lathies*, torches, lanterns, etc., had entered his house on the previous night, and carried away his valuables after having beaten and branded him with torches. Upon examination twenty-seven trifling injuries were found on several parts of his body which he could easily reach with his hands. These appeared to have been self-inflicted, and caused by the application of plumbago rosea to the skin. Of these injuries only one showed a slight abrasion due to destruction of the cuticle. The stains were of a reddish-brown colour and without raised or inflamed margins. The hair stood on them unsinged or uninjured. The man was found guilty of bringing a false charge of dacoity with self-inflicted injuries, and sentenced to four years' imprisonment.

Chemical Analysis.—The root of plumbago zeylanica is from $\frac{1}{4}$ to 2 or more inches thick. The dried bark is of a reddish-brown colour externally and brown and striated internally. The root of plumbago rosea is similar in structure, but much smaller.[2]

The following tests are employed for the detection of plumbagin in an organic mixture :—

1. Digest the mixture with alcohol and filter.
2. Evaporate the tincture to dryness.
3. Digest the residue with a small quantity of water rendered slightly alkaline with caustic potash solution.
4. Filter the solution obtained, acidulate with hydrochloric acid and shake with ether in a separating funnel.
5. Separate the ether. (Plumbagin passes into the ether).
6. Evaporate the ethereal extract.
7. Dissolve the residue (containing plumbagin) in caustic potash solution, when a bright crimson liquid is formed. On adding hydrochloric acid to this the colour is changed to yellow, and on standing for some time the liquid deposits yellow flocculi of plumbagin, which may be separated by shaking the acid liquid with ether. An alcoholic solution of plumbagin gives a crimson precipitate with a solution of basic lead acetate.

VERATRUM

There are three species of veratrum belonging to N. O. *Melanthaceæ*. These are Veratrum album (white hellebore), Veratrum viride (green hellebore) and Veratrum officinale (sabadilla). Several alkaloids have been obtained from these plants, the chief of which are veratrine, jervine, pseudo-jervine and cevadine. From among these, veratrine is a non-official preparation, having a dose of 1/64 to 1/16 grain to be given in pill form.

1. *Ind. Med. Gaz., Jan.*, 1900, *p.* 8.
2. *Dymmock, Pharmacographia Indica, Vol. II, p.* 331.

Veratrine.—Veratrine is a white, amorphous, inodorous powder, having an acrid, bitter taste, feebly soluble in water, but readily soluble in acids, alcohol and ether. Pure veratrine is crystalline in character. It is a powerful cardiac depressant.

Symptoms.—Burning pain in the throat, œsophagus and stomach; nausea; thirst; difficulty in swallowing; vomiting; diarrhœa, accompanied by tenesmus; tingling and itching of the skin which becomes reddened; salivation; sneezing and running of the eyes and nose; giddiness; feeble pulse; slow and gasping respirations; dilated pupils; cold and clammy skin. Death occurs from heart failure or from exhaustion due to incessant vomiting and prostration. Occasionally the tongue may be swollen, the throat may become sore, and spasms of the muscles and convulsions may occur.

Fatal Dose.—Uncertain. Three grains of veratrine have produced poisonous symptoms.[1] Eighteen grains of powdered white hellebore have caused death,[2] while half-an-ounce of the powder taken by mistake for cream of tartar has been recovered from.[3] Seventy minims of the fluid extract (U. S. P.) have proved fatal to a woman, aged 50 years, in 4 weeks.[4]

Fatal Period.—Uncertain. Death occurred in the case of an old peasant in 75 minutes after he had taken hellebore by mistake for liquorice powder.[5] Death has also occurred in 2 and 4 hours, but it may be delayed for several weeks.[6]

Treatment.—Administer emetics or wash out the stomach thoroughly with warm water. Tannic acid or vegetable astringents will precipitate the alkaloid. Give spirit ammonia aromatic and other stimulants, such as digitalis and strychnine. Keep the patient flat on the back, and start artificial respiration, if necessary. Opium may be given to check pain and diarrhœa.

Post-mortem Appearances.—These are not characteristic. The marks of acute inflammation may be found in the alimentary canal, and hyperæmia of the brain and its membranes may, sometimes, be present.

Tests.—Strong sulphuric acid gives a play of colours, viz., yellow, orange and lastly red. On heating, the colour becomes red at once, or the red colour is developed on adding bromine water.

Concentrated hydrochloric acid has no action in the cold, but on boiling the solution for a minute or two, it acquires a permanent bright red colour.

Weppen's Reaction.—One part of veratrine rubbed with six parts of cane sugar is moistened with a few drops of concentrated sulphuric acid. The colour developed is first yellow, dark green, then blue and lastly dirty violet.

Medico-Legal Points.—All parts of the veratrum plants are poisonous, but the chief source of poison is their root. It has been taken with a view to cause abortion. Powdered white hellebore has been mistaken for cream of tartar as mentioned in the case mentioned above.

Poisonous symptoms have been produced by the subcutaneous injection of veratrine, as also by the application of veratrine ointment.

COLCHICUM AUTUMNALE (COLCHICUM, MEADOW SAFFRON)

This plant belongs to N. O. *Liliaceœ*. All parts of the plant are poisonous, and are fatal to human beings as well as to cattle. The plant contains two active alkaloids, *colchicine*, $C_{22}H_{25}NO_6$ and *colchiceine*, $C_{21}H_{23}NO_6$. These appear to exert a similar pharmacological action, but the former is more poisonous than the latter. The corm (Colchici cormus) and the seeds (Colchici semina) are official in the

1. *Black, St. Geo. Hosp. Rep.*, 1870.
2. *Dixonmann, Forens. Med. and Toxic., Ed. VI, p.* 522.
3. *Giles, Lancet, Vol. II*, 1857, *p.* 9.
4. *Johnson, Buff. Med. and Surg. Jour., Nov.*, 1866, *p.* 133.
5. *Jour. Amer. Med. Assoc., May* 6, 1922, *p.* 1403.
6. *Witthaus, Med. Juris. and Toxic., Vol. IV, p.* 1075.

British Pharmacopœia, the dose of each being 2 to 5 grains. From the former are prepared an official preparation, *Extractum colchici siccum*, with the dose of $\frac{1}{4}$ to 1 grain and a non-official preparation, *Vinum colchici*, with the dose of 10 to 30 minims; from the latter are prepared official preparations, *Extractum colchici liquidum*, with the dose of 2 to 5 minims and *Tinctura colchici*, with the dose of 5 to 15 minims.

Colchicine is usually an amorphous, yellowish, bitter powder, but may be obtained in a yellow, crystalline form. It is soluble in water and alcohol. The non-official dose of colchicine is 1/120 grain.

Colchiceine occurs as lustrous white needles. It is slightly soluble in water, more readily in hot water, and dissolves easily in alcohol, chloroform and amyl alcohol, but is almost insoluble in ether and benzene.

Symptoms.—The symptoms usually supervene from one to three hours after swallowing a poisonous dose. There is burning pain in the mouth, throat, œsophagus and stomach. The mouth and throat are also dry and consequently swallowing is difficult. Intense thirst, nausea, vomiting and diarrhœa. The motions resemble very much choleraic stools except that they contain blood and shreds of mucous membrane, and that they are accompanied by tenesmus. A sensation of oppression is felt in the præcordial region with a feeling of vertigo. The patient is greatly prostrated and collapsed. The skin is cold; the face is pale or cyanosed. The pulse is small, irregular and imperceptible, and the respirations are slow and laboured. Towards death the pupils are dilated, twitchings of the muscles, spasms or convulsions occur, and the urine becomes scanty. The mind remains clear till death. In rare cases stupor may supervene before death.

Fatal Dose.—Uncertain; one grain of colchicine may be considered to be a fatal dose.[1] One-twentieth grain of colchicine injected hypodermically has caused death.[2] Three and-a-half drachms of vinum colchici taken in divided doses have proved fatal, though recovery has followed one ounce or more.[3] Casper[4] mentions a case in which four shoemakers died after each of them had swallowed about a wine glassful of tinctura colchici equivalent to two-fifths to half-a-grain of colchicine.

Fatal Period.—The average fatal period is about thirty hours. Two children ate colchicum bulbs. One of them died in a few minutes, and the other died in forty hours.[5] A man died in seven hours after he had taken by mistake $1\frac{1}{2}$ ounces of vinum colchici.[6] Death occurred on the fourth day in the case where $3\frac{1}{2}$ drachms of vinum colchici had been administered. A man of 43 years, who was suffering from gout, took about 1/20 grain of colchicine in divided doses within one hour, and died on the tenth day.[7]

Treatment.—Wash out the stomach with water containing tannic acid or tannin, which is a chemical antidote. Give mucilaginous drinks and hypodermic injections of morphine to allay pain and irritation. Administer glucose intravenously and strychnine and atropine hypodermically to combat collapse. Apply warmth and friction to the body. Resort to artificial respiration, if necessary.

Post-mortem Appearances.—Not characteristic. The blood may be found of a dark cherry-red colour, with the consistency of treacle. Inflammation of the mucous membrane of the stomach and intestines is usually found. The kidneys may be markedly congested.

Chemical Analysis.—The alkaloid is extracted by Stas' process from the acid solution by chloroform.

---

1. *Taylor, Princ. and Pract. of Med. Juris., Vol. II, Ed. IX, p.* 785.
2. *Klin.-Ther. Wchnschr.*, 1900, *VII, p.* 407; *Witthaus, Med. Juris. and Toxic., Vol. IV, p.* 911.
3. *Taylor, On Poisons, Ed. III, pp.* 510, 511.
4. *Forens. Med., Vol. II, E. T., pp.* 60 *and* 108, 109, 110.
5. *Rev. de Droit penal et Criminal, Mai*, 1926; *Sydney Smith, Forens. Med., Ed. VI, p.* 539.
6. *Taylor, On Poisons, Ed. III, p.* 510.
7. *Courtois-Suffit et Trastour., Bull. et mem. Soc. Med. des hop. de Paris*, 1903, *XX, p.* 254; *Witthaus, Manual of Toxic., Ed. II, p.* 911.

Tests.—1. Strong sulphuric acid forms with colchicine a bright yellow solution which, on adding nitric acid, changes to green, blue, violet and lastly to pale yellow. A brick red colour is produced, if now a strong solution of caustic potash be added.

2. Ferric chloride solution gives a dark green colour to an aqueous solution of colchicine. If hydrochloric acid solution of the alkaloid be boiled with a solution of ferric chloride, it is coloured greenish-black, and chloroform agitated with this is coloured garnet-red or brown.[1]

3. Tannic acid gives a white precipitate, readily soluble in acetic acid, alcohol and alkaline carbonates.

4. Chlorine water gives a yellow precipitate, which dissolves into an orange coloured solution, if ammonia is added to it.

Medico-Legal Points.—Colchicum and its preparations are used as remedies for gout. Hence accidental cases of poisoning, sometimes, occur from an overdose of their preparations. Poisonous cases have also been produced by the administration of Blair's gout pills and other proprietary remedies containing colchicum.[2]

Accidental cases of poisoning have also occurred from taking milk of the goats fed on the leaves of the colchicum plant.[3]

A woman[4] prepared a bitter alcoholic drink from herbs which contained 0.012 to 0.06 per cent. colchicine and gave it to her husband. He was taken ill with alarming general weakness, colic and diarrhœa. It took him three weeks to recover.

Cases of homicidal poisoning have occasionally occurred. In September, 1863, Catherine Wilson was convicted of the murder of a Mrs. Soames by administering colchicum. From the evidence at the trial it appeared that the accused had similarly destroyed three other persons.[5]

Colchicine has been detected in the bodies of animals exhumed four and-a-half to five and-a-half months after death.[6]

DELPHINIUM STAPHISAGRIA (STAVE SACRE)

This is a plant belonging to N. O. *Ranunculaceœ*. Its seeds (staphisagriæ semina) are non-official, and are used in the form of an ointment for destroying pediculi. They contain alkaloids, the chief of which are delphinine and staphisagrine, and also a fixed oil. They act both as an irritant and as a depressant poison. Poisoning by stave sacre seeds is very rare. A case[7] is recorded in which a man took two teaspoonfuls of a powder, two-thirds of which consisted of the seeds. The symptoms which he suffered from were a distended, painful abdomen, dilated pupils, slow, feeble pulse (35-40 p.m.), difficult and laborious breathing and severe collapse. Recovery occurred in a few hours.

HELLEBORUS NIGER (BLACK HELLEBORE, *KALI KATUKI*)

This plant belongs to N. O. *Ranunculaceœ*. All its parts are poisonous. The root is employed as a hydragogue, cathartic, emmenagogue and anthelmintic. It is given in five to ten-grain doses, but in larger doses it acts as an irritant-narcotic poison. The active principles are two glucosides, helleborin, and helleborein.

1. *Witthaus, Manual of Toxic., Ed. II, p.* 919.
2. *Lancet, Vol. I,* 1881, *p.* 368 ; *Brit. Med. Jour., Vol. II,* 1903, *p.* 1272.
3. *Ratti, Ph. J. and Tr.,* 1875, 3*s., VI, p.* 47.
4. *Compilation of Cases of Poisoning by H. Fuehrer, F. Hesse and F. Starkenstein, Vol. I, Nos.* 11 *and* 12 ; *Vol. II, No.* 1, 1930, *to* 1931 ; *Ars Medici, Jan.* 1932, *p.* 13.
5. *Taylor, On Poisons, Ed. III, p.* 512.
6. *Dixonmann, Forensic Med. and Toxic., Ed. VI, p.* 521 ; *Obolonski, vrtljschr. f. Ger. Med.,* 1888 ; *Ogier, Ann. d'Hyg.,* 1886.
7. *Friederich's Blatter, f. ger. Med.,* 1868 ; *Dixonmann, Forensic Med. and Toxic., Ed. VI, p.* 527.

Symptoms.—These are vomiting, pain in the abdomen, diarrhœa, profuse perspiration, convulsions, insensibility and death.

Fatal Dose.—Nearly 31 grains of an aqueous extract of the root have proved fatal to a man, 50 years old, within 8 hours.[1] A decoction of the root has caused death in less than 2 hours.[2] Recovery has occurred from a table-spoonful of the finely powdered root taken by mistake for rhubarb.[3]

Treatment.—Evacuate the stomach, administer stimulants and give morphine to allay pain and check diarrhœa.

Post-mortem Appearances.—Not characteristic. Inflammation of the mucous membrane of the stomach.

CYTISUS LABURNUM (*LABURNUM*)

This belongs to N. O. *Leguminoseœ*. It grows wild in gardens, shrubberies and woods in England. All parts of the plant, *viz.*, the bark, wood, seeds and flowers, produce toxic effects, when taken internally. The plant has a most nauseous and disagreeable odour and taste. The active principle is an alkaloid, *cytisine*, $C_{11}H_{14}N_2O$, which is the chief ingredient contained in Australian or Persian insect powder.

Symptoms.—Pain in the stomach, thirst, nausea, vomiting, purging, giddiness, collapse, drowsiness and coma. Occasionally convulsions and delirium have preceded death. The pupils are usually dilated, but may be found contracted. Death results from paralysis of the respiratory and vaso-motor centres.

G. F. Sydenham[4] reports two cases of poisoning by the seeds of laburnum on October 22, 1917, in children, aged respectively 3 and 5. They had been very sick and vomited up both husk and berries. They were unconscious with very dilated pupils, which did not react to light. The temperature was not raised, and the pulse was very feeble. The heart sounds were feeble. There were no convulsions and no diarrhœa. Both the children were comparatively well next morning.

Fatal Dose.—Not known. Three or four seeds are enough to produce toxic symptoms.[5]

Fatal Period.—Death has occurred from one to thirty hours. In one case it took place on the seventh day.[6]

Treatment.—Emetics, or wash out the stomach with warm water; stimulants and artificial respiration, if necessary.

Post-mortem Appearances.—Not characteristic. The brain and its membranes may be congested. The stomach and intestines may be inflamed.

Tests.—1. Strong sulphuric acid dissolves cytisine without effecting any change of colour, but on heating, the solution acquires a yellow colour.

2. A mixture of sulphuric and nitric acids produces a yellow colour.

3. Ferric chloride solution gives a blood-red colour, which disappears on adding hydrogen peroxide. On further heating, it assumes a blue colour.

Medico-Legal Points.—Accidental cases of poisoning have occurred among children owing to their having eaten the bark in mistake for liquorice, as well as the seeds which are sweet in taste.

Cytisine is eliminated largely in the urine, and to some extent in the fæces, as well as in the saliva.

---

1. *Morgagni, Blyth, Poisons, Ed. V, p.* 455.
2. *Guy and Ferrier, Forensic Med., Ed. VI, p.* 579.
3. *Taylor, On Poisons, Ed. III, p.* 514.
4. *Brit. Med. Jour., Nov.* 17, 1917, *p.* 676.
5. *Taylor, Princ. and Pract. of Med. Juris., Vol. II, Ed. IX, p.* 796.
6. *Brit. Med. Jour.*, 1875.

TAXUS BACCATA (*YEW*)

This is a large, evergreen tree of temperate Himalayas and belongs to N. O. *Coniferœ*. The poisonous symptoms are due to an alkaloid, *taxine*, $C_{37}H_{52}O_{16}N$, contained in the leaves and seeds of its berries. Taxine is an amorphous powder, hardly soluble in water, but dissolves in alcohol, ether and chloroform. The medicinal dose is 1/100 to 1/60 grain. The leaves are sold as *birmi* and *talispatra* in Indian towns.

Symptoms.—Giddiness, dilated pupils, vomiting, purging, pain in the abdomen, small irregular pulse, slow laboured breathing, collapse, convulsions, insensibility, delirium or coma.

Fatal Dose.—Unknown. One tea-spoonful of the leaves,[1] and four berries[2] have respectively caused death.

Fatal Period.—A girl, about five years old, died in four hours after she had eaten the berries, and a boy, aged four years, died on the nineteenth day after eating the berries.[3] A girl of nineteen years drank a tumblerful of a strong decoction of the leaves on four successive mornings to bring on the menses. She suffered from severe vomiting and delirium, and died eight hours after taking the last dose.[4]

Treatment.—Empty the stomach, keep the body warm, and use stimulants and artificial respiration, if necessary.

Post-mortem Appearances.—Inflammation of the mucous membrane of the stomach which may contain fragments of the seeds or leaves of the plant. A man, aged 26 years, a voluntary patient for a nervous break down in Maudsley hospital, who was cultivating the habit of chewing yew leaves to supersede the habit of smoking tobacco, died. At the necropsy a considerable quantity of the leaves was found in the alimentary canal.[5]

Medico-Legal Points.—Cases of poisoning occur accidentally among children or even among grown-up persons on account of their eating in mistake the leaves or fruits of the plant, and among women, who use an infusion of the leaves as an abortifacient owing to its emmenagogic properties.

The leaves and berries are also poisonous to cattle and cause death in a few hours, without producing vomiting and purging in some cases.

JUNIPERUS SABINUS (*SAVIN*)

This shrub belongs to N. O. *Coniferœ*, and yields a round purple fruit about the size of a currant. It has a peculiar strong odour, and an acrid taste. Its leaves and tops contain, as an active principle, an essential oil, oil of savin, which acts as a vesicant, when applied externally, and acts as an irritant, when administered by the mouth. The oil and infusion of the leaves have been often used as abortifacients, but they have no direct ecbolic action on the uterus. They cause abortion by producing congestion of the pelvic organs due to their irritating action, and consequently the death of the woman.

Symptoms.—Violent pain in the throat and abdomen; vomiting; purging, though rarely; hæmaturia; strangury; laboured and stertorous respiration; unconsciousness rapidly ending in death. Salivation occurs occasionally.

Fatal Dose.—The medicinal dose of oil of savin is 1 minim and that of the leaves is 7½ grains, the maximum being 15 grains during twenty-four hours,[6] but the fatal dose of the oil or the leaves is not known.

1. *Taylor, On Poisons, Ed. III, p.* 784.
2. *Prov. Jour., Dec.* 27, 1848, *p.* 708.
3. *Taylor,* Loc. Cit., *p.* 782.
4. *Ibid., p.* 783; *Lancet, Vol. II,* 1870, *p.* 471.
5. *Jour. Amer. Med. Assoc., Sep.* 17, 1932, *p.* 1005.
6. *Martindale, The Extra Pharmacopœia, Ed. XIX, p.* 883.

Fatal Period.—Death has occurred in 12[1] and 26[2] hours, and has been delayed for 5 days.[3]

Treatment.—Eliminate the stomach contents ; give heart stimulants, or administer chloral hydrate or morphine when necessary.

Post-mortem Appearances.—Acute inflammation of the œsophagus, stomach, intestines and kidneys. There may, sometimes, be patches of extravasation in the gastric mucous membrane and fragments of the leaves in the stomach contents.

Detection.—Perchloride of iron imparts a deep green colour to watery solutions of savin.

GAMBOGE (*REVENCHINO SHERO*)

This is a gum-resin obtained from Garcinia morella and Garcinia hanburii belonging to N. O. *Guttiferœ*. It is not an official drug of the British pharmacopœia, but is used as a drastic purgative, the dose being ½ to 2 grains. It is largely used by quacks, and forms one of the chief ingredients of several quack vegetable pills, which often produce the symptoms of irritant poisoning. It has occasionally proved fatal, when used as a purgative or as an abortifacient.

Symptoms.—Severe vomiting and purging, the dejected matter being of a deep yellow colour ; abdominal pain and tenesmus ; great weakness ; collapse and death.

Fatal Dose.—One drachm[4] has proved fatal. A case[5] is, however, recorded in which a female, 19 years old, who took about 3 drachms of pipe gamboge with a view to commit suicide, recovered slowly under proper treatment.

Treatment.—Wash out the stomach, and administer demulcent drinks and opiates. Combat collapse by giving cardiac and respiratory stimulants.

Post-mortem Appearances.—Signs of irritation of the alimentary tract and congestion of the liver, spleen and kidneys.

JALAP

This is a powder prepared from the dried tubercles of Ipomœa purga or Exogonium purga belonging to N. O. *Convolvulaceœ*, and is used as a hydragogue purgative in 5 to 20-grain doses. It is also contained in *Pulvis jalapœ compositus* (dose, 10 to 60 grains). The purgative properties are due to a resin contained in its root. The resin consists mainly of two anhydride glucosides, *jalapin* and *convolvulin* (*jalapurgin*), and is a non-official preparation, having a dose of 2 to 5 grains. In larger doses it acts as an irritant poison. It is, sometimes, used as an abortifacient, and produces toxic effects.

SCAMMONY

This is a gum resin obtained from the root of Convolvulus scammonia belonging to N. O. *Convolvulaceœ*. It is easily pulverized, and forms into an emulsion when mixed with emulsin. The resin is used as a drastic purgative in ½ to 3-grain doses. It contains an active principle, *jalapin*. In large doses it acts as a strong gastro-intestinal irritant, and may cause death, if administered to weak, debilitated persons.

KALADANA SEEDS (*PHARBITIS SEEDS*)

These are the seeds of Ipomœa hederacea cultivated in several places of India, and belonging to N. O. *Convolvulaceœ*. Their active principle is a pale yellowish resin, *pharbitisin*, corresponding in chemical action to jalapin, to which its irritant properties are chiefly due.

1. *Newth, Lancet, June* 14, 1845, *p.* 677.
2. *Blyth, Poisons, their Effects, and Detection, Ed. V, p.* 485.
3. *Med. Gazette*, 1845, *Vol.* 36, *p.* 646 ; *Taylor, On Poisons, Ed. III, pp.* 496, 497.
4. *Traill's "Outlines", p.* 150 ; *Taylor, On Poisons, Ed. III, p.* 522.
5. *Jamsetji, Bom. Med. and Phys. Trans. No.* 2, 1853-54, *p.* 340 ; *Chevers, Med. Juris., p.* 283.

The seeds are in the form of a segment of a sphere, about 5 mm. long and wide, and are nearly black except at the hilum where they are brown and hairy. The resin occurs in brownish opaque fragments, being translucent at the edges. The seeds and the resin were official in the British Pharmacopœia of 1914, having the doses of 30 to 45 grains and 2 to 8 grains respectively, but they are not included in the revised Pharmacopœia of 1932. In larger doses they produce symptoms of irritant poisoning.

The seeds are also contained in the non-official preparations of *Pulvis kaladanæ compositus* (dose, 10 to 60 grains) and *Tinctura kaladanæ* (dose, 30 to 60 minims).

IPOMŒA TURPETHUM (*NISHOTAR, PITHORI*)

This plant belongs to N. O. *Convolvulaceœ*, and is called an Indian jalap or *white turpeth*. Both the root and the bark are used as cathartic and laxative. The non-official dose of the root is 5 to 20 grains, but it can be given from ½ to 1½ drachms. Larger doses produce irritant symptoms. Another variety, known as *black turpeth*, is more drastic in its action and is, therefore, not used in medicine.

CUSCUTA REFLEXA (*AKASBEL*)

This is a parasitic, climbing plant, growing wild on certain hedges, and belonging to N. O. *Convolvulaceœ*. Its decoction is used as an abortifacient by "Dais" (utrained midwives), chiefly in the Punjab.[1] It is said that a decoction of 180 grains of the plant produces abortion, though at the same time it causes nausea, vomiting and depression.

EUPHORBIUM

This is an acrid, milky juice exuded from the stems of various euphorbious plants belonging to N. O. *Euphorbiaceœ*; the chief of these are Euphorbia antiquorum (*tidhara, sehund*), Euphorbia nerifolia (*thohar*) and Euphorbia tirucalli (milk hedge or Indian tree-sponge).

The juice produces vesication, when applied to the skin, and inflammation involving eye-sight, when dropped into the eyes. Internally, it acts as an irritant, causing vomiting, diarrhœa, convulsions and coma. It is used for procuring criminal abortion, but rarely for homicidal purposes. A Mahomedan girl, aged sixteen years, was convicted for administering to her husband in his food arsenic and sugar of lead mixed with the juice of Euphorbia antiquorum.[2] A tea-spoonful of the juice of Euphorbia officinarum or resinifera proved fatal to an adult in three days. On post-mortem examination gangrenous spots were observed in the stomach, and the spleen was found in a "rotten" condition.[3]

JATROPHA CURCAS (PHYSIC NUT, *JANGLI ARANDI*)

This evergreen plant belongs to N. O. *Euphorbiaceœ*. Its seeds contain a pale yellow, acrid oil, which has almost the same action as croton oil. Applied externally, it causes irritation, and has a purgative action, when administered internally. Twelve to fifteen drops of the oil produce alarming symptoms. Four seeds act as a violent cathartic and a few grains of the cake left after the expression of the oil may produce severe vomiting and purging.[4]

The active principle of the oil is jatrophic acid, but the seeds owe their toxic properties to a toxalbumin, called *curcin*, and analogous to ricin.

Symptoms.—Nausea, vomiting, diarrhœa, pain in the abdomen and general depression. A case[5] is recorded in which a young Englishman ate 15 or 20 *physic nuts*. In an hour and-a-half the patient complained of a burning sensation in the

1. *Dulipsigh, Ind. Med. Gaz., Jan.*, 1885.
2. *Bellasis's Bomb. S. F. A. Reports, p.* 37; *Chevers, Med. Juris., p.* 269.
3. *Jour. of Sci., Vol.* 3, *p.* 58.
4. *Taylor, Princ. and Pract. of Med. Juris., Vol. II, Ed. IX, p.* 824.
5. *Madras Med. Quar. Jour. of Med. Sci., July*, 1861, *p.* 37.

throat and stomach, followed by vomiting and diarrhœa. In another half-an-hour diarrhœa stopped, but convulsive twitchings of the muscles, especially of the back, were present together with deafness and impairment of sight. The skin was cold and clammy, the pulse was small, feeble and thready, and the features were sunken and contracted. There was also loss of memory. The patient was treated with hot brandy and water and anodyne frictions. He slowly recovered in three days.

In his annual report for 1927, the Bombay Chemical Analyser reports a case where three children were taken to the J. J. Hospital by their father who said that they had eaten some Jatropha seeds and thereafter been taken ill with vomiting and diarrhœa. One of the boys had eaten about six seeds, and he had ten attacks of vomiting and five or six offensive motions. The boys had rather rapid and feeble pulses and slightly dilated pupils. The respirations were hurried, and the surface of the body was cold particularly in those who had the severest symptoms.

No chemical tests are known for this plant.

JATROPHA MULTIPHIDA

This plant belongs to N. O. *Euphorbiaceœ*. Its fruit is known as the French physic nut. Three nuts[1] have produced violent vomiting, purging, intense burning pain in stomach and great prostration. Recovery occurred after the use of lime juice and stimulants.

JATROPHA URENS

This plant also belongs to N. O. *Euphorbiaceœ*. Its leaves are covered with hairs, which, if rubbed against the skin, produce irritation, inflammation and severe prostration.

ALOES (*ELWA* OR *ELIO*)

This is the inspissated juice derived from the leaves of Aloe Vulgaris and other species belonging to N. O. *Liliaceœ*. Its active principle is aloin. Aloes and aloin are both used as purgatives in doses of 2 to 5 grains and $\frac{1}{4}$ to 1 grain respectively. Aloes is also contained in the official preparations of *Pilula aloes*, *Pilula aloes et asafetidæ* and *Pilula aloes et ferri*. The dose of these three preparations is the same, *viz.*, 4 to 8 grains.

In large doses aloes acts as an irritant poison, 2 drachms having proved fatal to a woman in 12 hours.[2] The symptoms are chiefly colic, abdominal pains, diarrhœa with tenesmus and motions containing blood, great prostration and death. The chief post-mortem appearance is inflammation of the stomach and small intestine to some extent.

Aloes increases the menstrual flow reflexly by stimulating the uterus. It is, therefore, used as an abortifacient. Aloes is a leading ingredient in most quack aperient pills, and is one of the chief ingredients of Morison's pills, the other ingredient being colocynth. *Hierapicra* (holy bitter), a compound of four parts of aloes and one part of canella bark is, sometimes, employed for procuring abortion.

URGINEA SCILLA

This plant belongs to N. O. *Liliaceœ*. Its bulbous root cut into slices and dried is a pharmacopœial preparation, known as squill, and is given internally as a diuretic and expectorant in 1 to 3-grain doses. The other official preparations containing squill are *Acetum scillœ*, dose : 10 to 30 minims ; *Oxymel scillœ*, dose : 30 to 60 minims ; *Syrupus scillœ*, dose : 30 to 60 minims ; and *Tinctura scillœ*, dose : 5 to 30 minims.

In large doses squill or any of its preparations acts as a powerful gastro-intestinal irritant, and produces vomiting, purging, strangury, bloody urine and

1. *Chevers, Med. Juris., p.* 275.
2. *Taylor, On Poisons, Ed. III, p.* 524.

cardiac depression. Twenty-four grains of the powdered root have proved fatal.[1] Seventy-five grains of its alcoholic extract have also caused death in two days.[2]

The treatment consists in the administration of emetics or washing out of the stomach. The patient should be kept in a recumbent posture and should be treated symptomatically.

The post-mortem appearances may be inflammation of the alimentary canal and of the kidneys.

Squill owes its toxic properties to *scillitoxin* and *scillaren,* both glucosides, which are readily broken down by the digestive juices.

An Indian variety, called urginea Indica (*Jangli piaz*) is used as a substitute for squill.

GLORIOSA SUPERBA (*CARIHARI, KHADIYANAG*)

This belongs to N. O. *Leguminosœ.* It is an elegant climbing hedge plant growing in Bengal and in low jungles throughout India, and flowers about the end of the rains. Its root contains an active bitter principle, *superbine,* a glucoside. It is used as a tonic, stomachic and antiperiodic in 5 to 10-grain doses. Up to 12 grains it is not poisonous, but beyond that it has possibly the same poisonous action as squill. It is said to be used in India as an adulterant of aconite.

Symptoms.—Nausea, violent vomiting, purging, spasms, convulsions, profuse sweating and collapse with heart-failure. Chevers[3] mentions two fatal cases of poisoning by this plant. In one case a woman committed suicide by swallowing a *masha* of the fresh root, stalk and leaves. In the other about 2 *tolas* of the root were given with salt and red lead to a woman. A third case is recorded in which a female, 18 years old, took a quantity of the powdered root and died in 4 hours. The post-mortem appearances were inflammation of the gastric mucous membrane and congestion of the liver, kidneys, lungs and brain.[4]

A case[5] is recorded of a man, aged 45 years, who ate the root of gloriosa superba in order to commit suicide, suffered from violent gastro-intestinal symptoms and died within twelve hours.

Chemical Analysis.—On extracting the roots of gloriosa superba with rectified spirit under a soxhlet and adding ether to the alcoholic solution a whitish precipitate is obtained which, on separation and injection into frogs, proves fatal to them.[6] The precipitate is freely soluble in water but insoluble in absolute alcohol, ether, chloroform or amyl alcohol. Yellow oleander seeds, when treated in the same manner, yield a similar poisoncus precipitate which, however, is soluble in absolute alcohol as well as in amyl alcohol. Further, the precipitate from yellow oleander seeds yields a blue colour on boiling with dilute hydrochloric acid, but the precipitate from gloriosa superba does not yield any colour with hydrochloric acid.

ARUM MACULATUM (LORDS AND LADIES, CUCKOO-PINT, WAKE-ROBIN, THE PARSON IN THE PULPIT)

This plant belongs to N. O. *Araceœ, Sub Order, Aroideœ.* Its root, if eaten raw, produces irritant symptoms in addition to swelling of the tongue, salivation and dilatation of the pupils, but it loses its poisonous properties by soaking it in water, and then baking it. It is thus used as an article of food, constituting the Portland sago.

Cases of accidental poisoning have occurred among children from eating the leaves or berries which are bright red and succulent. Three children ate some leaves

1. *Lyon, Med. Juris., Ed. IX, p.* 569.
2. *J. de Chim. Med.,* 1842, *p.* 651.
3. *Med. Juris., pp.* 284-5.
4. *Ind. Med. Gaz.,* 1872, *p.* 153.
5. *Madras Chem. Exam.'s Ann. Rept.,* 1934, *p.* 6.
6. *Ibid., p.* 10.

of the plant. Two of them died in 12 and 16 days respectively, and the third recovered.[1] The root has caused death in 9 hours.[2]

The other arum varieties are Amorphophallus Campannalatus (*Suran*) and Arum Colocasia (*Kachu*).

CRINUM DEFLEXUM OR ASIATICUM (*SUKHADARSHAN, NAGDOWN*)

This is a large plant belonging to N. O. *Amaryllideœ* and much cultivated in Indian gardens. Its root and leaves are used as substitutes for ipecacuanha, and produce vesication if applied externally. They cause irritant symptoms if administered internally in large doses.

ARGEMONE MEXICANA (*DARURI, SIALKANTA*)

This is an American herbacious annual belonging to N. O. *Papaveraceœ*, but now growing wild in the cold season all over India. It has multi-lobed thorny leaves and produces bright yellow flowers. The extract of the whole plant contains *berberine* and *protopine*. The seeds of the plant are spherical and resemble black-mustard seeds. The oil expressed from the seeds is known as argemone oil (*katkar* oil). It contains about 40 per cent free glycerides of fatty acids. The oil is a valuable remedy as an aperient in 30 to 60 minims,[3] and is also used for the treatment of skin diseases, such as scabies and eczema. In larger doses it acts as an irritant giving rise to vomiting and diarrhœa. Sarkar[4] reports an outbreak of poisoning among the members of three families from the use of mustard oil adulterated with this oil. The earliest symptoms were excessive spitting and vomiting, which were followed by disturbance of the bowels and gradual œdema of the feet and legs. Twenty-four days later the symptoms complained of were intense pain all over the body, fever up to 101° F., profuse diarrhœa in some cases and constipation in others, and œdema of the lower extremities. Recovery was very slow. In the case of two girls who smeared the oil over their heads, the scalp was found to be inflamed with a burning sensation on the head on the same night, and later there was marked falling off of the hair.

Detection.—The oil is pale yellow, clear and limpid, and mixed with an equal volume of nitric acid, assumes a crimson colour.

COCCULUS SUBEROSUS (*KAKMARI, KAKPHAL*)

This belongs to N. O. *Menispermaceœ*, and is also known as Anamirta Cocculus. It grows in Southern and Eastern parts of India and in Burma. The berry has a dark brown wrinkled surface, and constitutes the Cocculus indicus or Levant nut of commerce. On section the berry contains a mushroom-shaped body which consists of a bitter seed on the top of a short stalk. The berry contains a poisonous, non-alkaloidal principle, *picrotoxin*, which exists as colourless shining prismatic crystals, and has an intensely bitter taste. It is soluble with difficulty in cold water, but dissolves freely in hot water, alcohol or chloroform. The shell or husk of the berry does not contain picrotoxin, but contains a non-poisonous principle, called *menispermine*. It is, therefore, possible that an entire berry, when swallowed, may pass through the body without causing poisonous symptoms.

Symptoms.—Bitter taste in the mouth, burning pain in the œsophagus and stomach, nausea, vomiting, diarrhœa, profuse sweating, intoxication, lethargic stupor and unconsciousness. The respirations are at first increased and afterwards become slow and laboured. The pulse is usually weak. The characteristic features in most cases are tetanic spasms with complete relaxation of the muscles during the intervals. The pupils are contracted during spasms and dilated during the interval of relaxation. Death occurs rapidly from failure of respiration or slowly from gastro-intestinal symptoms.

1. *Guy and Ferrier, Forens. Med., Ed. VI, p.* 586.
2. *Brit. Med. Jour., June* 23, 1861.
3. *Chopra, Indigenous Drugs of India,* 1933, *p.* 287.
4. *Ind. Med. Gaz., Feb.,* 1926, *p.* 62.

Fatal Dose.—Uncertain. Two berries, one of which was soon vomited, proved fatal to a girl, aged 9 years.[1] Two scruples of the berry caused the death of a boy, 12 years old, on the 19th day.[2] Recovery has taken place after swallowing a handful of the crushed berries.[3]

The medicinal dose of picrotoxin is 1/100 to 1/25 grain. The fatal dose is not definitely known, but from experiments on animals it is inferred that a dose of 2 to 4 grains would be dangerous for an adult person.[4]

Fatal Period.—The shortest fatal period is 30 minutes.[5] Death occurred in 3 hours in a case where the tincture intended as a remedy for vermin was taken internally.[6] A child, aged six years, died in about 6 hours after an alcoholic infusion of the berries was applied to the head.[7]

Treatment.—Wash out the stomach or administer emetics. Give chloroform inhalation or chloral hydrate by the mouth if the tetanic spasms are very severe. Artificial respiration, if neccesary.

Post-mortem Appearances.—Not characteristic. Congestion of the stomach, lungs and brain. There may be peritonitis in cases of delayed death.

Chemical Analysis.—Picrotoxin may be extracted from acidulated organic mixtures by ether. It is dissolved by strong sulphuric acid producing a yellow colour, which changes to violet on the addition of a trace of potassium bichromate and becomes brown on further adding the same. Picrotoxin may be mistaken for sugar as it reduces Fehling's solution. If picrotoxin is mixed with about three times the quantity of potassium nitrate, and the mixture is moistened with the smallest quantity of concentrated sulphuric acid, and then a strong solution of sodium or potassium hydroxide is added in excess, an intense red colour will appear.

Medico-Legal Points.—The powdered berries are used for poisoning fish in rivers. For this purpose the berries are mixed with flour and a little tobacco, made into a dough, and small pellets are thrown into water. They are also used for poisoning cattle. A decoction or extract of the berries is, sometimes, used to facilitate theft or rape, and to adulterate country liquor to increase its intoxicating effect. An ointment of picrotoxin is employed to destroy pediculi but care must be taken in its application, as it is absorbed through the abraded skin.

Accidental cases of poisoning have occurred from eating the berries, from drinking liquor impregnated with them and from application of the alcoholic infusion to the head.[8]

MORINGA PTERYGOSPERMA (*SHAJNA, SHARAGAVA*)

This tree belongs to N. O. *Moringeœ*, and grows wild in the Sub-Himalayan range. The fresh root of this tree closely resembles the common horse-radish in taste, smell and general appearances. The pods are used as a vegetable, and are considered preventive against intestinal worms. The root acts as a vesicant, if applied externally. The bark contains small quantities of an essential oil, having a very pungent odour. It also contains 0.105 per cent of alkaloidal bases, which closely resemble ephidrine in action. One of them is crystalline and is less active than the other which is amorphous. Both have a stimulant action on the heart, constrict the blood vessels and produce a marked and persistent rise of blood pressure. They relax the bronchioles, inhibit the tone and movements of the intestines and produce

1. *Amer. Jour. Med. Science,* 1851, *n. s., XXI, p.* 527 ; *Wharton and Stille, Med. Juris., Ed. V, Vol. II, p.* 531.
2. *Scholler, Canstatt, Jahresbericht.,* 1844, *Vol. V, p.* 291.
3. *Dutzmann, Wiener Med. Presse,* 1869 ; *Dixonmann, Forensic Med. and Toxic., Ed. VI, p.* 493.
4. *Blyth, Poisons, their Effects and Detection, Ed. V, p.* 478.
5. *Shaw, Med. News,* 1891, *Vol. LIX, p.* 38.
6. *Sozinsky, Ibid.,* 1883, *Vol. XLIII, p.* 485.
7. *Thompson, Med. Examiner, Phill.,* 1852, *n. s., VIII, p.* 227.
8. *Ibid., p.* 227.

contraction of the virgin, as well as the pregnant, uteri of guineapigs and rabbits.[1] The powdered bark is largely used as an abortifacient in Bengal, and has produced fatal results.

RUTA GRAVEOLENS (*SATAP*)

This plant belongs to N. O. *Rutaceœ*, and is commonly cultivated in Indian gardens. It yields, on distillation, a volatile oil, which is acrid bitter in taste, and is a valuable diuretic and emmenagogue in 2 to 5-minim doses. In large doses it acts as an abortifacient, and produces irritant symptoms.

SAPINDAS TRIFOLIATUS (*RHITHA*)

This tree belongs to N. O. *Sapindaceœ*, is common in Southern India and is cultivated in Bengal. Its fruits are known as soap-nuts, and are largely used for washing silk, etc. They contain a glucoside, *saponin*, a white amorphous powder, which dissolves in water, forming a froth like soap. It is insoluble in cold alcohol or ether, and strikes a red colour with sulphuric acid.

According to Blyth[2] 1½ to 3 grains of saponin administered by the mouth increases mucous secretion and causes nausea. Forty grains administered subcutaneously to an adult would endanger life, the symptoms being great muscular prostration, weakness of the heart's action and probably diarrhœa. The post-mortem appearances would probably be those of an irritant or inflammatory action on the gastric and intestinal mucous membranes.

TERMINALIA BELLERICA (BELLERIC MYROBALANS, *BAHERA*)

This tree belongs to N. O. *Combretaceœ*, and grows in Indian forests. Its fruits are oval and somewhat five angled. When fresh, they are of the size of a nutmeg, fleshy and covered with a grey silky down. When dry, they are of the size of a gall nut, dry brown in colour and astringent in taste. The stones are smooth and hard, and contain white kernels. These yield an oil which is used as a dressing for the hair. The dried ripe fruits are astringent and used in Indian medicine. The powder of their pericarp enters into the composition of *triphala*, the other two constituents being the chebulic and emblic myrobalans.

Accidental cases of poisoning by the belleric fruits have occurred, the symptoms being nausea, vomiting, headache, insensibility, normal pupils, quick, feeble pulse, slow, laboured respirations, trismus, convulsions and death. The post-mortem examination of the body of a girl, 5 years old, who died after eating some kernels, showed congestion of the stomach, which contained a greenish-black-fluid.[3] Chevers[4] mentions a case where three boys, from five to nine years of age, ate some dry nuts and suffered from poisonous symptoms, but recovered. One of them who had eaten the largest quantity of kernels, between 20 and 30, did not suffer from any symptoms for at least twelve hours. A Hindu boy, aged 4 years, died in about 24 hours after he had taken a *Bahera* fruit.[5] A boy of five years, who ate fresh kernels of *Bahera* fruits, died within forty-eight hours.[6] Windsor[7] reports a case where a family consisting of a man, his wife and four children partook of a *pilau* prepared with some kernels. In about an hour they were seized with nausea, vomiting and giddiness. After forty-eight hours' illness the man, his wife and two children recovered, but still felt dazed and giddy. The two younger children, aged 2 and 3½ years respectively, died within forty-eight hours, being unconscious throughout the illness.

1. *Chopra and De, Ind. Med. Gazette, March*, 1932, *p.* 128.
2. *Poisons, their Effects and Detection, Ed. V, p.* 461.
3. *Proc. Grant. Med. Coll. Soc., Bom., pt.* 8, 1880 ; *Collis Barry, Leg. Med., Vol. II, p.* 577.
4. *Med. Juris., Ed. III, p.* 273.
5. *Beng. Chem. Exam. Annual Report*, 1936, *p.* 11.
6. *Bhondoo Lal, Ind. Med. Gaz., May*, 1900, *p.* 180.
7. *Ibid., Oct.*, 1906, *p.* 406.

CHAPTER XXVII

IRRITANT POISONS—(*Contd.*)

II. ANIMAL POISONS

CANTHARIDES

The Spanish fly (Cantharis vesicatoria) or blister-beetle is ¾ to 1 inch long and ¼ inch broad, and is distinguished by the shining, metallic green colour of the head, legs and wing-sheaths. Under these sheaths there are two thin, brownish, transparent membranous wings. The powder of its dried body is greyish-brown, and contains shining, green particles. The active principle is *cantharidin*, $C_{10}H_{12}O_4$, the anhydride or lactone of cantharidic acid, which is a crystalline body, very slightly soluble in water, but freely soluble in alcohol, ether, chloroform and fixed oils. It is a powerful vesicant. The pharmacopœial preparations made from it are *Emplastrum cantharidini* (Blistering plaster) and *Liquor epispasticus* (Blistering Liquid) containing 0.2 and 0.4 per cent of cantharidin respectively.

The Indian fly (beetle) which yields cantharidin is known as Mylabris chicorii occurring abundantly in the rainy season in certain parts of North India and Kashmir. It is 1 inch long and about 1/3 inch broad. Its wing sheaths are black, marked with three broad, transverse, orange yellow, wavy bands, which contain scattered black, bristly hairs when viewed under a microscope.[1] Mylabris pustulata is another species which yields cantharidin. It is found in the fields of cereals and vegetables in the neighbourhood of Bangalore.[2]

Symptoms.—Locally applied to the skin, cantharides or cantharidin does not show any sign for two or three hours, and then produces redness and burning pain, followed soon by small vesicles, which later run together to form one large blister. It may be absorbed by the skin and cause poisoning.

Given internally, this substance produces an intense burning pain in the mouth and throat, quickly extending to the stomach and the whole of the abdomen, and accompanied by difficulty in swallowing, intense thirst, salivation due to the inflammation of the salivary glands, nausea, vomiting containing mucus, blood and shreds of mucous membrane mixed with shining, green particles, and diarrhœa of bloody stools with tenesmus. These are followed by pain in the loins, distressing strangury, passage of scanty urine containing blood and albumin, painful priapism in the male with swelling and inflammation of the genital organs and frequent seminal emissions, and abortion in pregnant women. The patient becomes extremely restless, with laborious respirations and a hard, quick pulse.

1. *Dutt, Ind. Med. Gaz., March,* 1922, *p.* 92.
2. *Iyer and Guha, Jour. Indian Industr. Science, Vol. XIV A, Part III,* 1931, *p.* 31.

In severe cases, headaches, delirium, convulsions and coma usually precede death.

Occasionally blisters occur in the mouth and other parts of the digestive tract with which it comes into contact. There is also redness of the eyes and lachrymation.

Fatal Dose.—Twenty-four grains[1] of powdered cantharides (non-official dose, 1/16 to ½ grain) have caused the death of a young woman, although 42 grains[2] and 2 drachms[3] have been recovered from. An ounce[4] of the non-official tincture (tinctura cantharidini, dose, 2 to 5 minims) has caused the death of a boy, aged 17 years; recovery has, however, followed a dose of six ounces.[5] About 1/50 grain[6] of crystalline cantharidin taken by a medical student out of misplaced curiosity produced poisonous symptoms, which persisted for 13 days. Eleven and-a-half grains of cantharidin have proved fatal.

Fatal Period.—The usual period is twenty-four to thirty-six hours. A man, 70 years old, died in 12 to 24 hours from a dose of 11½ grains of cantharidin.[7] A man,[8] aged 54, died in 2 days after he had taken some pills containing a large dose of cantharides with a view to promote success with his bride, aged 23 years. Death has also occurred in 4, as well as 14 days.[9]

Treatment.—Eliminate the poison by washing out the stomach. Give demulcent drinks and opium. Do not give oils or fats, as they dissolve cantharidin. Administer magnesium sulphate to empty the bowel and treat the renal damage by starvation followed by large quantities of water.

Post-mortem Appearances.—The green, shining particles of powdered cantharides may be found adherent to the mucous membrane of the stomach, which is softened, inflamed and ulcerated, showing patches of vesication or even gangrene. The same is the condition of the mucous membrane of the intestines. The spleen is hyperæmic and congested. The kidneys are congested and inflamed. The bladder is injected and ecchymosed.

Chemical Analysis.—Cantharidin should be extracted by Dragendorff's method, and should be tested by its blistering action on the skin.

When combined with a solution of caustic potash or soda, cantharidin gives a green precipitate with copper sulphate, and a red precipitate with cobalt sulphate.

Medico-Legal Points.—Cantharides has produced poisonous symptoms on account of its having been used as an aphrodisiac, or as a criminal

1. *Taylor, On Poisons, Ed. III, p.* 528.
2. *Beck, North Amer. Practitioner,* 1891, 3, *p.* 522.
3. *Med. Gazette, Vol.* 42, *p.* 873.
4. *Taylor, On Poisons. Ed. III, p.* 529.
5. *Pereira, Mat. Med., Vol.* 2, *pt.* 2, *p.* 750; *Taylor, Ibid.*
6. *C. H. Andrews, Lancet, Sep.* 24, 1921, *p.* 654.
7. *Ann. d'Hyg.,* 1892; *Witthaus, Med. Juris. and Toxic., Vol. IV, p.* 1113.
8. *Jour. Amer. Med. Assoc., Jan.* 1, 1921, *p.* 50.
9. *Taylor, On Poisons, Ed. III, p.* 529.

abortifacient. It is rarely used for homicidal purposes. Accidental poisoning has occurred from its external application as a vesicant, or from the use of a blistering paper (*Charta epispastica*). A case[1] is recorded in which an unmarried woman, aged 26, produced dermatitis artefacta by the application of cantharides plaster over the front of the neck and the chin down to the sternum and over the backs of the hands. The lesions were markedly angular and showed definite blisters in places.

According to the Law of England it is a criminal offence to administer a drug with the intention of exciting sexual passion, but not so according to the Penal Code of India.

The wings of the beetle resist putrefaction for a very long time; hence their shining particles may be visible on the gastric or intestinal mucous membrane by the aid of a lens many months after death has occurred.[2]

Cantharidin is eliminated in the urine and fæces. Cantharides does not affect fowls, but poisonous symptoms occur in a man, who eats the fowl that has been fed with cantharides.

SNAKES (OPHIDIA)

In India there are two chief varieties of snakes, poisonous and non-poisonous.

Poisonous Snakes.—These are again classified as Colubrine and Viperine.

The Colubrine snakes lay eggs. Their head is of about the same width as that of the neck, and the pupils of their eyes are circular. They are subdivided into the land (terrestrial) snakes (elapidæ), and the sea snakes (hydrophidæ).

The land snakes are the cobra (Naia tripudians), the king cobra or hamadryad (Naia bungarus), the common Krait (Bungarus cæruleus), and the banded Krait (Bungarus fasciatus). These land snakes have a round tail. The first two varieties are hooded, and the second two are non-hooded, and have a dorsal line of hexagonal scales.

Among the sea snakes the commonest is Enhydrina Valakadien. The sea snakes have a flattened tail, and the snout and crown are covered with large plate-like shields.

The viperine snakes are the Daboia or Russell's viper (Daboia or Vipera Russellii or Daboia elegans), and the Phoorsa (Echis Carinata). These snakes give birth to living young, and have a peculiar broad, lozenge-shaped head, small scales and a round tail. The pupils of their eyes are vertical slits.

These poisonous snakes possess two grooved or tubular fangs or poison teeth, communicating by means of a duct with the racemose glands secreting venom. These glands are the homologues of the parotid

1. *Frederick Gardiner, Brit. Med. Jour., Feb.* 15, 1930, *p.* 282.
2. *Gimlette, Malay Poisons and Charm Cures, Ed. II, p.* 132.

glands and situated below and behind the eyes, one on each side. The whole mechanism is so arranged, that all the venom secreted by the glands is discharged without any leakage at the moment the fangs penetrate the skin. The colubrines have very short and fine fangs, hence they cannot bite through the clothes; while the vipers have long and strong fangs, so that they can easily bite through the clothes. The colubrines must close the lower jaw before they can inject the venom, whereas the vipers can do so without closing the lower jaw.

Characteristics of the Snake Venom.—The fresh snake venom is a clear, transparent fluid, but loses 50 to 70 per cent of water and is converted into a yellowish granular mass which can be powdered when dried under bell jar in the sun or over concentrated sulphuric acid. The dried venom retains its toxic properties for an indefinite period. It dissolves rapidly in water. The cobra venom loses its poisonous property to a slight extent only, if heated to 73° C. for half an hour, but the daboia venom loses it altogether, if treated accordingly.

The snake venom is not a simple solution of one poisonous substance, but is a mixture of one or more of the following[1]:—

1. *A powerful fibrin ferment.*—This is separated at 75° C., because it is destroyed causing coagulation of the blood.

2. *An anti-fibrin ferment.*—This is not important. It causes permanent fluidity of the blood after death.

3. *A proteolytic ferment.*

4. *Cytolysins.*—These are present in a greater proportion in the viperine poison. They are capable of acting upon the red blood cells, leucocytes, endothelial cells of the vessels, nerve cells, and the cells of various other tissues. Hence in bites by viperines there is much hæmorrhage and sloughing.

5. *Agglutinin.*—This is for the red blood cells.

6. *Neurotoxins.*—These attach themselves to all the nerve cells and especially the cells of the respiratory centre. These are the chief constituents of the colubrine venom. Hence paralysis, especially of respiration, is a marked symptom. These substances vary greatly in different specimens.

7. *A substance*, which acts directly on the heart muscle, stimulating it and increasing its tone. This is also more marked in the colubrine venom.

Non-Poisonous Snakes.—There are several species of non-poisonous snakes inhabiting India. Their tails are not markedly compressed, and in most of the varieties their belly is covered with transverse plates, which, however, do not extend completely across it. They possess several small teeth attached to a short maxillary bone, and have no long and grooved fangs like the poisonous variety.

---

1. *For full particulars* vide *an article on Snake Venoms and Anti-venomous Sera by Major George Lamb, I.M.S., in Transactions of the Bom. Med. Congr.*, 1909, *p.* 242.

Symptoms of Snake Poisoning (Ophitoxaemia).—These vary according to the variety of the snake. If bitten by a cobra or krait, the symptoms are a considerable burning or stinging pain, irritation, redness and swelling at the site of the bite and, in some cases, gangrene of the skin and subjacent cellular tissue. In about an hour the patient begins to feel giddy, lethargic, and weak in the muscles and feels as if intoxicated. Salivation, nausea and vomiting are, sometimes, the early symptoms. Weakness of the muscles increases, and develops into paralysis, so that the patient is unable to stand. The tongue and larynx are also affected. Breathing becomes slower until it stops altogether, the heart continuing to beat for some time. Sometimes, convulsions may precede death.

In the case of viper or daboia bites there is a good deal of swelling and ecchymosis beyond the site of the bite, and bleeding from the apertures caused by the bite. Within a few seconds to fifteen minutes after the bite nausea, vomiting and the signs of collapse supervene with the cold, clammy skin, a small thready, imperceptible pulse, and dilated pupils which are insensible to light. These are followed by complete unconsciousness. In a case reported by Captain Coffin, R. A. M. C., 36 hours elapsed before the symptoms appeared. If the patient recovers from these effects, hæmorrhages occur from the mucous membranes of the rectum and other orifices of the body. Extensive local suppuration and sloughing, and malignant œdema or tetanus may supervene or death may occur from septitcæmia.

In some cases of snake bite death occurs from shock due to fright before the poisonous symptoms commence.

The snake poison, whether colubrine or viperine, has a hæmolytic action on blood, and reduces the power of its coagulability with the result that a thin bloody serum continues to ooze out from the wound for many hours.

Fatal Dose.—Fifteen to twenty milligrammes of the dried cobra venom. The amount of the dried venom yielded by a cobra in one bite is 200 to 370 milligrammes, and 150 to 250 milligrammes by a large daboia. From experiments conducted by Knowles[1] it was found that the amount of the cobra venom injected at a successful bite averaged from 172 to 211 milligrammes, while in the case of one cobra the amount of the venom injected at a single bite was 587 milligrammes or about 40 times the minimum lethal dose for man.

Fatal Period.—In the case of a cobra bite death occurs from 20 minutes to 30 hours, and in the case of a viper's bite death usually occurs in 2 to 4 days, but instantaneously[2] or within a few minutes[3] if the venom is injected into a vein. A case is recorded where the bite of a king cobra caused death in convulsions in three-quarters of an hour.[4]

Treatment.—Apply at once a ligature with a thick India rubber band at some distance above the site of the bite. The ligature should be tight

1. *Ind. Med. Gaz., Jan.*, 1922, *p.* 23.
2. *Ganguli, Ind. Jour. of Med., Vol. IV, No.* 2, 1923, *pp.* 70, 71.
3. *Chopra and Iswariah, Ind. Jour. of Med. Res., April,* 1931, *p.* 1113.
4. *Tropical Diseases Bulletin, May,* 1930, *p.* 360.

enough to stop the blood circulation in the part, but it should be slackened for a few seconds at regular intervals and should not be kept for more than half an hour.

2. Make free and deep incisions into the punctures, taking care not to cut any large blood vessel or injure any underlying bone. Suck the poison from the wound provided there are no sores about the mouth and lips, or neutralise the poison by washing the wound with a weak aqueous solution of potassium permanganate. If possible, inject hypodermically 15 grains of gold chloride dissolved in a minimum quantity of water at the site of the bite.

3. Inject hypodermically, preferably intravenously, 40 c.c. of antivenene as soon as possible and repeat the same dose if the symptoms do not abate. Antivenene is prepared at Kasauli from the combined venoms of the Cobra and the Russell's viper and is specific for the bite of either. Antivenene has also been found to neutralise the hæmorrhagin but not the other fractions of the Echis venom.[1]

4. Inject hypodermically pituitrin or adrenaline chloride, and intramuscularly 0.9 grain of calcium chloride in 20 minims of water.

5. Avoid alcohol if antivenene has been used, but give hot coffee or tea.

6. Promote warmth of the body by hot water bottles, and by friction with ginger or mustard.

7. Start artificial respiration, when necessary.

Post-mortem Appearances.—Lesions resulting from snake bite are, as a rule, two lacerated punctures about ½ inch deep in the case of colubrines and about 1 inch deep in the case of vipers. They may be so minute that they may not be visible to the naked eye, but may be seen with a lens. There is a good deal of swelling and cellulitis about the bitten part and hæmorrhage from the punctures, as well as from the mucous membranes of the body orifices. The areolar tissue round about the punctures is purple and infiltrated. The blood is extremely fluid and purple in colour. In cases of viperine bites solid clots may occur in the veins due to the fibrin ferment.

Chemical Analysis.—The following serum test,[2] as employed by Dr. Hankin, is used in the Government Laboratories, United Provinces, Agra :—

Make an aqueous solution or extract from a suspected rag, and inject it into a frog. If the frog dies, find out the lethal dose. Then take two more weighed frogs and inject into them their lethal dose, as follows :—

(*a*) The extract mixed with double the volume of freshly obtained antivenene (serum immunised against cobra and Russell's viper venom) after incubation for an hour.

(*b*) The extract under similar condition, but untreated with antivenene.

1. *Taylor and Mallick, Ind. Jour. Med. Res., XXIII, I, July,* 1935, *p.* 141.
2. *Chatterji, The Analyst, Nov.,* 1930.

Presence of snake venum is indicated by frog (*a*) being killed, and (*b*) remaining unaffected.

Medico-Legal Points.—Snake poisoning has not much medico-legal value except that in some cases of suicidal or homicidal deaths the alleged cause of death given by the relatives is snake-bite.

On the 28th August, 1919, the body of Musammat Kausalia, 20 years old, of Police Station Malihabad, was brought to the King George's Medical College Mortuary with a report that the deceased had been bitten by a snake. Upon examination of the body I found that the death was due to hanging. In another case, where a Hindu girl of 15 years was alleged to have died from a snake-bite on the 23rd July, 1922, the dissection revealed a rupture of the internal surface of the enlarged spleen.

The snake venom is seldom used for homicidal or suicidal purposes. A case[1] is recorded where an attempt at homicidal poisoning was made. A man attempted to throw some poison on the open wound of another, but missed the mark. The suspected poison was found to be cobra venom.

A case of suicide[2] by the injection of dried snake venom into a small wound is recorded. A man murdered his wife and to avoid the charge of murder attempted to commit suicide by taking arsenic by the mouth, and then as an additional precaution injected snake venom into a small wound on his left thigh, in a resolute attempt to commit suicide.

Cattle are, sometimes, criminally poisoned by introducing into the rectum rags impregnated with cobra poison. Dr. Hankin describes the process as follows :—A cobra is shut up in an earthen vessel with a banana and irritated. It bites the fruit, thus injecting its venom into the pulp, which is smeared on a rag. This rag is thrust, by the aid of a split bamboo, into the animal's rectum. Such rags are usually found post-mortem ; they should be dried, but never preserved in spirit, for this destroys the poison, which looks like a greasy substance of a dirty white colour like putty. It is also asserted that sometimes the snake is made to strike the victim directly.

The bodies of animals killed by snake poisoning may be eaten without any ill-effects, but their blood is poisonous, and destroys life, if injected into the human body.

The snake venom does not remain in the skin after the bite, but infiltrates into the areolar tissue and at some distance from the punctures owing to the free movement of the skin.

According to Frayer[3] the snake poison is excreted by the kidneys and mammary glands, and probably also by the salivary glands as well as the gastric mucous membrane. An infant died in two hours after it had sucked its mother who had been bitten by a venomous snake. This case also serves as an illustration that the snake venom may be absorbed by the stomach of an infant in a sufficient quantity to cause death.

1. *Madras Chemical Examiner's Annual Report*, 1929, p. 6.
2. *Madras Chemical Examiner's Annual Report*, 1933, p. 4.
3. *Thanatophidia of India*, p. 43.

POISONOUS INSECTS

Ants.—These produce pain, irritation and swelling at the seat of the bite owing to the action of formic acid secreted by certain glands situated in the tail.

Wasps, Bees and Hornets.—These secrete a poisonous fluid containing formic acid, when they sting. Single stings produce local irritation, burning pain and swelling; but multiple stings, sometimes, produce symptoms very much resembling heat apoplexy. These are unconsciousness, lividity of the face, jerky breathing, cold and clammy skin, and involuntary passage of the urine and fæces. Death may occur from shock as well. A case[1] is recorded in which a woman, aged 55 years, and of a highly nervous temperament, became unconscious soon after she had been stung by a bee behind the ear and died immediately from shock.

Another case[2] is recorded in which a lady stung by a bee on the ring finger suffered from severe symptoms. A few minutes after the sting the pulse was rapid and small, the face and neck were swollen and cyanosed, and breathing was obstructed by a similar swelling of the glottis and bronchioles.

Treatment.—The sting should be removed by lifting or scraping it out with the blade of a knife or the edge of a long finger nail, and the part should then be dabbed on with a solution of ammonia. Hot fomentations should be applied to relieve the pain of severe stings.

The wasp sting is alkaline in reaction, and should be treated with dilute vinegar.

Pugnat[3] recommends the early local application of a 20 per cent solution of powdered aloes in 60 per cent ethyl alcohol for the treatment of the stings of bees, wasps and hornets. The pain is checked almost immediately, and the swelling either does not appear or is greatly reduced.

Scorpions.—These possess a hollow sting in the last joint of their tail, communicating by means of a duct with the poisonous glands, which secrete poison on stinging. The poison coagulates the blood and causes hæmorrhage from injury to the capillary walls and leads to the formation of emboli due to the agglutination of the red blood corpuscles.

Symptoms.—The symptoms produced by the sting are severe local irritation and burning pain radiating from the site. Sometimes, there may be giddiness, faintness, muscular weakness, vomiting, diarrhœa, convulsions and mental disturbances. Very rarely death may occur, especially in the old and feeble. William O'Hara[4] reports two fatal cases of scorpion sting at Nellore. In one case a cart driver, 40 years old, was stung in the finger, and died in three hours and-a-half. In the other a Hindu lascar, 55 years old, was stung by a dark scorpion, about three inches long, in the great toe, and he died in four days. Sundaram[5] reports the case of a boy, 18 years old, who was stung by a scorpion on the left index finger on March 2, 1930 and died of acute pulmonary œdema on March 14, 1930. Krishnamurty[6] also records a case in which a boy, aged 15, was stung by a scorpion and died of acute pulmonary œdema in about 18 hours.

Treatment.—Apply a ligature above the site of the sting and incise it. Wash the wound with a weak solution of ammonia, borax or potassium permanganate or apply 5 to 10 minims of a 5 per cent solution of cocaine around the site of the wound. In cases of shock give hypodermic injections of caffeine and atropine sulphate, and administer subcutaneously or intravenously normal saline.

Case.—A young man, aged 22 years, was stung by a scorpian on the great toe of the left foot, and developed the following symptoms:—

---

1. *Lancet*, 1872, *Vol. II*, *p.* 135.
2. *F. W. L., Brit. Med. Jour., Aug.* 16, 1924, *p.* 303.
3. *Revue Medicale de la Suisse Romande, Lausanne, March* 25, 1929, *p.* 205; *Jour. Amer. Med. Assoc., June* 1, 1929, *p.* 1897.
4. *Ind. Med. Gaz., March*, 1884, *p.* 73.
5. *Ind. Med. Gazette, Sep.*, 1931, *p.* 510.
6. *Ind. Med. Gazette, Sep.*, 1931, *p.* 537.

The skin was cold, clammy, and showed cutis anserina. On the left great toe was a perforation from the scorpion sting, but the parts were not swollen. He vomited several times, then became semi-conscious, very restless, and began to froth at the mouth. In conscious moments he complained of severe pain throughout the body, but especially in the chest and the affected leg. There was much cough and expectoration of froth, which gradually became blood-stained. He showed all the symptoms of severe shock. The temperature was 96.2° F. The pulse was imperceptible at the wrist, but the apex beat was faintly heard with the stethoscope at the rate of 136 per minute. The respirations were much embarrassed, and the whole body was bathed in cold sweat. Examination of the chest revealed signs of generalised œdema of the lungs. The lips became cyanosed. In lucid moments the patient desired to void urine and fæces, but his efforts were unavailing for about one hour; he then passed a stool and somewhat later about 250 c.c. of urine. The urine was acid in reaction, and contained albumin. On microscopic examination it showed a few hyaline and finely granular casts, many leucocytes and an occasional red corpuscle. After about 2½ hours he improved sufficiently to be carried home and recovered completely within three days.[1]

FOOD POISONING

Food poisoning occurs occasionally as an acute illness in a number of individuals shortly after the consumption of the same food. It is due to infection of the food with living bacteria of the Salmonella group, *e.g.*, the *Bacillus enteritidis* of Gaertner and the *Bacillus aertrycke*. These bacteria are destroyed in the process of cooking but the toxins generated by them are resistant to heat and may be present in food even after boiling it for one hour.

Hæmolytic streptococci and staphylococci are reported to have produced outbreaks of food poisoning.

The foodstuffs which are responsible for causing this kind of poisoning are diseased meats, fish, eggs, milk, cheese, ice-creams, and tinned foods. Such foods often appear quite fresh and do not show any alteration in taste or smell to arouse one's suspicion of their poisonous nature.

Outbreaks of food poisoning were formerly described as cases of ptomaine poisoning on the assumption that the poisoning was caused by ptomaines produced by putrefactive changes occurring in meat or other food, but there is no evidence to show that these substances are the causative agents of food poisoning. Moreover, ptomaines are late degradation products and are never found until the food has become too nasty to be eaten.

Symptoms.—These usually commence soon after the ingestion of the food, when the toxins are the causative agents but they may be delayed for six to twelve or even twenty-four hours in cases where the living bacteria are the causative agents. The chief symptoms are headache, giddiness, intense thirst, acute vomiting, diarrhœa with colicky pain in the abdomen, dilatation of the pupils, ptosis, cold, clammy skin, rise of temperature to 101° and 103° or 104° F. with rigors, muscular weakness, cramps and paralysis of the lower limbs. The pulse becomes slow, weak and finally imperceptible. Death occurs from failure of the heart.

Diagnosis.—This is made by isolating the bacteria from the vomit, urine or fæces and the suspected foods or from the bowels and solid organs of the sufferer after death and identifying them by cultural characteristics and agglutination tests.

Treatment.—Wash out the stomach, and give brisk saline purgatives to empty the bowels. Give saline infusions to promote elimination of the toxins from the system. Use stimulants, if necessary.

Post-mortem Appearances.—The mucous membrane of the alimentary canal is swollen and acutely congested with submucous patechial hæmorrhages. The liver, spleen and kidneys are congested. The lungs are usually congested.

1. *Milton C. Lang, Ind. Med. Gaz., Nov.*, 1926, *p.* 553.

Food Allergy.—Owing to an inherent or acquired idiosyncrasy some individuals are hypersensitive to certain kinds of food which are ordinarily quite harmless and suffer from gastro-enteritis, local urticarial rashes or asthamatic attacks, whenever they take any of these articles. The foods which produce these symptoms are protein in nature and are meat, fish, shell-fish, prawns, eggs, milk, cheese, etc.

BOTULISM OR ALLANTIASIS

This is a form of food poisoning which is caused by the toxins of the *Bacillus Botulinus* contained in sausages, potted meats, tinned fish, canned fruits, etc.

Symptoms.—These commence generally within 24 hours after taking the unwholesome food, but may be delayed for 72 hours. There are dryness of the mouth, difficulty of swallowing, retching, vomiting, colic and diarrhœa followed by constipation. The nervous symptoms then appear with dilatation of the pupils, ptosis, diplopia, aphonia and a sense of suffocation. Marked muscular weakness and nervous prostration are the prominent symptoms. The pulse becomes weak, and the face becomes cyanosed. The temperature rises to 103° F., but falls below normal towards death, which is preceded by delirium or coma. Death may occur within 24 hours or may be delayed for a week.

Treatment.—Inject promptly the antitoxic (anti-botulinus) serum, if it is available. Wash out the stomach to prevent further infection. Give mild laxatives followed by high irrigation of the intestine with enemata. Administer morphine hypodermically to relieve vomiting and purging. It may be helpful to use stimulants and subcutaneous or intravenous injections of normal saline.

Post-mortem Appearances.—Hyperæmia of the alimentary tract. The other organs are found congested.

FISH POISONING

Certain kinds of fish belonging to the species, tetrodon, found in China and Japan, are very poisonous, and cause death within an hour.

Fish in sprawn is likely to produce poisonous symptoms. Of all the varieties of shell-fish, mussel is the chief, that gives rise to poisonous symptoms on account of a powerful toxin, *mytilotoxine*, which develops chiefly in its liver. The characteristic symptoms are urticaria and difficulty of breathing. Death may occur from collapse within two hours.

The symptoms of gastro-enteritis may occur from eating stale or decomposed oysters.

PTOMAINES

These are alkaloidal bodies produced by the action of saprophytic micro-organisms upon nitrogenous materials, probably during the process of decomposition. They are called *cadaveric alkaloids*, as they are generated in the dead tissues ; while alkaloids secreted by the living cells during the metabolic processes are called *leucomaines*.

Ptomaines exist as methylamine in the gaseous form, as ethylamine in the liquid form and as neurine in the solid form. They are unstable alkaline bases, forming salts when acted upon by acids.

These ptomaines resemble very closely vegetable alkaloids, such as veratrine, morphine, codeine, etc., inasmuch as they respond almost to the same chemical group reagents and physiological tests. At present there is no special test by which a cadaveric alkaloid can be distinguished from a vegetable alkaloid ; however, no cadaveric alkaloid will yield the same chemical reactions and will have the same physiological results, if injected into the body of a healthy animal, as any of the vegetable alkaloids.

In suspected poisoning, when one of the rare vegetable alkaloids, which does not ordinarily respond to chemical tests, has been detected in the body, the defence

pleader may set up a plea that the alkaloid was not a vegetable poison, but a ptomaine developed in the body after death. In this connection it should be remembered that most of the ptomaines that have been discovered are non-poisonous except *neurine* and *mydaleine,* which are actively poisonous, while a few more are poisonous in a much larger quantity than the lethal dose of the corresponding vegetable alkaloid. Again, neurine is not generated till the fifth or sixth day has elapsed since death, and mydaleine not until the seventh day and that too in traces only. It is not produced in a quantity sufficient for analytical purposes until the second or third week after death. Choline, which is a very weak poison, is the only alkaloid met with at the time, when medico-legal post-mortem examinations are ordinarily held.

The symptoms produced in animals by neurine are marked increase in salivation, lachrymation and nasal secretion; diarrhœa; contraction of the pupils; slowing of the heart and respiration; convulsions; paralysis of the limbs and death. These symptoms are analogous to those of muscarine, the active principle of the fly fungus. Atropine acts as an antidote and, if previously injected, will render the animal immune against its action.

Mydaleine produces increased lachrymal and nasal secretions, vomiting, diarrhœa, dilatation of the pupils and a slight rise of temperature to 99° or 100° F. Later, clonic spasms occur with paralysis and stupor ending in death. The pulse and respirations are first quickened and then become slow. The fatal dose is fifty milligrammes.

CHAPTER XXVIII

IRRITANT POISONS—(*Contd.*)

C. MECHANICAL (VULNERANT) POISONS

Mechanical irritants are actually not poisons, inasmuch as they do not produce any toxic symptoms by being absorbed in the blood, but they are included in the expression "unwholesome drug or other drug" of Section 328 of the Indian Penal Code, as they act mechanically by a local action, and cause irritation of the stomach and bowels with their angular edges or sharp points, when they are swallowed. The examples are powdered glass, diamond dust, pins, needles, nails, chopped animal and vegetable hair.

POWDERED GLASS

Symptoms.—Taken internally, powdered glass produces a sharp, burning pain in the throat and stomach and later in the intestines. This is followed by nausea and vomiting, the vomited matter containing streaks of blood. There is generally constipation, but sometimes there is diarrhœa. The motions are passed with pain and are usually mixed with blood. Death may occur from shock, especially if the stomach or intestine has been perforated.

At Agra, a young Mahomedan male, aged 20 years, was invited for breakfast at his father-in-law's house, where he was given powdered glass in the food. About 8 hours after the breakfast he complained of an intense burning pain in the pit of the stomach, and brought up mouthfuls of blood without any nausea or pain in the throat. The vomiting of blood was so very persistent that he became pale and had almost collapsed with a thready and imperceptible pulse, when ergot injection and saline infusion had to be tried. The symptoms abated after three days.

Fatal Dose.—Not known. A large tea-spoonful of powdered glass proved fatal to a child, 11 months old.[1] On the contrary, from his experiments Lesauvage[2] found that two and-a-half drachms of powdered glass given to a cat did not cause any harm, and a dog took six or seven ounces in eight days without suffering the slightest inconvenience, although it was administered when the animal was fasting, and the fragments were frequently a line in length. He himself swallowed a considerable number of fragments of glass, upto 2 m.m. (0.08 inch) long, without producing any deleterious effects.

Fatal Period.—Uncertain. A woman, 25 years old, of Mandi State, who swallowed powdered glass with the intent of committing suicide, died

1. *Hebb, Midland Med. and Surg. Reporter,* 1829, *Vol. I, p.* 47.
2. *Peterson, Haines and Webster, Leg. Med. and Toxic., Vol. II, Ed. II, p.* 889.

in 2 hours.[1] In a fatal case reported by the Chemical Analyser of Bombay death occurred in 48 hours,[2] and in another case recorded by Reichardt death took place in 6 days.[3]

Treatment.—Give bulky food, such as a large quantity of rice, and then give emetics, as well as purgatives. Give ice and morphine to relieve thirst and pain. Adopt such remedies as will combat collapse.

Post-mortem Appearances.—Erosions may be found in the mouth, pharynx, œsophagus, stomach and upper part of the small intestine. Fragments of glass may be found adherent to the mucous membrane of the stomach which is covered with tenacious mucus. The mucous membrane of the stomach and intestines is red, congested and streaked with blood.

In the case of the young woman of Mandi State who committed suicide by swallowing powdered glass, no excoriations were seen in the mouth or œsophagus at the post-mortem examination, but the stomach was highly congested, especially the greater curvature. The stomach contained undigested boiled rice mixed with pieces of glass. As much as 190 grains of powdered glass were collected from the stomach and the biggest piece weighed 3 grains. The small intestine was congested very much and particles of glass were found adherent to the mucous membrane. The ileo-cæcal valve was intensely congested. The mucous membrane of the stomach was leathery, but in the intestine the rugæ exhibited the appearance of minute scratches under the lens and fine particles were visible between the folds. The larger pieces of glass were found high up in the intestine.

Chemical Detection.—By straining the stomach contents and fæces through a muslin cloth, glass fragments may be detected with the naked eye, or they may be seen as transparent and amorphous pieces under the microscope.

The Chemical Examiner of Bengal reports a case referred to him by the Civil Surgeon of Howrah in which the cook of a European Guard attempted to poison his master with powdered glass mixed with the meat curry. The Guard took a portion of it but, suspecting something wrong with it, handed over the remainder to the police who forwarded it for chemical analysis along with the stool of the complainant which he passed after taking the meal. Coarsely powdered glass with many small sharp fragments were detected both in the curry as well as in the stool.[4]

Medico-Legal Points.—The popular belief is that glass is highly poisonous, so that it is frequently administered in a powdered or crushed form mixed with some article of food, such as rice, wheat, flour, sweets, etc. Usually a woman pounds her own glass bangle or a glass bottle and gives it to her husband in some dish with homicidal intent. Sometimes, glass is mixed with arsenic before administration. It is occasionally employed for destroying cattle, and is rarely selected for suicidal purposes.

1. *Punjab Chemical Examiner's Annual Report,* 1926, *p.* 2.
2. *Collis Barry, Legal Med., Vol. II, p.* 589.
3. *Arch. d. Pharm. Second Series, Vol. XCI, p.* 92; *Peterson, Haines and Webster, Leg. Med. and Toxic., Vol. II, Ed. II, p.* 893.
4. *Ind. Med. Gaz., Aug.,* 1915, *p.* 305.

A case[1] is recorded where a man swallowed powdered glass mixed with nitric acid and kerosene oil with a view to commit suicide, but he was removed to hospital where he recovered after proper treatment. The vomited matter contained particles of glass, nitrates and kerosene oil.

Glass does not produce the desired effect, if it gets entangled in the mucus or food in the stomach. Similarly it will not have any bad effects, if it is so well powdered as not to have any sharp points, or if it is so well chewed as to get well powdered before it is swallowed. This is the reason why professional exhibitors (*human ostriches*) do not come by any harm by swallowing glass. Some years ago, I saw in Bombay a gentleman and his wife both eating chimney glass without any ill-effects.

DIAMOND DUST

The proverbial method of commiting suicide by sucking a diamond ring is nothing but a myth, as a diamond, when swallowed, has no other action but mechanical on account of its prominent sharp angles. A man[2] swallowed in the morning eight powdered diamonds (size not known) with a view to commit suicide. An hour later he complained of pain in the stomach and was attended to by a doctor. His stomach was washed out and he was given butter and boiled rice. The stomach wash was found to contain minute transparent particles under the microscope.

Chevers[3] mentions several cases of attempted suicide by swallowing a whole diamond or diamond dust and records a homicidal case in which a woman killed her paramour by administering diamond dust in milk. In a Hyderabad poisoning case it was alleged that diamond powder was administered in *pansupari*, but it had no effect.[4]

In the famous Baroda case white arsenic and a very fine powder of diamond were mixed in a *sherbet* drink.

NEEDLES

These have been swallowed for suicidal purposes, and are known to have caused death.

CHOPPED ANIMAL HAIR

This is supposed to be poisonous, and has been given to cattle with the idea of destroying them, but it should not be forgotten that, sometimes, round boluses of hair are found in the stomach and intestines of animals dead from natural causes.

Finely chopped human hair is recognised as a slow poison and given in curry or other soft food in Singapore. It is also frequently used in Turkey and produces by continued irritation a chronic disease resembling cancer.[5]

In his annual report for the year 1932, the Chemical Examiner of Bengal reports a case where chopped hair mixed with dry plantain leaves and dust was given to a woman for administration of the same to her husband with food "to correct his temper and to make him love her." A case[6] is also recorded where a tuft of chopped hair and small fragments of human nails were administered in rice and vegetables to a Mahomedan male by his wife probably as love-philters.

Chopped human hair mixed with lime, earth or powdered bone is used as a cattle poison, particularly in the districts of Gaya and Hazaribagh in Bihar and in the district of Mymensingh in Bengal.[7]

1. *Madras Chem. Exam. Annual Rep.*, 1935, *p.* 6.
2. *Madras Chem. Exam. Annual Rep.*, 1935, *p.* 6.
3. *Med. Juris.*, *pp.* 289-90.
4. *Times of India*, Dec. 21, 1935.
5. *Chevers, Med. Juris.*, *p.* 291.
6. *Ben. Chem. Exam. Ann. Rep.*, 1936, *p.* 11.
7. *Beng. Chem. Exam. Ann. Rep.*, 1937, *p.* 16.

VEGETABLE HAIRS

Hairs of some vegetable plant, such as *mucuna pruriens*, the cow-itch or cow-hage and also known as "Russian fleas" (*Kavach*), produce local redness, intolerable itching, and even blisters when applied to the skin, and are liable to set up the symptoms of irritation, when swallowed by the mouth. A case of torture by mucuna pruriens is recorded in the annual report of the Chemical Examiner, Bengal, for 1909. A lad, 15 years old, threw some powder of the burnt pods of this plant on a female relation of his on account of some quarrel between them. The woman suffered from itching and burning all over the body attended with a swelling for two days, but ultimately recovered. On analysis the partly burnt pods were found covered with fine stiff hairs.[1] The Chemical Examiner of the United Provinces of Agra and Oudh mentions in his annual report for 1916, that an anonymous letter containing some of the hair of mucuna pruriens was sent. It leaked in the post office, and produced irritating symptoms on the hands of post office officials. In his annual report for 1931, the Chemical Analyser of Bombay also reports the case of two men who had a quarrel in the temple of Shri Vithoba at Pandharpur in Sholapur District, and one of them threw some powder over the other as a result of which he got almost unbearable itching of his body for which he was treated as an outpatient in the local dispensary. Some of the powder was seized by the police and sent for identification. It was found to contain numerous whitish hairs which were identified as those from the pods of the cowhage plant.

1. *Ind. Med. Gaz., Sept.*, 1910, *p.* 362.

CHAPTER XXIX

NEUROTIC POISONS

POISONS AFFECTING THE BRAIN (CEREBRAL, NARCOTIC)

A. SOMNIFEROUS POISONS

OPIUM (*AFIYUN*)

Opium is the juice obtained by incision from the unripe capsules of the white poppy, *Papaver Somniferum,* and inspissated by spontaneous evaporation. The white poppy belongs to N. O. *Papaveraceœ*, and is

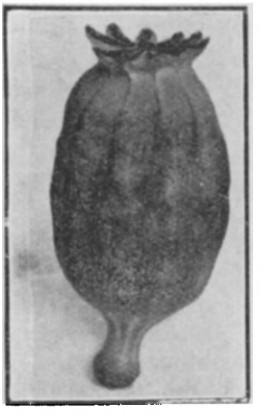


Fig. 139.—Poppy capsule—unincised.

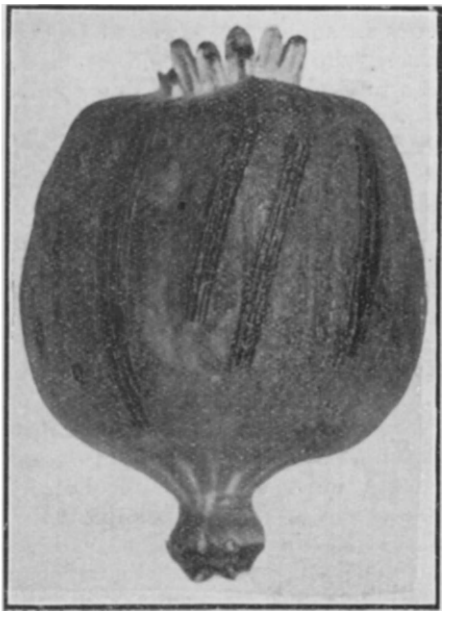


Fig. 140.—Poppy capsule—incised.

grown in India, Persia, Asia Minor, China and Egypt, but is cultivated in England and other cold countries.

Poppy capsules (*Post ka Doda*), when they are ripe and dry, contain a trace of opium and are, therefore, narcotic in action. Their warm decoction is used locally as a sedative fomentation and poultice.

Poppy seeds (*khas-khas*) are innocuous and white in colour and are used as food. They are sprinkled over some Indian sweets. They are regarded as demulcent and nutritive. They yield a bland oil, known as

poppy-seed oil (*khas-khas ka tel*), which is largely used for culinary and lighting purposes.

Opium occurs in more or less rounded, irregularly formed or flattened masses, weighing from 250 to 1,000 grammes, and having a heavy poppy-like smell and mawkish bitter taste. When fresh, it is plastic and internally moist, coarsely granular or nearly smooth and reddish or chest-nut brown, but becomes hard, brittle and dark brown on keeping. It is a highly complex body, containing about twenty-one alkaloids, combined with meconic, lactic and sulphuric acids. Of these the most important is morphine which occurs in combination with meconic acid. Next in importance are codeine, narcotine, papaverine and thebaine. Opium yields from 6 to 23 per cent of morphine, 0.3 to 2 per cent of codeine, 2 to 8 per cent of narcotine, 1 per cent of papaverine and 0.3 to 1 per cent of thebaine.

Of the several varieties of opium met with in India, the chief are Patna opium and Malwa opium. Patna opium yields 7 to 8 or even 10 per cent of morphine, and is chiefly prepared for medicinal purposes. It occurs in square packages of from two to four pounds in weight, usually covered with layers of talc. It is solid and brittle in the cold season, and has a brown colour and a fine odour. Malwa opium occurs in many varieties ; of these the superior quality yields from 6 to 8 per cent of morphine, and occurs in balls or cakes weighing about 10 ounces and covered with a coarse dust consisting of broken poppy petals. The inferior quality yields only from 3 to 5 per cent of morphine and occurs in flat circular cakes weighing about $1\frac{1}{2}$ pounds without any external covering. These are dull, opaque, blackish-brown externally, and somewhat dark and soft internally.

Morphine, $C_{17}H_{19}NO_3$.—This is the principal alkaloid to which the poisonous properties of opium are chiefly due. It occurs as a white powder or in white, shining crystals, having a bitter taste and alkaline reaction. It is very sparingly soluble in cold water, but soluble in 400 to 500 parts of boiling water. It is slightly soluble in ether and alcohol, but dissolves in acetic ether and amyl alcohol. It readily dissolves in dilute acids and in solutions of caustic alkalies and alkaline earths. It forms crystalline salts, of which morphine hydrochloride and morphine tartrate are pharmacopœial preparations and morphine acetate a non-official preparation, the dose of each being $\frac{1}{8}$ to 1/3 grain. These salts are bitter in taste, neutral in reaction and freely soluble in water.

Morphine has a specific action on the nerve-cells of the brain, and has a narcotic effect.

Heroin (Diacetyl-morphine or Diamorphine), Dionin (Ethyl-morphine) and Peronin (Benzoyl-morphine) are artificial alkaloids derived from morphine and used in medicine to allay cough in phthisis and asthma. Hydrochloride of heroin is now official under the name, *Diamorphinæ hydrochloridum*, the dose being 1/24 to $\frac{1}{8}$ grain. It is used sometimes by hypodermic injection and sometimes as a narcotic snuff like cocaine. It is liable to produce a habit after continued use. It is mainly

excreted by the kidneys. Medicinal doses have produced toxic symptoms, and 6.9 grains have produced death in 70 hours,[1] while recovery has occurred after 9 grains.[2]

Dilaudide (hydrochloride of dihydromorphinone) is an oxidation product of morphine. It is a colourless, crystalline substance, freely soluble in alcohol and water, but insoluble in ether. It is used as a substitute for morphine in 1/12-grain doses.

Codeine, $C_{18}H_{22}NO_3$.—This is chemically methylmorphine, and occurs in nearly colourless trimetric crystals. It is soluble in 120 parts of water, in 2 parts of alcohol and in chloroform. It is soluble in aqueous ammonia, but insoluble in excess of potash or soda solution. It dissolves easily in dilute acids, and forms neutral salts. Codeine and its salt, codeine phosphate, are pharmacopœial preparations, the dose of each being $\frac{1}{4}$ to 1 grain.

Codeine resembles morphine in its action, although it is much less poisonous. Four grains[3] have caused dangerous symptoms, and recovery has occurred even after 8 grains.[4] The symptoms produced by codeine poisoning are sometimes nausea, vomiting and abdominal pain, and thus differ from those caused by morphine poisoning. Codeine is excreted unchanged by the kidneys.

Narcotine, papaverine and thebaine are not important from a toxicological point of view. Narcotine and its salt, narcotine hydrochloride, are unofficial preparations, the dose of each being 1 to 3 grains. Narcotine is much less poisonous than either morphine or codeine, and produces toxic effects only in very large doses. The symptoms are chiefly convulsions.

Papaverine resembles codeine in its effects on man. One gramme might cause dangerous symptoms. It appears to undergo complete destruction in the tissues.

Thebaine has a convulsant action, and produces tetanic spasms resembling those caused by strychnine but is much less powerful. The fatal dose is not known. About 8 grains would produce poisonous symptoms.[5]

Dicodide (Bitatrate of dihydrocodeinone) and eukodal (hydrochloride of dihydroxycodeinone) are salts which occur as white crystals, are soluble in water, and are given in 1/16 to 1/12-grain doses.

Official Preparations.—The following are the official preparations of opium and its alkaloids:—

1. *Opium Pulveratum* (Powdered opium).—Synonym, *Pulvis Opii.* Dose, $\frac{1}{2}$ to 3 grains.

2. *Extractum Opii Siccum.*—Strength, 20 per cent of morphine. Dose, $\frac{1}{4}$ to 1 grain.

1. *Mc Nally, Jour. Lab. and clinic. Med.*, 1917, *Vol. II, p.* 571.
2. *Martindale and Westcott, Extra Pharmacopoeia, Vol. I, Ed. XX, p.* 559.
3. *Myrtle, Brit. Med. Jour., April*, 1874, *p.* 478.
4. *Walsh, Ibid.*, 1889, *Vol. II, p.* 718.
5. *Blyth, Poisons, their Effects and Detection, Ed. V, p.* 321.

3. *Pulvis Cretæ Aromaticus cum Opio.*—Strength, $2\frac{1}{2}$ per cent of opium. Dose, 10 to 60 grains.

4. *Pulvis Ipecacuanhæ et Opii* (*Pulv. Ipecacuanhæ Co*; Dover's Powder).—Strength, 10 per cent of opium. Dose, 5 to 10 grains.

5. *Suppositorium Plumbi cum Opio.*—Strength, 1 grain of opium in each.

6. *Tinctura Opii* (Laudanum).—Strength, 1 per cent of anhydrous morphine. Dose, 5 to 30 minims.

7. *Tinctura Opii Camphorata* (*Tinctura Camphoræ Co*; Paregoric or Paregoric Elixir).—Strength, 1/37 grain of morphine in 1 fluid drachm. Dose, 30 to 60 minims.

8. *Morphinæ Hydrochloridum.*—Dose, $\frac{1}{8}$ to 1/3 grain.

9. *Liquor Morphinæ Hydrochloridi.*—Strength, 1 per cent of morphine hydrochloride. Dose, 5 to 30 minims.

10. *Supppositorium Morphinæ.*—Strength, $\frac{1}{4}$ grain of morphine hydrochloride in each.

11. *Trochiscus Morphinæ et Ipecacuanhæ.*—Strength, 1/32 grain of morphine hydrochloride in each.

12. *Morphinæ Tartras.*—Dose, $\frac{1}{8}$ to 1/3 grain.

Proprietary Medicines.—The following are the proprietary medicines containing opium or morphine:—

1. *Atkinson's Infant Preserver.*—Strength, 3 minims of laudanum to 1 ounce.

2. *Battley's Liquor Opii Sedativus.*—Strength, 2 grains in 12 minims, i.e., double the strength of laudanum.

3. *Black Drop* (*Acetum Opii Crocatum*).—Strength, 3 times as strong as laudanum.

4. *Bœrhaave's Odontalgic Essence.*—Strength, about 3 per cent of opium.

5. *Chlorodyne.*—Strength, about 4 grains of morphine hydrochloride to an ounce. In addition to morphine it contains chloral, chloroform, hydrocyanic acid and tincture of cannabis indica.

6. *Dalby's Carminative.*—Strength, $2\frac{1}{2}$ minims of laudanum to 1 fluid ounce.

7. *Godfrey's Cordial.*—Strength, $\frac{1}{2}$ to $1\frac{1}{2}$ grains of opium to 1 ounce.

8. *Grimrod's Remedy for Spasms.*—Strength, 1 grain of morphine hydrochloride in 5 ounces.

9. *Le Maurier's Odontalgic Essence.*—Strength, 1 grain of morphine acetate to 1 ounce of cherry laurel water.

10. *Mrs. Winslow's Soothing Syrup.*—Strength, 1 grain of morphine in 1 ounce.

11. *Nepenthe.*—1/3rd less in strength than laudanum.

12. *Powell's Balsam Aniseed.*—Strength, 1/10 grain of morphine in 1 ounce.

13. *Sydenham's Laudanum.*—This is a tincture of opium flavoured with saffron.

Symptoms.—These commence usually in from half-an-hour to an hour after the poison has been taken. The symptoms commonly take more time to appear when opium is taken in a solid form than when it is taken in solution. Cases have, however, occurred where the symptoms have appeared almost immediately or within a few minutes, especially in children and after a hypodermic injection of morphine, or have been delayed for several hours. A lady took an ounce and-a-half of laudanum on an empty stomach, and no symptoms of narcotic poisoning appeared for four hours and-a-half.[1] A man took laudanum at 6-30 a.m. and became drowsy between 5 and 6 p.m.[2] A man who took an ounce and-a-half of laudanum, and six drachms an hour afterwards was perfectly sensible seven hours later and only became unconscious in eighteen hours.[3] In a case of compound poisoning by laudanum and antipyrin no marked symptoms of opium poisoning appeared for nineteen hours and-a-half.[4]

The symptoms manifest themselves in three stages; *viz.*, 1. stage of excitement; 2. stage of sopor; 3. stage of narcosis.

1. *Stage of Excitement.*—During this stage the symptoms are increased mental activity, loquacity, restlessness, or even hallucinations, flushing of the face and increased action of the heart. This stage is of a short duration, and may be absent if a large dose is taken. In children convulsions are a marked feature in the first stage. In adults a widely excited, and even a maniacal, condition may be seen in this stage. An Indian soldier at Poona who committed suicide by taking opium was very excited and noisy for about a quarter of an hour and then became deeply comatose.[5]

2. *Stage of Sopor.*—The nerve centres are depressed during this stage, which, sometimes, comes on quite suddenly. The symptoms are headache, giddiness, lethargic condition, drowsiness, and an uncontrollable desire to sleep, from which the patient may be roused by external stimuli. The pupils are contracted, the face and lips are cyanosed and an itching sensation is felt all over the skin. The pulse and respirations are still normal.

3. *Stage of Narcosis.*—The patient now passes into deep coma from which he cannot be roused. During this stage the muscles are relaxed,

1. *Taylor, On Poisons, Ed. III, p.* 551.
2. *Channing, Boston Med. and Sur. Jour.*, 1857, *Vol. LVI, p.* 449.
3. *Christison, Poisons, Amer. Ed., p.* 544.
4. *Gregory, Brit. Med. Jour., Vol. II,* 1897, *p.* 1000.
5. *Bombay Chemical Analyser's Annual Report,* 1929, *p.* 7.

and the reflexes are lost. All the secretions are almost completely suspended, except that of the skin, which feels cold and clammy. The face is pale, the lips are livid, and the lower jaw drops. The pupils are contracted to pin points and are insensible to light. The conjunctivæ are injected. The pulse is slow, small and compressible. The respirations are slow, laboured and stertorous.

At this stage recovery may take place by prompt and proper treatment; otherwise in the case of fatal termination lividity of the surface increases. The pulse becomes slower, more irregular and imperceptible. The respirations are slower, more feeble, and assume the character of Cheyne-Stokes, death occurring from asphyxia. The heart may continue to beat for a short time after respiration has stopped. Convulsive twitchings in groups of the muscles are observed, and the pupils become widely dilated towards the end. Sometimes, death occurs from failure of the heart. The odour of opium may be present in the breath throughout the illness. This is, sometimes, masked by the injudicious administration of alcohol by relatives.

Unusual Symptoms.—Vomiting and purging may be present in a few cases. In a case in which a sweeper in Lahore, an addict to opium, died from an overdose, one of the chief symptoms was vomiting. The vomited matter on chemical analysis showed the presence of opium.[1] A young Hindu of Pangaon in Barsi Taluka, who died after taking the food prepared by his wife, had symptoms of vomiting and purging. An analysis of the viscera revealed the presence of opium alone and no other poison.[2]

Convulsions of a tetanoid character are occasionally present, more frequent in children than in adults. In the case of a student of the Agra College, who died of opium poisoning, the prominent symptoms were convulsions and a rise of temperature, which misled a medical attendant very much in the correct diagnosis. In the case of a private soldier in the first Yemen Infantry at Aden who died of opium poisoning, the chief symptoms were remittent attacks of convulsions and a rise of temperature to 104° F. before death.[3] In the case[4] of a female, aged 32, who died of opium poisoning, the chief symptoms were muscular rigidity, violent delirium, frequent respirations (58 per minute) and a temperature of 106° F. Opium was detected by the Chemical Analyser in the stomach and its contents and in the other viscera, *viz.*, the liver, spleen and kidneys.

In a few cases the pupils may be found dilated in the earlier stage, especially when chlorodyne has been taken. The dilatation of the pupils is probably due to hydrocyanic acid contained in it.

Syncope may occur in some individuals after a subcutaneous administration of morphine. A case is recorded where one-sixth of a grain of morphine hydrochloride injected subcutaneously almost proved fatal to an old man.[5]

1. *Punjab Chemical Examiner's Annual Report*, 1925, p. 2.
2. *Bombay Chemical Analyser's Annual Report*, 1928, p. 4.
3. *Bombay Chemical Analyser's Annual Report*, 1925, p. 4.
4. *Shah, The Bombay Medical Jour., Vol. I, No. 5, p. 171.*
5. *Blyth, Poisons their Effects and Detection, Ed. V, p. 307.*

After an apparent remission of symptoms, sometimes it so happens that they return with more severity to end in death. This is explained by the fact that absorption is practically in abeyance during the stage of depression, and the poison is reabsorbed from the alimentary canal, when circulation has improved.

In January, 1859, the Honourable Mrs. Anson[1] swallowed, while fasting, an ounce and-a-half of laudanum by mistake. In a quarter of an hour emetics were given, but she did not vomit for half an hour; and she was not treated medically for two hours and-a-half. The matter then drawn from the stomach had no smell of laudanum. She was quite unconscious, and had lost the power of swallowing. After remaining in this comatose condition for more than nine hours she revived. The face became natural, and the pulse steady. She was able to recognise her daughters, and in a thick voice to give an account of the mistake. But this state lasted only five minutes, when she again became comatose, and died in fourteen hours after the poison had been taken.

Diagnosis.—Opium poisoning has to be diagnosed from apoplexy, uræmic coma, acute alcoholic poisoning, carbolic acid poisoning, and compression of the brain.

Apoplexy.—In apoplexy the patient affected is usually fat and old. The onset is sudden and abrupt. The chief symptoms are a slow, full pulse and paralysis, usually hemiplegia. The pupils are dilated except when the lesion is in the pons Varolii, when they are contracted but not symmetrical, and the temperature is raised to 103° or 104° F. A case[2] is reported where encephalitis lethargica was diagnosed as opium poisoning, as the patient was semi-comatose with the pupils fixed and contracted to pin points and his temperature was subnormal.

Uræmic Coma.—In uræmic coma there is always the previous history of a kidney disease with the presence of albumin and casts in the urine and anasarca. Epileptiform convulsions generally precede coma.

Acute Alcoholic Poisoning.—In acute alcoholic poisoning the chief symptoms are the congested face, injected eyes, dilated pupils, odour of alcohol in the breath and snoring respirations. The patient may be roused by loud shouts or vigorous shaking, and there is no paralysis.

Carbolic Acid Poisoning.—The most characteristic signs are white patches on the lips and mouth, characteristic odour of the breath and green coloured urine.

Compression of the Brain.—History of the accident, probably fracture of the skull bones. The pupils are unequal or dilated with subconjunctival hæmorrhages.

Fatal Dose.—The smallest dose that has proved fatal to a man, aged 32 years, is 2½ grains of the extract of opium, equivalent to 4 grains of

1. *Taylor, On Poisons, Ed. III, p.* 552.
2. *Toms, Brit. Med. Jour., Nov.* 1, 1924, *p.* 814.

crude opium.[1] The average fatal dose for a healthy adult, not addicted to opium, may be regarded as 30 grains of opium.[2] Hofmann-Haberda[3] considers the lethal dose of opium to be 15 to 30 grains. Recovery has, however, taken place after much larger doses, even as much as 360 grains.[4] The smallest fatal doses of the tincture of opium are twenty minims in the case[5] of a Parsi lady and two drachms in the case of a female, 17 years old,[6] but even 8 ounces have been recovered from under proper treatment.[7] The average fatal dose of morphine or its salt for a healthy non-addict is about 3 grains,[8] though death has occurred occasionally from the doses of 1 grain of morphine hydrochloride, while recovery has followed much larger quantities. Seventy-five grains of sulphate of morphine is the largest quantity that has so far been recovered from.[9] One-sixth of a grain of morphine hydrochloride injected subcutaneously nearly killed an old man.[10] Half-a-grain of morphine hydrochloride administered hypodermically by mistake for a hypodermic strychnine solution proved fatal in 3 hours to a man suffering from acute bronchitis.[11] On the other hand, recovery took place after the hypodermic injection of about twelve grains of morphine.[12]

In infants and young children much smaller doses have proved fatal. For example, 1 grain of Dover's powder equivalent to 1/10 grain of opium has killed a child, 4 months old, and a quantity of paregoric elixir containing 1/90 grain of opium has caused the death of an infant, 4 weeks old.[13] One[14] and two minims[15] of the tincture have respectively killed infants of seven and five days in 18 hours. One-twelfth of a grain of morphine hydrochloride has killed an infant.[16] Children have also recovered from the effects of very large doses, *e.g.*, $7\frac{1}{2}$ grains of opium,[17] 3 grains of morphine sulphate,[18] and 2 drachms of laudanum.[19] I have successfully treated several infants and children who were accidentally poisoned by overdoses of opium.

Fatal Period.—The shortest recorded period is 45 minutes in a woman, aged 52, from an ounce of laudanum.[20] The usual period is 8 to

1. *Med. Gaz., Vol. XXXVII, p.* 239 ; *Taylor, On Poisons, Ed. III, p.* 556.
2. *Sydney Smith, Forensic Med., Ed. VI, p.* 522 ; *Punjab Chem. Examiner's Annual Rept.*, 1931, *p.* 10.
3. *Lehrbuch der gerichtlichen Medizin, 11te Aufl.*, 1927, *p.* 898 ; *Webster, Leg. Med. and Toxic.*, 1930, *p.* 671.
4. *Maynard, Brit. Med. Jour., Vol. I,* 1896, *p.* 1194.
5. *Bombay Chemical Analyser's Annual Report,* 1929, *p.* 7.
6. *Wormley, Micro-Chemistry of Poisons, Ed. II, p.* 470.
7. *Lancet, March* 29, 1873, *p.* 468.
8. *Sydney Smith, Forens. Med., Ed. VI, p.* 522 ; *Punjab Chem. Examiner's Annual Rep.*, 1931, *p.* 10.
9. *W. F. Norris, Amer. Jour. of Med. Scie., Oct.*, 1862, *p.* 395.
10. *Blyth, Poisons, Ed. V, p.* 307.
11. *Lancet, March* 28, 1896 ; *Collis Barry, Leg. Med., Vol. II, p.* 524.
12. *Pope, Ibid., March* 17, 1894, *p.* 524.
13. *Taylor, On Poisons, Ed. III, p.* 558.
14. *Smith, Lancet, April* 15, 1854, *p.* 419.
15. *Prov. Med. and Surg. Jour., Oct.* 28, 1846, *p.* 519.
16. *Chemical News, Aug.* 22, 1863, *p.* 98.
17. *Amer. Jour. of Med. Scie., April*, 1854.
18. *Dow, Va. Med. Monthly,* 1877-78, *IV, p.* 670.
19. *Med. Rec., New York,* 1894, *p.* 345.
20. *Amer. Jour. of Med. Scie., Oct.*, 1854, *p.* 384.

12 hours. Recovery is probable, if a patient survives 24 hours. In rare cases, however, death has been delayed for 2 to 3 days. A child of three months died in 56 hours after an opiate had been administered to it.[1] A girl, aged 19 years, died in 3 days after having taken 10 grains of morphine with suicidal intent.[2]

Treatment.—Wash out the stomach first with warm water preserving the washing for chemical analysis, and then with a solution of potassium permanganate of the strength of 10 to 15 grains to the pint of water. This washing should not be preserved for transmission to the Chemical Examiner, as potassium permanganate oxidises opium and its alkaloids and renders them unidentifiable. It also converts morphine into oxydimorphine, which does not satisfy any of the tests for morphine.[3] Moor[4] has shown that 1 grain of potassium permanganate in one ounce of water oxidises 10 grains of opium or 1 grain of morphine or 1 drachm of laudanum or another preparation of the same strength. The oxidising action is increased by the addition of dilute sulphuric acid. To continue this action it is advisable to allow about half-a-pint of the solution to remain in the stomach. The practical test to stop the lavage of the stomach is to find the return water of a pink colour.

If potassium permanganate is not available the stomach may be washed out with an infusion of tea or tannic acid or a mixture of finely powdered animal charcoal and water. Mustard or zinc sulphate may be given as an emetic. A prompt emetic is the hypodermic injection of apomorphine hydrochloride, but it should be administered cautiously, as it may increase asthenia.

Even in poisoning by the hypodermic injection of morphine the stomach should always be washed out as, after absorption in the blood, morphine is excreted into the stomach from which it is again liable to be reabsorbed.

If the patient is seen in the earlier stage before coma has supervened, an attempt should be made to keep him awake by flicking a wet towel on the face, by cold affusions on the head and by making him walk about after he is well supported by two men, one on each side, but it is no use dragging him if he cannot use his muscles.

Injection of atropine sulphate in 1/40-grain doses hypodermically has been recommended as a physiological antidote, to be repeated until the pupils begin to dilate, but it should be remembered that this drug paralyses the medulla oblongata, and so may aid in bringing about the fatal termination.

The heart should be stimulated by hot applications to the præcordium and by hypodermic injections of caffeine, strychnine and sulphuric ether. Hot coffee or tea may be administered either by the mouth or by the rectum.

1. *Med. Times and Gaz., March*, 1858, *p.* 292.
2. *Med. Sentinel*, 1893, *Vol. I, p.* 199.
3. *Henry, Plant Alkaloids, Ed. II, p.* 262.
4. *Med. Rec., New York*, 1894, *XLV, p.* 200 ; 1895, *XLVII, p.* 266 ; *XLVIII, p.* 611.

A 25 per cent solution of coramine in doses of 5 to 15 c.c. may be administered intravenously or intramuscularly as a stimulant to the circulatory and respiratory systems. Intramuscular or intravenous injection of lobeline and administration of oxygen with 5 to 7 per cent of carbon dioxide should also be tried to combat the respiratory failure. Artificial respiration aided by the application of the faradic current should be resorted to when the coma is profound, and should be continued as long as the symptoms last.

Charles R. Box[1] reports a case in which a lady who had swallowed some 6 ounces of laudanum half-an-hour earlier was saved by a free venesection when she had got coma, stertorous breathing, deep cyanosis and commencing œdema of the lungs. Sen[2] also advises venesection, specially when the patient is cyanosed and has a feeble pulse. Fifteen ounces of blood should be drawn out at once to relieve the congested heart, when blood pressure is not very low, and the loss should be replenished by normal saline or 25 per cent glucose solution given intravenously. Adrenaline chloride solution should be given to guard against the fall of blood pressure.

Post-mortem Appearances.—The post-mortem appearances are not very characteristic, but the signs of asphyxia are prominent. The face and the finger-nails are livid. Froth is seen at the mouth and nostrils. The blood is usually dark and fluid.

When the stomach is opened, small, soft, brownish lumps of opium may be found in its contents, which may also look brown and viscid. and may give the smell of opium.

The smell of opium is often noticed, as soon as the chest is opened, but it disappears with the setting in of putrefaction. The trachea is rosy coloured, congested and covered with froth, if seen soon after death. The lungs are often engorged and œdematous, and exude frothy fluid blood on section. The bronchial tubes are also congested and contain froth. The right side of the heart is full of blood, and the left is empty. Sometimes, both the chambers are full with venous engorgement. The brain and its membranes are congested. Similarly, the abdominal organs are largely congested, and exude dark fluid blood on section. The bladder is generally full of urine.

In the case[3] of an adult Hindu who died of opium poisoning, about four ounces of partly clotted blood were found in the pericardial cavity and the substance of the brain was found congested. There was an extravasation of blood in the skin of the neck and chest and both sides of the abdomen. On the front of the chest the hæmorrhages were at intervals, while on the sides of the abdomen and neck the hæmorrhages were continuous. There were hæmorrhages in the skin at intervals on the face and forehead. There were also hæmorrhages in the skin of the back as far as the suprascapular regions, from the nape of the neck. There were a few hæmorrhages in the skin on both the feet.

1. *Lancet, April* 23, 1927, *p.* 899.
2. *Ind. Med. Gazette, Dec.*, 1934, *p.* 693.
3. *Bombay Chemical Analyser's Annual Report,* 1929, *p.* 7.

Chemical Analysis.—To ascertain whether the suspected article contains opium or not, it is necessary to detect the presence of meconic acid and morphine, if possible.

Tests for Meconic Acid.—1. A solution of ferric chloride gives a blood-red colour, which is not affected by dilute hydrochloric acid (distinction from acetates) or by mercuric chloride (distinction from sulphocyanides). If meconic acid is present in a minute trace the colour becomes orange.

2. Lead acetate gives a white precipitate, soluble in nitric acid.

Tests for Morphine.—1. Strong nitric acid gives an orange colour unchanged by sodium thiosulphate.

2. Ferric chloride gives a greenish-blue colouration.

3. Sulphuric acid and dichromate of potassium give a green colouration.

4. Sulphomolybdic acid gives a reddish-purple colouration which changes to blue.

5. Iodic acid turns yellow, when morphine is added, but assumes a blue colouration on the addition of starch owing to the liberation of iodine by morphine.

6. *Marquis's Test.*—A reagent containing 3 c.c. of concentrated sulphuric acid and 2 drops of 40 per cent formaldehyde produces with morphine a red-purple colour which gradually passes into violet and finally into blue. Codeine and apomorphine produce the violet colour changing to blue, but not the initial purple-red. Narcotine produces a violet colour, but it becomes olive-green and finally yellow. Oxydimorphine gives a green colour. Dionin gives a dark-blue violet colour, while heroin produces the same colours as morphine.

7. *Husemann's Test.*—Sulphuric acid is added to a mixture containing morphine, and allowed to stand for sixteen to eighteen hours. Nitric acid is then added to the mixture, when an intensely dark violet colour appears, which gradually changes to blood-red and finally to orange.

8. *Urotropine Test.*—Urotropine and concentrated sulphuric acid give a blue violet colouration.

9. *Porphyroxine Test.*—The alkaline ether extract obtained by Stas' process is allowed to evaporate spontaneously in a small porcelain dish. To the dry residue a few drops of hydrochloric acid are added, and the dish heated over a flame, when a rose pink colour shows the presence of porphyroxine, a neutral constituent of opium, first described by Merck. This test was thought to be peculiar to Indian opium only, but Bamford has shown that at least some specimens of both Turkish and Smyrna opium respond to this test.[1]

1. *The Analyst, LV,* 1930, *pp.* 445-46.

Medico-Legal Points.—Opium is about the commonest drug selected by suicides. Young men, who have lost money in speculation or gambling, or who have been scolded by their parents for some offence, frequently resort to its use. Similarly, women who have quarrelled with their husbands or relatives, or who have been disappointed in love, take opium either to terrify their relatives or to end their imaginary worries and miseries.

Suicides usually mix opium with mustard oil or asafœtida in the belief that these substances increase its absorptive power, but there is no foundation about this belief. However, it is true that the mustard oil makes it difficult to be eliminated even by washing out the stomach.

It is also believed that alcohol hastens the action of opium, but it does not do so in all cases. I saw an Anglo-Indian in Agra, who took a bottle of beer and opium, but he developed no other symptoms except dryness of the throat and drowsiness.

Suicide by morphine is comparatively rare in India. In his annual report for 1927, the Chemical Analyser of Bombay reports two cases of suicide by morphine as against 79 cases by opium.

Owing to its bitter taste, its peculiar smell and its dark brown colour opium is rarely used as a homicidal poison for adults, although it is, sometimes, used to destroy illegitimate infants.

A case[1] occurred in the District of Khulna, where a woman, aged 26 years, entertained a visitor, who gave her alcohol to drink. She died subsequently under suspicious circumstances. Opium was detected in the viscera.

Opium is, sometimes, used as a cattle poison. In his annual report for 1925, the Chemical Examiner of the U. P. reports a case in which opium was found in a pill intended for poisoning cattle. He also mentions a case of Ballia, where an attempt was made to poison an elephant with some *juar* leaves mixed with *gur*. The substance on examination was found to contain opium.[2]

Cases of poisoning occur among infants and children by their accidentally swallowing crude opium or opium pills meant for their parents or grand parents, who are in the habit of using the drug. They are also, sometimes, poisoned by an accidental overdose, as they are usually drugged with opium by their parents, especially of the labouring class with a view to lulling them to unnatural sleep.

Children are extraordinarily susceptible to the influence of opium. Hence great precaution should be used in prescribing the drug for them.

Henton White[3] records a curious case of poisoning by opium in which a child, aged 3 months, was poisoned by the teat of a feeding bottle being accidentally contaminated with laudanum. The mother was in the habit of moistening the teat of the feeding bottle in her mouth before giving it to the baby, and at the time she had put a pledget of cotton wool soaked in laudanum in her tooth which was aching.

1. *Beng. Chem. Exam. Annual Rep.*, 1931, p. 9.
2. *Annual Report*, 1926, p. 4.
3. *Brit. Med. Jour., July* 13, 1901, p. 78.

Mode of Administration.—Cases of poisoning, sometimes attended with fatal results, have occurred when opiate or morphine preparations have gained access to the system by channels other than the mouth, *e.g.*, application to an abraded surface or wound or even to the unbraded skin, hypodermic injection, or introduction into the rectum or vagina. A Burmese boy, about 9 or 10 years old, received a gaping wound on the forehead, which was stuffed with about a quarter *tola* (45 grains) of opium. In about forty hours he developed the symptoms of opium poisoning, but recovered under active treatment.[1] An ounce of laudanum applied on a poultice to the abdomen produced death.[2] An injection containing thirty grains of opium administered by the rectum proved fatal to a man.[3] Five minims of laudanum injected into the rectum killed a child, eighteen months old.[4] A woman died in ten hours after the application of thirty grains of morphine to the cancerous ulcers of her breasts.[5] A man, 40 years old, died in sixteen hours from the effects of an enema containing three grains of morphine administered to relieve the pain caused by a fistula.[6]

Elimination.—Opium is chiefly eliminated in the fæces and urine. It is, therefore, necessary to preserve urine for chemical analysis especially in non-fatal cases of poisoning where the stomach wash does not give the tests for opium owing to the stomach having been washed out with a solution of potassium permanganate. Opium is, sometimes, detected in the saliva and bile. That it is eliminated by the milk is proved by the occurrence of fatal poisoning in infants sucking their mothers, who have been poisoned by opium.[7] Elimination being very slow, a portion accumulates in the system and a certain amount may be oxidised into oxydimorphine, which is found in the urine.

Opium is said to withstand putrefaction in the presence of decomposing material. Stevenson detected morphine in the viscera two months after the death of a lady doctor.[8] The Chemical Analyser of Bombay reports a case where opium was detected in the viscera of a body exhumed five months after death.[9] M. Stas detected morphine in the viscera of a body after an interment of thirteen months.[10] Ogier[11] states that he has often failed to detect it in the putrefying viscera after two weeks to one month. The Chemical Examiner of the United Provinces of Agra and Oudh writes in one of his letters to me that " highly decomposed viscera, after being preserved in the usual manner, have shown evidence on analysis of the presence of morphine after 3 to 4 months. It is, however, conceivable that, under certain adverse circumstances, morphine may undergo a change beyond recognition. Cases also are known to happen

1. *Chevers, Med. Juris., Ed. III, p.* 228.
2. *Tardieu quoted by Blyth, Poisons, Ed. III, p.* 554.
3. *Orfila quoted by Taylor, On Poisons, Ed. III, p.* 554.
4. *Amer. Jour. of Med. Scie., Oct.*, 1854.
5. *Taylor, On Poisons, Ed. III, p.* 570.
6. *Anstie, Med. Times and Gaz.*, 1863, *Vol. I, p.* 134.
7. *Brit. Med. Jour.*, 1885, *Vol. II, p.* 1159 ; *Lancet*, 1861, *Vol. I, p.* 93 ; *Med. Times and Gaz.*, 1861, *Vol. I, p.* 70.
8. *Brit. Med. Jour.*, 1903, *Vol. II, pp.* 1105, 1356, 1381.
9. *Annual Report*, 1925, *p.* 4.
10. *Taylor, On Poisons, Ed. III, p.* 35.
11. *Chim. Tox.*, 1889, *p.* 567 ; *Witthaus, Manual of Toxicology, Ed. II, p.* 981.

where in undoubted opium poisoning cases no opium could be detected." A Mahomedan male child, about 5 months old, died of opium poisoning in the King George's Hospital at Lucknow on the 11th August, 1920. The post-mortem examination was held on the 12th August, 1920, 25 hours after death. The viscera were preserved and forwarded to the Chemical Examiner for analysis on the 25th August, 1920. In his letter, dated 13th September, the Chemical Examiner states that no opium or other poison could be detected. Haines[1] also reports a case in which a woman died in about eighteen hours after taking 10 to 15 grains of morphine, but the chemical analysis of the stomach immediately after death did not show the presence of morphine.

Opium Habit (*Opium Eating*).—The habit of taking opium is prevalent throughout India. Ordinarily crude opium is used but, on special festive occasions, *Kasoomba*, its decoction, is offered to the guests. Opium is also smoked in the form of *Madak*, *Chandu*, or opium dross.[2] In order to prevent the smoking of opium which is very much in vogue, especially in Calcutta, the Government of Bengal passed in June, 1933, the Bengal Opium Smoking Act, which provides for the registration of the existing smokers who should obtain a permit from the Excise Department. Any one found smoking without a permit after March, 1934, will be prosecuted and on conviction will have to undergo six months' imprisonment combined with a fine. As a result of the recommendation of the Opium Enquiry Committee in Bengal, since January 1, 1933, the limit of the possession of opium by a person has been reduced from one *tola* to 12 grains. Any one purchasing it in excess of the quantity upto 90 grains, *i.e.*, one and a half *tolas*, must obtain a permit from the Excise Department. These permits are to be issued only on the certificate of a medical practitioner and in no case a quantity exceeding 90 grains is to be sold to any one consumer.[3]

Similar opium smoking acts have also been passed in Bihar and the United Provinces of Agra and Oudh. These acts forbid the registration of habitual opium smokers under the age of 25 years. Recently the majority of the Provincial Governments in British India have declared certain dry areas where the use of intoxicating drugs, especially alcohol, opium and *bhang* (including *ganja* and *charas*) has been prohibited without special permits issued to the addicts on the recommendation of a registered medical practitioner.

An infusion[4] of poppy capsules is habitually drunk by some people in certain districts in the Punjab and parts of Rajputana States, especially Jaipur. A preparation, known as *Bhujri*, and made by frying green, ripe capsules in butter or *ghee* (clarified butter) is eaten by the addicts. A sweet called *Halwa* and prepared from the juice extracted from green poppy capsules is also used.

Opium is believed to increase the duration of the sexual act. Hence it is often taken by young men, who get accustomed to the drug by constant use. It is also used to steady the nerves for doing some bold deed

1. *Hamilton, Legal Medicine, Vol. I, p.* 446.
2. *R. N. Chopra and G. S. Chopra, Ind. Med. Gaz., March,* 1938, *p.* 132.
3. *The Leader, Nov.* 24, 1933.
4. *Chopra, Grewal and others, Indian Jour. of Medical Research, April,* 1930, *p.* 985.

requiring special courage. For instance, in ancient times the Rajputs used to take the drug before they took part in battles.

The morphine habit in Western countries is usually acquired by those who are advised to take the drug either by the mouth or subcutaneously as a remedy for some excruciating pain, as of sciatica. In India the habit is acquired usually by young people of certain classes in consequence of the belief that morphine produces the sense of euphoria and that it has remarkable power as an aphrodisiac. Once the habit is formed, it is difficult to give it up. In fact the victim has to take the drug in a larger dose to combat the feelings of lethargy and mental depression, as the symptoms of the first dose wear off.

It is a well-known fact that opium addicts can easily tolerate much larger quantities of the drug than an ordinary fatal dose. Chopra and Grewal ascertained in their investigations that Sikhs accustomed to opium in Calcutta took it in quantities, varying from 10 to 50 grains, in twenty-four hours.[1] In the Punjab it is not unusual for an addict to take 100 grains of opium a day and continue with it for years.[2] Cases are also on record in which individuals injected hypodermically 15 to 20 or more grains of morphine per day. A case[3] is reported from North-West Frontier Province in which 60 grains of morphine a day were taken by hypodermic injection. It should, however, be remembered that the opium addict may suffer from the symptoms of poisoning by the same drug, if he exceeds his usual limit or if he loses his power of toleration owing to unusual conditions of his system.

Unlike alcohol, opium does not seem to produce injurious effects on the system or to shorten life, if used in moderation ; but its abuse for a prolonged period leads to the derangement of appetite and digestion, disturbance of sleep, vomiting, sluggishness of the bowels, impotence, neurasthenic condition, mental weakness, perversion of morality, premature old age and mania. These symptoms are more evident in morphine eaters than in opium eaters, and are known as *morphinism* or *morphinomania.*

The best treatment for such a condition is the total deprivation of the drug from the patient, but this cannot be achieved without great moral control over one's mind which is not possible in such persons. Again, the sudden deprivation of the drug produces cerebral excitement, restlessness, relaxation of the bowels, pain in the stomach and a burning sensation in the back due to the formation of oxydimorphine, an acrid irritating substance, in the tissues. In order to prevent these symptoms it is advisable to administer lecithin and glucose before opium is completely withdrawn. A pill containing 10 grains of lecithin three times a day is given usually for the first five days and 25 c.c. of 25 per cent glucose are given intravenously each morning for the first three or four days. Glucose may then be administered by the mouth. The diet should consist of fluids only for the first two or three days and then light solids rich in protein and lecithin should be added gradually.[4]

1. *Ind. Jour. of Med. Research, July* 1927, *p.* 57.
2. *Punjab Chem. Exam. Annual Rep.,* 1931, *p.* 10.
3. *R. N. Chopra and G. S. Chopra, Ind. Med. Gaz., July,* 1933, *p.* 369.
4. *R. N. Chopra and G. S. Chopra, Ind. Med. Gaz., May,* 1937, *p.* 265.

CHAPTER XXX

CEREBRAL POISONS—(*Contd.*)

B. INEBRIANT POISONS

ALCOHOL (ETHYL ALCOHOL, ALCOHOL ETHYLICUM), C_2H_5OH

Pure ethyl alcohol is a transparent, colourless, mobile and volatile liquid, having a characteristic spirituous odour and a burning taste. It is very hygroscopic, boils at 78.4° C. (173.1° F.), and burns with a non-luminous flame. It dissolves resins, fats, volatile oils, bromine, iodine, etc., as also many salts and gases. When oxidised, it is converted into aldehyde and acetic acid.

Ethyl alcohol exists in alcoholic beverages in varying proportions. Absolute alcohol contains 99.5 per cent by weight of alcohol, and is used to prepare chloroform and liquor sodii ethylatis. Rectified spirit contains 90 per cent by volume of alcohol, and methylated spirit or denatured alcohol is a mixture consisting of rectified spirit and 10 per cent of wood spirit. Proof spirit is defined by the Act of Parliament as "being such as shall, at a temperature of 51° F., weigh exactly 12/13 part of an equal measure of distilled water." Weaker spirits are termed "under proof" and stronger spirits "over proof".

The following is the percentage of absolute alcohol by weight contained in various alcoholic beverages:—

Whisky	51 to 59 per cent
Rum, Gin and Strong Liqueurs	51 to 59 per cent
Proof spirit	57.09 per cent
Brandy	43 to 57 per cent
Port	20 to 30 per cent
Sherry and Medeira	16 to 22 per cent
Hock	9 to 12 per cent
Claret	8 to 12 per cent
Champagne	10 to 13 per cent
Cider	5 to 9 per cent
Strong Ale or Stout	5 to 9 per cent
Beer and Porter	2 to 5 per cent
Koumiss	1 to 3 per cent

Acute Poisoning.—This may result from inhaling the alcoholic vapours, or from swallowing the alcoholic liquid.

Symptoms.—These are confusion of ideas, muscular in-coordination, giddiness, staggering gait, flushed face, indistinct and foolish speech, and stupor. After a time recovery may occur, accompanied by nausea and vomiting, which are regarded as the early signs of recovery. These may be followed by sleep and severe headache.

If recovery does not occur, the patient passes gradually into unconsciousness and coma with slow, stertorous breathing and a full rapid pulse which then becomes slow and small. The breath smells of alcohol. The patient may be roused temporarily by a loud noise or a violent shake. The pupils are generally dilated, but may be contracted in exceptional cases. Their reaction to light is a hopeful sign. The temperature becomes subnormal. Death usually occurs from asphyxia due to respiratory paralysis, but it may occur from shock due to paralysis of the abdominal nerve centre, if a very large quantity of undiluted alcohol is taken. Sometimes, convulsions precede death. In some cases the patient regains sensibility on account of partial recovery, but a relapse occurs and the patient dies suddenly in a state of coma. He may also die later of pneumonia or œdema of the lungs.

A case[1] is recorded in which a boy, 8 years old, suffered from acute alcoholic poisoning due to the application of surgical spirit to the legs. His legs were shaved and washed with ether soap from the groin to the ankle. They were then covered from the groin to the ankle with a towel wrung out in surgical spirit, over which a dry towel was placed and kept in position by three rubber bands. The first dressing was applied at 12 noon on January 8, 1931, and it was repeated at 4 p.m. and 8 p.m. At 12-30 a.m. on January 9, 1931, he brought up food, but did not speak at all, and his mother thought that he was still asleep. He continued vomiting and retching on and off during the night, but did not wake up. When seen by the nurse first time in the morning, he was unconscious, the pulse rate being 160. The respirations were regular, 24 per minute, and the temperature was normal. At 10-30 a.m. the vomit was found to emit the smell of alcohol. The boy was unconscious, pale and warm, the pupils were small, reacted to strong light, and there was slight internal strabismus of the right eye. The corneal reflex was absent. The limbs were flaccid. There was no sweating. At 1 p.m. the patient was beginning to recover consciousness, but still vomited up small quantities of turbid brown liquid. He could move all the limbs, and complained of frontal headache. The pupils were of medium size and reacted to light. The pulse rate was now 142, the respirations 20 and the temperature 98° F. His bowels had opened involuntarily once in the morning. The vomit and the urine were found to contain alcohol on analysis.

Diagnosis.—Acute alcoholic poisoning has to be diagnosed from opium poisoning, apoplexy, cerebral compression, uræmic coma, diabetic coma and epilepsy.

Fatal Dose.—This is modified according to the habit and age of the patient, and the nature and strength of the liquor taken. Death occurs usually from a large quantity taken in a short space of time. Five fluid ounces of absolute alcohol are considered fatal for an adult. Two ounces of absolute alcohol are probably fatal to a child under 12, though very large doses may be tolerated by habit. Herter[2] reports a case where a child, $3\frac{1}{2}$ years old, accustomed to small drinks of whisky, recovered after taking 12 ounces of pure whisky. Robertson-Milne reports the case of a strong Hindu male, 30 years old, who drank a pint bottle of French brandy and shortly afterwards became unconscious, but recovered completely the next day.[3]

Fatal Period.—The usual fatal period is 12 to 24 hours, though death may occur in a few minutes or may be prolonged for 5 or 6 days. The

1. *Vincent C. James, Brit. Med. Jour., March* 28, 1931, *p.* 539.
2. *New York Med. Jour.*, 1896, *LXIV*, *p.* 608.
3. *Ind. Med. Gaz., June,* 1902, *p.* 208.

shortest fatal period on record is half-an-hour in the case of a man, who died after swallowing a bottle of gin.[1]

Treatment.—Eliminate the poison by emetics or the stomach tube. Apply cold affusions to the head. Maintain the body-temperature by covering the patient with warm blankets. Administer strong coffee with glucose either by the mouth or by the rectum. Wash out the colon with normal saline. Use hypodermic injections of strychnine and camphor. Resort to galvanism, artificial respiration, and oxygen inhalation.

Post-mortem Appearances.—Rigor mortis may last unusually long. Decomposition is also said to be retarded in cases where a very large quantity has been taken, but this is not always the case. The alcoholic odour is perceptible in the stomach, lungs and brain, unless putrefaction has set in. The mucous membrane of the stomach may be red, intensely congested and inflamed, or it may be only pale. The liver, lungs and brain are usually congested. The blood is generally fluid and dark.

The necessary viscera should be preserved in a saturated salt solution for chemical analysis.

Chronic Poisoning.—Habitual drunkards, who have been taking alcohol in one form or another for a long and continued time, suffer from many organic diseases.

The patient suffers from loss of appetite, nausea, vomiting, purging, jaundice, tremor of the tongue and hands, loss of memory, impaired power of judgment, dropsy and general anasarca. The symptoms of peripheral neuritis and dementia supervene in the last stage. Such patients generally die suddenly from coma. After death the gastric mucous membrane shows generally a deep reddish-brown colour with patches of congestion or effusion. The liver is congested and enlarged or cirrhosed and contracted. The kidneys are in a state of granular degeneration.

Delirium Tremens.—The chief important condition from a toxicological point of view is delirium tremens, which results from the long continued action of the poison on the brain. A temporary excess in the case of habitual drunkards is liable to bring on an attack. It sometimes develops in consequence of the sudden withdrawal of alcohol. It also occurs in chronic alcoholics suffering from shock after receiving an injury, such as the fracture of a bone, or from acute disease, such as pneumonia, influenza, erysipelas, etc.

This condition is characterized by disorientation of time and place and a peculiar kind of delirium of horrors owing to hallucinations of sight and hearing. The patient imagines that rats, mice and snakes are crawling on his bed. The patient gets a good deal of muscular tremors, suffers from insomnia and has a tendency to commit suicide or even homicide. Hence he has to be watched closely and carefully day and night.

Delirium tremens is considered unsoundness of mind, and not intoxication.

---

1. *Taylor, On Poisons, Ed. III, p.* 637.

Treatment.—The treatment of delirium tremens consists in the administration of large quantities of fluids, such as barley water with glucose. Irrigate the colon daily and administer warm saline by the rectum. Give potassium bromide in 20- to 30-grain doses. Give hypodermically 1/200 grain of hyoscine hydrobromide. One-twentieth grain of apomorphine hydrochloride may be given with caution.

Chemical Analysis.—Alcohol can be extracted by distillation from an organic mixture. If the organic mixture is highly acid, sodium carbonate should be added to neutralise it.

Tests.—1. Alcohol dissolves camphor.

2. Alcohol gives a deep blue colour when added to a mixture of molybdic acid and strong sulphuric acid heated in a porcelain basin.

3. On heating with caustic potash and iodine a yellow precipitate of iodoform is formed, which is known from its smell and from the hexagonal crystals seen under the microscope.

4. Dilute sulphuric acid and dichromate of potassium give a green colour, and emit the odour of aldehyde.

The following technique[1] is a simple modification of the Widmark test for determining alcohol in the blood and body fluids :—

One cubic centimetre of 0.33 per cent potassium dichromate solution in sulphuric acid (made by dissolving 333 mg. of potassium dichromate in 1 c.c. of water and diluting to 100 c.c. with concentrated sulphuric acid) is spread on the bottom of a 50 c.c. Erlenmeyer flask. Half a cubic centimetre of blood or other fluid supposed to contain alcohol is pipetted into a bit of filter paper and suspended over the potassium dichromate-sulphuric acid solution. The flask is heated at 100° C. for from fifteen to twenty minutes. After cooling, the contents of the flask are made up to 3 c.c. with distilled water. This will require about 1.7 c.c. of water (1 c.c. of the potassium dichromate-sulphuric acid solution + about 0.3 c.c. extracted from the unknown solution + about 1.7 c.c. of distilled water). This mixture is placed in a test tube, 6"$\times\frac{1}{2}$", and is compared with the standards.

The standards are prepared as follows :—

Fifteen test tubes are taken and in each is placed 1 c.c. of the potassium dichromate-sulphuric acid solution. In the first of these tubes is added 2 c.c. of distilled water. In the second is added sufficient alcohol to represent a concentration of 0.05 per cent. In the third, enough alcohol to represent a concentration of 0.10 per cent. In the fourth, a concentration of 0.15 per cent and so on, until the last tube represents a concentration of 0.7 per cent. All the standards are brought up to a total of 3 c.c. each by the addition of distilled water. The standard tubes are now heated to 100° C. for ten minutes. Now by comparing the colour of the unknown solution with the standards, the concentration of the alcohol in the unknown may be determined. The standards, if well sealed and protected from light, will remain accurate for two weeks. The first standard tube will represent a concentration of 0 per cent alcohol, and the last a concentration of 0.7 per cent, with 0.05 per cent gradations lying between.

Medico-Legal Points.—In European countries cases of alcoholic poisoning are very common and are mostly accidental. In India, they are more frequent in big cities than in towns and villages, but fatal cases are very rare. I have seen only two cases of death occurring from acute

1. *Abels, Proc. Soc. Exper. Biol. and Med., April,* 1936, *p.* 346 ; *Jour. Amer. Med. Assoc., July* 24, 1937, *p.* 294,

alcohol poisoning among passengers who were picked up dead from railway trains at Agra Station. Whisky bottles were found in the belongings of both. Probably their death was hastened owing to the excessive heat of the summer. I had also had occasion to hold a post-mortem examination on the body of a Hindu male, aged 30 years, who died from excessive drinking of alcohol in one night on or about the 2nd October, 1933. A case[1] occurred in Bombay where a Parsi, aged 50 years, committed suicide by taking a large quantity of alcohol.

Applied to the skin, alcohol produces redness and irritation, especially if it is prevented from evaporation. It has the power of abstracting water from the tissues and precipitating proteins.

Taken by the mouth, alcohol is quickly absorbed by the stomach and the small intestine, and circulates in the blood. The absorption of alcohol is facilitated if it is swallowed rapidly in a concentrated solution on an empty stomach, and it is delayed if a weaker solution is slowly drunk in the stomach full of food, especially bread and milk. Alcohol reaches its maximum concentration in the blood in about an hour after it is taken, and this concentration is ordinarily proportional to the amount consumed. It disappears very slowly, so that it is found in the blood for about twenty hours after it is drunk.[2] Over 95 per cent of the alcohol ingested is oxidised to carbonic acid and water, but the remaining portion is eliminated unchanged by the lungs and kidneys. It has been ascertained that alcohol appears in the urine within half-an-hour of ingestion.[3]

There is close relationship between the concentration of alcohol in the blood and the degree of alcoholic intoxication. Carter and Southgate[4] have also demonstrated that the concentration of alcohol in the urine is proportional to that of the blood under all conditions, and that a fairly constant ratio holds which enables them to deduce one from the other. Hence it is necessary to analyse the blood or urine for the estimation of alcohol concentration in cases where persons have been accused of being drunk while creating disturbance in streets or driving motor cars. Schweisheimer[5] has shown from experiments that 0.13 per cent alcohol in the blood produces mild intoxication in man and 0.225 per cent produces alcoholic coma. It may be generally assumed that persons with 0.2 per cent alcohol in the blood show symptoms of moderate intoxication, those with from 0.2 to 0.4 per cent are probably drunk and those with more than 0.5 per cent are dead drunk or deeply comatose. When the amount of alcohol approaches 0.6 to 0.7 per cent or more in the blood death usually ensues from asphyxia.

Alcohol acts differently on different individuals and also on the same individual at different times. The action depends mostly upon the environments and temperaments of the individuals and upon the degree of dilution of the alcohol consumed.

1. *The Free Press Jour., April* 15, 1933.
2. *Mellanby, Medical Research Council, Special Report Series, No.* 31.
3. *Carter and Southgate, Transactions, Med.-Leg. Society, Vol. XX, p.* 44.
4. *Transactions of the Medico-Legal Society, Vol. XX, p.* 54.
5. *Deut. Arch. f. Klin. Med.*, 1913, 109, *p.* 271.

In order to ascertain whether a particular individual is drunk or not the medical officer should bear the following points in mind :—

1. The quantity taken is no guide.

2. An aggressive odour of alcohol in the breath, unsteady gait, vacant look, dry and sticky lips, congested eyes, sluggish and dilated pupils, unsteady and thick voice, talks at random and want of perception of the passage of time are the usual signs of drunkenness.

3. Drunkenness does not come within the cognizance of the police, unless the man is dangerous to himself or to his property or that he is annoying or dangerous to others.

A special committee[1] of the British Medical Association was appointed to consider the question of the definition and diagnosis of drunkenness. This committee arrived at the following conclusions and recommendations in regard to persons accused of being " drunk " :—

I. That the word " drunk " should always be taken to mean that the person concerned was so much under the influence of alcohol as to have lost control of his faculties to such an extent as to render him unable to execute safely the occupation on which he was engaged at the material time.[2]

II. That it is desirable that a medical practitioner should base his opinion on the following considerations :—

(*a*) Whether the person concerned has recently consumed alcohol.

(*b*) Whether the person concerned is so much under the influence of alcohol as to have lost control of his faculties to such an extent as to render him unable to execute safely the occupation on which he was engaged at the material time.

(c) Whether his state is due, wholly or partially, to a pathological condition which causes symptoms similar to those of alcoholic intoxication, irrespective of the amount of alcohol consumed.

III. That in the absence of any pathological conditions a person is definitely under the influence of alcohol if there is a smell of alcoholic liquor in the breath and/or in the vomited matter (if any) provided there is a combination of all or most of the following groups of signs or symptoms :—

(*i*) A dry and furred tongue, or conversely, excessive salivation.

(*ii*) Irregularities in behaviour, such as insolence, abusive language, loquacity, excitement or sullenness, and disorder of dress.

(*iii*) Suffusion of the conjuctivæ and reaction of pupils. The pupils may vary from a state of extreme dilatation to extreme contraction and may be equal or unequal.

---

1. *Brit. Med. J., Feb.* 19, 1927, *Supplement, p.* 55.
2. *Under the Road Traffic Act,* 1930, *the word " drunk " was substituted by the phrase " under the influence of drink or a drug to such an extent as to be incapable of having proper control of a vehicle."*

In the opinion of many police surgeons when alcohol in toxic quantity has been consumed, the pupil reflex to *ordinary light* is absent, whereas the pupil will contract in a *bright light* and remain contracted for an abnormally long time, indicating the delayed reaction of the pupil.

(*iv*) Loss or confusion of memory, particularly as regards recent events and appreciation of time.

(*v*) Hesitancy and thickness in speech and impaired articulation.

(*vi*) Tremors and errors of co-ordination and orientation.

IV. That there is no single test by itself which would justify a medical practitioner in deciding that the amount of alcohol consumed had caused a person to lose control of his faculties to such an extent as to render him unable to execute safely the occupation on which he was engaged at the material time. A correct conclusion can only be arrived at by the result of the consideration of a combination of several tests or observations such as :—

General Demeanour ;

State of the clothing ;

Appearance of the conjuctivæ ;

State of the tongue ;

Smell of the breath ;

Character of the speech ;

Manner of walking, turning sharply, sitting down and arising, picking up a pencil or coin from the floor ;

Memory of incidents within the previous few hours and estimation of their time intervals ;

Reaction of the pupils ;

Character of the breathing, especially in regard to hiccup.

V. That the following are tests, upon which taken by themselves, little stress should be laid in deciding whether or not a person is under the influence of alcohol :—

Presence of tachycardia (rapid pulse) ;

Repetition of set words or phrases ;

Character of handwriting ;

Walking along a straight line ;

Failure of convergence of the eyes.

METHYL ALCOHOL (WOOD SPIRIT, PYROXYLIC SPIRIT OR WOOD NAPHTHA), CH_3OH

This is formed by the destructive distillation of wood or molasses. Owing to its peculiar nauseous odour it is used to render rectified spirit unpalatable for trade purposes. This mixture is known as methylated spirit, and is used in arts and manufactures under the name of denatured alcohol.

Symptoms.—The exhilarating effect is rapidly followed by vertigo, nausea, vomiting, abdominal pain, headache, dilated pupils, delirium, intense and persistent

coma and death. If recovery ensues, there is danger of blindness due to optic atrophy.

Fatal Dose.—This varies according to the susceptibility of individuals. One to two ounces have proved fatal.[1] Death has occurred from three, four and five ounces,[2] and often from six to eight ounces, but recovery has followed larger doses. Half-an-ounce[3] has caused blindness, although a much larger quantity has not produced any injury to the eyes.

Fatal Period.—Death may occur from twenty-four to thirty-six hours, or may be delayed for three or four days. In several cases death occurred between six and twenty hours and in one case it occurred in one hour.[4]

Treatment.—The stomach should be washed out with warm water. Hypodermic injections of camphor, caffeine and strychnine should be given. Morphine may be given hypodermically to relieve pain. Gettler and St. George[5] recommend the use of sodium bicarbonate by mouth or intravenously. Intravenous injection of normal saline may be used to promote its elimination.

Post-mortem Appearances.—The mucous membrane of the stomach and duodenum is hyperæmic and inflamed with small haemorrhages. The lungs are congested and œdematous. The brain and its meninges are congested. The mucosa of the bladder is often found congested.

Medico-Legal Points.—Methyl alcohol is less intoxicating than ethyl alcohol, but its after-effects are of longer duration, because it is exidised slowly and is retained in the body for a longer period. It many cases the symptoms appear after a latent period of several hours and days.

Methyl alcohol is eliminated by the breath, but a large portion of it is slowly oxidised to formic acid which is excreted in the urine, the excretion lasting for five or six days.

Owing to its peculiar and powerful odour no one can swallow it unknowingly.

Buller and Wood[6] report that in Canada and the United States poisonous symptoms resulting in blindness or death have frequently occurred from the use of methyl alcohol which is commercially known as "Columbian Spirits," Cologne Spirits and Eagle Spirits. Of 275 poisonous cases collected by them there were 122 deaths.

AMYL ALCOHOL (FUSEL OIL), $C_5H_{11}HO$

This is formed in the manufacture of ethyl alcohol from grain, potatoes and grapes. It is an oily liquid, very slightly miscible with water, and has an unpleasant odour and an acrid taste. Its vapours are more poisonous than the liquid.

Symptoms.—A man became unconscious four hours and-a-half after he swallowed half-a-pint of fusel oil.[7] The face was flushed, the pupils were contracted, the muscles were rigid and the teeth were tightly clenched. The skin was cold, the pulse was almost imperceptible and the respirations were slow and shallow, the breath having an odour resembling amyl nitrite or jargonelle pear. The patient recovered the next day after artificial respiration had been used.

When the vapours are inhaled, the symptoms are irritation to the lungs with headache, nausea, giddiness, choking sensation and inability to stand or walk.

Post-mortem Appearances.—In a fatal case which occurred from "faints," a mixture of amyl and other alcohols, the odour was noticed on opening the body.[8]

1. *Swadener, Jour. Amer. Med. Assoc.*, 1913, 60, *p.* 1479.
2. *Ring, Trans. Amer. Ophth. Soc.*, 1902, 8, *p.* 529.
3. *Raub, Ophthalm. Record*, 1899, 8, *p.* 169.
4. *Peterson, Haines and Webster, Leg. Med. and Toxic., Vol. II, Ed. II, p.* 609.
5. *Jour. Amer. Med. Assoc.*, 1918, 70, *p.* 145.
6. *Brit. Med. Jour., Feb.* 4, 1905, *p.* 262.
7. *Ord, Lancet*, 1889, *Vol. II, p.* 1225.
8. *Swain, Brit. Med. Jour.*, 1891, *Vol. I, p.* 903.

The stomach contained a grumous, bloody fluid and its mucous membrane was soft and thick. The ventricles of the brain were full of fluid which emitted the smell of amyl alcohol.

AMYL NITRITE, $C_5H_{11}NO_2$

This is produced by the interaction of nitrous acid and amyl alcohol that has been distilled between 262° and 270° F. It is a yellowish, volatile liquid, possessing a peculiar fruity and suffocative odour and is insoluble in water. The official dose for inhalation is from 2 to 5 minims.

Symptoms.—By swallowing large doses of amyl nitrite the stomach becomes eroded. The patient complains of a burning pain in the stomach, nausea and vomiting. Later his pulse becomes thready, and he gets convulsions, passes into a state of coma and dies. When inhaled, it causes dilatation of the arteries, flushing of the face, and a sense of fullness about the head. A tea-spoonful taken internally has caused poisonous symptoms.[1]

A retired medical man suffering from anginoid pain inhaled nine amyl nitrite capsules in less than an hour, and suffered from very severe symptoms, but eventually recovered.[2] Cadwallader[3] reports a case in which the inhalation of half-an-ounce of amyl nitrite proved fatal.

Treatment.—Wash out the stomach. Inject strychnine and digitalis hypodermically.

Post-mortem Appearances.—If administered quickly, the lungs and other organs are found blanched and free from blood. The right chamber of the heart is gorged with blood and the left empty. The brain is pale. If administered slowly, the brain is congested and both the chambers of the heart contain blood.[4]

Test.—Heated with caustic potash amyl nitrite forms amyl alcohol and potassium nitrite.

Medico-Legal Points.—Poisoning by amyl nitrite is mostly accidental, and rarely suicidal.

Amyl nitrite taken by the mouth is much less active than when inhaled, as the nitrous acid which is set free by the gastric juice is immediately decomposed. After absorption in the blood amyl nitrite undergoes partial oxidation, and appears in the urine as nitrates and nitrites of the alkalies.

FORMALDEHYDE, HCOH

This is formed when methyl alcohol vapour and air are passed over a red hot spiral of platinum wire. It is a colourless gas, possessing a strong pungent odour. It is soluble in water, a 40 per cent solution being a pharmacopœial preparation, *Liquor Formaldehydi*, commercially known as formalin. It is used as a disinfectant for the fumigation of rooms, as a preservative for pathological specimens, and in the preparation of artificial ivory, celluloid, and horn. It is also frequently used as a preservative for food, especially milk.

Symptoms.—The vapour, when inhaled, irritates the eyes and air-passages and causes painful irritation when it comes in contact with the skin. The liquid solution, when swallowed, produces a burning pain in the mouth, throat and abdomen, vomiting containing blood and mucus, contracted pupils, flushed face, and painful stools. The vomited matter and stools have a strong odour of formaldehyde. There may be suppression of urine. If urine is passed, it contains formic acid. Death may occur from dyspnœa and heart failure. In some cases the narcotic symptoms, *viz.*, giddiness, unconsciousness and stertorous breathing, are more prominent and supervene soon after the solution is swallowed. In a case reported by Moorhead

1. *Brit. Med. Jour.*, 1880, *Vol. II*, *p.* 859.
2. *Water Broadbent*, *Ibid.*, *Nov.* 3, 1923, *p.* 811.
3. *Medical Record*, 1896, 50, *p.* 816.
4. *Blyth*, *Poisons, their Effects and Detection*, *Ed. V*, *p.* 152.

unconsciousness supervened in three minutes after about 3 ounces of 4 per cent formaldehyde were taken.[1]

March[2] reports a case in which a boy, aged 7 years, drank half-an-ounce of commercial formalin in mistake for lemonade. In about 15 minutes he was somewhat collapsed, though quite sensible. He complained of a burning pain in his throat and epigastrium. His pulse was rapid and weak. He had vomited once, bringing up a quantity of clear, greenish fluid, and he was gasping for breath. He improved under the usual treatment, and on the following day he was in his usual health except that he complained of slight pain in the throat, and made an uneventful recovery since then.

Fatal Dose and Fatal Period.—Uncertain. A man, aged 69, died in 20 minutes after taking two to three ounces of commercial formalin.[3] A man, aged 63 years, died in less than 4 hours after he drank about an ounce of formalin containing 34 per cent of formaldehyde with intent to commit suicide.[4] Chooni Lal Bose[5] reports the case of an Anglo-Indian male, 47 years old, who died about 18 hours after swallowing about 3 ounces of 40 per cent formalin solution in a drunken state. On the other hand, recovery has occurred from a dose of 4 fluid ounces of formaldehyde.[6]

Treatment.—Wash out the stomach and administer a dilute solution of ammonia or liquor ammonii acetatis as a direct antidote. These unite with formaldehyde, and form a non-poisonous compound, hexamethylenetetramine, popularly known as urotropine or hexamine.[7] Inject hypodermically strychnine. It may be necessary to resort to artificial respiration.

Post-mortem Appearances.—The mucous membrane of the stomach may be red, inflamed and eroded or may be hard and tough like leather. In the above-mentioned case reported by C. L. Bose the post-mortem examination showed intense congestion of the stomach with erosions and extravasations of blood. The stomach contained half-an-ounce of blood. The intestines were congested. The liver was pale yellow and fatty. The lungs were congested. The chambers of the heart contained blood. The membranes of the brain were congested.

Chemical Analysis.—Formaldehyde may be recovered by distillation. The distillate, when treated with an ammoniacal solution of silver nitrate and gently heated, will produce a beautiful mirror of metallic silver on the inner side of the test-tube.

If 10 drops of a 5% aqueous solution of phenylhydrazine hydrochloride, 1 or 2 drops of a ½% solution of sodium nitroprusside and 10 drops of a 10% solution of sodium hydroxide be added to 2 c.c. of the distillate, a blue colour develops in the presence of formaldehyde. The blue colour changes to green and lastly to yellowish-red.

Medico-Legal Points.—Accidental and suicidal cases of poisoning by formaldehyde have been reported, and a few of them have also been fatal. A case[8] is recorded in which formaldehyde was used externally with criminal intent. A young wife of 15 years of age was severely beaten by her husband and father-in-law, and some quantity of about 31 per cent solution of formaldehyde was poured over her head. The solution caused her great pain and some hours later her hair was found to be falling out in locks and the skin of her scalp to be peeling off.

1. *Brit. Med. Jour.*, Nov. 23, 1912, p. 1470.
2. *Ibid.*, Oct. 15, 1927, p. 687.
3. *Levison, Jour. Amer. Med. Assoc.*, 1904, *Vol. XLII*, p. 1492.
4. *Watt, Brit. Med. Jour.*, Aug. 17, 1912, p. 350.
5. *Ind. Med. Gaz.*, April, 1905, p. 139.
6. *Hale, Jour. Amer. Med. Assoc.*, Feb. 12, 1922, p. 452.
7. *Therap. Montas*, Feb. 1901 ; *Brit. Med. Jour.*, 1901, *Vol. I, Suppl.*, p. 7.
8. *Madras Chem. Examiner's Annual Rep.*, 1931, p. 4.

ETHER (ÆTHER, SULPHURIC ETHER, ETHYLIC ETHER OR ETHYL OXIDE), $(C_2H_5)_2O$

This is prepared from ethyl alcohol by interaction with concentrated sulphuric acid. It is a colourless, mobile liquid, having a peculiar penetrating odour, and a sweetish, pungent taste. It dissolves freely in alcohol, chloroform and fixed and volatile oils, but sparingly in water. It is very volatile and highly inflammable, and its vapour forms an explosive mixture with air or oxygen. Hence it is dangerous to employ ether as an anæsthetic in operations where a naked flame is required or an actual cautery is to be used. An unusual case[1] occurred at Queen Mary's Hospital, Stratford, where a lad, aged 16, had a cycling accident resulting in a fracture of the jaw, and an operation was performed under the anæsthetic of ether and oxygen. In the course of the operation warm air had to be used to keep the patient's teeth dry. It was applied with a dental syringe. On the third application of the syringe an explosion occurred at the back of the lad's throat. Acute hæmorrhage followed, and he died within ten minutes. At the autopsy death was found to be due to rupture of the bronchi and collapse of the lungs; there was no sign of burning in the mouth. At the coroner's inquest it was stated in evidence that the light at which the syringe was warmed was fully six feet away from the operating table, and there was no naked flame near.

Ether is a pharmacopœial preparation, the dose being 15 to 60 minims. Its official preparations are *Æther Anœstheticus* for inhalation as an anæsthetic, and *Spiritus Ætheris*, dose 15 to 60 minims. *Spiritus Ætheris Compositus*, a non-official preparation, commonly known as Hoffmann's anodyne, is often used in medicine in 20 to 40-minim doses. *Injectio Camphoræ Ætheria*, B.P.C., is another non-official preparation, called Curschmann's solution, the dose of which is 4 to 15 minims.

Ether is, sometimes, taken internally as a substitute for alcoholic drinks. The habit of ether drinking has of late become prevalent in some parts of Ireland.[2]

Symptoms.—When swallowed, ether causes a burning pain in the throat and abdomen, and an intense degree of intoxication, which resembles that due to alcohol but is of a shorter duration.

Persons habituated to the use of ether as an intoxicating drink may suffer from chronic gastric troubles and nervous symptoms, such as trembling of the hands, muscular weakness, cramps, headache, palpitation, and ringing in the ears.

When inhaled, ether acts as a general anæsthetic just like chloroform, but its vapour is liable to cause more irritation of the air-passages and more secretion of mucus and saliva. The pulse and breathing become slow, and consciousness is soon lost. An overdose causes death by paralysis of the respiratory centre, but may, in some cases, cause death by failure of the heart, especially if it is diseased. Wilson[3] described

1. *Brit. Med. Jour.*, *Oct.* 7, 1925, *p.* 713.
2. *Hart, Brit. Med. Jour.*, 1890, *Vol. II*, *p.* 885.
3. *Lancet*, *May* 28, 1927, *p.* 1117.

convulsions as a new complication of ether anæsthesia in fatal and non-fatal cases. He investigated these cases and came to the conclusion that the convulsions were toxic in origin and due to the presence of impurities in the ether, such as acetaldehyde and " peroxide ".

Delayed poisoning does not occur after the inhalation of ether.

Fatal Dose.—Two to four fluid drachms, when taken internally, are likely to cause intoxication. One fluid ounce may prove fatal to an adult, although larger quantities can be borne by individuals accustomed to its use.

The concentration of ether necessary to produce anæsthesia reasonably quickly is about 6 per cent by volume or 15 per cent by weight in the inspired air. The concentration of ether reaching 0.14 per cent in the blood is sufficient to cause anæsthesia. When the concentration of ether reaches 11 per cent by volume in the inspired air, there is a distinct danger to life. Inhalation of two and-a-half ounces of ether has caused death.[1]

Fatal Period.—Death may occur at any time during ether administration, or it may occur from pulmonary complications hours or days after recovery from ether anæsthesia.

Treatment.—Lavage of the stomach and cardiac and respiratory stimulants are indicated, if ether has been swallowed. Fresh air, respiratory stimulants, such as ammonia, artificial respiration, inhalation of oxygen combined with carbon dioxide, and strychnine hypodermically are indicated in cases where respiration or the heart's action stops during ether anæsthesia.

Post-mortem Appearances.—The brain is pinker than normal and is slightly œdematous. A strong smell of ether is noticed on opening the thoracic cavity. The trachea contains a little frothy mucus. The lungs are congested, and exude a good deal of œdematous fluid strongly smelling of ether.[2]

In the case of a man who died in about ten minutes from the effects of the vapour the brain, lungs, heart, kidneys and spleen, on section, emitted a strong odour of ether at the post-mortem examination held twenty-two hours after death. The blood was dark, liquid and of a viscid character. The lungs were posteriorly congested, but in the anterior portion the air-tubes were found full of frothy mucus.[3]

Medico-Legal Points.—Accidental poisoning occurs from ether employed as an anæsthetic for general surgery. Ether is, sometimes, swallowed or inhaled for suicidal purposes. It is rarely used for homicidal purpose.

Ether is excreted largely through the lungs and partly through the kidneys.

1. *Holland, Med. Chem. and Toxic., Ed. V, p.* 406.
2. *Gerald Slot, Proceedings of the Royal Society of Medicine, May,* 1929, *p.* 903.
3. *Med. Gaz., Vol.* 41, *p.* 432.

CHLOROFORM (TRICHLOROMETHANE), $CHCl_3$

This is prepared largely by distilling ethyl alcohol, methylated spirit or acetone with bleaching powder. It is a heavy, colourless, volatile liquid, possessing a sweet, pungent taste and a characteristic ethereal odour. When heated, it burns with a green-edged flame, but it is not inflammable at the ordinary temperature. Exposed to air and light, pure chloroform gradually undergoes decomposition, and produces carbonyl chloride (phosgene gas), chlorine and hydrochloric acid, which are very poisonous. The addition of about 1 per cent alcohol and keeping it in a blue or amber coloured, well stoppered bottle prevents such decomposition.

Chloroform is soluble in 20 parts of water, and mixes in all proportions with absolute alcohol, ether, benzene and petroleum spirit. It dissolves fats, caoutchouc, resins, sulphur, phosphorus, iodine, various alkaloids and many other organic compounds. It is a pharmacopœial preparation, the dose being 1 to 5 minims. Its official preparations are—

1. *Aqua Chloroformi.*—1 in 400 of water. Dose ½ to 1 fluid ounce.

2. *Spiritus Chloroformi* (*Chloric ether* or *Spirit of Chloric ether*). 1 to 20 of rectified spirit. Dose 5 to 30 minims.

Tinctura chloroformi et morphinæ composita is a non-official preparation, and is intended to be a substitute for a proprietary medicine, chlorodyne. It contains ¾ minim of chloroform, 1|11 grain of morphine hydrochloride, and ½ minim of dilute hydrocyanic acid in ten minims. The dose is 5 to 15 minims.

Chloroform produces poisonous symptoms, when it is inhaled as a vapour, and also when it is swallowed as a liquid.

Symptoms when inhaled as a Vapour.—For convenience of description the symptoms are divided into the following three more or less distinct stages:—

1. Stage of Excitement.
2. Stage of Depression (Anæsthesia).
3. Stage of Paralysis.

1. *Stage of Excitement.*—As soon as a few whiffs of the vapour are inhaled, the patient experiences a sense of irritation in the throat and fauces, and a burning sensation in the eyes. The face becomes flushed, and a sense of warmth is felt over the whole body, with a creeping sensation in the skin. All the senses except those of sight and hearing are dulled, and the mind becomes confused. At this stage the patient gets delirious, begins to sing, laugh, cry, or use abusive and profane language. Sometimes, he struggles so violently that he is required to be held down by the assistants. The pupils are first dilated, but become contracted as in natural sleep. Frequently there is a tendency to vomit. The pulse and respirations are increased in frequency. This stage lasts rarely for more than four minutes.

2. *Stage of Depression* (*Anœsthesia*).—During this stage the patient becomes completely unconscious and loses all sensibility. The corneal

and other reflexes are lost. The pulse and respirations become slow and feeble. The pupils are contracted. The temperature is sub-normal and the skin is cold and moist. All the muscles are relaxed, and the limbs can be bent in any direction. Surgical operations are performed during this stage, which can be maintained for hours. If the inhalation is stopped, the condition may ordinarily last for twenty to forty minutes, although it may last for a much longer period in some cases. Sometimes, fatal results occur after the withdrawal of the inhalation.

3. *Stage of Paralysis.*—If the inhalation be still continued, the patient passes into the stage of paralysis. The muscular tone is abolished, and consequently the muscles become quite flaccid. The urine and fæces are passed involuntarily. The lips become blue. The surface is cyanosed and bathed in cold perspiration. The pupils are widely dilated. The respirations become slow and irregular with a long pause. The pulse is weak and irregular. Death occurs from stoppage of the heart's action or from respiratory paralysis. It may also occur at any stage, when it may be due to the heart's paralysis or asphyxia brought about by the passage of vomited matter or blood into the air-passages, or by the closure of the glottis from the pressure of the tongue, or possibly by status lymphaticus in the case of children.

Delayed chloroform poisoning occurs about a day or two after recovery from anæsthesia, especially if the quantity administered was large and continued for a long time. It is more common in persons suffering from acetonuria, rickets, hepatic disorders. and wasting diseases, and is especially frequent in children. The symptoms, which resemble those of phosphorus poisoning, are restlessness, violent and persistent vomiting, jaundice, tenderness over the liver, frequent pulse, delirium, coma and death. Sometimes, there may be cutaneous hæmorrhages. The post-mortem examination shows fatty degeneration of the liver, heart and kidneys.

Reichl[1] reports seven cases of late deaths from chloroform which occurred in his clinic in the course of nine days in May, 1925. After a so-called incubation period of 24 to 36 hours the patients became restless with a frequent pulse, somnolence, subicteric discoloration of the sclerotics and skin, delirium and a rise of temperature often as high as 104° F. They rapidly grew weaker, and death followed in 2½ to 3 days in children and in 4 or 5 days in adults. At the necropsy acute yellow atrophy of the liver was found in all the seven cases. The principal post-mortem finding was severe degeneration of the liver, shown by fatty degeneration of the liver cells with necrosis of the centre of the acini.

Fatal Dose.—It is difficult to ascertain the exact lethal dose. Large quantities have been inhaled during surgical operations without any deleterious effects. A concentration of two to three per cent of chloroform in air is the limit of safety for inducing surgical anæsthesia ; whereas a concentration of five per cent or more is considered dangerous. A concentration of 0.035 per cent by weight of chloroform in the blood produces anæsthesia, while a concentration of 0.06 per cent by weight in the blood causes death.[2]

1. *Med. Klin., June* 4, 1926, *p.* 899 ; *Brit. Med. Jour., Aug.* 14, 1926, *Epitome, p.* 23.
2. *Clark, Applied Pharmacology, Ed. V, p.* 164.

The inhalation of 15 or 20 drops of concentrated chloroform has caused death.[1] This was in all probability due to the idiosyncrasy of the patient for chloroform vapour.

Fatal Period.—The usual fatal period is less than ten minutes. The shortest period recorded is one minute after inhalation of thirty drops of chloroform.[2] Death from delayed poisoning occurs from ten hours to twenty days, the average period being four to five days.

Treatment.—Stop inhalation, lower the head and pull out the tongue forward either by a pair of forceps or by carrying the lower jaw forward. Start artificial respiration and apply the faradic current or use oxygen inhalation. Administer hypodermic injections of strychnine, caffeine or ether, and start cardiac massage by the sub-diaphragmatic route. Injection of adrenaline directly into the heart muscle often induces recovery.

The treatment of delayed chloroform poisoning consists in the administration of subcutaneous or intravenous injections of normal saline. Glucose may be administered by the mouth or per rectum to combat the acidosis.

Post-mortem Appearances.—Not characteristic. The brain is usually congested. The lungs are congested and emit the smell of chloroform. Gas bubbles may be found in the blood which is, as a rule, dark and fluid. The heart is often dilated. The liver, spleen and kidneys are, sometimes, congested.

Symptoms when swallowed as a Liquid.—First of all the symptoms of irritation appear and then coma supervenes. The patient complains of a burning pain in the mouth, throat and stomach ; this is followed by vomiting and purging. The vomited matter gives the smell of chloroform, and may contain blood. These symptoms are followed within ten minutes or so by unconsciousness and coma. The pupils are dilated. The surface is cyanosed. The skin is cold and bathed in perspiration. The pulse is feeble, frequent and irregular. The respirations are slow and stertorous. Death occurs from paralysis of the heart's action or respiration. It may, sometimes, occur from pulmonary œdema or gastritis. Cases that recover may show jaundice and enlargement of the liver.

Fatal Dose.—Uncertain. The smallest recorded fatal dose is one drachm in a boy, four years old.[3] Four drachms[4] as also six drachms[5] have respectively proved fatal to adults. Recovery has occurred even after five ounces[6] swallowed with suicidal intent.

Fatal Period.—The usual fatal period is 5 or 6 hours.[7] In one case death occurred in ten minutes[8] and in two other cases life was prolonged to the eighth day.[9]

1. *Taylor, On Poisons, Ed. III, p.* 59.
2. *Husband, Forens. Med., Ed. VI, p.* 390.
3. *Taylor, On Poisons, Ed. III, p.* 648.
4. *Luff, Forens. Med., Vol. I, p.* 240 *quoted by Collis Barry in Legal Med., Vol. II, p.* 473.
5. *Fox, Tr. Louisiani. Med. Soci.,* 1889, *XI, p.* 123.
6. *Dun, Glas. Med. Jour.,* 1898, *p.* 347.
7. *Taylor, Princ. and Pract. of Med. Juris., Vol. II, Ed. IX, p.* 619.
8. *Hamberg, Schmidt's Jahrb.,* 1882, *CXCIV, p.* 250 ; *Witthaus, Med. Juris. and Toxic., Vol. IV, p.* 1167.
9. *Bain, Lancet,* 1859, *Vol. I, p.* 400 ; *Witthaus, Ibid.*

Treatment.—Empty the stomach and wash it out with warm water and milk. Give demulcent drinks, and administer hypodermic injections of strychnine, digitalis, caffeine, atropine, brandy or ether. Give an enema containing whisky. Kep up the body heat by warmth and mustard plaster. Resort to artificial respiration and galvanism.

Post-mortem Appearances.—The mucous membrane of the pharynx and gullet is congested and inflamed. The stomach contents may give off the odour of chloroform. The gastric mucous membrane is red, softened and inflamed, and may show patches of erosion. The lungs are intensely congested. The heart, liver and kidneys may show fatty degeneration.

Chemical Analysis.—Chloroform is easily separated from organic mixtures by distillation with steam. In fatal cases of chloroform poisoning an examination of the blood is very necessary, as chloroform passes rapidly into the circulation.

Tests.—1. The vapour of chloroform, when passed through a red hot exit tube, is split up into chlorine and hydrochloric acid. Chlorine is known by its turning blue a piece of blotting paper moistened with starch and iodide of potassium. Hydrochloric acid gives a white precipitate to a solution of silver nitrate.

2. If *B*-naphthol dissolved in a small quantity of strong potassium hydroxide solution be added to a solution containing chloroform and heated, a blue colour is produced, which becomes green and finally brown.

3. Add an alcoholic solution of caustic potash and a drop of aniline to a mixture containing chloroform and heat. A disagreeable odour is given off due to the formation of isobenzonitrile (phenylisonitrile or phenylisocyanide). The equation representing the result is $CHCl_3 + 3KHO + C_6H_5NH_2 = C_6H_5NC + 3KCl + 3H_2O$. The odour is perceptible when chloroform is present in the proportion of 1 : 5000.

Medico-Legal Points.—Accidental deaths occurring during chloroform anæsthesia must at once be reported to the police, who should investigate into the cause of death for the satisfaction of the public and for exonerating the medical man from any fault or misadventure on his part. The law is not clear as regards the legal responsibility of the anæsthetist or surgeon in such accidental deaths. At any rate the surgeon is certainly responsible, if the anæsthetist happens to be non-qualified. Unfortunately this is usually the case in outlandish branch dispensaries in India.

Chloroform inhalation is occasionally used for suicidal purposes, but more often such deaths are accidental owing to its having been inhaled to relieve pain or to produce sleep. In his annual report for 1907, Rai Bahadur Chooni Lal Bose, Chemical Examiner of Bengal, mentions a case in which an Anglo-Indian woman committed suicide by inhaling chloroform.

Chloroform inhalation has been very rarely used as a homicidal agent. Casper[1] records only two cases. In March, 1856, a Berlin dentist killed his wife, his two children and himself by chloroform. In the second case a quack dentist killed his pregnant sweetheart by chloroform and

1. *Forens. Med., Vol. II, Eng., Tr., pp.* 284, 301.

then shot himself. In April, 1904, G. Hallam was tried at the Central Criminal Court in London for having administered chloroform by inhalation to his two children with intent to murder or do them bodily harm. He was convicted and sentenced to penal servitude for life. It has been frequently reported to the secular press that chloroform vapour is used to facilitate theft or rape, but it is doubtful if any authentic cases have occurred. However, in such cases two questions of medico-legal importance are likely to arise ; *viz.,* (1) whether an individual can be rendered insensible all at once by chloroform inhalation, and (2) whether a sleeping person can be anæsthetised without awaking.

1. *Whether an individual can be rendered insensible all at once by chloroform inhalation.*—Under ordinary circumstances it requires from two to ten minutes to anæsthetise a person with chloroform, properly diluted with air. Hence a person may resist if an attempt is made to chloroform him against his will, unless he is much weaker than his assailant or is overpowered by several assailants and rendered unfit to struggle. On the contrary, death is likely to result, if an attempt is made to render a person suddenly unconscious by a concentrated vapour of chloroform.

2. *Whether a sleeping person can be anœsthetised without awaking.* —It is a fact that operations have been performed on sleeping children after bringing them under chloroform anæsthesia without awaking them, but in the case of adults, it is possible to do so only by skilled and experienced anæsthetists, but that too in a very few cases.

In addition to the patient suffering from toxic symptoms, the anæsthetist and other attendants may be affected by poisonous symptoms resulting in death, if chloroform was used for a long time in an ill-ventilated room lighted by gas burners or lamps.

Owing to its taste and smell, liquid chloroform is rarely given by the mouth as a homicidal poison, though it is, sometimes, taken for suicidal purpose, but more often it is swallowed accidentally.

The Chemical Examiner of Bengal[1] reports the case of a prostitute, who was drugged with chloroform by two persons on the night of the 14th December, 1914. The history of the case showed that she drank liquor with these persons and shortly afterwards fell asleep. When she woke up, she found the visitors had gone and her ornaments were missing. A bottle left in the room was found to contain a small quantity of chloroform scented with essence of roses. In his annual report for 1931, the Chemical Examiner of Bengal also reports a case where a Madrasi male, 35 years old, was found dead in his room. Chloroform was detected in his stomach.

Chloroform is not infrequently swallowed as an intoxicant, and Hofman-Heberda[2] reports that several cases of poisoning occurred among the Russian prisoners during the last Great War by drinking chloroform as a substitute for alcohol.

Elimination.—Chloroform is eliminated mainly by the lungs, and may be detected there some days after death. A small quantity may be excreted in the urine. Chloroform may be resecreted in the stomach, even if introduced hypodermically into the system.

1. *Ind. Med. Gaz., Aug.,* 1915, *p.* 304.
2. *Lehrbuch der gerichtlichen Medizin, 11te. Aufl.,* 1927, *p.* 903 ; *Webster, Leg. Med. and Toxic.,* 1930, *p.* 706.

CARBON TETRACHLORIDE (TETRACHLOR-METHANE), CCl_4

This is a heavy, colourless, volatile, non-inflammable liquid with a chloroform-like odour. It is sparingly soluble in water, but dissolves freely in alcohol and ether. It is extensively used as a solvent for rubber, resins, sulphur and fats, and as a fire extinguisher. It is used as a dry-cleaning agent in the household. It can be used as a general anæsthetic like chloroform, but has twice its toxicity. It is a pharmacopœial preparation, called *Carbonei tetrachloridum*, and is largely used as an anthelmintic in ankylostomiasis in 30 to 60-minim doses. Poisoning has resulted frcm the inhalation and the internal administration of this drug.

Symptoms.—When inhaled, it causes burning pain in the eyes and throat, headache, nausea, sometimes vomiting, mental confusion, loss of consciousness and convulsions. Death occurs from failure of the circulatory and respiratory systems.

Persons employed in rubber works, where carbon tetrachloride is used as a solvent for rubber may suffer from chronic poisoning, which is characterised by irritation of the eyes, nose and throat, deramatitis, and loss of appetite and weight.[1] They also suffer from jaundice and anæmia.[2]

When taken by the mouth, it causes nausea, vomiting, abdominal pain, fine tremors, convulsions, coma and death. Gastric or intestinal hæmorrhages frequently occur.

The symptoms of poisoning are usually delayed for twenty to thirty-six hours after the ingestion of this drug, but Mitra[3] reports a case in which a prisoner, aged 30 years, and of good health, felt nausea one hour after the administration of a medicinal dose of 45 minims, and commenced vomiting in a few minutes. He was seized soon afterwards with colicky pains in the abdomen and was restless. The perspiration started on the forehead, the pulse was soft and slow and the respiration was laboured. An urticarial rash which soon became confluent appeared on the body. Recovery followed a hypodermic injection of 0.5 c.c. of pituitrin.

Fatal Dose and Fatal Period.—Uncertain. A young woman who used it as a hair wash, collapsed and died in a few minutes.[4] A dose of 1.5 c.c. has killed adults, and the administration of 0.18 to 0.92 c.c. has proved fatal to children.[5] A dose of 4 c.c. caused death in 27 hours,[6] and a dose of 3 c.c., in 40 hours.[7] On the other hand, a case is recorded in which a man took 40 c.c. without any symptoms whatever.[8]

Treatment.—If carbon tetrachloride has been inhaled, the patient must be removed at once to the open air, and artificial respiration must be started. Stimulants may be administered afterwards. In cases where the drug has been swallowed, the stomach should be washed out, and a saline purgative should be given immediately. Alcohol, fats or oils should be avoided, but calcium salts should be administered. The injection of 50 c.c. of 1 per cent methylene blue has been recommended.[9]

Post-mortem Appearances.—On opening the body the smell of carbon tetrachloride may be perceptible in the thorax and abdomen. There may be small hæmorrhagic patches in the kidneys and in the gastro-intestinal tract. There may also be inflammation of the small bronchial tubes and necrosis of the liver.

Chemical Analysis.—Like Chloroform, carbon tetrachloride is separated from organic mixtures by distillation. It responds to the isobenzonitrile (phenylisonitrile or phenylisocyanide) test, but not to the *B*-naphthol test. If it is heated with an

1. *Hamilton, Bull.* 179, *U. S. Bureau of Labour Statistics*, 1915, *p.* 32.
2. *A. D. H., Brit. Med. Jour., Sep.* 25, 1920, *p.* 497.
3. *Ind. Med. Gaz., Nov.*, 1928, *p.* 637.
4. *Veley, Lancet*, 1909, *Vol. II, p.* 1162.
5. *Lamson, Minot and Robbins, Jour. Amer. Med. Assoc., Feb.* 4, 1928, *p.* 345.
6. *Sydney Smith, Forensic Med., Ed. VI, p.* 501.
7. *Phelps and Hu, Jour. Amer. Med. Assoc.*, 1924, *Vol.* 82, *p.* 1254.
8. *Lamson, Minot and Robbins, Jour. Amer. Med. Assoc., Feb.* 4, 1928, *p.* 345.
9. *"Toxic Solvents," P. H. Report*, 1938; *Sydney Smith, Forensic Medicine, Ed. VI, p.* 502.

alcoholic solution of potassium hydroxide, it forms potassium chloride and potassium carbonate. If chloroform is similarly treated, it forms potassium chloride and potassium formate.

Medico-Legal Points.—Cases of poisoning by carbon tetrachloride are mostly accidental. Dingely[1] reports a case of poisoning due to the bursting of a patent fire extinguisher. A portion of the chemical came up into the face of the patient who first noticed burning of the eyes, and became unconscious in 45 minutes. The pulse was imperceptible and respiration was suspended. Artificial respiration was carried on for 20 to 25 minutes when the patient commenced to breathe with faint and shallow respirations. He was then given a few ounces of strong tea. His convalescence was uneventful.

When fire extinguishers of carbon tetrachloride are used in a closed room with a high temperature, highly poisonous phosgene gas is formed. It is, therefore, dangerous to use such fire extinguishers in closed rooms.

Khalil[2] has demonstrated that the toxicity of the drug is due to impurities, probably sulphur compounds, which can be got rid of by fractioning the carbon tetrachloride and throwing away the first portion of the distillate (about 1 per cent of the total quantity). Lamson and his co-workers[3] have shown that alcohol and fatty substances should not be given before or soon after the administration of this drug, as they greatly increase the rapidity of its absorption and its toxicity. They have also proved experimentally that calcium deficiency in the organism causes its increased toxicity.

BROMOFORM, $CHBr_3$

This is a limpid, colourless, volatile, sweet liquid, which is soluble in alcohol but almost insoluble in water. It is a non-official preparation, and is administered to children in $\frac{1}{2}$ to 2-minim doses for whooping cough. Being insoluble in water and a heavy liquid, it has a tendency to settle down at the bottom and, if taken without shaking the bottle, it is apt to produce poisonous symptoms. Almost all the cases of poisoning so far recorded have occurred among children.

Symptoms.—These are very similar to those caused by swallowing chloroform; the chief being vertigo, sleepiness, muscular relaxation, contracted pupils, insensibility, stertorous breathing, weak, feeble, irregular pulse, collapse and death.

Fatal Dose.—Three or four minims of bromoform, each administered to two children, aged 2 and 4 years respectively, produced poisonous symptoms.[4] Thirty-six minims of bromoform proved fatal to a girl, aged 5 years.[5] The drug was made up in a mixture with mucilage and water and dispensed in a bottle. It settled down to the bottom, hence the whole quantity was probably taken in the last dose from which she died. On the other hand, recovery has taken place after one and-a-half drachms swallowed by a girl of 6 years.[6]

Fatal Period.—Death occurred in 5 hours in the above-mentioned case.

Treatment.—Wash out the stomach with a solution of sodium carbonate or Condy's fluid. Use hypodermically stimulants, such as ether and strychnine. Apply electricity and start artificial respiration.

Post-mortem Appearances.—Odour of bromoform in the organs. Congestion of the stomach and duodenum.

IODOFORM (TRI-IODOMETHANE), CHI_3

Iodoform occurs as an amorphous powder or as small, lustrous, lemon-coloured hexagonal crystals, having a very penetrating, persistent and disagreeable

1. *Lancet, May* 29, 1926, *p.* 1036.
2. *Lancet, March* 13, 1926, *p.* 547.
3. *Jour. Amer. Med. Assoc., Feb.* 4, 1928, *p.* 345.
4. *Stokes, Brit. Med. Jour., May* 26, 1900, *p.* 1283.
5. *Lancet, Dec.* 31, 1898, *p.* 1816.
6. *T. Brown Darling, Brit. Med. Jour., June* 2, 1900, *p.* 1340.

odour and taste. It is soluble in ether, chloroform and fixed and volatile oils, but sparingly soluble in water and alcohol. The dose is ½ to 3 grains. Iodoform is a constituent of the pharmacopœial preparations, *Suppositorium iodoformi* and *Oculentum iodoformi*.

Iodoform is largely used as an antiseptic and disinfectant in surgical dressings. Poisoning has occurred from its use as a dressing for large, raw, ulcerated surfaces or from the injection of its ethereal solution in chronic abscess cavities, and also from its internal administration. The powdered form is more easily absorbed than the crystalline form. After absorption iodoform is decomposed into iodine and iodides, which are excreted slowly in the saliva, sweat and urine.

Symptoms.—These are giddiness, nausea, vomiting, abdominal pain, skin eruptions, elevation of temperature, dilated pupils, unconsciousness, quick pulse, stertorous breathing, coma and death. In some cases there may be convulsions, hallucinations, delirium and melancholia.

Fatal Dose.—Thirty grains taken internally have proved fatal, though recovery has ensued from larger doses. More than one drachm should not be applied to a wound at a time.[1]

Fatal Period.—Death may occur in one day or after several days. In one case death occurred on the 9th day after the injection of an ethereal solution containing 45 grains of iodoform.[2]

Treatment.—Wash out the wound. Treat the symptoms. Intravenous or subcutaneous injections of normal saline are regarded as beneficial.

If iodoform has been taken internally, the stomach should be washed out and large doses of sodium bicarbonate should be administered.

Post-mortem Appearances.—Œdema of the lungs and acute nephritis. Occasionally fatty degeneration of the heart, liver and kidneys.

Tests.—Warmed with an alcoholic solution of caustic potash, iodoform yields free iodine after it is acidified with nitric acid.

CHLORAL HYDRATE, $CCl_3 CH (OH)_2$

This is a colourless, crystalline substance, having a peculiar, pungent odour and a bitter taste. It is freely soluble in water, alcohol and ether, and forms a liquid when rubbed up with an equal weight of camphor. The pharmacopœial dose is 5 to 20 grains. Syrupus Chloral is a non-official preparation made from it, the dose being 30 to 120 minims. It contains 10.9 grains of chloral hydrate in one drachm.

Acute Poisoning.—This occurs from swallowing a large dose all at once.

Symptoms.—The patient complains of a burning pain in the mouth, throat and stomach immediately after swallowing a poisonous dose, but it is not marked if the drug is administered in a mucilaginous mixture. This is followed by drowsiness, unconsciousness, loss of reflexes, and deep sleep passing into coma. The face is cyanosed, the pulse is slow, feeble and irregular, the breathing is stertorous, the skin is cold with sub-normal temperature and the pupils are contracted. Sometimes, a scarlatinal or urticarial rash may be seen on the skin. Death usually occurs from paralysis of the respiratory centre. In a few cases death may occur from failure of the heart soon after swallowing the drug.

1. *Holland, Med. Chem. and Toxic., Ed. IV*, 391.
2. *Arch. de Med. et. de. Pharm.*, 1890 ; *Collis Barry, Leg. Med., Vol. II, p.* 472.

Chronic Poisoning.—This occurs among persons habituated to the use of the drug in medicinal doses for a long continued period.

Symptoms.—These are those of gastro-intestinal irritation with erythematous and urticarial eruptions on the skin, general weakness and dyspnœa. Clonic convulsions may, sometimes, occur. It has caused insanity and idiocy.

Fatal Dose.—Three grains of chloral hydrate killed a child, one year old.[1] Ten grains proved fatal to a woman, aged seventy years.[2] Twenty grains caused the death of an adult.[3] Thirty grains[4] proved fatal to a woman, aged 20, in thirty-five hours. A man,[5] suffering from delirium tremens, died in 1½ hours after the last of three doses of 15 grains each administered during 7 hours. A healthy woman[6] died from a dose of 60 grains. A girl,[7] 18 years old, died from the effects of two injections containing 40 grains each. On the other hand, recoveries have followed much larger doses. In one instance, recovery occurred after a dose of 430 grains.[8] In another case, a woman, aged thirty-four, recovered after she had taken an ounce of chloral hydrate dissolved in 2 ounces of water.[9] A man, about 35 years old, swallowed no less than 595 grains dissolved in lemon syrup, but recovered in about 3 days.[10]

Fatal Period.—The average fatal period is 10 to 12 hours. The shortest recorded period is 15 minutes after the last of two doses of ten grains each was taken.[11] Thirty minutes is the shortest fatal period recorded after a single dose of 20 grains.[12] Death has been delayed even for three days.[13]

Treatment.—Give emetics or wash out the stomach with warm water. Alkalies may be given to decompose chloral hydrate remaining in the stomach.[14] Keep up the body heat by the use of hot water bottles, blankets, massage, friction and galvanism. Keep the patient awake by flicking the face with wet towels, by shouting at him, or by administering strong coffee by the mouth or by the rectum. Give hypodermic injections of strychnine, caffeine, ether, etc. Perform artificial respiration and administer by inhalation oxygen with carbon dioxide, if necessary.

In chronic poisoning the drug should be withdrawn, and tonics with a liberal diet should be prescribed. It may be necessary to give stimulants, such as strychnine and digitalis.

1. *Phil. Med. and Surg. Rep.*, 1871.
2. *Dixonmann, Forens. Med. and Toxic., Ed. VI, p.* 455.
3. *Hart, Med. Rec.*, 1871-72, *VI, p.* 164.
4. *Fuller, Lancet*, 1871, *Vol. I, p.* 403.
5. *Fordyce, Weekly Med. Rev.*, 1883, *VIII, p.* 94.
6. *Finnell, Med. Record*, 1871-72, *VI, p.* 91.
7. *Ludlow, Med. and Surg. Reporter*, 1888, *LVIII, p.* 596.
8. *Stone, Louisville Med. News*, 1883, *XV, p.* 179.
9. *Colenso, Lancet*, 1894, *Vol. II, p.* 1034.
10. *Daley, Ind. Med. Gaz., Oct.*, 1905, *p.* 401.
11. *Ashbough, Chicago Med. Jour.*, 1877, *XXXIV, p.* 234.
12. *Hart, Loc. Cit.*
13. *Needham, Jour. Psychol. Med.*, 1871, *V, p.* 93.
14. *Dougall, Glas. Med. Jour.*, 1895, *XLIII, p.* 95.

Post-mortem Appearances.—Softening, reddening and erosion of the mucous membrane of the stomach. The lungs, as well as the brain, are congested and gorged with dark fluid blood. Fatty degeneration of the heart, liver and kidneys may be detected in chronic poisoning.

Chemical Tests.—1. A drop of ammonium sulphide added to a weak solution of chloral hydrate imparts an opalescent appearance to the mixture after some time, which subsequently assumes a yellowish or reddish-yellow appearance. This is a more delicate test.

2. Nessler's reagent gives a brick-red precipitate, which becomes lighter in colour and finally greenish-yellow.

3. Caustic potash produces chloroform known by its odour and potassium formate.

4. In organic mixtures it may be detected by distillation with caustic potash or soda and testing the distillate for chloroform by the phenylisonitrile or phenylisocyanide test.

Medico-Legal Points.—Chloral hydrate is often used as a hypnotic in medicine; hence accidental poisoning, followed by death in some cases, has resulted from its internal administration in too large doses. In 1908, a case occurred at Benares in which death resulted from an overdose of chloral hydrate.[1] In 1925, a man, aged 32, who was a victim to the opium habit, went to Lahore from Jullundur and bought some drug in the hope of curing himself of the habit. He took some of the drug and died immediately. The remaining portion of the medicine and the viscera removed from his body showed the presence of chloral hydrate.[2]

Accidental death resulted in one case in three hours after introduction of 5.86 grammes of chloral hydrate into the rectum and in another case in six minutes after injection of 6 grammes into a vein for the purpose of procuring surgical anæsthesia.[3]

Chloral hydrate has been used in a few cases for suicidal purposes. It has not been employed criminally with the intent of causing death but it has been administered with a view to stupefy the victim so as to facilitate the commission of rape or robbery, and has, sometimes, caused death. In October, 1931, a case came to my notice in which an Anglo-Indian administered chloral hydrate in an alcoholic drink to an Indian, and robbed him of his wrist watch and some cash when he became unconscious. 35.9 grains of chloral hydrate were detected in 1.4 ounces of an orange coloured liquid left in a phial found with the accused.

Chloral hydrate is known in certain districts of the Punjab as "Sukka sharab" or dry wine, and is often added to spirit to increase its potency. A party of two died together as the result of a drinking bout. Four deaths from Ludhiana District were recorded in which chloral hydrate was detected in the viscera.[4]

1. *U. P. Chemical Examiner's Annual Report*, 1908.
2. *Punjab Chemical Examiner's Annual Report*, 1925, *p.* iii.
3. *Blyth, Poisons, their Effects and Detection, Ed. V, p.* 167.
4. *Punjab Chem. Examiner's Annual Rept.*, 1929, *p.* 9.

Chloral hydrate is rapidly absorbed from the alimentary canal, and is carried to the central nervous system, where it has a depressing and eventually a paralysing effect. It is also absorbed from the skin. In the tissues chloral hydrate is converted into trichlorethyl alcohol which, combining with glycuronic acid, forms non-poisonous urochloralic acid, and is eliminated in this form in the urine. Some is eliminated unchanged by the kidneys, and some is resecreted in the stomach.

Bromidia.—This is a non-official preparation, a fluid drachm containing 15 grains each of chloral hydrate and potassium bromide ; the dose is ½ to 2 drachms. It is synonymous with *Liquor Bromo-chloral Compositus* of the B. P. C. It is used for procuring sleep and soothing the nervous system.

Accidental poisoning has occurred from its overdose, the toxic effects being chiefly due to chloral hydrate contained in it. Two suicidal cases are recorded, one of which was successful,[1] and the other unsuccessful.[2] Chronic poisoning has also occurred from its continued use for a long time. A woman of 32 years took one-half to one ounce of bromidia for nervousness, sleeplessness and pain for 18 months. She was confused, disoriented as to time, showed loss of memory for recent events and could not fix her attention. She answered voices and felt that people called her bad names, and that they were trying to kill her father and brother.[3]

PARALDEHYDE, $C_6H_{12}O_3$

This is a clear, colourless liquid, having a characteristic ethereal odour and an acrid nauseous taste. It dissolves in 9 parts of water and is soluble in ether and alcohol. It is an official preparation, known as *Paraldehydum*, the dose of which is 30 to 120 minims.

This drug acts chiefly on the cerebrum, inducing a light and natural sleep within ten to fifteen minutes, and is used as a hypnotic in the insomnia of cardiac and respiratory diseases and also in mental diseases. When taken in excess, it may produce acute poisoning.

Symptoms.—These are nausea, vomiting, headache, giddiness, contracted pupils, rapid pulse, unconsciousness, deepening into coma and death from respiratory failure.

Fatal Dose and Fatal Period.—Uncertain. In one case[4] six to seven teaspoonfuls of the drug killed a patient suffering from typhoid fever in four hours, and in another case 2 ounces proved fatal. A case[5] is also recorded where a man, aged 41, who was in the habit of taking one to two teaspoonfuls of the drug, took between 2½ to 3 ounces and went to bed at 11 p.m., and was found dead at 8 a.m. On the other hand, recoveries have followed the ingestion of 3½ ounces[6] and even larger doses.

Treatment.—Give emetics or wash out the stomach. Administer stimulants, such as caffeine, strychnine and digitalis. Resort to artificial respiration and oxygen inhalation, if necessary.

Post-mortem Appearances.—The mucous membrane of the stomach is hyperæmic and may be slightly inflamed. The other viscera are usually congested. There is generally a characteristic odour of paraldehyde when the cavities are opened.

Medico-Legal Points.—Accidental and suicidal cases of acute poisoning, by paraldehyde, though rare, have been recorded. A case[7] is recorded where a rectal injection of half an ounce of paraldehyde with three or four times its volume of water caused considerable sloughing of the rectal mucous membrane.

1. *Lincoln., Jour. Amer. Med. Assoc.*, 1887, *IX*, *p.* 55.
2. *Acker, New York State Jour. of Med.*, 1903, *III*, *p.* 445.
3. *Benett, Jour. Amer. Med. Assoc., Sept.* 23, 1922, *p.* 1048.
4. *Lancet, Vol. II*, 1890, *p.* 423.
5. *J. E. W. Mac Fall, Brit. Med. Jour., Aug.* 8, 1925, *p.* 255.
6. *Mackenzie, Brit. Med. Jour., Vol. II*, 1891, *p.* 1254.
7. *Robert Hutchison, Brit. Med. Jour., April* 12, 1930, *p.* 718.

Persons who take paraldehyde for a prolonged period, become addicted to its use and suffer from the symptoms of chronic poisoning similar to those seen in chronic alcoholism. These are digestive disturbances, muscular weakness, tremors of the hands, disturbance of speech, hallucinations, delusions and delirium.

Paraldehyde is eliminated in the breath and urine to which it imparts its characteristic ethereal odour.

SULPHONAL (DIMETHYL-METHANE-DIETHYL SULPHONE OR SULPHONMETHANE), $(CH_3)_2C(SO_2C_2H_5)_2$

This occurs in tasteless, odourless, colourless crystals or powder, soluble in 450 parts of cold water, in 15 parts of hot water, in 80 parts of 90 per cent alcohol, in 90 parts of ether and in 3 parts of chloroform. It is used in medicine as a hypnotic in 5 to 20-grain doses.

Symptoms.—The symptoms of acute poisoning caused by excessive doses are headache, mental confusion with ataxic gait and thick speech, stupor, insensibility, sometimes convulsions, feeble pulse, irregular and stertorous breathing, subnormal or elevated temperature and marked cyanosis. Death may occur from failure of respiration, or the urine is, sometimes, suppressed, and death may result from anuria. Eruptions may be noticed on the skin after a single large dose.

Sulphonal is not broken down in the body, but is excreted unchanged, the excretion lasting for several days. Hence it may produce chronic poisoning by cumulative effects, even if administered in small quantities for a prolonged period.

Chronic poisoning is characterised by pain in the stomach region, vomiting, constipation, erythematous rashes, headache, muscular weakness, ataxia, confusion of thought and hallucinations. The urine is reddish-brown or port-wine coloured, and contains hæmatoporphyrin, unchanged sulphonal and albumin.

Fatal Dose.—Uncertain. Seventy-five grains may be considered to be fatal to adults. Twenty grains is the smallest quantity that has caused death.[1] On the other hand, recovery has followed a dose of 3 ounces.[2]

Fatal Period.—Uncertain. Seventy hours in one case[3] and thirteen days in another.[4]

Treatment.—Elimination and washing out of the stomach; administration of stimulants; infusion of normal saline, or transfusion of blood.

Post-mortem Appearances.—Reddening and ecchymosis of the stomach and duodenum. Congestion of the liver and other internal organs. Fatty degeneration of the heart, liver and kidneys.

Chemical Tests.—1. Hydrogen sulphide is liberated if sulphonal be heated after adding iron powder and hydrochloric acid.

2. Sulphonal gives off a garlicky odour of mercaptan, if it is heated with charcoal in a test-tube.

Medico-Legal Points.—Accidental cases of poisoning by sulphonal have occurred from large doses or from the injudicious use of the drug by the patients themselves without consulting their physician. A few suicidal cases have also occurred. In November, 1909, an inquest was held by the Manchester City Coroner into the death of a well-known master spinner of Oldham. It was elicited in evidence that the deceased had swallowed in the evening two handfuls of tablets of sulphonal after taking a considerable quantity of whisky with intent to commit suicide. On becoming unconscious he was removed to the infirmary, where he died the following morning. The post-mortem examination showed a considerable number of undissolved tablets of sulphonal in the stomach. The verdict was suicide by sulphonal.[5]

1. *Lancet*, 1899, *Vol. I*, *p.* 811.
2. *Neisser, Deutesche Med. Wochenschr.*, 1891, *XVII*, *p.* 702.
3. *Munchener Med. Wochenschr.*, 1897.
4. *Reinfuss, Wiener Med. Blatter*, 1892; *Dixonmann, Forens. Med. and Toxic., Ed. VI*, *p.* 461.
5. *Brit. Med. Jour., Nov.* 13, 1909, *p.* 1431.

Trional (Methylsulphonal).—It occurs as a white, crystalline powder, with a slightly bitter taste. It is soluble in 320 parts of water and more soluble in dilute alcohol. It melts at 76° C. It is given as a hypnotic in 5 to 20-grain doses. It is similar in action to sulphonal, but acts more rapidly and induces sleep in from thirty to sixty minutes. It has a cumulative action and produces toxic symptoms when taken for a long time. The symptoms and treatment are similar to those of sulphonal poisoning.

Tetronal (Diethyl-sulphone-diethyl-methane).—It occurs in powder or in white crystals, having a camphoraceous bitter taste. It dissolves in 550 parts of water and 12 parts of alcohol. It melts at 85° C. It is used as a hypnotic in 10 to 20-grain doses. It is a dangerous drug and produces poisonous symptoms like sulphonal.

VERONAL (BARBITONE, BARBITAL, HYPNOGEN, MALONUREA, DIETHYL-MALONYL-UREA, DIETHYL-BARBITURIC ACID), $(C_2H_5)_2$ C (COHN)$_2$ CO

This is a white, crystalline powder, having no odour, but a faintly bitter taste. It is slightly soluble in cold water, more soluble in hot water and in 90 per cent alcohol and freely in aqueous alkaline solutions. The official dose is 5 to 10 grains.

Symptoms.—Nausea, vomiting, headache, drowsiness, ataxic gait, stupor deepening into coma, stertorous breathing and rise of temperature. In a case of fatal poisoning recorded by Russell and Parker the temperature rose to 107.2° F., and was brought down to 104.5° F. by cold packs.[1] Death occurs from respiratory failure. The lungs exhibit signs of consolidation and œdema, and may be mistaken for pneumonia. Very often a severe erythematous rash appears on the skin and the face is cyanosed. The urine may be suppressed or scanty, showing the presence of albumin and hæmatoporphyrin. The pupils are usually contracted and insensible to light, but may be dilated. Sometimes, the pupils may be found contracting and dilating alternately at brief intervals.[2] If recovery occurs, headache, dizziness, somnolence, diplopia, ptosis and ataxia may be observed for several days.

Fatal Dose.—Two powders, each containing 10 grains of veronal and ¼ grain of codeine, produced alarming symptoms in a woman.[3] The smallest quantity that has caused death is 15 grains which proved fatal to a barrister in 15 hours.[4] The usual fatal dose is 55 grains for an adult, but recovery has occurred after the doses of 125,[5] and 200 grains.[6] Tardieu and Camps[7] relate a case in which a Russian ex-airman, aged 30, took 360 grains of veronal in a single dose with intent to commit suicide, but he recovered completely after remaining in a comatose condition for five and-a-half days. A case is also recorded in which a Swede took 45 grains of veronal (15 in the morning and 30 in the evening) on the first day, took 90 grains on the second day and on the twelve succeeding days took 30 grains four times in twenty-four hours. During these two weeks he also took, on an average, ten whisky glasses except on the last day when he took only one glass. He suffered from headache, vertigo, marked in-coordination and nystagmoid movements of the eyeballs. The right pupil was larger than the left, both were slightly irregular, but responsive to light and accommodation. He recovered completely in 17 days.[8]

Fatal Period.—Death occurred in four hours and-a-half from a dose of 90 to 105 grains.[9] Death has also occurred in twenty hours and has been delayed for six to seven days.

1. *Brit. Med. Jour., April* 18, 1904, *p.* 853.
2. *Boenheim, Medizinische Klinik, Berlin, Oct.* 16, 1921, *p.* 1263 ; *Jour. Amer. Med. Assoc., Jan.* 7, 1922, *p.* 76.
3. *Lyons, Brit. Med. Jour., Feb.* 2, 1907, *p.* 259.
4. *Ibid., Nov.* 6, 1909, *p.* 1387.
5. *Chitty, Lancet, Mar.* 29, 1913, *p.* 917.
6. *Wells, Brit. Med. Jour., Nov.* 5, 1927, *p.* 826 ; *Sanderson, California and Western Med., S. Fransisco, Decr.*, 1930, *p.* 887.
7. *Bull. Soc. de Ther., Feb.* 13, 1924, *p.* 63 ; *Brit. Med. Jour., Epitome, June* 21, 1924, *p.* 89.
8. *Jerome Littel, Jour. Amer. Med. Assoc., Oct.* 22, 1921, *p.* 1333.
9. *Davis, Brit. Med. Jour., Oct.* 16, 1909, *p.* 1154.

Treatment.—Eliminate the poison from the stomach by washing it out with warm water, and then introduce castor oil and hot coffee. Use hypodermic injections of cardiac stimulants, such as strychnine, digitalis, camphor, and caffeine. Inject subcutaneously warm normal saline and give rectal injections of 15 ounces of normal saline containing 4 per cent glucose. In a case of attempted suicide by 200 grains of barbital recovery ensued after the intravenous injection of 800 c.c. of 20 per cent dextrose.[1]

Strychnine may be given intravenously, the initial dose being 10 mg. The subsequent dosage must depend on the behaviour of the reflexes, notably the jaw reflex. The injections should be repeated even oftener than once an hour. A man of 63 took about 8 grammes of veronal and recovered after he had been given 170 mg. of strychnine intravenously in several doses, 160 mg. having been given in the course of the first twenty-four hours.[2]

The most effective remedy that has recently been recommended is the intravenous injection of one ampoule of 5.5 c.c. of a 25 per cent solution of coramine; this should be followed by an intramuscular injection of another ampoule half to one hour later.[3] An intravenous injection of at least 30 per cent alcohol has also been recommended as an antidote to the barbituric group of drugs.[4]

In respiratory failure intravenous injections of lobeline and inhalations of oxygen with 5 per cent carbon dioxide are indicated. Frequent catheterization must not be neglected. The cerebro-spinal fluid is usually under raised pressure. Hence lumbar puncture should be performed and the fluid allowed to escape until the rate of flow —drop by drop—is almost normal. Intravenous injections of 2½ c.c. of a 0.2 aqueous solution of picrotoxin have been recommended in the treatment of barbiturate poisoning. Owing to its poisonous effects picrotoxin should be used very cautiously and only when a patient is comatose and possibly flaccid.[5]

Post-mortem Appearances.—Externally, there is cyanosis. Internally, the mucous membrane of the alimentary canal is congested. The kidneys show degeneration of the convoluted tubules. The lungs are congested and œdematous and are usually in a pneumonic condition. The other organs are congested.

Chemical Tests.—1. On dissolving the substance containing veronal in caustic potash and adding mercuric nitrate, a thick white precipitate is formed.

2. Nitric acid and Millon's reagent give a white, gelatinous precipitate, soluble in excess.

3. When copper sulphate is mixed with an alkaline solution of veronal, small lilac crystals separate out. The copper sulphate reagent is made as follows:—

Sixteen grammes of potassium bicarbonate are dissolved in 70 to 80 c.c. of water with gentle heating; 10 grammes of potassium carbonate and 8 grammes of copper sulphate are dissolved in the small amount of water and the solution is mixed with the bicarbonate. When the copper carbonate has gone into solution and the evolution of carbon dioxide has ceased, the liquid is made up to 100 c.c. The substance to be examined (about 0.1 gramme) is dissolved in 1 c.c. semi-normal sodium hydroxide, warming if necessary. To this solution 1 c.c. of the copper reagent is added, when in the presence of derivatives of the veronal group a lilac precipitate appears at once.[6]

1. *Sanderson, California and Western Medicine, San Fransisco, Dec.*, 1930, *p.* 877; *Jour. Amer. Med. Assoc., Feb.* 21, 1931, *p.* 642.
2. *Hansen, Nord. Med. Tidsskrift, Sep.* 1, 1934, *p.* 1118; *Brit. Med. Jour., Ep., Dec.* 22, 1934, *p.* 94; *Also* vide *Haggard and Greenberg, Jour. Amer. Med. Assoc., April* 2, 1932, *p.* 1133.
3. *Munchener Medizinische. Wochenschrift, Sept.* 2, 1932, *p.* 1430; *Practitioner, Oct.*, 1932, *p.* 513.
4. *Carrier, Huriez and Willoquet, Bull. de l'acad. de med.*, 1934, *No.* 18; *Lancet, June* 9, 1934, *p.* 1243.
5. *Murphy, Connerty, Connolly and Kopanyi, Jour. Lab. and Clin. Med.. Jan.*, 1937, *p.* 350.
6. *Lindberger, Pharm. Zentralh.*, 69, 32, *p.* 501; *Jour. of State Med., July*, 1929, *p.* 431.

Medico-Legal Points.—Veronal is a powerful hypnotic, and does not leave, as a rule, bad effects behind. Hence it is largely used by patients as a remedy for insomnia without consulting their physician. The result is accidental poisoning from large doses. Sometimes, it has been taken for suicidal purposes. In one case it was accidentally taken in mistake for *kamala*.

Veronal should be prescribed with great caution in renal diseases. Constipation must always be avoided when the drug is being administered, so that the poisonous symptoms may not develop. It is slowly eliminated by the kidneys, so that it may be found in the urine for the first four or five days, but has usually disappeared before ten days have elapsed. It has a cumulative action, and may lead to chronic poisoning if administered for a long time. There is also danger of possible addiction from the prolonged daily use of drugs of the veronal group.

Chronic Poisoning.—The symptoms of chronic poisoning are ataxia, tremors, thick and difficult speech, visual hallucinations and delirium.

Medinal (Barbitonum Solubile, Soluble Barbitone, Soluble Barbital or **Sodium Barbitone).**—This is a mono-sodium salt of diethyl-barbituric acid. It is a white, crystalline powder, soluble in 6 parts of water and possessing a bitter taste. The dose is 5 to 10 grains. It is similar in action to veronal, and produces fatal poisoning in the same way as veronal. The cumulative toxic effects of medinal are the same as those of veronal. The medinal habit (chronic medinal poisoning or *medinalism*) has the same toxic action and produces the same after-effects on the physical health and mental condition of the patients as chronic veronal poisoning. In his annual report for 1933, the Chemical Examiner of Bengal reports the case of a medical practitioner, who took medinal with intent to commit suicide and died on the third day. E. Stolkind[2] describes the case of a man who died thirty hours after taking medinal. The symptoms were smaller pupils not reacting to light, absence of corneal reflex, cyanosed lips, large amount of mucus in the mouth and moist skin, but the extremities were not cold. Breathing was stertorous, and the respirations were at first 24 per minute, and then became 45 per minute. The pulse was weak and regular but became frequent, the number being 150 per minute. The temperature was normal. The patient was in a comatose condition. At the post-mortem examination the lower lobes of the lungs were deeply congested and œdematous, and the spleen was soft.

Luminal (Gardenal, Phenobarbitonum, Phenyl-ethyl-barbituric acid or **Phenobarbital).**—This differs from veronal in that an ethyl group has been replaced by a phenyl radicle. It is a white, odourless powder with a slight bitter taste and almost insoluble in cold water, but soluble in alcohol. It is a more active hypnotic than veronal, and is given in $\frac{1}{2}$ to 2-grain doses, but according to Phillips[3] there is little difference between the therapeutic and the fatal dose, hence it should not be prescribed in single doses of $1\frac{1}{2}$ grains and not more than 3 grains should be taken in twenty-four hours. Luminal forms with sodium hydroxide a soluble salt, luminal-sodium (phenobarbitonum solubile, soluble phenobarbital or luminal soluble). It is an odorous, white, hygroscopic powder, the dose being $\frac{1}{2}$ to 2 grains.

A strong, well-nourished man experienced severe dizziness, nausea, clouded vision, and diplopia after taking a single dose of $4\frac{1}{2}$ grains of luminal.[4] A woman, who took $4\frac{1}{2}$ grains of luminal and another dose twenty-four hours afterwards slept until 10 o'clock the following morning. When she was awakened, she showed evidence of toxæmia, paraphasia, ataxia, inability to stand without support, dilated pupils and diminished knee jerks. These symptoms lasted twelve hours.[5]

Small doses of luminal show a cumulative effect. A woman,[6] aged 45 years, who was suffering from bronchial asthma, was ordered to take $1\frac{1}{2}$ grains of luminal

1. *Il Policlinico, Sez. Med., Nov.* 1, 1926, *p.* 674; *Brit. Med. Jour., Epitome, Feb.* 12, 1927, *p.* 29.
2. *Lancet, Feb.* 20, 1926, *p.* 391.
3. *Jour. Amer. Med. Assoc., April* 22, 1922, *p.* 1201.
4. *Stein, Therap. Halbmontashft, Vol.* 34, 1920, *p.* 387.
5. *Farnell, Jour. Amer. Med. Assoc., July* 19, 1913, *p.* 192.
6. *Phillips, Jour. Amer. Med. Assoc., April* 22, 1922, *p.* 1199.

at bed time. After she had taken 8 doses or 12 grains of this drug over a period of 8 days, the patient developed over the entire body morbilliform eruption. The face was considerably swollen and the conjunctivæ were reddened. The tongue was dry, coated and slightly swollen. The mucous membrane of the mouth and throat was bright red, the tonsils were swollen and the patient complained of a great deal of dryness and burning in the throat. The patient developed pain in the epigastrium with persistent nausea and vomiting which lasted four days. Diarrhœa was not present, but the stools contained some mucus. The urine contained albumin, a few red blood corpuscles, a few leucocytes, a number of epithelial cells and many finely granular casts. The temperature on the first day of the illness was 102° F., on the secoind day 103.6° F., and on the fourth day 105.2 °F., after which it gradually declined to normal. Recovery occurred in about seven days.

Weig[1] reports the case of a woman, 67 years old, who died in 29 hours after a dose of 1 gramme of luminal. A woman, aged 60 years, died after taking fifteen 1½-grain luminal tablets with suicidal intent.[2] In another case death occurred after a dose of 37 grains.[3] Recovery occurred in one case after swallowing 22 tablets of luminal[4] (2 grains in each), in another case after taking a mixture of luminal 10 grains and potassium bromide 50 grains for 12 days,[5] and in a third case after 30 grammes (450 grains).[6]

Luminal is excreted by the kidneys, but a portion is oxidised in the body. A case[7] is recorded in which a woman took 0.1 gramme of luminal for a few days, and her breast-fed child suffered from the symptoms of poisoning owing to the secretion of luminal in her milk.

Dial (Diallyl-barbituric acid or diallyl-malonyl-urea) and Didial, a combination of dial and ethyl-morphine (dionin), are other derivatives of the veronal group. Dial is a powerful hypnotic, the ordinary dose being 1½ to 3 grains. It is about five times as strong as veronal, but it is more rapidly oxidised in the body, and is, therefore, less likely to produce a cumulative effect. Didial is used to induce twilight sleep.

Nichol[8] records a case in which a patient remained in coma for twelve hours after taking about 18 grains of dial and 5½ drachms of paraldehyde, and recovered completely. Dargein and Dore report a case of acute poisoning by 2.7 grammes of dial followed by recovery and another case in which death followed a dose of 7.1 grammes of the drug. In a third case a man, aged 36, swallowed 5 grammes of veronal and 1 gramme of dial, and died after twenty-four hours. In this case the temperature rose to 106° F. before death. At the necropsy generalized congestion of the central nervous system was found, especially of the meninges, with discrete hæmorrhages throughout the brain. There was consolidation of the lower lobe of the right lung.[9]

Amytal, Bromural, Nembutal, Evipan, Proponal, Pernocton, Sodium amytal, Soneryl, etc., are proprietary drugs containing derivatives of barbituric acid and possessing hypnotic properties. Many of these have produced poisonous symptoms when taken in large doses and some have caused death. Six grains of nembutal caused the death of a person suffering from Graves' disease. A young nurse died after taking 75 grains of sodium amytal and 18 grains of nembutal. On the other hand, recoveries have followed the doses of 120 and 156 grains of sodium amytal.[10]

1. *Deutsche Medizinische Wochenschrift, Berlin, Feb.* 13, 1925, *p.* 272; *Jour. Amer. Med. Assoc., April* 11, 1925, *p.* 1159.
2. *White, Jour. Amer. Med. Assoc., April* 28, 1923, *p.* 1261.
3. *Ungar Wein Klin Wochenschr.*, 1914, *Vol.* 27, *p.* 847.
4. *N. C. Hypher, The Practitioner, May,* 1933, *p.* 612.
5. *Carlill, Lancet, Sep.* 19, 1925, *p.* 596.
6. *Boenheim, Medizinisch Klinik, Berlin, Oct.* 16, 1921, *p.* 1263; *Jour. Amer. Med. Assoc., Jan.* 7, 1922, *p.* 76.
7. *Frenedorf, Munchener Medizinische Wochenschrift, Munich, Feb.* 19, 1926, *p.* 322; *Jour. Amer. Med. Assoc., May* 15, 1926, *p.* 1591.
8. *Brit. Med. Jour., Aug.* 16, 1924, *p.* 277.
9. *Bull. et Mem. Soc. Med. des Hop. de Paris, Oct.* 27, 1927, *p.* 1392; *Brit. Med. Jour., Epitome, Dec.* 31, 1927, *p.* 103.
10. *W. J. Bleckwenn and M. G. Masten, Jour. Amer. Med. Assoc., Aug.* 6, 1938, *p.* 504.

ANTIFEBRIN, ANTIPYRIN, AND PHENACETIN

Antifebrin (Acetanilide), C_8H_9NO.—This is a colourless, odourless, crystalline substance, having a slightly pungent taste. It is soluble with difficulty in water but freely in alcohol, wine, ether and coloroform. It is a non-official preparation, the dose being 2 to 5 grains.

"Daisy" or "headache" powders sold in the chemist's shop contain from 4 to 10 grains of antifebrin. Exalgin (methylacetanilide) occurs in colourless crystals, and has a slight saline bitter taste. Dose ½ to 2 grains.

Antipyrin (Phenazone), $C_{11}H_{12}N_2O$.—This occurs in small, colourless, crystals, possessing no odour but a slightly bitter taste. It is freely soluble in water, alcohol or chloroform. Dose 5 to 10 grains.

Phenacetin (Acetphenetidin), $C_{10}H_{13}NO_2$.—This is a colourless, tasteless substance, having scaly crystals. It is very slightly soluble in water, insoluble in glycerine but soluble in 20 parts of alcohol. Dose 5 to 10 grains.

These drugs are used as antipyretics, analgesics and sedatives. Poisonous symptoms have occurred from the administration of doses larger than the medicinal ones. In large doses they destroy the red blood corpuscles, and induce the formation of methæmoglobin, setting it free in the blood plasma. Antifebrin and phenacetin are oxidised in the body to paramidophenol which, in combination with sulphuric or glycuronic acid, is eliminated by the kidneys. Antipyrin is not oxidised in the body, but is excreted in the urine combined with sulphuric acid.

Symptoms.—Vomiting, vertigo, cyanosis, great prostration, slow breathing, quick, irregular and imperceptible pulse, cold, clammy skin, subnormal temperature, collapse and death. Urticarial rashes may appear on the skin, especially in cases of poisoning by antipyrin.

Many persons become addicts through the long-continued use of these drugs, and may suffer from a form of chronic poisoning which is characterized by cyanosis, dyspnœa, weakness, anæmia, wasting, and dark-coloured urine. When these drugs are withdrawn suddenly, they may cause symptoms of acute mania.

Fisher[1] reports the case of a man, aged 47, who took as much as 8 grammes of antifebrin and 3 grammes of phenacetin daily over a long period. The first symptoms complained of were the marked cyanosis of a peculiar lavender hue of the face, weakness of the muscles, coarse tremors of the tongue and hands and some in-co-ordination. The temperature in the mouth varied from 96° F. to 98° F. The pulse rate ranged from 60 to 100 per minute and respirations from 14 to 20. The blood was of a peculiar dark brownish colour due to the presence of methæmoglobin. Marked mental symptoms developed after the withdrawal of the drugs. The patient became confused, disturbed and irrational, and soiled his clothing with urine and fæces. He developed ideas of reference and persecution, but no restraint was necessary. In the course of a week or two the mental symptoms gradually disappeared. Two months after the withdrawal he felt much better, gained 15 pounds in weight and was stronger. The tremors had disappeared.

Fatal Dose.—Uncertain. Three grains of antifebrin have produced insensibility in an infant, five months old.[2] Five grains are supposed to have proved fatal to patients suffering from fever.[3] Seven grains and a half of antifebrin have proved fatal.[4] Three powders, each containing 4½ grains of antifebrin and 0.38 grain of caffeine administered within 2 hours killed a boy, seven years old, in 4 hours.[5] Six powders containing 60 grains of antifebrin taken within a few hours caused the death of a man, thirty-seven years old, in 8 days.[6] A proprietary preparation containing 18

1. *Jour. Amer. Med. Assoc., March* 11, 1933, *p.* 736.
2. *Marenchaux, Deutsche Med. Wochenschr.,* 1889 ; *Dixonmann and Brend, Forensic Med. and Toxic., Ed. VI, p.* 475.
3. *Thomas, Indiana Med. Jour.,* 1890, *Vol.* 9, *p.* 67.
4. *Smedley, Jour. Amer. Med. Assoc.,* 1907, *Vol.* 48, *p.* 1433.
5. *Sanford and Wagman, Jour. Amer. Med. Assoc.,* 1907, *Vol.* 48, *p.* 1693.
6. *Brown, Amer. Jour. Med. Science,* 1901, 122, *p.* 770.

grains of antifebrin has caused death.[1] A single "Daisy" powder killed a woman, aged twenty-two years, in 1 hour.[2] On the other hand, recovery has followed the administration of 120 or more grains of antifebrin.

A dose of 5 grains of exalgin caused complete unconsciousness for 3 hours.[3] Eight grains caused fainting and sense of dying.[4] Six grains taken thrice daily for a week produced alarming symptoms in a woman, aged twenty-four years.[5] Recovery has also followed the administration of 150[6] and 248[7] grains of exalgin.

Five to fifteen grains of antipyrin have produced severe toxic symptoms. Fifteen grains proved fatal to patients suffering from angina pectoris, and 22 grains killed a consumptive patient.[8] Recovery has, however, occurred in the case of a woman, aged twenty-eight years, who took 15 grains of antipyrin four times a day for five days, and a similar dose on the sixth day when she became deeply cyanosed and collapsed. There was also a rash on the body.[9]

Five to ten grains of phenacetin have produced poisonous symptoms. Fifteen grains of phenacetin killed a girl, seventeen years old, in a few hours.[10] Recovery however, occurred after 11 grammes of phenacetin had been ingested by a woman, 48 years old, within 20 hours.[11]

Fatal Period.—Uncertain. Death may occur in a few hours or may be delayed for days.

Treatment.—Eliminate the poison by washing out the stomach and freely administer stimulants, such as digitalis, strychnine, camphor, etc.

Post-mortem Appearances.—Not characteristic.

Chemical Tests.—*Antifebrin.*—1. A play of colours from red changing to brown and then to green on the addition of a crystal of chromate of potassium and strong sulphuric acid.

2. It does not give any reaction with ferric chloride.

3. Heated with a solution of caustic potash, it is decomposed into aniline and potassium acetate.

Antipyrin.—1. A solution of ferric chloride produces a blood-red colour, destroyed by a mineral acid.

2. A mixture of potassium nitrite or sodium nitrite and sulphuric acid gives a green colour.

3. Heated with bleaching powder, it gives a brick-red precipitate.

Phenacetin.—1. Sulpho-vandalic acid produces an olive-green colour, turning to black on the application of heat.

2. Sodium persulphate produces a yellow colour on heating, and an orange colour on continued boiling.

3. To a mixture of equal parts of phenacetin and sodium nitrite, add a drop of strong sulphuric acid and heat on the water bath; it will turn green.

1. *Easley, Amer. Pract. and News*, 1891, *Vol.* 12, *p.* 178.
2. *Pharm. Jour.*, 1896, *Vol. II, p.* 14.
3. *Crookshank, Lancet*, 1895, *Vol. I, p.* 1307.
4. *Brit. Med. Jour.*, 1899 *Vol. I. p.* 1518.
5. *Bokenham and Jones, Brit. Med. Jour.*, 1890.
6. *Bell, Lancet*, 1899, *Vol. II, p.* 890.
7. *Weber, La Semaine Med.*, 1894; *Dixonmann and Brend, Forens. Med. and Toxic., p.* 475.
8. *Lewin, Berlin, Klin. Woch.*, 1895, 32, *p.* 727; *Nebenwirkungen d. Arzneimit*, 1899, *p.* 459; *Peterson, Haines and Webster, Leg. Med. and Toxic., Vol. II, Ed. II, p.* 740.
9. *Rapin, Revue Med. de la Suisse rom.*, 1888; *Dixonmann and Brend, Forens. Med. and Toxic., Ed. VI, p.* 476.
10. *Vereins-Beilage der Deutsch. Med. Wochenschr.*, 1895; *Dixonmann, Forens. Med. and Toxic., Ed. VI, p.* 476.
11. *Ann. de la Soc. de Med. de Grand*, 1905; *Ind. Med. Gaz., Jan.*, 1906, *p.* 32.

Medico-Legal Points.—Most of the poisonous cases have been accidental from overdoses or even from medicinal doses, especially if the heart happens to be diseased.

Antifebrin has produced fatal symptoms from its application as an antiseptic dressing to raw surfaces. Snow mentions the case of an infant who was thus poisoned after the drug had been used as a dusting powder for the unhealed umbilicus.[1]

It is reported that four drachms of antipyrin were used subcutaneously as a last resort by Clark of Agra to murder Fulham, after he had been unsuccessfully drugged with arsenic, gelsemine and probably with cocaine and belladonna.

CINCHOPHEN (PHENYLQUINOLENE CARBOXYLIC ACID)

This is a yellowish, cream-coloured, amorphous powder, insoluble in water and slightly bitter in taste. It is a pharmacopœial preparation and is known as atophan, phenoquin, agotan, atocin, nylofanol, etc., and is given in 5 to 15-grain doses as an analgesic in lumbago and sciatica. It is also said to increase the elimination of uric acid from the blood in gout and rheumatic affections.

The drug is a dangerous poison and should be used with great care. Small doses administered for a prolonged period may produce chronic degenerative changes in the liver, while large doses may cause acute fatty degeneration, or even acute yellow atrophy, of the liver.

The symptoms of poisoning are general malaise, headache, gastro-intestinal disturbance, urticaria, urobilinuria, albuminuria and jaundice.[2] Death has occurred in some cases from necrosis of the liver.

The treatment consists of the withdrawal of the drug and administration of dextrose and insulin. The drug has a cumulative effect, hence there should be frequent rest periods during its administration.

A man, aged 52, who received 118 grammes of atophan in 41 days on account of chronic rheumatism, recovered gradually after he was given 60 grammes of dextrose and 20 units of insulin twice daily. Later on, duodenal lavage with magnesium sulphate was instituted.[3]

SULPHANILAMIDE (PRONTOSIL)

This is a term adopted by the American Council of Pharmacy and Chemistry as a nonproprietary name for para-aminobenzenesulphonamide. It is a white, odourless, crystalline substance, is slightly bitter with a sweetish after taste and is but slightly soluble in water and alcohol. It is a chemotherapeutic agent and was originally intended for use in hæmolytic streptococcal infections, but is now largely used in the treatment of erysipelas, puerperal sepsis, tonsillitis, peritonitis, meningitis, gonorrhœa, pneumonia, otitis media and osteomyelitis.

The derivatives of sulphanilamide are sold under different proprietary names, such as Prontosil Soluble, Prontosil Album, Proseptasine, Soluseptasine, M and B 693 (Sulphapyridine), Bacteramide, Streptocide, Sulphonamide-P, etc.

Sulphanilamide is generally administered by the mouth, the daily dose for an adult being 1 gm. for each 20 lb. of body weight, but the total dose for a day should not exceed 5 gm. The drug may be administered hypodermically or intramuscularly. It may also be given per rectum or intrathecally in an 0.8 per cent solution in normal saline. The treatment should be continued only for two to three weeks and the dose should be reduced as the condition improves.

The administration of the drug for a prolonged period or in fevers of uncertain nature usually gives rise to toxic effects and may cause death. Owing to idiosyncrasy poisonous symptoms may appear in some cases after the administration of an ordinary therapeutic dose. It should be remembered that children bear it well.

1. *Arch. of Pediatrics*, 1897.
2. *Worster-Drought, Brit. Med. Jour., Vol. I*, 1923, *p.* 148.
3. *K. Eimer, Deut. Med. Woch., Sept.* 25, 1931, *p.* 1663; *Brit. Med. Jour., Ep., Nov.* 14, 1931, *p.* 85.

Symptoms.—These may be classified as mild and severe. The mild symptoms consist of general malaise, headache, anorexia, vertigo, nausea, slight cyanosis and dyspnœa. The severe symptoms are abdominal pain, diarrhœa, numbness and tingling of the face, hands and feet, skin eruptions, fever, acidosis, cyanosis, methæmoglobinæmia, or sulphæmoglobinæmia and agranulocytosis.

Young[1] reports a case where a man, aged 53, who was given daily 3 gm. of prontosil album for 18 days for an acute rheumatic infection, developed agranulocytosis and died on the 23rd day. The post-mortem examination revealed complete myeloid aplasia.

Treatment.—Sodium bicarbonate should be given to prevent acidosis. Methylene-blue in doses of 1-2 mg. per kilogramme of the body weight should be given intravenously in cases of cyanosis. It also prevents the formation of methæmoglobinæmia when administered with sulphanilamide.[2] Blood transfusion is recommended when there is danger to life.

Patients should be kept in bed during the course of sulphanilamide therapy and should be watched daily by their physician, who should do the white blood cell count at frequent intervals. Magnesium or sodium sulphate should be avoided, as its administration concurrently with, or within two or three days preceding, administration of sulphanilamide gives rise to sulphæmoglobinæmia.[3] Coal-tar derivatives should also be avoided during the treatment with this drug. Low residue diet should be prescribed and liquid paraffin should be administered daily to keep the bowels free.[4]

ANILINE (MONO-PHENYLAMINE OR ANILINE OIL), $C_6H_5NH_2$

This is a coal-tar derivative, and is prepared by reducing nitrobenzene by means of nascent hydrogen. It is a colourless, oily liquid, becoming brown on exposure to the air. It has a peculiar aromatic odour and a burning taste. It is soluble with difficulty in water, but freely in alcohol, ether and chloroform. It is chiefly used in the arts for making several aniline dyes. It is also a basis of some synthetic drugs, such as phenazone and exalgin. Commercial aniline contains aniline, toluidine, nitrobenzene, and other benzene derivatives.

Symptoms.—These usually appear immediately after swallowing a poisonous dose, but may, sometimes, be delayed for an hour or more. The symptoms are nausea, vomiting, headache, giddiness, drowsiness soon deepening into coma, slow laboured breathing, small, feeble and irregular pulse, and remarkable cyanosis of the lips, face, fingers and toes, and sometimes of the whole body, largely due to the formation of methæmoglobin. The skin is cold and clammy; the pupils are usually dilated, but are contracted in some cases. Very often convulsions occur before death.

A boy[5] aged 12, swallowed at 9-45 a.m. in a school some distilled aniline from a flask which contained less than 2 c.c. of a mixture of aniline and water. At 12-30 he took train to his home and arrived at his station without mishap, but collapsed outside at about 12-45, and was carried home. He was then unconscious, the finger tips were blue and the lips almost black. He vomited continuously from 2-30 onwards and at 3 o'clock was roused enough to say that he had taken aniline but immediately relapsed into unconsciousness. At 10 p.m. he was lying peacefully in bed with his eyes shut. He was very drowsy but could be roused with difficulty when he would open his eyes but would not answer questions. The skin was a pale lilac colour all over except that the lips, ears, tongue, and the nails of the fingers and toes were of a deep leaden blue colour. The respirations were slow and very shallow. The pupils were dilated and reacted sluggishly to light. Recovery occurred under oxygen inhalation, although headache persisted for three days.

1. *Brit. Med. Jour., July* 17, 1937, *p.* 105.
2. *Hartmann, Perley and Barnett, J. Clin. Invest.,* 1939, 17, *p.* 699; *Lancet, Feb.* 18, 1939, *p.* 403.
3. *Paton and Eaton, Lancet, May* 15, 1937, *p.* 1159.
4. *Archer and Briscombe, Lancet,* 1937, *Vol. II, p.* 432.
5. *J. Inkster, Lancet, Oct.* 9, 1926, *p.* 752.

Fatal Dose.—Six drachms[1] have proved fatal, but a smaller dose may cause death.

Fatal Period.—Uncertain. Seven hours in a case,[2] where a man had poisoned himself with aniline. Twelve hours in a second case after swallowing 3 ounces of marking-ink consisting chiefly of aniline,[3] and 2 days in a third.[4]

Treatment.—Give emetics or wash out the stomach as quickly as possible. Administer stimulants hypodermically or per rectum. Inhalation of oxygen and artificial respiration. Venesection, saline infusion and transfusion of blood may be necessary in severe cases.

Post-mortem Appearances.—Not characteristic. Hyperæmia and congestion of the bronchial tubes, as well as the stomach. The blood is chocolate coloured. In the case[5] where a man died within seven hours, the post-mortem appearances were vacuolation and swelling of the ganglion cells of the cortex and in the wall of ventricle with perivascular hæmorrhages.

Chemical Tests.—1. A solution of calcium hypochlorite added to an aqueous solution of aniline produces a deep purple colour, changing to brownish-red.

2. If a drop of strong sulphuric acid be mixed with a drop of aniline on a porcelain slab, a dirty white mass is formed. On the addition of water and potassium dichromate, it acquires a blue-violet colouration, which rapidly changes to blue and then to black (blue changing to purple and red in the case of strychnine).

3. Heated with chloroform and alcoholic potash the offensive odour of phenylisocyanide is noticed.

Medico-Legal Points.—Aniline is a blood poison. It disintegrates the red blood corpuscles and causes the formation of methæmoglobin, which may be readily recognised by its characteristic spectroscopic appearance. Engelhardt[6] has shown that aniline is partly changed in the human body into aniline black. In severe aniline poisoning fine blue-black granules may be seen in every drop of the blood and also in the urine. Aniline is oxidised in the tissues to para-amino-phenyl-sulphuric acid, which is then eliminated in the urine as an alkaline salt. A part of aniline may be found unchanged in the urine.

Aniline is occasionally taken internally for the purpose of committing suicide, but does not seem to have been used for homicidal purposes. Kreuser[7] mentions that workmen exposed to the vapour of aniline suffer from dry spasmodic bronchial cough, and ulceration on the scrotum and extremities. Poisonous symptoms have also occurred from its absorption through the unbroken skin. Rayner[8] relates a case where newly-born infants in a work house showed the blue colouration of the lips, gums and palate from the use of napkins stamped with marking-ink containing aniline chloride. The aniline was absorbed by the vulvæ and buttocks. A similar instance is quoted by Landouzy and Brouardel, where ten children suffered from poisonous symptoms after wearing boots, which had been covered with a yellow pigment containing 90 per cent of aniline.[9] Arthur J. Pathek[10] also reports three cases in which a girl, aged 13, and two brothers, aged 11 and 13, suffered from poisonous symptoms after wearing shoes dyed with a colour which contained aniline. Henry H.

1. *Muller, Deutsche Med. Woch.*, 1887 ; *Collis Barry, Leg. Med., Vol. II, p.* 484.
2. *Raschewskaja, Deuts. Zeits. f. d. ges. gerichtl. Med.*, 1932, *XIX, pp.* 23-25 ; *The Med.-Leg. and Criminolog. Rev., Vol. I, Part I,* 1933, *p.* 81.
3. *Smith, Lancet, Jan.* 13, 1894, *p.* 89.
4. *Ibid., Dec.* 7, 1898.
5. *Raschewskaja, Loc. Cit.*
6. *Contributions to the Toxicology of Aniline, Franz. Diss. Dorpat,* 1888 ; *Warren, Autenrieth's Detection of Poisons, Ed. VI, p.* 72.
7. *Edin. Month. Jour., Aug.*, 1864, *p.* 172.
8. *Brit. Med. Jour., Vol. I,* 1886, *p.* 294.
9. *Bulletin de l' Academie de Medecine ; Brit. Med. Jour., Sep.* 29, 1900, *p.* 946.
10. *Jour. Amer. Med. Assoc., March* 27, 1926, *p.* 944.

Haft[1] reports a case of poisoning by shoe dye, in which a youth, aged 19, complained of frequency of micturition with dysuria and the passage of very bloody urine.

Chronic poisoning occurs among those who are exposed to its fumes in industrial arts. The symptoms are eczematous ulcerations, cough, nervous symptoms and blindness.

It is mentioned in the Extra Pharmacopœia of Martindale and Westcott that aniline sulphate should be administered cautiously from ½ to 3-grain doses, but it does not seem to be poisonous, inasmuch as 406 grains of the drug were taken by a patient in a few days without exhibiting any untoward symptoms.[2]

COAL-TAR NAPHTHA

Coal-tar Naphtha is a term generally applied to the first distillates when coal-tar is distilled. It is inflammable, and has a most disagreeable smell.

Symptoms.—Inhaled as a vapour, coal-tar naphtha produces headache, giddiness, difficulty in speech, irritation of the respiratory tract, and broncho-pneumonia.

Taken internally, it produces burning pain in the mouth, throat and stomach, vomiting, thirst, colic, restlessness, shallow respirations, weak pulse, insensibility, collapse and death.

A boy,[3] aged 12, swallowed inadvertently about three ounces of coal-tar naphtha and suffered from delirium, vomiting, stertorous breathing, cold, clammy skin, contracted pupils, and insensibility and died from collapse in less than three hours. At the post-mortem examination held on the fourth day after death a strong odour of naphtha was perceived throughout the tissues. The stomach contained a pint of semi-fluid matter from which an ounce of a dark coloured liquid was separated possessing the characteristic properties of naphtha; viz., insolubility, lightness and inflammability.

A girl, aged 5, who had taken from between two to three ounces of coal-tar naphtha, recovered from the immediate symptoms, but died from acute bronchitis on the 11th day.[4]

Treatment.—Emetics or washing out of the stomach with warm water; purgatives, especially magnesium sulphate; stimulants and artificial respiration if necessary.

NAPHTHALENE (NAPHTHALIN, TAR CAMPHOR), $C_{10}H_8$

This is a hydrocarbon contained in the middle oil distillate of coal-tar. It occurs in large, lustrous crystalline plates, having a persistent odour. It melts at 79° C., boils at 218° C., but sublimates at a lower temperature. It is insoluble in water, but dissolves freely in ether, chloroform, alcohol and oils.

Naphthalene is chiefly used in the manufacture of indigo and certain azo-dyes, as a repellant to moths and as a deodorant in closets. It is used in medicine as an intestinal disinfectant and as a vermifuge, the dose being 3 to 12 grains.

Symptoms.—Taken internally, naphthalene produces headache, nausea, vomiting, abdominal pain, staggering gait, pain on micturition with dark brown urine containing albumin and hæmoglobin, drowsiness, muscular twitchings, cyanosis, coma and death.

A Mahomedan male, who took some naphthalene in place of an Indian sweet, suffered from severe jaundice, marked anæmia, hyperthermia, hemiplegia and coma. He died three days after swallowing the poison. It is possible that, in the metabolism of naphthalene, naphthylamine (an amino-derivative) was formed and was responsible for the rise of temperature to 103° F.[5]

1. *Jour. Ame. Med. Assoc., March* 10, 1928, *p.* 742.
2. *Lethby, Proc. Roy. Soc.*, 1863, *p.* 563; *Med. Times and Gaz.*, 1862, *I, p.* 239.
3. *Lancet, Vol. II*, 1856, *p.* 230.
4. *Lancet, Vol. I*, 1901, *p.* 245.
5. *N. R. Konar, H. K. Roy and M. N. De, Ind. Med. Gaz., Dec.*, 1939, *p.* 723.

Inhaled as a vapour, it causes chiefly malaise, headache and vomiting. Inhalation of the vapour for a prolonged period may produce chronic poisoning. Evers[1] records a case where persons sleeping under bedclothing dusted over with naphthalene as a moth powder suffered from loss of appetite, headache and eczema of both legs. Lutz[2] also records three cases of chronic poisoning by naphthalene vapour, in which the symptoms were headache, mental depression, digestive disturbances with irritation of the bladder and olive-green colour of the urine.

Fatal Dose and Fatal Period.—Not known. Seven grains[3] of naphthalene have produced severe symptoms of poisoning. A boy,[4] 6 years old, died in two days after taking 1.75 grammes of naphthalene in seven doses as an anthelmintic. A case occurred to Dr. Vyas, in which a boy of 2 years died on the third day after he had swallowed a naphthalene ball (moth ball) weighing about 40 grains. In this case the symptoms supervened two days after swallowing the ball when castor oil was administered. The patient soon collapsed, and became comatose, with dilated pupils. The urine contained albumin, blood and hyaline and epithelial casts. A boy,[5] 12 years old, who had eaten two naphthalene camphor tablets ("bon-bons") each containing 2 grammes of pure nephthalene, suffered from the symptoms much resembling those of alcoholic intoxication. Recovery occurred on the fifth day.

Treatment.—Wash out the stomach and administer purgatives, since the drug is absorbed slowly. Avoid fats and castor oil, which dissolve it.

Post-mortem Appearances.—Jaundice and acute nephritis may be present.[6] The gastric mucous membrane may be congested and inflamed. The other viscera are congested.

The post-mortem examination of the case of the Mahomedan male quoted above showed that the skin was yellow. The brain and spinal cord were stained yellow. The larynx and trachea contained frothy mucus which was stained yellow. The lungs were congested and exuded yellow froth on section. The heart contained fluid blood in both the chambers. The peritoneum was stained yellow. The mucous membrane of the gastro-intestinal tract was yellow. The spleen was congested. The liver was soft and pale and showed patchy necrosis chiefly in the central zone. The gall bladder was full of bile. The kidneys were pale. Naphthalene was detected in the urine.

Chemical Analysis.—Naphthalene may be separated by distillation with steam and extracting the distillate with ether. The ethereal solution thus obtained forms a yellow crystalline compound with picric acid.

Medico-Legal Points.—Accidental cases of poisoning by naphthalene have occurred from the inhalation of its vapour, from its internal administration or from its application to wounds,[7] and among children who had swallowed naphthalene balls.[8]

Naphthalene is oxidised in the tissues to beta-naphthol, which is then excreted in the urine in combination with glycuronic and sulphuric acids.

BENZENE (BENZOL), C_6H_6

This is one of the constituents of coal-tar naphtha, and is obtained by the fractional distillation of the latter. It is a colourless, volatile liquid, and has a suffocating, disagreeable odour, resembling that of coal-gas. It is highly inflammable, and gives off a vapour which is explosive when mixed with air. It is insoluble in water, but mixes with alcohol, chloroform and ether. It is used in "dry cleaning",

1. *Berliner klin, Wochenschr.*, 1884, *Vol. II, p.* 593.
2. *Verhandl. d. Soc. Scientif., S. Paulo*, 1906; *Dixonmann and Brend, Forens. Med. and Toxic., Ed. VI, p.* 477.
3. *Dixonmann and Brend, Forens. Med. and Toxic., Ed. VI, p.* 477.
4. *Prochownik, Therap. Montash.*, 1911, 25, *p.* 489.
5. *Zangerle, Therap. Montash., Feb.*, 1899, 13, *p.* 122.
6. *Heine, Med. Klinik*, 1913, 9, *p.* 62.
7. *Fromuller, Memorabilien*, 1883, *p.* 257.
8. *Nash, Brit. Med. Jour., Vol. I*, 1903, *p.* 251.

and is also used extensively as a solvent for India-rubber. It is a narcotic poison which, when inhaled or swallowed, produces toxic symptoms.

Symptoms.—When inhaled as a vapour, the symptoms are dizziness, flushing, ringing in the ears, unconsciousness, cyanosis, dyspnœa, stupor and death. When the vapour is inhaled in a concentrated form, coma may supervene at once, and death may result in a few minutes. A concentration of 19,000 parts per million of the atmosphere is sufficient to cause death.

When taken by the mouth, the symptoms are a burning pain in the stomach, giddiness, flushing of the face, restlessness, excitement, dilated pupils, rapid and feeble pulse, slow and laboured respiration, cold, clammy skin, stupor, coma and death from respiratory failure. Twitchings of the muscles, convulsions, hallucinations and delirium may occur in some cases.

Fatal Dose.—The medicinal dose is 5 to 20 minims. Three or four drachms have produced toxic symptoms,[1] while one ounce has caused death.[2]

Fatal Period.—Ten minutes in the case of a child, 2 years old, who swallowed a mouthful of benzene.[3] A woman, 26 years old, who took one ounce of benzene, died in twelve hours.[4] Another woman, aged 26 years, died in 50 hours after swallowing benzene.[5]

Treatment.—When the vapour is inhaled, the patient should at once be removed to the open air, and artificial respiration should be performed. Oxygen inhalation and restoratives should be used, if necessary.

When it is swallowed as a liquid, the stomach should be washed out, and stimulants, such as ether and strychnine, should be injected hypodermically. Galvanism, artificial respiration and oxygen inhalation should be used, if necessary.

Post-mortem Appearances.—Purpuric spots may be visible on the skin.[6] An odour like that of coal gas emanates from the body cavities. Hæmorrhages in the mucous membranes. Hyperæmia of the stomach and other organs. Œdema of the lungs may be present.

Chronic Poisoning.—This may occur among workers who are directly or indirectly exposed to the fumes of benzene in factories. The symptoms are headache, excessive fatigue, dizziness, nausea, loss of appetite, weakness, nervousness, disturbances of sensation, such as numbness and tingling in the extremities, bleeding from the gums and nose, disturbed sleep, menstrual irregularities among women, indigestion, frequent urination, leukopœnia, and a tendency towards a diminution in the polymorphonuclear leucocytes.[7] Aplasia of the granulocytes of the osseum medulla is a frequent and constant symptom. The post-mortem examination will show submucous hæmorrhages, aplasia of the bone marrow and fatty degeneration of the heart and liver.

A case[8] is recorded where fifty cases of poisoning by benzene occurred among young women within a few weeks of their employment in a rubber goods factory. Of these seven died. The treatment consisted of blood transfusion, ingestion of fresh liver or liver extract, and large quantities of fresh air. In severe cases extirpation of the spleen was tried with success. Heliotherapy, natural or artificial, is often effective.

1. *Averill, Brit. Med. Jour,* 1889.
2. *Kelynack, Med. Chr.,* 1893, *p.* 112; *Gaz. Med. de Paris,* 1893, *p.* 541.
3. *Falk, Vierteljahrsschr, f. ger. Med.,* 1892; *Dixonmann, Forens. Med. and Toxic., Ed. VI, p.* 467.
4. *Kelynack, Loc. Cit.*
5. *Spurr, Lancet,* 1899, *Vol. I, p.* 1488.
6. *In John Hopkins Hospital Bulletin of* 1910, *Selling describes two fatal cases of purpura among girls employed in a benzene factory.*
7. *H. R. Smith, Jour. of Industrial Hygiene, March,* 1928, *p.* 73.
8. *Correspondent of Vienna, Jour. Amer. Med. Assoc., July* 19, 1930, *p.* 215.

Detection.—Benzene is separated from organic mixtures by distillation and can be recognised from its odour and from its boiling point, which is 80° C.

Medico-Legal Points.—Poisoning by this drug is mostly accidental. A few cases are recorded, where it was taken with a view to commit suicide.

Benzene is oxidised in the body to phenol and dihydroxybenzenes, and is excreted partly by the kidneys in combination with sulphuric and glycuronic acids and partly unchanged by the lungs. Taken internally, it causes a marked fall in the number of the leucocytes of the blood, and is, therefore, recommended in the treatment of some forms of leukæmia.

NITROBENZENE (NITROBENZOL), $C_6H_5NO_2$

This substance is formed by the action of strong nitric acid on benzene. It is a yellow, oily liquid, having a pleasant odour like that of oil of bitter almonds. It is insoluble in water, but freely soluble in alcohol. It is commercially known as artificial oil of bitter almonds, or oil or essence of mirbane. It is largely used in the manufacture of aniline and explosives, in the preparation of perfumery, and for making boot polish, scenting soaps and flavouring confectionery. The liquid as well as its vapours are poisonous. When applied to the skin, it is absorbed rapidly and produces toxic symptoms.

Symptoms.—The symptoms are usually delayed from one to three hours or even longer after swallowing the poison. These are a burning taste in the mouth, numbness of the tongue, salivation, nausea, vomiting, giddiness, headache, cyanosis, cold and moist skin, weak and rapid pulse, hurried breathing, drowsiness and coma. The pupils are contracted first and then dilated. The urine is dark coloured. Convulsions may occur before death.

The symptoms produced by inhalation of its vapours are almost precisely the same as those produced when swallowed. A man, aged 43, spilled a quantity of nitrobenzene over his clothes, and went about several hours breathing an atmosphere saturated with nitrobenzene. After some time he became drowsy, his expression was stupid, and his gait unsteady; he had the appearance of a person who had been drinking. In four hours the stupor gradually deepened into profound coma which ended in death after five hours.[1]

Fatal Dose.—Eight to nine minims have caused death.[2] Twenty drops have also proved fatal. On the other hand, recovery has occurred, under prompt and efficient treatment, from one ounce[3] as also from three and-a-half ounces.[4]

Fatal Period.—The average fatal period is 7 hours. Death occurred in 75 minutes when a woman swallowed less than half-an-ounce of oil of mirbane.[5] Death may be delayed for 2 or 3 days.

Treatment.—Use emetics or the stomach tube. Give stimulants, such as strychnine or digitalin, but avoid alcohol, oils and milk. Use oxygen inhalation, saline infusion, venesection and blood transfusion.

Post-mortem Appearances.—The smell of nitrobenzene is discernible on the cavities being opened. All the organs are greatly congested. The mucous membranes of the stomach and duodenum are diffusely reddened and occasionally show patches of ecchymoses. The blood is fluid, chocolate coloured, and shows the spectrum of methæmoglobin, and an absorption band between the yellow and the red, which does not correspond to any of the hæmoglobin products.

Chronic Poisoning.—This occurs in persons working in factories where nitrobenzene is used. It is characterised by languor, anæmia with the red blood corpuscles

1. *Letheby, London Hosp. Rep.*, 1865, *Vol. II*, p. 34; *Taylor, On Poisons, Ed. III*, p. 666.
2. *Lond. Hosp. Rep.*, 1865.
3. *Dixonmann, Forensic Medicine and Toxic., Ed. VI*, p. 469.
4. *Cissel, abs. Lancet*, 1894, *Vol. I*, p. 1521.
5. *Graham Grant, Brit. Med. Jour., April* 12, 1913, p. 778.

reduced to less than half the normal, dyspnœa and jaundice with superimposed cyanosis producing a yellowish colour and even a blue-black colour in severe cases. The liver is damaged and resembles that of acute yellow atrophy in appearance. Nodular skin eruptions appear in some cases.

Chemical Tests.—1. Nitrobenzene is converted into aniline by reduction with nascent hydrogen generated by the action of dilute hydrochloric acid on zinc according to the following equation:—

$$C_6H_5NO_2 + 3H_2 = C_6H_5NH_2 + 2H_2O$$

The aniline boiled with caustic potash with the addition of a few drops of chloroform gives the characteristic unpleasant smell of phenylisocyanide or phenylisonitrile.

2. Strong sulphuric acid does not produce any change in colour, but it gives a crimson colour to oil of bitter almonds.

3. Ferrous sulphate does not produce Prussian blue with nitrobenzene, but it does so with hydrocyanic acid.

Medico-Legal Points.—Poisoning by nitrobenzene is generally accidental. A few suicidal cases have been recorded. It does not appear to have been used for homicidal purposes, though it has been used as an abortifacient.

Accidental poisoning has occurred from the inhalation of the vapour,[1] from soap scented with nitrobenzene used in a hot bath,[2] from wearing shoes polished with a blacking containing it,[3] as also from its application to the skin to cure itch or destroy parasites.[4] It has also been swallowed accidentally in mistake for lemonade[5] or spirit.[6] A man,[7] aged 65, who was suffering from habitual constipation, swallowed about half-an-ounce of nitrobenzene with milk in mistake for almond oil. On reaching home in half-an-hour after taking the medicine, he felt ill, was losing control of his muscles and could not walk upstairs. Fifteen minutes later, he was found with a weak and intermittent pulse, rapid respiration, anxious look and inability to speak. The abdomen was tympanitic, and there was incontinence of urine. In half-an-hour the vomit smelt strongly of bitter almonds. The patient gradually became more and more deeply comatose, lost consciousness and ultimately died 22 hours after taking the oil. On post-mortem examination the stomach contained about 10 ounces of dark brown fluid smelling strongly of bitter almonds. The mucous membrane was thickened and hardened, and there were petechial hæmorrhages on the lesser curvature. The small intestine was healthy, and the large intestine was loaded with fæces. The bladder was normal and healthy. There was slight congestion of the lungs. The brain, liver, kidneys and spleen were normal.

Nitrobenzene stimulates, then paralyses, the central nervous system. It also acts upon the blood, deforming or destroying some of the red blood corpuscles and converting hæmoglobin into methæmoglobin. The blood loses the power of carrying and imparting oxygen to the tissues and contains a much smaller amount of oxygen than normally. In some cases it may contain but 1 per cent of oxygen instead of the normal 17 per cent. These changes in the blood lead to a diminution of the oxidation of the tissues and to the appearance of abnormal products in the urine. Some part of the nitrobenzene is reduced in the system to aniline, which in turn is oxidised to paraminophenol, which appears in the urine. A portion of the nitrobenzene is also eliminated by the lungs.

1. *Letheby, London Hosp. Rep.*, 1865, *Vol. II, p.* 34; *Taylor, On Poisons, Ed. III, p.* 666.
2. *Nicolson, Lancet*, 1862, *Vol. I, p.* 135.
3. *Stone, Jour., Amer. Med. Assoc.*, 1904, *Vol. XLIII, p.* 977.
4. *Linnosset, Union Med.*, 1874, *XXVIII, p.* 209; *Sterne, Rev. Med., de l'est*, 1905, *XXXVII, p.* 444; *Witthaus, Med. Juris. and Toxic., Vol. IV, p.* 1213.
5. *Gregory, Lancet*, 1906, *Vol. I, p.* 1242.
6. *G. Grant, Brit. Med. Jour., April* 12, 1913, *p.* 778; *Vizard, Lancet*, 1906, *Vol. I, p.* 88.
7. *Thomas, Ind. Med. Gaz., May*, 1926, *p.* 229.

DINITROBENZENE (DINITROBENZOL), $C_6H_4(NO_2)_2$

This occurs in three forms, *viz.*, ortho-, meta-, and para-dinitrobenzene. It is a yellow, crystalline solid, and is used in the manufacture of the explosives, roburite, bellite and sicherite, employed for blasting in coal mines. Poisonous symptoms have been produced among workmen employed in factories where it is used, either by inhaling its vapours or by handling it in their hands.

Acute Poisoning.—The symptoms are similar to those produced by nitrobenzene poisoning.

Chronic Poisoning.—The symptoms are pain in the stomach, nausea, vomiting, anorexia, headache, giddiness, staggering gait, insomnia, pale face, blue lips and nails, cold, clammy and yellow skin, dark coloured urine, amblyopia and occasionally peripheral neuritis.

Fatal Dose.—Unknown.

Fatal Period.—Unknown.

Treatment.—Same as in poisoning by nitrobenzene.

Post-mortem Appearances.—Not characteristic. Congestion of the organs. Chocolate-coloured blood.

Chemical Test.—In the presence of zinc and hydrochloric acid dinitrobenzene is converted into phenylene-diamine which is rendered alkaline by adding caustic soda or potash and evaporated after shaking it up with ether. The residue gives a brown colour with sodium nitrite and acetic acid.

TRINITROTOLUENE (TROTYL), $C_6H_2CH_3(NO_2)_3$

This is a high explosive, commonly called T. N. T., and obtained by nitrating toluene, a product of coal-tar distillation. It is a fine crystalline, yellow powder, sometimes used by shell fillers in the form of fused yellowish-brown lumps. It melts at about 80° C. It is soluble in oils and greases as well as in acetone, ether, benzene and xylol. It stains the skin and hair a characteristic yellow or tawny orange colour which is not removed by ordinary washing. It is actively toxic, and may produce poisonous effects by absorption through the skin, gastro-intestinal tract, or lungs. Poisoning, sometimes, attended with fatal results, occurred among workers who were engaged in the manufacture of this substance during the last Great War and among those who handled it in filling shells, mines and grenades.

Symptoms.—These[1] may be considered under the following heads:—

1. Dermatitis.
2. Toxic Gastritis.
3. Blood Changes.
4. Toxic Jaundice.

1. **Dermatitis.**—This appears in the form of a papular or erythematous rash over the hands, wrists, face, neck and feet, and is most prominent where sweating and mechanical friction are greatest. The rash on the hands is most frequently of the cheiropompholyx type, seen on the webs of the fingers and on the palms. There is intense pruritus, and the character of the rash is often altered by a secondary infection. Fine desquamation follows the rash, and in rare cases the skin is exfoliated in large flakes.

Toxic Gastritis.—This is characterised by a bitter taste in the mouth, spasmodic pain in the epigastrium, anorexia, acid eructations, nausea, vomiting, constipation, later diarrhœa with pain and tenesmus.

1. *Livingstone-Learmonth and Cuningham, Lancet, Aug.* 12, 1916, *p.* 261; *Ibid., Dec.* 16, 1916, *p.* 1026.

3. **Blood Changes.**—These are hæmolysis of the red blood corpuscles and conversion of hæmoglobin into methæmoglobin, which cause the symptoms of pallor or cyanosis, dizziness, breathlessness and the passage of dark urine. Aplastic anæmia occurs in severe poisoning, which often ends fatally.

4. **Toxic Jaundice.**—This occurs in cases of severe poisoning, and often appears suddenly without preliminary warning. Sometimes, there may be premonitary symptoms of dizziness, fatigue and headache. Toxic jaundice is associated in its early stages with enlargement of the liver, and later with shrinkage; ascites was observed in one case in which considerable shrinkage of the liver was found. In fatal cases coma and delirium supervene suddenly, usually about three weeks after the first appearance of jaundice.

Toxic jaundice appears to occur more frequently in young adults who, when attacked, are very prone to die. It usually develops between the fifth and sixteenth week after exposure to T. N. T.,[1] but a long latent period may, sometimes, supervene before toxic jaundice occurs. Legge reports a case in which the latent period was seven months, and Glynn reports one in which it was nine months.[2]

Treatment.—Where jaundice is absent, the treatment consists of rest in bed for a day or two with a liberal diet of milk, fruit and green vegetables, demulcent drinks and vegetable laxatives. A mixture containing sodium sulphate, potassium citrate and sodium bicarbonate may be given as a routine measure.

In the treatment of cases with jaundice absolute rest in bed is essential. Milk should be given and the bowels must be kept loose by aperients. Alkalies, such as citrates and bicarbonates, should be given to counteract the tendency to acid intoxication. Rectal and intravenous saline infusions are recommended in severe cases.

Preventive Measures.—These are—

1. Employment in T. N. T. factories of healthy persons above 18 years of age.
2. Medical inspection of the workers at least once a week.
3. Efficient ventilation of factories.
4. Mechanical devices for preventing the accumulation of dust and getting rid of fumes.
5. Protection of the workers by the use of special clothing, such as gauntletted gloves, respirators and veils.
6. Thorough washing of the hands and face before leaving the factories and before taking meals.
7. Liberal supply of milk.

Post-mortem Appearances.—The liver shows extensive necrosis and atrophy. The kidneys show cloudy swelling and fatty degeneration. The myocardium is soft, pale and flabby. Petechial and diffuse hæmorrhage are generally found beneath the endocardium, pericardium and peritoneum.

Chemical Analysis.—If the urine of a suspected case of trinitrotoluene poisoning be mixed with an equal volume of 20 per cent sulphuric acid solution, and shaken out with ether, the ether is separated and washed free of acid with water and then treated with alcoholic potash, a pink colour will indicate the evidence of trinitrotoluene poisoning (Webster's test).

NITROGLYCERINE, (TRINITROGLYCERINE, TRINITRIN, GLONOIN OIL, NOBEL'S BLASTING OIL), $C_3H_5[(NO_2)O]_3$.

This is a trinitrate of glyceryl, and is a colourless, oily liquid with a sweet, aromatic pungent taste. It is slightly soluble in water and rapidly in alcohol, ether,

1. *Lancet*, Dec. 16, 1916, *p.* 1026.
2. *Medical Research Council, Special Report Series*, No. 58, 1921; *Brit. Med. Jour.*, *March* 12, 1921, *p.* 395.

chloroform, oil and fats. It is highly explosive, and forms dynamite, when mixed with an infusorial earth.

Physiologically nitroglycerine is similar in action to amyl nitrite, but its effect is more lasting. It is an extremely active drug, the medicinal dose being 1/200 to 1/80 grain. A 1 per cent solution of nitroglycerine is known in the Pharmacopœia as *Liquor glycerylis trinitratis,* the dose being ½ to 2 minims. Another official preparation is *Tabella glycerylis trinitratis,* each containing 1/120 grain of nitro-glycerine. The dose is 1 or 2 tablets.

The vapours of nitroglycerine are highly poisonous.

Symptoms.—A burning sensation in the throat, nausea, vomiting, colicky pain in the abdomen, sometimes diarrhœa, painful throbbing of the arteries all over the body, severe headache, giddiness, flushing of the face and skin, perspiration, oppression in the heart, hurried and difficult breathing, marked cyanosis, complete paralysis and unconsciousness. Death occurs from respiratory paralysis. Muscular twitchings and delirium may, sometimes, be observed.

Fatal Dose.—This is uncertain. Two mouthfuls of nitroglycerine swallowed in mistake proved fatal to a miner,[1] but a few drops of the undiluted drug would probably cause death. On the other hand, recovery occurred after a dose of a tablespoonful of dynamite to which a few drops of nitroglycerine were added.[2]

Fatal Period.—This is not known, but about one ounce caused death in 4 hours.[3] Death occurred in 2 hours in another case.[4]

Treatment.—Emetics or stomach tube; adrenaline chloride or atropine hypodermically; black coffee to relieve headache; fresh air and artificial respiration in poisoning by vapour inhalation.

Post-mortem Appearances.—Ecchymosis and congestion in the stomach and intestines. The lungs are œdematous, and the other organs are congested. The blood may be of a chocolate colour due to the formation of methæmoglobin.

Chemical Tests.—1. When treated with aniline and a drop of strong sulphuric acid, nitroglycerine produces a red colour. The same reaction is obtained when treated with brucine and strong sulphuric acid.

2. Nitroglycerine explodes violently when struck with a hammer.

Medico-Legal Points.—Most of the cases of poisoning occur among those who are engaged in the manufacture of nitroglycerine, dynamite or other high explosives. Accidental poisoning may also result from an overdose of nitroglycerine administered as a remedy for angina pectoris, cardiac dyspnœa, etc. It should, however, be remembered that tolerance for the drug is often too readily acquired. Stewart[5] describes cases in which a single dose of 5 grains and 20 minims of pure nitroglycerine per day were taken without any ill-effects.

Nitroglycerine has been administered in alcoholic drinks for criminal purposes, and two bobbins of dynamite, 4" by ¾", were taken internally with a view to commit suicide.[6]

PETROLEUM (ROCK OIL)

This is an oily liquid found under the ground in several parts of the earth, and consists of a mixture of hydrocarbons of the paraffin series. This crude oil contains inflammable and explosive products, which are removed by distillation and

1. *Taylor, On Poisons, Ed. III, p.* 671.
2. *Dixonmann and Brend, Forens. Med. and Toxic., Ed. VI, p.* 454.
3. *Dixonmann and Brend. Forens. Med. and Toxic., Ed. VI, p.* 454.
4. *Sydenham Soc. Biennial Retrosp.,* 1867-68, *p.* 453.
5. *Jour. Amer. Med. Assoc., May* 27, 1905; *Brit. Med. Jour., Sep.* 30, 1905, *Ep., p.* 52.
6. *Dixonmann and Brend, Forensic Med. and Toxic., Ed. VI, p.* 454.

purification so as to render it fit for household use. The refined oil is called kerosene. During the process of purification several other products are separated which cannot be used in lamps. Those which are lighter and boil at a lower temperature than kerosene are known as gasoline, petrol, naphtha, benzine, etc. From the heavier portions or those which boil at higher temperatures than kerosene the lubricating oils, vaseline, and paraffin are made.

Symptoms.—The symptoms produced by inhaling the fumes are dizziness, headache, nausea, burning sensation in the chest, mental confusion, inability or disinclination to move, cyanosis, insensibility and convulsions. Death may occur from failure of the heart and respiration.

A coolie[1] while working at the manhole of a large petrol tank, apparently overcome by the petrol fumes, fell into the tank, the bottom of which was covered with petrol to a depth of not more than two inches. One hour later, he was removed from the tank and was at once taken to the Indian Military Hospital, Quetta, where he was found quite unconscious. His pulse was imperceptible and breathing was laboured. Blisters, most of which had burst, had already formed on every part of his body, much more than half the superficial skin area being burnt. The patient's clothes were all soaked in petrol. He was kept in the open, and oxygen inhalations with injections of strychnine and digitalis were given. Four hours after he was removed from the tank he regained consciousness, was very restless and complained of severe thirst, to relieve which copious draughts of water were given. The temperature was 97° F. The pulse, 80 per minute, improved in volume and tension. The respirations were 22 per minute. The patient did not complain of pain. There were no signs of œdema of the larynx, the lungs were clear, and the urine was passed freely and was free from albumin. The burns which were of the second degree were dressed with half per cent picric acid solution. On the following morning small discrete ulcers were seen to be forming on both corneæ. On the third day he developed severe diarrhœa, which was readily controlled with bismuth salicylate. On the fourth day the eye condition was much worse, the eyes presenting the appearance of traumatic conjuctivitis with lachrymation and photophobia, and with superimposed dermatitis of the lids. The dull greyish-white necrosed areas on the corneæ spread until the whole surface of both corneæ was involved. There was no perforation but considerable shrinkage of the eye balls. On the fifth day the general condition of the patient was very much worse, and from that day he went rapidly down hill, and died on the ninth day.

The symptoms produced by the ingestion of the products of petroleum, especially kerosene, are burning pain in the throat, feeling of warmth in the stomach, thirst, vomiting, colic, giddiness, heaviness in the head, pale or cyanosed face, drowsiness deepening into stupor, coma and death. The breath, vomit and urine give off the peculiar smell of kerosene. The pupils are at first contracted, but become dilated when coma supervenes. Convulsions may occur in some cases. A relapse and death may follow an apparent recovery.

Fatal Dose and Fatal Period.—Uncertain. A dose of half-an-ounce proved fatal in four hours. A child, aged 3 years, drank a mouthful of kerosene and died in seven hours.[2] An ounce and-a-quarter of paraffin oil killed a child, fourteen months old, in one hour and fifty minutes.[3] Recovery has taken place after a pint of petroleum swallowed by a woman for the purpose of suicide.[4]

Treatment.—If the fumes have been inhaled, the patient should at once be removed to the open air and artificial respiration should be started. The body should be kept warm.

If the poison has been swallowed, emetics should be administered or the stomach should be washed out with warm water. Purgatives and stimulants should then be administered and artificial respiration may be resorted to, if necessary.

1. *Cruickshank and Chowdry, Ind. Med. Gaz., June,* 1930, *p.* 320.
2. *Sydney Smith, Forensic Med., Ed. VI, p.* 504.
3. *M'Dougall, Med. Chron.,* 1898.
4. *Jour. Amer. Med. Assoc., April,* 1873, *p.* 566.

Post-mortem Appearances.—The usual signs of asphyxia may be present. The smell of petroleum may be noticed in the lungs, stomach, intestines and in the urine. The stomach may be pale or congested.

Chemical Tests.—The oil is known by its oily feeling when rubbed between two fingers, and by its characteristic odour and inflammability. When treated with an alkali, it does not saponify, a distinguishing feature from animal and vegetable fats and oils.

Medico-Legal Points.—Petroleum is not an active poison. In India, accidental cases of poisoning by kerosene occur among children who crawl on the floor and manage to drink the contents from small tin lamp cans kept within their easy approach. I have seen a few such cases, which recovered under proper treatment. Accidental cases occur also among men who drink it in mistake for country liquor. A case[1] is recorded where a Hindu woman, aged 60, who had a quarrel with her daughter-in-law, committed suicide by drinking a quantity of kerosene oil. The post-mortem examination showed that the stomach contained partially digested rice smelling of kerosene oil. The liver, pancreas, spleen and kidneys were congested.

The use of about a pint of petrol as a hair wash has produced poisonous symptoms in a strong, healthy woman, about 35 years old.[2] Its fumes have given rise to toxic symptoms among chauffeurs and others working with motor-engines.[3] Those exposed to the fumes for a long time suffer usually from polioneuritis. Skin eruptions are noticed on those who work constantly in petroleum distilleries.

A person attempted to commit suicide by drinking petrol which resulted in the vomiting of blood and mucus and pain in the mouth, throat and abdomen.[4]

Gasoline killed an infant, 18 months old, in 30 minutes, but the quantity that was taken is not known.[5]

OIL OF TURPENTINE (SPIRIT OF TURPENTINE)

This is distilled from common turpentine, an oleo-resin obtained from various species of Pinus, N. O. *Coniferæ*. It is a colourless and transparent oily liquid, a mixture of several hydrocarbons of the turpene series. It has a strong peculiar odour and a pungent, bitter taste. It is insoluble in water, but soluble in alcohol, ether, chloroform and carbon bisulphide. It is extensively used to dissolve varnish. The pharmacopœial preparation is *Oleum terebinthinæ* (Rectified oil of turpentine), the dose of which is 3 to 10 minims and 120 to 240 minims as an anthelmintic. It is contained in the official preparations of *Linimentum terebenthinæ* (65%) and *Linimentum terebinthinæ aceticum* (44.5%).

When purified by distillation with lime, it is known as camphene.

Sanitas is a watery solution of turpentine oxidised by exposure to the air. Hydrogen peroxide is its active principle.

Symptoms.—A poisonous dose of oil of turpentine causes a burning pain in the mouth, throat and stomach, thirst, vomiting, diarrhœa, giddiness, drowsiness, cold skin, muscular spasms, coma and death. Owing to its irritating action on the kidneys the patient complains of pain in the loins, difficulty of micturition, strangury and passes scanty high-coloured urine, which contains blood and albumin, and possesses a smell of violets. When a large quantity is taken, the urine may be completely suppressed.

Applied to the skin, oil of turpentine produces redness and irritation, followed by vesication.

1. *Beng. Chem. Exam. Annual Rep.*, 1930, *p.* 10.
2. *Houghton, Brit. Med. Jour., Dec.* 12, 1908, *p.* 1747.
3. *Box, Ibid., April* 4, 1908, *p.* 807 ; *Petrie, Ibid., April* 25, 1901, *p.* 987.
4. *Madras Chemical Examiner's Annual Report*, 1929, *p.* 4.
5. *Biller, New York Med. Jour.*, 1889.

Inhalation of turpentine vapour produces irritation of the eyes, headache, dizziness and irritation of the respiratory passages or even pneumonia. It, sometimes, causes irritation of the kidneys.

Fatal Dose.—Four and six ounces of oil of turpentine have respectively killed adults.[1] A teaspoonful killed a child, 5 months old, and half-an-ounce killed a child, 14 weeks old.[2] Recovery has followed half-a-pint in the case of a woman,[3] and four ounces in the case of an infant.[4]

Fatal Peirod.—The adult who took 4 ounces died in 12 hours and the child who had half-an-ounce poured down his throat by his brother, aged 8 years, died in 15 hours.

Treatment.—Give emetics or wash out the stomach. Administer demulcents and castor oil. Keep up the warmth of the body.

Post-mortem Appearances.—The stomach usually shcws hæmorrhagic spots, sometimes, with erosions of its mucous membrane. The stomach contents may smell strongly of turpentine. In the case[5] of an adult male, aged 39, who died after drinking six ounces of spirit of turpentine the post-mortem examination showed that the stomach contained four ounces of turpentine, and its mucous membrane was completely macerated and lying in small pieces in the gastric cavity. The wall of the stomach felt like leather due to the action of the turpentine.

Chemical Tests.—Oil of turpentine forms a red cclour with hydrochloric acid and ferric chloride; the colour changes to violet and blue on standing. It is also known by its odour.

Medico-Legal Points.—Oil of turpentine is not an active poison. A few accidental cases of poisoning have occurred from its medicinal use as an anthelmintic or from its administration by mistake. It has been taken to procure abortion, but has been very rarely used for homicidal purposes. A woman was charged with an attempt to murder her infant by pouring oil of turpentine down its throat. She was acquitted on the plea that the oil was administered as a cure for the child's cough.[6] A case of attempted suicide is reported in which a large quantity of camphene was taken by a woman, aged 22 years, who recovered in 8 days.[7]

Toxic symptoms occurring from continued inhalation of turpentine vapour are occasionally observed in painters or in persons sleeping in a newly varnished room. Reinhard describes a case, where a man who was occupied in a room in filling small vessels out of a large vessel containing turpentine, began to feel dizzy on the first day, dryness of the mouth and mental depression on the second day and complained of painful micturition on the third day. The urine contained blood and albumin and continued to emanate the odour of violets for 7 days after he ceased to inhale the vapour.[8] Seamen who were engaged in painting in enclosed spaces on one of H. M. ships suffered from turpentine poisoning by inhaling its vapours. The symptoms arose after one or two days' work, seven men reporting sick within a week. They complained of scalding pain at the end of micturition, and in some cases, of frequency; the urine contained blood and had the odour of violets. They all recovered after some time.[9]

Turpentine is eliminated by the lungs, and imparts its characteristic odour to the breath. It is eliminated by the kidneys and appears in the urine in combination with glycuronic acid. The urine acquires a smell of violets and reduces Fehling's solution. Turpentine is also excreted to some extent by the skin.

1. *Dixonmann, Forens. Med. and Toxic., Ed. VI, p.* 529.
2. *Taylor, On Poisons, Ed. III, p.* 658.
3. *Stanwell, Brit. Med. Jour.*, 1901, *Vol. I, p.* 640.
4. *Taylor, Loc. Cit.*
5. *Maitland, Brit. Med. Jour., July* 11, 1931, *p.* 77.
6. *Reg.* v. *Rodanbosh, C. C. C., Dec.* 1856; *Taylor, On Poisons, Ed. III, p.* 658.
7. *Horn's Vierteljahr*, 1866, *Vol. II, p.* 537; *Ibid.*
8. *Deutsche. Med. Wochenschr.*, 1887; *Dixonmann, Foren. Med. and Toxic., Ed. VI, p.* 529.
9. *H. Wilks, Jour. Royal Nav. Med. Service, Jan.*, 1930, *p.* 53; *Lancet, Feb.* 8, 1930, *p.* 307.

EUCALYPTUS OIL

This is distilled from the fresh leaves of Eucalyptus globulus, Eucalyptus dumosa and other species of Eucalyptus. It is a colourless or pale yellow, volatile oil, which becomes darker and thicker by exposure. It has an aromatic, camphoraceous odour and a pungent taste, leaving a sensation of cold in the mouth. It is soluble in alcohol. It is a pharmacopœial preparation, known as *Oleum eucalypti*. The dose is 1 to 3 minims. *Unguentum eucalypti* is a non-official preparation containing eucalyptus oil in the proportion of 1 in 10.

Symptoms.—Applied to the skin, eucalyptus oil is less irritant than other volatile oils but, if its vapour is confined, it will produce redness, irritation, vesication and even pustulation.

Taken by the mouth in a large dose, eucalyptus oil acts both as an irritant and as a narcotic poison, and causes nausea, vomiting, purging, abdominal pain, headache, foam at the mouth, cyanosis, contracted pupils, cold clammy skin, cramps, rapid pulse, slow, stertorous breathing, albumin and blood in urine, unconsciousness and coma. Death occurs from respiratory paralysis.

Fatal Dose and Fatal Period.—Uncertain. Two drachms of eucalyptus oil produced toxic symptoms in a boy, aged $2\frac{3}{4}$ years.[1] Three[2] and four[3] drachms have caused poisonous symptoms in adults. Six drachms[4] killed a cabdriver, aged 34 years, in 40 hours. On the other hand, recovery has followed a large dose of one ounce and a half.[5]

Treatment.—This consists in the lavage of the stomach, hypodermic administration of stimulants, such as strychnine, caffeine, etc., and inhalation of oxygen.

Post-mortem Appearances.—The mucous membrane of the stomach is red and congested, and may, sometimes, be inflamed. The mucous membrane of the trachea and bronchi is red and congested. The lungs are congested. The kidneys are acutely congested.

Medico-Legal Points.—Poisoning by eucalyptus oil is not common, although a few accidental cases have occurred from it having been swallowed in mistake for some medicine. A case[6] is recorded where a girl, 16 years old, drank some eucalyptus oil in mistake for her fever mixture. She was taken to the hospital in a drowsy condition. Her breath smelt of eucalyptus oil. She recovered after her stomach was washed out in the hospital. Eucalyptus oil was detected in the stomach wash.

Rarely, suicidal cases have occurred. In his annual report for 1932, the Chemical Examiner of Madras reports a suspected case of suicide, where a ticket collector and a girl with whom he was living swallowed about an ounce each of eucalyptus oil. They had vomitings and purgings, but recovered under treatment in the hospital. The vomitings and purgings showed eucalyptus oil on analysis.

Eucalyptus oil is excreted by the kidneys, and imparts to the urine an odour of violets. It is also eliminated by the skin and the lungs.

---

1. *Orr, Brit. Med. Jour., May* 12, 1906, *p.* 1085.
2. *Garrett, Brit. Med. Jour., June* 27, 1925, *p.* 1172.
3. *Sewell, Brit. Med. Jour., May* 16, 1925, *p.* 922; *Gibbin, Brit. Med. Jour., June* 4, 1927, *p.* 1005.
4. *Myott, Brit. Med. Jour., March* 10, 1906, *p.* 558.
5. *Bombay Chem. Analyser's Annual Report*, 1932, *p.* 6.
6. *Madras Chem. Examiner's Annual Rep.*, 1933, *p.* 7.

CHAPTER XXXI

CEREBRAL POISONS—(*Contd.*)

C. DELIRIANT POISONS

DATURA ALBA AND DATURA FASTUOSA (*SAFED AND KALA DHATURA*)

These plants belonging to N. O. *Solanceœ* grow commonly on waste places all over India. Datura stramonium (the thorn apple) grows in India at high altitudes throughout the temperate Himalayas, and in England on waste places and dung heaps. All the parts of these plants are poisonous but the seeds and fruit are considered to be the most noxious.

Fig. 141.—Datura Alba (Dhatura) × ½.

They yield an active principle, *daturine*, chemically identical with atropine and hyoscyamine, which is a crystalline substance having an acrid, bitter taste. It is a powerful narcotic, anodyne and anti-spasmodic. Two other varieties, Datura atrox and Datura metel are met with. The former is

found about the coast of Malabar, and the latter occurs in many parts of India and in Eastern and West Indian Colonies.

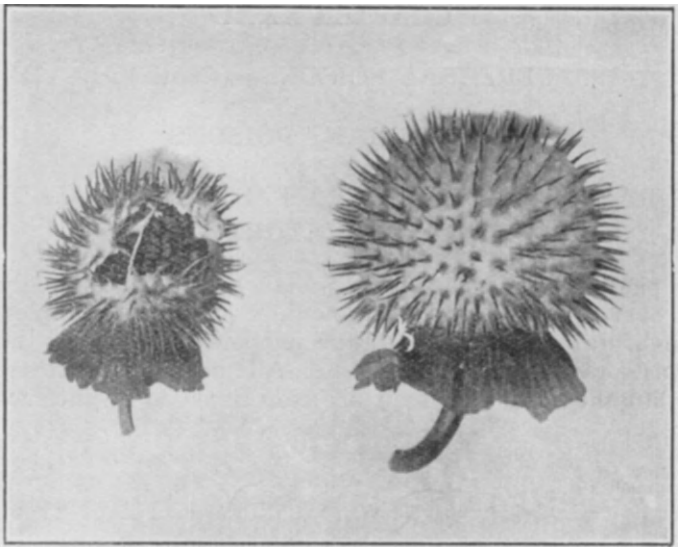


Fig. 142.—Datura Alba: Fruits

The dried leaves (Daturæ folia) of *Datura fastuosa* and of *Datura metel* and the dried seeds (Daturæ semina) of *Datura fastuosa* were official in the British Pharmacopœia of 1914, but are not included in the British Pharmacopœia of 1932. The non-official preparation made from the seeds is *Tinctura daturæ seminum* (strength 1 in 4), dose 5 to 15 minims.

Symptoms.—The symptoms usually appear within half-an-hour after swallowing the poison. Vomiting often occurs immediately after taking the seeds, especially when crushed, as they produce gastric irritation. A bitter taste, dryness of the mouth and throat, burning pain in the stomach and dysphagia are the first symptoms that are complained of. These are followed by giddiness, staggering gait, in-coordination of the muscles, peculiar flushed appearance of the face, dry, hot skin, diplopia, dilated pupils, red and injected conjunctivæ and drowsiness. Sometimes, the temperature of the body is raised very high. In three cases of poisoning in the District of Hissar the temperatures were noted 105.4°, 107.4° and 108° F. respectively.[1] In three cases which came under my observation in King George's Hospital, Lucknow, during 1932, the temperatures were found to be 102°, 104° and 105° F. respectively. The pulse is full and bounding but, later becomes weak, irregular and intermittent. The patient now becomes restless and delirious. Delirium is of a peculiar character. He is silent or mutters indistinct and inaudible words but, usually he is noisy, tries to run away from his bed, picks at the bed clothes, tries to pull imaginary threads from the tips of his fingers, and is subject to dreadful hallucinations of sight and hearing. In fatal cases drowsiness passes

1. *Black, Punjab Chemical Examiner's Annual Report,* 1916.

into stupor, convulsions and coma. Death occurs from paralysis of the heart or respiration. In cases, which recover, stupor passes away, and secondary delirium develops, which lasts for some hours.

In some cases insensibility occurs almost immediately after the poison is administered either in solution, or in very fine powder. A man drank two mouthfuls of a liquid poisoned with datura, complained of a bitter taste and fell down insensible within forty yards of the spot where he had drunk, and did not recover his senses until the third day. Another man was struck down so suddenly that his feet were scalded by some hot water which he was carrying.[1]

Fatal Dose.—Uncertain. Four datura fruits pounded and mixed with flour were given to six men, four of whom died.[2] A ripe fruit weighs, on an average, about 2 drachms, and contains the seeds which weigh about $1\frac{1}{2}$ drachms. One hundred dried datura seeds weigh 20 to $20\frac{1}{2}$ grains. A decoction of 125 seeds of datura stramonium proved fatal to a woman.[3] One hundred stramonium seeds weighing 16 grains killed a child, 2 years old.[4] On the other hand, a girl, aged 8, recovered after swallowing 237 of these seeds.[5]

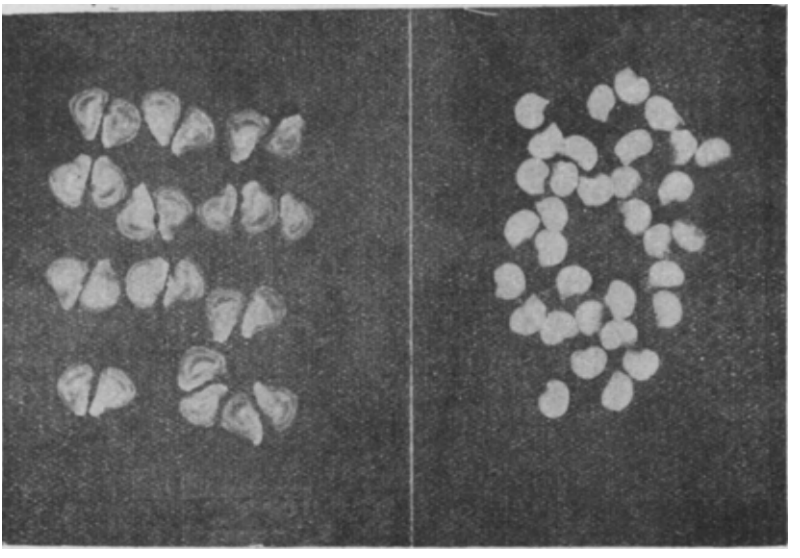
Fatal Period.—In a majority of fatal cases death usually occurs within twenty-four hours. A boy died in three or four hours after drinking a large quantity of datura mixed in *sharbat*.[6] A man, 22 years old, died in four or five hours after datura seeds had been administered to him in sweets, known as *Peras*.[7] The woman who took a decoction of stramonium seeds died in seven hours.

Treamtent.—Emetics should be given or the stomach should be washed out with a weak solution of potassium permanganate or a solution of tannic acid (20 grains in 4 ounces of water). Pilocarpine nitrate in doses of 1/10 to $\frac{1}{4}$ grain hypodermically is recommended as an antidote, but it is doubtful whether it can antagonise the action of datura on the brain. Morphine in $\frac{1}{4}$-grain doses hypodermically is also regarded as a physiological antidote, but it should be administered with caution, as it has a depressant action on the respiratory centre. Soporifics, such as bromides and barbiturates, may be given to control the delirium, but the administration of chloroform by inhalation is considered more beneficial. Cold affusions may be applied to the head. Stimulants, such as caffeine, should be given and artificial respiration should be adopted when necessary. A large hot enema may be given with advantage to the patient, for it acts as a stimulant and by flushing the body helps the elimination of the absorbed poison.

Post-mortem Appearances.—Datura seeds or their fragments may be found in the stomach and intestines. The œsophagus, stomach, duodenum and other internal organs are mostly congested. In rare cases the mucous membrane of the stomach may be found slightly inflamed.

1. *Chevers, Med. Juris., Ed. III, p.* 210.
2. *K. E. v. Sumeran Das, Gorakhpur District, Appeal No.* 572 *of* 1921, *Allahabad High Court.*
3. *Taylor, On Poisons, Ed. III, p.* 774.
4. *Ibid.; Duffin, Lond. Med. Gaz.,* 1834, *Vol. XV, p.* 194.
5. *Friedman, Jahrb. F. Kinderh.,* 1891, *XXVIII, p.* 354.
6. *Lahore High Court, Criminal Appeal No.* 828 *of* 1929; *Criminal Law Jour., Feb.,* 1930, *p.* 140.
7. *Allahabad High Court, Criminal Appeal No.* 870 *of* 1930.

Detection.—The seeds of Datura alba are often mistaken for those of Capsicum. The seeds of Datura alba are very hard, flattened, kidney-shaped, and 1/6 inch broad and 1/25 inch thick. They are bitter in taste



A. B.

Fig. 143.—A. Datura Seeds × 3. B. Capsicum Seeds × 3.

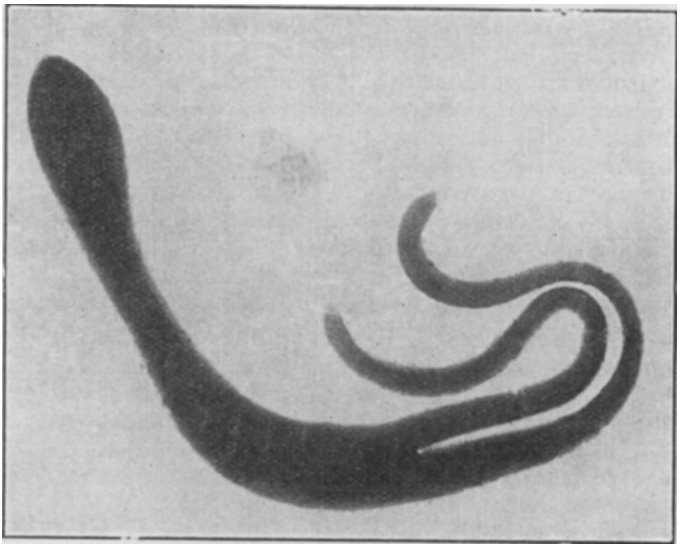


Fig. 144.—Microphotograph of Section of Datura seed showing embryo.

and have a double-ridged convex border. The testa is dark or yellow brown in colour, is finely pitted and reticulated. On longitudinal section the seeds show the embryo curving outwards at the hilum.

The seeds of Capsicum are thin, smooth, roundish, the convex border being single and sharper. They have a sharp pungent taste. The testa is of a pale yellow colour. On section the seeds show the embryo curved inwards.

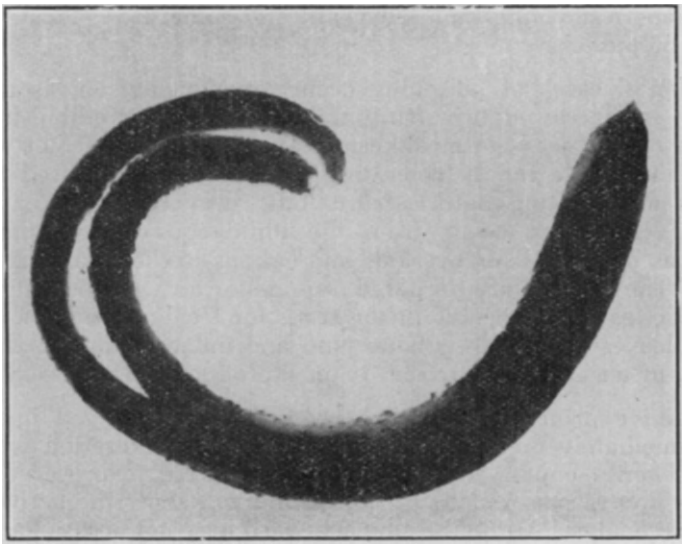


Fig. 145.—Microphotograph of Section of Capsicum seed showing embryo.

Test.—Digest the seeds or the suspected material for about half-an-hour in warm rectified spirit, filter and evaporate on an open water bath to dryness. Rub the residue with about half-a-drachm of distilled water acidulated with sulphuric acid and evaporate to dryness on the water bath. Take up the residue with a few drops of distilled water, and instil a drop of this into the eye of a cat. After about half-an-hour the pupil will be found dilated.

Medico-Legal Points.—Datura is commonly used in India for criminal purposes. The seeds are generally used by road poisoners to stupefy travellers to facilitate robbery and theft and rarely to destroy life. A case[1] is, however, recorded, in which one Musammat Maiki of District Kheri administered to Musammat Chitana, her mother-in-law, datura poison in her food. When Musammat Chitana lost her senses after taking the food, the accused (Musammat Maiki) killed her by throttling her neck with her foot. The seeds are, sometimes, given to children with a view to kidnap them when they become unconscious or delirious.

The seeds are given whole, or more often crushed, mixed with rice, *dal,* or wheat or *bajra* flour and, sometimes, with liquor. The seeds, as well as the leaves, are also mixed with tobacco or *ganja* and smoked in a *chilum* (pipe) for the same purpose. A decoction of the seeds is at times added to liquor or toddy with a view to enhance its intoxicating property.

1. *Leader, Sep.* 13, 1930.

Cases of suicidal poisoning by datura are very rare. In his annual report for 1907, Rai Chooni Lal Bose Bahadur, Chemical Examiner of Bengal, mentions the case of a Hindu female, who committed suicide by taking datura seeds. In his annual report for 1928, the Chemical Examiner of the United Provinces of Agra and Oudh reports a case from Jaunpur where a young man, 20 years old, committed suicide by taking datura and opium.

Accidental cases of poisoning occur among children, as also among adults, by eating raw datura fruits mistaking them as edible fruits or by eating dry datura seeds in mistake for capsicum seeds. I met with a case in which the whole family consisting of 8 members suffered from toxic symptoms after eating datura fruits but recovered the next morning. Accidental cases also occur from the injudicious use of the seeds in medicine, as they are used by *vaids* and *hakims* in the treatment of several diseases. The seeds are reputed to have an aphrodisiac property. Medicated *ghee* is prepared with the seeds for local application. The juice of datura leaves is used to subdue pain and inflammation in rheumatism. If applied to an abraded surface, it may produce poisonous symptoms.

The active principle of datura is excreted unchanged in the urine, almost immediately on its administration and the excretion is completed in ten to twenty hours. It is, therefore, advisable to preserve the urine in cases of datura poisoning, since the urine will show the active principle on chemical analysis, while the stomach wash may not occasionally respond to the tests.

The seeds of Datura resist putrefaction for a long time, but the mydriatic principle contained in them appears to be destroyed by putrefactive changes in the body, although it can be obtained after some lapse of time in the vomit or from the earth upon which the patient has vomited. Five persons of Police Station Mohanlalganj, Lucknow District, *viz.*, a Hindu Brahmin, 40 years old, a Hindu female, aged 30 years, 2 boys of 12 and 16 years respectively and a girl, 7 years old, who were administered datura by their comrade, died on the 16th May, 1921. The post-mortem examination on the bodies of these persons was held on the 18th May, 48 hours after death. The viscera were decomposed, but preserved in the usual manner. They were forwarded to the Chemical Examiner at Agra on the 28th July, 1921. He detected a substance having the properties of datura in the viscera of the male, female and the boy, aged 16 years, but failed to detect it in the viscera of the other two. Datura was also found in the viscera of a Mahomedan male whose body was exhumed after five days of burial.[1]

Illustrative Cases.—1. *Homicidal Poisoning by Datura.*—In 1921, one Musammat Khazanu, 16 years old, was convicted by the first Additional Sessions Judge at Bulandshahr of having committed the murder of her husband, Net Ram, 18 years old, by administering datura seeds in his food. On the evening of the 10th May, 1921, she mixed datura seeds in the flour and prepared some loaves. Next morning Net Ram took these loaves with him and went out to work at his field. On the way he ate them with onion at 8 a.m., and worked in his field till 10 a.m., when he felt giddy and his legs began to reel. After a short while he became unconscious and was taken home, where he died at 5 p.m.,—nine hours after he had eaten the poisonous loaves.

1. *Sind Chemical Analyser's Annual Report,* 1924, *p.* 24.

On post-mortem examination, a few suspicious seeds were sticking to the inner surface of the œsophagus which was rather congested. The stomach was congested. It contained four ounces of brownish fluid in which several similar seeds were floating. The duodenum was congested, the contents being similar to those of the stomach. The large intestine was distended with flatus. Th Civil Surgeon of Bulandshahr forwarded the viscera to the Chemical Examiner for analysis. A substance having the properties of datura was detected in the viscera.—*K. E.* v. *Mt. Khazanu, Allah. H. Crt. Cr. Appl. No.* 645, 1921.

2. *Datura Administered as a Love Philter.*—A Mahomedan boy, aged 16 years, was convicted of poisoning with datura five or six women, and sentenced to one year's rigorous imprisonment. The poison was administered in *peras* (sweets) as a love philter which would turn a girl, 12 years old, with whom he became infatuated, and other women of the house in his favour.—*Leader, June* 13, 1923.

3. *Datura Administered for Robbery.*—(*a*) In April, 1919, Janki Bania met a Mahomedan passenger at Charbagh Railway Station, and volunteered to purchase some sweets for him. The Mahomedan, soon after eating the sweets, became unconscious and was removed to Balrampur Hospital where he appeared to be suffering from the symptoms of datura poisoning. He was treated with hypodermic injections of pilocarpine nitrate, and he recovered on the third day. The accused was arrested. On his person were found a powder and two datura seeds. Datura was detected in the powder on chemical analysis.

(*b*) In December, 1920, one Chhida Ahir administered datura in vegetable to two passengers who had been waiting at the musafarkhana near Charbagh Railway Station. They were afterwards found lying unconscious, and removed to the Police Hospital, where their stomachs were washed out and hypodermic injections of pilocarpine nitrate were administered. The following day when they recovered they complained that they had lost all the cash and clothes which were with them. The stomach washings were forwarded for chemical analysis; a substance having the properties of datura was detected in them. The accused was arrested and convicted to a term of imprisonment.

(*c*) A Brahmin was sentenced to five years' rigorous imprisonment under section 328, I.P.C., for administering datura in cooked *puries* and potatoes to not less than nine persons of a family residing at Nimsar. All of them were taken ill and were unconscious, but recovered in three days.—*Ind. Daily Teleg., Aug.* 10. 1923.

ATROPA BELLADONNÁ (DEADLY NIGHT-SHADE)

This plant belongs to N. O. *Solanceœ,* and grows wildly in England near villages or on old ruins. All parts of this plant, *viz.*, the leaves berries and root are poisonous. They contain two alkaloids, atropine and hyoscyamine, but the poisonous properties are chiefly due to the former.

Atropine, $C_{17}H_{23}NO_3$, crystallizes in odourless and colourless prismatic needles, and has a bitter taste. It is sparingly soluble in water (1 in 500), but freely in ether, alcohol and chloroform. Its aqueous solution has an alkaline reaction, and is readily decomposed by keeping. It can be chemically split up by strong acids and alkalies into tropine and tropic acid, and may be reconstructed synthetically from these substances. The official dose of atropine as also of atropinæ sulphas [atropine sulphate, $(C_{17}H_{23}NO_2)_2H_2SO_4$] is 1/240 to 1/60 grain.

The following are the pharmacopœial preparations of belladonna:—

1. *Belladonnæ Folium.*—Balladonna leaf. It must not yield less than 0.3 per cent of the alkaloids.

2. *Belladonna Pulverata.*—Pulvis Belladonnæ. Dose, ½ to 3 grains.

3. *Extractum Belladonnæ Siccum.*—It contains 1 per cent of the alkaloid. Dose, ¼ to 1 grain.

4. *Tinctura Belladonnæ.*—It contains 0.03 per cent of the alkaloid. Dose, 5 to 30 minims.

5. *Belladonnæ Radix.*—Belladonna root. Dose, ½ to 2 grains.

6. *Emplastrum Belladonnæ.*—It contains 0.25 per cent of the alkaloids.

7. *Extractum Belladonnæ Liquidum.*—It contains 0.75 per cent of the alkaloids of the root. Dose, ¼ to 1 minim.

8. *Linimentum Belladonnæ.*—It conatins 0.375 per cent of the alkaloids.

9. *Suppositorium Belladonnæ.*—Each contains 1|60 grain of the alkaloids.

The following official preparations are derived from atropine sulphate :—

1. *Lamella Atropinæ.*—Each disc contains 1|5000 grain of atropine sulphate.

2. *Oculentum Atropinæ.*—It contains 0.25 per cent of atropine sulphate.

3. *Oculentum Atropinæ cum Hydrargyri Oxido.*—It contains 0.125 per cent of atropine sulphate and 1 per cent of yellow mercuric oxide.

Symptoms.—These are dryness of the mouth and throat with diminution of saliva, intense thirst, difficulty in swallowing and hoarseness in speaking. Nausea, vomiting and diarrhœa may, sometimes, be present. The face is flushed, the eyes are suffused, the eyelids are swollen and the vision is impaired or double. The pupils are widely dilated and insensible to light, but a case is recorded in which the pupils were normal,[1] while Philips cites a case in which the pupils were contracted.[2] The skin is hot and dry with a marked rise in temperature which may reach 107° or 108° F. in cases of severe poisoning. Sometimes, a scarlatinal rash or exfoliation of the skin is seen over most of the body. The pulse is at first slow, full and bounding, and afterwards becomes feeble and rapid. The respirations are slowed at first, and later become quicker and somewhat deeper. The patient feels giddy, staggers, is unable to walk from in-coordination of the muscles and, sometimes, suffers from convulsions. These symptoms are followed by delirium of fantastic hallucinations and delusions, drowsiness, stupor, coma and death. Usually the patient has no recollection of the symptoms after recovery.

Fatal Dose.—This is variable. A decoction of 80 grains of belladonna root used as an enema caused the death of a woman, 27 years old, in 5 hours.[3] Three berries have proved fatal to a child, nine months old,[4] and 14 berries have caused the death of an old man.[5] On the other hand, recovery has occurred after eating 50 berries.[6] A tea-spoonful of belladonna liniment,[7] a drachm of the tincture[8] and the same quantity of the extract[9] have respectively caused death. Recovery has, however, followed the ingestion of larger doses of these pharmacopœial preparations. Usually one grain of atropine, taken internally, is considered a fatal dose, although

1. *Wood, Brit. Med. Jour., Feb.* 21, 1885, *p.* 377.
2. *Ophthalmic Record, Jan.*, 1903, *XII, p.* 5 ; *Peterson, Haines and Webster, Leg. Med. and Toxic., Vol. II, Ed. II, p.* 23.
3. *Casper, Wochens., Feb.* 8, 1845, *Taylor, On Poisons, Ed. III, p.* 761.
4. *Lancet, Aug.* 29, 1846.
5. *Dixonmann, Forens. Med. and Toxic., Ed. VI, p.* 503.
6. *Med. Times, Aug.* 24, 1874.
7. *Beddoc, Lancet, July* 16, 1870.
8. *Buchanan, Forens. Med. and Toxic., Ed. VI, p.* 372.
9. *Brit. Med. Jour., Nov.* 20, 1869.

half-a-grain of atropine has proved fatal.[1] The smallest quantity of atropine reported to have proved fatal is 0.006 grain. It was dropped into the eye of a child, 4 years old.[2] One-thirteenth to one-tenth grain of atropine sulphate taken in solution has caused the death of a woman, 57 years old.[3] One-twentieth grain of atropine injected hypodermically in mistake for strychnine has killed an adult.[4] Recoveries have, however, taken place after the administration of much larger doses, even as much as 7.5[5] and 7.7[6] grains of atropine sulphate. An ointment containing 0.21 gramme of atropine applied to an abraded skin has caused death.[7]

Fatal Period.—In rapidly fatal cases death occurs in 3 to 6 hours. Ordinarily it occurs within 24 hours, although it may be delayed for days.

Treatment.—The same as in datura poisoning.

Post-mortem Appearances.—Belladonna seeds or fragments of the leaves and other parts of the plant may be found in the contents of the stomach and intestines. The stomach may or may not be congested, but its mucous membrane may be found reddened, if death has been caused by belladonna berries. The throat, trachea and œsophagus may be hyperæmic. The lungs are usually congested or œdematous. The liver is slightly congested. The brain and its membranes are usually hyperæmic.

Tests.—1. *Physiological Test.*—The alkaline ether extract (dry) by Stas' process is taken up with a few drops of distilled water and applied to a cat's eye. Dilatation of the pupil is produced.

2. *Vitali's Test.*—A little atropine is treated with a few drops of fuming nitric acid, heated to boiling, and evaporated to dryness on a water-bath. The residue is then moistened with a drop of freshly prepared alcoholic potassium hydroxide solution, when a purple-violet colouration is produced, changing slowly to dark red and finally disappearing. The colour may be made to reappear by adding more alcoholic solution of potassium hydroxide.

3. Auric chloride gives a citron yellow precipitate to a solution containing atropine. If the precipitate be recrystallised from boiling distilled water, acidified with hydrochloric acid, it will show a minutely crystalline appearance, and when dry will appear dull and pulverulent. It has a melting point of about 137° C.

4. An alcoholic solution of bromine gives a yellow, crystalline precipitate with atropine.

Medico-Legal Points.—Poisoning by belladonna occurs accidentally from an overdose of its pharmacopœial preparations or from swallowing

1. *Taylor, Princ. and Pract. of Med. Juris., Vol. II, Ed. IX, p.* 754.
2. *Burrenich, Ann. et bull. Soc. Med. de Gand.*, 1891, *p.* 288 ; *Witthaus, Manual of Toxic., Ed. II, p.* 873.
3. *Jaenicke, Deut. Arch. f. Klin. Med.*, 1877, *XX, p.* 617 ; *Witthaus, Manual of Toxic., Ed. II, p.* 873.
4. *Fabris, quoted by Modica, Gazz. d. Osp.*, 1898, *XIX, p.* 683 ; *Witthaus, Med. Juris. and Toxic., Ed. II, Vol. IV, p.* 873.
5. *Comroe, Jour. Amer. Med. Assoc., Aug.* 5, 1933, *p.* 446.
6. *Macchiavelli, Gazz. med. it. lomb.*, 1880 ; *Witthaus, Loc. Cit.*
7. *Ploss. Zeits. f. Chir.*, 1863 ; *Glaister, Med. Juris. and Toxic., Ed. VI, p.* 657.

"eye drops" in mistake. Sometimes, children suffer from poisoning by eating accidentally the berries or seeds, though they are relatively less susceptible than adults. Cases of accidental poisoning have also occurred owing to idiosyncrasy from the external application of belladonna liniment or plaster. Knight Rayson[1] reports a case in which poisonous symptoms appeared on the application to the loin of 3 drachms of belladonna liniment. I have seen a case in which a solution of atropine dropped into the eyes to dilate the pupils for retinoscopic examination produced mild symptoms of poisoning. Jones[2] describes a case where eight children suffered from the symptoms of poisoning after 5 drops were instilled into each eye of a 2 per cent solution of atropine which was dispensed by a druggist in place of homatropine. Plummer[3] reports the case of a woman, aged 67, who was poisoned within half-an-hour after 3 drops only of half-a-per cent solution of atropine sulphate had been instilled into each lower conjuctival sack. One hour and-a-half later, she was pulseless and much collapsed, but recovered after several hours. George Heller[4] records a case in which a boy, aged 6 years, suffered from toxic symptoms after two drops of a 1 per cent aqueous solution of atropine sulphate had been dropped into each nostril at 2 p.m. and again at 6 p.m. in place of a 3 per cent aqueous solution of ephedrine sulphate. An inhabitant[5] of Dohad in Gujarat instilled into his ears ear drops containing belladonna. The same night he was seized with severe headache, vomiting and diarrhœa, became unconscious and died. Atropine was detected in the viscera and in the residue of the ear drops.

Firth and Bentley[6] report three cases of belladonna poisoning resulting from eating the flesh of a rabbit which had been feeding on belladonna leaves. Winder and Manley[7] also record the case of a woman, aged 46 years, who suffered from symptoms of belladonna poisoning after she had taken ½ ounce of the liquid extract of liver as well as 2 drachms of the extract as a remedy for pernicious anæmia. On analysis the liver extract showed the presence of atropine of the strength of 1/25 grain per fluid ounce. She was thus poisoned by 1/50 grain as also by 1/100 grain of the alkaloid. It appears that belladonna leaves and fruit had been eaten by the animals from whose livers the extract was manufactured.

Suicidal cases have occurred from swallowing the liniment or extract. Homicidal cases are very rare. In one case a man mixed the seeds in soup, which he took to bring a false charge of poisoning against his wife.[8] A case is recorded in which a woman, aged 50 years, was first drugged with atropine and then murdered by her throat being cut with a sharp cutting instrument.[9] Atropine with cocaine was supposed to have been given by Clark of Agra to Fulham to simulate the symptoms of heat apoplexy.

1. *Brit. Med. Jour., April* 25, 1908, *p.* 987.
2. *Jour. Amer. Med. Assoc., March* 19, 1921, *p.* 813.
3. *Brit. Med. Jour., Feb.* 1, 1930, *p.* 226.
4. *Jour. Amer. Med. Assoc., March* 9, 1929, *p.* 800.
5. *Bombay Chem. Analyser's Annual Report*, 1927, *p.* 4.
6. *Lancet, Oct.* 29, 1921, *p.* 901.
7. *Brit. Med. Jour., Feb.* 29, 1936, *p.* 413.
8. *Bachner, Friedreich's Blatter f. Ger. Med.*, 1887; *Dixonmann, Forens. Med., and Toxic., Ed. VI, p.* 504.
9. *Bengal Chem. Exam. Annual Rep.*, 1922, *p.* 6.

Atropine is eliminated from the system chiefly by the kidneys. Consequently it can be detected in an unchanged condition in the urine.

Homatropine hydrobromide (Homatropinae hydrobromidum).—This is a salt of homatropine, an artificial or synthetic alkaloid, prepared by the condensation of tropine with mandelic acid in the presence of hydrochloric acid. It is a white, crystalline powder, soluble in 6 parts of water and in 18 parts of alcohol (90%). The dose is 1/64 to 1/32 grain. It is a constituent of *Lamella homatropinæ*, each disc containing 1/100 grain. It is largely used in ophthalmic practice, as its effects subside more quickly than those of atropine. A case occurred in the King George's Hospital, Lucknow, where 20 to 25 drops of a 1 per cent solution of homatropine hydrobromide instilled into the eyes for a period of 3 hours caused some poisonous symptoms.

COCAINE (METHYL BENZOYL-ECGONINE), $C_{17}H_{21}NO_4$

Cocaine is an alkaloid derived from the leaves of Erythroxylum Coca and its varieties (N. O. *Linaceæ*), growing in South America, but now cultivated in the tea districts of India, Ceylon and Java.

Cocaine is a colourless, odourless, crystalline substance and has a bitter taste, causing numbness of the tongue and mucous membrane of the mouth. It is soluble with great difficulty in water, but dissolves readily in alcohol, ether, chloroform and benzene, the solution being alkaline. It is a pharmacopœial preparation, known as *Cocaina*, the dose being $\frac{1}{8}$ to $\frac{1}{4}$ grain. The B.P.C. ointment, *Unguentum cocainæ*, contains 4 per cent of cocaine.

In the form of cocaine hydrochloride it is largely used as a local anæsthetic in ophthalmic practice, and in dental and minor operative surgery. Cocaine hydrochloride exists in colourless, acicular crystals. It is soluble in water, alcohol, chloroform and glycerine. Its solution is neutral and decomposes in a short time, but keeps better if mixed with half-a-per cent solution of boric acid. The official dose is $\frac{1}{8}$ to $\frac{1}{4}$ grain.

Cocaine hydrochloride is contained in the official preparations of *Oculentum cocainæ* (0.25 per cent), *Lamella cocainæ* (1/50 grain in each disc) and *Trochiscus krameriæ et cocainæ* (each containing 1/20 grain).

Synthetic substitutes, such as beta-eucaine, stovaine, novocaine, etc., are frequently used in surgical practice as local anæsthetics, and although much less toxic than cocaine, have produced poisonous symptoms followed, sometimes, by fatal results.

Novocaine is largely added to cocaine as an adulterant or is used as a substitute for the same. Addicts have to consume a large quantity of novocaine and suffer from its poisonous effects, as it does not produce the same effects as their usual dose of cocaine. A case occurred in Patna, where a Hindu male, aged about 30, who happened to be a cocaine eater, took a large quantity of novocaine which was sold to him as cocaine, became unconscious in half an hour and died in about four hours.[1]

Besides novocaine, boric acid, carbonate and bicarbonate of soda, lime, chalk, aspirin, antifebrin, antipyrin, and starch are also used as adulterants of cocaine.

1. *Bengal Chem. Examiner's Annual Rep.*, 1936, *p.* 13.

Acute Poisoning.—This is marked by excitement with delirium of a noisy character, followed by depression, as cocaine, when absorbed into the blood, first stimulates and then paralyses the nerve centres of the brain and spinal cord.

Symptoms.—Dryness of the mouth and throat; dysphagia; feeling of tingling and numbness in the tongue, hands and feet; nausea but rarely vomiting; cramps in the stomach; headache; giddiness; faintness; marked cyanosis; dilated pupils; quick, irregular and imperceptible pulse; shallow, gasping and convulsive respirations; profuse perspiration chiefly on the forehead; convulsions; paralysis. Death occurs from respiratory paralysis or from heart failure. Delirium and hallucinations may occur.

A case occurred in King George's Hospital, Lucknow, where a young student of 20 years suffered from symptoms of acute poisoning within an hour after his nose had been plugged for ten minutes with a pledget of cotton wool soaked in a 4 per cent solution of cocaine hydrochloride and sprayed twice with the same solution for submucous cauterisation of the inferior turbinate. In all about a drachm of the solution had been used. The patient became excited and restless, and began to shout and talk at random. He was delirious. His mouth and throat were very dry, and his face became flushed. He complained of intense thirst. The temperature was 101° F. The pupils were dilated. The respirations were hurried and the pulse was feeble and rapid. An attempt was made to produce vomiting, but of no avail. He was then given a tablet of luminal and became quiet within an hour (4 hours after the onset of the symptoms). The mouth became moist 5 hours later. He recovered completely the next morning.

Fatal Dose.—Two-thirds of a grain injected hypodermically caused the death of a woman, 71 years old,[1] and 20 minims of a 4 per cent solution injected into the urethra proved fatal.[2] Twenty-two grains of cocaine injected into the rectum killed a young girl.[3] The average fatal dose may be considered to be 15 grains taken by the mouth, though 10 and 12 grains have respectively produced fatal results.[4] It should, however, be remembered that much larger doses can be tolerated by habit.

Fatal Period.—Death usually ocurs early and suddenly. It occurred in one case in 3 minutes after the injection into the urethra of 30 minims of a 10 per cent solution of cocaine hydrochloride,[5] and in 20[6] and 40[7] minutes respectively in two other cases. A young woman in Bareilly died in 3 to 4 hours as a result of having taken cocaine in excess.[8] A Parsi lad,[9] 16 years old, died in 3 hours and 50 minutes after the use of cocaine as a local anæsthetic for the removal of the tonsils. The operation was successful.

Treatment.—Use emetics or wash out the stomach with warm water containing finely powdered charcoal if cocaine has been taken by the mouth. Wash out the mucous membrane, if it has been applied to the

1. *Dixonmann, Forens. Med. and Toxic., Ed. VI, p.* 511.
2. *Mathieson, Dublin Jour. Med. Sci.,* 1895.
3. *Vinogradoff, Lancet, Sep.* 28, 1889, *p.* 656.
4. *Mannheim, Ztschr, F. K. Med.,* 1890, *XVI, p.* 380; *Witthaus, Med. Juris. and Toxic., Vol. IV, p.* 901.
5. *Tivy, Brit. Med. Jour., Oct.* 6, 1906, *p.* 866.
6. *Zambianchi, Gazz. degli Ospidali,* 1888; *Dixonmann, Loc. Cit.*
7. *Johnston, Brit. Med. Jour., Vol. II,* 1895, *p.* 1162.
8. *U. P. Chemical Examiner's Annual Report,* 1925, *p.* 4.
9. *Gaekwar, Med.-Leg. Jour., July-August,* 1931, *p.* 119.

nose or to the throat. Try to ligate off the part as far as possible, if it has been taken hypodermically. Keep the patient in a recumbent posture. Administer stimulants, such as ammonia, digitalis, caffeine, strychnine, and coramine. Administer chloroform to combat the convulsions but do not give morphine, which may endanger life, as it hastens respiratory failure.

Amyl nitrite is considered an antidote and should, therefore, be given by inhalation. Carry on artificial respiration, if necessary.

Mayer[1] advises the use of calcium chloride to inhibit the toxic action of cocaine. M. Reese Guttman[2] has found that phenobarbital (luminal) is the best remedial agent in the treatment of cocaine poisoning, and suggests the prophylactic use of three grains by the mouth 30 minutes before anæsthesia. It can also be given hypodermically. Tatum and his co-workers[3] recommend the use of 100 mg. of soluble barbital dissolved in 5 c.c. of a saturated solution of paraldehyde per kilogramme of body weight.

Post-mortem Appearances.—Marked hyperæmia of the brain, spinal cord and other internal organs.

A woman, about 30 years old, was found alive at about 11 p.m. on the 16th June, 1914, and was found dead on the following morning at 11-30 a.m., when her paramour came to visit her. The post-mortem examination showed that the mucous membrane of the stomach was slightly congested. The stomach contained a quantity of semi-digested foodstuffs with no particular smell. The pupils were slightly dilated and the internal organs were congested. Cocaine in marked quantity was detected in the viscera.[4]

Chronic Poisoning (Cocainophagia or Cocainomania).—This occurs among those who have been accustomed to its use either by internal administration or by subcutaneous injection.

Symptoms.—Insomnia; digestive derangements; wasting; emaciation; rapid pulse; impotence; defective memory; physical and moral degeneration; derangement of the special senses; visual and other hallucinations; melancholia and mania with delusions of persecution.

The characteristic symptom, known as Magnan's symptom, and complained of by the patient, is a feeling as if grains of sand were lying under the skin, or some small insects (cocaine bugs) were creeping on the skin, giving rise to itching sensation. The tongue and teeth of the habitual cocaine eater in India are jet black, probably due to the chemical change brought about by lime and saliva acting upon cocaine.[5]

H. Hartmann[6] reports that homosexuality is often seen among cocaine addicts, and cites several cases of men and women who got into this habit after they took to cocaine and the perversion disappeared after the drug was stopped.

---

1. *Schw. Med. Wchnschr., Aug.* 18, 1921, *p.* 767; *Jour. Amer. Med. Assoc., Oct.* 15, 1921, *p.* 1290; *Fabry, Munch. Med. Wchnschr.*, 1922, *Vol.* 69, *p.* 969.
2. *Jour. Amer. Med. Assoc., March* 10, 1928, *p.* 753.
3. *Jour. Amer. Med. Assoc., March* 10, 1928, *p.* 754.
4. *Bengal Chem. Exam. Annual Rep.; Ind. Med. Gaz., Aug.*, 1915, *p.* 304.
5. *K. C. Bose, Ind. Med. Gaz., March*, 1902, *p.* 85.
6. *Deutsche Medizinische Wochenschrift, Berlin, Feb.* 27, 1928, *p.* 268; *Jour. Amer. Med. Assoc., April* 28, 1928, *p.* 1418.

Treatment.—This consists in the gradual withdrawal of the drug from cocaine addicts and in the treatment of gastric derangements, etc., with appropriate remedies.

Detection.—1. *Physiological Test.*—Cocaine produces numbness and local anæsthesia at the point of application. The condition lasts for about half-an-hour.

2. *Giesel's Test.*—A solution of potassium permanganate gives a fine bright, violet precipitate, which shows rhombic crystals arranged in rosettes when seen under the microscope.

3. *Hankin's Test.*—Cocaine is dissolved in a saturated or semi-saturated solution of alum. A drop of potassium permanganate solution is spread out, and dried on a glass slide. A drop of the alum solution is placed in the permanganate film and covered with a cover slip. The characteristic crystals of permanganate of cocaine form almost immediately. Under the microscope these crystals are seen to be rectangular in shape and pale pink in colour.

This test is of such delicacy that it can be used to reveal the presence of cocaine on a small piece of paper in which this substance has been

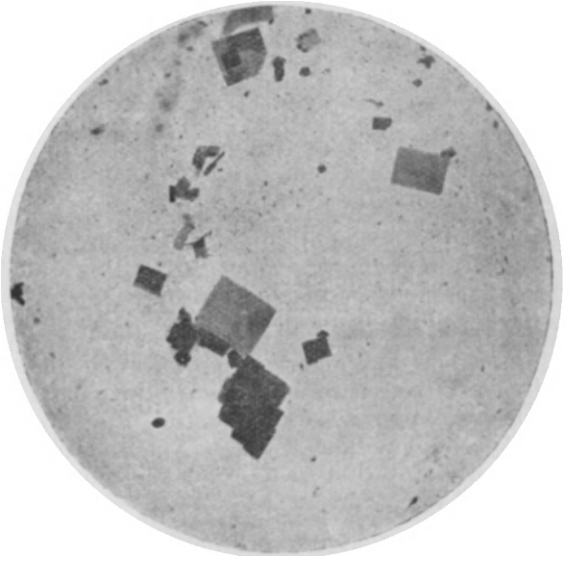


Fig. 146.—Microphotograph of Cocaine crystals × 200 (*R. B. Dr. K. N. Bagchi*). (*Obtained by Hankin's test*).

Fig. 147.—Microphotograph of Cocaine crystals × 200 (*R. B. Dr. K. N. Bagchi*). (*Obtained by Bagchi's modified method of Hankin's test*).

wrapped. Similar but easily distinguishable crystals are also formed by Alypin, Tropacocaine, and Scopalamine. No crystals are formed by Beta-eucaine, Stovaine, Novocaine, Holocaine, and Nirvanine.[1] Antipyrin which is often mixed with cocaine interferes with the test and should be

1. *U. P. Chem. Exam. Annual Rep.*, 1911; *Analyst, Jan.*, 1911, *Vol. XXXVI, p.* 2.

removed. The powder should, therefore, be dissolved in water and ammonia added to it, when cocaine would be precipitated. This should then be filtered and the residue should be tested.

Dr. Bagchi, Chemical Examiner to the Government of Bengal, has adopted the following modification in the method of this test :—

If a trace of cocaine or cocaine hydrochloride is dissolved in a few drops of a saturated solution of alum and a small drop of this solution is added to a drop of a saturated solution of potassium permanganate on a microscopic slide and the two are mixed together by gently rubbing on the slide for about a minute or two and then covered with a cover slip and examined under the microscope, small characteristic crystals of cocaine permanganate are seen. If the slide is left aside for about fifteen minutes much larger crystals are formed and are easily seen under the microscope.

If the drops of cocaine and potassium permanganate solutions are mixed gently and without rubbing and the slide is left uncovered and allowed to evaporate almost to dryness, the same crystals but of a very large size are formed.

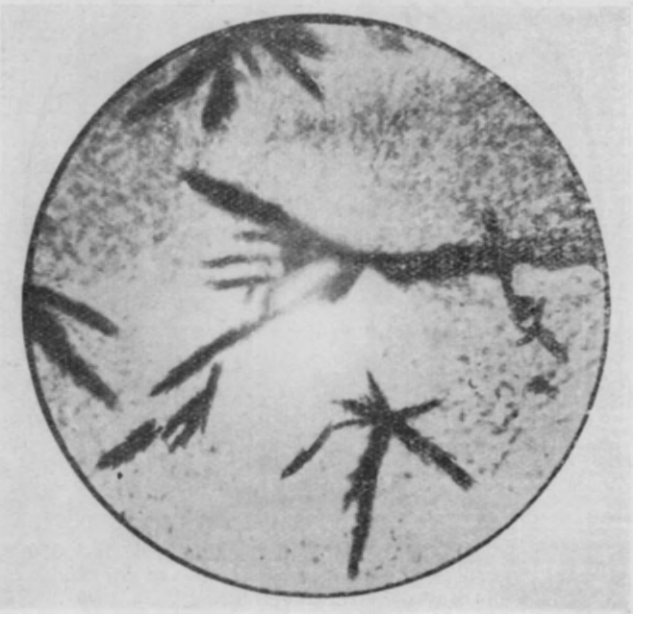


Fig. 148.—Microphotograph of Cocaine crystals × 150 (*K. B. Dr. N. J. Vazifdar*). (*Obtained by gold chloride test with cocaine solution*, 1 *in* 500).

If the solution of cocaine is very weak, rubbing on the slide helps to form the crystals within a minute, otherwise longer time is required.

This modification of Hankin's method is useful in obtaining the crystals quickly in a dilute solution of cocaine and in developing larger crystals in stronger solutions in a shorter time.

4. *Gold Chloride Test.*—A 5 per cent solution of gold chloride in distilled water gives a precipitate with a solution containing cocaine. The precipitate is at first amorphous, but rapidly becomes crystalline. Viewed under the miscroscope, the crystals are found to be delicate rosettes, or long rods resembling fern-fronds, generally with a stellate arrangement. This is a delicate test and a few crystals are formed even with a solution of 1 in 20,000.

Gold chloride solution also gives a crystalline precipitate with novocaine, but the novocaine gold chloride compound is soluble in dilute hydrochloric acid while the cocaine gold chloride compound is insoluble in the same acid.

Bagchi and his collaborators[1] have made use of this fact in devising a method of carrying out the determination of a small quantity of cocaine in a sample adulterated with novocaine.

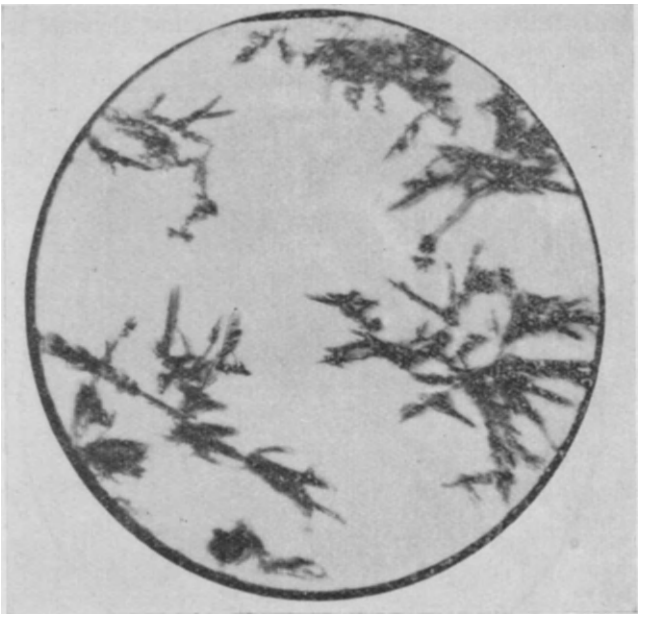


Fig. 149.—Microphotograph of Cocaine crystals × 150 (*K. B. Dr. N. J. Vazifdar*). (*Obtained by gold chloride test with cocaine solution,* 1 *in* 1000).

The presence of chalk, antifebrin, aspirin, starch, etc., along with novocaine does not interfere with the determination. They are easily removed from the solution by a preliminary filtration, but the presence of alkaline carbonates and lime necessitates the use of stronger (20 per cent) hydrochloric acid. In other cases 10 per cent acid is quite good.

5. *Chromic Acid Test.*—A 5 per cent solution of chromic acid or a 7.5 per cent solution of potassium bichromate, added drop by drop to a

1. *Indian Med. Gaz., Jan.,* 1939, *p.* 29.

solution of cocaine hydrochloride, produces a yellow precipitate which disappears immediately on shaking. If 1 c.c. of concentrated hydrochloric acid is then added to the clear solution, a more or less crystalline orange precipitate is formed.

Medico-Legal Points.—Accidental cases of poisoning by cocaine have occurred from internal use, from hypodermic injection, and from urethral, vesical and rectal injections.

A few cases of suicide have been recorded. Like opium, cocaine is believed to be an aphrodisiac and to increase the duration of the sexual act by paralysing the sensory nerves of the glans penis. Hence young men indulge in its use. It may be used for this purpose by local application, but it is ordinarily taken in *prepared pan.* The habit once established is difficult to be given up. About a grain of cocaine hydrochloride is first taken, but the craving for the drug soon increases and the daily ration is increased to 30 grains or even more. K. C. Bose[1] reports a case in which a man, aged 52 years, was taking daily a few grains less than two drachms and another case of a Mahomedan boy, 12 years old, who was in the habit of taking 12 grains every day. This pernicious habit has become so very common that Government forbids the possession of cocaine without a licence.[2] When, owing to the Great War, supplies of smuggled cocaine became difficult to procure, anæsthesin (ethylester of para-amido-benzoic acid), a preparation of cocaine, was used instead. A solution of it was applied to the glans penis before intercourse. It is possible that a 5 per cent solution of this drug, thus used, might be found of benefit in cases in which owing to excessive excitability, the sexual act cannot be properly performed.

Prostitutes, sometimes, inject a solution of cocaine into the vagina by means of a douche can. This gives the individual a sense of local constriction and the general systemic effects appear immediately.[3]

In England, some persons are accustomed to use cocaine hypodermically. In Paris, certain classes of people use it in the form of snuff, and addicts use large quantities—about a drachm a day on an average. The snuff produces irritation of the nasal mucous membrane. The irritation causes inflammation and ulceration which may occasionally lead to perforation of the nasal septum.

Cocaine is rarely smoked with the cigarette or pipe tobacco. During the smoking one observes " a euphoric mood, and an agreeable feeling of lightness and coolness in the head."[4]

A very small portion of cocaine is eliminated in the urine. It is largely decomposed in the human system ; hence it is difficult to be detected in the viscera.

1. *Ind. Med. Gaz., March,* 1902, *pp.* 86-87.
2. Vide *Appendix X.*
3. *R. N. Chopra and G. S. Chopra, Indian Jour. of Med. Research, Jan.,* 1931, *p.* 1013.
4. *Erich Leschke, Clin. Tox., Engl. Trans. by Stewart and Dorrer,* 1934, *p.* 208.

HYOSCYAMUS NIGER (HENBANE, *KHORASANI AJWAYAN*)

This plant belongs to N. O. *Solanaceæ*, and grows wild throughout the Himalayan range. All parts of the plant are poisonous, but the seeds are more poisonous. The seeds, leaves, and green flowering tops yield two active principles, hyoscyamine and hyoscine, and a fixed oil.

Hyoscyamine occurs both as a crystalline and as an amorphous alkaloidal substance. It is slightly soluble in water but freely in alcohol (90%), chloroform and ether. It is isomeric with atropine, into which it can be readily converted. It may be split up into hyoscine and hyoscinic acid. Hyoscine is a syrupy alkaloid synonymous with scopolamine. It is slightly soluble in water, but readily dissolves in alcohol (90%), ether, chloroform and dilute acids. It is considered five times more powerful therapeutically than hyoscyamine. Its official preparation, *Hyoscinæ hydrobromidum* (Hyoscine or scopolamine hydrobromide), occurs in colourless, transparent, rhombic crystals, having a slightly bitter taste. It is soluble in water and alcohol (90%). The dose is 1/200 to 1/100 grain. It is contained in *Oculentum hyoscinæ* (strength, 0.125 per cent).

Pharmacopoeial Preparations.—The following are the official preparations of hyoscyamus :—

1. *Hyoscyamus.*—Hyoscyamus leaves or henbane leaves. Dose, 3 to 6 grains.

2. *Extractum hyoscyami liquidum.*—It contains 0.05 per cent of the alkaloids. Dose 3 to 6 minims.

3. *Extractum hyoscyami siccum.*—It contains 0.3 per cent of the alkaloids. Dose, ¼ to 1 grain.

4. *Pilula colocynthidis et hyoscyami.*—It contains 12.5 parts of dry extract of hyoscyamus. Dose, 4 to 8 grains.

5. *Tinctura hyoscyami.*—It contains 0.005 per cent of the alkaloid, hyoscyamine. Dose, 30 to 60 minims.

Symptoms.—These are the same as in belladonna poisoning, but delirium is not so marked, while there is greater tendency to sleep, insensibility and general paralysis of the nervous system. In addition to these, nausea, vomiting, purging, spasmodic contractions of the muscles and hallucinations may be present.

A case[1] is recorded in which a man, 52 years old, suffered from the following symptoms during and shortly after the course of treatment with the hypodermic injection of hyoscine hydrobromide grain 1/100 once a day, then twice a day and eventually 1/75 grain twice a day, though the treatment was occasionally omitted for a few days :—

Twitchings of the arms and legs, profuse sweating of the body and limbs, pallor of the nose, closed eyes, dilated pupils, stuporose condition, no loss of rigidity, picking movements of fingers, no response to questions. After recovery the patient had no remembrance of having any indisposition at all.

A druggist took, for suicidal purposes, 500 mg. of scopolamine and a few minutes later became unconscious. His face became markedly red, the mucous membrane of his mouth very dry and his pupils were widely dilated. His extremities were flaccid, a trace of Babinski reflex was present in the right foot, and twitchings were present in the right arm. The tendon reflexes were normal, and the pupil reaction was less. The stomach was washed out, and twenty hours after the poison was taken the patient was conscious and quite sensible.[2]

Fatal Dose.—Not certain. Owing to an individual idiosyncrasy medicinal doses of the alkaloids have produced toxic symptoms. One-fourth to half-a-grain of hyoscine hydrobromide may be regarded as a fatal dose.[3] Seven grains of the non-official

1. *Stanley E. Denyer, Brit. Med. Jour., Jan.* 1, 1927, *p.* 16.
2. *F. Lickint, Munch. Med. Woch.*, 1931, *II*, *p.* 1991 ; *The Med.-Leg. and Criminol. Rev., Jan.*, 1933, *p.* 83.
3. *Willcox, Brit. Med. Jour., Oct.* 29, 1910, *p.* 1375.

extract of hyoscyamus (dose 2 to 8 grains) have produced alarming symptoms, and the tincture has also given rise to the poisonous effects after three or four doses of 10 minims administered every six hours.[1]

Fatal Period.—Not known.

Treatment.—Emetics or stomach tube; tannin; tea; coffee; pilocarpine hypodermically.

Post-mortem Appearances.—Not characteristic. The seeds may be found in the stomach. Congestion of the brain and lungs has been found.

Detection.—The seeds are kidney-shaped, about $\frac{1}{8}$" in diameter, covered with small projections and are of a brown or grey colour.

Tests.—1. Hyoscyamine and hyoscine are mydriatic alkaloids; hence a solution of either of them, if put into the eye of a cat, causes dilatation of the pupil.

2. Hyoscyamine forms with auric chloride solution a gold double salt, which melts at 165° and 200° C.

Hyoscine treated with auric chloride solution yields a yellow precipitate, which, recrystallised from water, forms bright yellow glistening needles, having a melting point of 165° and 200° C.

3. An alcoholic solution of bromine in hydrobrombic acid forms needleshaped crystals with a solution of hyoscyamine, but round spheres with a solution of hyoscine.

Medico-Legal Points.—An accidental fatal case has occurred in 24 hours from the root used as a vegetable in mistake for parsnip.[2] The seeds have been mistaken for celery seeds, and have produced poisonous symptoms.

The dried leaves and flowers are smoked like *ganja* by depraved persons and *Fakirs* in Sind. The juice of the fresh leaves, and the dried leaves are used in the treatment of irritable affections of the lungs, bowels and genito-urinary organs. The juice and oil are also used for external applications. In 1910, hyoscine hydrobromide was used by Crippen, an American homœopathic doctor, for killing his wife. Two-fifths of a grain of the salt were estimated to be present in the organs submitted to Willcox for analysis. This amounted to more than half-a-grain in the whole body.[3]

Hyoscine (Scopolamine) is used in combination with morphine in producing the so-called twilight sleep. It has caused toxic effects, followed by a few deaths. One-eighth grain of morphine and 1/100 grain of scopolamine hydrobromide injected hypodermically has caused death.[4]

The following plants belonging to N. O. *Solanaceæ* have produced poisonous symptoms which are due to solanine, an active principle, contained in them. It is readily hydrolised by mineral acids into solanidine. It acts as a gastro-intestinal irritant and narcotic:—

1. Solanum Dulcamara (Woody nightshade).—The berries are known as *Inab-es-salib*. Two cases[5] of cattle poisoning by these berries are reported. In one case one foal died, and in the other several cows died.

2. Solanum Indicum (*Barhanta, Dolimoola*).

3. Solanum Jacquinii (*Katai, Bhooiringni*).

4. Solanum Nigrum (*Kakmachi, Makoi*).

5. Solanum Tuberosum (*Alu*).

1. *Taylor, On Poisons, Ed. III, p.* 674.
2. *Wibmer, Pois. Vegetables of Great Brit., p.* 3.
3. *Brit. Med. Jour., Oct.* 29, 1910, *p.* 1375.
4. *Ely, New York Med. Jour.*, 1906, *LXXXIV, p.* 799.
5. *H. Lowe, Analyst*, 1929, *p.* 153; *Jour. of State Med., June*, 1929, *p.* 368.

Symptoms.—Nausea ; vomiting ; diarrhœa ; colic ; tenesmus ; giddiness ; widely dilated pupils ; cramps in the legs ; muscular spasms ; drowsiness ; delirium ; coma. Death occurs from respiratory paralysis.

Fatal Dose and Fatal Period.—Uncertain. Two berries of solanum dulcamara [1] have caused the death of a child, four years old, in thirty-two hours. Brown reports a case in which 5 children died after eating the berries of solanum nigrum.[2]

Treatment.—Wash out the stomach. Give morphine hypodermically. Keep up the body warmth and use stimulants.

Chemical Analysis.—Solanine is extracted from the viscera by the Stas-Otto method, but as it is practically insoluble in ether and chloroform, warm amyl alcohol is used for a final extraction from ammoniacal solution. The following are the most useful tests[3] :—

1. A concentrated solution of the alkaloid in amyl alcohol sets to a jelly-like consistence.
2. Phosphomolybdic acid gives a cream-coloured precipitate.
3. Nitric acid gives a purple colour on warming.
4. Ethyl sulphuric acid gives a red colour.
5. Concentrated sulphuric acid with bromine water gives a red colour forming in streaks.

CANNABIS SATIVA OR INDICA (INDIAN HEMP)

This plant belongs to N. O. *Urticaceœ*, and is cultivated largely all over India. It yields an amorphous resin, *cannabinone*, which contains a phenol-aldehyde, *cannabinol* and some substances allied to choline.

The forms in which cannabis indica is used in India are—

1. *Bhang, Siddhi* or *Sabji.*—This consists of the dried leaves and fruiting shoots. It is used as an infusion in the form of a beverage, which produces intoxication of a sensuous character. It is prepared by rubbing on a stone slab, sugar, black pepper and dried leaves, and is taken in the form of a bollus or pill, or is mixed with water and strained through a muslin cloth before it is drunk. This is the favourite beverage, especially of the Hindus, in the Northern parts of India.

The intoxication produced by it is of the most cheerful kind causing the individual to sing and dance, to eat food with great relish and to seek sexual enjoyment. The intoxication lasts about three hours when sleep supervenes.

2. *Majun.*—This is a sort of confection prepared from *bhang* after treating it with sugar, flour, milk and butter. It has an agreeable odour and a sweet taste. It is sold in the *bazaar* in small lozenge-shaped pieces. One to three drachms are enough to intoxicate a person, who feels great appetite and a sexual desire. He also feels quite happy and contented as though he belongs to some *Raja's* family, and has got all what he wants. Sometimes *datura* is mixed with *majun.*

3. *Ganja.*—This consists of the flowering or fruiting tops of the female plant coated with resin and grown on the plains. It is mixed with a little

1. *Lancet, June* 28, 1855.
2. *Dymmock, Pharmaco. Ind., Vol. II, p.* 555.
3. *H. Lowe, Analyst,* 1929, *p.* 153 ; *Jour. of State Med., June,* 1929, *p.* 368.

tobacco, and is usually smoked in a pipe (*chilam*). The person using the smoke feels heavy and lazy, and indulges in pleasant reveries, though he is able to discharge his ordinary duties. It is largely indulged in by *Sadhus* and *Fakirs*.

The flowering tops (*Ganja*) are the constituents of the non-official preparations of *Extractum cannabis* (dose $\frac{1}{4}$ to 1 grain), *Tinctura cannabis* (dose 5 to 15 minims), and *Cannabinœ Tannas* (dose 4 to 8 grains).

4. *Charas.*—This is commonly known as *hashish*, and is the resin exuding from the leaves and stems of the plants, which grow on mountainous regions from six to eight thousand feet above the sea-level. It is smoked with tobacco in a pipe or a *hukka*, and is the most potent of all the forms.

Symptoms.—Persons not accustomed to its use or from an overdose suffer from toxic symptoms. They appear soon after smoking *ganja* or *charas* and within half an hour after swallowing *bhang*, and are chracterized by two stages ; stage of intoxication and stage of narcosis.

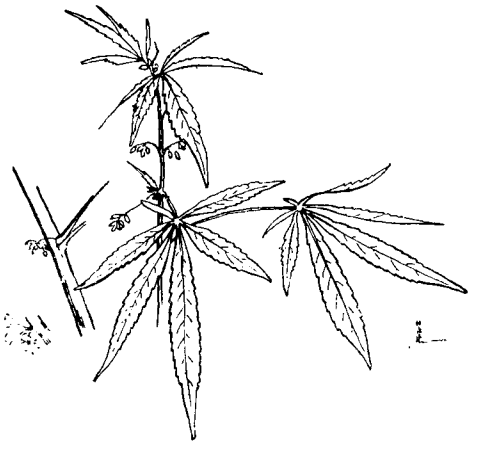


Fig. 150.—Cannabis Sativa.

The first stage is characterized by excitement with hallucinations, laughing and foolish talk. The patient loses all perception of time and space, gets dreadful hallucinations, becomes wildly delirious and, sometimes, has a homicidal tendency. He feels giddy, complains of tingling and numbness of the skin, becomes drowsy, suffers from muscular weakness and then passes into the second stage of narcosis with dilated pupils. In severe poisoning there may be general anæsthesia. Recovery usually follows a deep sleep. Death, although extremely rare, may occur from respiratory failure.

Chronic Poisoning.—This occurs from the excessive consumption of cannabis indica in one or more of its forms for a prolonged period. The symptoms are loss of appetite, general weakness, trembling, loss of sexual power, slothfulness, hallucinations and insanity (mania and dementia).

Fatal Dose.—Unknown. Seven minims of the tincture of cannabis indica have produced toxic symptoms in a female, aged 30 years.[1] Twelve pills, each containing half-a-grain of the extract taken by a woman to cure headache produced poisonous symptoms.[2] Eight ounces of Neurosine equivalent to 4.8 grains of cannabis indica taken in two days and-a-half produced alarming symptoms in a woman.[3]

1. *Lancet, Sep.* 30, 1871.
2. *Baxter-Tyrie, Lancet, Vol. II,* 1897, *p.* 1452.
3. *G. Creswell Burns, Jour. Amer. Med. Assoc., April* 11, 1931, *p.* 1225.

Fatal Period.—Death is very rare but it has ensued in twelve hours. Death has also been delayed till the nineteenth day.[1]

Treatment.—Evacuation of the stomach, cold affusions to the head, strychnine hypodermically and artificial respiration.

Post-mortem Appearances.—Not characteristic.

Fig. 151.—Microphotograph of *ganja* hairs × 250.
(*Rai Bahadur Dr. K. N. Bagchi*).

Detection.—Extract the resin by digesting the suspected material in alcohol. The resin, when given to a dog, will produce narcosis with swaying of the head from side to side.

Bhang and *ganja* can be detected under the microscope as fragments of green leaves covered thickly with curved claw-shaped, sharp pointed hairs.

Beam's Test.—The suspected material extracted with petroleum ether and evaporated to dryness in a small porcelain capsule gives a rich purple or reddish-purple colour when a few drops of weak alcoholic solution of potash or soda are added.

This test is obtained with the resinous material or *hashish* but not with the preparations of cannabis indica. Hence Beam devised the following modified test to which cannabis indica and its preparations could respond:—

The petroleum ether extract is prepared and evaporated in a short test tube. To the residue are addded a few cubic centimetres of a reagent

1. *Dixonmann, Forensic Med. and Toxicology, Ed. VI, p.* 509.

prepared by passing dry hydrogen chloride gas through absolute alcohol to saturation. In the presence of cannabis extract the liquid strikes a bright cherry-red colour, which disappears on dilution with alcohol or water.[1]

The Chemical Examiner, Madras, has found that the resin of *ganja*, which yields Beam's test with alcoholic hydrochloric acid, gives also a characteristic purple colour in chloroform solution with acetic anhydride and a drop of sulphuric acid. He, therefore, recommends the use of this latter reaction as an additional test to Beam's test. He has adopted the following method for detecting *ganja* in viscera :—

The minced viscus is macerated with a mixture of barium chloride and barium hydroxide solutions and the aqueous filtrate is extracted with petroleum ether. The aqueous solution is drawn off, acidulated with acetic acid and again extracted with petroleum ether. This second petroleum ether solution contains the resin and may be used directly for the tests. A few calcium chloride granules are added to the petroleum ether solution and after shaking to remove any water, the petroleum ether is divided into two portions : (1) To one portion a solution of anhydrous hydrochloric acid in absolute alcohol is added and gives, in the presence of *ganja*, a pink colour (Beam's test). (2) To another portion a little chloroform is added followed by a drop of concentrated sulphuric acid and a few drops of acetic anhydride, when a purple colour develops in the solution.[2]

Medico-Legal Points.—Poisoning by *bhang* is mostly accidental. *Majun* and *charas* have been occasionally used by road poisoners to stupefy persons to facilitate robbery. Chevers[3] mentions a case in which one Luxmee of Ahmadnagar gave *majun* to a boy, aged 7 years, and then murdered him for the sake of his ornaments. A case[4] is recorded in which one Sankothi Thakur was sentenced to four years' rigorous imprisonment on the charge of administering *charas* to a fellow passenger and stealing his purse in a railway train. The accused and the complainant entrained at Howrah for their homes in Ballia and Muzaffarpur. At the complainant's request for a *biri* the accused mixed *charas* with a half-burnt cigarette. He first smoked it and next the complainant. The latter felt intoxicated and uneasy, and slept in the train by the side of the accused. Awaking at Mokameh, he found his money missing and demanded it from the accused. At Patna City railway station, the accused was arrested, and on a search by the police, the amount alleged to have been stolen was found on his person.

In his annual report for the year 1934, the Chemical Examiner of Madras describes a case in which cannabis indica was administered for homicidal purpose. A father killed his son by administering a powder containing cannabis indica leaves, as there was enmity between them.

1. *Wellcome Tropical Research Laboratories, Chemical Section Bulletin No.* 3, *April*, 1915 ; *Lucas, Forensic Chemistry and Scientific Criminal Investigation, Ed. III, p.* 344.
2. *Madras. Chem. Exam. Annual Rep.*, 1937, *p.* 7.
3. *Med. Juris., p.* 225.
4. *Leader, June* 21, 1933.

Sometimes, people take cannabis indica to steady their nerves before committing a crime. A case is recorded in which a Mahomedan male took a pill of *bhang* or *majun* to get intoxicated and then hacked his wife to pieces with a sword.[1] Rarely, people, after the continued use of cannabis indica, run *amok*, *i.e.*, they first kill a person or persons against whom they have entertained fancied or real enmity and then go on killing everybody that comes in their way until the homicidal tendency lasts. They then commit suicide or quietly submit themselves to the police. It must be remembered that people, sometimes, run *amok*, even though they are not addicted to the use of cannabis indica. The following is a typical example[2] of running *amok* at Allahabad, where four innocent persons were murdered and several wounded in the course of about 15 minutes :—

On the evening of January 29, 1931, a Punjabi Muslim was selling tumblers made of sulphur when a constable of the C. I. D. sitting on a shop suspected him to be a person wanted in connection with a counterfeit coining case. The constable thereupon caught hold of his hand and asked him to go with him, when he dropped the tumblers and whipped out a knife. Seeing the knife the constable released the accused and on the latter running away, the constable shouted 'chor chor.' As the accused took a turn into the passage between the cloth and vegetable markets, a coolie caught him when the accused struck him with the knife and the coolie left him shouting 'chor chor'. The coolie, however, escaped with a few cuts on his hand. The accused resumed the flight, and near the end of the same passage he stabbed a young Hindu lad who was standing on the way, perhaps making some purchases, and while taking the Garhi-ki-sarai road he stabbed another Hindu youth, who was going with his cycle. During his flight on this road he wounded some people and stabbed two more men who were Mahomedans. Proceeding further the accused was encountered by a Hindu male who managed to throw him on the ground. The accused, however, succeeded in getting up again and as he resumed the flight several people attacked him with *lathis*. At this stage the C. I. D. constable who was pursuing him arrived and caught hold of the accused's hands, when the accused bit the constable's nose. Eventually with the help of the public the man was secured.

A case[3] also occurred in Bombay, where a Bhayya, who was addicted to the smoking of *charas*, used to squat on Hornby road, plying his trade as a palmist. On the night of December 18, 1938, he had a quarrel with his client, and then suddenly started running along the road with a spring knife in his hand and stabbing persons that came in his way till he was grappled, disarmed and arrested by two police constables, who happened to be passing along Bori Bunder tram junction. During his mad career he stabbed ten persons, one after the other, four of whom died subsequently.

Neurosine, which contains 6/10 grain (0.04 gm.) of cannabis indica in each fluid ounce may cause poisonous symptoms, when taken in large doses. A woman[4] was ordered to take a drachm of neurosine three or four times a day but instead, she took it every two hours day and night for two days and a half. In this way she took 8 ounces of neurosine, and lapsed into deep sleep which lasted twenty-four hours. She was aroused and subsequently she was delirious and irrational. The axillary temperature on the fifth day was 97° F., the pulse rate was 92 per minute and respirations 24. She was unable to respond to questions and was completely out of touch with her environment. The skin was warm and flushed. The pupils were equally dilated and reacted promptly to light. The lips were

1. *Chevers, Med. Juris., p.* 790.
2. *Leader, Feb.* 1, 1931.
3. *Times of India, Dec.* 19, 1938, *p.* 9.
4. *G. Creswell Burns, Jour. Amer. Med. Assoc., April* 11, 1931, *p.* 1225.

parched. There was incontinence of fæces and urine. The knee jerks were equal and moderately exaggerated. She was given ampoules of caffeine sodiobenzoate by the mouth and physiological solution of sodium chloride intravenously. She recovered ultimately.

ARTEMISIA MARITIMA (WORM-WOOD, *KIRMANI OWA*)

This plant belonging to N. O. *Compositæ* grows on the coasts of England, and yields an active principle, *santonin*, chiefly from santonica or wormseed, the dried unexpanded flower heads. The other varieties, Artemisia brevifolia and Artemisia vulgaris, grow in Kashmere and the hilly tracts of the United Provinces of Agra and Oudh.

Santonin is a glucoside and occurs as flat, glittering, prismatic crystals. It is either tasteless or faintly bitter. It is colourless, but becomes yellow on exposure. It is slightly soluble in water, but is easily soluble in alcohol, chloroform and alkalies. The dose is 1 to 3 grains.

Symptoms.—Giddiness, pain in the stomach, vomiting, yellow vision (xanthopsia), dilated pupils, cold skin bathed in perspiration, feeble and slow pulse and respirations, convulsions, delirium, stupor, coma, and death ending the scene from failure of the heart or respiration. The urine is usually increased in quantity, and has an intensely yellow colour, if acid, but it assumes a purplish-red colour, if alkaline or if an alkaline solution is added to the acid urine. Sometimes, strangury and hæmaturia are observed owing to irritation of the kidneys.

A girl, 4 years old, was given $2\frac{1}{2}$ grains of santonin. About two hours later, she complained of abdominal pain, and became uneasy and restless. Subsequently she vomited several times, and fell into a semi-comatose condition with dilated pupils and distended tympanitic abdomen. Death took place about 48 hours after the administration of the drug. At the post-mortem examination the stomach and intestines were yellowish stained internally and filled with blackish material with a strongly ammoniacal odour.[1]

Fatal Dose and Fatal Period.—One hundred and fifty-five grains of wormseed proved fatal to a girl, aged 16 years, in about three days.[2] Two grains of santonin administered twice killed a child, five and-a-half years old, in twelve hours.[3] A Hindu girl, aged 16 years, died in about an hour after taking an overdose of some "worm powder" containing santonin.[4] Recovery has taken place in the case of a child after ten grains[5] and in the case of an adult after an ounce.[6]

Treatment.—Give emetics or wash out the stomach by the stomach tube, and give stimulants to combat collapse. In cases of convulsions, potassium bromide and chloral hydrate should be administered.

Post-mortem Appearances.—Not characteristic. In a case[7] where a man, aged 22, died shortly after he had taken santonin as a remedy for some disease, the post-mortem examination showed that the stomach was anæmic and empty, and the liver and kidneys were congested.

Tests.—Strong H_2SO_4 + Heat + Ferric chloride forms a red colour, becoming yellow and lastly brown.

Medico-Legal Points.—Cases of poisoning occur accidentally among children from an overdose given as an anthelmintic to remove round worms from the intestines. A case is recorded in which a man was poisoned after taking one-fourth of a pint of

1. *Bombay Chemical Analyser's Annual Report*, 1924, *p.* 2.
2. *Taylor, On Poisons, Ed. III, p.* 682.
3. *Dixonmann, Forens. Med. and Toxic., Ed. VI, p.* 536.
4. *Bom. Chem. Analyser's Annual Rep.*, 1935, *p.* 5.
5. *Dixonmann, Forens. Med. and Toxic., Ed. VI, p.* 536.
6. *Ibid., Annali Univ. di. Med.*, 1882.
7. *Punjab Chem. Examiner's Annual Rept.*, 1934, *p.* 11.

the infusion as an aphrodisiac.[1] An adult took an ounce of santonin in mistake for Epsom salts, but recovered as mentioned above.

Santonin is eliminated slowly by the kidneys, and has a tendency to accumulate in the system. Hence it may act as a poison, if administered for a prolonged period even in medicinal doses. A boy, 11 years old, was given santonin for months, and consequently suffered from paralysis, twitchings, dizziness, pain in the head, vomiting, yellow and violet vision, sparks before the eyes, and loss of speech. Under proper medical treatment he was able to walk in six weeks, but regained the power of speech after nine weeks.[2]

Oil of absinthe (oil of wormwood) extracted by distillation from artemisia absinthum is used as an abortifacient, sometimes with fatal results. A woman died in three-quarters of an hour after swallowing 100 grains of oil of absinthe, which she had procured for the purpose of terminating her pregnancy. A few minutes after she took the drug, she was found lying speechless.[3] A case is also recorded, in which a man took half-an-ounce probably for the cure of worms and was found perfectly insensible, convulsed and foaming at the mouth. The pulse was weak, slow and compressible. From time to time he uttered incoherent expressions and attempted to vomit. He recovered after repeated administrations of stimulants, sal volatile and water, lime water and an emetic containing mustard or zinc sulphate.[4]

A French liqueur, called absinthe, contains this oil with a large proportion of alcohol, and is largely used in France. When taken in excess, it acts as a narcotic poison, the chief symptoms being digestive disturbances, giddiness, tingling in the ears, and illusions of sight and hearing followed by numbness in the limbs, loss of intellect, general paralysis and death. According to Magnan epileptic attacks are the special features in poisoning by absinthe.[5]

CAMPHOR (*KAFOOR*), $C_{10}H_{16}O$

This is stearoptene obtained from the wood of Cinnamomum camphora (Camphora officinarum) belonging to N. O. *Lauraceæ*. It is artificially produced by the direct union of oil of turpentine and hydrochloric acid. It occurs as white, translucent crystals or crystalline masses, having a pungent, bitter taste and a peculiar, fragrant, penetrating odour. It floats on water in which it is almost insoluble, but it is dissolved by alcohol, ether, chloroform, milk and oils. It is extremely volatile and inflammable, burning with a bright light and much smoke. The dose is 2 to 5 grains and 1 to 3 grains by subcutaneous injection. It is a constituent of the following official preparations:—

1. *Aqua camphoræ*.—Strength, $\frac{1}{2}$ grain to 1 fluid ounce. Dose, $\frac{1}{2}$ to 1 fluid ounce.

2. *Linimentum camphoræ*.—It is commonly known as camphorated oil. Strength, 20% of camphor.

3. *Linimentum camphoræ ammoniatum*.—It is commonly known as compound liniment of camphor. It contains 12.5 per cent of camphor.

4. *Spiritus camphoræ*.—Strength, 1 in 10. Dose, 5 to 30 minims.

5. *Tinctura opii camphorata*.—It is also called Paregoric or Tincture camphor compound. It contains 3 parts of camphor in 1000 parts of alcohol. Dose, 30 to 60 minims.

Camphor is widely used as a personal disinfectant and as a preservative of clothing against an attack of moths. When rubbed into the skin, camphor acts as an irritant, causing redness and heat. When taken internally in poisonous doses, it acts as an irritant to the stomach, and after absorption it acts first as a stimulant and then as a depressant to the nerve centres.

1. *Robinson, Lancet, Vol. I*, 1889, *p.* 770.
2. *Rey, Therap. Montashefte*, 1889; *Dixonmann and Brend, Forensic Medicine and Toxicology, Ed. VI, p.* 536.
3. *Brit. Med. Jour., Aug.* 16, 1902, *p.* 504.
4. *Taylor, On Poisons, Ed. III, p.* 681.
5. *Ibid.*

Borneo Camphor or *Borneol*, $C_{10}H_{18}O$, is derived from Dryobalanops aromatica, and is ordinarily met with in commerce in place of camphor, from which it can be distinguished by sinking in water.

Symptoms.—Burning pain in the mouth and stomach, nausea, vomiting, flushed face, cyanosed lips, dilated pupils, vertigo, convulsions, delirium, unconsciousness, coma and death. The breath, vomit and urine have the odour of camphor.

Fatal Dose.—Twenty grains have proved fatal to an adult, though recovery has followed larger doses.[1] One drachm of camphorated oil killed a child, 5 years old, while a tea-cupful of the oil containing between one and two hundred grains administered to a child was recovered from.[2]

Fatal Period.—Uncertain. A child, 2 years old, died in 4 hours after swallowing about 15 grains of camphor.[3] A child of 16 months died in 7 hours after having a tea-spoonful of camphorated oil containing 12 grains of camphor.[4] A child[5] of the same age died in about 12 hours after swallowing at least half-an-ounce of camphorated oil. A child, aged 2 years and 8 months, died in 18 hours after eating a solid piece of camphor, weighing about 30 grains.[6]

Treatment.—Evacuation of the stomach, warmth to the body, cold affusions to the head, saline purgatives, stimulants, and artificial respiration if necessary.

Post-mortem Appearances.—Signs of gastro-intestinal inflammation with erosion or ulceration of the stomach. The stomach contents may show a solid piece of camphor, or may give off the odour of camphor.

At the post-mortem examination of the body of the child,[7] who died after swallowing a tea-spoonful of camphorated oil containing 12 grains of camphor, the skin showed a profuse hæmorrhagic eruption all over the body, and the mucous membrane of the mouth and lips was slightly excoriated. The stomach and intestines showed a few petechial hæmorrhages under the peritoneal coat and mucous membrane. There were numerous small hæmorrhages in the cortex, just under the capsule in both kidneys.

Detection.—Camphor may be separated by distillation from organic fluids. The distillate is shaken out with benzene and evaporated on a warm water-bath, when the camphor remains as a residue. The residue is then purified by several recrystallisations from 50 per cent alcohol.

There are no chemical tests for camphor, but it can be identified by its bitter, pungent taste, characteristic odour and spontaneous volatility. It melts at 176° C.

Medico-Legal Points.—Accidental cases of camphor poisoning have occurred from the pharmacopœial preparations (liniment and spirit) having been drunk in mistake for other preparations, such as castor oil, etc. Sen[8] mentions a case in which a female, aged about 18 years, swallowed about a drachm or more of camphor in water and suffered from poisonous symptoms. Among others the chief symptoms were twitchings of the fingers with tingling sensation, lock-jaw and delirium. She recovered in seven or eight days. A female child, 6 years old, swallowed about four drachms of camphor oil at 7-30 p.m. on September 16, 1930, and immediately she felt nausea. Her throat was tickled to make her vomit and she brought up a little of the oil. But she became drowsy in a short time, and was removed to King George's Hospital, Lucknow, at 8 p.m. At the time of admission she was found in a drowsy condition with the eyes closed. The pupils were dilated. There were muscular twitchings all over the body. The pulse was rapid and feeble, and the respirations were slow and laboured. The stomach was first washed out with saline and then with

1. *Glaister, Med. Juris. and Toxic., Ed. VI, p.* 685.
2. *Wilkinson, Brit. Med. Jour., Vol. I,* 1898, *p.* 299.
3. *Finley, Medical Record,* 1887, *Vol. I, p.* 125.
4. *Clark, Brit. Med. Jour., March* 15, 1924, *p.* 467.
5. *Barker, Brit. Med. Jour., April* 16, 1910, *p.* 921.
6. *Davies, Brit. Med. Jour., Vol. I,* 1887, *p.* 726.
7. *Clark, Brit. Med. Jour., March* 15, 1924, *p.* 467.
8. *Ind. Med. Gaz., Sep.,* 1917, *p.* 336.

potassium permanganate, when the twitchings became less. The pulse became very feeble, and ½ c.c. pituitrin was administered hypodermically. The pulse improved and the twitchings gradually subsided. Next morning the lips were found swollen and the buccal mucosa necrosed at places. Magnesium sulphate was given as a purgative, and the patient was discharged cured after 24 hours.

A case of suicide is recorded in which a European female, 39 years old, swallowed about 2 ounces of camphor liniment, but recovered under prompt treatment.[1] A case[2] is also reported where a man took camphor with a view to commit suicide and died soon afterwards.

Poisonous symptoms resulting in death in some cases have followed its use as an abortifacient.

POISONOUS FUNGI (MUSHROOMS)

The common varieties of poisonous fungi are Amanita muscaria and Amanita phalloides. Amanita muscaria is known as the fly agaric, because its decoction is used for killing flies. It grows singly in sandy soil and attains a large size. It has a hollow stalk which is solid and bulbous at the base and has gills which are always of a pure white colour. The pileus varies in colour from yellow to orange and red, and is covered by warty scales.

The fungus owes its poisonous properties to an alkaloid, *muscarine*, which is a crystalline substance, soluble in water and alcohol, but insoluble in chloroform and ether. It is alkaline and deliquesces in the air forming a syrupy liquid. It contracts the pupils when administered internally but dilates them when applied locally.

Amanita Phalloides is commonly called the deadly agaric, and is white in colour, having an unpleasant taste and giving off a fœtid odour when old. It grows to a height of about four to six inches in woody places. It has a hollow stalk with a prominent bulb at the base, the upper margin of which is formed into a vulva or cup. The pileus is usually white but may vary in colour from pale dull yellow to olive, and has gills covered with white spores on its under surface.

The fungus is a powerful poison and contains two active principles, *amanita hæmolysin* (*Phallin* of Kobert) and *amanita toxin*.[3] Amanita hæmolysin is a hæmolytic glucoside, which is precipitated by alcohol and is completely destroyed when heated to 70° C., or when digested with pepsin as well as pancreatin. Amanita toxin is the chief poisonous principle which is dissolved by alcohol, but is not destroyed by heat or digestive ferments.

Symptoms.—These are divided into two groups, irritant and neurotic.

1. *Irritant Symptoms.*—The symptoms are usually delayed for six to ten hours or for thirty hours in some cases. These are constriction of the throat, burning pain in the stomach, nausea, painful retchings, vomiting, and diarrhœa, the stools containing blood ; the urine may contain blood and albumin. These are followed by cyanosis, small pulse, laboured respirations, convulsions, profuse sweating, collapse and death. Sometimes there may be anuria.

2. *Neurotic Symptoms.*—These are giddiness, headache, delirium, diplopia, dilatation, sometimes contraction of the pupils, tetanic spasms, insensibility and coma.

In some cases irritant symptoms may be present, and in others neurotic only. The predominance of one or the other group of symptoms depends on the nature of th alkaloids present.

Fatal Dose.—Uncertain.

Fatal Period.—Death usually occurs within twenty-four hours. Plowright[4] records four fatal cases of poisoning by amanita phalloides at King's Lynn. The

1. *Ind. Med. Gaz., June*, 1902, *p.* 210.
2. *Free Press Jour., Nov.* 25, 1932.
3. *Ford, Brit. Med. Jour., Dec.* 1, 1906, *p.* 1541.
4. *Brit. Med. Jour., Sep.* 9, 1905, *p.* 541.

first, a boy, aged 12, ate about a third of the uncooked pileus (top) of the fungus and died after 81½ hours. The second, a boy of 5, died 68 hours after eating an unknown quantity of the fungus. In the third case a man, aged 32, gathered some amanita phalloides, which he and his family consisting of the wife, aged 22 years, a daughter, 7 years, and a son, aged 22 months, ate for their tea, the total quantity being three-quarters of a pound. During the preparation of the meal the mother and the boy ate a certain quantity of the raw fungi. They all became ill. The boy died fifty-four hours after eating the mushrooms, and the mother died on the fourth day. The father and the daughter recovered.

Fig. 152.—Amanita Muscaria.

Treatment—Evacuate the stomach by giving common salt as an emetic or by lavaging it with water containing potassium permanganate or finely powdered charcoal. Give castor oil to clear the bowels. Atropine is considered a physiological antidote to muscarine, and should be administered hypodermically. Morphine may be administered hypodermically to relieve pain. Give stimulants and normal saline subcutaneously. Repeated intravenous infusions of 300 to 500 c.c. of 10 to 20 per cent sugar solutions with eventual addition of calcium salts are considered very useful.[1]

Limcusin and Petit[2] recommend the administration of the fresh stomach and brain of a rabbit in poisoning by amanita phalloides. A family of four persons partook of A. phalloides, and one died. The other three had serious symptoms. They were given each three fresh rabbit stomachs mashed and some fresh brains, and a rapid recovery ensued.

Post-mortem Appearances.—The signs of inflammation of the mucous membrane of the alimentary canal are present, if the irritant signs have been predominant.

1. *Zeitschrift fur Klinische Medizin, Berlin, March* 3, 1926; *Jour. Amer. Med. Assoc., May* 1, 1926, *p.* 1407.
2. *Bull. de l' Acad. de Med., May,* 1932, *p.* 24; *Med. Annual,* 1933, *p.* 500.

Fatty degeneration of the liver, kidneys and heart may also be found. In cases of neurotic symptoms congestion of the brain vessels, and subpleural and subpericardial hæmorrhages are likely to be met with.

Fig. 153.—Amanita Phalloides.

Medico-Legal Points.—Chevers[1] records the case of an Assistant Collector who felt drunk and laughed ludicrously in open Court after having eaten amanita mushrooms at his breakfast. In this connection it may be mentioned that the poor people of Siberia and Kamschatka manufacture an intoxicating beverage from the same fungus. The poison is excreted by the urine which possesses intoxicating properties and is drunk by persons to produce intoxication.

Some poisonous fungi lose their toxic properties when they are boiled, or when they are steeped in salt and vinegar for some time, while the edible ones become poisonous by being warmed some time after they have been cooked once.

Some edible fungi are rich in water and albumen and are, therefore, apt to decompose and may thus produce poisonous symptoms. It is also possible that owing to idiosyncrasy some persons may be poisoned by eating edible mushrooms. Frossard[2] records the case of a healthy young woman of 30 years who died in about five hours after eating some of the raw mushrooms which she was preparing for family breakfast.

1. *Med. Juris., p.* 280.
2. *Brit. Med. Jour., Feb.* 10, 1906, *p.* 319.

Along with her the other members of the family ate them after they had been properly cooked, but none of them devloped any poisonous symptoms.

POISONOUS FOOD GRAINS

Lathyrus Sativus (*Kesari Dal, Teora,* or *Buttorah ka Dal*).—This is a variety of pulse, belonging to N. O. *Leguminosœ,* and is used as an article of diet by the common people in Sind, United Provinces and some parts of Central India. Its continued use gives rise to a disease characterized by spastic paraplegia, known as lathyrism or vetch-poisoning. It was supposed to owe its toxic properties to water-soluble amines present in the *dal,* which increase in quantity during germination of the grain.[1] But Anderson, Howard and Simonsen[2] have carried out investigations, and are of opinion that *Kesari Dal* (*Lathyrus Sativus*) is by itself harmless and that the danger of the disease lies in its contamination with *Akta,* a leguminous weed called *Vicia Sativa.* In this connection it may be mentioned that Acton and Chopra[3] have now been able to confirm the work of Anderson and his co-workers by carrying out further investigations.

Symptoms.—The onset of the disease often comes on suddenly. On waking up in the morning or whilst working in the field, the patient may notice weakness in his legs and difficulty in sitting down and getting up from a squatting position. He is then unable to walk without the aid of a stick, and later assumes a spastic gait owing to the rigidity of the muscles of the calves and thighs. Lastly complete paraplegia of the lower limbs occurs. There is no atrophy or loss of the tone of the muscles and no reaction of degeneration. Sensation is normal, although there is muscular pain. The knee-jerks are increased, ankle clonus is well-marked and Babinski's sign is present. There is no loss of consciousness, nor is there any involvement of the bladder and rectum.

Prophylaxis.—The remedy for the prevention of lathyrism is to grow *Kesari Dal* in pure culture by removing *Akta* in the early stages so that when the crop flowers, it is practically pure.

Post-mortem Appearances.—Death in the acute stage is very rare. There may be sclerosis of the lateral columns of the spinal cord.

Lolium Temulentum (*Darnel, Mochni*).—This is a kind of grain belonging to N. O. *Graminaceœ.* Its seeds contain a glucoside, loluin, to which its poisonous properties are believed to be due. Accidental cases of poisoning have occurred from these seeds being ground in mistake with wheat or rye, and then made into bread.

Symptoms.—Giddiness, muscular weakness, tremors, symptoms of gastro-intestinal irritation, dilatation of the pupils and stupor. No case of death has yet been recorded.

Stigmata Maides (Maize, Indian Corn, *Maccai* or *Butta*).—This corn belongs to N. O. *Graminœ* and is cultivated everywhere. It is affected by a special kind of fungus, which causes pellagra, when eaten. However, pellagra is now regarded as a deficiency disease due to lack of fat-soluble A vitamin in maize.

Paspalam Scrobiculatum (*Kodro* or *Kodon*).—The poison is supposed to reside in the husk of the grain, which is often used by poor people as an article of food. The poison is removed by boiling.

Symptoms.—These are giddiness, intoxication, dilated pupils, tremors, delirium, convulsions, stupor and coma.

A family consisting of a woman, aged 50, a man, aged 22, and two boys, aged 9 and 12, was attacked by vomiting and giddiness about an hour and-a-half after taking an evening meal consisting of bread made from some flour of *kodon.* They then became unconscious. The pulse was small and quick, and the extremities cold. They regained consciousness in about an hour, but the young man was unconscious for some time. They all had tremors, and recovered the following morning.[4]

1. *Acton, Ind. Med. Gaz., July,* 1922, *p.* 241.
2. *Indian Jour. of Med. Research, April,* 1925, *p.* 613.
3. *Causation of Lathyrism by Vicia Sativa. Abstract of Papers, Far Eastern Association of Tropical Medicine,* 1927, *p.* 104.
4. *A. Swarup, Ind. Med. Gaz., July,* 1922, *p.* 257.

CHAPTER XXXII

SPINAL POISONS

STRYCHNOS NUX VOMICA (*KUCHILA*)

This tree belongs to N. O. *Loganiaceæ*, and grows in the jungles of Manbhoom, in the Madras Presidency, Malabar and Coromandel Coasts.

Its ripe fruit contains nux vomica seeds, which are poisonous. They are flat, circular discs, or slightly concave on one side and convex on the other, being $\frac{7}{8}$" to 1" in diameter, and $\frac{1}{4}$" in thickness. They are ash-grey in colour and have a shining surface with short satiny hairs. Internally they are tough, horny and slightly translucent, having no odour but possessing a bitter taste. They yield two principal alkaloids, *strychnine* and *brucine*, united with strychnic or igasuric acid as igasurates. Besides, the seeds contain to a small extent a glucoside, named *loganin*. The bark, wood and leaves contain brucine, but no strychnine. The dose of the powdered seeds (*Nux vomica pulverata* or *Pulvis nucis vomicæ*) is 1 to 4 grains.

The following trees belonging to N. O. *Loganiaceæ* also contain the same alkaloids :—

1. Strychnos Colubrina (Snake wood, *Kuchila-lata* or *Gogari lakdi*).

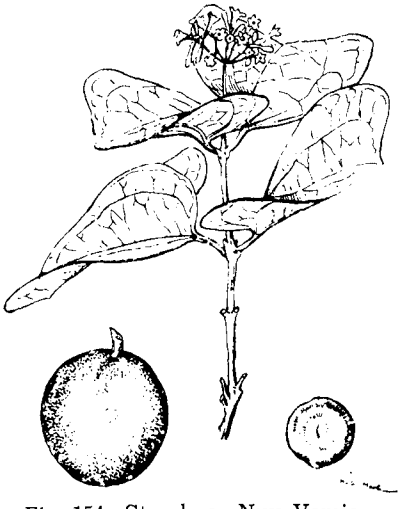


Fig. 154—Strychnos Nux Vomica.

2. Strychnos Ignatii (St. Ignatius' Beans, *Papita*).

3. Strychnos Tieute (Upas tree).—This is used in making arrow and dart poisons by the jungle tribes of the Malay Peninsula.[1]

Strychnine, C_{21} H_{22} N_2 O_2.—This crystallises in white, inodorous, rhombic prisms, having an intensely bitter taste. It dissolves in 5,760 parts of cold water, and in 2,500 parts of boiling water, but dissolves readily in acidulated water. It is a B. P. C. preparation, the dose being 1|32 to $\frac{1}{8}$ grain.

Strychnine is very stable, and does not change in the process of putrefaction, if present in the dead body. Hence it can be detected some years after death.

1. *Gimlette, Malay Poisons and Charm Cures, p.* 168.

Strychnine is largely used to destroy wild animals and vermin and, therefore, forms one of the chief constituents of several " vermin killers," which are usually mixed with some colouring material, such as Prussian blue, indigo or soot. These are sold in the chemist's shop as " Barber's Battle's, Butler's, Gibson's, Hunter's and Marsden's vermin killers and Miller's rat powders."

Brucine, C_{23} H_{26} N_2 O_4, $4H_2O$.—This occurs in colourless, prismatic crystals, with an intensely bitter taste. It is soluble in 3,200 parts of cold water, in 150 parts of boiling water, and freely in chloroform and amyl alcohol, but not in ether. It resembles strychnine closely in action, but is much weaker and less toxic, from 30 to 40 times as large a dose being required to produce the same effect.[1]

Both strychnine and brucine form salts, many of which are soluble in water.

Pharmacopoeial Preparations.—The pharmacopœial preparations of nux vomica and strychnine are—

1. *Extractum Nucis Vomicæ Siccum.*—Standardised to contain 5 per cent of strychnine. Dose $\frac{1}{4}$ to 1 grain.

2. *Extractum Nucis Vomicæ Liquidum.*—Standardised to contain 1.5 per cent of strychnine. Dose 1 to 3 minims.

3. *Tinctura Nucis Vomicæ.*—Standardised to contain 0.125 per cent of strychnine. Dose 10 to 30 minims.

4. *Strychninæ Hydrochloridum.*—Dose 1|32 to $\frac{1}{8}$ grain.

5. *Liquor Strychninæ Hydrochloridi* (*Liquor strychninæ*).—It contains 1 per cent of strychnine hydrochloride. Dose 3 to 12 minims.

6. *Syrupus Ferri Phosphatis cum Quinina et Strychnina* (Easton's syrup).—Each fluid drachm represents 1|60 grain of strychnine hydrochloride. Dose 30 to 60 minims.

Symptoms.—These supervene immediately after, or within five or ten minutes after, swallowing the poison ; sometimes they may be delayed for an hour or more.[2] A hot and intensely bitter taste is experienced during the act of swallowing if it happens to be in solution. This is followed by a choking sensation in the throat. The most marked effects due to its direct action on the spinal cord are the convulsions affecting all the muscles at a time. These are at first clonic, but eventually become tonic, as the intervals become shorter and the paroxysms longer. During the paroxysms the face becomes cyanosed, and wears an anxious look, the eyes are staring, the eye-balls prominent and the pupils are dilated. The features are drawn into a grin (the *risus sardonicus*), and the mouth is covered with froth, frequently stained with blood. The body is arched

1. *Cushny, Pharmacology and Therapeutics, Ed. VIII, p.* 284.

2. *In a case reported in the American Journal of Medical Science of April,* 1848, *the symptoms developed in two hours and-a-half and in the case of a boy of* 12 *years they developed* 3 *hours after he took a pill containing* 3 *grains of strychnine which had been prepared eight months ago* (*Lancet,* 1861, *Vol. II, p.* 480).

back in the position of *opisthotonos*, the unfortunate patient resting on his heels and occiput. The spasms of the diaphragm, drawing upon the ensiform cartilage, cause epigastric pains. The contractions of the respiratory muscles produce a sense of suffocation, which may end in asphyxia. Sometimes, the spasms of the abdominal muscles may bend the body forward (*emprosthotonos*) while, less frequently, the body may be flexed to the side (*pleurosthotonos*). The mind usually remains clear to the end of life, and the patient is conscious of the pain and impending danger of death. The reflex excitability is so great that the slightest movement of the patient, a sudden noise or the touch of a glass of water to the lips or even a flash of light is enough to induce the convulsions. Vomiting is readily induced, and persists when once excited. Death may occur from asphyxia during the first paroxysm, or any subsequent attack, or from exhaustion during the intervals as a result of painful spasms.

In cases ending in recovery, the convulsions become shorter and less active, and the period of intermissions is much longer.

Fatal Dose.—The usual fatal dose for an adult is ½ to 2 grains[1] of strychnine. The smallest amount of strychnine known to have proved fatal to a woman of 36 years is ¼ grain.[2] Half-a-grain of sulphate of strychnine has proved fatal.[3] One drachm of liquor strychninæ hydrochloridi containing 0.52 grain of strychnine killed a naval officer in 45 minutes.[4] Two, possibly three, of the tabloids of Easton's syrup equal to 1/32 and 1/20 grain of strychnine respectively proved fatal to a boy, three and-a-half years old, in about one and-a-half hours.[5] One-sixteenth of a grain of strychnine has killed a child between two and three years of age in four hours.[6] Three-fourths of a grain caused the death of a child of seven years and-a-half in half-an-hour.[7] On the other hand, recoveries after prompt treatment have ensued from very large doses, as much as, 10 grains of strychnine hydrochlorate,[8] 20 grains of strychnine sulphate[9] and even 40 grains of strychnine.[10] Dixonmann[11] mentions a case in which 22 grains of strychnine remained in the stomach for two hours, before vomiting occurred, yet recovery took place.

Thirty grains of powdered nux vomica equal to one seed in weight (1/3 grain of strychnine) given in two doses of 15 grains each proved fatal.[12] Recoveries have, however, followed larger doses. A woman,

1. *Taylor, On Poisons, Ed. III, p.* 712.
2. *Med. Times and Gaz., April* 15, 1854, *VIII (n. s.) p.* 376; *Collis Barry, Leg. Med., Vol. II, Ed. II, p.* 513.
3. *Warner, Poisoning by Strychnine, pp.* 138, 139.
4. *Littlejohn, Transactions of the Medico-Legal Society, Vol. XIX, p.* 13.
5. *Ibid., p.* 14.
6. *Taylor, Loc. Cit.*
7. *Ann. d.' Hyg.,* 1861, *t., I, p.* 133.
8. *Surbadhicarry, Ind. Med. Gaz., July,* 1894, *p.* 270.
9. *Wallace and Mc Rae, Brit. Med. Jour., July* 23, 1892, *p.* 179.
10. *Med. Gaz., Sep.* 21, 1865, *p.* 267 (*quoted by Glaister, Med. Juris. and Toxic., Ed. VI, p.* 666); *Wilson, Amer. Jour. of Med. Sc.,* 1864 *m. s., XLVIII, p.* 70 (*quoted by Witthaus, Med. Juris. and Toxic., Vol. IV, p.* 1030).
11. *Forensic Med. and Toxic., Ed. VI, p.* 489.
12. *Christison, Poisons, p.* 901.

aged 23, recovered after swallowing 120 grains of powdered nux vomica,[1] a Hindu male student, aged 19 years, recovered after taking six nux vomica seeds and a European male, aged 30, recovered after eating 8 nux vomica seeds.[2] Owing to the presence of the hard insoluble testa, the entire seeds may pass out of the bowel without producing poisonous symptoms. Three grains of the extract of nux vomica[3] and 6 drachms of the tincture[4] have respectively produced fatal results. Thirty drops of extract nux vomica liquid equivalent to $\frac{3}{4}$ grain of strychnine administered in mistake for extract ergot liquid proved fatal to a European woman of Calcutta in 2 hours and 45 minutes on April 28, 1923.

Fatal Period.—The usual fatal period is one to two hours. In a few cases death has occurred soon after swallowing the poison. In one recorded case a man died immediately after taking $1\frac{3}{4}$ grains of strychnine.[5] In another case death occurred in 5 minutes,[6] and in a third case it occurred in 10 minutes.[7] In the case of Cook (*Reg.* v. *Palmer*), death occurred in 20 minutes after the commencement of the symptoms. A lady, 25 years of age, died in 30 minutes after taking 1 grain of strychnine in mistake for exalgin.[8] In rare cases death has been delayed for several hours. An adult died in 6 hours from a dose of 3 grains of strychnine.[9] A man died in nine hours after taking about 7 or 8 grains of strychnine.[10] In a homicidal case death occurred in 12 hours after the administration of the poison in coffee.[11] A young girl died in 18 hours after a dose of 1 grain of strychnine taken with suicidal intent.[12]

Dignosis.—Strychnine poisoning has to be diagnosed from tetanus. The chief distinguishing points between the two are as follows:—

STRYCHNINE POISONING	TETANUS
1. Onset, sudden.	1. Onset, gradual.
2. All the muscles are affected at a time.	2. The muscles of the neck and lower jaw are affected first (Lock-jaw).
3. During the intervals the muscles are relaxed.	3. During the intervals the muscles are rigid.
4. Death takes place within a few hours. If death does not occur within four to six hours, the probability of recovery is great.	4. Death rarely takes place within 24 hours and may be delayed for several days.

1. *Taylor, On Poisons, Ed. III, p.* 693.
2. *Robertson Milne, Ind. Med. Gaz., June,* 1902, *p.* 211.
3. *Christison, Loc. Cit.*
4. *Hale, Brit. Med. Jour.,* 1899, *Vol. II, p.* 10.
5. *Amer. Jour. of Med. Sc.,* 1854, *p.* 557.
6. *Gray, Glas. Med. Jour.,* 1870-71, *III, p.* 167.
7. *Hunter, Med. Times and Gaz.,* 1867.
8. *Littlejohn, Transactions of the Medico-Legal Society, Vol. XIX,* 1925, *p.* 11.
9. *Guy's Hosp. Report,* 1857, *p.* 483.
10. *Henry, Australian Medical Gazette,* 1893, *XII, p.* 73.
11. *Lesser, Vrtljschr. f. ger. Med.,* 1898, 3 *F. XV, pp.* 268, 272; *Witthaus, Manual of Toxic., Ed. II, p.* 1033.
12. *Tardieu and Roussin, "Empoisonnement", Ed. II, p.* 1173; *Witthaus, Ibid., p.* 1033.

Treatment.—Give chloroform inhalation to check the spasms, and then introduce the stomach tube to wash out the stomach with warm water containing potassium permanganate, animal charcoal, tannic acid or tannin. In the absence of the tube, evacuate the stomach contents by the hypodermic administration of 1/10 to 1/5 grain of apomorphine hydrochloride. It also tends actually to quiet and prevent the convulsions. Three cases are reported in which the hypodermic use of apomorphine hydrochloride was followed by recovery in human beings who had taken presumably lethal quantities of strychnine.[1]

Large doses of potassium bromide and chloral hydrate should be given internally at frequent intervals. Chloral hydrate may be given in 30-grain doses by the rectum or in 5-grain doses hypodermically. Urethane (dose 1 to 4 drachms) is considered useful in controlling the convulsions.[2] Gentle narcosis, perfect quiet and dark surroundings are very essential. Nitrite of amyl and carbogen (a mixture of oxygen and 5% of carbon dioxide) may be administered by inhalation. Artificial respiration may be attempted, if respiratory paralysis supervenes.

Haggard and Greenberg[3] recommend the intravenous injection of phenobarbital sodium as a true antidote for strychnine poisoning. Wheelock[4] reports the recovery of a case of strychnine poisoning by the intravenous injection of a 5-grain ampoule of phenobarbital sodium and a 15-grain ampoule of sodium amytal dissolved in 10 c.c. of water. Fenton[5] describes the case of a man, aged 33, who had taken 15 grains of strychnine. He was cured by the intravenous administration of 5 grains of sodium amytal preceded by 6 grains administered orally.

Post-mortem Appearances.—Rigor mortis sets in more rapidly and may persist for a long time. In the case of Cook, the rigidity of the body and limbs was well marked on exhumation after two months' interment.[6] Usually the muscles are relaxed at the time of death and soon become extremely rigid, but in some cases the tetanic spasm may pass into cadaveric rigidity without the initial stage of relaxation. Livid patches may be observed on the body, and may be mistaken for bruises caused by violence.

The mucous membrane of the stomach and duodenum occasionally shows patches of ecchymosis or congestion. The liver and kidneys are generally congested. The heart is usually empty and contracted, but its right side is, sometimes, gorged with dark fluid blood. The lungs are congested. The brain and its membranes and the upper part of the spinal cord are found congested.

In a fatal case by strychnine poisoning which occurred at Lucknow on the 11th December, 1929, I found the following post-mortem appearances :—

1. *J. S. Martin, cited by Haggard and Greenberg, Jour. Amer. Med. Assoc., April* 2, 1932, *p.* 1134.
2. *Dixon, Manual of Pharmacology, Ed. VI, p.* 124.
3. *Jour. Amer. Med. Assoc., April* 2, 1932, *p.* 1133.
4. *Jour. Amer. Med. Assoc., No.* 26, 1932, *p.* 1862 ; *See also Kempf, Mc. Callum and Zerfas, Jour. Amer. Med. Assoc., Feb.* 25, 1933, *p.* 550.
5. *Jour. Amer. Med. Assoc.. Oct.* 21, 1933, *p.* 1133.
6. *Taylor, On Poisons, Ed. III, p.* 707.

The stomach was contracted and contained about an ounce of a pinkish fluid. Some mucus was adherent to the mucous membrane of the stomach which was congested. There were some submucous hæmorrhagic points along the greater curvature. The same appearances were found in the duodenum. The chambers of the heart were empty. The large vessels were gorged with blood. There were some subendothelial hæmorrhagic points on the surface of the right chamber of the heart. The lungs were slightly collapsed and were congested especially towards the base. On section they exuded dark fluid blood. The lining membrane of the larynx and trachea was cyanosed, congested and covered with froth towards the lower part. The pharynx was cyanosed, and so was the œsophagus in its upper part. The brain and the upper part of the cord were congested. The vessels of the cortex were engorged with blood. The liver, spleen and kidneys were congested.

Tests—Strychnine.—1. A bitter taste will be perceptible in a solution of 1 in 70,000 of water.

2. Sulphuric or nitric acid does not produce any colour change.

3. Strong sulphuric acid and potassium bichromate produce a violet colour, which gradually changes to bright red, then to rose pink and lastly to yellow. This is a delicate test, and reveals 1 : 50,000. Instead of potassium bichromate, potassium permanganate, manganese dioxide, lead peroxide or any other oxidising agent may be used.

Physiological Test.—A few drops of the suspected mixture injected subcutaneously into the abdomen or thoracic cavity of a frog will induce tetanic spasms.

Brucine.—1. Strong sulphuric acid and a crystal of potassium nitrate or nitric acid give a blood-red colour. On evaporating the mixture on a water bath, a brown residue is obtained which turns violet.

2. *Blyth's Test.*—Added to an alcoholic solution of brucine, methyl iodide produces in a few minutes circular, rosette-shaped crystals composed of methyl brucine iodide. Strychnine does not respond to this test, nor does it interfere with the test, if present along with brucine.

Medico-Legal Points.—Strychnine is one of the most deadly poisons. Accidental poisoning has resulted from an overdose or from it having been dispensed in medicine in mistake for some other harmless drug, such as quinine, salicin, jalapin, caffeine, etc. In 1919, a case occurred at John's Mills in Agra, where strychnine was accidentally dispensed instead of quinine with the result that seven persons died within an hour. The dose taken was probably ten grains. Of those who took the doses one is said to have had vomiting and blistering on the lips and to have recovered.[1] A similar case[2] occurred in Hoshiarpur District, where a medical man gave some tablets supposed to be of quinine to a family. The head of the family took four tablets, and distributed three tablets each to four members of his family. They all became ill and suffered from convulsions. The head of the family died and the other members

1. *U. P. Chemical Examiner's Annual Report*, 1919.
2. *Punjab Chemical Examiner's Annual Report*, 1928, *p.* 10.

fortunately recovered. The viscera of the deceased revealed the presence of strychnine on analysis. The remaining tablets were examined and found to be of pure strychnine. A case[1] is also recorded where a man died of strychnine poisoning. It was administered to him with jaggery as a quack antidote to ringworm.

Poisonous symptoms have also occurred accidentally from incompatible prescriptions containing potassium iodide or liquor arsenicalis and strychnine, when the latter precipitates to the bottom of the mixture and is taken with the last dose. A lady, 36 years old, consulted her medical attendant, who prescribed a mixture of ½ ounce of liquor arsenicalis and ½ ounce of liquor strychninæ hydrochloridi, six drops to be taken three times a day. One morning three weeks later she did not feel well, and thought a dose of the medicine would do her good. She had, however, finished the contents of the bottle, but noticing a little whitish deposit at its bottom added some water and drank off the contents. About an hour afterwards she suffered from strychnine poisoning, and died in two hours and twenty minutes after taking the medicine.[2]

Suicidal cases are common in England on account of the facility with which packages of "vermin killers" containing strychnine are obtainable in a chemist's shop. Owing to the ignorance of the people about strychnine suicidal poisoning is rare in India, though a few cases have lately been reported. In the annual report for 1921, the Chemical Examiner, U. P., reports a case in which a woman committed suicide by taking strychnine. In his annual report for 1923, the Chemical Examiner of Bengal describes a case in which an Anglo-Indian lady took a teaspoonful of strychnine at 5 a.m. with intent to commit suicide owing to a quarrel with her husband. She had convulsions for the first time at 6-30 a.m., and soon died. In the annual report for 1926, the Chemical Analyser of Sind reports the case of a woman of Shadapur who committed suicide by taking strychnine. In his annual report for 1927, the Chemical Analyser of Bombay cites a case in which three brothers in Malvan, District Ratnagiri, boiled nux vomica seeds in milk and took that with a view to commit suicide. Two died and one recovered. Fragments of nux vomica seeds were found in the stomachs of both the deceased, and strychnine was detected on analysis of the viscera. The Chemical Examiner of Bengal[3] reports the case of a Hindu male, 22 years old, who committed suicide by taking strychnine. He was picked up from the Eden Gardens and removed to the Medical College Hospitals, Calcutta. He seemed to be conscious, but could not speak and died within fifteen minutes. This case is interesting from the fact that there was no history of spasms or convulsions. The Chemical Examiner of Madras[4] reports two cases of suicide. In one of these a man and his wife drank liquor strychninæ hydrochloridi. In the other case a decoction of nux vomica leaves was taken. A case[5] is also recorded in which a European committed suicide by taking strychnine hydrochloride mixed in a glass containing whisky. Eighteen grains of

1. *U. P. Chemical Examiner's Annual Report,* 1930, *p.* 5.
2. *Littlejohn, Transactions of Medico-Legal Society, Vol. XIX,* 1925, *p.* 12.
3. *Annual Report,* 1929, *p.* 11.
4. *Annual Report,* 1929, *p.* 4.
5. *Bombay Chem. Analyser's Annual Report,* 1931, *p.* 4.

strychnine hydrochloride were isolated from the glass. Strychnine was also detected in the viscera of the deceased.

Homicidal cases by the administration of strychnine are reported to have occurred in England and other western countries. Of these the most famous are those of William Palmer, a medical practitioner, who was convicted at the Central Criminal Court at London in 1856 of having murdered John Parsons Cook at Rugeley in Staffordshire by administering two pills containing strychnine, and of Thomas Neill, or Neill Cream, who was convicted in the same Court on October 21, 1892, of the murder of four women and the attempted murder of a fifth woman by giving strychnine.

Homicidal poisoning by strychnine is rare in India. In 1891, a case occurred in the presidency of Bombay, in which five persons were murdered by strychnine given in milk.[1] A case occurred in Seoni, in which a man suffered from the effects of poisoning as a result of taking betels offered to him at a singing party by two persons with whom he was not on good terms. Strychnine was detected in the washings of the stomach, as well as in the scrapings of the soil in which the man had spat.[2] A case[3] is recorded in which one Singhe administered strychnine in a cup of wine to one Amrat who died in about 3 hours. A case[4] is also reported in which the adopted son of a Hyderabad millionaire was killed by the administration of pills containing strychnine.

Nux vomica seeds are, sometimes, used for destroying cattle.[5]

Accidental cases of poisoning by nux vomica have occurred from an overdose, for it is largely used in medicinal practice by *vaids* and *hakims*. An adult male was nearly killed by taking internally a mixture containing equal parts of the powdered root of strychnos nux vomica, sugar and black pepper for the treatment of gonorrhœa.[6] In his annual report for 1927, the Chemical Examiner of Bengal also reports two cases of accidental poisoning. In one case two female children, aged 3 and 5 years respectively, were given some powder as a quack remedy for worms, and both died from convulsions within half-an-hour. In the other case a woman was given some stuff which was alleged to be opium, and she died. It turned out to be nux vomica as the viscera showed the presence of strychnine and brucine.

The bark of the tree (strychnos nux vomica) has been mistaken for *kurchi* bark (holarrhena antidysenterica) or for angostura bark.

Not only has poisoning occurred from the administration of strychnine by the mouth or hypodermically, but also from its application to the eye[7] or from the inhalation of steam issuing from a hot mixture containing

1. *R. V. Buchu, Bom. High Court,* 1891 ; *Chem. and Druggist,* 1891, *Vol. XXXVIII, p.* 380.
2. *U. P. Chem. Examiner's Annual Rept.,* 1923.
3. *K. E. v. Singhe, All. High Court Appeal No.* 733 *of* 1932.
4. *Times of India, Dec.* 14, 1935.
5. *Bombay Chem. Analyser's Annual Report,* 1918, *p.* 2.
6. *J. V. Swamy, Ind. Med. Gaz., April,* 1889, *p.* 113.
7. *Lancet, Vol. I,* 1879, *p.* 333 ; *Schuler, Gaz. Med. de Paris,* 1861, 3*s., XVI, p.* 98.

strychnine.[1] It has also resulted from the application of nux vomica paste to a wound.[2]

The poisonous effects depend on an individual idiosyncrasy, and tolerance is established by habitually taking the drug for a long time. In India nux vomica is taken as an aphrodisiac. According to Baker those who get into the habit of taking it begin with $\frac{1}{8}$th of a grain morning and evening and gradually increase it to about 20 grains.[3]

When strychnine is taken together with an opiate, its symptoms are delayed, or are modified; in some cases they alternate with those of the opium preparation. A woman, aged 23 years, who swallowed $1\frac{1}{2}$ grains of strychnine and one ounce of laudanum developed the characteristic symptoms of opium poisoning in three hours and those of strychnine poisoning in eight hours. Later on, the symptoms alternated with each other, those of strychnine poisoning lasting longer than those of opium poisoning, until recovery occurred.[4] Occasionally the symptoms of morphine poisoning have supervened, when it has been administered in the treatment of strychnine poisoning.

Strychnine is eliminated unchanged mainly in the urine. Elimination begins even in the first hour of ingestion, continues for two to three days and ceases entirely from three to eight days. It is excreted to some extent in the bile and saliva and possibly in the sweat. An infant may be affected by sucking its mother, who has been taking medicinal doses of strychnine. Three cases have been reported in which infants were poisoned by strychnine absorbed in their mother's milk, one of them dying subsequently.[5] Strychnine is also said to act as a cumulative poison, as it tends to stop its own elimination by contracting the renal arteries.[6]

A small portion of strychnine is taken up by the liver and undergoes oxidation. In cases of fatal poisoning strychnine is found especially in the liver and kidneys, and an unabsorbed portion of it is generally found in the stomach and its contents. According to Bakunin and Majone,[7] the amount of strychnine found in the organs of animals is usually very small and rarely exceeds a tenth of the quantity administered. Traces of strychnine have been detected in the organs in fatal cases of non-strychnine poisoning where strychnine had been administered as a remedial agent two or three days prior to death. It is, therefore, essential for a medical jurist to bear these points in mind before he draws an inference from a very small quantity of strychnine in the organs found by the Chemical Examiner.

Strychnine is not destroyed for a long time in putrefactive changes occurring in a body after death and has often been detected in exhumed bodies. Thus, it was found in an exhumed body after nine months[8] and

1. *Newbocker, Jour. Amer. Med. Assoc.*, 1904, *Vol. XLII, p.* 310.
2. *Chatterji, Ind. Med. Gaz.*, 1872, *p.* 251.
3. *Chevers, Med. Juris., p.* 241.
4. *Macredy, Lancet*, 1882, *Vol. II, p.* 724.
5. *Lancet*, 1869, *Vol. II, p.* 241; *Vol. I, pp.* 733 *and* 872.
6. *Lauder Brunton, Action of Medicine, p.* 651.
7. *Toxicological Experiments with Strychnine, Gaz. chim. ital.* 36 (1905), 227; *Warren, Autenrieth's Detection of Poisons and Powerful Drugs, Ed. VI, p.* 166.
8. *Lancet, Sep.* 23, 1899.

in the stomach, liver and intestines of a body exhumed one year and three days after death.[1] Littlejohn and Drinkwater detected it in viscera two years after their first analysis.[2] Richter found it in a putrid mass of the heart, lungs and liver which had been exposed in open vessels for 11 years.[3] It must, however, be borne in mind that in cases of death from undoubted strychnine poisoning the alkaloid may not be detected in the body. Haines[4] failed to detect the poison in the viscera of two children who died suddenly with all the symptoms of strychnine poisoning. Dr. N. J. Vazifdar, late Chemical Analyser of Bombay, once informed me that he failed to detect it in a case in which there was ample evidence that death occurred from poisoning by strychnine.

PHYSOSTIGMATIS SEMINA (CALABAR BEAN)

This is the ripe seed of Physostigma Venenosum, belonging to N. O. *Leguminosæ*. It is known as the Ordeal Bean of West Africa, as it is used there as a test in suspected witchcraft. It is blackish-brown in colour, and slightly kidney-shaped, having a black groove along its convex border, measures $1\frac{1}{4}'' \times \frac{3}{4}'' \times \frac{1}{2}''$ and weighs about $1\frac{1}{2}$ to 2 drachms. It has no odour, nor has it any distinctive taste. If cut longitudinally, it is seen to consist of a brown rind, containing two hard, white, brittle cotyledons which adhere to the shell.

The poisonous properties are due to two alkaloids, *Physostigmine* or *eserine* and *calabarine*, contained in the cotyledons of the seed.

Physostigmine (Eserine), $C_{15}H_{21}N_3O_2$.—In a pure state this is a white, crystalline substance, but becomes yellowish on exposure to air and light. It is bitter in taste and alkaline in reaction. It is slightly soluble in water, but readily dissolves in alcohol, chloroform and ether. With acids it forms salts, which are soluble in water. Of these physostigminæ salicylas (physostigmine or eserine salicylate) is a pharmacopœial preparation, the dose being 1/100 to 1/50 grain. It enters into the composition of the pharmacopœial preparations of *Lamella physostigminæ* and *Oculentum physostigminæ*.

Symptoms.—These are giddiness, salivation, thirst, pain in the stomach, vomiting, sometimes diarrhœa, slow, feeble and irregular pulse, cold, clammy skin, contracted pupils, muscular twitchings and paralysis of the voluntary muscles. The intellect remains clear to the last. Death occurs from asphyxia due to paralysis of the respiratory centre.

Fatal Dose and Fatal Period.—Desiring to try the effects of the seed on himself, Christison chewed and swallowed the fourth part of a seed equivalent to twelve grains, and in twenty minutes was seized with alarming symptoms. In August, 1864, fifty to sixty children were accidentally poisoned at Liverpool by eating the beans. In two hours forty were taken to the hospital. One of them, aged 6, who had eaten six beans died soon after his admission. The rest recovered.[5] According to Blyth 6 mgms. of physostigmine would be likely to be dangerous and about 205 mgms. or 3 grains would be much beyond the least fatal dose.[6] A patient was given after an operation for hernia 0.1 gramme of eserine sulphate (a non-official preparation, dose being 1/64 to 1/32 grain) to stimulate peristalsis, but he got convulsions and cyanosis and died from failure of respiration and of the heart's action.[7]

Treatmment.—Give emetics or wash out the stomach with charcoal and tannic acid. Atropine and chloral hydrate are both regarded as physiological antidotes. Give stimulants, and artificial respiration may be resorted to, if necessary.

---

1. *Prescott, Org. Analysis*, 1887.
2. *Edin. Med. Jour.*, 1907, *XXII*, *p.* 112.
3. *Ztschr. f. anal. chem.*, 1868, *VII*, *p.* 400 ; *Blyth, Poisons, Ed. V, p.* 344.
4. *Hamilton, System of Legal Medicine*, 1894, *I*, *p.* 459.
5. *Taylor, On Poisons, Ed. III, pp.* 793, 794.
6. *Poisons, Their Effects and Detection, Ed. V, pp.* 422, 423.
7. *Ars Medici, Jan.*, 1932, *p.* 14.

Post-mortem Appearances.—Not characteristic. The mucous membrane of the stomach may be red and congested, and may, sometimes, be covered with a tenacious mucus. The lungs are generally congested and œdematous. The brain is slightly hyperæmic.

Tests.—1. Bromine water produces a red orange-coloured turbid solution which will clear away on heating.

2. Chlorine water gives a red colour.

3. Two or three drops of a very weak solution of physostigmine dropped into a cat's eye will produce contraction of the pupil.

Medico-Legal Points.—Accidental cases of poisoning have occurred among children from eating the beans. Accidental poisoning has also resulted from eserine solution having been instilled into the eyes or sprayed into the nose.

Suicidal cases have occurred, but no homicidal case has yet been recorded.

Physostigmine increases the irritability of the voluntary and involuntary muscles, causing muscular twitchings and peristaltic movements of the intestines. It contracts the pupils by stimulating the ends of the third nerve. It increases the secretions by stimulating the peripheral nerve endings. It augments the irritability of the peripheral terminations of the vagus in the heart and thus causes slowing of the heart beat. It depresses the motor centres in the cord and then in the brain. It causes death by failure of the respiratory centre. It is excreted in the urine, and has been found in the saliva and bile.

Calabarine acts as a stimulant to the cord, and produces convulsions just like strychnine.

Case.—M. S., aged 32, was admitted to the London Hospital suffering from psoriasis. Inadvertently the patient was given $\frac{1}{4}$ grain of eserine sulphate in 15 minims of water by intravenous injection. Within a minute she became unconscious, with laboured breathing, imperceptible pulse, cyanosis, fibrillary twitchings of all muscles, and wide dilatation of the pupils. There was incontinence of urine and fæces, and a great flow of saliva, and the throat and larynx were fiilled with secretion. Immediately the mistake was recognised an injection of atropine 1/100 grain and strychnine 1/60 grain was given, and oxygen was administered by inhalation. An hour later the pulse could just be felt, the throat was clearer, but breathing was still laboured. A second injection of atropine 1/100 grain was given, and the inhalatation of oxygen continued.

Shortly afterwards the patient began to regain consciousness, but it was at least an hour later before she spoke. She complained of deafness and blindness. The breathing was now calm and deep, and the twitchings had almost stopped. The pulse was weak and irregular. The oxygen was stopped; saline (2 pints) was injected by the rectum and retained. Gradually the sight and hearing returned, and the general condition steadily improved. The following day the patient felt rather weak, but was none the worse.—*Slater, Brit. Med. Jour., Dec.* 9, 1922, *p.* 1120.

GELSEMIUM SEMPERVIRENS OR NITIDUM (YELLOW OR CAROLINA JESSAMINE OR JASMINE)

This is a plant belonging to N. O. *Loganiaceæ*, and grows in North America. Its root is official and yields active principles; *viz.*, gelsemine, gelseminine and gelsemic acid.

Gelsemine.—This is a white, very bitter, inodorous, amorphous alkaloidal substance, sparingly soluble in water, but freely in alcohol and ether. With acids it forms crystalline salts.

Gelseminine.—This is a highly poisonous alkaloid occurring as a yellowish-brown, amorphous powder, or in yellowish-white minute crystals. It is slightly soluble in water, but freely soluble in alcohol and ether. Its salts are freely soluble in water. Gelsemine hydrochloride is a non-official preparation, the dose being 1/60 to 1/20 grain.

Gelsemic Acid.—This is a colourless, tasteless, odourless, crystalline substance. It is slightly soluble in cold water, more in hot water and freely in ether and chloroform. It forms salts, but with few metals.

Symptoms.—Nausea, frontal headache, giddiness, ptosis, strabismus, diplopia, dilatation of the pupils, great muscular weakness, in-coordination, paralysis, difficulty of articulation and swallowing due to paralysis of the mouth and throat, depression of the temperature, pulse and respirations, and general prostration. Death occurs from respiratory failure, the mind remaining clear. Sometimes, clonic convulsions may be seen.

Fatal Dose.—Thirty-five drops of the tincture of gelsemium (dose 5 to 15 minims) proved fatal to a woman, 25 to 30 years old, within two hours,[1] although recovery has followed a dose of two drachms of the tincture.[2] Three tea-spoonfuls of the fluid extract of gelsemium (equal to 1|6 grain of gelsemine) caused the death of a pregnant woman in seven hours and-a-half.[3] Twelve minims of the fluid extract proved fatal to a child, 3 years old,[4] and about thirty drops of the same preparation killed a boy, 18 months old.[5]

Fatal Period.—The average fatal period is about three hours. The shortest is one hour,[6] and the longest is seven hours and-a-half.

Treatment.—Emetics or thorough washing out of the stomach. Hypodermic injections of digitalis and atropine. Digitalis will strengthen the heart, and atropine the respiration. Hot applications to the epigastrium and extremities. Oxygen inhalation and artificial respiration, if necessary.

Post-mortem Appearances.—No characteristic appearances. There may be congestion of some of the organs.

Analysis.—1. *Chemical Test.*—The alkaloids of gelsemium, when touched with concentrated alcohol on a white plate, yield a yellow-brown colour. A fragment of potassium chromate changes the colour to red, purple, and lastly to green.

2. *Physiological Test.*—Administered to frogs, cats or rabbits, the alkaloids cause prostration, convulsions, dilated pupils and asphyxia.

Medico-Legal Points.—Poisoning by gelsemium is generally accidental. During the investigation of the Clark-Fulham murder case in Agra in 1912, it was suspected that Clark had administered gelsemine to Fulham with criminal intent.

Gelsemine paralyses the spinal cord and respiratory centre, but has no action on the heart and brain. Sometimes, it causes tetanic spasms.

Gelsemine is eliminated in the urine.

---

1. *"The Legality of drug provings recognised" (Report of the trial of Dr. E. A. Lodge, etc., Detroit,* 1862) ; *Lancet,* 1878, *Vol. I, p.* 892 ; *Witthaus, Med. Juris. and Toxic., Vol. IV, p.* 938.
2. *Wood, Brit. Med. Jour., Feb.* 7, 1885, *p.* 279.
3. *Wormley, Am. Jour. of Phar., Jan.,* 1870, *p.* 14.
4. *Peterson, Haines and Webster, Leg. Med. and Toxic., Vol. II, Ed. II, p.* 508.
5. *Harris, Chicago Med. Jour.,* 1868, *XXV, p.* 760.
6. *Freeman, Hatfield, quoted by Witthaus, Med. Juris. and Toxic., Vol. IV, p.* 940.

CHAPTER XXXIII

CARDIAC POISONS

NICOTIANA TABACUM (TOBACCO, *TAMBAKU*)

This belongs to N. O. *Solanceœ*, and is originally a native plant of America, but is now cultivated largely all over India.

The dried leaves of tobacco are used in India as articles of luxury by almost all classes of people, who use them either in the form of smoke or snuff, or chew them with lime alone or with lime and *pan*. The leaves are manufactured as cigars (cheroots) in Trichinopoly and Burma.

The leaves yield two active principles ; *nicotine* and *nicotianin*.

Nicotine, $C_{10}H_{14}N_2$.—This exists in all parts of the tobacco plant, but notably in the leaves, which contain from 0.6 to 8 per cent in combination with malic and citric acids. It is a colourless, volatile, oily liquid, turning brown and resinous on exposure to the air. It has a burning acrid taste, and a penetrating disagreeable odour. It is soluble in water, alcohol and ether, the solution being alkaline in reaction.. It first stimulates and then depresses the vagal and vasomotor ganglia. Similarly, it first stimulates and then paralyses the cerebral and spinal centres. In smaller doses it contracts the pupils but, when toxic symptoms develop, it dilates them.

Nicotinine.—This is also known as tobacco camphor, and is a volatile, crystalline substance, unimportant from a medico-legal point of view.

Duboisia Hopwoodii, belonging to N. O. *Solanaceœ*, and growing in Australia, contains piturine, a volatile liquid alkaloid, acting exactly like nicotine.

Symptoms.—These are burning, acrid sensations in the mouth and throat, which spread down the œsophagus to the stomach, and are followed by salivation, nausea, vomiting, sometimes diarrhœa, giddiness, faintness, numbness, muscular weakness, tremors, cold, clammy skin, and partial or complete unconsciousness. The pupils are at first contracted, but later on become dilated. The pulse is generally slow at first, and then becomes very rapid. After very large doses, the pulse may be first accelerated and then slow and feeble. The respirations are at first rapid and laboured, and afterwards slow and sighing. Death occurs from paralysis of the respiratory centre, the heart continuing to beat for some time afterwards. Sometimes, there may be delirium and convulsions. In some instances death may occur very rapidly, the symptoms being those of sudden paralysis of the central nervous system.

Chronic Poisoning.—This occurs from overindulgence in tobacco for a long time. It may also occur amongst workmen employed in tobacco factories.

Symptoms.—These are cough, loss of appetite, vomiting, diarrhœa, anæmia, faintness, cardiac irritability and weakness, quick, irregular

pulse, tremors and impaired memory. Eye-sight may be affected, leading to amblyopia.

Fatal Dose.—Kobert[1] has deduced from experiments and records that one grain (0.06 gm.) of nicotine is about the smallest fatal dose to an adult. It is probable that two or three drops of nicotine[2] taken in the stomach might prove fatal to an adult not accustomed to the use of tobacco. A man,[3] however, recovered after taking 4 gm. of purest nicotine as he had vomited much of the poison.

Half an ounce to one ounce of crude tobacco taken by the mouth has caused the death of a lunatic sailor in seven hours.[4] An infusion of thirty grains of dry tobacco leaves given as an enema has resulted in death,[5] but recovery has occurred after the use of an enema in which half an ounce of snuff and five leaves were mixed.[6] A young man, 19 years old, suffered from very serious symptoms after smoking two consecutive pipes,[7] and two brothers died after smoking seventeen and eighteen German pipefuls of tobacco.[8] A drachm of the tobacco juice removed from pipes and mixed with an alcoholic drink proved fatal to a drunken student.[9]

Fatal Period.—Nicotine, when swallowed, may cause death almost immediately or within a few minutes. In the Count Bocarme case death occurred in five minutes.[10] Two men died within five minutes after drinking in mistake for whisky about 30 c.c. each of a germicide containing 39.84 per cent of nicotine.[11] An infusion of tobacco used as an enema resulted in death in fifteen minutes.[12] In some cases death has been delayed for four[13] and five days.[14]

Treatment.—Elimination by washing out the stomach with warm water containing finely powdered charcoal, tannin, or a solution of iodine in potassium iodide. These drugs render the alkaloid insoluble. Keep the patient in a recumbent posture, apply warmth to the body and cold affusions to the head. Administer hypodermically diffusible stimulants, such as strychnine, ether, etc. Oxygen inhalation, artificial respiration and galvanism must be resorted to, when necessary.

Post-mortem Appearances.—The odour of tobacco is usually noticed on opening the stomach, which may contain fragments of the leaves. The mucous membrane of the stomach and intestines is congested and inflamed,

1. *Lehrbich der Intoxikationen*, 1906, *II*, p. 1064; *Peterson, Haines and Webster, Leg. Med. and Toxic., Vol. II, Ed. II*, p. 558.
2. *Witthaus, Manual of Toxic., Ed. II*, p. 1004.
3. *Ars. Medici, Jan.*, 1932, p. 14.
4. *Edin. Med. Jour.*, 1855-56, *Vol. I*, p. 643.
5. *Pereira, Mat. Med., Vol. II*, p. 594; *Collis Barry, Leg. Med., Vol. II*, p. 535.
6. *Glaister, Med. Juris. and Toxic., Ed. VI*, p. 681.
7. *Marshall Hall, Edin. Med. and Surg. Jour., Vol. XII*, 1816; *Blyth, Poisons, Ed. V*, p. 282.
8. *Ibid.*
9. *Collis Barry, Leg. Med., Vol. II*, p. 518.
10. *Orfila, Toxic., Vol. II*, p. 498.
11. *Mc Nally, Jour. Lab. and Clin. Med.*, 1920, *V*, p. 213.
12. *Beck, Med. Juris., Vol. II*, p. 298.
13. *Brit. Med. Jour.*, 1877, *Vol. II*, p. 389.
14. *Morgan, Ibid.*, 1875, *Vol. II*, p. 487.

if death has not ensued rapidly. The brain, lungs and liver are usually congested. The blood is dark and fluid.

The following post-mortem appearances were found in the body of a woman, aged 29 years, who died soon after she had taken a mixture of nicotine with soft soap and water[1] :—

There was marked congestion of the gastric mucosa, and the stomach contained a small quantity of fluid with a very offensive smell. There was some pulmonary œdema. The right side of the heart was dilated. The heart weighed 12½ ounces. There was slight congestion of the meninges. The bladder contained a small quantity of urine. The other organs were all normal.

Chemical Tests.—1. Nicotine gives an orange colour with nitric acid, and a violet colour with hydrochloric acid.

2. A solution of iodine in ether mixed with an ethereal solution of nicotine yields an oily resin of a brownish colour, which in a few minutes crystallizes in long needles of a ruby-red colour by transmitted, and dark blue by reflected, light. These are called "Roussin's crystals."

3. Mercuric chloride gives a white precipitate, becoming crystalline yellow on standing for some time.

Medico-Legal Points.—Poisoning by tobacco has occurred accidentally from excessive smoking, from the infusion given as an enema, or from the application of the leaves or their juice to a wound, an abraded surface or even to the unbroken skin. Taylor[2] mentions the case of a smuggler who was poisoned by having secreted tobacco leaves next his skin in order to avoid the custom duty. A similar case is reported where a squadron of Hussars was poisoned by smuggling tobacco leaves next the skin.[3] A small girl[4] suffered from symptoms of tobacco poisoning following a vigorous rubbing of her trunk and limbs with a mixture of writing ink and scrapings from an old tobacco pipe as a remedy for a very diffuse attack of ringworm. A convict admitted to Liverpool prison, who secreted an ounce of cut Cavendish tobacco in his rectum in order to convey it past searchers, suffered from very severe symptoms four hours later.[5] Children have, sometimes, been poisoned accidentally by sucking the juice of a tobacco pipe, or by drinking *hookah* water.

Accidental cases of poisoning have, sometimes, occurred from nicotine, which, diluted with soft soap and water, is used largely as a germicide and insecticide, especially in agricultural districts. In 1926, a labourer[6] of Kent, who had used nicotine as an insecticide and kept it on a shelf in the kitchen with other bottles containing non-poisonous medicine, took some of the nicotine by mistake and died immediately. The following cases are the examples of severe nicotine poisoning as a result of absorption through the skin :—

1. *A. Douglas Cowburn, The Med.-Leg. and Criminol. Rev., April,* 1933, *pp.* 118-119.
2. *Princ. and Practice of Med. Juris., Vol. II, Ed. IX, p.* 833.
3. *Blyth, Poisons, Their Effects and Detection, Ed. V, p.* 282.
4. *Jones and Morris, Brit. Med. Jour., April* 24, 1926, *p.* 739.
5. *Gill, Brit. Med. Jour., Vol. I,* 1901, *p.* 1544.
6. *A. Douglas Cowburn, The Med.-Leg. and Criminolog. Rev., April,* 1933, *p.* 120.

1. In the process of making an insecticide a girl, 22 years old, accidentally spilt about 2 drachms of a 95 per cent solution of nicotine on her overall sleeve. She changed the overall and washed her arm under the hot tap, dried herself, wiped her damp jumper sleeve, and went on with her job. Twenty minutes later she collapsed.[1]

2. A man, aged 35, sat down in a chair on the seat of which some "Nico-Fume Liquid" (a 40% solution of free nicotine) had been spilled. He felt the solution wet through his clothes to the skin over the left buttock, an area of about the size of a palm, and recognised what it was by its characteristic odour. In about 15 minutes he was seized with severe symptoms of poisoning and recovered in 4 days.[2]

Soldiers, sometimes, apply tobacco to the skin with a view to become sick and thus to escape military duty. The usual method of malingering is to soak two strong cheroots in water for some hours, and to place at bed time one in each axilla, which is held in position by a bandage. The following morning poisonous symptoms supervene, so that the malingerer is unable to attend to duty. In order to ensure greater certainty of the effects, the water in which the cigars have been soaked is taken internally. Deacon[3] describes the case of an Italian soldier who thus suffered from tobacco poisoning at the time of expiry of his leave, so that he was reported sick.

Suicidal and homicidal cases of tobacco poisoning have been very rare indeed. Douglas Cowburn[4] reports the case of a woman who took an insecticide consisting of a mixture of nicotine apparently with suicidal intent, and who was subsequently found dead in a field with the empty bottle by her side which had contained the poison. In the celebrated case of Count Bocarme, nicotine was administered to the brother of the Countess by force. Chevers[5] records two cases of infanticide by tobacco, which used to be a common practice in the districts of Agra and Gwalior. Tobacco has also been used to procure abortion.[6]

Nicotine is eliminated partly by the lungs, but chiefly in the urine, the secretion of which it increases. It is also to be detected in the saliva and sweat. In nursing mothers who smoke excessively nicotine may be found in the breast milk. Lessage[7] asserts that wet nurses who chew or smoke tobacco can poison the babies they nurse, and the symptoms produced are digestive disturbances, restlessness, dyspnœa, bradycardia, syncope, collapse and death.

A case[8] is recorded where a breast-fed infant, six weeks old, whose mother smoked twenty cigarettes a day, suffered from restlessness, insomnia, spastic vomiting, diarrhœa, rapid pulse and circulatory disturbances. The infant recovered after the

1. *Lockhart, Brit. Med. Jour., Feb.* 11, 1933, *p.* 246.
2. *Faulkner, Jour. Amer. Med. Assoc., May* 27, 1933, *p.* 1664.
3. *Brit. Med. Jour., July* 10, 1926, *p.* 61.
4. *The Med.-Leg. and Criminolog. Rev., April*, 1933, *pp.* 120-21.
5. *Med. Juris., p.* 240.
6. *Landerer, Schweiz. Ztschr. f. Pharm.*, 1868, *No. II*, 72; *Witthaus, Med. Juris. and Toxic., Vol. IV, p.* 1002.
7. *Jour. Amer. Med. Assoc., June* 5, 1926, *p.* 1787.
8. *Wyckerheld Bisdan, Maandschrift voor Kindergeneskunde, Leyden, May*, 1937, *p.* 332: *Jour. Amer. Med. Assoc., July* 10, 1937, *p.* 178.

mother's milk was discontinued. Nicotine was detected in the mother's milk. Such a result may, however, be a great rarity.

Putrefaction has no effect on nicotine, which can be detected in the body some years after death.

A non-poisonous alkaloid resembling nicotine has been isolated from the human body, and a ptomaine similar to nicotine has been found but is not so poisonous.

LOBELIA INFLATA (LOBELIA, INDIAN TOBACCO)

The herb belongs to N. O. *Lobeliaceœ* and grows in North America.

Lobelia nicotianœ folia (*Dhawal*) belonging to the same natural order grows in Southern and Western India and the mountain ranges of Ceylon. Its leaves are serrated and hairy, and are very much like tobacco leaves.

Both these plants contain two alkaloids, *lobeline* and *lobelanidine,* and *lobelic acid.*

Lobeline.—This is a volatile, oily, yellow, liquid alkaloid, possessing a pungent taste and an odour like that of tobacco. It is slightly soluble in water, but freely in ether. It resembles nicotine very closely in action.

The official preparations are *Lobelia* (the dried flowering herb), dose, 1 to 3 grains and *Tinctura lobeliœ aetherea* (strength 1 in 5), dose 5 to 15 minims.

Symptoms.—Burning pain in the stomach, vomiting, distressing nausea, headache, giddiness, small, feeble and rapid pulse, pupils contracted and insensible to light, muscular twitchings, unconsciousness, collapse and death ending the scene. Diarrhœa and dysuria are, sometimes, present.

Fatal Dose.—Uncertain. A drachm of the powdered leaves has caused death.[1]

Fatal Period.—Uncertain. The shortest fatal period is half-an-hour.[2] The longest is thirty-six hours.[3]

Treatment.—Produce vomiting, if it has not already set up. Wash out the stomach. Recumbent posture. External warmth and hypodermic stimulants, such as strychnine.

Post-mortem Appearances.—Softening and inflammation of the mucous membrane of the stomach and intestines. Congestion of the vessels of the brain.

Tests.—1. Strong sulphuric acid gives a red colour.

2. Sulphomolybdic acid produces a deep violet colour which, after many hours, passes into a brown and then yellow colour.

Medico-Legal Points.—Lobelia is largely used in England and the United States, and has consequently given rise to fatal accidental poisoning. It has also proved fatal when administered as an abortifacient.

DIGITALIS PURPUREA (DIGITALIS OR FOXGLOVE)

This is a poisonous plant, belonging to N. O. *Scrophulariaceœ,* and growing wild in the hedges in the South of England. It is now cultivated in India and many other parts of the world.

Its root, leaves and seeds contain as active principles the glucosides, digitoxin, digitalin, digitalein, digitonin and some other bodies of an

1. *Pharm. Times, May* 1, 1847, *p.* 182.
2. *Wharton and Stille, Med. Juris., p.* 522.
3. *Pharm. Times, Loc. Cit.*

alkaloidal nature. All these glucosides are met with in commerce, and are used in medicine.

Digitoxin.—This is the most active, poisonous and cumulative of all the four glucosides. It is contained in digitalis leaves in the proportion of 0.22 to 0.4 per cent. It occurs commercially in a crystalline form. It is insoluble in water, slightly soluble in ether, but readily in alcohol and chloroform. It is a non-official preparation, the dose being 1/250 to 1/60 grain. It is also sold as an aqueous solution mixed with glycerine, the dose being 15 minims by the mouth. Digalen is a solution of amorphous digitoxin, and is freely miscible with water and suitable for administration by the mouth or rectum, as well as by hypodermic or intravenous injection.[1]

Digitalin.—This is a crystalline glucoside, and is also known as *digitalin verum.* It is almost insoluble in water, slightly soluble in ether, and readily in chloroform and in a mixture of chloroform and alcohol. It is the chief constituent of Homolle's digitaline, the dose being 1/60 to 1/30 grain in granules. Nativelle's digitalin consists mostly of digitoxin, and occurs in fine white needles. It is insoluble in water, but soluble in alcohol (90%) and in chloroform. The dose is 1/240 to 1/100 grain.

Digitalein (Gitalin).—This is an amorphous, bitter glucoside, soluble in water and alcohol, but soluble with difficulty in ether, chloroform and benzene. It is given hypodermically in 1/100-grain doses. It is said to be non-cumulative.

Digitonin.—This glucoside is both amorphous and crystalline. The amorphous form is soluble in water, but not the crystalline. It is a cardiac depressant, and hence antagonistic to the first three active principles, which have a stimulating action on the heart.

Pharmacopoeial Preparations.—The pharmacopœial preparations of digitalis are as follows:—

1. *Digitalis Pulverata* (Powdered digitalis).—Dose, ½ to 1½ grains (repeated) and 3 to 10 grains for a single dose.

2. *Infusum Digitalis Recens.*—It must be freshly prepared and used within 12 hours, as it loses its activity, if kept long. Dose, 90 to 300 minims (repeated) and 1 to 4 fluid ounces for a single administration.

3. *Tinctura Digitalis.*—Dose, 5 to 15 minims (repeated) and 30 to 90 minims for a single dose.

Symptoms.—The toxic symptoms produced by digitalis are gastro-intestinal at first, and are then referable to its action on the heart. These are thirst, nausea, vomiting followed by severe abdominal pain and perhaps watery diarrhœa, vertigo, severe headache, fainting and oppression in the præcordial region. The pulse is at first accelerated and then slowed, intermittent, irregular and small, the beat falling even to 25 per minute. The respirations are slow and sighing. The pupils are dilated, and visual derangements, such as dimness of vision and changes in the perception of colour, are present. These are followed by drowsiness and coma.

1. *Brit. Med. Jour., May* 22, 1909, *p.* 1243.

There may be delirium or hallucinations and convulsions. The urine may, in some cases, be suppressed. Death usually occurs from sudden syncope. It may take place on slight exertion during apparent convalescence.

Fatal Dose.—Uncertain. Thirty-eight grains of the powdered leaves and nine drachms of the tincture have proved fatal, though recovery has ensued from much larger doses.[1] One-fourth to half-a-grain of digitalin and one-sixteenth grain of digitoxin might cause the death of an adult.

Fatal Period.—Death rarely occurs in less than twenty-two hours, but in one case it occurred in forty-five minutes.[2] In another case a woman, aged 31, died in twelve hours after she had swallowed about 6 to 8 ounces of the tincture of digitalis with a view to commit suicide.[3] Death has been delayed for five,[4] twelve[5] and sixteen[6] days.

Treatment.—This consists in the use of the stomach tube, or emetics, followed by aperients, and the free use of vegetable infusions containing tannin. Tea or coffee may also be given with advantage. Keep the patient in a recumbent posture, administer stimulants, and apply sinapism to the chest. Give cautiously aconite, a physiological antidote, because it exhausts the nerve centres.

Post-mortem Appearances.—Not characteristic. There may be fragments of digitalis leaves in the stomach, which may be found congested and inflamed.

Chemical Tests.—Strong sulphuric acid produces a green colour, changing to purple red on the addition of bromine water. The physiological test should be tried on a frog or a dog.

Medico-Legal Points.—Poisoning by digitalis is not a frequent occurrence. A few accidental cases have occurred from an overdose of one of the medicinal preparations or from eating the leaves by mistake.

Digitalis is rarely used for suicidal purposes, and has been used only once as a homicidal poison, when a homœopathic physician, La Pomerais, killed a widow in Paris in 1864, by giving her digitalin. He had pecuniary interest in her death.

Digitalis is not excreted by the kidneys as fast as it is absorbed into the system, hence it is regarded as a cumulative poison. Persons, who have been taking it for a long time, may suddenly develop the symptoms of poisoning without any subsequent increase in the dose. In such cases the quantity of the urine should be measured to find out if it is diminished, or digitalis should be prescribed with a diuretic, or should be omitted for one week in every four weeks.

1. *Dixonmann, Forens. Med. and Toxic., Ed. VI, p.* 519.
2. *Bull. Soc. d' anat., Paris,* 1849, *XXIV, p.* 89.
3. *Mc Gurie, Johnson and Richards, Amer. Heart Jour., July,* 1936, *pp.* 109-112; *Jour. Ind. Med. Assoc., Jan.,* 1937, *p.* 203.
4. *Edward, Lancet,* 1849, *Vol. II, p.* 31.
5. *Causse, Ann. d' hyg.,* 1859, 2 *s., XI, p.* 464.
6. *Duroziez, Union. Med.,* 1879, 3 *s., XXVII, p.* 991; *Witthaus, Manual of Toxicology, Ed. II, p.* 1083.

QUININE (QUININA), $C_{20}H_{24}N_2O_2$

This is an alkaloid which exists in combination with cinchonine and other alkaloids as salts of quinic or chinic and quinotannic or cinchotannic acids in the barks of various species of cinchona plants, N. O. *Rubinaceæ*. These plants are native to Western South America, but are now cultivated in Java and India.

Quinine occurs in white, acicular, inodorous crystals, having a bitter taste. It is insoluble in water, but dissolves in alcohol, ether, chloroform, benzene and carbon disulphide. It reacts like an alkali, and forms neutral and acid salts with acids. Thus, quinine sulphate, quinine bisulphate, quinine hydrochloride and quinine dihydrochloride are official preparations, the dose of each being 1 to 10 grains. Dissolved in water, these salts have a blue fluorescence when sulphuric acid is present. Of these salts quinine sulphate enters into the preparations of *Syrupus ferri phosphatis cum quinina et strychnina* (dose, 30 to 60 minims) and *Liquor quininæ ammoniata* (dose, 30 to 60 minims).

Quinine is a protoplasmic poison and reduces the metabolism of the body. In sufficiently large doses it paralyses and destroys all forms of living matter.

Symptoms.—These are giddiness, headache, ringing in the ears and deafness, disorders of vision, difficulty of speech, pain in the abdomen, vomiting, diarrhœa, mental depression, confusion of thought, muscular weakness, itching, erythematous or urticarial rash on the skin, hæmoglobinuria, cold, clammy skin, gasping respiration, slow and imperceptible pulse, collapse, cyanosis and death from respiratory failure. Delirium and convulsions have, sometimes, been observed.

Fatal Dose and Fatal Period.—Uncertain. Thirty grains of quinine sulphate swallowed in tablet form have caused deafness and loss of perception of light lasting for months.[1] Sixty grains of quinine sulphate taken in hot whisky and water have caused alarming symptoms.[2] Of five soldiers who drank a solution of 16 grammes of quinine hydrochloride in mistake for sodium sulphate, three died within 10 to 20 minutes and the other two younger ones vomited and awoke from their coma after two days.[3] A child, aged 5 years, died in 2 hours and 15 minutes after swallowing 8 sugar-coated quinine pills of 5 grains each.[4] Willimot[5] describes the case of two sisters, aged 5 and 2½ years, who consumed, in mistake for sweets, 5 and 26 sugar-coated five-grain quinine tablets respectively. Within an hour of taking the tablets both girls suffered from severe pains in the stomach, complained of nausea and refused all food. The elder child, after administration of an emetic, made a permanent recovery in about 4 hours. The younger child, however, began vomiting with purging and later had convulsions. She died in about 3½ hours after she had swallowed the tablets. One hundred and twenty grains of quinine hydrochloride have caused death, and 225 grains of quinine sulphate have proved fatal to a woman, 22 years old, in about seventeen hours.[6] A woman,[7] aged 50, died after she had taken overnight forty-eight 5-grain tablets of quinine bisulphate "in order to ensure sleep." On the other hand, much larger doses have been recovered from. In one case one ounce produced only some confusion and noises in the ears.[8]

Treatment.—Administer emetics or wash out the stomach, and give hypodermic injections of strychnine, digitalin and camphor. Give hot infusions of coffee and apply warmth to the body. Resort to artificial respiration, if necessary.

Post-mortem Appearances.—There may be congestion of the organs. There is usually hæmolysis of the red blood corpuscles. In the abovementioned case where the three soldiers died after swallowing a solution of quinine hydrochloride, the post-

1. *Plummer, Brit. Med. Jour., Dec.* 5, 1925, *p.* 1062.
2. *Gooch, Brit. Med. Jour., July* 17, 1926, *p.* 115.
3. *L. Lavier, Presse Medicale, No.* 35, 1931 ; *Ars. Medici, Sep.*, 1931, *p.* 395.
4. *Beng. Chem. Examiner, Ann. Rep.*, 1931, *p.* 6.
5. *Lancet, Nov.* 21, 1931, *p.* 1133.
6. *Autenrieth, Detection of Poisons, Ed. VI (Engl. Trans. by Warren), pp.* 212, 213.
7. *Raven, Brit. Med. Jour., July* 9, 1927, *p.* 59.
8. *Cushny, Pharmacology and Therapeutics, Ed. XI, p.* 690.

mortem examination of the bodies showed that the gastric mucous membrane was macerated, both kidneys were hyperæmic and the bases of the lungs were congested.

Chemical Analysis.—Quinine may be extracted from aqueous alkaline solutions by ether, benzene or chloroform. Upon evaporation, a resinous, amorphous residue is left in which quinine may be recognised by the following test :—

When the residue is dissolved in a little dilute acetic acid and 5 to 10 drops of saturated chlorine water are added, a green colour due to thalleioquin is obtained on the addition of ammonia is excess if quinine is present.

Medico-Legal Points.—Cases of poisoning by quinine are mostly accidental. Owing to idiosyncrasy, even the medicinal doses have, sometimes, produced poisonous symptoms. Thus, Bannerji[1] reports the case of his younger brother who used to complain of toxic symptoms even after the administration of ¼ grain of quinine. Krishnamurty[2] records the case of a male, 30 years old, whose face became swollen and flushed and who complained of inordinate itching all over the body within ten minutes after he had taken 5 grains of quinine. Cruikshank[3] also quotes the case of a woman, 63 years old, who nearly collapsed after an injection of 1 c.c. solution containing 0.3 gramme quinine and 0.065 gramme urethane. He suggests the following test to be performed where there is reason to suspect idiosyncrasy to quinine :—

If a drop of a 1 per cent solution of quinine hydrochloride is placed on the forearm and the skin under it is scarified, a definite wheal surrounded by erythema appears ten minutes later in individuals susceptible to quinine. A control of sterile water shows only slight redness.

Quinine was taken with suicidal intent in the case where 225 grains proved fatal. Goldmann describes the case of a man, aged 20, who recovered after he had taken 19 grammes of quinine sulphate in solution with a view to commit suicide.[4]

Quinine excites the pregnant uterus and occasionally causes abortion ; hence it is often used as an abortifacient, and has, sometimes, produced poisonous symptoms.

Quinine is eliminated for the most part unchanged in the urine. It may be detected in the urine within fifteen to thirty minutes after its administration by the mouth, and excretion may continue for forty-eight to seventy-two hours. Traces may also be found in the saliva, sweat and milk.[1]

NERIUM ODORUM (WHITE OR SWEET-SCENTED OLEANDER, *KARAN* OR *KANER*)

This plant belongs to N. O. *Apocynaceæ* and is grown in gardens in India for its beautiful white or pink flowers, which are given as offerings to gods by Hindu worshippers.

All parts of the plant are poisonous. Rai Chooni Lal Bose Bahadur[5] isolated from the plant three active principles, neriodorein, neriodorin and karabin (named after *karabi*, the Bengali term for this plant). Of these the first two had already been isolated by Greenish. Neriodorin and karabin are glucosides and have a paralysing action on the heart like digitalin and a stimulating action on the spinal cord like strychnine. Neriodorein is a saponin only, and has much milder toxic properties.

1. *Ind. Med. Gaz., Sep.*, 1928, *p.* 533.
2. *Ind. Med. Gaz., March*, 1927, *p.* 142.
3. *Brit. Med. Jour., Jan.* 19, 1929, *p.* 104.
4. *Erich Leschke, Clin. Toxic., Eng. Trans. by Stewart and Dorrer*, 1934, *p.* 191.
5. *Ind. Med. Gaz., Nov.*, 1901, *p.* 408.

From investigations[1] carried out in the Chemical Examiner's laboratory at Madras it has been ascertained that Nerium odorum yields only

Fig. 155.—Nerium Odorum.

one active principle, which is a pure, white, crystalline glucoside, named, nerin, and that the three active principles isolated by Rai Chooni Lal Bose Bahadur are the same poisonous principle in different degrees of purity.

1. *Madras Chem. Examiner's Annual Rep.*, 1937, *p.* 8.

Nerin is sparingly soluble in water and very sparingly soluble in ether, petroleum ether and benzene. It readily dissolves in alcohol, acetone and chloroform. It melts at 123° C. One-fiftieth of a milligramme is about the average minimum fatal dose to a frog, weighing about 10 grammes, producing paralysis and death. Eight milligrammes, injected into the abdomen of a dog, weighing about 4.1 kilogrammes, killed it in one hour. About one-eighth of a milligramme of strychnine is about the average minimum fatal dose to a frog, weighing about 10 grammes; hence nerin appears to be about six times as poisonous as strychnine to a frog.

Symptoms.—Difficulty of swallowing and articulation, abdominal pain, vomiting, profuse frothy salivation and diarrhœa. The pulse is first slowed and later becomes rapid and weak. The respirations are hurried from the beginning. These are followed by dilated pupils, muscular twitchings, tetanic spasms, drowsiness, unconsciousness, coma and death. Lock-jaw is frequently present, while diarrhœa is occasionally absent.

Fatal Dose.—Uncertain. More than a quarter of a tola (1 tola = 180 grains) of the fresh root bark have produced poisonous symptoms.[1] Two hundred and fifty grains (about half an ounce) of the root may be considered to be an average fatal dose for an adult.[2] Four ounces of the decoction of the root caused the death of a man, 35 years old, in five days.[3]

Fatal Period.—Uncertain. A man, 50 years old, who swallowed some of the root with mustard oil with intent to commit suicide died in about twenty-four hours.[4] A Hindu female, aged 20 years, who took the oleander root as an abortifacient, died in two to three hours.[5]

Treatment.—Evacuation by washing out the stomach. Give stimulants, such as ether, and treat the symptoms.

Post-mortem Appearances.—Not characteristic. There may be congested patches in the stomach and upper portion of the small intestine.

Tests.—The Chemical Examiner of Madras mentions in his annual report for the year 1937 that a minute fragment of the acid alcoholic extract of the leaves of Nerium odorum, dissolved in concentrated sulphuric acid, produces an immediate purple colour which assumes a deeper tint on standing. It also gives Keller's test, a bright green colour appearing in the acetic acid layer and a purple colour in the sulphuric acid layer. Both the colours are stable for several days. Keller's test is performed by dissolving the extract in 1 c.c. of glacial acetic acid containing 5 per cent of ferric sulphate and floating this solution on the surface of a mixture of concentrated sulphuric acid (100 parts) and 5 per cent ferric sulphate (1 part).

This extract does not reduce Fehling's solution but, after hydrolysis either with hydrochloric acid or with emulsin, it reduces Fehling's solution.

1. *Ind. Med. Gaz., Sep.*, 1866, *p.* 258 *a.*
2. *C. L. Bose, Indian Med. Gaz., Nov.*, 1901, *p.* 412.
3. *Murray, Ind. Med. Gaz.*, 1877, *p.* 319.
4. *Dr. Greig of Sitapur quoted in Chevers' Med. Juris., p.* 253.
5. *Bengal Chemical Examiner's Annual Report*, 1929, *p.* 13.

Medico-Legal Points.—The root is used internally by ignorant people as a remedy for venereal diseases. Hence accidental poisonings, sometimes with fatal consequences, have occurred from the administration of the root or its decoction. Two Mahomedans drank each a cupful of a strong decoction of the root as an anodyne for pain in the loins. One of them died in about 26 hours, and the other recovered after prolonged illness.[1]

In the form of a paste the root is used in the treatment of cancers and ulcerations. The decoction of the leaves is applied externally to reduce swellings.

Criminally the root is used as an abortifacient both as a local application and as an internal administration. The root and the leaves are often used as a paste or decoction for suicidal purposes, especially in western and southern India. A case is recorded in which a man first took oleander and then to hasten his death he hanged himself on the same tree of oleander.[2] A man of Parner in Ahmednagar District committed suicide by taking the juice of *Kaner* root (Nerium odorum).[3]

Nerium odorum is not often used for homicidal purposes, but it has caused death when administered as a love potion. As a cattle poison the juice of the root is, sometimes, smeared on a rag, which is then inserted into the anus of the animal intended to be killed.

Oleander was detected in the viscera of the female bodies which were completely burnt externally at the cremating place in Midnapur and Bhubneshwar (Puri).[4]

CERBERA THEVETIA OR THEVETIA NERIIFOLIA (EXILE OR YELLOW OLEANDER, *PILA KANER*)

This is a plant belonging to N. O. *Apocynaceæ*, and is widely cultivated as an ornamental shrub in gardens in the plains of India. The plant is highly poisonous and contains an active principle, *thevetin*, which is a glucoside. It resides in the milky juice which exudes from all parts of the plant. It is soluble in 124 parts of water at 14°, and is freely soluble in spirit and chloroform but not in ether. Its action is very much like digitalin, but has a convulsant effect also.

In 1919, De and Chowdhury[5] were able to isolate *thevetin* in a pure form with a melting point of 189° to 190° C. and gave it a formula, $C_{72}H_{124}O_{36}$. They also found that this glucoside on hydrolysis broke up into an amorphous substance, called *thevetidin*, which was stated to be more toxic than *thevetin*. In 1932, Ghatak,[6] working at the Chemical Laboratory of the Allahabad University under Dr. Dutt, isolated two glucosides from the kernel of the seeds. One of these was named as *thevetin* after de Vrij who was the first to isolate it. On crystallization from dilute alcohol, it was obtained as snow-white slender needles melting

1. *Kali Mohan Sen, Ind. Med. Gaz., April,* 1899, *p.* 118.
2. *Bengal Chemical Examiner's Annual Report,* 1923.
3. *Bombay Chemical Analyser's Annual Report,* 1927, *p.* 5.
4. *Bengal Chemical Examiner's Annual Report,* 1923.
5. *Calcutta University Thesis.*
6. *Bull. Acad. Sci., U. P., II, No.* 2.

at 192° C. and having a formula, $C_{20}H_{30}O_6$. This substance is insoluble in water but easily dissolves in most of the organic solvents. When perfectly pure, it is quite tasteless, but in the presence of only traces of impurities it has a pronounced bitter taste. The second glucoside, on twice recrystallization from hot water, was obtained as slender, shining silky needles melting at 178° C. and having a formula, $C_{16}H_{24}O_6$. It differs from *thevetin* by its solubility in water and intense bitter taste. It dissolves in strong sulphuric acid and forms an orange colour, which intensifies in about five minutes and becomes deep red. It is insoluble in most of the organic solvents, except alcohol in which it is very soluble. It was named as *thevetoxin* on the supposition that it was highly poisonous, but Bhatia and Lal[1] have demonstrated from experiments carried out at the Pharmacological Laboratory of the Lucknow University that it is less toxic than *thevetin,* although it is toxic enough to prevent its safe use as a therapeutic agent. It closely resembles in action the drugs of the digitalis group, but has no local irritant action. It has a stimulant action on the plain muscles of the intestines, blood vessels, bronchi, uterus and heart.

Fig. 156.—Cerbera Thevetia.

Symptoms.—Burning pain in the mouth and dryness of the throat, tingling and numbness of the tongue, vomiting and often diarrhœa, headache, dizziness, dilated pupils, and fainting. The pulse is soft and slow,

1. *Ind. Jour. of Med. Research, XXI,* 3, *Jan.* 1934, *p.* 608.

later becomes rapid, weak and irregular. Collapse sets in, and death occurs from heart failure. Tetanic convulsions are sometimes observed.

Fatal Dose.—Uncertain. One seed killed a child, about 4 years old.[1] Eight to ten seeds would prove fatal to an adult.

Fatal Period.—Uncertain. The child who ate one seed died in 2 hours. A young man died in 2 to 3 hours after he took his meal mixed with the powdered root.[2]

Treatment.—Same as in white oleander poisoning. Ether hypodermically.

Post-Mortem Appearances.—Not characteristic. In the case of a Hindu male who died shortly after taking some yellow oleander the mucous coat of the stomach was thrown into exaggerated folds, the general surface of which was congested, and of a deep red colour; scattered about the folds were some inflammatory spots of a lighter colour than the general surface, somewhat glistening and stellate in appearance. Several irregular fragments like millet seeds were found scattered in the mucous folds of the stomach, which on analysis were found to be those of yellow oleander. The duodenum to the extent of four inches was brick red in colour, and had an irregular dark purple patch in the centre. The liver was congested.[3]

Tests.—1. If the pericarp or the kernel is boiled with dilute hydrochloric acid, a blue colour is formed.

2. If an alcoholic extract of the seeds is warmed with dilute hydrochloric acid, a deep bluish-green colour is produced. The colour disappears on adding a solution of potassium permanganate.

3. If the extract is treated with strong sulphuric acid, a cherry red colour is developed.

4. The alkaline ether extract (dry) from Stas' process, when rubbed on the tip of the tongue, produces a pricking sensation and rawness lasting for about ½ to 1 hour.

Medico-Legal Points.—The root and the seeds are often used for procuring criminal abortion, and occasionally for suicidal and homicidal purposes. In his annual report for the year 1927, the Chemical Analyser of Bombay reports a case in which the powdered seeds were given to a woman to be administered to her husband as a love philter, as a result of which he would become a mere puppet in her hands. The seeds are also commonly used for poisoning cattle, especially in the Presidencies of Bombay and Madras.

Jadub Kristo Sen[4] reports four cases of poisoning by the seeds of this plant. In one case a young Hindu widow rubbed two seeds with treacle on a mortar and swallowed them to procure abortion. She suffered

1. *Chevers, Med. Juris., p.* 260.
2. *S. Bannerjea, Ind. Med. Gaz., Jan.,* 1923, *p.* 22.
3. *Daley, Ind. Med. Gaz., August,* 1903, *p.* 296.
4. *Ind. Med. Gaz., Nov.,* 1901, *pp.* 412-13.

from poisonous symptoms, gave birth to a healthy male child on the fourth day and ultimately recovered. In two other cases a Mahomedan female and a Hindu widow attempted to commit suicide by taking two grains of the seeds. The fourth was a case of homicidal poisoning, in which a man developed toxic symptoms after taking a meal of stale rice left in an open pot by his wife with whom he was not on good terms. S. Bannerji[1] also reports a case, in which a woman administered the powdered root to her husband in a meal containing *pakhal* (stale rice) and bean curry.

Bhupendra Mohan Roy[2] reports the case of a Hindu male, aged 22, who was suffering from toxic heart block due to Cerbera thevetia. When he saw him on the 17th April, 1927, the patient was in a stuporose condition, and could not raise or hold up his head. The arms and legs were flaccid, and saliva and ropy mucus were flowing from the angles of the mouth. At intervals he tossed his head from side to side and threw up his arms and legs. He could not answer questions. The pulse was feeble, slow and 36 per minute with marked irregularity. The respiration was slow and hurried. The pupils were normal and reacted to light. The blood pressure was 120|76. After inhaling a few whiffs of ammonia the patient opened his eyes and said that in order to end his miserable existence he had taken 8 seeds of kaner (yellow oleander) squashed up with one pice worth of vermilion at 9 a.m. After three hours he vomited once. After the stomach was washed out, 1|100 grain of atropine was injected hypodermically every four hours, and a dose of magnesium sulphate was given to clear the bowels. Next day the patient was spitting bilious fluid, and felt hungry. Atropine was continued for eight or nine days until dilated pupils, dry skin and great thirst were noticed. After complete recovery on the 25th April, he was handed over to the police to take his trial in the law courts.

A case is recorded where yellow oleander could be detected in the stomach and contents of the two bodies that had been exhumed and had undergone decomposition.[3]

The bark is used as an antipyretic in small doses, 2 grains of the powdered bark being equivalent to an ordinary dose of cinchona. In large doses it acts as an emetic and purgative and produces toxic effects.

ACONITUM NAPELLUS (ACONITE, MONK'S HOOD, WOLFSBANE OR BLUE ROCKET; *MITHA ZAHAR OR DUDHIA BISH*)

This is a plant, 2 to 6 feet high, belonging to N. O. *Ranunculaceæ*, and growing in England and the temperate Himalayan region. All parts of the plant are poisonous, but the root is chiefly used as a poison in this country. The dry root is more or less conical or tapering in shape, presenting scars or bases of broken rootlets, and is usually arched and shrivelled with longitudinal wrinkles. It is usually 2 to 4 inches long, ½ to ¾ inches thick at the upper extremity, dark-brown externally and whitish and starchy internally. It has no odour; when chewed, it imparts a sensation of tingling and numbness to the tongue and lips.

1. *Ind. Med. Gaz., Jan.*, 1923, *p.* 22.
2. *Ibid., August*, 1927, *p.* 450.
3. *Madras Chem. Exam. Annual Rep.*, 1933, *p.* 9.

The root of Aconitum napellus is official in the British Pharmacopœia, which gives no dose, but the United States Pharmacopœia gives 1 grain as the dose. The pharmaceutical preparation derived from the root is *linimentum aconiti* (strength 0.2% ether-soluble alkaloid), while the non-official preparations made from the root are *extractum aconiti radicis alcoholicum*, the maximum single dose being ½ grain, *chloroformum aconiti* (B.P.C.), and *tinctura aconiti* (strength 1 in 10), dose 2 to 5 minims. Fleming's tincture is almost of the same strength as the liniment, and is, sometimes, known as *tinctura aconiti fortior*. The A.B.C. liniment (*Linimentum aconiti compositum*) is a mixture of equal parts of aconite, belladonna and chloroform liniments.

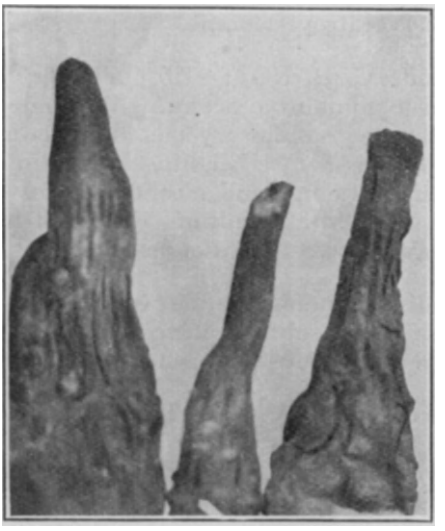


Fig. 157.—Dried Aconite roots.

The root and other parts of the plant contain a number of alkaloids, the chief of which is aconitine.

Aconitine (*Acetyl-Benzoyl-Aconine*), $C_{34}H_{45}NO_{11}$.—This is one of the most deadly known poisons and occurs in Aconitum napellus in combination with aconitic acid. Pure aconitine forms colourless, transparent, rhombic crystals, readily soluble in alcohol and chloroform, less readily in ether and almost insoluble in water, the dose being 1|400 (U. S. P.). With acids it forms crystalline salts, of which the nitrate is used in medicine in 1|600 grain-doses hypodermically. The non-official preparation of the pure alkaloid is *unguentum aconitinæ* (strength 1 in 50). Aconitine splits up on hydrolysis into acetic acid, benzoic acid and aconine.

Aconitine first stimulates and then paralyses the peripheral terminations of the sensory and secretory nerves. It produces the same effect on the motor nerves and the centres of the medulla and cord, but it does not seem to affect the higher centres of the brain, for consciousness usually remains till the end.

Aconitum Ferox (*Bish, Telyabish or Bachhnak*).—This is another poisonous variety very similar to Aconitum napellus. It grows from 2 to 5 feet high in the temperate and sub-Alpine regions of the Himalayas. Its root, when dried and steeped in oil, is a black, plump, heavy tuber, 2 to 4 inches long, and has a disagreeable odour like hydraceum, and a reddish-brown resinous fracture. It is extensively used both externally and internally by *Vaids* and *Hakims* in the treatment of muscular rheumatism, neuralgia and paralysis. It is administered in $\frac{1}{4}$ to $\frac{1}{2}$ grain-doses, after it is soaked in cow's urine for at least three days, whereby it loses much of its poisonous property.

Aconitum ferox yields a number of alkaloids, the chief of which is pseudo-aconitine or veratroyl-aconine.

Pseudo-Aconitine, $C_{36}H_{51}NO_{12}$.—This alkaloid exists in Aconitum Ferox, which is said to contain no aconitine. Pseudo-aconitine crystallises in transparent needles or granular crystals, but it is usually obtained as an amorphous or syrupy mass. It is slightly soluble in water, but it is more soluble than aconitine in ether and alcohol. In its chemical reactions it presents a close analogy with aconitine. Thus on hydrolysis it splits up into acetic acid, veratric acid and pseudo-aconine.

Pharmacologically pseudo-aconitine is identical to aconitine in its action, but in consequence of its molecules being larger more pseudo-aconitine is required to produce the same effects as aconitine.[1]

Aconitum Heterophyllum (*Atis*).—This plant grows in the sub-Alpine and Alpine zone of the Himalayas. Its tuberous root is used in Indian medicine as a tonic and anti-periodic. The root contains a non-poisonous, amorphous alkaloid, *atisin*, and aconitic acid.

Symptoms.—The symptoms supervene immediately, or within a few minutes, after swallowing a poisonous dose of aconite or aconitine or any of its preparations. These are severe burning and tingling of the lips, tongue, mouth and throat, followed by numbness and anæsthesia of these parts. Nausea, salivation, pain in the abdomen and vomiting usually occur, but diarrhœa is rare. Later tingling and formication spread over the whole body, causing great uneasiness to the patient. The pupils contract and dilate alternately, and vision is impaired. The patient complains of vertigo, restlessness, great prostration, and pain and weakness of the muscles with twitchings and spasms. The pulse is slow, feeble and irregular, and the respirations are first rapid, but soon become slow, laboured and shallow. The skin is cold and damp, with sub-normal temperature. Death occurs usually from syncope, or in some cases from asphyxia. In most cases consciousness is retained till near the end, but sometimes delirium or convulsions, insensibility and coma have been observed.

Fatal Dose.—Four grains of the alcoholic extract, sixty grains of the root (Aconitum napellus) and twenty-five minims of Fleming's tincture

1. *Dymmock, Pharmacographica Indica, Vol. I. p.* 6.

have each caused death.[1] Thirty-five grains of the root eaten by mistake for horse radish have also proved fatal to an adult.[2] Two grains of the non-alcoholic extract and one drachm of the tincture have proved fatal.[3] Eighty drops of the tincture taken in ten doses during a period of four days proved fatal to Dr. Male of Birmingham, the last dose being ten drops.[4] Recovery, however, has taken place after much larger doses. In one instance one ounce of Fleming's tincture did not prove fatal,[5] and in two other cases one ounce of the U. S. P. tincture failed to cause death.[6] Twenty minims of the liniment would probably prove fatal, but recovery has followed a dose of two ounces.[7] Chevers[8] mentions a case in which fifteen grains of the root of Aconitum ferox produced poisonous symptoms. One-fiftieth grain of aconitine nearly proved fatal to an elderly lady,[9] and it is probable that one-thirty-second grain of pure aconitine would prove fatal to an adult, although recovery has occurred after a dose of two grains and-a-half.[10] One-fifteenth grain of aconitine nitrate has produced a fatal result.[11]

Fatal Period.—The average fatal period is from one to five hours, but may, sometimes, be delayed. The shortest recorded period is less than seven minutes in the case of a man who swallowed the liniment.[12] A boy, 12 years old, died in thirty minutes after having been given some sweet containing aconite by one of the "antiparty".[13] In non-fatal cases the symptoms of numbness and tingling may persist for a long time after the severe toxic symptoms have subsided. Thus, in the case of a girl who recovered from the effects of five granules of aconitine administered for hysterical coxalgia the tingling persisted in both legs for several days and in another case for fifteen days.[14]

Treatment.—Use emetics or wash out the stomach with a solution of iodine in potassium iodide, or a solution containing animal charcoal or tannic acid. Maintain the recumbent posture, administer amyl nitrite by inhalation or atropine and diffusible stimulants, such as digitalis, strychnine and ether hypodermically. Keep up the body heat by hot water bottles, friction and covering the patient with blankets. Artificial respiration may be resorted to, if necessary.

Hypertonic saline may be administered intravenously to combat collapse. A woman swallowed about one ounce of A. B. C. liniment, and suffered from symptoms of acute poisoning. She soon got into a collapsed

1. *Amer. Med. Monthly, March,* 1854, *p.* 223.
2. *Taylor, On Poisons, Ed. III, p.* 750.
3. *Ibid., pp.* 750, 756.
4. *Prov. Med. and Surg. Jour., Aug.* 20, 1845, *p.* 535.
5. *Bradley, Med. Rec., N. Y.,* 1887, *Vol. XXXII, p.* 155.
6. *Witthaus, Med. Juris. and Toxic., Vol. IV, p.* 855.
7. *Ibid.*
8. *Med. Juris., p.* 141.
9. *Pereira, Mat. Med., Vol. II, Part II, p.* 695.
10. *Med. Gaz., Vol.* 41, *p.* 30.
11. *Dixonmann and Brend, Forensic Med. and Toxic., Ed. VI, p.* 524.
12. *Bennet, Monthly Jour. of Med. Scie.,* 1852, *Vol. XV, p.* 69.
13. *Bengal Chemical Examiner's Annual Report,* 1927, *p.* 13.
14. *Jour. d. Med. d. Bordeaux,* 1893; *Witthaus, Med. Juris. and Toxic., Vol. IV, Ed. II, p.* 857.

condition, but recovered after an intravenous injection of 3 pints of hypertonic saline.[1]

Post-Mortem Appearances.—Not characteristic. Fragments of the root may be found in the stomach contents. The mucous membrane of the stomach and small intestine may be congested and inflamed. There is usually marked general venous congestion with dark fluid blood.

Tests.—1. Tingling and numbness of the tongue and lips for several hours is produced, if a small drop of a solution of aconitine is placed on the tongue or the smallest fragment of aconitine root is chewed between the front teeth.

2. A solution containing a very minute quantity of aconitine injected under the skin of a frog or a mouse will produce the toxic effects and will cause the death of the animal, usually within an hour.

3. Acetic acid and potassium permanganate produce a red crystalline precipitate but this chemical test is not reliable.

Medico-Legal Points.—Accidental poisoning by aconite is not a rare occurrence, seeing that it is largely used in Indian medicine.

On the 5th June, 1923, five persons, *viz.*, three males and two females, ate *chutnie* with their breakfast, and suffered from poisonous symptoms. On admission to the King George's Hospital, Lucknow, in the afternoon the symptoms were tingling and numbness of the tongue, pain in the throat and abdomen, vomiting, weakness of the muscles and marked prostration. They all recovered on the fourth day. It appears that aconite root was powdered by mistake with *amture* in preparing the *chutnie*.

The root has, sometimes, been eaten by mistake for horse radish and produced fatal results although there is no similarity between the two roots. The tincture has been swallowed in overdoses, and the liniment has been taken internally in mistake. The external application of neuraline, a preparation containing Fleming's tincture, has caused death.[2] Inhalation of its dust[3] while powdering the root has produced toxic symptoms. A case of multiple poisoning by aconite illustrating the danger of careless labelling is reported by the Chemical Examiner of Bengal.[4]

A medical man made up a drink supposed to contain citric acid, tinctura aurantii and sugar. Eight persons including himself partook of the drink, and all developed poisonous symptoms in two hours. All recovered. On examination the bottle labelled tinctura aurantii in Bengali was found to contain tincture of aconite. A similar case of multiple poisoning occurred at Shalimar. A *Kholasi* of the Bengal Nagpur Railway goods shed found a bottle containing tincture of aconite on the railway line, and mistaking it for brandy brought it to the cooly lines, where his friends also thought that it contained brandy. Nine men partook of the contents of the bottle, and all of them showed typical symptoms of aconite poisoning and one of them died.[5] In a third case ten people drank some liquor mixed with soda water from a bottle labelled "Beehive Brandy," which was purchased by one of them with several other empty bottles. They all felt an immediate irritation in their throat, and vomited. They were removed to the hospital, where their stomachs were washed out. Four died and the

1. *Talukdar, Ind. Med. Gaz., Nov.* 1935, *p.* 628.
2. *Pharm. Jour., Jan.*, 1872, *p.* 618.
3. *Med. Times and Gaz., Jan.* 14, 1860.
4. *Ind. Med. Gaz., Sep.*, 1910, *p.* 363.
5. *Beng. Chem. Exam. Ann. Rep.*, 1922, *p.* 6.

others recovered. Aconite was detected in the viscera of two and the stomach washings of all the victims. It was also detected in the liquor contained in the bottle labelled "Beehive brandy".[1]

In a case which occurred at Gorakhpur, some supposed catechu served with a betel leaf was responsible for the poisoning of five persons in a marriage party. Three of them spat out the betel on experiencing some unusual sensation in their mouth, but the other two ate up their shares, developed the symptoms of irritant poisoning and died within ten hours. The supposed catechu, on examination, was found to consist of aconite root.[2]

Cases of suicidal and homicidal poisoning by aconite often occur in India, although they are rare in European countries.

A young Hindu woman, aged 20 years, took a piece of the root with intent to destroy herself, but recovered under the prompt treatment at the King George's Hospital, Lucknow.

A woman administered aconite to her son-in-law in cooked rice with curds coloured with turmeric. After taking the food he fell ill, and died soon afterwards. The herb of aconite was found in the house of the woman, who also confessed that she had poisoned her son-in-law. She was convicted and sentenced to death.[3]

On the night of the 11th June, 1923, one Phulmani Mundain[4] administered to her husband aconite powder in the *dhal* as a love potion with the object of influencing his heart. At about 10 o'clock that night the husband complained that he was feeling ill and that there were burnings in his body ; he also started vomiting. He was given some medicine but he could not swallow it, and vomited it out. His condition grew worse, and early in the morning, at about 4 a.m. he died. At the post-mortem examination there were general venous congestion and hæmorrhages in the mucous membrane of the stomach while the heart was full of dark clotted blood. Aconite was detected in the vomited matter, in the brass cup which had contained the *dhal* consumed by the husband, and in the portions of the viscera submitted to the Chemical Examiner. The woman was found guilty and convicted under Section 304-A, Indian Penal Code.

A case[5] of multiple homicide by aconite is recorded. A woman administered the poison with food to her husband, her step-son, aged 14, and her step-daughter, aged 6. Pain in the throat, vomiting and purging were observed and they died within 2 hours after the administration. Aconite was detected in the viscera, vomited matters and in the brownish substances said to have been used.

The celebrated case in which aconitine was used for the purpose of criminal poisoning is that of Dr. Lamson, who was convicted and sentenced to death by the Central Criminal Court for administering the drug to his brother-in-law, Percy Malcolm John, and thereby causing his death. Lamson had a reversionary interest through his wife, in a sum of £1,500, which would come to him on the death of his brother-in-law. The latter was a sickly lad of 18 years of age, and was paralysed in his lower limbs from old-standing spinal disease. At the beginning of December, 1881, Lamson went to the school, where his brother-in-law had been placed as a boarder, had an interview with him, and in the presence of the headmaster, filled a gelatine capsule with a white powder presumed to be sugar and gave it to him. Directly after seeing him swallow it Lamson took his departure. Within a quarter of an hour, the lad became unwell, saying he felt the same as when Lamson had given him a quinine pill on a former occasion, also adding, "My skin feels all drawn up and my throat burning." Violent vomiting soon set in, and he was unable to swallow. He was very restless and delirious, and died in three hours and three-quarters. At the post-mortem examination the brain was found slightly congested. The lungs

1. *Bengal Chemical Examiner's Annual Report*, 1927, p. 13.
2. *U. P. Chemical Examiner's Annual Report*, 1926, p. 4.
3. *Leader*, Sep. 7, 1923.
4. *Patna High Court Cr. Appeal* No. 194 *of* 1923 ; *Criminal Law Jour., June*, 1924, p. 449.
5. *Bengal Chemical Examiner's Annual Report*, 1932, p. 14.

were congested, especially towards the bases. The stomach was red and congested at the cardiac end and at the greater curvature. The duodenum was also red and congested. Stevenson and Dupre[1] detected aconitine in the vomit, stomach contents and viscera. During the trial it was proved that the accused had purchased 2 grains of aconitine on the 24th November.

A case[2] of ordeal by aconite root is recorded. A gold necklace was lost in a house. A man who was said to be an adept in the occult arts was brought by the owner to trace out the culprit. Ten possible suspects were assembled and each of them was given a piece of aconite root to chew in betel leaves. One of them had vomitings and purgings but recovered in hospital.

A case[3] is recorded in which aconite was intended to be given as an abortifacient. One aunt-in-law procured a few aconite pills for administration to a daughter-in-law in a case of illicit pregnancy. The mother-in-law of the girl having noticed the same in time prevented her from taking the pills and sent the same to the police for investigation.

A case[4] occurred at the Darbhanga railway station, where aconite was administered apparently for the purpose of committing robbery. At a sweetmeat stall a batch of six passengers was taking their meals of curd and *chura*, when a man belonging to another group of three became friendly with them and distributed some sugar from a packet among the six passengers. Five of them took this sugar with their meals and soon afterwards "began to feel burning sensation and pain in the stomach." They all suffered from vomiting but had no purging, and four of them died in a few hours.

Aconite is, sometimes, added to Indian liquors to increase the intoxicating effect. A case is recorded in which seventy men were poisoned in Benares by mowrah liquor containing aconite. Eighteen out of them died.[5] In a case referred by the Civil Surgeon of Gaya to the Chemical Examiner of Bengal two men died after drinking toddy drugged with aconite.[6] In Birbhum District five persons died after drinking *pachai* (a kind of country drink) adulterated with aconite.[7] In the Burmese war of 1842, the retreating Burmese poisoned the water of a tank with bruised aconite in the hope that the British soldiers in their pursuit would be poisoned by drinking it.[8]

The root of Aconitum ferox is occasionally used as a cattle poison. A case[9] occurred at Karwi, in which aconite was detected in a substance "found on the generative canal of a she-buffalo."

A root is largely used as an arrow poison by the hill people of Nepal, Assam and neighbouring districts. The Lepchas of Sikkim describe the root as being "useful to sportsmen for destroying elephants and tigers, useful to the rich for putting troublesome relations out of the way and useful to jealous husbands for the purpose of destroying faithless wives." In the Aka expedition of 1884, poisoned arrows were used against British soldiers. Some of these were chemically examined by Lieutenant-Colonel

1. *Johnston, Poison Mysteries in History, Romance and Crime*, 1923, *p.* 298.
2. *Madras Chem. Exam. Annual Rep.*, 1934, *p.* 4.
3. *Beng. Chem. Exam. Annual Rep.*, 1934, *p.* 11.
4. *Bengal Chem. Exam. Ann. Rep.*, 1937, *p.* 13.
5. *Ind. Jour. of Med. Scie., Vol. I, p.* 286.
6. *Ind. Med. Gaz., Aug.*, 1915, *p.* 305.
7. *Beng. Chem. Exam. Annual Rep.*, 1922, *p.* 6.
8. *Chevers, Med. Juris., Ed. III, p.* 136.
9. *U. P. Chem. Exam. Annual Rep.*, 1907.

Waddell, I.M.S., and the heads were found to be smeared over with a paste containing aconite.[1]

Aconitine is eliminated mainly by the urine. Traces have also been found in the saliva, bile and sweat.

Aconitine is extremely unstable and is destroyed by putrefactive processes. Hence it is often difficult to detect it after death. It is also decomposed by alkali. Wood ashes which are usually added to vomit destroy aconite owing to the presence of alkali. Dr. Hankin, therefore, recommends the mixing of the vomit and wood ashes with alcohol and acetic acid which have the power of checking this decomposition. In a criminal case[2] of aconite poisoning in the district of Gorakhpur the Chemical Examiner of the United Provinces of Agra and Oudh failed to detect it in the viscera of the murdered woman or in the dejecta or guavas (in which the poison was mixed). In his letter to the Superintendent of Police he wrote that it was probable that aconite had never been detected either by him or any one else after absorption into the viscera. It might rarely be detected in the contents of the stomach before absorption and also in vomit. Aconite being a virulent poison, only small doses are used and the amount present is, therefore, very little and this fact obviously adds to the difficulty of detection. In a case where the accused had murdered her husband by administering aconite in his food on the 27th March, 1925, no aconite was detected in the viscera which were despatched to the Chemical Examiner, U. P., on the 3rd April, 1925.[3] On the other hand, in a case where one Ali Baksh killed one Khidir by giving aconite mixed with spices in food on the night of April 15, 1924, aconite was detected by the Chemical Examiner in the viscera, vomited matter containing earth and reddish-brown powder.[4] Palet[5] has shown by experiments on white rats injected subcutaneously with lethal doses of aconitine and buried in metallic boxes, that the alkaloid could be readily detected after a lapse of two months.

HYDROCYANIC ACID (HYDROGEN CYANIDE OR PRUSSIC ACID), H C N

This is obtained by distilling potassium ferrocyanide with dilute sulphuric acid. The pure, anhydrous acid is a colourless, volatile liquid, possessing a characteristic odour similar to that of bitter almonds or peach kernels. It solidifies at — 14° C., boils at 26° C., and is more or less rapidly decomposed by exposure to light. It is a powerful poison, and is not found in commerce, but is only met with in chemical laboratories. It is chiefly used to fumigate houses, ships, railway carriages and warehouses for the destruction of rats and vermin.

1. *Beng. Chem. Exam. Ann. Rep.*, 1885.
2. *K. E.* v. *Mathura, Allahabad High Court, Cr. Appeal No.* 91 *of* 1922.
3. *K. E.* v. *Mt. Beatrice and David Masih, Allahabad High Court Cr. Appeal No.* 758 *of* 1925.
4. *K. E.* v. *Ali Baksh, Judicial Commissioner's Court of Oudh Cr. Appeal No.* 608 *of* 1924.
5. *Semana Med.*, 1919, *XXVI, p.* 166; *Peterson, Haines and Webster, Leg. Med. and Toxic., Vol. II, Ed. II, p.* 443.

According to the international agreement of 1930 an aqueous solution of hydrocyanic acid, known as dilute hydrocyanic or prussic acid, should contain 2 per cent by weight of the pure acid. It is a pharmacopœial preparation under the name of *Acidum hydrocyanicum dilutum* with a dose of 2 to 5 minims. Scheele's acid contains from 4 to 5 per cent of the pure acid.

Hydrocyanic acid is widely distributed in nature. It occurs in combination in the leaves of the cherry-laurel, in bitter almonds, in the kernels of the common cherry, plum, peach and other stone fruits, and in many other plants belonging to N. O. *Rosaceœ*. These plants contain a crystalline glucoside, known as amygdalin, which, in the presence of water and a natural enzyme, called emulsin, is readily decomposed into hydrocyanic acid, glucose and benzaldehyde.

The crude essential oil of bitter almonds contains 5 to 14 per cent of hydrocyanic acid, and the non-official preparation of cherry-laurel water (*Aqua laurocerasi*) should contain 0.1 per cent hydrocyanic acid, but it loses strength by keeping. The dose of cherry-laurel water is 30 to 120 minims.

Hydrocyanic acid forms cyanides with metals. Of these potassium cyanide, mercuric cyanide and silver cyanide are used in photography, electroplating and dyeing. These are soluble in water, alkaline in reaction and highly poisonous.

The double cyanides, such as potassium ferrocyanide and potassium ferricyanide, are practically non-toxic, but they give off hydrocyanic acid under certain conditions and act as poisons. Thus, potassium ferrocyanide may produce poisonous symptoms and cause death, when it is taken in association with acids. A case is recorded where death occurred after a dose of potassium ferrocyanide and then one of tartaric acid. Another instance is also reported of the death of a man who took potassium ferrocyanide along with a mixture of equal parts of nitric and hydrochloric acids.[1] In his annual report for the year 1936, the Chemical Examiner, Bengal, reports that a Mahomedan male, aged about 22, committed suicide by taking potassium ferrocyanide. On inspection of the body a big patch of submucous hæmorrhage was noticed in the cardiac end of the stomach.

Symptoms.—This is the most rapid of all poisons. Hence with a large dose the symptoms usually appear within a few seconds or even during the act of swallowing. They are rarely delayed beyond one or two minutes. During the interval the patient may be able to walk or speak or perform some volitional act. The first symptoms are the odour of hydrocyanic acid from the breath, loss of muscular power and giddiness. The patient staggers about, the eyes are wide open, bright and shining, and the pupils are dilated and do not react to light. Consciousness is lost. The respirations become slow and stertorous, with sudden and short inspirations and prolonged expirations. Tonic convulsions affect the jaw rendering it stiff. The pulse is quick and feeble and later becomes inperceptible. These symptoms are followed by cyanosis, cold, clammy skin and relaxation of the sphincters. Death occurs from failure of respiration. Some say that

1. *Blyth, Poisons, Their Effects and Detection, Ed. V, p.* 223.

it is due to the failure of internal tissue respiration brought on by a change of the blood due to the formation of cyanmethæmoglobin, which yields a spectrum resembling that of reduced hæmoglobin, *i.e.*, a thick band between the lines D and E.

When a small poisonous dose is taken, the patient experiences a hot, bitter taste and constriction of the throat and complains of salivation, giddiness, nausea, headache, confusion of ideas, sense of oppression in the chest, loss of muscular power and insensibility. The face is suffused or bloated, and the mouth is covered with froth ; the eyes are glassy and prominent with dilated pupils ; the finger-nails are blue or purple. Convulsions of a tentanic character and involuntary evacuations precede death. Vomiting is occasionally observed and is, sometimes, the beginning of reocvery.

The spasmodic or piercing cry, which is commonly observed in cattle poisoning, is rarely met with in human poisoning.

Inhalation of the vapours of hydrocyanic acid produces a sense of constriction about the throat and chest, dizziness, vertigo, insensibility and death from respiratory failure. Tatham reports a case in which there were disturbances of vision.[1]

Potassium cyanide, which is strongly alkaline and frequently contains potassium carbonate as an impurity, has a corrosive effect on the mouth, throat and stomach and causes epigastric pain and vomiting. The other symptoms are cyanosis of the face, neck and hands, white froth about the lips, dilated pupils, imperceptible pulse, slow and shallow respirations, incontinence of urine, coma and death. Sometimes, convulsions may precede death. Williams[2] reports non-fatal cases of acute and severe gastro-enteritis in hotels from cyanide poisoning apparently from silver polish containing sodium cyanide to the extent of 20.54 per cent.

Chronic poisoning occurs among photographers, gilders and workmen who are constantly engaged in preparing or handling either hydrocyanic acid or potassium cyanide. The symptoms are headache, vertigo, loss of appetite, nausea, constipation, fœtid breath, dyspnœa, and anæmia.

Fatal Dose.—The smallest quantities that have proved fatal are half a drachm of dilute hydrocyanic acid[3] and 20 minims of Scheele's acid[4] equivalent to 0.6 grain and 1 grain of the anhydrous acid respectively. One drachm of dilute hydrocyanic acid would commonly be sufficient to destroy the life of an adult. Recovery has, however, occurred after taking 4 drachms of the dilute acid equivalent to 4.8 grains of the anhydrous acid.[5] Two grains and a half of pure potassium cyanide may be regarded as a minimum fatal dose. A dose of 5 grains of potassium cyanide has proved fatal in some cases,[6] though recovery has followed much larger

1. *Brit. Med. Jour.*, 1884, *Vol. I, p.* 409.
2. *Jour. Amer. Med. Assoc., March* 1, 1930, *p.* 627.
3. *Garstang, Lancet,* 1888, *Vol. II, p.* 15.
4. *Med. Gaz., Vol. XXXV, p.* 896.
5. *Shively, Amer. Jour. Med. Scie., n. s., c.* 1890, *p.* 47.
6. *Taylor, On Poisons, Ed. III, p.* 624.

doses of 20[1], 40[2] and even 50 or 60[3] grains. Seventeen as well as thirty drops of oil of bitter almonds have produced fatal results,[4] but recovery has taken place after doses of from 4 to 6 drachms in some cases.[5] Twelve hundred grains of bitter almonds proved fatal to a woman, 31 years old.[6] Death has also occurred from a handful of bitter almonds in two hours,[7] while recovery has taken place after a dose of two handfuls.[8] One and-a-half to two ounces of cherry laurel water have caused death.[9]

A concentration of one volume of hydrocyanic acid gas in 2,000 parts of air is generally fatal to animals. A concentration of 0.2 to 0.3 mlg. of the gas per litre of air is regarded as sufficient to kill men almost immediately, while a concentration of 0.13 mlg. per litre of air and an exposure of over an hour are sufficient to prove fatal to men.

Fatal Period.—Two to ten minutes. It is possible that life may be prolonged for three and-a-half hours,[10] but in most cases the patient will recover, if death does not occur within an hour.

In poisoning by potassium cyanide death may take place as rapidly as in hydrocyanic acid poisoning. Powell[11] reports a case in which death occurred from commercial potassium cyanide in seven to twelve minutes. A student of Lucknow University died within 10 to 15 minutes after taking potassium cyanide. Taylor[12] cites cases in which death took place in fifteen minutes, twenty minutes and two hours. In a few cases death has been delayed for twenty-four hours.[13]

Treatment.—There is hardly time for treatment, if strong hydrocyanic acid is taken. In the case of potassium cyanide or dilute hydrocyanic acid poisoning, wash out the stomach immediately with a dilute solution of hydrogen peroxide or potassium permanganate. Vinegar may be added if the poison is potassium cyanide. If the stomach tube is not available produce vomiting by mustard and water aided by tickling the fauces or the hypodermic injection of apomorphine hydrochloride.

Cold affusions to the head and chest, and inhalation of ammonia should be followed by the hypodermic injection of 1/50 grain of atropine and ether or brandy, and artificial respiration.

If death is delayed, a mixture of ferrous and ferric sulphates with carbonate of potassium may be given as a chemical antidote to produce

1. *Higgins, Medical Record,* 1891, *XL, p.* 687.
2. *Ord., Lancet,* 1886, *Vol. II, p.* 1174.
3. *Gillebrand, Lancet,* 1876, *Vol. II, p.* 223.
4. *Prov. Med. Jour., Sep.* 11, 1844, *Taylor, Loc. Cit., p.* 613.
5. *Assoc. Med. Jour.,* 1854, *Vol. II, p.* 885; *Lancet,* 1839, *Vol. II, p.* 930; *Int. Med. Mag., Phila.,* 1893, *Vol. II, p.* 126.
6. *Blyth, Poisons, Their Effects and Detection, Ed. V, p.* 223.
7. *Zemke, Munch. Med. Woch., No.* 24, 1905, 52, *p.* 1172; *Peterson, Haines and Webster, Leg. Med. and Toxic., Ed. II, Vol. II, p.* 683.
8. *Baker, Brit. Med. Jour.,* 1881, *Vol. I, p.* 12.
9. *Taylor, On Poisons, Ed. III, p.* 619; *Casper, Forensic Med., Vol. II (Engl. Trans. by Balfour), p.* 88.
10. *Brit. Med. Jour.,* 1883, *Vol. I, p.* 131.
11. *Ind. Med. Gaz., Aug.,* 1902, *p.* 306.
12. *On Poisons, Ed. III, pp.* 624-625.
13. *Witthaus, Manual of Toxic., Ed. II, p.* 806.

the innocuous Prussian blue. Martin and O'Brien[1] recommend the use of 1 ounce of a twenty-three per cent solution of ferrous sulphate, 1 ounce of a five per cent solution of caustic potash and 30 grains of powdered magnesium oxide. Intravenous injection of 10 c.c. of a ten per cent solution of sodium hyposulphite (thiosulphate) has been recommended, as it combines with hydrocyanic acid and forms non-poisonous sulphocyanate of sodium. Intravenous injections of glucose or glucose and insulin are regarded as beneficial.

In poisoning by potassium cyanide intravenous injection of 50 c.c. of a one per cent sterile aqueous solution of methylene blue (methyl thionine chloride, U. S. P.) after lavaging the stomach with water containing sodium bicarbonate has been recommended as an antidote. Methylene blue converts the hæmoglobin of the blood into methæmoglobin, which combines with the free cyanide, thereby removing it from the reaction. Cases are recorded where recoveries by this method of treatment occurred after 15 and 100 grains of potassium cyanide had been taken.[2]

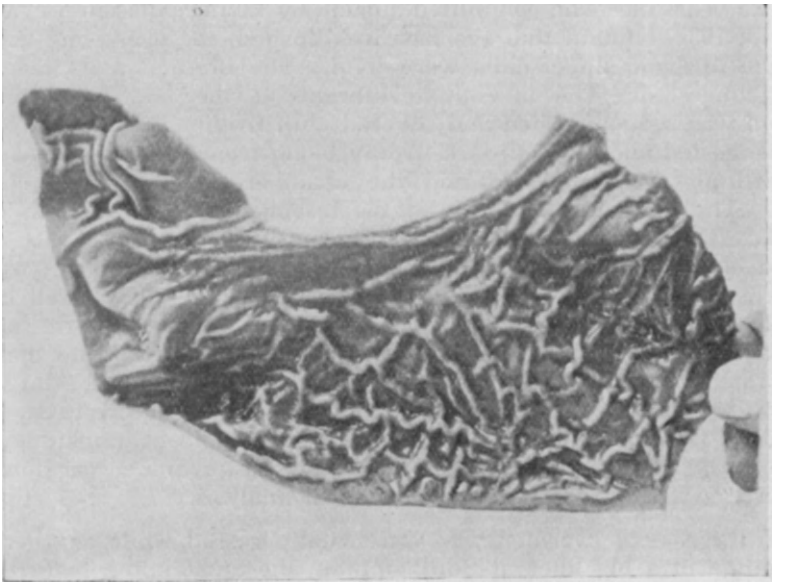


Fig. 158.—Stomach in poisoning by potassium cyanide. (*From the Pathological Museum, Grant Medical College, Bombay*).

Post-mortem Appearances.—The skin presents a livid or violet colour. The post-mortem stains are bright red or pink, due to the formation of cyanmethæmoglobin and to the fact that the tissues cannot take up the oxygen of the blood, leaving it bright red even in the veins. The fingers are clenched, the finger-nails are blue, the jaws are firmly closed and there

1. *Proc. Soc. Chem. Ind., Victoria,* 1901, *Vol. I, p.* 119.
2. *Geiger, Jour. Amer. Med. Assoc., Dec.* 3, 1932, *p.* 1944; *Jour. Amer. Med. Assoc., July* 22, 1933, *p.* 269.

is froth at the mouth. The eyes may be bright, glistening and prominent with dilated pupils. Rigor mortis sets in early and lasts longer.

The odour of hydrocyanic acid may be noticed on opening the body. There may be bloody froth in the trachea and bronchi. The right side of the heart is full and engorged with venous blood, which is fluid and bright red. The lungs are congested. The serous cavities are ecchymosed.

In poisoning of hydrocyanic acid, the mucous membrane of the stomach and duodenum is often red and congested, although it may be normal in some cases. In cases where potassium cyanide has been taken, the lips and mouth may be corroded, and the mucous membrane of the stomach and duodenum may be bright red, inflamed, softened, and even ulcerated. In the case reported by Powell the angles of the lips, the mucosa of the tongue, cheeks, pharynx and œsophagus were whitened, feeling soapy to the touch. The mucosa of the stomach was dissolved leaving a raw surface. On section the epithelium was found to have disappeared except from the bottom of a few follicles.[1] At the autopsy on the body of a Hindu male, 25 years old, who committed suicide by taking potassium cyanide on July 13, 1932, I found that the face was flushed, the eyes were congested and the lips and finger-nails were livid. The brain and its membranes were congested. The mucous membrane of the larynx, trachea and bronchi was red, congested and covered with froth. The lungs were dark red, congested and exuded dark, frothy blood from the cut surfaces. The pericardium was congested and the chambers of the heart contained blood. The mucous membrane of the œsophagus was red with injected vessels. The stomach was empty. Its mucous membrane was red, inflamed and presented a velvety appearance. The stomach wall was thickened, corrugated and the vessels were injected. The small intestine contained reddish liquid matter. The mucous membrane in its upper part was red, inflamed with hæmorrhagic patches under it and the vessels were injected. The lower part of the small intestine and the large intestine were normal. The large intestine contained liquid fæcal matter. The liver was dark red and congested and the spleen and kidneys were congested. The bladder was empty. Potassium cyanide was detected in the viscera usually preserved for chemical analysis.

In the case of two men who were fatally gassed while engaged in the fumigation of a big liner at Southampton, the outstanding features noted at the post-mortem examination held two days later were the absence of smell before opening the body, gaseous distension of the peritoneal cavity and intestines, smell of hydrocyanic acid in all serous cavities, chiefly in the lateral ventricles of the brain, marked lividity of the face, blue nails, pink colour of the gastric and intestinal mucosa, bright pink staining of the intima of the aorta, uniform blue-green colour of the liver, green colour of the grey matter of the brain, lividity and engorgement of the buccal, pharyngeal, œsophageal and respiratory mucous membranes, flabbiness of the myocardium and extravasation of blood into the tissues of the stomach wall posteriorly in the region of the fundus.[2]

1. *Ind. Med. Gaz., Aug.*, 1902, *p.* 306.
2. *Seager Thomas, Lancet, June* 16, 1923, *p.* 1210.

Analysis.—It is very necessary that the analysis of the viscera should be made as soon as practicable after death, as hydrocyanic acid, being a volatile and unstable compound, is readily decomposed, especially if the conditions favouring putrefaction are present. However, the acid has been detected in putrefied viscera a long time after death. Thus, Autenrieth[1] detected its presence after 60 days when the organs were in a high state of decomposition. Jollyman[2] found potassium cyanide in the stomach contents of a Negro six months after death.

Tests.—1. Silver nitrate produces a white precipitate of silver cyanide, soluble in strong, boiling nitric acid. It does not turn dark on exposure to light, but shows prismatic needles under the microscope while silver chloride is amorphous, and turns dark on exposure to light.

2. *Prussian Blue Test.*—Caustic potash, ferrous sulphate and ferric chloride produce a brown precipitate, which becomes deep blue (Prussian blue) on the addition of hydrochloric acid.

3. *Liebig's Test.*—If a little ammonia and ammonium sulphide be added to a solution containing hydrocyanic acid and heated to dryness, a blood-red colour will be developed on further adding a drop of ferric chloride; the colour will disappear if treated with mercuric chloride.

4. If a strip of white paper is moistened with copper-benzidine solution and dipped into the suspected gastric contents, the paper assumes a distinct blue colour if hydrocyanic acid is present. Copper-benzidine solution is prepared by adding 1 c.c. of a three per cent solution of copper acetate and 5 c.c. of a saturated solution of benzidine in glacial acetic acid to 15 c.c. of water.

Medico-Legal Points.—Hydrocyanic acid and various cyanides are often used for suicidal purposes, as their swift and sure action is generally known.

A Bengali student who failed at the University examination swallowed the contents of a bottle of hydrocyanic acid. There was time to remove him to the Medical College Hospital where he died in about 15 minutes.[3] The Chemical Examiner of Madras reports two cases of suicidal poisoning by potassium cyanide. In one a man and his wife took the poison together after going to bed, and died in about 40 minutes. There was neither vomiting nor purging. On post-mortem examination the mucous membranes of the stomachs were congested but not very markedly so. In the other case death was almost instantaneous.[4] The Chemical Analyser of Bombay[5] reports a case in which two young persons, a young man and his wife, were lying in a room in the city of Bombay apparently in a dying condition. They were removed to J. J. Hospital, but expired on the way. The post-mortem examination revealed an intensely congested and hæmorrhagic condition of the stomachs and other viscera, and white masses smelling of hydrocyanic acid were found in the stomachs. Chemical analysis confirmed this finding.

These preparations are rarely used with homicidal intent, as they are easy of detection owing to their characteristic odour and perceptible taste.

1. *Detection of Poisons, Ed. VI, p.* 36 *(Eng. Trans. by Warren).*
2. *Ibid., p.* 37; *Chemilker-Zeitung Jahrgg.*, 1905, 29, *p.* 350.
3. *Choonilal Bose, Ind. Med. Gaz., Aug.*, 1915, *p.* 304.
4. *Annual Report*, 1922, *p.* 3.
5. *Annual Report*, 1928, *p.* 6.

In the report of the Chemical Examiner, Bengal, for the year 1906, Choonilal Bose[1] records a case of theft and murder in which a woman of the town of Calcutta was seen drinking with a stranger in her room one evening; shortly afterwards she was discovered by the other inmates of the house lying on the floor and she died soon afterwards. The stranger had already absconded. The post-mortem appearances were consistent with death from heart failure. Hydrocyanic acid was detected in the viscera. Hydrocyanic acid and alcohol were also detected in the viscera of a public woman, aged 25, of Calcutta. She entertained visitors with drink till midnight and was found dead in her room on the following morning with all her ornaments missing.[2] The motive of the crime was apparently theft.

A man, aged 23, tried to poison a girl, aged 18, by the contents of a so-called Indian "poison bladder" which contained in one 2 c. cm. ampoule 0.3 gramme of hydrocyanic acid in solution and then murdered her by hanging.[3]

A few homicidal cases of poisoning by potassium cyanide have also been recorded. A Hindu female child, aged about 10 years, was said to have been poisoned by her father with potassium cyanide. He afterwards committed suicide by taking the same poison. Two persons used to commit murders especially of prostitutes by administering potassium cyanide in liquor and then used to deprive them of their money and jewellery.[4] In order to advance his career Lieutenant Hofrichter of Linz sent "tonics" containing potassium cyanide to various officers, and in 1909 poisoned Captain Møder in Vienna with 2 grammes of potassium cyanide.[5] In October, 1932, the chief witness for the prosecution in an action against Communists was poisoned at Chemnitz, on the day before the trial, by chocolates containing potassium cyanide which were sent to him.[5]

Oil of bitter almonds and cherry-laurel water (*aqua laurocerasi*) are used as flavouring agents, and have caused accidental poisoning. In the case of *Rex* v. *Captain Donellan*, the accused was charged with having caused the murder of his brother-in-law, Sir T. Boughton, by administering two ounces of cherry-laurel water in place of a purgative draught.[6]

Accidental poisonings have occurred from the inhalation of the vapour of the acid used as a fumigating agent, from the ingestion of the pharmacopœial acid in mistake for some other drug, from its application to a wound or raw surface,[7] and from the injection of potassium cyanide into the rectum.[8]

Cases of cattle poisoning are known to occur through eating *juar kadvi* and *alsi* (linseed) plant. This is due to the natural development of a cyanogenetic glucoside, particularly in the young plants, which, under certain circumstances, breaks up and yields hydrocyanic acid. Bagchi and Ganguli[9] have demonstrated that the linseed plant (Linum usitatissimum) contains a cyanogenetic glucoside in all stages of its growth, but the linseed flowers with immature seeds contain the maximum amount of the cyanogenetic glucoside producing as much as 0.69 per cent of free hydrocyanic acid, and about half a pound of these flowers is sufficient

1. *Ind. Med. Gaz., Oct.*, 1907, *p.* 394.
2. *Bengal Chemical Examiner's Annual Report*, 1929, *p.* 11.
3. *Schwarzacher Beitr. gerichtl. Med.*, 1931, *XI*, 48-53; *abstr*: *Deuts. Zeits. f. d. ges. gerichtl. Med.*, 1932, *XIX*, 27; *The Med.-Leg. and Criminol. Rev., Jan.*, 1933, *p.* 85.
4. *Beng. Chem. Exam. Annual Rep.*, 1930, *pp.* 11-12.
5. *Erich Leschke, Clinic., Toxic., Eng. Trans. by Stewart and Dorrer*, 1934, *p.* 144.
6. *Taylor, On Poisons, Ed. III, p.* 620.
7. *Wilkes, Lancet*, 1904, *Vol. II, p.* 1058.
8. *Bull. gen. de ther.*, 1869, *LXXVII, p.* 458; *Witthaus, Med. Juris. and Toxic., Vol. IV, p.* 800.
9. *Ind. Jour. Veter. Sc. and Animal Husbandry, Vol. IX, Part I, March*, 1939, *p.* 61.

to prove fatal to a bullock. The fatal dose for a bullock is about 20 grains of pure hydrocyanic acid. Very dilute acids and alkalis only retard the liberation of hydrocyanic acid, but a normal acid or alkali stops it altogether. A strong solution of an alkali (sodium carbonate) is, therefore, recommended as an antidote in poisoning by the linseed plant.

Five buffaloes died after eating *juar* or young *jonera* plants, which yielded hydrocyanic acid on analysis.[1] In Jodhpur 95 cattle and 2 goats were affected by eating from two bundles of *juar kadvi*. Twenty-four of the cattle and the goats died. On examination of the remnants of the two bundles of the *kadvi* hydrocyanic acid amounting to 0.0112 and 0.0073 per cent respectively was found.[2] In Wasaliganj, Gaya, 21 cattle became ill after eating the dry linseed plant and its dry fodder. Of these seven died.[3]

Hydrocyanic acid is rapidly absorbed from all surfaces, even from the unabraded skin. Part of the acid thus absorbed is eliminated unchanged by the lungs. Another part is changed in the tissues to sulphocyanides, which are excreted in the urine. It is also partially eliminated by the skin.

The toxic action of potassium cyanide depends largely upon the hydrochloric acid content of the stomach. It is said that Rasputin was given a large amount of potassium cyanide in pastries, but he did not suffer from any ill-effects, as owing to alcoholic gastritis the liberation of hydrocyanic acid was inhibited by the lack of free hydrochloric acid in the stomach, and absorption was hindered by the thickening of the gastric mucous membrane.[4]

1. *Beng. Chem. Exam. Ann. Rep.; Ind. Med. Gaz., Sep.*, 1910, *p.* 863.
2. *U. P. Chem. Exam. Ann. Rep.*, 1926, *p.* 4.
3. *Beng. Chem. Exam. Ann. Rep.*, 1936, *p.* 16.
4. *Leschke, Clin. Toxic., Eng. Trans. by Stewart and Dorrer*, 1934, *p.* 145.

CHAPTER XXXIV

ASPHYXIANTS (IRRESPIRABLE GASES)

CARBON DIOXIDE (CARBONIC ACID GAS, CARBONIC ANHYDRIDE), CO_2

This is a colourless, suffocating gas, having a slightly acid taste and a faint and pleasantly pungent smell. It is a constituent of the atmospheric air in which it exists to an extent of 0.04 per cent and the air containing 2 per cent causes discomfort, while more than 20 per cent causes death, but if the air is contaminated with carbon dioxide from respiration or combustion, 5 per cent of it may be fatal. It is given off in the process of respiration, combustion, fermentation and putrefaction of animal matter. It forms the choke damp or after damp of the coal mines. It is also evolved in the neighbourhood of lime kilns on account of decomposition of carbonates. Being heavier than air it tends to accumulate at the bottoms of old wells, damp cellars, mine shafts, brewers' vats, grain pits, etc.

Carbon dioxide (*Carbonei dioxidum*) is included in the British Pharmacopœia of 1932. It acts as a mild rubifacient when applied externally in the form of a solution, but in a solid form it acts as a caustic.

Symptoms.—These are heaviness in the head, throbbing of the temporal arteries, giddiness, ringing of the ears, a sense of oppression, muscular weakness, drowsiness and insensibility passing into coma with stertorous breathing. Death occurs from asphyxia or apoplexy. Sometimes there may be convulsions and delirium.

When inhaled in a concentrated form, immediate insensibility occurs followed by death from spasm of the glotis causing suffocation.

Treatment.—The patient must at once be removed into the open air, and artificial respiration should be started with inhalation of oxygen. This ought to be assisted by galvanism and friction of the extremities. After breathing is established, the body should be well covered with blankets, and coffee or brandy should be administered internally. If a patient is seen lying unconscious at the bottom of a well or pit used for storing grain, an attempt should be made to discharge oxygen from an oxygen holder into the bottom of the well or pit by means of a hose that it may not only revivify the patient but displace the carbon dioxide, so that others can descend to render him help.

Post-mortem Appearances.—The body heat is retained for a longer period. The face is usually pale and placid, but may be swollen and caynosed. The pupils are dilated. The brain and lungs are usually congested. The right side of the heart contains dark fluid blood with venous engorgement, and the left is empty. Ecchymosed patches are noticed in the small intestine.

A male child, 4 months old, was shut up in a small steel trunk on or about the 29th November, 1930, and died consequently. At the post-mortem examination held by me on the next day the face was flushed, and the lips and finger-nails were cyanosed. The mucous membrane of the larynx and trachea was congested and covered with froth. The lungs were congested and exuded frothy blood from the cut surfaces. The liver, spleen and kidneys were congested.

Tests.—1. Carbon dioxide makes lime-water milky.

2. A burning candle will be extinguished in the air containing more than 6 per cent of carbon dioxide.

3. Barium nitrate gives a white precipitate of barium carbonate with carbonic acid, soluble with effervescence in hydrochloric or nitric acid.

4. Silver nitrate gives a white precipitate of silver carbonate.

Medico-Legal Points.—Cases of poisoning by carbon dioxide are mostly accidental. A thief in Agra died from carbon dioxide poisoning by sleeping near a lime kiln.

CARBON MONOXIDE (CARBONIC OXIDE GAS), CO

This is prepared by the decomposition of certain organic substances, such as oxalic and formic acids, by means of sulphuric acid, and is formed whenever carbon is burned with an insufficient supply of air or oxygen. It is found in the gaseous products from charcoal stoves, salamanders, blast furnaces, lime kilns, gas engines and burning houses. It is generated in a large amount when gunpowder or dynamite is exploded, and when explosions occur in coal mines. It is a constituent of coal gas, the amount varying from 6 to 12 per cent. It is found in the proportion of 30 to 40 per cent in water gas which is obtained by blowing steam through red hot coal or coke. It is present in quantities varying from 6 to 9 per cent in the exhaust gases of motor cars. The quantity of carbon monoxide produced per minute by a 20 horse-power motor car is approximately 1 cubic foot, which is enough to render the atmosphere of a small closed garage of 10 by 10 by 20 feet deadly in less than ten minutes.[1] Carbon monoxide also occurs in tobacco smoke. Dixon[2] has showed that cigarette smoke obtained by means of an aspirator contains 410 c.c. of carbon monoxide in 100 grammes of tobacco consumed.

Carbon monoxide is a colourless, tasteless, inodorous gas. It is almost insoluble in water and alcohol. It burns with a blue flame, forming carbon dioxide, and explosive mixtures with air or oxygen. It combines with metals, such as nickel and iron, and forms colourless liquids, known as carbonyls. Combined with chlorine, it forms carbonyl chloride, commonly called phosgene, which was used as a poisonous gas during the last Great War.

Carbon monoxide is a highly poisonous gas, as it readily combines with the hæmoglobin of the red blood-corpuscles to form a stable

1. *Henderson, Brit. Med. Jour., Jan.* 9, 1926, *p.* 44.
2. *Manual of Pharmacology, Ed. VI, p.* 106.

compound, known as carboxyhæmoglobin, and thus reduces the oxygen-carrying power of the blood. The affinity of carbon monoxide for the blood is about 200 times greater than that of oxygen, so that as long as carbon monoxide is present in the atmosphere, it becomes fixed cumulatively in the blood.

Symptoms.—When the gas is inhaled in a concentrated form, sudden insensibility supervenes immediately followed by coma and death. Coma may last for three, four or five days even after the patient has been removed from the gas. In one case a patient remained comatose for eight days and died on the twelfth day after the fatal inhalation.[1] In such cases there may be broncho-pneumonia or œdema of the lungs.

When inhaled in a diluted form, the symptoms are dizziness, headache, noises in the ears, nausea, sometimes vomiting, muscular weakness, drowsiness, dilated pupils, retarded breathing, coma and death. In some cases tremors and convulsions may precede death.

Nervous and mental symptoms are occasionally manifested after recovery from the effects of the gas. The symptoms may be cerebral hæmorrhage, encephalitis, optic neuritis, chorea, spastic paraplegia, retrograde amnesia, aphasia, mental confusion, transient mania, and dementia.

It should be remembered that the symptoms are not noticeable until the hæmoglobin is about 20 per cent saturated with carbon monoxide when shortness of breath is observed. When the saturation increases to 30 per cent, there is a slight increase in the rate of the pulse and respiration, followed by headache, nausea and faintness. From experiments made upon himself Haldane[2] has found that the loss of memory, mental confusion and inco-ordination of movement are the marked symptoms when the hæmoglobin reaches a saturation of 30 to 40 per cent. Forty to fifty per cent saturation causes collapse and inability to move the limbs, and 60 to 70 per cent saturation causes unconsciousness and rapid death. According to Haldane[3] the hæmoglobin is about 80 per cent saturated in deaths from carbon monoxide poisoning. In persons in ill-health death may occur with a much lower percentage of carbon monoxide in the hæmoglobin. Spilsbury[4] cites a case of suicide in which a young woman suffering from chronic tuberculosis of the lungs died when her blood reached a saturation of only 45 per cent. In two other cases, in which the fatal percentage was about 50, one was an old feeble person, and the other was suffering from cancer of the stomach.

According to Gruber[5] the air containing 0.02 per cent carbon monoxide is at the limit of toxicity, while the air containing 0.05 per cent causes distinct toxic symptoms. The Board of Trade reported in 1924, that an atmosphere containing 0.25 per cent carbon monoxide or 3 to 3.3 per cent coal gas would prove fatal to a healthy adult in about four hours. A smaller percentage would be fatal if the exposure was over a prolonged

1. *Blyth, Poisons, Ed. V, p.* 75.
2. *Brit. Med. Jour., July* 5, 1930, *p.* 16.
3. *Ibid.*
4. *Brit. Med. Jour., July* 5, 1930, *p.* 16.
5. *Autenrieth, Laboratory Manual for the Detection of Poisons and Powerful Drugs (English Translation by Warren), Ed. VI, p.* 324.

period. The air containing 1 per cent carbon monoxide would cause 50 per cent saturation of the blood in fifteen minutes, and 80 per cent in twenty-three minutes, when death would result. However, if the victim exerted himself while absorbing the first part of the carbon monoxide, he might be breathing four or five times as much, and reach the 50 per cent saturation in five minutes.[1] Henderson and Haggard[2] from their experiments have laid down a standard for calculating the toxic action of carbon monoxide, which depends upon the amount of the gas and the time of exposure. When the time of exposure in hours multiplied by the concentration of carbon monoxide in parts per 10,000 of air equals 3, there is no perceptible physiological effect. When the product equals 6, there is a just perceptible effect, perhaps a slight headache and lassitude ; when it equals 9, severe headache and nausea result ; when it equals 15, the condition is dangerous, and when it rises above 15, the conditions are such as will be quickly fatal.

Chronic Poisoning.—This form of poisoning is found in persons, who are constantly exposed to the action of the gas in gas houses and automobile workshops, and in those inhabiting ill-ventilated rooms, in which fire is burning.

Symptoms.—These are headache, nausea, digestive disturbances, dyspnœa, dizziness, mental torpidity, loss of memory, wasting of the muscles, anæmia, and in some cases symptoms of peripheral neuritis and glycosuria.

Treatment.—Remove the patient into the fresh air. Commence artificial respiration and supplement it by the administration of oxygen containing 5 per cent of carbon dioxide. This mixture stimulates the respiratory centre, deepens the respirations, assists the oxygen absorption and eliminates rapidly the carbon monoxide from the hæmoglobin. Inject hypodermically 0.5 to 1 c.c. of adrenaline and intravenously 5 c.c. of 25 per cent coramine solution and repeat them, if necessary, at intervals of from half an hour to an hour. Keep the patient warm by applying hot-water bottles to the extremities and covering the body with blankets.

In severe collapse administer subcutaneously normal saline or resort to blood transfusion.

After breathing is established, watch the patient carefully for the after-effects of carbon monoxide poisoning, and give him hot tea or coffee when he is conscious enough to swallow.

Post-mortem Appearances.—Externally, the lips and finger-nails have a bright red colour. Irregular patches of a bright red colour are scattered over the anterior surface of the body, and the post-mortem stains appearing on the dependent parts have also the same bright red colour. Internally, the blood is fluid and of a bright cherry-red colour due to the formation of carboxyhæmoglobin. The internal organs are hyperæmic and are bright red owing to the colour of the blood. The mucous membrane of the air-passages is bright red, and is often covered with froth. The

1. *Haldane, Brit. Med. Jour., July* 5, 1930, *p.* 16.
2. *Jour. Industrial and Engineer. Chemi.*, 1922, *Vol.* 16, *p.* 229 ; *Brit. Med. Jour., Jan.* 9, 1926, *p.* 45.

lungs are congested, and may, occasionally, be œdematous. There is serous effusion into the ventricles of the brain.

Punctiform hæmorrhages and softening in the cortex and the lenticular nuclei may be found in chronic carbon monoxide poisoning. There may be fatty degeneration of the heart and kidneys.

Tests.—1. *Spectroscopic Test.*—The spectrum of the blood will show two absorption bands similar to those of oxyhæmoglobin, but placed nearer the violet end. The addition of ammonium sulphide does not alter the spectrum.

2. *Hoppe-Seyler's Test.*—Caustic soda of specific gravity 1.3 produces a greenish colour, if added to normal blood, but retains the bright red colour, if carbon monoxide is present in the blood.

3. *Kunkel's Test.*—The blood, diluted with 4 volumes of water, is mixed with 3 times its volume of 1 per cent tannic acid solution and shaken well. Carbon monoxide blood forms a crimson-red coagulum, which retains its colour for several months. Normal blood forms a coagulum which is at first red, becomes brown in the course of one to two hours and then becomes grey in twenty-four to forty-eight hours. The blood saturated even with 10 per cent carbon monoxide responds to this test.

4. *Potassium Ferrocyanide Test.*—If 15 c.c. of blood is mixed with an equal amount of 20 per cent potassium ferrocyanide solution and 2 c.c. of dilute acetic acid and shaken gently, a bright red coagulum will form, if the blood contains carbon monoxide, while a dark brown coagulum will form if the blood is normal.

The Reversion Spectroscope method designed by Professor Hartridge is very convenient for the quantitative determination of carbon monoxide present in blood.[1]

Medico-Legal Points.—Poisoning by carbon monoxide is mostly accidental. Accidents may occur in connection with incomplete combustion of wood, charcoal or coal in ill-ventilated rooms, leaky gas pipes and taps in dwellings, and motor car exhausts in small garages or even in narrow streets where motor traffic is very dense.

On the night of the 10th February, 1924, a family consisting of a man, aged 35 years, his wife, aged 25 years and a son, aged 10 years, went to sleep in a closed room, where coal was kept burning to ward off cold. Next morning the boy was found dead in bed, and the man and the woman were found in a state of unconsciousness. They were immediately removed to the King George's Hospital, Lucknow. On admission they were found in a comatose condition, and the limbs were rigid and the reflexes were exaggerated. They gradually recovered in six or seven days. The blood of the three victims showed the presence of carbon monoxide in the spectroscope.

Sherman, Swindler and Mc Ellroy[2] describe three cases of collapse under the use of ethylene as an anæsthetic, of which two proved fatal. The blood from the patients showed 50 to 60 per cent saturation with

1. *For details see Sydney Smith and Glaister, Recent Advances in Forensic Medicine, Ed. II, p.* 201.
2. *Jour. Amer. Med. Assoc., June* 5, 1926, *p.* 1765.

carbon monoxide. The cylinder of ethylene was found to contain carbon monoxide concentration of 0.7 per cent.

Suicidal poisoning by carbon monoxide frequently occurs in England and other Western countries. The victim generally shuts himself up in a room after placing smouldering fire and after closing all the doors and windows. Sometimes, the suicide sleeps in a room where a gas tap is turned on or he may attach a tube to the gas tap and then put it in his mouth. In India, suicide by carbon monoxide is rare. An Anglo-Indian, 65 years old, and resident of Lucknow, was found dead on a couch in a small room of his house at about 4 p.m. on October 25, 1929. The room had been closed from inside, all openings to allow ventilation had been closed and charcoal had been kept burning in an *angethi* (stove). A case[1] also occurred in Bombay where a European committed suicide by sitting in a chair near a gas stove in his kitchen after opening the gas tap and then covering his head and the gas stove with a rug and a sheet. The door and windows of the kitchen had also been shut.

The use of carbon monoxide for homicidal poisoning is very rare, although a few cases have been recorded. A murderer may turn on a gas tap when his victim is asleep in his bed-room, and thus suffocate him to death without disturbing him.

The elimination of the gas from the blood after the patient has been removed from the atmosphere containing carbon monoxide is very slight for the first hour and-a-half, but becomes rapid after that and provided the patient lives, all the carbon monoxide would have been eliminated from five to six hours.[2]

A case[3] is recorded in which the post-mortem appearances which simulated very closely those of carbon monoxide poisoning were due to the formation of nitric-oxide-hæmoglobin (nitroxyhæmoglobin) after death. A man employed at a colliery in stoking the boiler furnaces died after an illness of nine days. At the post-mortem examination held within a few hours, the whole of the blood, in whatever part of the body, including the heart, spleen, kidneys, muscles and lungs, had a bright red colour exactly similar to that seen in death from carbon monoxide. The blood also responded to the usual tests of carboxyhæmoglobin. Hence a verdict was given at the inquest that the death was due to carbon monoxide poisoning. On further investigation it was, however, found that the red colour of the blood was due to the development of nitric-oxide-hæmoglobin probably by the action of a nitrifying infective organism in the body. A solution of the blood containing nitric-oxide-hæmoglobin can be distinguished by boiling, since it gives a pink coagulum, while the blood containing oxyhæmoglobin and carboxyhæmoglobin gives a dull grey coagulum.

Carbon monoxide retards putrefaction, and may be detected in the blood several days after death from poisoning by this gas. Autenrieth[4] detected carbon monoxide in the blood of an adult two months after he

1. *Times of India*, Dec. 13, 1934.
2. *Henderson, Brit. Med. Jour., Jan.* 9, 1926, *p.* 45 ; *Douglas J. Kerr, Ibid., March* 5, 1927, *p.* 415.
3. *Banham, Haldane and Savage, Brit. Med. Jour., Aug.* 1, 1925, *p.* 187.
4. *Detection of Poisons, Ed. VI, English Trans. by Warren, p.* 325.

died from poisoning by coal gas. Laguna[1] describes a case in which carbon monoxide was detected chemically and spectroscopically in the fluid contents of the pleura and abdomen of a woman whose body was exhumed seven months after death which occurred suddenly from poisoning by carbon monoxide from a defective oven. On the other hand, Dr. Mathur of the Physiological Department of the King George's Medical College, Lucknow, has come to the conclusion from investigations carried out on rats in January, 1933, that in cases of deaths by carbon monoxide the organs begin to decompose after the third day and the blood, after the fourth day.

CARBON DISULPHIDE (CARBON BISULPHIDE), CS_2

This is a colourless, highly refractive, volatile, mobile liquid, with a disgusting odour. It boils at 46° C. Being highly inflammable, it burns with a blue flame, forming carbon dioxide and sulphur dioxide. It is not miscible with water, but freely dissolves in alcohol, ether, chloroform, hydrocarbons of the benzene family and most of the essential oils. It is used in the arts as a solvent for caoutchouc, India-rubber, phosphorus, sulphur, etc., and for extracting essential oils, spices and perfumes.

Acute Poisoning.—This form of poisoning occurs from swallowing the liquid or from inhaling its vapour.

Symptoms.—Intense burning pain in the throat, headache, giddiness, drowsiness, unconsciousness, dilated pupils, cyanosed lips, cold, damp skin, laboured respirations, muscular weakness and the odour of carbon disulphide in the breath, urine and fæces. These are followed by convulsions, coma and death.

Fatal Dose and Fatal Period.—A man swallowed about half-an-ounce of carbon bisulphide and died in two hours and-a-quarter.[2] In another case a man took two ounces, but recovered in five days.[3] It has been ascertained that a concentration of about 600 parts per million produces serious symptoms if inhaled for one hour, whilst double this concentration is dangerous in thirty minutes.[4]

Treatment.—Wash out the stomach. Warmth, stimulants and artificial respiration. Remove the victim at once into the fresh air, if carbon bisulphide has been inhaled.

Post-mortem Appearances.—The odour of carbon disulphide on opening the body cavities. The blood is dark and fluid. Congestion and punctiform hæmorrhages in the stomach.

Analysis.—Carbon bisulphide may be separated from organic mixtures by distillation, and recognised by the following test:—On heating with lead acetate and caustic potash, it produces a black precipitate.

Chronic Poisoning.—This form of poisoning occurs among workmen by constant exposure to its fumes in ill-ventilated India-rubber and caoutchouc factories.

Symptoms.—Nausea; anorexia and sometimes vomiting with abdominal pain; headache; noises in the ears; tremors; muscular weakness; ataxia; tingling, numbness and burning sensation in the hands and feet (peripheral neuritis); paralysis of the extensor muscles; delirium; mania and even dementia. Amblyopia with optic atrophy occurs in some cases.

A man,[5] aged 38, worked during 1926 and 1927 in an artificial silk works, attending to churns in which wood pulp was acted upon by carbon bisulphide.

---

1. *Deut. Zeit. f. d. ges. gerichtl. Med.* 1933, *XXI*, *p.* 512; *Med.-Leg. Criminol. Review*, 1934, *Vol. II, Part I, p.* 95.
2. *Foreman, Lancet*, 1886.
3. *Davidson, Med. Times and Gaz.*, 1878.
4. *Brit. Med. Jour., June* 2, 1934, *p.* 998.
5. *F. M. R. Walshe, Proceedings of the Royal Society of Medicine, Vol. XXIII, No.* 2, *Dec.*, 1929, *p.* 89.

Structural alterations in the churns were followed by a constant slight leak of their contents so that the workers were subjected to the fumes of carbon bisulphide. He became ill after six months of this exposure. He first noticed a difficulty in concentrating his attention on his work, and could not remember at what stage of the process he had arrived. He could not overcome a feeling of listlessness and indifference. At the same time he began to suffer from attacks of diarrhœa, nausea and vomiting, with abdominal pain and loss of weight. Several severe attacks of epistaxis also occurred at this time. The prominence of his eyes developed about three months after the initial symptoms ; at first the left eye alone was affected, and this has always been more prominent than the right. Simultaneously his grasp became weak and his gait unsteady. At one time he could not hold a knife or fork with sufficient force to feed himself. He could only walk a few yards without resting and fell down on several occasions. Wasting of the hands and forearms became marked during the latter months of 1928. All these symptoms were maximal in the spring of 1929, and since that time have steadily improved.

HYDROGEN SULPHIDE (SULPHURETTED HYDROGEN), H_2S

This is a colourless, transparent gas, having a somewhat sickly sweetish taste and the odour of rotten eggs. It dissolves in water, forming an acid solution, which is, sometimes, called sulphydric acid. It burns in air with a pale blue flame, forming sulphur dioxide and water.

Hydrogen sulphide is formed during the decomposing process of organic substances containing sulphur, and may be formed as a bi-product in some of the sulphur industries. It is often found in large quantities in sewers, cesspools, privy vaults and tannery vats. It is also found in many chemical industries, such as artificial silk works, sulphur dye works, gas works, tar distillation works, etc. It is a highly poisonous gas, acting as a local irritant and affecting the central nervous system. It causes death by respiratory failure.

Hydrogen sulphide is not a cumulative poison. When inhaled, it passes into solution in the blood, where it is rapidly oxidised by the oxygen of the hæmoglobin to harmless or relatively non-toxic substances. It does not combine with oxyhæmoglobin, but combines with methæmoglobin and changes it to sulphmethæmoglobin, especially after death.

Symptoms.—When inhaled in its pure state, this gas is almost immediately fatal, causing unconsciousness at once and stoppage of respiration after a few seconds. When diluted with air, it produces irritation of the eyes, nose, throat and air-passages, followed by dizziness, headache, nausea, vomiting. abdominal pain, cyanosis, dilated pupils, cold extremeties, muscular prostration, laboured breathing, irregular pulse, tetanic convulsions, delirium, stupor, coma and death. When largely diluted, it gives rise to langour and sleepiness, and proves fatal without sensibility being restored. When very largely diluted, it may, sometimes, produce febrile symptoms somewhat resembling typhoid fever. Air containing 0.02 per cent hydrogen sulphide is sufficient to produce local irritation in man, and an atmosphere containing 0.05 per cent can be inhaled only for about thirty minutes, and then gives rise to alarming symptoms.[1] Air containing 0.07 per cent hydrogen sulphide is sufficient to cause death after the lapse of some time, and an atmosphere containing 0.18 per cent causes death immediately.[2]

Chronic Poisoning.—This occurs in workmen who are exposed to the constant inhalation of this gas for a prolonged period. According to Haggard[3] the prolonged inhalation of a concentration of the gas even as low as 0.01 per cent is sufficient to induce symptoms of chronic poisoning.

Symptoms.—These are conjuctivitis, headache, gastric disturbances, anæmia, and furunculosis. Nervous disturbances are also present.

1. *Lehman, Archiv. fur Hygiene,* 14 (1892), *p.* 135; *Autenrieth's Laboratory Manual of the Detection of Poisons, etc., Ed. VI (Translated by Warren), p.* 304.
2. *Haggard, Jour. of Industrial Hygiene, March,* 1925, *p.* 113.
3. *Jour. of Industrial Hygiene, March,* 1925, *p.* 113.

Treatment.—Fresh air, inhalation of oxygen with 5% of carbon dioxide, artificial respiration, warmth to the extremities and stimulants. The cautious use of chlorine by inhalation is recommended, but should not be tried, as it is a powerful irritant.

Post-mortem Appearances.—Putrefaction sets in much more rapidly. The offensive smell is noticed on opening the body. The blood is liquid and dark-brown in colour from the conversion of hæmoglobin into sulphmethæmoglobin, which is characterised by an absorption spectrum of two bands, consisting of one band in the red between C and D, and a fainter band between D and E. The lungs are œdematous. The other organs are dark and congested.

Tests.—1. Hydrogen sulphide is recognised by its offensive smell, which is perceptible when one part is present in 10,000 of air.

2. A piece of white filter paper moistened with lead acetate or carbonate turns black on bringing it into contact with the stomach or other organs containing the gas.

NITROGEN MONOXIDE (NITROUS OXIDE OR LAUGHING GAS), N_2O

This is a colourless gas, obtained by heating ammonium nitrate, and has a sweetish taste and a characteristic odour. It does not break and give oxygen to the body. It is a pharmacopœial preparation, called *Nitrogenii monoxidum.*

Symptoms.—When inhaled mixed with 20% of air, it produces after a few seconds a condition of hysterical excitement often accompanied by noisy laughter and gay intoxication ; hence it is known as *laughing gas.* When pushed beyond this hysterical stage, it causes anæsthesia, and is used in minor surgery, especially dentistry. It is very rarely fatal. Death occurred in two cases in 60 and 74 hours from the commencement of its inhalation.[1] A case of accidental death from self-administration of nitrous oxide as an anæsthetic is recorded, wherein a dentist was found dead in his operating chair with the mask applied to his face.[2]

When inhaled in the pure state, it at first causes the abovementioned symptoms followed immediately by unconsciousness, cyanosis, cold, clammy sweats, dyspnœic and stertorous breathing, and death from respiratory paralysis. The heart may continue to beat for some time after the stoppage of respiration.

Treatment.—This consists in the inhalation of oxygen, artificial respiration and stimulants.

Post-mortem Appearances.—There may be degenerative changes in the cortex of the brain and in the parenchyma of the basal ganglia. The blood is dark in colour.

Tests.—The gas supports combustion, but not life. It dissolves in alcohol.

SULPHUR DIOXIDE (SULPHUROUS ACID GAS OR SULPHUROUS ANHYDRIDE), SO_2

This is formed by burning sulphur or certain metallic sulphides, such as iron pyrites, in air or oxygen, and is a bi-product in the manufacture of sulphuric acid. It is met with in the gaseous emanations from volcanoes. It is present in noticeable amount in the air of towns, being derived from the combustion of the sulphur compounds present in coal.

It is a heavy, colourless gas, possessing a pungent suffocating smell of burning sulphur, and dissolving freely in water. An aqueous solution containing 5 per cent by weight of sulphur dioxide is a non-official preparation known as *Acidum sulphurosum,* dose 30 minims to 60 minims.

Sulphur dioxide is a powerful antiseptic and disinfectant, being largely used for fumigating infected rooms and furniture. It is very destructive to vegetable life, and intensely poisonous to mammalin and insect life.

1. *K. Lowenberg, R. Waggoner and T. Zainden, Ann. Surg., November,* 1936, *p.* 801 ; *Lancet, Jan.* 16, 1937, *p.* 158.
2. *Holmes and Visick, Lancet, Dec.* 4, 1920, *p.* 1167.

Symptoms.—When inhaled in the pure state, it acts as an irritant to the air-passages, causing immediately coughing and sneezing, accompanied by a feeling of suffocation, spasm of the glottis, dyspnœa, opacity of the cornea, cyanosis and convulsions. Even in dilutions of 5 parts per 10,000 of air, it produces sneezing, coughing and lachrymation. Habit produces a marked tolerance for this gas. Lehman found that air containing from 0.03 to 0.04 gramme per thousand did not affect workmen more severely than did from 0.01 to 0.02 those unaccustomed to breathing the gas.[1]

When an aqueous solution of sulphur dioxide is taken internally, it is easily oxidised to sulphuric acid, and has, therefore, a local corrosive action. It has also a remote action on the blood, causing its reduction and decomposition, as shown by the formation of hæmatin with brown colouration.

Treatment.—Removal into the fresh air and artificial respiration. Masks containing a wet sponge should be used as a prophylactic measure by workmen who are exposed to the fumes of this gas. There should be proper ventilation, and mild alkalies should be used as antidotes.

Post-mortem Appearances.—These are chiefly due to asphyxia. The blood has a strikingly dark colour, and has an acid reaction. The lungs may be œdematous.

Tests.—Starch-paper moistened with a solution of iodic acid turns blue on exposure to sulphur dioxide. One part of the gas contained in 3,000 parts of air responds to this test.

Medico-Legal Points.—The pungent, suffocating odour of the gas prevents the occurrence of accidental poisoning. Sulphur dioxide has been used for murder only once, and sulphurous acid only once for suicidal purpose.[2]

WAR GASES

The term, "gas," as used in chemical warfare, denotes a chemical compound, whether gaseous, liquid or solid, which is employed to produce poisonous or irritant effects on the enemy forces or even the civil population. The gases which are likely to be used in time of war may be described under the following heads :—

I. Vesicants or Blistering Gases.
II. Asphyxiants or Lung Irritants.
III. Lachrymators or Tear Gases.
IV. Sternutators or Nasal Irritants.
V. Paralysants.

I. **Vesicants or Blistering Gases.**—These are chiefly mustard gas (dichlorodiethyl sulphide) and lewisite (chlorovinyl-dichloroarsine). Mustard gas is also known as "Yellow Cross" or "Yperite", and was largely used in the last Great War. Mustard gas is a heavy, dark-coloured, oily liquid, having a mustard-like or garlicky odour and giving off a vapour at the ordinary temperature of the air. It is almost insoluble in water and evaporates slowly so that it persists for a long time after it is discharged. It dissolves freely in paraffin, petrol, ether, benzene, rubber, alcohol, acetone, and carbon bisulphide and readily penetrates clothing, leather, wood, bricks, etc.

Mustard gas is extremely dangerous both in the liquid and in the vaporous state. It is insidious on its onset and produces poisonous symptoms usually after the lapse of two or three hours and occasionally after twenty-four or forty-eight hours. It causes irritation of the eyes with profuse lachrymation and nasal secretion, laryngitis involving the trachea and bronchi, nausea, vomiting and gastric pain. It enters deeply into the skin through the clothes and produces intense itching, redness, vesication and ulceration. It attacks chiefly the axillæ, groins, perineum and scrotum which are

1. *Archiv. fur Hygiene*, 1893, *XVIII*, *p.* 180 ; *Peterson, Haines and Webster, Leg. Med. and Toxic., Vol. II, Ed. II, p.* 340.
2. *Kobert, Kompendium der Toxikologie Funfte Auflage*, 1912, *p.* 147 ; *Peterson, Haines and Webster, Ibid, p.* 341.

moist due to perspiration. Owing to secondary infection these ulcers are often difficult to heal. The skin of the exposed parts, such as the face, neck and hands is also affected.

In severe cases there may be œdema of the eyelids, suppuration and destruction of the conjuctiva, cornea and even the eye-ball. Death may occur from septic bronchitis, or broncho-pneumonia.

The treatment consists in prompt removal of all clothing and washing the body with soap and water. The eyes should be washed with warm water, normal saline or 2 per cent sodium bicarbonate solution. The nose should be irrigated with 5 per cent sodium bicarbonate solution. Mustard gas should be removed from the skin by applying cotton wool swabs dipped in petrol, kerosene or methylated spirit and then by rubbing into the cleansed area bleach cream prepared by mixing one part of bleaching powder to two parts of water. Tannic acid jelly or solution should also be used. The respirator which will afford protection for the eyes, nose, face and lungs should be used. The respirator is a mask with valves for the intake of air and the escape of expired air, with a container, in which is activated charcoal and a filter of celluloid through which the outer air has to pass before it enters the lungs. It will also give protection against any other type of gas. Protective clothing and boots which prevent the penetration of mustard gas should be worn.

Lewisite is a heavy, oily, dark liquid, having an odour of geraniums. It is insoluble in water, but hydrolyses rapidly ; this action is increased by heat and alkalis. It dissolves in oils, benzene and ordinary organic solvents. It is both a vesicant and an asphyxiant, and is more rapid in action than mustard gas and produces more discomfort on inhalation and more irritation on coming into contact with the skin. It must be remembered that a vesicle caused by lewisite is clearly defined, covers the whole erythematous area and is filled with a cloudy fluid containing arsenic and leucocytes. While a blister produced by mustard gas is surrounded by a zone of erythema and contains a clear yellow serum but does not contain mustard.[1]

II. **Asphyxiants or Lung Irritants.**—These are chlorine,[2] phosgene (carbonyl chloride or carbon oxychloride), diphosgene (trichloromethyl-chloroformate) and chloropicrin (nitro-chloroform). They exert their main action on the pulmonary alveoli through the upper respiratory passages.

Phosgene is a colourless gas at ordinary temperature and pressure, possessing a smell of musty hay. It is three times and a half as heavy as air and is decomposed by water into hydrochloric and carbonic acids. It is one of the most dangerous poison gases, being practically ten times more toxic than chlorine, but owing to its poor solubility its action is very slow. Hence it may, sometimes, produce poisonous symptoms a few hours after exposure and during the interval the patients may be able to carry on their work.

Diphosgene is an oily liquid having a smell of phosgene. It is heavier than phosgene, is as toxic as phosgene and is intensely lachrymatory. Both phosgene and diphosgene are known as "Green Cross."

Chloropicrin is a yellow, oily liquid, smells like chlorine and is about four times more toxic than chlorine. It is destroyed by a solution of sodium sulphite in alcohol (50 per cent).

When inhaled, these gases cause watering of the eyes, coughing, dyspnœa, feeling of pain and constriction in the chest, headache, retching and vomiting. These symptoms are followed by rapid and stertorous respirations, cyanosis and collapse. Death occurs from acute pulmonary œdema within twenty-four to forty-eight hours or later from broncho-pneumonia.

The treatment consists in absolute rest, administration of oxygen by inhalation and venesection. Codeine may be given to relieve the irritating cough and intramuscular injections of calcium gluconate may be tried to prevent the pulmonary œdema.

1. *E. M. Cowell, Brit. Med. Jour., Oct.* 14, 1939, *p.* 778.
2. Vide *p.* 536.

On post-mortem examination the lungs are found heavy and œdematous, exuding frothy, dark fluid blood on section. There are petechial hæmorrhages on the upper surface of the lungs and serous effusion in the pleural cavity.

III. **Lachrymators or Tear Gases.**—These are chiefly chloro-acetophenone (C.A.P.), ethyl-iodoacetate (K.S.K.) and bromobenzyl cyanide (B.B.C.)

Chloro-acetophenone is a colourless, crystalline solid. It is very slightly soluble in water, but dissolves in ether, alcohol or benzene and a hot aqueous solution of sodium carbonate. Ethyl-iodoacetate is a dark brown, oily liquid with a smell like that of "pear drops." Bromobenzyl cyanide is a heavy, oily, dark brown liquid, having a penetrating, bitter-sweet odour. It is very persistent.

Exposure to the vapours of any of these substances causes intense irritation of the eyes with a copious flow of tears, spasm of the eyelids and temporary blindness. When the concentration is high, the vapour causes irritation of the respiratory passages and lungs, and produces a burning sensation in the throat and discomfort in the chest. In cases where the exposure is continued for a long time, there may be nausea, vomiting, tracheitis, bronchitis, and blistering of the skin. In rare cases there may be keratitis and corneal opacities. The effects are, as a rule, transitory, incapacitating persons for some hours only and are not dangerous to life.

The treatment consists in washing the eyes with warm normal saline and removing the patient into the fresh air. The respirator is a sufficient protector of the eyes and lungs against all tear gases of any concentration.

IV. **Sternutators or Nasal Irritants.**—These are solid organic compounds of arsenic, which are dispersed by heat or detonation in the form of very fine, particulate clouds or smokes. The compounds which may be used during war are—

1. Diphenylchloroarsine (D.A.), a colourless, crystalline solid. It is slightly soluble in water, but dissolves in phosgene and chloropicrin.

2. Diphenylamine-chloroarsine or diphenylarsine-chloroarsine (D.M.), a yellow, almost odourless, crystalline solid. It is not soluble in phosgene and tarnishes metals.

3. Diphenylcyanarsine (D.C.), a colourless, odourless, crystalline solid.

The vapours of these substances, when inhaled, cause intense pain and irritation in the nose and sinuses with excessive sneezing, malaise, headache, painful gums, salivation, nausea, vomiting, pain and tightness in the chest and temporary prostration. The effects are temporary, lasting for an hour or two, but are quite effective in destroying the morale of the enemy's troops.

Water and food contaminated by these substances may give rise to symptoms of arsenic poisoning.

The treatment is fresh air. The nose should be irrigated with a 5 per cent solution of sodium carbonate. Gargles of the same solution may be used if there is irritation of the throat. A few whiffs of chloroform inhalation may be given if there is severe pain in the sinuses.

V. **Paralysants.**—These are hydrocyanic acid and sulphuretted hydrogen,[1] but they are not very useful in chemical warfare, as it is difficult to obtain them in their lethal concentrations during the time of war.

1. Vide *pp.* 797 *and* 813.

CHAPTER XXXV

PERIPHERAL (NEURAL) POISONS

CONIUM MACULATUM (COMMON OR SPOTTED HEMLOCK)

This plant belongs to N. O. *Umbelliferæ*, and grows generally in hedgerows and in waste places in Europe, America and the temperate regions of Asia. It has a peculiar mousy odour which is intensified by rubbing the leaves or other part of the plant in a mortar with a little solution of caustic potash or soda. The plant owes its poisonous properties to the two liquid alkaloids, *Coniine* and *Methyl-coniine*. It also contains a crystalline alkaloid, *Conhydrine*, allied to methyl-coniine, and *Conic acid*.

Coniine, $C_8H_{17}N$.—This exists in all parts of the plant, but is extracted chiefly from the fruit and leaves by distillation with soda. It is a colourless, volatile oil, but changes to brown on exposure to air. It has an acrid, bitter taste and a penetrating, mousy odour. It is slightly soluble in water but freely in alcohol, ether and chloroform. Its salts are stable and crystalline, and are soluble in water and alcohol. It paralyses the motor nerve endings and subsequently the motor centres.

Methyl-Coniine, $C_9H_{19}N$.—This is a colourless, volatile, oily, liquid alkaloid, possessing an odour similar to that of coniine.

Symptoms.—Burning sensation in the mouth, constriction of the throat, profuse salivation, dizziness, headache, staggering gait, weakness or paralysis of the extremities, great prostration, dilated pupils, ptosis, convulsions and coma. Death occurs from paralysis of the respiratory muscles. The intellect remains clear to the last. Nausea and vomiting may occur during the course of the symptoms.

Fatal Dose.—Uncertain. Half to one grain of coniine is likely to cause serious symptoms, and 2 grains will probably prove fatal. A dose of 10 or 15 drops of coniine (0.42 to 0.63 gramme) killed a woman in a few minutes.[1] One ounce of Succus Conii (a non-official preparation, dose 1 to 2 drachms), has caused death.[2]

Fatal Period.—Death has occurred in a few minutes. The usual fatal period is one to three hours, though death occurred in three hours and a quarter in one case,[3] in seven hours in another case[4] and in fifty-two hours in a third case.[5]

Treatment.—Give emetics or wash out the stomach after giving tannic acid or vegetable astringents. Administer strychnine hypodermically, and

1. *Husemann, Die Pflanzenstoffe, p.* 269 ; *Taylor. Princ. and Pract. of Med. Juris., Vol. II, Ed. IX, p.* 789.
2. *A. Robertson, Med. Juris. and Toxic., Ed. V, p.* 396.
3. *Bennet, Ed. Med. and Sur. Jour., July,* 1845, *p.* 169.
4. *Pepper, Med.-Leg. Jour.,* 1885-86, *III, p.* 179.
5. *Armstrong, Trans. Med. Soc., New Jersey,* 1880, *CXIV, p.* 249.

then give general stimulants, such as strong coffee, alcohol, etc. Perform artificial respiration, if necessary.

Post-mortem Appearances.—Not characteristic. The mucous membrane of the stomach may be reddened and ecchymosed. The other organs are congested with venous engorgement and dark fluid blood. The lungs may, sometimes, be œdematous.

Tests.—1. Coniine is recognised by its mousy odour. A drop of coniine dissolved in just sufficient cold water has a clear appearance, but becomes turbid on heating and again becomes clear on cooling.

2. Alloxan produces a purple-red colour, which forms white needle-shaped crystals on standing. These crystals, touched with caustic potash, turn purple and give off a mousy odour.

3. Warmed with sulphuric acid and potassium bichromate, coniine produces butyric acid, which is known by its peculiar odour.

Medico-Legal Points.—Poisoning by conium maculatum is very rare in India. In Enrope and America accidental poisoning has occurred from the leaves having been made into salad in mistake for parsnip, or from the root having been used for parsley, fennel and asparagus. Children have also been poisoned from using whistles made of its stem.[1] The seeds have been accidentally mixed with caraway, anise and dill seeds. The non-official preparations have also caused accidental and homicidal poisoning.

The ancient Greeks were familiar with the toxic properties of conium maculatum, and used its juice or an infusion of the leaves as a state-poison. Socrates was put to death by drinking the infusion.

Coniine is rapidly eliminated in the urine so that its action passes off very soon, if death does not occur.

CURARE (CURARA, WOORARA, URARI OR WOORALI)

This is a blackish-brown resinous extract produced from various species of strychnos and other plants of the N. O. *Loganiaceœ*. It has a bitter taste and is nearly soluble in water. The non-official dose of curare is 1|20 to ½ grain subcutaneously. A non-official preparation from curare is *Injectio curarœ hypodermica,* dose 1 to 6 minims subcutaneously.

Curare is used as an arrow poison by South American Indians. It contains an active principle, curarine or curarina, which is a most powerful poison and occurs as a yellowish-brown powder or in deliquescent prisms, with an intensely bitter taste. It is soluble in water and alcohol.

Symptoms.—Curare is extremely poisonous, and exerts its toxic properties, when injected into the blood stream by means of a hypodermic syringe or through a wound. When swallowed, it is supposed to be almost inert like the snake-venom, provided there is no abraded surface in the mouth or throat.

Curare acts on the motor nerve endings causing paralysis of the voluntary muscles, and causes death by paralysis of respiration. It was formerly used in physiological laboratories for the purpose of abolishing voluntary movements of animals required for experiments but its use is now prohibited by the Vivisection Act. It has been given hypodermically in hydrophobia, tetanus and strychnine poisoning.

1. *Lancet, Sep.* 20, 1851, *Vol. II, p.* 276.

Fatal Dose.—Three-quarters to a grain of curare would probably prove fatal.[1]

Treatment.—This consists in the use of stimulants, such as strychnine hypodermically, and artificial respiration, if necessary. If the poison is applied to a wound, a ligature should be applied at once at its proximal end, and the poison should be sucked from the wound provided there are no abrasions about the mouth and lips or the poison should be neutralized by washing the wound with a solution of potassium permanganate. Substance 36 (methyl-phenyl-carbamic ester of 3-oxyphenyl-trimethyl-ammonium-methyl sulphate) which is an analogue of eserine and closely related to Prostigmin is considered to possess antagonistic properties to curare.[2]

Tests.—1. Sulphuric acid and potassium bichromate produce first a blue colour, then violet and lastly cherry-red. Strychnine undergoes the same reaction, but takes a longer time.

2. Sulphuric acid imparts a red colour to curarine.

Medico-Legal Points.—It is reported that in 1917 a plot was laid to poison Mr. Lloyd George, the Prime Minister, with curarine.

Curare is rapidly excreted unchanged by the kidneys.

1. *Taylor, Princ. and Pract. of Med. Juris., Vol. II, Ed. IX, p.* 795.
2. *Grace Briscoe, Lancet, March* 13, 1937, *p.* 621.

APPENDIX I

ORDERS PERTAINING TO MEDICO-LEGAL WORK

(FROM MANUAL OF GOVERNMENT ORDERS, U. P., VOL. I, DEPT. VI)

DYING DECLARATIONS

(1) Dying declarations[1] should, if possible, be written by the persons making them. Such statements should be signed or marked by the declarant and attested by respectable witnesses.

(2) Where a dying declaration is recorded by a police or medical officer, it should be recorded in full detail in the vernacular in the words of the declarant in the form of question and answer, and in the presence of respectable witnesses. It should then be read over to the declarant, who should affix his signature or mark to it. The accused or his pleader, if present, should be allowed to put questions to the declarant. The declaration when concluded should be signed by the police or medical officer recording it, who should also obtain the signatures of respectable witnesses. It should then be forwarded in a sealed envelope direct to the Magistrate who would ordinarily hold inquiry into the case. If it can be avoided, no police officer who is engaged in the investigation into the case should be present, when the dying declaration is recorded.

Procedure to be observed in recording a Dying Declaration.—

(*a*) **For Police.**—The officer investigating a case in which a person has been seriously injured should, if there is any probability of the person dying before he can reach a dispensary where his dying declaration can be recorded, at once himself record the declaration in the presence of two respectable witnesses, obtaining the signature or the mark of the declarant at the foot of the declaration.

The Prosecuting Inspector, on being warned that a dying declaration is to be taken should at once go to the hospital with the police papers of the case, and, if possible, arrange for the attendance of the accused and his pleader.

(*b*) **For Magistrates.**—The District Magistrate, or Senior Magistrate present in the station, on receiving notice that a dying declaration is necessary, should at once himself proceed to take it, or depute some stipendiary Magistrate, if possible above the rank of Tahsildar to take it. He should at the same time cause the Prosecuting Inspector to attend with the police papers of the case.

Every Magistrate on receiving an order or requisition to take a dying declaration from the District Magistrate or medical authority, must at once proceed to the hospital or dispensary to record the dying declaration.

(*c*) **For the Medical Authorities.**—(1) The Civil Surgeon or the Assistant Surgeon in charge of the sadar hospital, should at once call on the District Magistrate or the Senior Magistrate present at the station, to arrange for the record of the dying declaration of such persons as are likely to die and are in a fit state to make a statement.

If, in the opinion of the Civil Surgeon, or Assistant Surgeon, there is no time to call on the District Magistrate or the Senior Magistrate present at the station, the nearest Magistrate may be sent for to take the dying declaration.

If, in the opinion of the Civil Surgeon, or Assistant Surgeon, there is no time to call any Magistrate, he may himself record the declaration.

---

1. *Chapter XXVI, paras* 854 *to* 857.

In cases where there is no time to call on the District Magistrate or Senior Magistrate present at the sadar, he should be informed of the action taken.

(2) The Sub-Assistant Surgeon of an outlying dispensary should at once call on the Tahsildar or in his absence, the nearest Honorary Magistrate, to record the dying declarations of such persons as are likely to die and are in a fit state to make a statement.

If there is, in his opinion, no time to call on the Tahsildar or an Honorary Magistrate, he may record the dying declaration himself.

FROM POLICE REGULATIONS

SECTION 118-A.—Where the identity of a corpse or of a person killed by an accident or who has met with death under suspicious circumstances or in the commission of crime, has not been fully ascertained by ordinary enquiries, the finger prints should be taken on search slip forms, and sent to the Bureau for search. Ordinarily there is not much difficulty in taking impressions from the fingers of a corpse, but it sometimes happens that the skin of the fingers is so contracted and wrinkled that decipherable prints cannot be obtained. In such cases the medical officer holding the post-mortem should be asked to remove the skin from the fingers. He should place each piece in a separate sealed envelope marking on the outside the finger to which it belongs. These envelopes should then be sent to the Finger Print Bureau at Allahabad for opinion.

The finger prints of unidentified bodies should invariably be taken under the supervision of an officer not below the rank of a Sub-Inspector. Finger prints of all digits must be taken, and the supervising officer shall certify by his signature on the search slip that impressions have been correctly taken in his presence. The supervising officer will further note in the remarks column of the scarch slip the condition of the body, whether in an advanced stage of decomposition or otherwise.

SECTION 121. (Cf. 858 Para, Manual of Government Orders, U. P., Vol. I).—The following instructions prescribe the procedure to be observed by the police when sending in a dead body to the civil medical officer for post-mortem examination, and for the conduct of such examination by that officer :—

1. The body shall be laid in the shell in the state in which it has been found.

2. The body sent in for examination shall be accompanied by a police constable and a chaukidar. If the *thana* is over twenty miles distant from headquarters, the constable and chaukidar may be relieved at one or more intermediate stations, but the number of reliefs should be kept as low as possible.

3. The names of the police constable and of the chaukidar and of the relieving constables and chaukidars, if any, shall be always entered in police form No. 13.

4. They shall be instructed by the *Thanedar* sending in the body to make it over, on arrival at the police headquarters, to the civil medical officer after despatch of the usual requisition, but will remain in charge of the body until the medical officer has completed the examination, and will arrange for the disposal of the remains in the absence of relatives of the deceased.

5. Before commencing the examination, the medical officer shall ask the police constable and the chaukidar whether the body to be examined is the body which they accompanied from the police station, and their replies shall form part of the civil medical officer's declaration.

6. After the formal identification by the police constable and the chaukidar, the medical officer shall compare the body with the *hulia* or descriptive roll sent from the police station, and he shall certify that the body about to be examined agrees with the descriptive roll with it.

7. The greatest care and precision is enjoined on all police officers in describing the body ; any marks or natural conditions by which it may be readily identified should be noted ; and this descriptive roll should contain particulars for identification distinct from any injuries that may be apparent.

8. The officer in charge of the station from which the body is despatched, shall not send both copies of the descriptive roll with the police constable, who accompanies

the body, but shall send only one copy of the roll by the constable and forward the other by post to the officer in charge of the police headquarters.

9. The medical officer shall be furnished with a detailed translation on the appearance and the situation of the body when it was first discovered, and on the cause of death, as far as ascertainable at the time.

Note.—Superintendents of police are authorized to call on civil surgeons to make post-mortem examinations.

10. The post-mortem examination in cases in which an Indian has been killed by a soldier (British) shall be conducted by the civil surgeon himself, and the police shall give that officer the information necessary to show him that the case is one in which his personal conduct of the post-mortem is required.

SECTION 121-A.—1. All substances or articles connected with the commission of an offence and required to be put in as evidence at the trial should be sent under a sealed cover with the contents noted outside, by the investigating officer under a *chalan* or invoice to the Prosecuting Inspector. The Prosecuting Inspector shall sign the *chalan* in token of having received the parcel and shall enter it in his register. The *chalan* should show the name of the constable who brought the sealed cover.

2. If the article is one in which no medical or Chemical Examiner's examination is required the Prosecuting Inspector shall retain the article until it is wanted for production in Court.

3. If the article is one of which examination by the Civil Surgeon or Chemical Examiner appears to be necessary, the Prosecuting Inspector shall send it on to the Civil Surgeon with a letter requesting him to examine it. The Prosecuting Inspector shall note in his register the agency by which the parcel was sent, and shall obtain the Civil Surgeon's receipt for it.

4. The Civil Surgeon on receiving the article shall open and inspect it. If he finds that he can examine it, he can do so, and return the article unless it be offensive matter, such as stomach washing, vomited matter, viscera, etc., with the report of his examination to the Prosecuting Inspector who shall then produce the report, and where it has been returned, the article, when required by the Court. If the article is of such a nature that examination by the Chemical Examiner appears desirable, the Civil Surgeon shall inform the Court to that effect, and retain the article pending orders from the Court.

5. On the receipt of orders from the Court requiring him to send the article to the Chemical Examiner, the Civil Surgeon shall proceed as laid down in the existing rules on the subject (paragraphs 868, 886, Manual of Government Orders).

6. If the Court intimates that it does not consider an examination by the Chemical Examiner necessary, the Civil Surgeon shall return the article to the Court for disposal, obtaining a receipt for it from the Court.

The Civil Surgeon shall be the custodian of substances of an offensive nature as long as the medical analysis is under consideration.. Once the analysis has been made and the report and substances presented in Court, the police should take charge of the exhibits, the *malkhana* being the repository for such matter.

In accordance with G. O. No. 585 VI—82C, dated 3rd March 1904, such substances should be destroyed by the Civil Surgeon without orders from the Court after being detained for six months but the District Magistrate's assent must first be obtained.

SECTION 123.—The Medical Officer should give an abstract of his report to the constable accompanying the injured person or dead body for communication to the investigating officer. The constable should be instructed by the investigating officer to ask for such an abstract.

SECTION 125.—No person may be sent by the police for medical examination against his or her will. No person should be sent for examination to an employee of the Dufferin Fund.

CIRCULAR No. 24 of 1903 OF THE INSPECTOR-GENERAL, CIVIL HOSPITALS, UNITED PROVINCES

1. The form of certificate in use in these Provinces for the reports of Medical Officers to the police in cases of "hurt" requires that an opinion should be expressed as to whether the injury is "*simple*" or "*grievous*" and definitions of these terms, as explained in the Indian Penal Code, are printed on the form for information.

2. It has been found in practice that the police usually press for an immediate opinion as to whether an injury is "*simple*" or "*grievous,*" to enable them to decide whether to take cognizance of an offence or not, with the result, in the case of Hospital Assistants particularly, that wrong opinions are often given, and much subsequent trouble caused both to the police and to the medical subordinates. The latter are frequently suspected, and sometimes accused, of having been bribed by interested parties if their sworn evidence in Court differs from that given in their original certificate.

3. It goes without saying, that in some cases it is impossible for the most experienced surgeon to give an opinion *at once* as to whether an injury is "*simple*" or "*grievous,*" as defined by the Indian Penal Code; while in many, probably in most cases, it is perfectly possible to do so.

4. Medical Officers of the subordinate grades should understand that what is wanted from them is a description of the injury presented to them, an opinion as to how it was caused, and what will be the *probable* immediate and remote result. If they are unable to diagnose the actual injury, or to say how it was caused, or to give an immediate and reliable prognosis as to results, they are perfectly justified in reporting to that effect, and are hereby authorized to do so. No pressure from subordinate police officials should induce them to make a report founded on imperfect knowledge or insufficient observation.

5. If these instructions are borne in mind, there will be fewer cases of Hospital Assistants adhering in Court to an obviously incorrect original opinion, or being suspected or accused of dishonesty, because they honestly changed their views as to the nature of a case between the time of their original examination and that of giving evidence in Court.

CIRCULAR No. 17 of 1920 OF THE I. G. C. H., U. P. (G. O. No. 1409|VI-898, DATED THE 6th MARCH 1920)

All classes of Medical Officers holding the appointment of a Civil Surgeon are entitled to a fee of Rs. 16 and Rs. 10 for conducting post-mortem and medico-legal examinations respectively otherwise than in the course of their ordinary duties. When such examinations are conducted by medical officers other than Civil Surgeons, otherwise than in the course of their ordinary duties, a fee of Rs. 4 is admissible.

THE REFORMATORY SCHOOLS ACT, 1897 (ACT NO. VIII OF 1897)

SECTION 4.—A youthful offender is any boy who has been convicted of any offence punishable with transportation or imprisonment and who, at the time of such conviction, was under the age of fifteen years (sixteen years in the Presidency of Bombay, vide Bombay Act No. XIII of 1924).

SECTION 8.—Whenever any youthful offender is sentenced to transportation or imprisonment, and is, in the judgment of the Court by which he is sentenced, a proper person to be an inmate of a Reformatory School, the Court may, subject to any rules made by the Local Government, direct that, instead of undergoing his sentence, he shall be sent to such a school, and be there detained for a period which shall not be less than three and more than seven years.

NOTIFICATION NO. 611|VI-4003 REPUBLISHING NOTIFICATION OF GOVERNMENT OF INDIA, HOME DEPARTMENT JAILS, NO. 173, DATED 14|3|1889.—RULES FOR REGULATING THE PERIODS FOR WHICH YOUTHFUL OFFENDERS MAY BE SENT TO A REFORMATORY SCHOOL.—"No boy shall be sent to a Reformatory School, if under ten years of age, for a less period than seven years; if over ten years for a less period than five years, unless he shall sooner attain the age of eighteen years."

APPENDIX II

SPECIAL RULES OF EVIDENCE

(FROM OUDH CRIMINAL DIGEST)

MEDICAL EVIDENCE IN CRIMINAL CASES

105. The following instructions in regard to obtaining and taking medical evidence in criminal cases are to be followed (Sections 509 and 510 of Cr. P. Code) :—

(1) When a case arises requiring medical opinion, the subject shall be forwarded to the proper medical officer, with such a general description of what is known of the case that the attention of the medical officer may be turned in the right direction. This description may be in English or Vernacular, as the case may be.

(2) The Medical Officer shall reply by letter or memorandum on the back of the reference, or at any rate attach his reply to the reference, in order that there may be no doubt of the case to which his remarks apply.

(3) Whenever the cause of death, or the nature of the injury, is the subject of inquiry, the Medical Officer shall be summoned as a witness and examined by the Magistrate.

(4) The Magistrate shall look into the case, and make himself acquainted with its particular features, before the Medical Officer is examined, in order that the proper questions may be asked.

(5) The deposition of the Medical Officer shall be recorded on a separate piece of paper. The Magistrate shall set out accurately the official designation of the witness, and before the witness leaves the Court his deposition shall be translated and read over to the accused, who shall be allowed to cross-examine, and the deposition shall show that he has had the opportunity of doing so. The translation shall form part of the record.

(6) The report (if any) of the Chemical Examiner or Assistant Chemical Examiner shall be put on the record.

(7) The reply of the Medical Officer shall be put on the record, and may be used to refresh the memory of the Medical Officer at the time of giving his deposition, and so assist the Magistrate or Judge in framing his questions. It is not sufficient to read it over to the Medical Officer and swear him to the truth of it, his deposition must be fully recorded in the presence of the accused.

(8) If, in any particular case, the evidence of a medical witness is not to be had, the details, such as fact of death, symptoms, appearances, wounds, must be made out as correctly as possible from the evidence of non-medical eye-witnesses. The Court cannot assume any such facts from mere reports not admissible as evidence, and it is of no use proceeding in the trial until the *corpus delicti* is made out. Police officers can always be put into the witness-box to bear testimony to what they saw.

(9) In cases committed to the Court of Session, the medical witness shall not be bound over by the Magistrate to appear before the Court of Session, but the Court, if it think fit, may on its own motion or on the application of either side, summon and examine such witness.

107. **Expenses payable to Witnesses.**—The following rules have been made under Section 544, Criminal Procedure Code, by the Local Government with the

previous sanction of the Governor-General in Council, for the payment of the expenses of complainants and witnesses attending before any Criminal Court for the purpose of an inquiry or a trial provided that no such payment shall be made from public funds to any witness in cases where under the provisions of any law in force the reasonable expenses of such witness have by order been deposited in Court as a condition precedent to the issue of process to compel his attendance[1] :—

1. (*a*) For the ordinary labouring class of Indians two annas per diem.

(*b*) For Indians of higher rank in life, four annas per diem.

(*c*) For Europeans and Eurasians and Indians of superior rank, a diet allowance according to circumstances up to about of Rs. 3 per diem.

2. Diet money shall be paid for the days of actual detention as well as for the time occupied in the journeys to and from the Court. The number of days which should be allowed for the journey to and fro will be determined by the officer ordering payment in each case.

3. Travelling expenses shall be given only when the journey could not, with reasonable care and expedition, have been performed on foot, or in the case of persons whose age, position and habits of life render it impossible for them to walk. In such cases, in addition to diet allowance, travelling allowance shall be given at the following rates :—

(*a*) When journey is by rapid dak by road the actual expenses incurred up to a maximum limit of four annas a mile, or in the case of Europeans, Eurasians and Indians of superior rank up to eight annas a mile. In towns where licensed hackney carriages ply for hire the actual cost of hiring a vehicle suited to the rank of a witness may be allowed if, in the opinion of the presiding officer of the Court, the use of such a vehicle was necessary.

(*b*) Where the journey is wholly or partially by rail—

(*i*) For Indians generally, railway fare for the lowest class ;

(*ii*) For Europeans, Eurasians and Indians of higher rank, intermediate or second class railway fare according to circumstances ; but the Court may, in its discretion allow first class railway fare when the persons concerned, from their social position, would ordinarily travel by that class.

4. Notwithstanding anything contained in the foregoing rules—

(*a*) Government servants (not the police officers on duty as witnesses, patwaries, or rural jamadars or chaukidars) shall receive nothing beyond actual expenses.

(*b*) Police officers on duty as witnesses shall not receive from the Court any allowances or expenses whatsoever. The Court shall, upon application, give such officers a certificate of attendance, and they will receive departmentally any travelling allowance to which they may be entitled under the provisions of article 1133 read with article 1038 of Civil Service Regulations.

(*c*) Witnesses following any profession, such as medicine or law, shall receive a special allowance according to circumstances and custom.

N. B. The following are the rules regarding the fee which a Civil Surgeon can claim for giving evidence in a Magistrate's Court as an expert witness (vide G. O. No. 627|VI-13, dated the 25th February 1913 ; I. G. C. H. Circular No. 30 of 1913) :—

(*a*) In summons cases under section 244 (3) of the Criminal Procedure Code and for the defence in warrant cases under section 257 (2) of the Criminal Procedure Code, the Court may require his reasonable expenses to be deposited before summoning a witness. It appears to be customary for the Courts to require the usual fee to be deposited before summoning the Civil Surgeon as an expert witness, and this custom should be followed unless special reasons to the contrary exist.

---

1. *Manual of Government Orders, U. P., Vol. I, VI, Judicial (criminal) Dept., Ch. XXXIX, paras* 900, 901, 902, 903.

(*b*) For the prosecution in warrant cases the Court has no power to demand from the complainant payment of a witness's expenses in advance, nor under the rules issued by Government with G. O. No. 896|VI-300, dated the 18th April 1911, can such a witness claim from the Court anything more than his actual travelling expenses. In these cases, therefore, the Civil Surgeon cannot claim his fee as an expert. Cases of this kind, however, in which the Civil Surgeon's evidence is required are not numerous, and it is not necessary to make any changes in the existing rules on this account.

These orders have been extended by the Local Government to cover the cases of all Government medical officers summoned as expert witnesses in summons cases under section 244 (3) of the Criminal Procedure Code and for the defence in warrant cases under section 257 (2) of the Criminal Procedure Code (vide G. O. No. 1298 VI-13, dated the 24th March 1917 ; I. G. C. H. Circular No. 21, dated the 3rd April 1917).

5. (*a*) Government servants summoned to give evidence in their official capacity shall, except in the cases specified hereafter, be given a certificate of attendance. Any fees or expenses deposited in such cases for the travelling and subsistence allowance of Government servants must, therefore, be credited to Government, unless the case falls under one or the other exceptions.

The exceptions are—

(1) Cases in which Government servants have to give evidence at a Court situate not more than five miles from their headquarters. Rule 26, Chapter III of the Travelling Allowance Rules (Financial Hand-book, Volume II), debars the grant of travelling allowance in such cases, and as the certificate system would operate somewhat harshly, it is left to the discretion of the Courts to pay the actual travelling expenses incurred in cases where they consider this necessary.

(2) Cases in which the salary of the official summoned (such officer not being a police constable) does not exceed Rs. 10 per mensem. These officials shall receive their expenses from the Court.

(*b*) The term "reasonable expenses" in Section 244 (3) of the Code of Criminal Procedure must be held to mean the travelling and halting allowance to which such Government servants are entitled under the Travelling Allowance Rules.

(*c*) When Government servants are summoned in other circumstances, the provisions of Rule 59 (2), Chapter IV of the Travelling Allowance Rules, shall be observed (Financial Hand-book, Volume III).[1]

1. *The Oudh Criminal Rules, 1928, Chapter V, p.* 13.

APPENDIX III

QUESTIONS TO BE PUT TO MEDICAL WITNESSES

(FROM OUDH CRIMINAL DIGEST)

No. I

QUESTIONS THAT MAY BE PUT TO A MEDICAL WITNESS IN A CASE OF SUSPECTED POISONING AFTER POST-MORTEM EXAMINATION OF THE BODY

1. Did you examine the body of , a late resident of and, if so, what did you observe ?

2. What do you consider to have been the cause of death ? State your reasons.

3. Did you find any external marks of violence on the body ? If so, describe them.

4. Did you observe any unusual appearances on further examination of the body ? If so, describe them.

5. To what do you attribute these appearances ; to disease, poison or other cause ?

6. If to poison, then to what class of poisons ?

7. Have you formed an opinion as to what particular poison was used ?

8. Did you find any morbid appearances in the body besides those which are usually found in cases of poisoning by.....? If so, describe them.

9. Do you know of any disease, in which the post-mortem appearances resemble those which you observed in this case ?

10. In what respect do the post-mortem appearances of that disease differ from those which you observed in the present case ?

11. What are the symptoms of that disease in the living ?

12. Are there any post-mortem appearances usual in case of poisoning by .....but, which you did not discover in this instance ?

13. Might not the appearances you mention have been the result of spontaneous changes in the stomach after death ?

14. Was the state of the stomach and bowels compatible or incompatible with vomiting and purging ?

15. What are the usual symptoms of poisoning by.....?

16. What is the usual interval between the time of taking the poison and the commencement of the symptoms ?

17. In what time does.....generally prove fatal ?

18. Did you send the contents of the stomach and bowels (or other matters) to the Chemical Examiner ?

19. Were the contents of the stomach (or other matters) sealed up in your presence immediately on removal from the body ?

20. Describe the vessel in which they were sealed up, and what impression did the seal bear ?

21. Have you received a reply from the Chemical Examiner ? If so, is the report now produced that which you received ?

22. (If a female adult) what was the state of the uterus ?

No. II

QUESTIONS THAT MAY BE PUT TO A NON-PROFESSIONAL WITNESS IN A CASE OF SUSPECTED POISONING

1. Did you know , a late resident of ? If so, did you see him during his last illness and previously ?

2. What are the symptoms from which he suffered ?

3. Was he in good health previous to the attack ?

4. Did the symptoms appear suddenly ?

5. What was the interval between the last time of eating or drinking and the commencement of the symptoms ?

IF DEATH OCCURRED

6. What was the interval between the commencement of the symptoms and death ?

7. What did the last meal consist of ?

8. Did any one partake of this meal with.....?

9. Were any of them affected in the same way ?

10. Had he ever suffered from a similar attack before ?

If any of the following symptoms have been omitted in answer to question 2, special questions (11-14) may be asked regarding them as follows :—11. Did vomiting occur ? 12. Was there any purging ?

13. Was there any pain in the stomach ? 14. Was—very thirsty ?

15. Did he become faint ?

16. Did he complain of headache or giddiness ?

17. Did he appear to have lost the use of his limbs ?

18. Did he sleep heavily ?

19. Had he any delirium ?

20. Did convulsions occur ?

21. Did he complain of any peculiar taste in the mouth ?

22. Did he notice any peculiar taste in his food or water ?

23. Was he sensible in the intervals between the convulsions ? (This is with reference to Nux Vomica).

24. Did he complain of burning or tingling in the mouth and throat, or of numbness and tingling in the limbs ? (Aconite).

No. III

QUESTIONS WHICH MAY BE PUT TO A MEDICAL WITNESS IN A CASE OF SUPPOSED DEATH BY WOUNDS OR BLOWS AFTER POST-MORTEM EXAMINATION OF THE BODY

1. Did you examine the body of , a late resident of , and if so, what did you observe ?

2. What do you consider to have been the cause of death? State your reasons.

3. Did you find any external marks of violence on the body? If so, describe them.

4. Are you of opinion that these injuries were inflicted before or after death? Give your reasons.

5. Did you examine the body internally? Describe any unnatural appearance which you observed.

6. You say that in your opinion — was the cause of death; in what immediate way did it prove fatal?

7. Did you find any appearance of disease in the body?

8. If so, do you consider that, if the deceased had been free from this disease, the injuries would still have proved fatal?

9. Do you believe that the fact of his suffering from this disease lessened his chance of recovery from the injuries sustained?

10. Are these injuries taken collectively, or any one of them ordinarily and directly dangerous to life?

11. Have they been caused by manual force or with a weapon?

12. Did you find any foreign matters in the wound?

13. By what sort of weapon has the wound been inflicted?

14. Could the injuries have been inflicted by the weapon now before you (No. — in the police charge sheet)?

15. Could the deceased have walked (so far) or spoken, etc., after the receipt of such an injury?

16. Have you, chemically or otherwise, examined the stains (or the weapon, clothes, etc.,) now before you (No.————in the police charge sheet)?

17. Do you believe the stains to be those of blood?

18. What time do you think elapsed between the receipt of the injuries and death?

19. What was the direction of the wound, and can you form an opinion as to the position of the person inflicting such a wound with respect to the person receiving it?

20. Is it possible for such a wound to have been inflicted by any one on his own person? Give your reasons.

21. (In gun shot wounds), give precise direction of the wound.

22. Did the appearance of the wound indicate that the gun had been discharged close to the body or at some distance from it?

23. Did you find any slug, bullet, wadding, etc., in the wound or had — made its exit?

24. Did you think it possible that you could have mistaken the aperture of entrance for that of exit?

No. IV

QUESTIONS THAT MAY BE PUT TO A MEDICAL WITNESS IN A CASE OF SUPPOSED INFANTICIDE AFTER POST-MORTEM EXAMINATION OF THE BODY

1. Did you examine the body of a (male or female) child sent to you by the District Superintendent of Police on the————of————19————? And, if so, what did you observe?

2. Can you state whether the child was completely born alive, or born dead? State the reasons for your opinion.

3. What do you consider to have been the cause of death? Give your reasons.

4. What do you believe to have been the uterine age of the child? State your reasons.

5. What do you believe to have been the extra-uterine age of the child? Give reasons.

6. Did you find any marks of violence or other unusual appearances externally? If so, describe them accurately.

7. Did you find any morbid or unusual appearances on examination of the body internally? If so, describe them accurately.

8. Do you believe the injuries you observed to have been inflicted before or after death? Give reasons.

9. Can you state how they were inflicted? Give reasons.

10. Do you consider that they were accidental or not? Give reasons.

11. Had the infant respired fully, partially, or not at all?

12. Did you examine the person of the alleged mother of the infant? If so, have you reason to suppose that she was recently delivered of a child? Can you state approximately the date of her delivery? Give reasons.

No. V

QUESTIONS THAT MAY BE PUT TO A MEDICAL WITNESS IN A CASE OF SUPPOSED DEATH BY HANGING OR STRANGULATION

1. Did you examine the body of , a late resident of , and if so, what did you observe?

2. What do you consider to have been the cause of death? State reasons for your opinion.

3. Did you observe any external marks of violence upon the body?

4. Did you observe any unnatural appearance on examination of the body internally?

5. Was there any rope or other such article round the neck when you saw the body?

6. Can you state whether the mark or marks you observed, were caused before or after death?

7. By what sort of articles do you consider the deceased to have been hanged (or strangled)?

8. Could the marks you observed have been caused by the rope or other article now before you (No..... of the police charge sheet)?

9. Do you think that this rope could have supported the weight of the body?

10. If strangulation, would great violence be necessary to produce the injuries you describe?

11. What, as far as you can ascertain, were the general characteristics of his previous disposition?

12. Does he appear to have had any previous attacks of insanity?

No. VI

QUESTIONS THAT MAY BE PUT TO A MEDICAL WITNESS IN A CASE OF SUPPOSED DEATH BY DROWNING AFTER POST-MORTEM EXAMINATION OF THE BODY

1. Did you examine the body of , a late resident of , and if so, what did you observe?

2. What do you consider to have been the cause of death? State your reasons.

3. Were there any external marks of violence upon the body? If so, describe them.

4. Describe any unnatural appearances which you observed in further examination of the body.

5. Did you find any foreign matter, such as weeds, straw, etc., in the hair, or clenched in the hands of the deceased or in the air-passages or attached to any other part of the body ?

6. Did you find any water in the stomach ?

No. VII

QUESTIONS THAT MAY BE PUT TO A MEDICAL WITNESS IN A CASE OF ALLEGED RAPE

1. Did you examine the person of Musammat — ? If so, how many days after the alleged rape did you make the examination and what did you observe ?

2. Did you observe any marks of violence about the vulva or adjacent parts ?

3. Are these injuries such as might have been occasioned by the commission of rape ?

4. Was the hymen ruptured ?

N.B.—This question is only to be asked in the case of the rape of a girl of tender years.

5. Did you observe any further marks of violence upon the person of the woman ?

6. Has she passed the age of puberty ?

7. Can you state approximately what her age is ?

8. Did you find her to be a strong, healthy woman, or so weakly as to be unable to resist an attempt at rape ?

9. Did you examine the person of the accused ?

10. Did you observe any marks of violence upon his body ?

11. Was he suffering from any venereal disease ?

12. Did you find the woman to be suffering from a similar or other venereal disease ?

13. Had a sufficient time elapsed when you examined the person of the woman, for venreal disease to have made its appearance, in case of her having been infected ?

14. Can you state approximately how long the accused had been suffering from this complaint ?

15. Can you state approximately how long the woman had been suffering from this (venereal) complaint ?

16. Have you examined the stained articles forwarded to you and now in Court (No.....of police charge sheet) ?

17. What is the result of your examination ?

18. Do you believe that rape has been committed or not ? State your reasons.

No. VIII

QUESTIONS THAT MAY BE PUT TO A MEDICAL WITNESS IN A CASE OF INSANITY

1. Have you examined — ?

2. Have you done so on several different occasions, so as to preclude the possibility of your examination having been made during lucid intervals of insanity ?

3. Do you consider him to be capable of managing himself and his personal affairs ?

4. Do you consider him to be of "unsound mind"; in other words intellectually insane ?

5. If so, do you consider his mental disorder to be complete or partial ?

6. Do you think he understands the obligation of an oath ?

7. Do you consider him in his present condition, competent to give evidence in a Court of Law ?

8. Do you consider that he is capable of pleading to the offence of which he now stands accused ?

9. Do you happen to know how he was treated by his friends (whether as a lunatic, an imbecile or otherwise) prior to the present investigation and the occurrences that have led to it ?

10. Is he subject to insane delusions ?

11. If so, what is the general character of these ? Are they harmless or dangerous ? How do they manifest themselves ?

12. Might such delusion or delusions have led to the criminal act of which he is accused ?

13. Can you discover the cause of his reason having become affected ? In your opinion was it congenital or accidental ?

14. If the latter, does it appear to have come on suddenly, or by slow degrees ?

15. Have you any reason for believing that his insanity is of hereditary origin ? If so, please to specify the grounds for such an opinion ; and all the particulars bearing on it as to the insane parents or relations of the accused ; the exciting cause of his attack ; his age when it set in ; and the type which it assumed.

16. Have you any reason to suspect that he is, in any degree, feigning insanity ? If so, what are the grounds for this belief ?

17. Is it possible, in your opinion, that his insanity may have followed the actual commission of his offence, or been caused by it ?

18. Have you any reason to suppose that the offence could have been committed during a lucid interval during which he could be held responsible for his act ? If so, what appears to you to have been the duration of such lucid interval ? Or, on the contrary, do you believe his condition to be such as altogether to absolve him from legal responsibility ?

19. Does he now display any signs of homicidal or of suicidal mania or has he ever done so to your knowledge ?

20. Do you consider it absolutely necessary, from his present condition, that he should be confined in a lunatic asylum ?

21. Do you think that judicious and unremitting supervision, out of an asylum, might be sufficient to prevent him from endangering his own life or the property of others ?

No. IX

QUESTIONS THAT MAY BE PUT TO A MEDICAL WITNESS IN A CASE OF ALLEGED CAUSING MISCARRIAGE

1. Did you examine the person of Musammat — ? If so, when ? What did you observe ?

2. Are you of opinion that a miscarriage has occurred or not ? Give your reasons.

3. In what mode do you consider the miscarriage to have been produced, whether by violence per vaginam or by external violence, or by the use of irritants internally ? Give your reasons.

4. It is alleged that a drug called ——— was used, state the symptoms and effects which the administration internally of this drug would produce. Do you consider that it would produce miscarriage ?

5. Can you state whether the woman was quick with child when miscarriage was produced ? State your reasons.

6. Did you see the fœtus ? If so, at what period of gestation do you consider the woman to have arrived ?

No. X

QUESTIONS THAT MAY BE PUT TO A MEDICAL WITNESS IN A CASE OF GRIEVOUS HURT

1. Have you examined — ? If so, state what you observed ?

2. Describe carefully the marks of violence which you observed.

3. In what way do you consider the injuries to have been inflicted ? If by a weapon, what sort of weapon do you think was used ?

4. Do you consider that the injuries inflicted could have been caused by the weapon now shown to you (No. — of police charge sheet) ?

5. What was the direction of the wound, and can you form an opinion as to the position of the person inflicting such a wound with respect to the person receiving it ?

6. Is it possible for such a wound to have been inflicted by any one on his own person ? Give your reasons.

7. Do you consider that the injuries inflicted constitute any of the forms of "grievous hurt" defined in S. 320 of the Indian Penal Code ? If so, which of them ? Give your reasons. The Magistrate in putting this question will show the Indian Penal Code to the witness, or the Magistrate may vary the form of the question so as to elicit the required information without calling the witness's attention to the code.

8. Do you consider that the person injured is now out of danger ?

9. It is alleged that the injuries were caused by —. Could they have been caused in the manner indicated ?

10. Have you chemically or otherwise examined the stains on the weapon, clothes, etc., now before you (No. — in the police charge sheet) ?

11. Do you believe the stains to be those of blood ?

N. B.—In case of the injuries being gun shot wounds, questions 21 to 24 under the head of No. III (Death by wounds) may be put to the witness.

No. XI

QUESTIONS THAT MAY BE PUT TO A MEDICAL WITNESS IN A CASE OF DEATH FROM THE RUPTURE OF SPLEEN

1. What appearances of external violence were perceptible on the body ?

2. What was the size and weight of the spleen after death ?

3. How far did it project beyond the ribs ?

4. What was the consistency of the spleen—hard, firm, soft, pulpy or diffluent ?

5. How long after death was the body exhumed, and what was the temperature of the air ?

6. Was the body much putrefied ?

7. What was the position of the rupture ?

8. What was the length and depth of the rupture ?

9. Is it your opinion that rupture was caused by external violence or not ? State your reasons for your opinion.

10. Were there any adhesions about the spleen, if so, were they older than the rupture or not ?

APPENDIX IV

FORMS FOR MEDICO-LEGAL REPORTS

POLICE FORM No. 34|34-A

UNITED PROVINCES POLICE

No.....

From

THE SUPERINTENDENT OF POLICE,
THE OFFICER IN CHARGE OF POLICE STATION,

.....

To

THE CIVIL SURGEON OF
THE SUB-ASSISTANT SURGEON IN CHARGE OF DISPENSARY,

.....

Dated.....193 .

Sir,

I have the honour to request the favour of your examining.....

.....

.....sent to hospital on the.....

193 , and of your furnishing me with a report on the reverse of the nature and extent of bodily injury sustained by the said.....

I have the honour to be,

Sir,

Your most obedient servant,

Superintendent of Police.
Officer in charge of Police Station.

1	2	3	4	5	6	7
Nature of injury, whether cut, wound, bruise, fracture or dislocation.	Size of each injury in inches, being length, breadth and depth.	On what part of the body inflicted.	Simple, grievous or dangerous (see foot-note I).	By what weapon inflicted.	Whether the weapon was dangerous or not (Vide foot-note II).	Remarks.

Civil Surgeon.

Sub-Assistant Surgeon in Charge of Dispensary.

NOTE No. I.—Description of grievous hurt—

Firstly.—Emasculation.

Secondly.—Permanent privation of the sight of either eye.

Thirdly.—Permanent privation of the hearing of either ear.

Fourthly.—Privation of any member or joint.

Fifthly.—Destruction or permanent impairing of the powers of any member or joint.

Sixthly.—Permanent disfiguration of head or face.

Seventhly.—Fracture or dislocation of a bone or tooth.

Eighthly.—Any hurt which endangers life, or which causes the sufferer to be, during the space of twenty days, in severe bodily pain or unable to follow his ordinary pursuits.

NOTE No. II.—Description of dangerous weapon—

Any instrument for shooting, stabbing or cutting, or any instrument which, used as a weapon of offence, is likely to cause death.

OUDH JUDICIAL FORM PART VII (A) No. B-51

LETTER REQUESTING CIVIL SURGEON TO EXAMINE AND REPORT INJURIES TO A WOUNDED PERSON

No......

From

THE MAGISTRATE OF

.....

To

THE CIVIL SURGEON OF

.....

Sir,

I have to request you to examine.....sent to the hospital on the..... Be so good as to fill up the columns on the reverse of this letter and to return it to me, with such remarks as you may consider necessary to show clearly your opinion of the nature and cause of the injury.

You are|are not entitled to charge a fee in this case.

The object of this examination is to ascertain as far as possible.....

I have the honour to be,
Sir,
Your most obedient servant,

Magistrate's Office,
The.....,193 .

MAGISTRATE.

2

1	2	3	4	5	6
Nature of injury, that is, whether a cut, a bruise or a burn, etc.	Size of each injury in inches, that is length, breadth and depth.	On what part of the body inflicted.	Simple, grievous or dangerous.	By what kind of weapon inflicted.	Remarks.

NOTE.—Description of grievous hurt—

Firstly.—Emasculation.

Secondly.—Permanent privation of the sight of either eye.

Thirdly.—Permanent privation of the hearing of either ear.

Fourthly.—Privation of any member or joint.

Fifthly.—Destruction or permanent impairing of the powers of any member or joint.

Sixthly.—Permanent disfiguration of head or face.

Seventhly.—Fracture or dislocation of a bone or tooth.

Eighthly.—Any hurt which endangers life, or which causes the sufferer to be, during the space of twenty days, in severe bodily pain or unable to follow his ordinary pursuits.

Hospital,
The.....193 .

Civil Surgeon.

FORM OF MEDICAL EXAMINATION OF WOUNDED PERSONS FOR THE PROVINCE OF BIHAR

No.....

Dated.....193 .

To

THE CIVIL SURGEON.....District
MEDICAL OFFICER IN CHARGE.....Hospital

Sir,

I have the honour to request the favour of your examining..... son/daughter of.....resident of.....sent to the hospital on.....after satisfying yourself that he/she consents to examination. The question (s) and answer (s) on this point may kindly be certified at the place provided at the end of this form.

The columns of the form should be filled in and the form returned in duplicate with such remarks as you consider necessary to show clearly your opinion of the cause of the injuries.

Should there be any fear of the case terminating fatally or should unfavourable symptoms develop at any time, immediate information should be given to the Court Sub-Inspector and to me so that steps may be taken to have the dying declaration recorded by a Magistrate.

All that is known of the case at present is as follows :—

I have the honour to be,
Sir,
Your most obedient servant,

Sub-Inspector of Police.

To

THE SUB-INSPECTOR OF POLICE,

.....Police Station.

Sir,

I have the honour to forward herewith the result of my examination of.....son/daughter of....., resident of.....

1	2	3	4	5	6
Nature of injury, *i.e.*, whether a cut, a bruise, or a burn, etc.	Size of each injury in inches, *i.e.*, length, breadth and depth.	On what part of the body inflicted.	Whether "simple" or "grievous."	By what kind of weapon inflicted.	Remarks.

I certify that the said.....was asked the question (s) noted below and gave the answer (s) recorded :—

I have the honour to be,
Sir,
Your most obedient servant,

Question (s) **asked** :—

Reply given :—

(*Designation*).....

POLICE FORM No. 238

ABSTRACT IN THE VERNACULAR OF REPORT OF MEDICAL OFFICER'S EXAMINATION TO BE MADE OVER TO THE CONSTABLE ACCOMPANYING THE INJURED PERSON

No. and date of injury letter to Civil Surgeon.	Name of Police Station.	Name of injured person.	Abstract of examination report in Vernacular.

POLICE FORM No. 33

UNITED PROVINCES POLICE

No......

From

THE SUPERINTENDENT OF POLICE,

To

THE CIVIL SURGEON OF

Dated.....193 .

Sir,

You are requested to hold a post-mortem examination on the body of .....
sent herewith and to furnish a report of your examination in the accompanying form. A translation of the police officer's report on the appearance and situation of the body and on the cause of death as far as ascertainable at the time is annexed.

I have the honour to be,
Sir,
Your most obedient servant,

Superintendent of Police.

N.B.—In case of poisoning, (1) the date and hour of onset of symptoms, (2) the date and hour of patient's death, and in cases of exhumation, (3) the dates of burial and exhumation should be submitted with this to the Civil Surgeon.

Report of the post-mortem examination

On the body of

Place Date Time

Body identified by Police Constable

No. and Chaukidar

Probable age.

Probable time since death.

A.—EXTERNAL EXAMINATION

1. Condition of body as regards muscularity, stoutness, emaciation, rigor mortis and decomposition.
2. Marks of identification, especially in the case of the body of an unknown person.
3. Eyes.
4. State of natural orifices, ears, nostrils, mouth, anus, urethra, vagina.
5. Injuries—nature, exact position and measurements including direction, especially in incised wounds.
6. Bones and Joints.
7. External organs of generation.
8. Additional remarks.

B.—INTERNAL EXAMINATION

I.—*Head and Neck*

1. Scalp, skull bones (Vertex).
2. Membranes.
3. Brain.
4. Base of skull.
5. Vertebræ.
6. Spinal Cord.[1]
7. Additional remarks.

1. *Spinal Cord need not be examined unless any indications of disease, strychnia poisoning or injury exist.*

II.—*Thorax*

A. Walls, ribs, cartilages.
B. Pleuræ.
C. Larynx, Trachea and Bronchi.
D. Right lung.
E. Left lung.
F. Pericardium.
G. Heart with weight.
H. Large vessels.
I. Additional remarks.

III.—*Abdomen*

1. Walls.
2. Peritoneum.
3. Cavity.
4. Buccal cavity, teeth, tongue, and pharynx.
5. Œsophagus.
6. Stomach and its contents.
7. Small intestine and its contents.
8. Large intestine and its contents.
9. Liver (with weight) and gall bladder.
10. Pancreas.
11. Spleen with weight.
12. Kidneys with weight.
13. Bladder.
14. Organs of generation.
15. Additional remarks with, where possible, medical man's deduction from the state of the contents of the stomach as to time of death and last meal.

C.—DATE AND HOUR OF ONSET OF SYMPTOMS. To be answered
Do. OF DEATH in cases of poisoning*

D.—OPINION AS TO CAUSE AND MANNER OF DEATH.

Place.

Date.

Medical Officer.

---

* *In case of exhumation the dates of burial and exhumation should be furnished.*

POLICE FORM No. 289

ABSTRACT OF POST-MORTEM TO BE FORWARDED TO LOCAL POLICE WITHOUT DELAY

Name of thana.	Name of deceased.	Date of Post-mortem.	Probable cause of death. (To be recorded in English by the Civil Surgeon immediately after holding post-mortem).	Urdu translation of column 4.

APPENDIX V

THE INDIAN EVIDENCE ACT

(ACT I OF 1872)

SEC. 3. INTERPRETATION CLAUSE.—In this Act the following words and expressions are used in the following senses, unless a contrary intention appears from the context :—

"Court" includes all Judges and Magistrates, and all persons except arbitrators, legally authorized to take evidence.

"Fact" means and includes—(1) anything, state of things, or relation of things, capable of being perceived by the senses ; (2) any mental condition of which any person is conscious.

"Document" means any matter expressed or described upon any substance by means of letters, figures or marks, or by more than one of those means, intended to be used or which may be used, for the purpose of recording that matter.

"Evidence" means and includes—

(1) All statements which the Court permits or requires to be made before it by witnesses in relation to matters of fact under inquiry ; such statements are called oral evidence.

(2) All documents produced for the inspection of the Court ; such documents are called documentary evidence.

SEC. 32. CASES IN WHICH STATEMENT OF RELEVANT FACT BY PERSON WHO IS DEAD OR CANNOT BE FOUND, ETC., IS RELEVANT.—Statements written or verbal, of relevant facts made by a person who is dead, or who cannot be found, or who has become incapable of giving evidence, or whose attendance cannot be procured without an amount of delay or expenses which, under the circumstances of the case, appears to the Court unreasonable, are themselves relevant facts in the following cases :—

When the statement is made by the person as to the cause of his death, or as to any of the circumstances of the transaction which resulted in his death, in cases in which the cause of the person's death comes into question.

Such statements are relevant whether the person who made them was or was not, at the time when they were made, under expectation of death, and whatever be the nature of the proceeding in which the cause of his death comes into question.

SEC. 33. RELEVANCY OF CERTAIN EVIDENCE FOR PROVING IN SUBSEQUENT PROCEEDING, THE TRUTH OF FACTS, THEREIN STATED.—Evidence given by a witness in a judicial proceeding, or before any person authorized by law to take it, is relevant for the purpose of proving, in a subsequent judicial proceeding, or in a later stage of the same judicial proceeding, the truth of the facts which it states, when the witness is dead or cannot be found or is incapable of giving evidence, or is kept out of the way by the adverse party, or if his presence cannot be obtained without an amount of delay or expense which, under the circumstances of the case, the Court considers unreasonable ;

Provided—

that the proceeding was between the same parties or their representatives in interest ;

that the adverse party in the first proceeding had the right and opportunity to cross-examine ;

that the questions in issue were substantially the same in the first as in the second proceeding.

SEC. 45. OPINIONS OF EXPERTS.—When the Court has to form an opinion upon a point of foreign law, or of science or art, or as to identity of handwriting or finger impressions, the opinions upon that point of persons specially skilled in such foreign law, science or art or in questions as to identity of handwriting or finger impressions are relevant facts. Such persons are called experts.

SEC. 46. FACTS BEARING UPON OPINIONS OF EXPERTS.—Facts, not otherwise relevant, are relevant if they support or are inconsistent with the opinions of experts, when such opinions are relevant.

SEC. 59. PROOF OF FACTS BY ORAL EVIDENCE.—All facts, except the contents of documents, may be proved by oral evidence.

SEC. 60. ORAL EVIDENCE MUST BE DIRECT.—Oral evidence must, in all cases whatever, be direct; that is to say—

if it refers to a fact which could be seen, it must be the evidence of a witness who says he saw it;

if it refers to a fact which could be heard, it must be the evidence of a witness who says he heard it;

if it refers to a fact which could be perceived by any other sense or in any other manner, it must be the evidence of a witness who says he perceived it by that sense or in that manner;

if it refers to an opinion or to the grounds on which that opinion is held, it must be the evidence of the person who holds that opinion on those grounds:

Provided that the opinions expressed in any treatise commonly offered for sale, and the ground on which such opinions are held, may be proved by the production of such treatises if the author is dead or cannot be found, or has become incapable of giving evidence, or cannot be called as a witness without an amount of delay or expense which the Court regards as unreasonable:

Provided also that, if oral evidence refers to the existence or condition of any material thing other than a document, the Court may, if it thinks fit, require the production of such material thing for its inspection.

SEC. 61. PROOF OF CONTENTS OF DOCUMENTS.—The contents of documents may be proved either by primary or by secondary evidence.

SEC. 62. PRIMARY EVIDENCE.—Primary evidence means the document itself produced for the inspection of the Court.

Explanation 1.—Where a document is executed in several parts, each part is primary evidence of the document.

Where a document is executed in counterpart, each counterpart being executed by one or some of the parties only, each counterpart is primary evidence as against the parties executing it.

Explanation 2.—Where a number of documents are all made by one uniform process, as in the case of printing, lithography, or photography, each is primary evidence of the contents of the rest; but where they are all copies of a common original, they are not primary evidence of the contents of the original.

SEC. 63. SECONDARY EVIDENCE.—Secondary evidence means and includes—

1. Certified copies given under the provisions hereinafter contained;
2. Copies made from the original by mechanical processes which in themselves insure the accuracy of the copy, and copies compared with such copies;
3. Copies made from or compared with the original;
4. Counterparts of documents as against the parties who did not execute them;

5. Oral accounts of the contents of a document given by some person who has himself seen it.

SEC. 107. BURDEN OF PROVING DEATH OF PERSONS KNOWN TO HAVE BEEN ALIVE WITHIN THIRTY YEARS.—When the question is whether a man is alive or dead, and it is shown that he was alive within thirty years; the burden of proving that he is dead is on the person who affirms it.

SEC. 108. BURDEN OF PROVING THAT PERSON IS ALIVE WHO HAS NOT BEEN HEARD OF FOR SEVEN YEARS.—[Provided that when] the question is whether a man is alive or dead, and it is proved that he has not been heard of for seven years by those who would naturally have heard of him if he had been alive, the burden of proving that he is alive is shifted to the person who affirms it.

SEC. 112. BIRTH DURING MARRIAGE CONCLUSIVE PROOF OF LEGITIMACY.—The fact that any person who was born during the conitnuance of a valid marriage between his mother and any man or within 280 days after its dissolution, the mother remaining unmarried, shall be conclusive proof that he is the legitimate son of that man, unless it can be shown that the parties to the marriage had no access to each other at any time when he could have been begotten.

SEC. 118. WHO MAY TESTIFY.—All persons shall be competent to testify unless the Court considers that they are prevented from understanding the questions put to them, or from giving raitonal answers to those questions, by tender years, extreme old age, disease, whether of body or mind, or any other cause of the same kind.

Explanation.—A lunatic is not incompetent to testify, unless he is prevented by his lunacy from understanding the questions put to him and giving rational answers to them.

SEC. 119. DUMB WITNESS.—A witness who is unable to speak may give his evidence in any other manner in which he can make it intelligible, as by writing, or by signs; but such writing must be written and the signs made in open Court. Evidence so given shall be deemed to be oral evidence.

SEC. 124. OFFICIAL COMMUNICATION.—No public officer shall be compelled to disclose communications made to him in official confidence, when he considers that the public interests would suffer by the disclosure.

SEC. 126. PROFESSIONAL COMMUNICATION.—No barrister, attorney, pleader, or vakil shall at any time be permitted, unless with his client's express consent to disclose any communication made to him in the course and for the purpose of his employment as such barrister, attorney, pleader or vakil, by or on behalf of his client, or to state the contents or condition of any document with which he has become acquainted in the course and for the purpose of his professional employment, or to disclose any advice given by him to his client in the course and for the purpose of such employment.

Provided that nothing in this section shall protect from disclosure

(1) any such communication made in furtherance of any illegal purpose;

(2) any fact observed by any barrister, pleader, attorney or vakil, in the course of his employment as such, showing that any crime or fraud has been committed since the commencement of his employment.

It is immaterial whether the attention of such barrister, pleader, attorney, or vakil was or was not directed to such fact by or on behalf of his client.

Explanation.—The objection stated in this seciton continues after the employment has ceased.

SEC. 132. WITNESS NOT EXCUSED FROM ANSWERING ON GROUND THAT ANSWER WILL CRIMINATE.—A witness shall not be excused from answering any question as to any matter relevant to the matter in issue in any suit or in any civil or criminal proceeding, upon the ground that the answer to such question will criminate or may tend directly or indirectly to criminate such witness, or that it will expose or tend directly or indirectly to expose, such witness to a penalty or forfeiture of any kind: Provided that no such answer which a witness shall be

compelled to give, shall subject him to any arrest or prosecution, or be proved against him in any criminal proceeding, except a prosecution for giving false evidence by such answer.

SEC. 137. EXAMINATION-IN-CHIEF.—The examination of a witness by the party who calls him shall be called his examination-in-chief.

CROSS-EXAMINATION.—The examination of a witness by the adverse party shall be called his cross-examination.

RE-EXAMINATION.—The examination of a witness, subsequent to the cross-examination, by the party who called him shall be called his re-examination.

SEC. 138. ORDER OF EXAMINATIONS.—Witnesses shall be first examined-in-chief, then—if the adverse party so desires—cross-examined, then—if the party calling him so desires—re-examined.

The examination and cross-examination must relate to relevant facts, but the cross-examination need not be confined to the facts, to which the witness testified in his examination-in-chief.

DIRECTION OF RE-EXAMINATION.—The re-examinaiton shall be directed to the explanation of matters referred to in cross-examination, and if new matter is, by permission of the Court, introduced in re-examination, the adverse party may further cross-examine upon that matter.

SEC. 141. LEADING QUESTIONS.—Any question suggesting the answer which the person putting it wishes or expects to receive is called a leading question.

SEC. 142. WHEN THEY MUST NOT BE ASKED.—Leading questions must not, if objected to by the adverse party, be asked in an examination-in-chief, or in a re-examination, except with the permission of the Court.

The Court shall permit leading questions as to matters which are introductory or undisputed, or which have, in its opinion, been already sufficiently proved.

SEC. 143. WHEN THEY MAY BE ASKED.—Leading questions may be asked in cross-examination.

SEC. 146. QUESTIONS LAWFUL IN CROSS-EXAMINATION.—When a witness is cross-examined, he may in addition to the questions hereinbefore referred be asked any questions which tend

(1) to test his veracity,

(2) to discover who he is and what is his position in life,

(3) to shake his credit, by injuring his character, although the answer to such question might tend directly or indirectly to expose him to a penalty or forfeiture.

SEC. 152. QUESTIONS INTENDED TO INSULT OR ANNOY.—The Court shall forbid any question which appears to it to insult or annoy, or which though proper in itself, appears to the Court needlessly offensive in form.

SEC. 157. FORMER STATEMENTS OF WITNESS MAY BE PROVED TO CORROBORATE LATER TESTIMONY AS TO SAME FACT.—In order to corroborate the testimony of a witness, any former statement made by such witness relating to the same fact, at or about the time when the fact took place, or before any authority legally competent to investigate the fact, may be proved.

SEC. 159. REFRESHING MEMORY.—A witness may, while under examination, refresh his memory by referring to any writing made by himself at the time of the transaction concerning which he is questioned, or so soon afterwards that the Court considers it likely that the transaction was at that time fresh in his memory.

The witness may also refer to any such writing made by any other person, and read by the witness within the time aforesaid, if when he read it, he knew it to be correct.

Whenever a witness may refresh his memory by reference to any document, he may, with the permission of the Court, refer to a copy of such document:

Provided the Court be satisfied that there is sufficient reason for the non-production of the original.

An expert may refresh his memory by reference to professional treatises.

SEC. 160. TESTIMONY TO FACTS STATED IN DOCUMENT MENTIONED IN SEC. 159.—A witness may also testify to facts, mentioned in any such document in Section 159, although he has no specific recollection of the facts themselves if he is sure that the facts were correctly recorded in the document.

SEC. 161. RIGHT OF ADVERSE PARTY AS TO WRITING USED TO REFRESH MEMORY.—Any writing referred to under the provisions of the two last preceding sections must be produced and shown to the adverse party if he requires it; such party may, if he pleases, cross-examine the witness thereupon.

SEC. 165. JUDGE'S POWER TO PUT QUESTIONS OR ORDER PRODUCTION.—The Judge may, in order to discover or to obtain proper proof of relevant facts, ask any question he pleases, in any form, at any time, of any witness, or of the parties about any fact relevant or irrelevant, and may order the production of any document or thing; and neither the parties nor their agents shall be entitled to make any objection to any such question or order, nor without the leave of the Court to cross-examine any witness upon any answer given in reply to any such question:

Provided that the judgment must be based upon facts declared by this Act to be relevant, and duly proved.

Provided also that this section shall not authorise any Judge to compel any witness to answer any question, or to produce any document which such witness would be entitled to refuse to answer or produce under Sections 121 to 131, both inclusive, if the questions were asked or the documents were called for by the adverse party; nor shall the Judge ask any question which it would be improper for any other person to ask under Section 148 or 149; nor shall he dispense with primary evidence of any document, except in the cases hereinbefore excepted.

SEC. 166. POWER OF JURY OR ASSESSORS TO PUT QUESTIONS.—In cases tried by jury or with assessors, the jury or assessors may put any questions to the witnesses, through or by leave of the Judge, which the Judge himself might put and which he considers proper.

APPENDIX VI

THE CODE OF CRIMINAL PROCEDURE

(ACT V OF 1898) AS AMENDED BY THE CRIMINAL LAW AMENDMENT ACT, XII OF 1923 AND CRIMINAL PROCEDURE CODE AMENDMENT ACT, XVIII OF 1923, AND AS AMENDED UPTO DATE WITH THE CRIMINAL LAW AMENDMENT ACT, 1933

SEC. 4. (*f*) "Cognizable offence" means an offence for, and "cognizable case" means a case in which a police-officer, within or without the presidency towns, may, in accordance with the second schedule, or under any law for the time being in force, arrest without warrant.

SEC. 6. CLASSES OF CRIMINAL COURTS.—Besides the High Courts and the Courts constituted under any law other than this Code for the time being in force, there shall be five classes of Criminal Courts in British India, namely :—

I.—Courts of Session.

II.—Presidency Magistrates.

III.—Magistrates of the first class.

IV.—Magistrates of the second class.

V.—Magistrates of the third class.

SEC. 29-A. TRIAL OF EUROPEAN BRITISH SUBJECTS BY SECOND AND THIRD CLASS MAGISTRATES.—No Magistrate of the second or third class shall inquire into or try any offence which is punishable otherwise than with fine not exceeding fifty rupees where the accused is an European or British subject who claims to be tried as such.

SEC. 29-B. JURISDICTION IN THE CASE OF JUVENILES.—Any offence other than one punishable with death or transportation for life, committed by any person who at the date when he appears or is brought before the Court is under the age of fifteen years, may be tried by a District Magistrate or a Chief Presidency Magistrate, or by any Magistrate specially employed by the Local Government to exercise the powers conferred by Section 8, Sub-section (1) of the Reformatory Schools Act, 1897, or, in any area in which the said Act has been wholly or in part repealed by any other law providing for the custody, trial or punishment of youthful offenders, by any Magistrate empowered or under such law to exercise all or any of the powers conferred thereby.

SEC. 31. SENTENCES WHICH HIGH COURTS AND SESSIONS JUDGES MAY PASS.—(1) A High Court may pass any sentence authorised by law.

(2) A Sessions Judge or Additional Sessions Judge may pass any sentence authorised by law; but any sentence of death passed by any such Judge shall be subject to confirmation by the High Court.

(3) An Assistant Sessions Judge may pass any sentence authorised by law, except a sentence of death or of transportation for a term exceeding seven years, or of imprisonment for a term exceeding seven years.

SEC. 32. SENTENCES WHICH MAGISTRATES MAY PASS.—(1) The Courts of Magistrates may pass the following sentences, namely :—

(*a*) Courts of Presidency Magistrates and Magistrates of the first class;	Imprisonment for a term not exceeding two years, including such solitary confinement as is authorised by law; Fine not exceeding one thousand rupees; Whipping.
(*b*) Courts of Magistrates of the second class;	Imprisonment for a term not exceeding six months, including such solitary confinement as is authorised by law; Fine not exceeding two hundred rupees.
(c) Courts of Magistrates of the third class;	Imprisonment for a term not exceeding one month; Fine not exceeding fifty rupees.

(2) The Court of any Magistrate may pass any lawful sentence, combining any of the sentences which it is authorised by law to pass.

SEC. 33. POWER OF MAGISTRATES TO SENTENCE TO IMPRISONMENT IN DEFAULT OF FINE.—(1) The Court of any Magistrate may award such term of imprisonment in default of payment of fine as is authorised by law in case of such default:

Provided that—

(*a*) the term is not in excess of the Magistrate's powers under this Code:

(*b*) in any case decided by a Magistrate where imprisonment has been awarded as part of the substantive sentence, the period of imprisonment awarded in default of payment of the fine shall not exceed one-fourth of the period of imprisonment which such Magistrate is competent to inflict as punishment for the offence otherwise than as imprisonment in default of payment of the fine.

(2) The imprisonment awarded under this section may be in addition to a substantive sentence of imprisonment for the maximum term awarded by the Magistrate under Section 32.

SEC. 34. HIGHER POWERS OF CERTAIN DISTRICT MAGISTRATES.—The Court of a Magistrate specially empowered under Section 30, may pass any sentence authorised by law except a sentence of death or of transportation for a term exceeding seven years or of imprisonment for a term exceeding seven years.

SEC. 34-A. SENTENCES WHICH COURTS AND MAGISTRATES MAY PASS UPON EUROPEAN BRITISH SUBJECTS.—Notwithstanding anything contained in Sections 31, 32 and 34—

(*a*) no Court of Sessions shall pass on any European British Subject any sentence other than a sentence of death, penal servitude, or imprisonment with or without fine, or of fine, and

(*b*) no District Magistrate or other Magistrate of the first class shall pass on any European British Subject any sentence other than imprisonment which may extend to two years or fine which may extend to one thousand rupees or both.

IMPRISONMENT.—There are two grades of imprisonment, *viz.*, rigorous (hard labour) and simple. Hard labour consists chiefly in grinding corn, oil pressing, soorkhi pounding, paper pounding and polishing, digging and carrying earth, drawing water, cutting firewood, latrine work, bowing wool, blacksmith's work, etc. Simple imprisonment means nothing more than confinement in jail, subject to the jail rules as to diet, etc., and prisoners undergoing such a sentence cannot lawfully be put to any work against their will. There is, however, no objection to their being employed on any trade or occupation permitted by the jail rules, if they, of their own free will, express their desire for employment.

WHIPPING.—In the Indian Act there is no provision as to how the punishment of whipping has to be inflicted on youthful offenders, but the Local Government is authorised to prescribe it. According to the English law whipping has to be carried

out in private by a policeman in the presence of an Inspector and the number of stripes to be inflicted has been fixed according to the age. Thus, six stripes for a child of six, twelve for a boy under fourteen. The Government of the United Provinces of Agra and Oudh[1] have prescribed the following rules to be observed as regards the place and mode of inflicting the punishment of whipping :—

(1) All judicial whippings shall in future be inflcited in private, either at a jail or in an enclosure near the court-house ;

(2) Wherever it is possible to do so Magistrates shall secure the presence of a medical officer at the whipping ;

NOTE.—The presence of a superior medical officer is not necessary at whipping. It will be sufficient if a competent sub-assistant surgeon is present but in any doubtful case the man to be whipped should be sent beforehand to the civil surgeon for inspection. The civil surgeon will have to certify whether the man can stand a whipping. Any advanced organic disease should be regarded as a contra-indication. It should also be remembered that the medical officer might be asked during the whipping if the person's health was likely to suffer from its completion. Severe hæmorrhage in the case of a hæmophiliac or signs of physical collapse should be the signal to stop the punishment.

(3) the practice shall invariably be adopted of spreading a thin cloth soaked in some antiseptic over the prisoner's buttocks during the operation ;

(4) the cane employed shall never exceed the legal minimum of half inch in diameter in the case of persons of over sixteen years of age [Section 392 (i) of the Criminal Procedure Code] ; and in the case of juvenile offenders a still lighter cane shall be employed.

SEC. 42. PUBLIC WHEN TO ASSIST MAGISTRATES AND POLICE.—Every person is bound to assist a Magistrate or police-officer reasonably demanding his aid, whether within or without the presidency-towns,—

(*a*) in the taking or preventing the escape of any other person whom such Magistrate or police-officer is authorised to arrest ;

(*b*) in the prevention or suppression of a breach of the peace, or in the prevention of any injury attempted to be committed to any railway, canal, telegraph or public property.

SEC. 44. PUBLIC TO GIVE INFORMATION OF CERTAIN OFFENCES.—(1) Every person, whether within or without the Presidency towns, aware of the commission of, or of the intention of any other person to commit any offence punishable under any of the following Sections of the Indian Penal Code (namely), 121, 121-A, 122, 123, 124, 124-A, 125, 126, 130, 143, 144, 145, 147, 148, 302, 303, 304, 382, 392, 393, 394, 395, 396, 397, 398, 399, 402, 435, 436, 449, 450, 456, 457, 458, 459 and 460, shall, in the absence of reasonable excuse, the burden of proving which will lie upon the person so aware, forthwith give information to the nearest magistrate or police-officer of such commission or intention.

(2) For the purpose of this section the term "offence" includes any act committed at any place out of British India which would constitute an offence if committed in British India.

Punishment.—Omission to give information under the section is punishable under Sections 118, 176 and 202, Indian Penal Code.

SEC 45. VILLAGE HEADMEN, ACCOUNTANTS, LANDHOLDERS AND OTHERS BOUND TO REPORT CERTAIN MATTERS.—(1) Every village headman, village accountant, village watchman, village police officer, owner or occupier of land, and the agent or any such owner or occupier in charge of the management of that land and every officer employed in the collection of revenue or rent of land on the part of the Government or the Court of Wards, shall forthwith communicate to the nearest magistrate or to the officer in charge of the nearest police station, whichever is the nearer, any information which he may possess respecting :—

1. *Manual of Government Orders, Vol. I, Part VI, para* 924.

(*a*) the permanent or temporary residence of any notorious receiver or vendor of stolen property in any village of which he is headman, accountant, watchman, or police officer, or in which he owns or occupies land, or is agent, or collects revenue or rent;

(*b*) the resort to any place within, or the passage through, such village of any person whom he knows, or reasonably suspects, to be a thug, robber, escaped convict or proclaimed offender;

(*c*) the commission of, or intention to commit, in or near such village any non-bailable offence or any offence punishable under Sections 143, 144, 145, 147 or 148 of the Indian Penal Code;

(*d*) the occurrence in or near such village of any sudden or unnatural death or of any death under suspicious circumstances or the discovery in or near such village of any corpse or part of a corpse, in circumstances which lead to a reasonable suspicion that a non-bailable offence has been committed in respect of such person;

(*e*) the commission of, or intention to commit, at any place out of British India near such village any act which, if committed in British India, would be an offence punishable under any of the following Sections of the Indian Penal Code, namely, 231, 232, 234, 235, 237, 238, 302, 304, 382, 393, 394, 395, 396, 397, 398, 399, 402, 435, 436, 449, 450, 457, 458, 459, 460, 489-A, 489-B, 489-C and 489-D;

(*f*) any matter likely to affect the maintenance of order or the prevention of crime or the safety of person or property respecting which the District Magistrate, by general or special order made with the previous sanction of the Local Government, has directed him to communicate information.

(2) In this section—

(*i*) "village" includes village-lands; and

(*ii*) the expression "proclaimed offender" includes any person proclaimed as an offender by any Court or authority established or continued by the Governor-General in Council in any part of India, in respect of any act which, if committed in British India, would be punishable under any of the following Sections of the Indian Penal Code, namely, 302, 304, 382, 397, 398, 399, 402, 435, 436, 449, 450, 457, 458, 459 and 460.

(3) Subject to rules in this behalf to be made by the Local Government, the District Magistrate may from time to time appoint one or more persons with his or their consent to perform the duties of a village-headman under this section whether a village-headman has or has not been appointed for that village under any other law.

SEC. 54. WHEN POLICE MAY ARREST WITHOUT WARRANT.—(1) Any police-officer may, without an order from a Magistrate and without a warrant arrest—

First, any person who has been concerned in any cognizable offence or against whom a reasonable complaint has been made or credible information has been received, or a reasonable suspicion exists of his having been so concerned;

Secondly, any person having in his possession without lawful excuse, the burden of proving which excuse shall lie on such person, any implement of house-breaking;

Thirdly, any person who has been proclaimed as an offender under this Code or by order of the Local Government;

Fourthly, any person in whose possession anything is found which may reasonably be suspected to be stolen property and who may reasonably be suspected of having committed an offence with reference to such thing;

Fifthly, any person who obstructs a police-officer while in the execution of his duty, or who has escaped, or attempts to escape, from lawful custody;

Sixthly, any person reasonably suspected of being a deserter from Her Majesty's Army or Navy or of belonging to Her Majesty's Indian Marine Service and being illegally absent from that service;

Seventhly, any person who has been concerned in, or against whom a reasonable complaint has been made or credible information has been received or a reasonable suspicion exists of his having been concerned in, any act committed at any place out of British India, which, if committed in British India, would have been punishable as an offence, and for which he is, under any law relating to extradition or under the Fugitive Offenders Act, 1881, or otherwise, liable to be apprehended or detained in custody in British India ;

Eighthly, any released convict committing a breach of any rule made under Section 565, Sub-section (3) ; and

Ninthly, any person for whose arrest a requisition has been received from another police-officer, provided that the requisition specifies the person to be arrested and the offence or other cause for which the arrest is to be made and it appears therefrom that the person might lawfully be arrested without a warrant by the officer who issued the requisition.

(2) This section applies also to the police in the town of Calcutta.

THE POWERS OF POLICE TO INVESTIGATE

SEC. 154. INFORMATION IN COGNIZABLE CASES.—Every information relating to the commission of a cognizable offence, if given orally to an officer in charge of a police station, shall be reduced to writing by him or under his direction, and be read over to the informant ; and every such information, whether given in writing or reduced to writing as aforesaid, shall be signed by the person giving it and the substance thereof shall be entered in a book to be kept by such officer in such form as the Local Government may prescribe in this behalf.

SEC. 155. INFORMATION IN NON-COGNIZABLE CASES.[1]—(1) When information is given to an officer in charge of a police-station of the commission, within the limits of such station, of a non-cognizable offence, he shall enter in a book to be kept as aforesaid the substance of such information and refer the informant to the Magistrate.

(2) No police-officer shall investigate a non-cognizable case without the order of a Magistrate of the first or second class having power to try such case or commit the same for trial, or of a Presidency Magistrate.

(3) Any police-officer receiving such order may exercise the same powers in respect of the investigation (except the power to arrest without warrant) as an officer in charge of a police-station may exercise in a cognizable case.

SEC. 156. INVESTIGATION INTO COGNIZABLE CASES.—(1) Any officer in charge of a police station may, without the order of a Magistrate, investigate any cognizable case which a Court having jurisdiction over the local area within the limits of such station would have power to inquire into or try under the provisions of Chapter XV relating to the place of inquiry or trial.

(2) No proceeding of a police-officer in any such case shall at any stage be called in question on the ground that the case was one which such officer was not empowered under this section to investigate.

(3) Any Magistrate empowered under Section 190 may order such an investigation as above-mentioned.

SEC. 157. PROCEDURE WHERE COGNIZABLE OFFENCE SUSPECTED—(1) If, from information received or otherwise, an officer in charge of a police-station has reason to suspect the commission of an offence which he is empowered under Section 156 to investigate, he shall forthwith send a report of the same to a Magistrate empowered to take cognizance of such offence upon a police-report, and shall proceed in person, or shall depute one of his subordinate officers not being below such rank as the Local Government may by general or special order prescribe in this behalf to proceed, to the spot to investigate the facts and circumstances of

1. *This section so far as it applies to the police in the town of Bombay, is repealed by S.* 211 *and Schedule A to the City of Bombay Police Act,* 1902 (*Bombay Act IV of* 1902).

the case, and, if necessary, to take measures for the discovery and arrest of the offender :

Provided as follows : —

(*a*) when any information as to the commission of any such offence is given against any person by name and the case is not of a serious nature, the officer in charge of a police-station need not proceed in person or depute a subordinate officer to make an investigation on the spot ;

(*b*) if it appears to the officer in charge of a police station that there is no sufficient ground for entering on an investigation, he shall not investigate the case.

(2) In each of the cases mentioned in clauses (*a*) and (*b*) of the proviso to sub-section (1) the officer in charge of the police-station shall state in his said report his reasons for not fully complying with the requirements of that sub-section, and in the case mentioned in clause (*b*) such officer shall also forthwith notify to the informant, if any, in such manner as may be prescribed by the Local Government, the fact that he will not investigate the case or cause it to be investigated.

SEC. 160. POLICE-OFFICERS' POWER TO REQUIRE ATTENDANCE OF WITNESSES.—Any police-officer making an investigation under this Chapter may, by order in writing, require the attendance before himself of any person being within the limits of his own or any adjoining station, who, from the information given or otherwise, appears to be acquainted with the circumstances of the case, and such person shall attend as so required.

SEC. 161. EXAMINATION OF WITNESSES BY POLICE.—(1) Any police-officer making an investigation under this Chapter (power to investigate) or any police-officer not below such rank as the Local Government may, by general or special order, prescribe in this behalf, acting on the requisition of such officer, may examine orally any person supposed to be acquainted with the facts and circumstances of the case.

(2) Such person shall be bound to answer all questions relating to such case put to him by such officer, other than questions the answers to which would have a tenedency to expose him to a criminal charge or to a penalty of forfeiture.

SEC. 164. POWER TO RECORD STATEMENTS AND CONFESSIONS.—(1) Any Presidency Magistrate, any Magistrate of the first class and any Magistrate of the second class specially empowered in this behalf by the Local Government may, if he is not a police-officer, record any statement or confession made to him in the course of an investigation under this Chapter or at any time afterwards before the commencement of the inquiry or trial.

(2) Such statements shall be recorded in such of the manners hereinafter prescribed for recording evidence as is, in his opinion, best fitted for the circumstances of the case. Such confessions shall be recorded and signed in the manner provided in Section 364 and such statements or confessions shall then be forwarded to the Magistrate by whom the case is to be inquired into or tried.

(3) A Magistrate shall before recording any such confession explain to the person making it that he is not bound to make a confession and that if he does so it may be used as evidence against him and no Magistrate shall record any such confession unless, upon questioning the person making it, if he has reason to believe that it was not made voluntarily ; and when he records any confession, he shall make a memorandum at the foot of such record to the following effect : —

"I have explained to (name) that, he is not bound to make a confession and that if he does so, any confession he may make, may be used as evidence against him and I believe that this confession was voluntarily made. It was taken in my presence and hearing, and was read over to the person making it and admitted by him to be correct, and it contains a full and true account of the statement made by him.

(Signed) A. B.
Magistrate."

Explanation.—It is not necessary that the Magistrate receiving and recording a confession or statement should be a Magistrate having jurisdiction in the case.

SEC. 174. POLICE TO INQUIRE AND REPORT ON SUICIDE, ETC.—(1) The officer in charge of a police-station, or some other police officer[1] specially empowered by the Local Government in that behalf, on receiving information that a person—

(a) has committed suicide, or

(b) has been killed by another, or by an animal, or by machinery, or by an accident, or

(c) has died under circumstances raising a reasonable suspicion that some other person has committed an offence,

shall immediately give intimation thereof to the nearest Magistrate empowered to hold inquests, and, unless otherwise directed by any rule prescribed by the Local Government, or by any general or special order of the District or Sub-divisional Magistrate, shall proceed to the place where the body of such deceased person is, and there, in the presence of two or more respectable inhabitants of the neighbourhood, shall make an investigation and draw up a report of the apparent cause of death, describing such wounds, fractures, bruises and other marks of injury as may be found on the body, and stating in what manner, or by what weapon or instrument (if any), such marks appear to have been inflicted.

(2) The report shall be signed by such police-officer and other persons, or by so many of them as concur therein, and shall be forthwith forwarded to the District Magistrate or the Sub-divisional Magistrate.

(3) When there is any doubt regarding the cause of death, or when for any other reason the police-officer considers it expedient so to do, he shall, subject to such rules as the Local Government may prescribe in this behalf, forward the body, with a view to its being examined, to the nearest Civil Surgeon, or other qualified medical man appointed in this behalf by the Local Government, if the state of the weather and the distance admit of its being so forwarded without risk of such putrefaction on the road as would render such examination useless.

(4) In the Presidencies of Fort St. George and Bombay, investigation under this section may be made by the head of the village, who shall then report the result to the nearest Magistrate authorised to hold inquests.

(5) The following Magistrates are empowered to hold inquests, namely, any District Magistrate, Sub-divisional Magistrate, or Magistrate of the First Class and any Magistrate specially empowered in this behalf by the Local Government or the District Magistrate.

Scope.—When the body cannot be found or has been buried, there can be no investigation under Section 174. This section is intended to apply to cases in which an inquest is necessary, which presupposes that the corpse must be available.—*Gul Hasan,* 1908, *P. R.* 27, 9 *Cr. L. J.*, 105.

N.B.—1. Whenever information of the sudden or unnatural death of a European is reported at a police station, the officer in charge of the station shall send an urgent immediate information to the District Superintendent and the Inquest Report required by S. 174 of Act X of 1882, shall be taken by a European officer; and unless death has been caused by violence, the marks of which are apparent, no native officer shall have the right of examination of the body. Under no circumstances shall any examination be taken when the deceased is of the female sex. In such case a police officer, not below the rank of Head Constable, will remain with or accompany the body till receipt of orders from the Magistrate.—*Reg. and Ord. N. W. P., S.* 10, *Art.* 156, *p.* 275.

2. It appears to the Government of India that it will be better, if inquiries into cases of sudden and unnatural deaths of soldiers are made by Magistrates and

1. *Head Constables specially selected by the Superintendent of Police are empowered by the Local Government to make inquiries* (vide *U. P. Govt., No.* 75 *VI,* 103-1906 *dated March* 12*th,* 1917).

not by the police. The police should, however, report all such occurrences to the Magistrate.—*No.* 1398, *dated* 10*th October,* 1878.

SEC. 175. POWER TO SUMMON PERSONS.—(1) A police-officer proceeding under Section 174 may, by order in writing, summon two or more persons as aforesaid for the purpose of the said investigation, and any other person who appears to be acquainted with the facts of the case. Every person so summoned shall be bound to attend and to answer truly all questions other than questions the answers to which would have a tendency to expose him to a criminal charge, or to a penalty or forfeiture.

(2) If the facts do not disclose a cognizable offence to which Sectoin 170 applies, such persons shall not be required by the police-officer to attend a Magistrate's Court.

THE CORONER'S ACT.—In the Presidency towns of Bombay and Calcutta the Coroner's Act IV of 1871 is in force. The following are some of the sections of the Act in connection with the investigation :—

8. *Jurisdiction to enquire into deaths.*—When a Coroner has reason to believe that the death of any person has been caused by accident, homicide, suicide, or suddenly by means unknown or that any person, being a prisoner has died in prison, and that the body is lying within the place for which the Coroner is so appointed, the Coroner shall enquire into the cause of death. Every such enquiry shall be deemed a judical proceeding within the meaning of Section 193 of the Indian Penal Code.

9. *Coroner to be sent for when prisoner dies.*—Whenever a prisoner dies in a prison situate within the place for which a Coroner is also appointed, the Superintendent of the Prison shall send for the Coroner before the body is disposed of. Any Superintendent failing herein shall, on conviction before a Magistrate, be punished. with fine not exceeding five hundred rupees. Nothing in the former part of this Section applies to cases in which the death has been caused by cholera or other epidemic diseases.

10. *Power to hold inquests on bodies within local limits whenever cause of death occurred.*—Whenever an inquest ought to be held on any body lying dead within the local limits of the jurisdiction of any Coroner, he shall hold such inquest, whether or not the cause of death arose within his jurisdiction.

11. *Power to order body to be disinterred.*—A Coroner may order a body to be disinterred within a reasonable time after the death of the deceased person, either for the purpose of taking an original inquisition where none has been taken, or a further inquisition where the Coroner considers it necessary or desirable in the interests of justice to take a further inquisition.

12. *Summoning jury.*—On receiving notice of any death mentioned in Section 8, the Coroner shall summon five, seven, nine, eleven, thirteen, or fifteen respectable persons to appear before him at a time and place to be specified in the summons, for the purpose of enquiring when, how, and by what means the deceased came by his death. Any inquest under this Act may be held on a Sunday.

14. *Jurors to be sworn.*—When a sufficient jury is in attendance, he shall administer an oath to each juror to give a true verdict according to the evidence, and shall then proceed with the jury to view the body.

15. *View of the body.*—The Coroner and the jury shall view and examine the body at the first sitting of the inquest, and the Coroner shall make such observations to the jury as the appearance of the body requires.

Provided that the Coroner may, with the concurrence of a majority of the jury, dispense with a view of the body, if he is satisfied, from medical evidence or medical certificates, that no advantage should result from such viewing.

17. *Summoning witnesses.*—It shall be the duty of all persons acquainted with the circumstances attending the death to appear before the inquest as witnesses ; the Coroner shall enquire of such circumstances and the cause of death ; and if, before or during the inquiry he is informed that any person, whether within or without the local limits of his jurisdiction, can give evidence or produce any document material

thereto, may issue a summons requiring him to attend and give evidence or produce such document on the inquest.

Any person disobeying such summons shall be deemed to have committed an offence under S. 174, S. 175 or S. 176 of the Indian Penal Code, as the case may be.

For the purpose of causing prisoners to be brought up to give evidence the Coroner shall be deemed a Criminal Court within the meaning of Part IX of the Prisoners' Act, 1900.

18. *Post-mortem examinations: Fees to medical witnesses.*—The Coroner may direct the performance of a post-mortem examination, with or without an analysis of the contents of the stomach or intestines, by any medical witness summoned to attend the inquest; and every medical witness other than the Chemical Examiner to Government, shall be entitled to such reasonable remuneration as the Coroner thinks fit.

19. *Evidence to be on oath: Evidence on behalf of accused.*—All evidence given under this Act shall be on oath, and the Coroner shall be bound to receive evidence on behalf of the party (if any) accused of causing the death of the deceased person.

Questions suggested by jury.—After each witness has been examined the Coroner shall enquire whether the jury wish any further questions to be put to the witness; and if the jury wish that any such questions should be put, the Coroner shall put them accordingly.

20. *Coroner to take down evidence in writing.*—The Coroner shall commit to writing the material parts of the evidence given to the jury, and shall read or cause to be read over such parts to the witness, and then procure his signature thereto.

Any witness refusing so to sign shall be deemed to have committed an offence under S. 180 of the Indian Penal Code. Every such deposition shall be subscribed by the Coroner. For the purpose of S. 26 of the Indian Evidence Act, a Coroner shall be deemed to be a Magistrate.

22. *Coroner to sum up to jury.*—When all the witnesses have been examined, the Coroner shall sum up the evidence to the jury, and the jury shall then consider their verdict.

23. *Coroner to draw up inquisition.*—When the verdict is delivered, the Coroner shall draw up the inquisition according to the finding of the jury, or when the jury is not unanimous, according to the opinion of the majority.

24. *Contents of inquisitions.*—Every inquisition under this Act shall be signed by the Coroner with his name and style of office and by the jurors, and shall set forth—(1) where, when, and before whom, the inquisition is holden, (2) who the deceased is, (3) where his body lies, (4) the names of the jurors, and that they present the inquisition upon oath, (5) where, when, and by what means, the deceased came by his death, and (6) if his death was occasioned by the criminal act of another who is guilty thereof. If the name of the deceased be unknown, he may be described as a certain person to the jurors unknown.

25. *Procedure where death is found due to an act amounting to an offence.*—When the jury or a majority of the jury find that the death of the deceased person was occasioned by an act which amounts to an offence under any law in force in British India, the Coroner shall immediately after the inquest forward a copy of the inquisition, together with the names and addresses of the witnesses, to the Commissioner of Police.

26. *Power to arrest and commit for trial.*—The Coroner may also, where the verdict justifies him in so doing, issue his warrant for the apprehension of the person who is found to have caused the death of the deceased person, and send him forthwith to a Magistrate empowered to commit him for trial.

28. *Warrant for burial.*—When the proceedings are closed, or before, if it be necessary to adjourn the inquest, the Coroner shall give his warrant for the burial of the body on which the inquest has been taken.

38. *Power to appoint deputy.*—Every Coroner may, from time to time with the previous sanction of the Local Government, appoint by writing under his hand, a proper person to act for him as his deputy in the holding of inquests. All inquests taken and other acts done by any such deputy, under or by virtue of any such appointment, shall be deemed to be the acts of the Coroner appointing him: Provided that no such deputy shall act for any such Coroner except during the illness of the said Coroner, or during his absence for any lawful and reasonable cause.

SEC. 176. INQUIRY BY MAGISTRATE INTO CAUSE OF DEATH.—(1) When any person dies while in the custody of the police, the nearest Magistrate empowered to hold inquests shall, and, in any other case mentioned in Section 174, clauses (a), (b) and (c) of sub-section (1), any Magistrate so empowered may, hold an inquiry into the cause of death, either instead of, or in addition to, the investigation held by the police-officer; and, if he does so, he shall have all the powers in conducting it which he would have in holding an inquiry into an offence. The Magistrate holding such an inquiry shall record the evidence taken by him in connection therewith in any of the manners hereinafter prescribed according to the circumstances of the case.

(2) *Power to disinter corpses.*—Whenever such Magistrate considers it expedient to make an examination of the dead body of any person who has already been interred, in order to discover the cause of his death, the Magistrate may cause the body to be disinterred and examined.

The following two sections, *viz.*, 243 and 244 are meant for the trial of summons cases by Magistrates:—

SEC. 243. CONVICTION ON ADMISSION OF TRUTH OF ACCUSATION.—If the accused admits that he has committed the offence of which he is accused, his admission shall be recorded as nearly as possible in the words used by him; and, if he shows no sufficient cause why he should not be convicted, the Magistrate may convict him accordingly.

SEC. 244. PROCEDURE WHEN NO SUCH ADMISSION IS MADE.—(1) If the Magistrate does not convict the accused under the preceding section or if the accused does not make such admission, the Magistrate shall proceed to hear the complaint (if any), and take all such evidence as may be produced in support of the prosecution, and also to hear the accused and take all such evidence as he produces in his defence:

Provided that the Magistrate shall not be bound to hear any person as complainant in any case in which the complaint has been made by a Court.

(2) The Magistrate may, if he thinks fit, on the application of the complainant or accused, issue a summons to any witness directing him to attend or produce any document or other thing.

(3) The Magistrate may, before summoning any witness on such application require that his reasonable expenses, incurred in attending for the purposes of the trial, be deposited in Court.

The following section, *viz.*, 257 is meant for the trial of warrant cases by Magistrates:—

SEC. 257. PROCESS FOR COMPELLING PRODUCTION OF EVIDENCE AT THE INSTANCE OF ACCUSED.—(1) If the accused, after he has entered upon his defence, applies to the Magistrate to issue any process for compelling the attendance of any witness for the purpose of examination or cross-examination, or the production of any document or other thing the Magistrate shall issue such process unless he considers that such application should be refused on the ground that it is made for the purpose of vexation or delay or for defeating the ends of justice. Such grounds shall be recorded by him in writing: Provided that, when the accused has cross-examined or had the opportunity of cross-examining any witness after the charge is framed, the attendance of such witness shall not be compelled under this section, unless the Magistrate is satisfied that it is necessary for the purposes of justice.

(2) The Magistrate may, before summoning any witness on such application, require that his reasonable expenses incurred in attending for the purposes of the trial be deposited in Court.

SEC. 267. TRIALS BEFORE HIGH COURT TO BE BY JURY.—All trials under this Chapter before a High Court shall be by jury; and, notwithstanding anything herein contained, in all criminal cases transferred to a High Court under this Code or under the Letters Patent of any High Court established under the Indian High Court Act, 1861, or the Government of India Act, 1915, the trial may, if the High Court so directs, be by jury.

SEC. 268. TRIALS BEFORE COURT OF SESSION TO BE BY JURY OR WITH ASSESSORS.—All trials before a Court of Session shall be either by jury, or with the aid of assessors.

SEC. 269. LOCAL GOVERNMENT MAY ORDER TRIALS BEFORE COURT OF SESSION TO BE BY JURY.—(1) The Local Government may, by order of the Official Gazette, direct that the trial of all offences, or of any particular class of offences, before any Court of Session, shall be by jury in any district and may revoke or alter such order.

(2) The Local Government, by like order, may also declare that, in the case of any district in which the trial of any offence is to be by jury, the trial of such offences shall, if the judge, on application made to him or of his own motion, so directs, be by jurors summoned from a special jury list, and may revoke or alter such order.

(3) When the accused is charged at the same trial with several offences of which some are and some are not triable by jury, he shall be tried by jury for such of those offences as are triable by jury, and by the Court of Session, with the aid of the jurors as assessors, for such of them as are not triable by jury.

SEC. 274. NUMBER OF JURY.—In trials before the High Court the jury shall consist of nine persons.

In trials by jury before the Court of Session the jury shall consist of such uneven number, not being less than five, or more than nine, as the Local Government, by order applicable to any particular district or to any particular class of offences in that district, may direct:

Provided that where an accused person is charged with an offence punishable with death, the jury shall consist of not less than seven persons and, if practicable, of nine persons.

SEC. 275. JURY FOR TRIAL OF EUROPEAN AND INDIAN BRITISH SUBJECTS AND OTHERS.—(1) In a trial by jury before the High Court or Court of Session of a person who has been found under the provisions of this Code to be an European or Indian British Subject, a majority of the jury shall, if such person before the first juror is called and accepted so requires, consist, in the case of an European British Subject, of persons who are Europeans or Americans and, in the case of an Indian British Subject, of Indians. (2) In any such trial by jury of a person who has been found under the provisions of this Code to be an European (other than an European British Subject) or an American, a majority of the jury shall, if practicable and if such European or American before the first juror is called and accepted so requires, consist of persons who are Europeans or Americans.

SEC. 276. JURORS TO BE CHOSEN BY LOT.—The jurors shall be chosen by lot from the persons summoned to act as such, in such manner as the High Court may from time to time by rule direct:

Provided that—

Firstly, pending the issue under this section of rules for any Court, the practice now prevailing in such Court in respect to the choosing of jurors shall be followed;

Secondly, in case of a deficiency of persons summoned, the number of jurors required may, with the leave of the Court, be chosen from such other persons as may be present;

Thirdly, in a trial before any High Court in the town which is the usual place of sitting of such High Court,

(*a*) if the accused person is charged with having committed an offence punishable with death, or

(b) if in any other case a Judge of the High Court so directs, the jurors shall be chosen from the special jury list hereinafter prescribed ; and

Fourthly, in any district for which the Local Government has declared that the trial of certain offences may be by special jury, the jurors, shall, in any case in which the Judge so directs, be chosen from the special jury list prescribed in Section 325.

SEC. 280. FOREMAN OF JURY.—(1) When the jurors have been chosen, they shall appoint one of their number to be foreman.

(2) The foreman shall preside in the debates of the jury, deliver the verdict of the jury, and ask any information from the Court that is required by the jury or any of the jurors.

(3) If a majority of the jury do not, within such time as the Judge thinks reasonable, agree in the appointment of a foreman, he shall be appointed by the Court.

SEC. 284. ASSESSORS HOW CHOSEN.—When the trial is to be held with the aid of assessors, not less than three and, if practicable, four shall be chosen, from the persons summoned to act as such.

SEC. 305. VERDICT IN HIGH COURT WHEN TO PREVAIL.—(1) When in a case tried before a High Court the jury are unanimous in their opinion, or when as many as six are of one opinion and the Judge agrees with them, the Judge shall give judgment in accordance with such opinion.

(2) When in any such case the jury are satisfied that they will not be unanimous, but six of them are of one opinion, the foreman shall so inform the Judge.

(3) If the Judge disagrees with the majority, he shall at once discharge the jury.

(4) If there are not so many as six who agree in opinion, the Judge shall, after the lapse of such time as he thinks reasonable, discharge the jury.

SEC. 306. VERDICT IN COURT OF SESSION WHEN TO PREVAIL.- (1) When in a case tried before the Court of Session the Judge does not think it necessary to express disagreement with the verdict of the jurors or of a majority of the jurors, he shall give judgment accordingly.

(2) If the accused is acquitted, the Judge shall record judgment of acquittal. If the accused is convicted, the Judge shall, unless he proceeds in accordance with the provisions of Section 562, pass sentence on him according to law.

SEC. 307. PROCEDURE WHERE SESSIONS JUDGE DISAGREES WITH VERDICT.—(1) If in any such case the Judge disagrees with the verdict of the jurors, or a majority of the jurors, on all or any of the charges on which any accused person has been tried, and is clearly of opinion that it is necessary for the ends of justice to submit the case in respect of such accused to the High Court, he shall submit the case accordingly, recording the grounds of his opinion, and when the verdict is one of acquittal, stating the offence which he considers to have been committed and in such case, if the accused is further charged under the provisions of Section 310, shall proceed to try him on such charge as if such verdict had been one of conviction.

(2) Whenever the Judge submits a case under this section, he shall not record judgment of acquittal or of conviction on any of the charges on which such accused has been tried, but he may either remand such accused to custody or admit him to bail.

(3) In dealing with the case so submitted the High Court may exercise any of the powers which it may exercise on an appeal, and subject thereto it shall after considering the entire evidence and after giving due weight to the opinions of the Sessions Judge and the jury, acquit or convict such accused of any offence of which the jury could have convicted him upon the charge framed and placed before it; and, if it convicts him, may pass such sentence as might have been passed by the Court of Session.

SEC. 309. DELIVERY OF OPINIONS OF ASSESSORS.—(1) When, in a case tried with the aid of assessors, the case for the defence and the prosecutor's reply (if any) are concluded, the Court may sum up the evidence for the prosecution and defence, and shall then require each of the assessors to state his opinion orally, on all the charges on which the accused has been tried and shall record such opinion, and for that purpose may ask the assessors such questions as are necessary to ascertain what their opinions are. All such questions and the answers to them shall be recorded.

(2) The Judge shall then give judgment, but in doing so shall not be bound to conform to the opinions of the assessors.

(3) If the accused is convicted, the Judge shall, unless he proceeds in accordance with the provisions of Section 562, pass sentence on him according to law.

SEC. 312. NUMBER OF SPECIAL JURORS.—The High Court may prescribe the number of persons whose names shall be entered at any one time in the special jurors' list :

Provided that no definite number of Europeans or Americans or of Indians shall be so prescribed.

SEC. 320. EXEMPTIONS.—The following persons are exempt from liability to serve as jurors or assessors, namely :—

(*a*) officers in civil employ superior in rank to a District Magistrate ;

(*aa*) members[1] of either Chamber of the Indian Legislature and members of a Legislative Council constituted under the Government of India Act ;

(*b*) salaried Judges ;

(*c*) Commissioners and Collectors of Revenue or Customs ;

(*d*) police-officers and persons engaged in the Preventive Service in the Customs Department ;

(*e*) persons engaged in the collection of the revenue whom the Collector thinks fit to exempt on the ground of official duty ;

(*f*) persons actually officiating as priests or ministers of their respective religions ;

(*g*) persons in His Majesty's Army, Navy or Air Force, except when, by any law in force for the time being, they are specially made liable to serve as jurors or assessors ;

(*h*) surgeons and others who openly and constantly practise the medical profession ;

(*i*) legal practitioners (as defined by the Legal Practitioners' Act, 1879) in actual practice ;

(*j*) persons employed in the Post-Office and Telegraph Departments ;

(*k*) persons exempted from personal appearance in Court under the provisions of Civil Procedure Code, Section 640 and 641 ;

(*l*) other persons exempted by the Local Government from liability to serve as jurors or assessors.

SEC. 345. COMPOUNDING OFFENCES.—(1) The offences punishable under the sections of the Indian Penal Code, such as 323, 334[2] are compoundable by the person to whom the hurt is caused.

(2) The offences of causing hurt, and grievous hurt, punishable under Section 324, Section 325, Section 335, Section 337, or Section 338[3] of the Indian Penal Code, may, with the permission of the Court before which any prosecution for such is pending, be compounded by the person to whom the hurt has been caused.

---

1. *This clause has been added by the Legislative Members Exemption Act XXIII of* 1925.

2. *For original wording and fuller detail of other sections of the Indian Penal Code* vide *the Criminal Procedure Act*, 1923.

3. *Ibid.*

(3) When any offence is compoundable under this section, the abetment of such offence or an attempt to commit such offence (when such attempt is itself an offence) may be compounded in like manner.

(4) When the person who would otherwise be competent to compound an offence under the section is under the age of eighteen years or is an idiot or a lunatic, any person competent to contract on his behalf may, with the permission of the Court, compound such offence.

(5) When the accused has been committed for trial or when he has been convicted and an appeal is pending, no composition for the offence shall be allowed without the leave of the Court to which he is committed, or, as the case may be, before which the appeal is to be heard.

(5-A) A High Court acting in the exercise of its powers of revision under Section 439 may allow any person to compound any offence which he is competent to compound under this section.

(6) The composition of an offence under this section shall have the effect of an acquittal of the accused with whom the offence has been compounded.

(7) No offence shall be compounded except as provided by this section.

SEC. 374. SENTENCE OF DEATH TO BE SUBMITTED BY COURT OF SESSION.—When the Court of Session passes sentence of death, the proceedings shall be submitted to the High Court, and the sentence shall not be executed unless it is confirmed by the High Court.

SEC. 382. POSTPONEMENT OF CAPITAL SENTENCE ON PREGNANT WOMAN.—If a woman sentenced to death is found to be pregnant, the High Court shall order the execution of the sentence to be postponed, and may, if it thinks fit, commute the sentence to transportation for life.

The pregnancy of a woman shall be certified by a civil surgeon.—*Bombay Gazette,* 1879, *p.* 471.

SEC. 390. TIME AND PLACE OF EXECUTION OF SENTENCE OF WHIPPING ONLY.—When the accused is sentenced to whipping only, the sentence shall, subject to the provisions of Section 391, be executed at such place and time as the Court may direct.

SEC. 391. EXECUTION OF SENTENCE OF WHIPPING ONLY, OR OF WHIPPING IN ADDITION TO IMPRISONMENT.—(1) When the accused—

(*a*) is sentenced to whipping only and furnishes bail to the satisfaction of the Court for his appearance at such time and place as the Court may direct, or

(*b*) is sentenced to whipping in addition to imprisonment,

the whipping shall not be inflicted until fifteen days from the date of the sentence, or if an appeal is made within that time until the sentence is confirmed by the Appellate Court, but the whipping shall be inflicted as soon as practicable after the expiry of the fifteen days, or, in case of an appeal, as soon as practicable after the receipt of the order of the Appellate Court confirming the sentence.

(2) The whipping shall be inflicted in the presence of the officer in charge of the jail, unless the Judge or Magistrate orders it to be inflicted in his own presence.

(3) No accused person shall be sentenced to whipping in addition to imprisonment when the term of imprisonment to which he is sentenced is less than three months.

SEC. 392. MODE OF INFLICTING PUNISHMENT.—(1) In the case of a person, of or over sixteen years of age, whipping shall be inflicted with a light rattan not less than half-an-inch in diameter, in such mode, and on such part of the person as the Local Government directs; and in the case of a person under sixteen years of age, it shall be inflicted in such mode, and on such part of the person, and with such instrument, as the Local Government directs.

(2) In no case shall such punishment exceed thirty stripes and in the case of a person under sixteen years of age it shall not exceed fifteen stripes.

SEC. 393. WHIPPING NOT TO BE EXECUTED BY INSTALMENTS AND EXEMPTIONS.—No sentence of whipping shall be executed by instalments; and none of the following persons shall be punishable with whipping namely:—

(*a*) Females;

(*b*) Males sentenced to death, or to transportation or to penal servitude, or to imprisonment for more than five years;

(*c*) Males whom the Court considers to be more than forty-five years of age.

SEC. 394. WHIPPING NOT TO BE INFLICTED IF OFFENDER NOT IN A FIT STATE OF HEALTH.—(1) The punishment of whipping shall not be inflicted unless a medical officer, if present, certifies, or if there is not a medical officer present, unless it appears to the Magistrate or officer present, that the offender is in a fit state of health to undergo such punishment.

(2) If, during the execution of a sentence of whipping, a medical officer certifies or it appears to the Magistrate or officer present, that the offender is not in a fit state of health to undergo the remainder of the sentence, the whipping shall be finally stopped.

SEC. 395. PROCEDURE IF PUNISHMENT CANNOT BE INFLICTED UNDER SECTION 394.—(1) In any case in which, under Section 394, a sentence of whipping is, wholly or partially, prevented from being executed, the offender shall be kept in custody till the Court which passed the sentence can revise it; and the said Court may, at its discretion either remit such sentence, or sentence the offender in lieu of whipping, or in lieu of so much of the sentence of whipping as was not executed, to imprisonment for any term not exceeding twelve months or to a fine not exceeding five hundred rupees, which may be in addition to any other punishment to which he may have been sentenced for the same offence.

(2) Nothing in this section shall be deemed to authorise the Court to inflict punishment for a term or a fine of an amount exceeding that to which the accused is liable by law, or that which the said Court is competent to inflict.

SEC. 399. CONFINEMENT OF YOUTHFUL OFFENDERS IN REFORMATORIES.—(1) When any person under the age of fifteen years is sentenced by any Criminal Court to imprisonment for any offence, the Court may direct that such person, instead of being imprisoned in a criminal jail, shall be confined in any reformatory established by the Local Government as a fit place for confinement, in which there are means of suitable discipline and of training in some branch of useful industry, or which is kept by a person willing to obey such rules as the Local Government prescribes with regard to the discipline and training of persons confined therein.

(2) All persons confined under this section shall be subjected to the rules so prescribed.

(3) This section shall not apply to any place in which the Reformatory Schools Act, 1897, is for the time being in force.

LUNATICS

SEC. 464. PROCEDURE IN CASE OF ACCUSED BEING LUNATIC.—(1) When a Magistrate holding an inquiry or a trial has reason to believe that the accused is of unsound mind and consequently incapable of making his defence, the Magistrate shall inquire into the fact of such unsoundness, and shall cause such person to be examined by the Civil Surgeon of the district or such other medical officer as the Local Government directs, and thereupon shall examine such surgeon or other officer as a witness, and shall reduce the examination to writing.

(1-A) Pending such examination and inquiry, the Magistrate may deal with the accused in accordance with the provisions of Section 466.

(2) If such Magistrate is of opinion that the accused is of unsound mind and consequently incapable of making his defence, he shall record a finding to that effect and shall postpone further proceedings in the case.

SEC. 465. PROCEDURE IN CASE OF PERSON COMMITTED BEFORE COURT OF SESSION OR HIGH COURT BEING LUNATIC.—(1) If any person committed for trial before a Court of Session or a High Court, appears to the Court at his trial to be of unsound mind and consequently incapable of making his defence, the jury or the Court with the aid of assessors, shall, in the first instance, try the fact of such unsoundness and incapacity, and if the Jury or Court, as the case may be, is satisfied of the fact, the Judge shall record a finding to that effect and shall postpone further proceedings in the case and the jury, if any, shall be discharged.

(2) The trial of the fact of unsoundness of mind and incapacity of the accused shall be deemed to be part of his trial before the Court.

SEC. 466. RELEASE OF LUNATIC PENDING INVESTIGATION OR TRIAL. —(1) Whenever an accused person is found to be of unsound mind and incapable of making his defence, the Magistrate or Court, as the case may be, whether the case is one in which bail may be taken or not, may release him on sufficient security being given that he shall be properly taken care of and shall be prevented from doing injury to himself or to any other person, and for his appearance when required before the Magistrate or Court or such officer as the Magistrate or Court appoints in this behalf.

(2) If the case is one in which, in the opinion of the Magistrate or Court, bail should not be taken, or if sufficient security is not given, the Magistrate or Court, as the case may be, shall order the accused to be detained in safe custody in such place and manner as he or it may think fit, and shall report the action taken to the Local Government:

Provided that no order for the detention of the accused in a lunatic asylum shall be made otherwise than in accordance with such rules as the Local Government may have made under the Indian Lunacy Act, 1912.

SEC. 467. RESUMPTION OF INQUIRY OR TRIAL.—(1) Whenever an inquiry or a trial is postponed under Section 464 or Section 465, the Magistrate or Court, as the case may be, may, at any time, resume the inquiry or trial of and require the accused to appear or be brought before such Magistrate or Court.

(2) When the accused has been released under Section 466, and the sureties for his appearance produce him to the officer whom the Magistrate or Court appoints in this behalf, the certificate of such officer that the accused is capable of making his defence shall be receivable in evidence.

SEC. 468. PROCEDURE ON ACCUSED APPEARING BEFORE MAGISTRATE OR COURT.—(1) If, when the accused appears or is again brought before the Magistrate or the Court, as the case may be, the Magistrate or Court considers him capable of making his defence, the inquiry or trial shall proceed.

(2) If the Magistrate or Court considers the accused to be still incapable of making his defence, the Magistrate or Court shall again act according to the provisions of Section 464 or Section 465 as the case may be, and if the accused is found to be of unsound mind and incapable of making his defence, shall deal with such accused in accordance with the provisions of Section 466.

SEC. 469. WHEN ACCUSED APPEARS TO HAVE BEEN INSANE.—When the accused appears to be of sound mind at the time of inquiry or trial, and the Magistrate is satisfied from the evidence given before him that there is reason to believe that the accused committed an act which, if he had been of sound mind, would have been an offence, and that he was, at the time when the act was committed, by reason of unsoundness of mind, incapable of knowing the nature of the act, or that it was wrong or contrary to law, the Magistrate shall proceed with the case, and, if the accused ought to be committed to the Court of Session or High Court, send him for trial before the Court of Session or High Court, as the case may be.

SEC. 470. JUDGMENT OF ACQUITTAL ON GROUND OF LUNACY.—Whenever any person is acquitted upon the ground that, at the time at which he is alleged to have committed an offence, he was, by reason of unsoundness of mind, incapable of knowing the nature of the act alleged as constituting the offence, or that it was wrong or contrary to law, the finding shall state specifically whether he committed the act or not.

SEC. 471. PERSON ACQUITTED ON SUCH GROUND TO BE DETAINED IN SAFE CUSTODY.—(1) Whenever the finding states that the accused person committed the act alleged, the Magistrate or Court before whom or which the trial has been held, shall, if such act would, but for the incapacity found, have constituted an offence, order such person to be detained in safe custody in such place and manner as the Magistrate or Court thinks fit, and shall report the action taken to the Local Government :

Provided that no order for the detention of the accused in a lunatic asylum shall be made otherwise than in accordance with such rules as the Local Government may have made under the Indian Lunacy Act, 1912.

(2) The Local Government may empower the officer-in-charge of the jail in which a person is confined under the provisions of Section 466 or this section, to discharge all or any of the functions of the Inspector-General of Prisons under Section 473 or Section 474.

SEC. 472. LUNATIC PRISONERS TO BE VISITED BY INSPECTOR-GENERAL.—Repealed by Act IV of 1912. Cf. Section 30 of Act IV (Lunacy) of 1912—(1) When any person is confined under the provisions of Section 466 or Section 471 of the Code of Criminal Procedure, 1898, the Inspector-General of Prisons, if such person is confined in a jail or the visitors of the asylum or any two of them, if he is confined in an asylum, may visit him in order to ascertain his state of mind ; and he shall be visited once at least in every six months by such Inspector-General or by two such visitors as aforesaid ; and such Inspector-General or visitors shall make a special report as to the state of mind of such person to the authority under whose order he is confined.

(2) The Local Government may empower the officer-in-charge of the jail in which such person may be confined to discharge all or any of the functions of the Inspector-General under sub-section (1).

SEC. 473. PROCEDURE WHERE LUNATIC PRISONER IS REPORTED CAPABLE OF MAKING HIS DEFENCE.—If such person is detained under the provisions of Section 466 and in the case of a person detained in a jail, the Inspector-General of Prisons, or, in the case of a person detained in a lunatic asylum, the visitors of such asylum or any two of them shall certify that, in his or their opinion, such person is capable of making his defence, he shall be taken before the Magistrate or Court as the case may be, at such time as the Magistrate or Court appoints, and the Magistrate or Court shall deal with such person under the provisions of Section 468 ; and the certificate of such Inspector-General or visitors as aforesaid shall be receivable as evidence.

SEC. 474. PROCEDURE WHERE LUNATIC DETAINED UNDER SECTION 466 OR 471 IS DECLARED FIT TO BE RELEASED.—(1) If such person is detained under the provisions of Section 466 or Section 471, and such Inspector-General or visitors shall certify that, in his or their judgment, he may be released without danger of his doing injury to himself or to any other person, the Local Government may thereupon order him to be released or to be detained in custody, or to be transferred to a public lunatic asylum, if he has not been already sent to such an asylum ; and in case it orders him to be transferred to an asylum, may appoint a Commission consisting of a judicial and two medical officers.

(2) Such Commission shall make formal inquiry into the state of mind of such person, taking such evidence as is necessary, and shall report to the Local Government, which may order his release or detention as it thinks fit.

SEC. 475. DELIVERY OF LUNATIC TO CARE OF RELATIVE OR FRIEND. —(1) Whenever any relative or friend of any person detained under the provisions

of Section 466 or Section 471 desires that he shall be delivered to his care and custody, the Local Government may, upon the application of such relative or friend and on his giving security to the satisfaction of such Local Government that the person delivered shall—

(*a*) be properly taken care of and prevented from doing injury to himself or to any other person, and

(*b*) be produced for the inspection of such officer, and at such times and places, as the Local Government may direct, and

(c) in the case of a person detained under Section 466, be produced when required before such Magistrate or Court,

order such person to be delivered to such relative or friend.

(2) If the person so delivered is accused of any offence the trial of which has been postponed by reason of his being of unsound mind and incapable of making his defence, and the inspecting officer referred to in sub-section (1), clause (b) certifies at any time to the Magistrate or Court that such person is capable of making his defence, such Magistrate or Court shall call upon the relative or friend to whom such accused was delivered to produce him before the Magistrate or Court; and upon such production, the Magistrate or Court shall proceed in accordance with the provisions of Section 468, and the certificate of the inspecting officer shall be receivable as evidence.

SPECIAL RULES OF EVIDENCE

SEC. 509. DEPOSITION OF MEDICAL WITNESS.—(1) The deposition of a Civil Surgeon or other medical witness, taken and attested by a Magistrate in the presence of the accused, or taken on Commission under Chapter XL, may be given in evidence in any inquiry, trial or other proceeding under this Code, although the deponent is not called as a witness.

(2) The Court may, if it thinks fit, summon and examine such deponent as to the subject-matter of this deposition.

Note.—In order to ensure that the medical officer's deposition may, in all cases, be admissible under this section, the Magistrate must sign at the foot of it a certificate in the following form :—" The foregoing deposition was taken in the presence of the accused (name), who had an opportunity of cross-examining the witness. The deposition was explained to the accused, and was attested by me in his presence." This is, of course, necessary when the deposition is taken in an inquiry preparatory to the commitment to the Sessions. Where the attestation is wanting, the Sessions Judge should summon such witness to give his evidence.

SEC. 510. REPORT OF CHEMICAL EXAMINER.—Any document purporting to be a report under the hand of any Chemical Examiner or Assistant Chemical Examiner to Government, upon any matter or thing duly submitted to him for examination or analysis and report in the course of any proceeding under this Code, may be used as evidence in any inquiry, trial or other proceeding under this Code.

SEC. 512. RECORD OF EVIDENCE IN ABSENCE OF ACCUSED.—(1) If it is proved that an accused person has absconded, and that there is no immediate prospect of arresting him, the Court competent to try or commit for trial such person for the offence complained of may, in his absence, examine the witnesses (if any) produced on behalf of the prosecution, and record their depositions. Any such deposition may, on the arrest of such person, be given in evidence against him on the inquiry into, or trial for, the offence with which he is charged, if the deponent is dead or incapable of giving evidence or his attendance cannot be procured without an amount of delay, expense or inconvenience which, under the circumstances of the case, would be unreasonable.

(2) If it appears that an offence punishable with death or transportation has been committed by some person or persons unknown, the High Court may direct that any Magistrate of the first class shall hold an inquiry and examine any witnesses who can give evidence concerning the offence. Any depositions so taken

may be given in evidence against any person who is subsequently accused of the offence, if the deponent is dead or is incapable of giving evidence or beyond the limits of British India.

SEC. 540. POWER TO SUMMON MATERIAL WITNESS, OR EXAMINE PERSON PRESENT.—Any Court may, at any stage of any inquiry, trial, or other proceeding under this Code, summon any person as a witness, or examine any person in attendance, though not summoned as a witness or recall and re-examine any person already examined; and the Court shall summon and examine or recall and re-examine any such person if his evidence appears to it essential to the just decision of the case.

SEC. 544. EXPENSES OF COMPLAINANTS AND WITNESSES.—Subject to any rules made by the Local Government, any Criminal Court may, if it thinks fit, order payment, on the part of Government, of the reasonable expenses of any complainant or witness attending for the purpose of any inquiry, trial or proceeding before such Court under this Code.

APPENDIX VII

THE INDIAN PENAL CODE

SEC. 32. WORDS REFERRING TO ACTS INCLUDE ILLEGAL OMISSION.—In every part of this code, except where a contrary intention appears from the context, words which refer to acts done extend also to illegal omissions. (An act includes illegal omissions, which must be intentional and conducive to bad or harmful result).

34. ACT DONE BY SEVERAL PERSONS IN FURTHERANCE OF COMMON INTENTION.—When a criminal act is done by several persons, in furtherance of the common intention of all, each of such persons is liable for that act in the same manner, as if it were done by him alone.

44. INJURUY.—The word injury denotes any harm whatever illegally caused to any person in body, mind, reputation, or property.

51. OATH.—The word "oath" includes a solemn affirmation substituted by law for an oath, and any declaration required or authorised by law to be made before a public servant or to be used for the purpose of proof, whether in a Court of Justice or not.

52. GOOD FAITH.—Nothing is said to be done or believed in good faith which is done or believed without due care and attention.

53. PUNISHMENTS.—The punishments to which offenders are liable under the provisions of this code are—

First,—Death ;

Secondly,—Transportation ;

Thirdly,—Penal servitude ;

Fourthly,—Imprisonment, which is of two descriptions, namely :—

(1) Rigorous, that is, with hard labour.

(2) Simple.

Fifthly,—Forfeiture of property ;

Sixthly,—Fine ;

Seventhly,—Whipping added by the Whipping Act as in the case of a "juvenile offender" who is under sixteen years.

80. ACCIDENT IN DOING A LAWFUL ACT.—Nothing is an offence which is done by accident or misfortune, and without any criminal intention or knowledge in the doing of a lawful act in a lawful manner by lawful means and with proper care and caution.

81. ACT LIKELY TO CAUSE HARM, BUT DONE WITHOUT CRIMINAL INTENT, AND TO PREVENT OTHER HARM.—Nothing is an offence merely by reason of its being done with the knowledge that it is likely to cause harm, if it be done without any criminal intention to cause harm, and in good faith for the purpose of preventing or avoiding other harm to person or property.

82. ACT OF A CHILD UNDER SEVEN YEARS OF AGE.—Nothing is an offence which is done by a child under seven years of age.

83. ACT OF A CHILD ABOVE SEVEN AND UNDER TWELVE, OF IMMATURE UNDERSTANDING.—Nothing is an offence which is done by a child above seven years of age and under twelve, who has not attained sufficient maturity

of understanding to judge of the nature and consequences of his conduct on that occasion.

According to the English law 14 years is the limit instead of twelve ; and it is left to the jury to decide whether the offence was committed by the prisoner and if so, whether at the time of the offence the prisoner had a guilty knowledge that he was doing wrong, if he was indicted for felony between 7 and 14 years of age. In cases of murder an infant may be convicted of the capital punishment, if it appeared to the Court and jury and if it was proved that the infant could discern between good and evil.

Cf. SEC. 130 OF THE INDIAN RAILWAYS ACT (ACT IX OF 1890).—(1) If a minor under the age of twelve years is, with respect to any railway, guilty of any of the acts or omissions mentioned or referred to in any of the four Sections 126, 127, 128 and 129, he shall be deemed notwithstanding anything in Section 82 or Section 83 of the Indian Penal Code, to have committed an offence, and the Court convicting him may, if it thinks fit, direct that the minor, if a male, shall be punished with whipping, or may require the father or guardian of the minor to execute, within such time as the Court may fix, a bond binding himself, in such penalty as the Court directs, to prevent the minor from being again guilty of those acts or omissions.

(2) The amount of the bond, if forfeited, shall be recoverable by the Court as if it were a fine imposed by itself.

(3) If a father or guardian fails to execute a bond under sub-section (1) within the time fixed by the Court, he shall be punished with fine which may extend to fifty rupees.

Offences under sections—

126.—Maliciously wrecking or attempting to wreck a train.

127.—Maliciously hurting or attempting to hurt persons travelling by railway.

128.—Endangering safety of persons travelling by railway by wilful act or omission.

129.—Endangering safety of persons travelling by railway by way of rash or negligent act or omission.

84. ACT OF A PERSON OF UNSOUND MIND.—Nothing is an offence which is done by a person who, at the time of doing it, by reason of unsoundness of mind, is incapable of knowing the nature of the act, or that he is doing what is either wrong or contrary to law.

85. ACT OF A PERSON INCAPABLE OF JUDGMENT BY REASON OF INTOXICATION CAUSED AGAINST HIS WILL.—Nothing is an offence which is done by a person who, at the time of doing it, is, by reason of intoxication, incapable of knowing the nature of the act, or that he is doing what is either wrong or contrary to law ; provided that the thing which intoxicated him was administered to him without his knowledge or against his will.

86. OFFENCE REQUIRING A PARTICULAR INTENT COMMITTED BY ONE WHO IS INTOXICATED.—In cases where an act done is not an offence unless done with a particular knowledge or intent, a person who does the act in a state of intoxication shall be liable to be dealt with as if he had not been intoxicated unless the thing which intoxicated him was administered to him without his knowledge or against his will.

87. ACT NOT INTENDED AND NOT KNOWN TO BE LIKELY TO CAUSE DEATH OR GRIEVOUS HURT DONE BY CONSENT.—Nothing which is not intended to cause death or grievous hurt, and which is not known by the doer to be likely to cause death or grievous hurt, is an offence by reason of any harm which it may cause, or be intended by the doer to cause, to any person, above eighteen years of age, who has given consent, whether express or implied, to suffer that harm ; or by reason of any harm which it may be known by the doer to be likely to cause to any such person who has consented to take the risk of that harm.

88. ACT NOT INTENDED TO CAUSE DEATH, DONE BY CONSENT IN GOOD FAITH FOR PERSON'S BENEFIT.—Nothing which is not intended to cause

death, is an offence by reason of any harm which it may cause, or be intended by the doer to cause, or be known by the doer to be likely to cause, to any person for whose benefit it is done in good faith, and who has given a consent, whether express or implied, to suffer that harm, or to take the risk of that harm.

89. ACT DONE IN GOOD FAITH FOR THE BENEFIT OF CHILD OR INSANE PERSON, BY OR BY CONSENT OF GUARDIAN.—Nothing which is done in good faith for the benefit of a person under twelve years of age, or of unsound mind, by or by consent, either express or implied, of the guardian or other person having lawful charge of that person, is an offence by reason of any harm which it may cause, or be intended by the doer to cause, or be known by the doer to be likely to cause, to that person :

Provided—

First.—That this exception shall not extend to the intentional causing of death, or to the attempting to cause death ;

Secondly.—That this exception shall not extend to the doing of anything which the person doing it knows to be likely to cause death, for any purpose other than the preventing of death or grievous hurt, or the curing of any grievous disease or infirmity ;

Thirdly.—That this exception shall not extend to the voluntary causing of grievous hurt, or to the attempting to cause grievous hurt, or the curing of any grievous disease or infirmity ;

Fourthly.—That this exception shall not extend to the abetment of any offence, to the committing of which offence it would not extend.

90. CONSENT KNOWN TO BE GIVEN UNDER FEAR OR MISCONCEPTION. —A consent is not such a consent as is intended by any section of this code, if the consent is given by a person under fear of injury, or under a misconception of fact, and if the person doing the act knows, or has reason to believe, that the consent was given in consequence of such fear or misconception ; if the consent is given by a person who, from unsoundness of mind or intoxication, is unable to understand the nature and consequence of that to which he gives his consent ; or, unless the contrary appears from the context, if the consent is given by a person who is under twelve years of age.

91. EXCLUSION OF ACTS WHICH ARE OFFENCES INDEPENDENTLY OF HARM CAUSED.—The exceptions in Sections 87, 88 and 89 do not extend to acts which are offences independently of any harm which they may cause or be intended to cause, or be known to be likely to cause, to the person giving the consent, or on whose behalf the consent is given.

92. ACT DONE IN GOOD FAITH FOR THE BENEFIT OF A PERSON WITHOUT CONSENT.—Nothing is an offence by reason of any harm which it may cause to a person for whose benefit it is done in good faith, even without that person's consent, if the circumstances are such that it is impossible for that person to signify consent, or if that person is incapable of giving consent. and has no guardian or other person in lawful charge of him from whom it is possible to obtain consent in time for the thing to be done with benefit : Provided—

First.—That this exception shall not extend to the intentional causing of death or the attempting to cause death ;

Secondly.—That this exception shall not extend to the doing of any thing which the person doing it knows to be likely to cause death, for any purpose other than the preventing of death or grievous hurt or the curing of any grievous disease or infirmity ;

Thirdly.—That this exception shall not extend to the voluntary causing of hurt, or to the attempting to cause hurt, for any purpose other than the preventing of death or hurt ;

Fourthly.—That this exception shall not extend to the abetment of any offence, to the committing of which offence it would not extend.

176. OMISSION TO GIVE NOTICE OR INFORMATION TO PUBLIC SERVANT BY PERSON LEGALLY BOUND TO GIVE IT.—Whoever, being legally bound to give any notice or to furnish information on any subject to any public servant, as such, intentionally omits to give such notice or to furnish such information in the manner and at the time required by law, shall be punished with simple imprisonment for a term which may extend to one month, or with fine which may extend to five hundred rupees, or with both ; or

if the notice or information required to be given respects the commission of an offence, or is required for the purpose of preventing the commission of an offence, or in order to the apprehension of an offender, with simple imprisonment for a term which may extend to six months, or with fine which may extend to one thousand rupees, or with both.

191. GIVING FALSE EVIDENCE.—Whoever being legally bound by an oath or by any express provision of law to state the truth, or being bound by law to make a declaration upon any subject, makes any statement which is false, and which he either knows or believes to be false, or does not believe to be true, is said to give false evidence.

192. FABRICATING FALSE EVIDENCE.—Whoever causes any circumstance to exist, or makes any false entry in any book or record, or makes any document containing a false statement, intending that such circumstance, false entry, or false statement may appear in evidence in a judicial proceeding, or in a proceeding taken by law before a public servant as such, or before an arbitrator and that such circumstance, false entry, or false statement, so appearing in evidence, may cause any person who in such proceeding is to form an opinion upon the evidence, to entertain an erroneous opinion touching any point material to the result of such proceeding, is said to " fabricate false evidence."

193. PUNISHMENT FOR FALSE EVIDENCE.—Whoever intentionally gives false evidence in any stage of a judicial proceeding, or fabricates false evidence for the purpose of being used in any stage of a judicial proceeding, shall be punished with imprisonment of either description for a term which may extend to seven years, and shall also be liable to fine ;

and whoever intentionally gives or fabricates false evidence in any other case, shall be punished with imprisonment of either description for a term which may extend to three years, and shall also be liable to fine.

Explanation 1.—A trial before a Court-martial is a judicial proceeding.

Explanation 2.—An investigation directed by law preliminary to a proceeding before a Court of Justice, is a stage of a judicial proceeding, though that investigation may not take place before a Court of Justice.

197. ISSUING OR SIGNING FALSE CERTIFICATE.—Whoever issues or signs any certificate required by law to be given or signed, or relating to any fact of which such certificate is by law admissible in evidence, knowing or believing that such certificate is false in any material point, shall be punished in the same manner as if he gave false evidence.

201. CAUSING DISAPPEARANCE OF EVIDENCE OF OFFENCE, OR GIVING FALSE INFORMATION TO SCREEN OFFENDER.—Whoever, knowing or having reason to believe that an offence has been committed, causes any evidence of the commission of that offence to disappear, with the intention of screening the offender from legal punishment, or with that intention gives any information respecting the offence which he knows or believes to be false,

shall, if the offence which he knows or believes to have been committed is punishable with death, be punished with imprisonment of either description for a term which may extend to seven years, and shall also be liable to fine ;

and if the offence is punishable with transportation for life, with imprisonment which may extend to ten years, shall be punished with imprisonment of either description for a term which may extend to three years, and shall also be liable to fine ;

and if the offence is punishable with imprisonment for any term not extending to ten years, shall be punished with imprisonment of the description provided for the offence, for a term which may extend to one-tenth part of the longest term of the imprisonment provided for the offence, or with fine or with both.

202. INTENTIONAL OMISSION TO GIVE INFORMATION OF OFFENCE BY PERSON BOUND TO INFORM.—Whoever, knowing or having reason to believe that an offence has been committed, intentionally omits to give any information respecting that offence which he is legally bound to give, shall be punished with imprisonment of either description for a term which may extend to six months, or with fine, or with both.

284. NEGLIGENT CONDUCT WITH RESPECT TO POISONOUS SUBSTANCE. —Whoever does, with any poisonous substance, any act in a manner so rash or negligent as to endanger human life, or to be likely to cause hurt or injury to any person, or knowingly or negligently omits to take such order, with any poisonous substance in his possession as is sufficient to guard against probable danger to human life from such poisonous substance, shall be punished with imprisonment of either description for a term which may extend to six months, or with fine which may extend to one thousand rupees, or with both.

299. CULPABLE HOMICIDE.—Whoever causes death by doing an act with the intention of causing death, or with the intention of causing such bodily injury as is likely to cause death, or with the knowledge that he is likely by such act to cause death, commits the offence of culpable homicide.

Explanation 1. Any person who causes bodily injury to another who is labouring under a disorder, disease or bodily infirmity, and thereby accelerates the death of that other, shall be deemed to have caused his death.

Explanation 2.—When the death is caused by bodily injury, the person who causes such bodily injury shall be deemed to have caused the death, although by resorting to proper remedies and skilful treatment the death might have been prevented.

Explanation 3.—The causing of the death of a child in the mother's womb is not homicide. But it may amount to culpable homicide to cause the death of a living child, if any part of that child has been brought forth, though the child may not have breathed or been completely born.

300. MURDER.—Except in the cases hereinafter excepted, culpable homicide is murder, if the act by which the death is caused is done with the intention of causing death, or—

Secondly.—If it is done with the intention of causing such bodily injury as the offender knows to be likely to cause the death of the person, to whom the harm is caused, or—

Thirdly.—If it is done with the intention of causing bodily injury to any person and the bodily injury intended to be inflicted is sufficient in the ordinary course of nature to cause death, or—

Fourthly.—If the person committing the act knows that it is so imminently dangerous that it must in all probability cause death or such bodily injury as is likely to cause death and commits such act without any excuse for incurring the risk of causing death or such injury as aforesaid.

Exception 1.—Culpable homicide is not murder if the offender, whilst deprived of the power of self-control by grave and sudden provocation, causes the death of the person who gave the provocation, or causes the death of any other person by mistake or accident.

The above exception is subject to the following provisos :—

First.—That the provocation is not sought or voluntarily provoked by the offender as an excuse for killing or doing harm to any person.

Secondly.—That the provocation is not given by anything done in obedience to the law, or by a public servant in the lawful exercise of the powers of such public servant.

Thirdly.—That the provocation is not given by anything in the lawful exercise of the right of private defence.

Explanation.—Whether the provocation was grave and sudden enough to prevent the offence from amounting to murder is a question of fact.

Exception 2.—Culpable homicide is not murder if the offender, in the exercise in good faith of the right of private defence of person or property, exceeds the power given to him by law and causes the death of the person against whom he is exercising such right of defence without premeditation, and without any intention of doing more harm than is necessary for the purpose of such defence.

Exception 3.—Culpable homicide is not murder if the offender being a public servant or aiding a public servant acting for the advancement of public justice, exceeds the powers given to him by law, and causes death by doing an act which he, in good faith, believes to be lawful and necessary for the due discharge of his duty as such public servant and without ill-will towards the person whose death is caused.

Exception 4.—Culpable homicide is not murder if it is committed without premeditation in a sudden fight in the heat of passion upon a sudden quarrel and without the offender's having taken undue advantage or acted in a cruel or unusual manner.

Explanation.—It is immaterial in such cases which party offers the provocation or commits the first assault.

Exception 5.—Culpable homicide is not murder when the person whose death is caused, being above the age of eighteen years, suffers death or takes the risk of death with his own consent.

N.B.—The law of British India, differing from the law of England, does not regard every case of homicide as *prima facie* murder; it throws on the prosecution the burden of proving a certain intent or knowledge.

301. CULPABLE HOMICIDE BY CAUSING DEATH OF PERSON OTHER THAN PERSON WHOSE DEATH WAS INTENDED.—If a person, by doing anything which he intends or knows to be likely to cause death, commits culpable homicide by causing the death of any person, whose death he neither intends nor knows himself to be likely to cause, the culpable homicide committed by the offender is of the description of which it would have been if he had caused the death of the person whose death he intended or knew himself to be likely to cause.

302. PUNISHMENT FOR MURDER.—Whoever commits murder shall be punished with death, or transportation for life, and shall also be liable to fine.

303. PUNISHMENT FOR MURDER BY LIFE CONVICT.—Whoever, being under sentence of transportation for life, commits murder, shall be punished with death.

304. PUNISHMENT FOR CULPABLE HOMICIDE NOT AMOUNTING TO MURDER.—Whoever commits culpable homicide not amounting to murder, shall be punished with transportation for life, or imprisonment of either description for a term which may extend to ten years, and shall also be liable to fine if the act by which the death is caused is done with the intention of causing death, or of causing such bodily injury as is likely to cause death; or with imprisonment of either description for a term which may extend to ten years, or with fine, or with both, if the act is done with the knowledge that it is likely to cause death, but without any intention to cause death or to cause such bodily injury as is likely to cause death.

304. A. CAUSING DEATH BY NEGLIGENCE.—Whoever causes the death of any person by doing any rash or negligent act not amounting to culpable homicide shall be punished with imprisonment of either description for a term which may extend to two years, or with fine, or with both.

305. ABETMENT OF SUICIDE OF CHILD OR INSANE PERSON.—If any person under eighteen years of age, any insane person, any delirious person, any idiot, or any person in a state of intoxication commits suicide, whoever abets the commission of such suicide, shall be punished with death, or transportation for life, or imprisonment for a term not exceeding ten years, and shall also be liable to fine.

306. ABETMENT OF SUICIDE.—If any person commits suicide, whoever abets the commission of such suicide, shall be punished with imprisonment of either description for a term which may extend to ten years and also be liable to fine.

307. ATTEMPT TO MURDER.—Whoever does any act with such intention or knowledge and under such circumstances, that if he by that act caused death, he would be guilty of murder, shall be punished with imprisonment of either description for a term which may extend to ten years, and shall also be liable to fine; and if hurt is caused to any person by such act, the offender shall be liable either to transportation for life, or to such punishment as is hereinbefore mentioned.

When any person offending under this section is under sentence of transportation for life, he may, if hurt is caused, be punished with death.

308. ATTEMPT TO COMMIT CULPABLE HOMICIDE.—Whoever does any act with such intention or knowledge, and under such circumstances that if he by that act caused death, he would be guilty of culpable homicide not amounting to murder, shall be punished with imprisonment of either description for a term which may extend to seven years, or with fine, or with both.

309. ATTEMPT TO COMMIT SUICIDE.—Whoever attempts to commit suicide and does any act towards the commission of such offence, shall be punished with simple imprisonment for a term which may extend to one year, or with fine, or with both.

312. CAUSING MISCARRIAGE.—Whoever voluntarily causes a woman with child to miscarry, shall, if such miscarriage be not caused in good faith for the purpose of saving the life of the woman, be punished with imprisonment of either description for a term which may extend to three years, or with fine, or with both; and if the woman be quick with child, shall be punished with imprisonment of either description for a term which may extend to seven years, and shall also be liable to fine.

Explanation.—A woman who causes herself to miscarry is within the meaning of this section.

NOTE.—"WITH CHILD" means pregnant, and it is not necessary to show that quickening, that is, perception by the mother of the movements of the fœtus has taken place, or that the embryo has assumed a fœtal form. The stage to which pregnancy has advanced and the form which the ovum or embryo may have assumed are immaterial.

"MISCARRIAGE" means the premature expulsion of the child or fœtus from the mother's womb at any period of pregnancy, before the term of gestation is completed.

"QUICK WITH CHILD".—When the woman has felt the child move within her.

313. CAUSING MISCARRIAGE WITHOUT WOMAN'S CONSENT.—Whoever commits the offence defined in the last preceding section without the consent of the woman, whether the woman is quick with child or not, shall be punished with transportation for life, or with imprisonment of either description for a term which may extend to ten years, and shall also be liable to fine.

314. DEATH CAUSED BY ACT DONE WITH INTENT TO CAUSE MISCARRIAGE.—Whoever, with intent to cause the miscarriage of a woman with child, does any act which causes the death of such woman, shall be punished with imprisonment of either description for a term which may extend to ten years, and also be liable to fine, and if the act is done without the consent of the woman, shall be punished either with transportation for life, or with the punishment above mentioned.

Explanation.—It is not essential to this offence that the offender should know that the act is likely to cause death.

315. ACT DONE WITH INTENT TO PREVENT CHILD BEING BORN ALIVE OR TO CAUSE IT TO DIE AFTER BIRTH.—Whoever before the birth of any child does any act with the intention of thereby preventing that child from being born alive or causing it to die after its birth, and does by such act prevent that child from being born alive, or causes it to die after its birth, shall, if such act be not caused in good faith for the purpose of saving the life of the mother, be punished with

imprisonment of either description for a term which may extend to ten years, or with fine, or with both.

Cf. English Law.—If a person intending to procure abortion does an act which causes a child to be born so much earlier than the natural time, that it is born in a state much less capable of living, and afterwards dies in consequence of its exposure to the external world; the person who by his mis-conduct so brings the child into the world and puts it thereby in a situation in which it cannot live, is guilty of murder; and the mere existence of a possibility that something might have been done to prevent the death will not render it the less a murder.

316. CAUSING DEATH OF QUICK UNBORN CHILD BY ACT AMOUNTING TO CULPABLE HOMICIDE.—Whoever does any act under such circumstances, that if he thereby caused death he would be guilty of culpable homicide, and does by such act cause the death of a quick unborn child, shall be punished with imprisonment of either description for a term which may extend to ten years, and shall also be liable to fine.

317. EXPOSURE AND ABANDONMENT OF CHILD UNDER TWELVE YEARS, BY PARENT OR PERSON HAVING CARE OF IT.—Whoever, being the father or mother of a child under the age of twelve years, or having the care of such child, shall expose or leave such child in any place with the intention of wholly abandoning such child, shall be punished with imprisonment of either description for a term which may extend to seven years, or with fine, or with both.

Explanation.—This section is not intended to prevent the trial of the offender for murder or culpable homicide, as the case may be, if the child die in consequence of the exposure.

318. CONCEALMENT OF BIRTH BY SECRET DISPOSAL OF DEAD BODY. —Whoever, by secretly burying or otherwise disposing of the dead body of a child, whether such child die before or after or during its birth, intentionally conceals or endeavours to conceal the birth of such child, shall be punished with imprisonment of either description for a term which may extend to two years, or with fine, or with both.

319. HURT.—Whoever causes bodily pain, disease, or infirmity to any person is said to cause "hurt".

320. GRIEVOUS HURT.—The following kinds of hurt only are designated as "grievous":—

First.—Emasculation.

Secondly.—Permanent privation of the sight of either eye.

Thirdly.—Permanent privation of the hearing of either ear.

Fourthly.—Privation of any member or joint.

Fifthly.—Destruction or permanent impairing of the powers of any member or joint.

Sixthly.—Permanent disfiguration of the head or face.

Seventhly.—Fracture or dislocation of a bone or tooth.

Eighthly.—Any hurt which endangers life or which causes the sufferer to be, during the space of twenty days, in severe bodily pain, or unable to follow his ordinary pursuits.

321. VOLUNTARILY CAUSING HURT.—Whoever does any act with the intention of thereby causing hurt to any person, or with the knowledge that he is likely thereby to cause hurt to any person, and does thereby cause hurt to any person, is said "voluntarily to cause hurt".

322. VOLUNTARILY CAUSING GRIEVOUS HURT.—Whoever voluntarily causes hurt, if the hurt which he intends to cause or knows himself to be likely to cause is grievous hurt, and if the hurt which he causes is grievous hurt is said "voluntarily to cause grievous hurt".

Explanation.—A person is not said voluntarily to cause grievous hurt except when he both causes grievous hurt, and intends or knows himself to be likely to cause grievous hurt. But he is said voluntarily to cause grievous hurt if, intending or knowing himself to be likely to cause grievous hurt of one kind, he actually causes grievous hurt of another kind.

323. PUNISHMENT FOR VOLUNTARILY CAUSING HURT.—Whoever, except in the case provided for by Section 334, voluntarily causes hurt, shall be punished with imprisonment of either description for a term which may extend to one year, or with fine which may extend to one thousand rupees or with both.

324. VOLUNTARILY CAUSING HURT BY DANGEROUS WEAPONS OR MEANS.—Whoever, except in the case provided for by Section 334, voluntarily causes hurt by means of any instrument for shooting, stabbing, or cutting or any instrument, which, used as a weapon of offence, is likely to cause death or by means of fire or any heated substance, or by means of any poison or any corrosive substance, or by means of any explosive substance, or by means of any substance which it is deleterious to the human body to inhale, to swallow, or to receive into the blood, or by means of any animal, shall be punished with imprisonment of either description for a term which may extend to three years, or with fine, or with both.

325. PUNISHMENT FOR VOLUNTARILY CAUSING GRIEVOUS HURT.—Whoever, except in the case provided for by Section 335, voluntarily causes grievous hurt, shall be punished with imprisonment of either description for a term which may extend to seven years, and shall also be liable to fine.

326. VOLUNTARILY CAUSING GRIEVOUS HURT BY DANGEROUS WEAPONS OR MEANS.—Whoever, except in the case provided for by Section 335, voluntarily causes grievous hurt by means of any instrument for shooting, stabbing, or cutting or any instrument which, used as a weapon of offence, is likely to cause death, or by means of fire or any heated substance, or by means of any poison or any corrosive substance, or by means of any explosive substance, or by means of any substance which it is deleterious to the body to inhale, to swallow, or to receive into the blood, or by means of any animal, shall be punished with transportation for life, or with imprisonment of either description for a term which may extend to ten years, and shall also be liable to fine.

327. VOLUNTARILY CAUSING HURT TO EXTORT PROPERTY, OR TO CONSTRAIN TO AN ILLEGAL ACT.—Whoever voluntarily causes hurt for the purpose of extorting from the sufferer, or from any person interested in the sufferer, any property or valuable security, or of constraining the sufferer or any person interested in such sufferer to do anything which is illegal or which may facilitate the commission of an offence, shall be punished with imprisonment of either description for a term which may extend to ten years, and shall also be liable to fine.

328. CAUSING HURT BY MEANS OF POISON, ETC., WITH INTENT TO COMMIT AN OFFENCE.—Whoever administers to or causes to be taken by any person any poison or any stupefying, intoxicating, or unwholesome drug, or other thing, with intent to cause hurt to such person or with intent to commit or to facilitate the commission of an offence, or knowing it to be likely that he will thereby cause hurt, shall be punished with imprisonment of either description for a term which may extend to ten years, and shall also be liable to fine.

329. VOLUNTARILY CAUSING GRIEVOUS HURT TO EXTORT PROPERTY OR TO CONSTRAIN TO AN ILLEGAL ACT.—Whoever voluntarily causes grievous hurt for the purpose of extorting from the sufferer or from any person interested in the sufferer, any property or valuable security, or of constraining the sufferer or any person interested in such sufferer to do anything that is illegal or which may facilitate the commission of an offence. shall be punished with transportation for life or imprisonment of either description for a term which may extend to ten years, and shall also be liable to fine.

330. VOLUNTARILY CAUSING HURT TO EXTORT CONFESSION, OR TO COMPEL RESTORATION OF PROPERTY.—Whoever voluntarily causes hurt, for the purpose of extorting from the sufferer or any person interested in the sufferer any confession or any information which may lead to the detection of an offence or misconduct, or for the purpose of constraining the sufferer or any person interested in the sufferer to restore or cause the restoration of any property or valuable security

or to satisfy any claim or demand, or to give information which may lead to the restoration of any property or valuable security, shall be punished with imprisonment of either description for a term which may extend to seven years, and shall also be liable to fine.

331. VOLUNTARILY CAUSING GRIEVOUS HURT TO EXTORT CONFESSION, OR TO COMPEL RESTORATION OF PROPERTY.—Whoever voluntarily causes grievous hurt for the purpose of extorting from the sufferer or any person interested in the sufferer any confession or any information which may lead to the detection of an offence or misconduct, or for the purpose of constraining the sufferer or any person interested in the sufferer to restore or to cause the restoration of any property or valuable security, or to satisfy any claim or demand or to give information which may lead to the restoration of any property or valuable security shall be punished with imprisonment of either description for a term which may extend to ten years, and shall also be liable to fine.

332. VOLUNTARILY CAUSING HURT TO DETER PUBLIC SERVANT FROM HIS DUTY.—Whoever voluntarily causes hurt to any person being a public servant in the discharge of his duty as such public servant or with intent to prevent or deter that person or any other public servant or in consequence of anything done or attempted to be done by that person in the lawful discharge of his duty as such public servant, shall be punished with imprisonment of either description for a term which may extend to three years, or with fine, or with both.

333. VOLUNTARILY CAUSING GRIEVOUS HURT TO DETER PUBLIC SERVANT FROM HIS DUTY.—Whoever voluntarily causes grievous hurt to any person being a public servant in the discharge of his duty as such public servant, or with intent to prevent or deter that person or any other public servant from discharging his duty as such public servant or in consequence of anything done or attempted to be done by that person in the lawful discharge of his duty as such public servant, shall be punished with imprisonment of either description for a term which may extend to ten years, and shall also be liable to fine.

334. VOLUNTARILY CAUSING HURT ON PROVOCATION.—Whoever voluntarily causes hurt on grave and sudden provocation, if he neither intends nor knows himself to be likely to cause hurt to any person other than the person who gave the provocation, shall be punished with imprisonment of either description for a term which may extend to one month, or with fine which may extend to five hundred rupees, or with both.

335. VOLUNTARILY CAUSING GRIEVOUS HURT ON PROVOCATION.—Whoever voluntarily causes grievous hurt on grave and sudden provocation, if he neither intends nor knows himself to be likely to cause grievous hurt to any person other than the person who gave the provocation, shall be punished with imprisonment of either description for a term which may extend to four years, or with fine which may extend to two thousand rupees, or with both.

Explanation.—The last two sections are subject to the same provisos as exception 1, section 300.

336. ACT ENDANGERING LIFE OR PERSONAL SAFETY OF OTHERS.—Whoever does any act so rashly or negligently as to endanger human life or the personal safety of others, shall be punished with imprisonment of either description for a term which may extend to three months, or with fine which may extend to two hundred and fifty rupees, or with both.

337. CAUSING HURT BY ACT ENDANGERING LIFE OR PERSONAL SAFETY OF OTHERS.—Whoever causes hurt to any person by doing any act so rashly or negligently as to endanger human life, or the personal safety of others, shall be punished with imprisonment of either description for a term which may extend to six months or with fine which may extend to five hundred rupees, or with both.

338. CAUSING GRIEVOUS HURT BY ACT ENDANGERING LIFE OR PERSONAL SAFETY OF OTHERS.—Whoever causes grievous hurt to any person by doing any act so rashly or negligently as to endanger human life, or the personal safety of others, shall be punished with imprisonment of either description for a term which may extend to two years, or with fine which may extend to one thousand rupees, or with both.

351. ASSAULT.—Whoever makes any gesture, or any preparation, intending or knowing it to be likely that such gesture or preparation will cause any person present to apprehend that he who makes that gesture or preparation is about to use criminal force to that person, is said to commit an assault.

Explanation.—Mere words do not amount to an assault. But the words which a person uses may give to his gestures or preparations such a meaning as may make those gestures or preparations amount to an assault.

Cf. English Law.—An assault consists in an attempt or offer by a person having present ability, with force, to do any hurt or violence to the person of another. Battery means any least hurt or violence unlawfully and wilfully or culpably done to the person of another. Striking at another with a cane, stick, or fist although the blow misses, drawing a sword or bayonet, or throwing a bottle or glass with intent to wound or strike, presenting a loaded gun at a man within range, or any other act indicating an intention to use violence against the person of another, is an assault.

352. PUNISHMENT FOR ASSAULT OR CRIMINAL FORCE OTHERWISE THAN ON GRAVE PROVOCATION.—Whoever assaults or uses criminal force to any person otherwise than on grave and sudden provocation given by that person, shall be punished with imprisonment of either description for a term which may extend to three months, or with fine which may extend to five hundred rupees, or with both.

Explanation.—Grave and sudden provocation will not mitigate the punishment for an offence under this section, if the provocation is sought or voluntarily provoked by the offender as an excuse for the offence, or if the provocation is given by anything done in obedience to the law, or by a public servant, in the lawful exercise of the powers of such public servant, or if the provocation is given by anything done in the lawful exercise of the right of private defence.

Whether the provocation was grave and sudden enough to mitigate the offence, is a question of fact.

359. KIDNAPPING.—Kidnapping is of two kinds: Kidnapping from British India, and kidnapping from lawful guardianship.

360. KIDNAPPING FROM BRITISH INDIA.—Whoever conveys any person beyond the limits of British India without the consent of that person or of some person legally authorised to consent on behalf of that person, is said to kidnap that person from British India.

361. KIDNAPPING FROM LAWFUL GUARDIANSHIP.—Whoever takes or entices any minor under fourteen years of age, if a male, or under sixteen years of age, if a female, or any person of unsound mind, out of the keeping of the lawful guardian of such minor, or person of unsound mind, without the consent of such guardian, is said to kidnap such minor or person from lawful guardianship.

Explanation.—The words "lawful guardian" in this section include any person lawfully entrusted with the care or custody of such minor or other person.

Exception.—This section does not extend to the act of any person who in good faith believes himself to be the father of an illegitimate child, or who in good faith believes himself to be entitled to the lawful custody of such child, unless such act is committed for an immoral or unlawful purpose.

362. ABDUCTION.—Whoever by force compels, or by any deceitful means induces, any person to go from any place, is said to abduct that person.

363. PUNISHMENT FOR KIDNAPPING.—Whoever kidnaps any person from British India or from lawful guardianship, shall be punished with imprisonment of either description for a term which may extend to seven years, and shall also be liable to fine.

364. KIDNAPPING OR ABDUCTING IN ORDER TO MURDER.—Whoever kidnaps or abducts any person in order that such person may be murdered or may be so disposed of as to be put in danger of being murdered, shall be punished with transportation for life, or rigorous imprisonment for a term which may extend to ten years, and shall also be liable to fine.

365. KIDNAPPING OR ABDUCTING WITH INTENT SECRETLY AND WRONGFULLY TO CONFINE PERSON.—Whoever kidnaps or abducts any person with intent to cause that person to be secretly and wrongfully confined, shall be punished with imprisonment of either description for a term which may extend to seven years, and shall also be liable to fine.

366. KIDNAPPING OR ABDUCTING WOMAN TO COMPEL HER MARRIAGE, ETC.—Whoever kidnaps or abducts any woman with intent that she may be compelled, or knowing it to be likely that she will be compelled, to marry any person against her will, or in order that she may be forced or seduced to illicit intercourse, shall be punished with imprisonment of either description for a term which may extend to ten years, and shall also be liable to fine.

And whoever, by means of criminal intimidation as defined in this Code or of abuse of authority or any other method of compulsion, induces any woman to go from any place with intent that she may be, or knowing that it is likely that she will be, forced or seduced to illicit intercourse with another person shall also be punishable as aforesaid.[1]

366-A. PROCURATION OF MINOR GIRL.—Whoever, by any means whatsoever, induces any minor girl under the age of eighteen years to go from any place or to do any act with intent that such girl may be, or knowing that it is likely that she will be, forced or seduced to illicit intercourse with another person shall be punishable with imprisonment which may extend to ten years, and shall also be liable to fine.

365-B. IMPORTATION OF GIRL FROM FOREIGN COUNTRY.—Whoever imports into British India from any country outside India any girl under the age of twenty-one years with intent that she may be, or knowing it to be likely that she will be, forced or seduced to illicit intercourse with any person,

and whoever with such intent or knowledge imports into British India from any State in India any such girl who has with the like intent or knowledge been imported into India, whether by himself or another person,

shall be punishable with imprisonment which may extend to ten years, and shall also be liable to fine.[1]

367. KIDNAPPING OR ABDUCTING IN ORDER TO SUBJECT PERSON TO GRIEVOUS HURT, SLAVERY, ETC.—Whoever kidnaps or abducts any person in order that such person may be subjected, or may be so disposed of, as to be put in danger of being subjected to grievous hurt, or slavery, or to the unnatural lust of any person, or knowing it to be likely that such person will be so subjected or disposed of, shall be punished with imprisonment of either description for a term which may extend to ten years, and shall also be liable to fine.

368. WRONGFULLY CONCEALING OR KEEPING IN CONFINEMENT, KIDNAPPED OR ABDUCTED PERSON.—Whoever, knowing that any person has been kidnapped or has been abducted, wrongfully conceals or confines such person shall be punished in the same manner as if he had kidnapped or abducted such person with the same intention or knowledge, or for the same purpose as that with or for which he conceals or detains such person in confinement.

369. KIDNAPPING OR ABDUCTING CHILD UNDER TEN WITH INTENT TO STEAL FROM ITS PERSON.—Whoever kidnaps or abducts any child under the age of ten years with the intention of taking dishonestly any movable property from the person of such child, shall be punished with imprisonment of either description for a term which may extend to seven years, and shall also be liable to fine.

372. SELLING MINOR FOR PURPOSES OF PROSTITUTION, ETC.—Whoever sells, lets to hire or otherwise disposes of any minor under the age of sixteen years with intent that such minor shall be employed or used for the purpose of prostitution or for any unlawful and immoral purpose, or knowing it to be likely that such minor will be employed or used for any such purpose, shall be punished with imprisonment of either description for a term which may extend to ten years, and shall also be liable to fine.

---

1. *See Act No. XX of* 1923.

373. BUYING MINOR FOR PURPOSES OF PROSTITUTION, ETC.—Whoever buys, hires, or otherwise obtains possession of any minor under the age of sixteen years with intent that such minor shall be employed or used for the purpose of prostitution, or for any unlawful and immoral purpose, or knowing it to be likely that such minor will be employed, or used for any such purpose, shall be punished with imprisonment of either description for a term which may extend to ten years, and shall also be liable to fine.

375. RAPE.—A man is said to commit "rape", who except in the case hereinafter excepted, has sexual intercourse with a woman under circumstances falling under any of the five following descriptions:—

First.—Against her will.

Secondly.—Without her consent.

Thirdly.—With her consent, when her consent has been obtained by putting her in fear of death, or of hurt.

Fourthly.—With her consent, when the man knows that he is not her husband, and that her consent is given because she believes that he is another man to whom she is or believes herself to be lawfully married.

Fifthly.—With or without her consent, when she is under fourteen years of age.

Explanation.—Penetration is sufficient to constitute the sexual intercourse necessary to the offence of rape.

Exception.—Sexual intercourse by a man with his own wife, the wife not being under thirteen years of age, is not rape.

376. PUNISHMENT FOR RAPE.—Whoever commits rape shall be punished with transportation for life, or with imprisonment of either description for a term which may extend to ten years, and shall also be liable to fine, unless the woman raped is his own wife and is not under twelve years of age, in which case he shall be punished with imprisonment of either description for a term which may extend to two years, or with fine, or with both.

English Law.—A boy under fourteen years of age cannot be convicted of rape, as at that age he is under a physical incapacity to commit the offence. In India, the potency of a person charged with the offence has to be proved by evidence in each case, as unlike the English law there is no limit of age laid down, under which the law presumes a person physically incapable of committing rape.

377. UNNATURAL OFFENCES.—Whoever voluntarily has carnal intercourse against the order of nature with any man, woman, or animal, shall be punished with transportation for life, or with imprisonment of either description for a term which may extend to ten years, and shall also be liable to fine.

According to the English law if the passive agent is under fourteen, it is not felony in him, but only in the active agent. If both be of the age of discretion, *i.e.*, above fourteen years, it is felony in both. A married woman who consents to her husband's committing an unnatural offence with her is an accomplice.

Explanation.—Penetration is sufficient to constitute the carnal intercourse necessary to the offence described in this section.

394. VOLUNTARILY CAUSING HURT IN COMMITTING ROBBERY.—If any person in committing, or attempting to commit, robbery voluntarily causes hurt, such person, and any other person jointly concerned in committing or attempting to commit such robbery, shall be punished with transportation for life, or with rigorous imprisonment for a term which may extend to ten years, and shall also be liable to fine.

396. DACOITY WITH MURDER.—If any one of five or more persons who are conjointly committing dacoity, commits murder in so committing dacoity, every one of those persons shall be punished with death, or transportation for life, or rigorous imprisonment for a term which may extend to ten years, and shall also be liable to fine.

397. ROBBERY OR DACOITY WITH ATTEMPT TO CAUSE DEATH OR GRIEVOUS HURT.—If at the time of committing robbery or dacoity, the offender uses any deadly weapon, or causes grievous hurt to any person, or attempts to cause death or grievous hurt to any person, the imprisonment with which such offender shall be punished shall not be less than seven years.

459. GRIEVOUS HURT CAUSED WHILST COMMITTING LURKING HOUSE-TRESPASS OR HOUSE-BREAKING.—Whoever, whilst committing lurking house-trespass or house-breaking, causes grievous hurt to any person or attempts to cause death or grievous hurt to any person, shall be punished with transportation for life, or imprisonment of either description for a term which may extend to ten years, and shall also be liable to fine.

460. ALL PERSONS JOINTLY CONCERNED IN LURKING HOUSE-TRESPASS OR HOUSE-BREAKING BY NIGHT PUNISHABLE WHERE DEATH OR GRIEVOUS HURT CAUSED BY ONE OF THEM.—If, at the time of the committing of lurking house-trespass by night or house-breaking by night, any person guilty of such offence shall voluntarily cause, or attempt to cause, death or grievous hurt to any person, every person jointly concerned in committing such lurking house-trespass by night or house-breaking by night, shall be punished with transportation for life or with imprisonment of either description for a term which may extend to ten years, and shall also be liable to fine.

499. DEFAMATION.—Whoever, by words either spoken or intended to be read, or by signs or by visible representations, makes or publishes any imputation concerning any person intending to harm, or knowing, or having reason to believe that such imputation will harm, the reputation of such person, is said, except in the case hereinafter excepted, to defame that person.

Explanation 1.—It may amount to defamation to impute anything to a deceased person, if the imputation would harm the reputation of that person if living, and is intended to be hurtful to the feelings of his family or other near relatives.

Explanation 2.—It may amount to defamation to make an imputation concerning a company or an association or collection of persons as such.

Explanation 3.—An imputation in the form of an alternative or expressed ironically, may amount to defamation.

Explanation 4.—No imputation is said to harm a person's reputation, unless that imputation directly or indirectly in the estimation of others, lowers the moral or intellectual character of that person, in respect of his caste or of his calling or lowers the credit of that person, or, causes it to be believed that the body of that person is in a loathsome state or in a state generally considered as disgraceful.

500. PUNISHMENT FOR DEFAMATION.—Whoever defames another shall be punished with simple imprisonment for a term which may extend to two years, or with fine, or with both.

511. PUNISHMENT FOR ATTEMPTING TO COMMIT OFFENCES PUNUISHABLE WITH TRANSPORTATION OR IMPRISONMENT.—Whoever attempts to commit an offence punishable by this Code with transportation or imprisonment, or to cause such an offence to be committed, and in such attempt does any act towards the commission of the offence, shall, where no express provision is made by this Code for the punishment of such attempt, be punished with transportation or imprisonment of any description provided for the offence, for a term of transportation or imprisonment which may extend to one half of the longest term provided for that offence, or with such fine as is provided for the offence, or with both.

APPENDIX VIII

FORMS REQUIRED BY THE INDIAN LUNACY ACT, 1912

FORM I.

APPLICATION FOR RECEPTION ORDER

In the matter of A. B.,[1] residing at , by occupation , son of , a person alleged to be a lunatic.

To Presidency Magistrate, for [or District Magistrate of , or Sub-Divisional Magistrate of or Magistrate especially empowered under Act IV of 1912 for].

The Petition of C. D.,[1] residing at , by occupation , son of , in the town of [or Sub-Division of in the district of].

1. I am years of age.[2]

2. I desire to obtain an order for the reception of A. B. as a lunatic in the asylum of situate at[3]

3. I last saw the said A. B. at on the day[4] of

4. I am the[5] of the said A. B.

Or if the petitioner is not a relative of the patient state as follows:

I am not a relative of the said A. B. The reasons why this petition is not presented by a relative are as follows: (*State them*).

The circumstances under which this petition is presented by me are as follows: (*State them*).

5. The persons signing the medical certificates which accompany the petition are[6]

6. A statement of particulars relating to the said A. B. accompanies this petition.

7. (*If that is the fact*). An application for an inquiry into the mental capacity of the said A. B. was made to the on the and a certified copy of the order made on the same petition is annexed hereto. (*Or if that is the fact*).

No application for an inquiry into the mental capacity of the said A. B. has been made previous to this application.

---

1. *Full name, caste and titles.*

2. *Enter the number of completed years. The petitioner must be at least eighteen or twenty-one whichever is the age of majority under the law to which the petitioner is subject.*

3. *Insert full description of the name and locality of the asylum (mental hospital) or the name, address and description of the person in charge of the asylum (mental hospital).*

4. *A day within 14 days before the date of the presentation of the petition is requisite.*

5. *Here state the relationship with the patient.*

6. *Here state whether either of the persons signing the medical certificates is a relative, partner or assistant of the lunatic or of the petitioner and, if a relative of either, the exact relationship.*

The petitioner therefore prays that a reception order may be made in accordance with the foregoing statement.

Dated (Sd.) C. D.

The statements contained or referred to in paragraphs are true to my knowledge; the other statements are true to my information and belief.

Dated (Sd.) C. D.

STATEMENT OF PARTICULARS

(*If any of the particulars in this statement is not known, the fact to be so stated*).

The following is the statement of particulars relating to the said A. B.

Name of patient at length.

Sex and age.

Married or single or widowed.

Previous occupation.

Caste and religious belief, as far as known.

Residence at or immediately previous to the date hereof.

Names of any near relatives to the patient who are alive.

Whether this is a first attack of lunacy.

Age (if known) on first attack.

When and where previously under care and treatment as a lunatic.

Duration of existing attack.

Supposed cause.

Whether the patient is subject to epilepsy.

Whether suicidal.

Whether the patient is known to be suffering from phthisis or any form of tubercular disease.

Whether dangerous to others, and in what way.

Whether any near relative (stating the relationship) has been afflicted with insanity.

Whether the patient is addicted to alcohol or the use of opium, ganja, charas, bhang, cocaine or other intoxicant.

[The statements contained or referred to in paragraphs are true to my knowledge. The other statements are true to my information and belief].

[Signature by person making the statement].

FORM 2.

RECEPTION ORDER PETITION.

(See Sections 7, 10).

I, the undersigned E. F., being a Presidency Magistrate of

[*or* the District Magistrate of —*or* the Sub-Divisional Magistrate of—*or* a Magistrate of the first class specially empowered by the Government to perform the functions of a Magistrate under Act IV of 1912] upon the petition of C. D. of[1] in the matter of A. B.,[1] a lunatic, accompanied by the medical certificates of G. H., a medical officer, and of J. K., a medical practitioner [*or* medical officer], under the said Act, hereto annexed, hereby authorise you to receive the said A. B. into your asylum. And I declare that I have (*or* have not) personally seen the said A. B. before making this order.

To [2] (Sd.) E. F.

Designation as above.

1. *Address and description.*

2. *To be addressed to the officer or person in charge of the asylum.*

FORM 3.

MEDICAL CERTIFICATE.

In the matter of A. B. of[1] in the town of [*or* the sub-division of in the district of], an alleged lunatic.

I, the undersigned C. D., do hereby certify as follows:—

1. I am a gazetted medical officer [*or* a medical practitioner declared by a holder of[2] [*or* declared by Local Government to be a medical government to be medical officer under Act IV of 1912] and I am in the practitioner under Act IV of 1912] actual practice of the medical profession.

2. On the day of 19 at[3] in the town / village of [*or* the sub-division of in the district] [separately from any other practitioner][4], I personally examined the said A. B., and came to the conclusion that the said A. B., is a lunatic and a proper person to be taken charge of and detained under care and treatment.

3. I formed this conclusion on the following grounds, *viz.*:—

(a) Facts indicating insanity observed by myself, *viz.*:—

(b) Other facts (if any) indicating insanity communicated to me by others, *viz.*:— *Here state the information and from whom.*

(Sd.) C. D.

Designation as above.

FORM 5.

Reception order in case of wandering or dangerous lunatics or lunatics not under proper control or cruelly treated sent to an asylum established by Government.

(See sections 14, 15 and 17.)

I, C. D., Presidency Magistrate of [or Commissioner of Police for] [*or* the District Magistrate of—*or* the Sub-divisional Magistrate of—*or* a Magistrate specially empowered by Government under Act IV of 1912] having caused A. B. to be examined by E. F., a Medical officer under the Indian Lunacy Act, 1912, and being satisfied that A. B. [describing him] . . . is a lunatic who was wandering at large (or is a person dangerous by reason of lunacy) [or is a lunatic not under proper care and control or is cruelly treated or neglected by the person having the care or charge of him] and a proper person to be taken charge of and detained under care and treatment, hereby direct you to receive the said A. B. ..... into your asylum.

Dated the 19

(Sd.) C. D.

Designation as above.

To

The officer in charge of the asylum at

1. *Insert residence of patient.*

2. *Insert qualification to practise medicine and surgery registrable in the United Kingdom.*

3. *Insert place of examination.*

4. *Omit this where only one certificate is required.*

APPENDIX IX

THE HIPPOCRATIC OATH AND THE VEDIC PRINCIPLES OF MEDICINE

I. THE HIPPOCRATIC (UNANI) OATH, 400 B.C.

I swear by Apollo the Physician and Æsculapius and Hygeia and Panaces, while I take to witness all the Gods and Goddesses that I shall fulfil according to my ability and judgment this oath and this covenant.

That I shall esteem my teacher in this art as I do my parents, and contribute towards his subsistence, and share my goods with him if he is in need, and regard his offspring as my own brothers and teach them this art, without fee and covenant, should they wish to study it, and impart its general precepts and oral lessons and all the rest of the science to my sons, and those of my teachers and to students who have been registered and sworn according to the law of Medicine, but to none other.

I will follow the dietic regimen for the benefit of the sick according to my ability and my judgment and will withhold that which would be for their injury and hurt.

I will not administer a deadly drug to any one though solicited thereto, nor will I suggest any such advice.

In like manner I will not administer abortive drugs to women.

Purely and holily will I live and practise my art.

Nor will I use the knife on those suffering from stone, but will leave them to specialists in this operation.

Into whatever house I enter I will go with the object of helping the sick, holding aloof from all voluntary and all other hurtful wrong doing and from licentious practices, whether with women or with men, free or bond, and regarding the things I see or hear, in the exercise of my art, or outside its exercise, in my intercourse with men, which ought not to be divulged I will keep silent regarding them as inviolable secrets.

If then I fulfil this oath, without any violation, may I enjoy life and the fruits of my art held in honour by all men for evermore, but, if I transgress it and commit perjury may quite the contrary befall me.

II. THE VEDIC PRINCIPLES OF MEDICINE

Thus said the illustrious son of Atri: If an intelligent man, impelled by proper reason, desires to become a Physician, the following should be the qualifications of him that should be selected as preceptor. He should be:

One whose doubts have been all cleared in respect of Medical scriptures—possessed of experience—clever in the practice of his profession—compassionate towards those who approach him—clean in person and clothing—have a practised hand in Surgery—possessed of all the implements of his profession with his organs of sense perfect—conversant with nature—his knowledge of medical science supplemented with a knowledge with other branches of study—without malice—of a peaceful disposition—capable of bearing privations and pain—well affected towards disciples and disposed to teach them—capable of communicating his ideas—

Approaching such a preceptor, the pupil should attend on him with heedfulness like one revering one's sacrificial fire, or one's deity, or one's king, or one's father, or one's patron.

The preceptor should examine his pupil who should be of a mild disposition—noble by nature—not mean in acts—with eyes, mouth and nasal line straight—tongue thin, red and not slimy—teeth and lips without deformity—voice of good tone—possessed of intelligence—free from pride—endowed with a large understanding—with a power of judgment and memory—having a liberal mind—belonging to a medical family—devoted to truth—without defect in his limbs—having all his senses perfect—disposed to solitude—free from haughtiness—of thoughtful disposition—free from the faults of "Vyasana",—not prone to wrath—endowed with purity of behaviour and compassion for all—devotedly attached to the study of medicine—free from cupidity—without sloth—seeking the good of all creatures—prepared to obey all his preceptor's commands and attached to him.

Unto one adorned with such qualifications the preceptor should say—thou shouldst always regard me as the foremost of persons—holding thyself in subjection to me—bearing thyself in a way that is agreeable and beneficial to me—behaving as a son, as a slave, as a suppliant, towards me whilst being taught by me.

Thou shouldst be free from impatience and always attentive, doing everything with a mind concentrated upon thy work—behaving with humility, and acting after reflection—never murmuring or finding fault with thy instructor but willingly carrying out my orders.

Thou shouldst, with thy whole heart, strive to bring about the cure of those that are ill—not even for thy life's sake extorting their substance. Thou shouldst not, even in imagination, know another man's wife, and similarly thou shouldst not appropriate the possession of others.

Thou shouldst never administer medicines unto those that have incurred the displeasure of the king or those that are ill-disposed towards him or those that have incurred the displeasure of the great or those bearing ill-will towards them. So also thou shouldst not administer medicines to those that are of exceedingly perverse or wicked disposition or those that are exceedingly poor, or those that never vindicate their character when it is aspersed, or those that are on the point of death or those that have not their masters near them, or those women that have not their husbands or other guardians near them.

Thou shouldst never gossip of the practices of a patient's house. Even if possessed of sufficient knowledge thou shouldst not boast of that knowledge.

There is no end in the scienc of medicine. Hence heedfully and carefully thou shouldst devote thyself to it conducting thyself as I direct and without feeling of humiliation acquiring practice in the art.

Unto men, possessed of intelligence the entire world acts as a preceptor. Unto men, destitute of intelligence, the entire world occupies the position of an enemy.

The preceptor saying these words the pupil should answer, "yes". If the pupil does as he is commanded then should he be taught. If he behaves otherwise he should be rejected as unworthy.

APPENDIX X

THE POISONS ACT (ACT No. XII), 1919 AND THE RULES IN THE UNITED PROVINCES OF AGRA AND OUDH

Whereas it is expedient to consolidate and amend the law regulating the importation, possession and sale of poisons throughout British India; It is hereby enacted as follows:—

1. (1) This Act may be called the Poisons Act, 1919.

(2) It extend to the whole of British India, including British Baluchistan and Sonthal Parganas.

2. (1) Subject to the control of the Governor-General in Council, the Local Government may, by rule, regulate within the whole or any part of the territories under its administration the possession for sale and the sale, whether wholesale or retail, of any specified poison.

(2) In particular, and without prejudice to the generality of the foregoing power, such rules may provide for—

(*a*) the grant of licenses to possess any specified poison for sale, wholesale or retail, and the fixing of the fee (if any) to be charged for such licenses;

(*b*) the classes of persons to whom alone such licenses may be granted;

(c) the classes of persons to whom alone such poison may be sold;

(*d*) the maximum quantity of any such poison which may be sold to any one person;

(*e*) the maintenance by vendors of any such poison of registers of sales, the particulars to be entered in such registers, and the inspection of the same;

(*f*) the safe custody of such poisons and the labelling of the vessels, packages or coverings in which any such poison is sold or possessed for sale; and

(*g*) the inspection and examination of any such poison when possessed for sale by any such vendor.

3. The Governor-General in Council may, by notification in the Gazette of India, prohibit, except under and in accordance with the conditions of a license, the importation into British India of any specified poison and may, by rule, regulate the grant of licenses.

4. (1) The Local Government, with the previous sanction of the Governor-General in Council, may by rule regulate the possession of any specified poison in any local area in which the use of such poison for the purpose of committing murder or mischief by poisoning cattle appears to it to be of such frequent occurrence as to render restrictions on the possession thereof desirable.

(2) In making any rule under sub-section (1), the Local Government may direct that any breach thereof shall be punishable with imprisonment for a term which may extend to one year, or with fine which may extend to one thousand rupees, or with both, together with confiscation of the poison in respect of which the breach has been committed, and of the vessels, packages or coverings in which the same is found.

5. Any substance specified as a poison in a rule made or notification issued under this Act shall be deemed to be a poison for the purposes of this Act.

6. (1) Whoever—(*a*) commits a breach of any rule made under section 2, or (*b*) imports into British India without a license any poison, the importation of which is for the time being restricted under section 3, or (c) breaks any condition of a license for the importation of any poison granted to him under section 3, shall be punishable,—(i) on a first conviction, with imprisonment for a term which may extend to three months, or with fine which may extend to five hundred rupees, or with both, and (ii) on a second or subsequent conviction, with imprisonment for a term which may extend to six months, or with fine which may extend to one thousand rupees or with both.

(2) Any poison in respect of which an offence has been committed under this section together with the vessels, packages or coverings in which the same is found, shall be liable to confiscation.

7. (1) The District Magistrate, the Sub-divisional Magistrate and, in a Presidency Town, the Commissioner of Police may issue a warrant for the search of any place in which he has reason to believe or suspect that any poison is possessed or sold in contravention of this Act or any rule thereunder, or that any poison liable to confiscation under this Act is kept or concealed.

(2) The person to whom the warrant is directed may enter and search the place in accordance therewith, and the provisions of the Code of Criminal Procedure, 1898, relating to search-warrants shall, as far as may be, be deemed to apply to the execution of the warrant.

8. (1) In addition to any other power to make rules hereinbefore conferred, the Governor-General in Council or, subject to the control of the Governor-General in Council, the Local Government may make rules generally to carry out the purposes and objects of this Act.

(2) Every power to make rules conferred by this Act shall be subject to the condition of the rules being made after previous publication.

(3) All rules made by the Governor-General in Council or by the Local Government under this Act shall be published in the Gazette of India or the Local Official Gazette, as the case may be, and on such publication shall have effect as if enacted in this Act.

9. (1) Nothing in this Act or in any license granted or rule made thereunder shall extend to or interfere with, anything done in good faith in the exercise of the profession as such by a medical or veterinary practitioner.

(2) Notwithstanding anything hereinbefore contained, the Local Government may in its discretion by general or special order declare that all or any of the provisions of this Act shall be deemed not to apply to any article or class of articles of commerce specified in such order, or to any poison or class of poisons, used for any purpose so specified.

(3) The authority on which any power to make rules under this Act is conferred may, by general or special order, either wholly or partially—

(*a*) exempt from the operation of any such rules, or

(*b*) exclude from the scope of the exemption provided by sub-section (1),

any person or class of persons either generally or in respect of any poisons specified in the order.

10. The Poisons Act, 1904, is hereby repealed.

RULES[1] MADE BY THE GOVERNMENT OF UNITED PROVINCES OF AGRA AND OUDH UNDER SECTION 2 OF THE POISONS ACT (ACT No. XII) OF 1919

1. In these rules "the Act" means the Poisons Act, 1919.

---

1. *U. P. Gazette Notifications, Nos.* 631|*VI*-1149 *dated* 15*th Feb.,* 1921, 1859|*VI*-935 *dated June* 1, 1926, 1991|*VI*-1261 *dated May* 1, 1933, 1273|*VI*-1766 *dated Sep.* 29, 1933, 2135|*VI*-2019 *dated Jan.* 8, 1934, 1971|*VI*-1776-1930 *dated Sep.* 5, 1934, 1000|*VI*-1766-1930 *dated April* 15, 1935 *and* 2023|*VI*-1766-1930 *dated Oct.* 11, 1935.

2. SCHEDULE OF POISONS.—The following substances are deemed to be poisons within the meaning of the Act.

1. Aconite, nux vomica, stramonium (dhatura), and ergot.

2. Perchloride of mercury (corrosive sublimate), cyanide of potash, and prussic acid.

3. Aconitine, strychnine, hyoscyamine, hyoscine, and gelsemine, or gelseminine, whether as free alkaloids or as salts of these alkaloids.

4. Arsenic and its compounds.

A.—SULPHIDES OF ARSENIC

I. Red sulphide (realgar). (Vern.) Mansil.

II. Yellow sulphide (orpiment). (Vern.) Hartal.

B.—IMPURE SULPHIDES OF ARSENIC

I. Black Arsenic. (Vern.) Kala sankhia.

II. Impure orpiments.

III. White oxide. (Vern.) Safed sankhia.

IV. Pink sulphide. (Vern.) Gulabi sankhia.

V. Brown sulphide. (Vern.) Bhura sankhia.

C.—GREEN ARSENIC

I. Arsenite of copper (Scheele's green). (Vern.) Hirwa.

II. Aceto-arsenite of copper (Schweinfurth's green). (Vern.) Hirwa.

5. Oxalic acid, Picric Acid, Barium carbonate, Plumbago rosea (Lal Chitra) and Plumbago zeylanica (Chitra).

6. The following poisonous preparations of British Pharmacopœia :—

1. Atropine—its salts and B. P. preparations.

2. Chloroform and all preparations containing more than 20 per cent of chloroform.

3. Cocaine, its salts and B. P. preparations.

4. B. P. preparations of Dhatura.

5. Diethyl-Barbituric acid and such derivatives, as Veronal, Proponal, Medinal.

6. Digitalis and its B. P. preparations.

7. Homatropine hydrobromide and its preparations.

8. Hyoscine hydrobromide.

9. Hyoscyamine sulphate.

10. All B. P. preparations of Nux Vomica containing more than 0.2 per cent of strychnine, and all its alkaloids with their salts and preparations.

11. Oxalic Acid.

12. Phosphorus and all preparations containing 0.005 or more per cent of free phosphorus.

13. Physostigmine sulphate.

14. Pilocarpine nitras.

15. Prussic acid and all preparations containing more than 0.1 per cent of it.

Note.—"Preparations" or "B. P. preparations" in this list refer to preparations official in the British Pharmacopœia, 1932 Edition except item No. 4 (preparations of Dhatura) included only in the schedule of poisonous preparations of the 1914 edition of the British Pharmacopœia.

7. Tetra-ethyl lead except as provided in rule 16.

3. No person not exempted under the provisions of the Act shall sell or possess for sale any poison specified in the schedule, except under a license granted in that behalf by the District Magistrate.

4. The grant or withdrawal of a licence to any applicant shall be at the discretion of the District Magistrate, whose decision shall be final. Medical practitioners not possessing registrable qualifications should not be granted licenses for the sale of any of the poisons mentioned in item No. 6 in the schedule.

5. Subject to the provisions of rules 6 and 7, a license granted under rule 3 shall remain in force for one year from the first January, or the date of issue, if later than the first January to the thirty-first December following. Every applicant for the grant or renewal of a licence shall make a written application to the District Magistrate, and such application shall bear a court-fee stamp of rupee one.

6. A licence shall terminate on the death of the licence-holder, or if granted to a firm or company, on the winding up or transfer of the business of such firm or company.

7. The District Magistrate may, at any time, for any sufficient cause, revoke or cancel any licence granted unler rule 3.

8. Every sale of poison shall, as far as possible, be conducted by the licence-holder in person or where the licence-holder is a firm or company, through, or under the supervision of, an accredited representative of such firm or company.

9. A licence-holder shall not sell any poison to any person unless the latter is personally known to him or identified to his satisfaction. He shall not sell any poison to any person who appears to him to be under the age of 18 or to any person who does not appear to him to be in full possession of his faculties, or to any wandering mendicant.

He shall not sell or dispense the poisonous preparations of the British Pharmacopœia enumerated in item No. 6 of the schedule except in a prescription given by a medical practitioner possessing qualifications registrable under the United Provinces Medical Act, 1917.

9-A. A licence-holder shall not sell powdered white arsenic to any person unless the same is, before the sale thereof, mixed with soot in the proportion of one ounce of soot at least to one pound of white arsenic. or with indigo or Prussian blue in the proportion of half an ounce of indigo or Prussian blue to one pound of white arsenic, and so on in proportion for any greater or less quantity.

Provided that the licensing authority may, after full investigation and reference, if necessary, to higher authorities, permit on such condition and with such restrictions as it thinks necessary any licence-holder to sell white arsenic without any admixture.

10. (i) Every licence-holder shall maintain a register in which he shall enter all sales of poison except those issued in prescriptions.

The following particulars shall be entered in such register in respect of each such sale, namely :—

(*a*) Name of poison.

(*b*) Quantity sold.

(c) Date of sale.

(*d*) Name and address of purchaser.

(*e*) Purpose for which the poison was stated by the purchaser to be required.

(*f*) Signature of purchaser (or thumb-impression if illiterate) or in case of purchase by post, date of letter or written order and reference to the original in the file in which it is preserved.

(*g*) Signature of vendor.

(ii) In a separate portion of the register shall be entered in separate columns for each poison, the quantity of each sold daily and these entries shall be filled up, from day to day, and totalled daily.

(iii) The signature under item (*g*) of the register shall be that of the licence-holder himself, or when the licence-holder is a firm or company, that of an accredited representative of such firm or company, and shall be entered at the time of sale or despatch to the purchaser. Such signature shall be held to imply that the writer has satisfied himself that the requirements of rule 7 have been fulfilled.

(iv) All letters or written orders referred to in head (*f*) of the register shall be preserved in original by the licence-holder for a period of not less than two years from the date of the sale.

11. (i) A licence-holder shall maintain in respect of each poison specified in the schedule a stock register which shall contain the following particulars :—

(*a*) Serial number.

(*b*) Date.

(c) Amount received.

(*d*) Name and address of person from whom received.

(*e*) Amount sold.

(*f*) Balance in stock.

(*g*) Remarks.

(ii) The stock register shall be balanced daily.

12. A licence-holder shall maintain a register containing copies of all prescriptions which contain any scheduled poison. The register shall contain the following particulars:—

(1) Serial number.

(2) Date.

(3) Name of prescriber.

(4) Name of person for whom prescribed.

(5) Copy of prescription.

(6) Detail of poison in the prescription—(*a*) Name of poison, and (*b*) Total quantity.

(7) Signature of dispenser.

13. Any Magistrate or police officer of or above the rank of inspector, any Revenue officer of or above the rank of tahsildar, or any medical officer of or above the rank of assistant surgeon may, at any time, visit and inspect the premises of a licence-holder where poison is kept for sale and may inspect all poisons found therein and registers maintained under rules 10, 11 and 12.

14. All poisons kept for sale by any licence-holder under these rules shall be kept in a box, almirah, room or building (according to the quantity maintained) which shall be secured by lock and key and in which no substance shall be placed other than poisons possessed in accordance with a licence granted under the Act, and each poison shall be kept, within such box, almirah, room or building, in a separate closed receptacle of glass, metal or earthenware. Every such box, almirah, room or building, and every such receptacle, shall be marked with the word "Poison" in red characters, both English and Vernacular, and in the case of receptacles containing separate poisons with the name of such poison.

15. When any poison is sold, it shall be securely packed in a closed receptacle or packet (according to quantity) and every such receptacle or packet shall be labelled by the vendor with a red label bearing the name of the poison in English and Vernacular, and the number and date of the entry in the register of sales kept for the purpose.

16. Petrol containing ethyl fluid (an admixture of tetra ethyl lead) is exempted from the provisions of the Act and these rules provided :

(i) the cans and pumps containing the fuel are labelled to indicate the presence of tetra ethyl lead in the fuel to warn the user both in English as well as in the Vernaculars to avoid spillage and not to use the fuel for applying to any part of the body or for any purposes other than as a motor fuel ;

(ii) the fuel is dyed as an additional check against use otherwise than as a motor fuel; and

(iii) the amount of lead tetra ethyl does not exceed one part in 1,300 parts by volume or one in 650 by weight.

THE U. P. EXCISE ACT IV OF 1910, AMENDED BY ACT III OF 1913

Section 3 (12).—Intoxicating drug means—

(i) the leaves, small stalks and flowering or fruiting tops of the Indian hemp plant (Cannabis sativa L.), including all forms known as *bhang, siddhi,* or *ganja;*

(ii) *charas,* that is, the resin obtained from the Indian hemp plant, which has not been submitted to any manipulations other than those necessary for packing and transport;

(iii) any mixture, with or without neutral materials, of any of the above forms of hemp or any drink prepared therefrom; and

(iv) any other intoxicating or narcotic substance which the Chief Commissioner (Local Government), may, by notification, declare to be an intoxicating drug such substance not being opium, coca leaf, or a manufactured drug, as defined in section 2 of the Dangerous Drugs Act, 1930.

Section 3 (23).—Cocaine includes coca leaves, any alkaloid or substance prepared from the coca plant and an admixture of any of the above.

The possession of cocaine is prohibited throughout the United Provinces of Agra and Oudh.

Provided that this prohibition shall not extend to the possession of cocaine by—

(1) Licensed vendors of cocaine to the extent of one ounce or such larger quantity as may in special cases be sanctioned by the Excise Commissioner;

(2) Medical practitioners registered under the United Provinces Medical Act, 1917, military or civil assistant surgeons, sub-assistant surgeons, gazetted officers of the Army Veterinary Corps and the Indian Civil Veterinary Department possessing cocaine in exercise of their profession to the extent of half-an-ounce or such larger quantity as may in special cases be sanctioned by the Excise Commissioner provided that retired military or veterinary officers shall not possess cocaine unless they are registered under the above-mentioned Act;

(3) private individuals to the extent of such quantity as they may have purchased on the prescription of a medical or veterinary practitioner exempted under clause (2);

(4) veterinary assistants in charge of Government, Municipal board or District board veterinary hospitals to such an amount not exceeding half an ounce as they may be authorised to possess by their superior authorities.

(5) officers in charge or managers, as the case may be, of the recognized hospitals and dispensaries for use in their hospitals and dispensaries to the amounts varying from one ounce to sixteen ounces.

II. Provided also that the Chief Revenue authority may exclude from the operation of the above proviso any person belonging to the class described in clause (2) thereof who in his opinion has abused the privilege conferred thereby.—*Rule* 698, *Excise Manual, Vol. I, as amended.*

Cocaine is understood to include anæsthesin.—*Rule* 693, *Excise Manual, Vol. I.*

The import, export or transport of cocaine by the inland post, otherwise than on Government account, is prohibited. This prohibition does not apply to the preparations containing not more than one-tenth per cent of cocaine which are exempted under section 76 of the U. P. Excise Act.—*Rule* 693*A*, *Excise Manual, Vol. I.*

APPENDIX XI

THE DANGEROUS DRUGS ACT, 1930 (ACT NO. II OF 1930) AS AMENDED BY THE DANGEROUS DRUGS AMENDMENT ACT, 1933 (ACT NO. XXVI OF 1933), AND THE DANGEROUS DRUGS AMENDMENT ACT, 1938 (ACT NO. III OF 1938)

(Some of its important Sections)

Whereas India participated in the Second International Opium Conference, which was convoked in accordance with the resolution of the Assembly of the League of Nations dated the 27th day of September, 1923, met at Geneva on the 17th day of November, 1924, and on the 19th day of February 1925, adopted the Convention relating to Dangerous Drugs (hereinafter referred to as the Geneva Convention) ;

And whereas the Contracting Parties to the said Geneva Convention resolved to take further measures to suppress the contraband traffic in and abuse of Dangerous Drugs, especially those derived from opium, Indian hemp and coca leaf, such measures being more particularly set forth in the articles of the said Geneva Convention ;

And whereas for the effective carrying out of the said measures it is expedient that the control of certain operations relating to Dangerous Drugs should be centralised and vested in the Governor-General in Council ;

And whereas it is also expedient that the penalties for certain offences relating to Dangerous Drugs should be increased, and that all penalties relating to certain operations should be rendered uniform throughout British India ;

It is hereby enacted as follows :—

1. (1) This Act may be called the Dangerous Drugs Act, 1930.

(2) It extends to the whole of British India, including British Baluchistan and the Sonthal Parganas.

(3) It shall come into force on such date as the Governor-General in Council may, by notification in the Gazette of India, appoint.

2. In this Act, unless there is anything repugnant in the subject or context,—

(*a*) "coca leaf" means—

(i) the leaf and young twigs of any coca plant, that is, of the *erythroxylon coca* (*Lank.*) and the *Erythroxylon novo-granatense* (*Hiern.*) and their varieties and of any other species of this genus which the Governor-General in Council may, by notification in the Gazette of India, declare to be coca plants for the purpose of this Act ; and

(*ii*) any mixture thereof, with or without neutral materials ;

but does not include any preparation containing not more than 0.1 per cent of cocaine ;

(*b*) "coca derivative" means—

(i) crude cocaine, that is, any extract of cocaine leaf which can be used, directly or indirectly, for the manufacture of cocaine ;

(ii) ecgonine, that is, lævo-ecgonine having the chemical formula $C_9H_{15}NO_3.H_2O$, and all the derivatives of lævo-ecgonine from which it can be recovered ;

(iii) cocaine, that is, methyl-benzoyl-lævo-ecgonine having the chemical formula $C_{17}H_{21}NO_4$, and its salts ; and

(iv) all preparations, officinal and non-officinal, containing more than 0.1 per cent of cocaine ;

(*c*) "hemp" means—

(i) the leaves, small stalks and flowers or fruiting tops of the Indian hemp plant (*Cannabis sativa L.*), including all forms known as *bhang, siddhi,* or *ganja ;*

(ii) *charas,* that is, the resin obtained from the Indian hemp plant, which has not been submitted to any manipulations other than those necessary for packing and transport ; and

(iii) any mixture, with or without neutral materials, of any of the above forms of hemp or any drink prepared therefrom ;

(*d*) "medicinal hemp" means an extract or tincture of hemp ;

(*e*) "opium" means—

(i) the capsules of the poppy (*Papaver somniferum L.*) ;

(ii) the spontaneously coagulated juice of such capsules which has not been submitted to any manipulations other than those necessary for packing and transport ; and

(iii) any mixture, with or without neutral materials, of any of the above forms of opium ;

but does not include any preparation containing not more than 0.2 per cent of morphine ;

(*f*) "opium derivative" means—

(i) medicinal opium, that is, opium which has undergone the process necessary to adapt it for medicinal use in accordance with the requirements of the British Pharmacopœia, whether in powder form or granulated or otherwise or mixed with neutral materials ;

(ii) prepared opium, that is, any produce of opium, obtained by any series of operations designed to transform opium into an extract suitable for smoking, and the dross or other residue remaining after opium is smoked ;

(iii) morphine, that is, the principal alkaloid of opium having the chemical formula $C_{17}H_{19}NO_3$, and its salts ;

(iv) diacetylmorphine, that is, the alkaloid, also known as diamorphine or heroin, having the chemical formula $C_{21}H_{23}NO_5$, and its salts ; and

(v) all preparations, officinal and non-officinal, containing more than 0.2 per cent of morphine, or containing any diacetylmorphine ;

(*g*) "manufactured drug" includes—

(i) all coca derivatives, medicinal hemp and opium derivatives ; and

(ii) any other narcotic substance which the Governor-General in Council may, by notification in the Gazette of India made in pursuance of a recommendation under Article 10 of the Geneva Convention, or in pursuance of any international convention supplementing the Geneva Convention, declare to be a manufactured drug ;

but does not include any preparation which the Governor-General in Council may, by notification in the Gazette of India made in pursuance of a finding under Article 8 of the Geneva Convention, declare not to be a manufactured drug ;

(*h*) "dangerous drug" includes coca leaf, hemp and opium, and all manufactured drugs ;

(*i*) "to import into British India" means, subject to the provisions of clause (*j*) to bring into British India by land, sea or air; and includes the bringing into any port or place in British India of a dangerous drug intended to be taken out of British India without being removed from the ship or conveyance in which it is being carried.

(*j*) "to import inter-provincially" means to bring into one province from another, and includes—

(i) the bringing of a dangerous drug into a province from any territory of a Prince or Chief in India which is adjacent to or enclosed by the territories of such province, which the Governor-General in Council may, by notification in the Gazette of India, declare to be inter-provincial import; and

(ii) bringing into one province from another, in the course of a continuous journey, by sea or through the territory of a Prince or Chief in India;

(*k*) "to export from British India" means, subject to the provisions of clause (*l*), to take out of British India by land, sea or air;

(*l*) "to export inter-provincially" means to take out of one province into another and includes—

(i) the taking of a dangerous drug out of a province into any territory of a Prince or Chief in India which is adjacent to or enclosed by the territories of such province, which the Governor-General in Council may, by notification in the Gazette of India, declare to be inter-provincial export; and

(ii) taking out of one province into another, in the course of a continuous journey, by sea or through the territories of a Prince or Chief in India;

(*m*) "to transport" means to take from one place to another in the same province; and

(*n*) "territory of a Prince or Chief in India" includes any territory in which the Governor-General in Council exercises powers or jurisdiction by virtue of the Indian (Foreign Jurisdiction) Order in Council, 1902.

3. The Governor-General in Council may make rules prescribing the method by which percentages in the case of liquid preparations shall be calculated for the purpose of clauses (*a*), (*b*), (*e*) and (*f*) of section 2:

Provided that, unless and until such rules are made such percentages shall be calculated on the basis that a preparation containing one per cent. of a substance means a preparation in which one gramme of the substance, if a solid, or one millilitre of the substance, if a liquid, is contained in every one hundred millilitres of the preparation, and so in proportion for any greater or less percentage.

4. (1) No one shall—

(*a*) cultivate any coca plant, or gather any portion of a coca plant,

(*b*) manufacture or possess prepared opium, unless it is prepared from opium lawfully possessed for the consumption of the person so possessing it, or

(*c*) import into British India, export from British India, tranship or sell prepared opium;

Provided that this section shall not apply to the cultivation of any coca plant or to the gathering of any portion thereof on behalf of Government.

(2) The Local Government may make rules restricting and regulating the manufacture and possession of prepared opium from opium which is lawfully possessed under clause (*b*) of sub-section (1).

5. (1) No one shall—

(*a*) cultivate the poppy (*Papaver somniferum L.*) or

(*b*) manufacture opium,

save in accordance with rules made under sub-section (2) and with the conditions of any license for that purpose which he may be required to obtain under those rules.

(2) The Governor-General in Council may make rules permitting and regulating the cultivation of the poppy (*Papaver somniferum L.*) and the manufacture of opium, and such rules may prescribe the form and conditions of licenses for such cultivation and manufacture, the authorities by which such licenses may be granted, the fees that may be charged therefor, and any other matter requisite to render effective the control of the Governor-General in Council over such cultivation and manufacture.

(3) The Governor-General in Council may also make rules permitting and regulating the sale of opium from Government factories for export or to Local Governments or to manufacturing chemists.

6. (1) No one shall manufacture any manufactured drug, other than prepared opium, save in accordance with rules made under sub-section (2) and with the conditions of any licence for that purpose which he may be required to obtain under those rules.

(2) The Governor-General in Council may make rules permitting and regulating the manufacture of manufactured drugs, other than prepared opium, and such rules may prescribe the form and conditions of licenses for such manufacture, the authorities by which such licenses may be granted and the fees that may be charged therefor, and any other matter requisite to render effective the control of the Governor-General in Council over such manufacture.

(3) Nothing in this section shall apply to the manufacture or medicinal opium or of preparations containing morphine, diacetylmorphine or cocaine from materials which the maker is lawfully entitled to possess.

7. (1) No one shall—

(*a*) import into British India,

(*b*) export from British India,

(*c*) tranship

any dangerous drug, other than prepared opium, save in accordance with rules made under sub-section (2) and with the conditions of any license for that purpose which he may be required to obtain under those rules.

(2) The Governor-General in Council may make rules permitting and regulating the import and export from British India and the transhipment of dangerous drugs, other than prepared opium, and such rules may prescribe the ports or places at which any kind of dangerous drug may be imported, exported or transhipped, the form and conditions of licenses for such import, export or transhipment, the authorities by which such licenses may be granted, the fees that may be charged therefor, and any other matter requisite to render effective the control of the Governor-General in Council over such import, export and transhipment.

8. (1) No one shall—

(*a*) import or export inter-provincially, transport, possess or sell any manufactured drug, other than prepared opium, or coca leaf, or

(*b*) manufacture medicinal opium or any preparation containing morphine, diacetylmorphine or cocaine, save in accordance with rules made under sub-section (2) and with the conditions of any license for that purpose which he may be required to obtain under those rules.

(2) The Local Government may, subject to the control of the Governor-General in Council, make rules permitting and regulating—

(*a*) the inter-provincial import and export into and from the territories under its administration, the transport, possession and sale of manufactured drugs, other than prepared opium, and of coca leaf ; and

(*b*) the manufacture of medicinal opium or of any preparation containing morphine, diacetylmorphine or cocaine from materials, which the maker is lawfully entitled to possess.

Such rules may prescribe the form and conditions of licenses for such import, export, transport, possession, sale and manufacture, the authorities by which such licenses may be granted and the fees that may be charged therefor, and any other matters requisite to render effective the control of the Local Government over such import, export, transport, possession, sale and manufacture.

(3) Save in so far as may be expressly provided in rules made under sub-section (2), nothing in this section shall apply to manufactured drugs which are the property and in the possession of Government:

Provided that such drugs shall not be sold or otherwise delivered to any person who, under the rules made by the Local Government under this section, is not entitled to their possession.

9. No one shall engage in or control any trade whereby a dangerous drug is obtained outside British India and supplied to any person outside British India, save in accordance with the conditions of a license granted by and at the discretion of the Local Government.

10. Whoever—

(*a*) cultivates any coca plant or gathers any portion of a coca plant,

(*b*) manufactures or possesses prepared opium otherwise than as permitted under section 4, or

(*c*) imports into British India, exports from British India, transhipps or sells prepared opium,

shall be punished with imprisonment which may extend to two years, or with fine, or with both:

Provided that this section shall not apply to the cultivation of any coca plant or to the gathering of any portion thereof on behalf of Government.

11. Whoever, in contravention of section 5, or any rule made under that section, or of any condition of a license granted thereunder,

(*a*) cultivates the poppy, or

(*b*) manufactures opium,

shall be punished with imprisonment which may extend to two years, or with fine, or with both.

12. Whoever, in contravention of section 6, or any rule made under that section, or any condition of a license granted thereunder, manufactures any manufactured drug, shall be punished with imprisonment which may extend to two years, or with fine, or with both.

13. Whoever, in contravention of section 7, or any rule made under that section, or any condition of a license granted thereunder,

(*a*) imports into British India,

(*b*) exports from British India, or

(*c*) transhipps

any dangerous drug, shall be punished with imprisonment which may extend to two years, or with fine, or with both.

14. Whoever, in contravention of section 8, or any rule made under that section, or any condition of a license issued thereunder,

(*a*) imports or exports inter-provincially, transports, possesses or sells any manufactured drug or coca leaf, or

(b) manufactures medicinal opium or any preparations containing morphine, diacetylmorphine or cocaine,

shall be punished with imprisonment which may extend to two years, or with fine, or with both.

15. Whoever, being the owner or occupier or having the use of any house, room, enclosure, space, vessel, vehicle, or place, knowingly permits it to be used for the commission by any other person of an offence punishable under section 10, section 12, section 13, or section 14, shall be punished with imprisonment which may extend to two years, or with fine, or with both.

16. Whoever, having been convicted of an offence punishable under section 10, section 12, section 13, or section 14, is guilty of any offence punishable under any of those sections, shall be subject for every such subsequent offence to imprisonment which may extend to four years, or to fine, or to both.

17. Whoever, having been convicted of an offence punishable under section 15, is again guilty of an offence punishable under that section, shall be subject for every such subsequent offence to imprisonment which may extend to four years, or to fine, or to both.

INDEX

A

Abandoning of infants, 388.
Abdomen, changes in, during delivery, 323, 324.
—enlargement of, during pregnancy, 319.
—examination of, 71.
—injuries of, 288.
Abducting, 42.
Abortifacients used in India, 359.
Abortion, 354; criminal, 41, 45, 356.
Abortionists, 360.
Abortion sticks, 360.
Abrasions, 68, 217, 221.
—difference between ante-mortem and post-mortem, 223.
Abrin, 636.
Abrus precatorius, 636.
—poisoning by, 636.
—seeds, 636.
Absinthe, 758.
—oil of, 758.
Accident following rape, 339.
—from criminal abortion, 361.
—in burns, 209.
—in drowning, 188.
—in hanging, 159.
—in starvation, 194.
—in strangulation, 170.
—in suffocation, 176.
Accidental causes in infanticide, 379.
Accidental wounds, 252.
Accused persons, examination of, in abortion cases, 362; in infanticide cases, 369; in rape, 339; in sodomy, 352.
Acetanilide, poisoning by, 716.
Acetic acid, poisoning by, 519.
Aceto-arsenite of copper, 544.
Acetphenetidin, poisoning by, 716.
Acid, acetic, 519.
—acetyl salicylic, 519.
—arsenic, 543.
—arsenious, 542.
—boracic, 540.
—cacodylic, 544.
—carbazotic, 516.
—carbolic, 510.
—carbonic, 806.
—chromic, 614.
—citric, 522.
—crotonoleic, 628.
—hydrochloric, 502.
—hydrocyanic, 797.
—hydrofluoric, 503.
—jatrophic, 652.
—meconic, 674, 683.
Acid, nitric, 499.
—osmic, 626.
—oxalic, 504.
—picric, 516.
—prussic, 797.
—salicylic, 517.
—strychnic, 764.
—sulphuric, 493.
—sulphurous, 814.
—tartaric, 521.
Acid of sugar, 504.
Acids, burns by, 198.
—corrosive, 491.
—mineral, 491.
—organic, 504.
—treatment of, 492.
Aconite, poisoning by, 790.
Aconitine, 791.
Aconitum, ferox, 792.
—heterophyllum, 792.
—napellus, 790.
Acute insanities, 393, 398.
—rheumatism, in life assurance, 438.
Adipocere, 124, 146.
—conditions favouring, 147.
—illustrative cases of, 148.
—time of, 147, 148.
Adolf Beck's Case, 63, 64.
Ærugo, 587.
Æther, poisoning by, 698.
Affiliation cases, 327.
Affirmation, 13.
Afiyun, 673.
Age, 24, 32.
—in life assurance, 439.
—medico-legal aspect of, 41.
—minor signs of, 40.
—viable, 45.
Agglutinable factors, 102.
Agglutinin, 102.
Agglutinogens, 102.
Agoraphobia, 406.
Agotan, 718.
Agraphia in will-making, 424.
Aid, omission to call in medical, 386.
Air, in stomach as a sign of respiration, 376.
—in middle ear, 377.
Airol, 608.
Ak, in abortion, 359.
Akasbel, 652.
Akdo, 640.
Akta, 763.
Alcohol, 688.
Alcohol, absolute, 688.
Alcohol, amyl, poisoning by, 695.

Alcohol, ethyl, diagnosis of poisoning by, 689.
—methyl, poisoning by, 694.
—post-mortem appearances of, 690.
—symptoms of poisoning by, 688.
—tests for, 691.
Alcoholic intoxication, 692.
Alkalies, 522.
—poisoning by, 522.
Alkaloidal group reagents, 483.
Alkaloids, cadveric, 667.
Allantiasis, 667.
Aloes, 653; Aloin, 653.
—poisoning by, 653.
Alu, 751.
Alum, 619.
Aluminium, 619.
—poisoning by, 619.
Alsi, 804.
Amanita muscaria, poisoning by, 760.
—phalloides, poisoning by, 760.
Amentia, 393, 394.
Ammonia, 522.
—poisoning by, 523.
—tests for, 525.
Ammonium carbonate, poisoning by, 523.
Amok, run, 756.
Amount of illumination for identification, 25, 62.
Amygdalin, 798.
Amyl alcohol, poisoning by, 695.
—nitrite, poisoning by, 696.
Anacardium occidentale, 638.
Anæsthesia, in chloroform, 700.
—in rape, 342.
Anæsthesin, 749.
Anæsthetics, responsibility in death from, 455.
Androgynæ, 29.
Androgyni, 29.
Anethum graveolens, 359.
Angostura bark, 771.
Aniline, 719.
—oil, 719.
Animal poisons, 470, 658.
Antagonists, 489.
Anthropometry, 24, 48.
Anthropophagy, 339.
Antidotes, 486, 488.
Antifebrin, poisoning by, 716.
Antigen, 99.
Antimoniuretted hydrogen, 568.
Antimony, 567.
—acute poisoning by, 568.
—cases, 574.
—chemical tests for, 572.
—chronic poisoning by, 571.
—hydride, 568.
—medico-legal points in poisoning by, 572.
—organic preparations of, 568.
Antimony, post-mortem appearances in poisoning by, 570, 571.
—tartaratum, 567.
—treatment in poisoning by, 570, 571.
—trichloride, 567.
—trioxide, 567.
—trisulphide, 568.
Antipyrin, poisoning by, 718.
Ants, abrasions caused by bites of, 223.
—poisoning by bites of, 665.
Anus in sodomy, 349, 351.
Aorta, decomposition of, 143.
—examination of, 70.
—wounds of, 288.
Aphasia, in relation to testamentary capacity, 424.
Apomorphine hydrochloride, as emetic, 488.
Apoplexy, diagnosis from opium poisoning, 679.
—in drowning, 179.
—in hanging, 155.
—heat, 196.
—in strangulation, 161.
Aqua fortis, 499.
Arandi, 627.
Areolæ of breasts, in delivery, 323.
—in pregnancy, 318.
—in virgins, 315.
Argemone mexicana, poisoning by, 655.
Argyria, 611.
Argyrol, 610.
Arrow poison, 631, 796.
Arsamin, 545.
Arsacetin, 545.
Arsenates, 543.
Arsenic, 542.
—acid, 543.
—acute poisoning by, 546.
—chemical tests for, 554.
—chronic poisoning by, 553.
—compounds of, 542.
—diagnosis in poisoning by, 548.
—fatal dose of, 548.
—fatal period of, 548.
—hydride, 544.
—medico-legal points of, 558
—post-mortem appearances of, 551, 554.
—proprietary articles of, 545.
—sulphide, 544.
—treatment of, 549, 554.
—trichloride, 544.
—triodide, 544.
—trioxide, 542.
Arsenious acid, 542.
—anhydride, 542.
—iodide, 544.
Arsenious oxide, 542.
Arsenites, 543.
Arseniuretted hydrogen, 544.
Arsenobenzol, 545.

Arsenophagists, 562.
Arsine, 544.
Arsphenamine, 545.
Arthralgia, 595.
Artificial inflation, 375.
Artificial respiration in drowning, 180.
Artificial verdigris, 586.
Artemisia maritima, 757.
Arum colocasia, 655.
Arum maculatum, poisoning by, 654.
Aschheim-Zondek test, 320.
Asphyxia, 119, 120.
—causes of death from, 120.
—post-mortem appearances of, 120, 121.
—symptoms of, 120.
—traumatic, 281.
Asphyxiants, 470, 806, 815.
Aspirin, poisoning by, 519.
Assessors, 9, 10, 14.
Assurance, accident, 441.
—life, 436.
Asthenia, 119.
Asthma, in life assurance, 437.
Asylum ear, 393.
Atelectasis, 374.
Atis, 792.
Atocin, 718.
Atophan, 718.
Atoxyl, 545.
Atropa belladonna, 739.
—poisoning by, 740.
Atropine, 739.
Attainment of majority, 41, 43.
Auric chloride, 624.
Australian insect powder, 649.
Autopsy, 65.
Azoospermia, 306, 307.

B

Bachhnak, 792.
Bahera, 657.
Ballottement, 320.
Bansdola, in strangulation, 160, 161.
—in suffocation, 176.
Barberios' test, 112.
Barber's vermin-killer, 765.
Barbital, 712.
Barbitone, 712.
Barbitonum solubile, 714.
Barhanta, 751.
Barium, poisoning by, 621.
—carbonate, 621.
—chemical tests for, 622.
—chloride, 621.
—nitrate, 621.
—sulphate, 621.
—sulphide, 621.
Bark, angostura, 771.
Bastard, 326.
Battery fluids, 614.
Battle's vermin killer, 765.
Beam's test, 754.
Beans, calabar, 773.
—St. Ignatius, 764.
Beer, arsenic in, 559.
Bees, stings by, 665.
Belladonna, 739.
—poisoning by, 739.
Belleric myrobalans, 657.
Benzene, poisoning by, 722.
Benzidine solution, 93.
—test, 92, 93.
Benzol, poisoning by, 722.
Benzoyl-methyl-ecgonine, 743.
Bertillonage, 48.
Bertillon system, 48.
Bestiality, 353.
Bhang, 752.
Bhilawan, 220, 300, 638.
Bhooi ringani, 751.
Bhujri, 686.
Biological test for, arsenic, 558.
—blood, 99.
—pregnancy, 320.
—semen, 116.
Binoxalate of potassium, poisoning by, 509.
Birmi, 650.
Birth, concealment of, 388.
Birth marks, 55.
Bish, 792.
Bismarsen, 608.
Bismuth, breath, 608.
—carbonate, 608.
—poisoning by, 608.
—salicylate, 608.
—subnitrate, 608.
Bistoval, 608.
Bitter almonds, 797.
Bitter Apple, 631.
Black, antimony, 568.
—drop, 676.
—hellebore, 648.
—turpeth, 652.
Blackening of the hand by fire-arms, 256.
Bladder, at birth, 376.
Bladder, decomposition of, 142.
—examination of, 73.
—rupture of, 297.
Blindness in life assurance, 438.
Blister-beetle, 658.
Blistering gases, 815.
Blood, 89.
—arterial, 91.
—corpuscles, 95.
—human, 96.
—menstrual, 91.
—venous, 91.
Blood stains, 89.
—age of, 91.
—appearances of, 91.
—due to crushing of insects, 92.
—examination of, 90.
—from an assailant, 90, 91, 92.
—from a victim, 90, 91, 92.

Blood stains, grouping test, 101.
—groups, technique for determining, 103.
—on leather, 95.
—on wood, 95.
Blue, line on gums, 582.
—rocket, 790.
—stone, 586.
—vitriol, 586.
Blyth's test for brucine, 769.
Bodies, exhumation of, 85.
Body, cooling of, 123, 127.
—external examination of, 66.
—in cases of poisoning, 71.
—internal examination of, 69.
Bones, 35, 68, 78.
—age from, 82.
—burnt, 82, 83.
—cause of death from, 83.
—contusion of, 301.
—decomposition of, 83.
—examination of, 78.
—fractures of, 301.
—identification by, cases, 84.
—injuries of, 301.
—nutrient canals of, 83.
—ossification of, 35.
—specific gravity of, 82.
—time of death from, 83.
—weight of, 74.
Book as evidence, 20.
Boot mark, 55.
Boracic or boric acid, poisoning by, 540.
—tests for, 541.
Borax, 358; poisoning by, 540.
Born alive, 369.
Borneo camphor, 759.
Borneol, 759.
Boron, 540.
Botulism, 667.
Brain, 70, 269.
—compression of, 271.
—concussion of, 270.
—contusion of, 270.
—decomposition of, 141, 142.
—laceration of, 270.
—ventricles of, 70.
—weight of, 74.
Braxton Hick's sign, 319.
Breasts, in abortion, 362.
—in delivery, 323, 324.
—in pregnancy, 318.
—in virginity, 315.
Breslau's second life test, 376.
Brides of the bath, 188.
Bromide of ammonium, 537.
—of potassium, 537.
—of sodium, 537.
Bromidia, 710.
Bromine, poisoning by, 537.
—tests for, 538.
—treatment of, 538.
Bromism, 537.
Bromoform, poisoning by, 706.
Bronzing liquid, 567.
Brucine, 765.
—tests for, 769.
Bruises, 217.
—age of, 219.
—difference between accidental, homicidal and self-inflicted, 219.
—difference between ante-mortem and post-mortem, 221.
—difference from post-mortem staining, 129.
Bruises, result of, 219.
Buchanan's formula, 440.
Buggery, 347.
Bullet, 67, 229.
Burdwan case, 63.
"Burking", 176.
Burnett's fluid, 604.
Burns, 198.
—ante-mortem, 205, 206.
—causes of death from, 200.
—classification of, 199.
—effects of, 200.
—fatal period in, 202.
—illustrative cases of, 208.
—on a dead body, 68.
—period of, 207.
—post-mortem, 205, 206.
—post-mortem appearances in, 202.
Butler's vermin-killer, 765.
Butta, 763.
Butter of antimony, 567.
Buttorah ka Dal, 763.

C

Cacodylic acid, 544.
Cadaveric alkaloids, 667.
—changes in muscles, 124, 130.
—hypostasis, 123, 128.
—lividity, 123, 128.
—rigidity, 131.
—spasm, 132, 133.
Cadmium, 623.
—chloride, 623.
—poisoning by, 623.
—sulphide, 623.
Calabar bean, 773.
—poisoning by, 773.
Callus, time of appearance, 243.
Calomel, 578.
Calotropis gigantea and procera, 359, 640.
—poisoning by, 640.
—tests for, 642.
Camphene, 730.
Camphor, poisoning by, 758.
Cancer in life assurance, 439.
Cannabis Indica (Sativus), 752.
—poisoning by, 753.
Cannibalism, 339.
Cantharides, poisoning by, 658.
—treatment of, 659.

Cantharidin, 658.
Cantharis Vesicatoria, 658.
Capacity, testamentary, 422.
Capsaicin, 635.
Capsicum annum (frutescens), 635.
—poisoning by, 635.
—seeds, 636.
Caput Succedaneum, 377.
Carbazotic acid, poisoning by, 516.
Carbolic acid, chemical tests for, 512.
—medico-legal points in poisoning by, 513.
—poisoning by, 510.
—treatment of, 511.
Carbolism, 510.
Carbon, bisulphide, poisoning by, 812.
—dioxide, poisoning by, 806.
—disulphide, poisoning by, 812.
—monoxide, poisoning by, 807.
—tetrachloride, poisoning by, 705.
Carbonyl of nickel and cobalt, 626.
Carbonyls, 807.
Cardiac poisons, 470, 776.
Carica Papaya, 359.
Carihari, 654.
Carnal knowledge, 333.
Carrot seeds, as abortifacients, 359.
Carunculæ hymenealis (myrtiformes), 337.
Caryophyllus aromaticus, 359.
Cashew nut, 638.
Cassel yellow, 591.
Castor oil, poisoning by, 627.
—seeds, 627; detection of, 628.
Catamite, 348.
Catatonia, 397, 398, 406.
Cattle poisoning, 469.
Caucasian race, 27.
Caustic potash, 522.
Caustic soda, 522.
Cayenne pepper, 635.
Celastrus paniculata, 359.
Cephal-hæmatoma, 265.
Cephalic index, 27.
Cerbera thevetia, poisoning by, 787.
Cerebral poisons, 673, 733.
Certificates, medical, 14.
—in insanity, 414.
Cervix uteri, 319.
—changes in, 319, 323, 324.
Cessation, of circulation, 123, 124; tests for, 125, 126.
—of menses, 316.
—of respiration, 123, 124; tests for, 126.
Chandi, 610.
Chandu, 686.
Chapman case, 574.
Charas, 753.
Charcoal, in poisoning, 489.
Cheena sindur, 577.
Chemical analysis, viscera to be preserved for, 71, 72.
—of poisons, 478.
Chemical antidote, 488.
Chemical examination of blood, 90.
Chemical Examiner's report, 17, 18.
Cherry-laurel water, 798, 804.
Chest, injuries of, 281.
—pressure on, 173.
—post-mortem examination of, 70.
—shape of, 370.
Child, quick with, 317.
Children, supposititious, 328.
Chillies, 300, 635.
Chitra, 220, 644.
Chloral hydrate, poisoning by, 707.
Chlorine, poisoning by, 536.
—treatment of, 536.
Chloroform, poisoning by, 700.
Chloroacetophenone, 817.
Chloropicrin, 816.
Choke damp, 806.
Chopped animal hair, poisoning by, 671.
Chromate of lead, 591.
Chrome holes, 615.
Chrome yellow, 591.
Chromic, acid, 614.
—anhydride, 614.
—trioxide, 614.
Chromium, 614.
—acute poisoning by, 614.
—chronic poisoning by, 615.
Cicatrix, 55.
Cinchophen, poisoning by, 718.
Cinnabar, 577.
Circular insanity, 403.
Citric acid, poisoning by, 522.
Civil responsibility, 419.
Classification of poisons, 470.
Claustrophobia, 406.
Clothes and ornaments, 25, 61.
Cloquet's needle test, 125.
Coagulation of blood after death, 129.
Coal gas, 807.
Coal-Tar Naphtha, 721.
Cobalt, poisoning by, 626.
—tests for, 626.
Cobra, poisoning by, 662.
Cocaine, 743.
—acute poisoning by, 744.
—bugs, 745.
—chronic poisoning by, 745.
—hydrochloride, 743.
—tests for, 746.
Cocainomania, 745.
Cocainophagia, 745.
Cocculus Indicus, 655.
Cocculus Suberosus, poisoning by, 655.
Codeine, 675.
Colic in lead poisoning, 595.
Colchicum, 646.
Colchicum autumnale, 646.
—poisoning by, 646.
—tests for, 646.
Cold, 190, 194.
—death from, 195.

Cold, medico-legal aspect in, 195.
—post-mortem appearances in, 195.
—stiffening, 132, 133.
—symptoms in, 194.
—treatment in, 135.
Collargol, 610.
Colliquative putrefaction, 139.
Colocynth, 631.
—poisoning by, 631.
Colocynthin, 632.
Colour changes, in ecchymosis, 219.
Colour changes, in post-mortem staining, 129.
—in putrefaction, 135.
Colostrum, 318.
Colubrine snakes, 660.
Coma, 119, 121.
—causes of, 121.
—post-mortem appearances of, 122.
—symptoms of, 121.
Combustion, spontaneous (preternatural), 209.
Common salt, as an emetic in poisons, 488.
Common witness, 18, 19.
Complexion, 24, 45.
—illustrative cases, 46, 47.
—powders, 543.
Compression of brain, 271.
Concealed sex, 30.
Concealment, of birth, 388.
—of pregnancy, 317.
Concussion, of brain, 270.
—of spine, 281.
Condemned woman, 315.
Conduct-money, 11.
Condy's fluid, 526.
Congenital diseases, as a cause of death of infants, 378.
Coniine, 818.
Conium maculatum, poisoning by, 818.
Consent, age of, 43, 333.
—in examination of person, 23.
—validity of, 422.
Contract, validitv of, 421.
Contusions, 68, 217.
Cooling of body after death, 123, 127.
—conditions influencing, 127, 128.
—rate of, 127.
Copper, 586.
—aceto-arsenite of, 543.
—acute poisoning by, 587.
—arsenite, 543.
—carbonate, 587.
—chemical tests for, 589.
—chronic poisoning by, 588.
—constituent of body, 590.
—in tinned peas, 589.
—medico-legal points in poisoning by, 589.
—subacetate of, 587.
—sulphate, 586.
—tests for, 589.
Copper, treatment for poisoning of, 588.
Copperas, 612.
—white, 604.
Cord, 67.
—in strangulation, 162.
—mark of, in hanging, 156.
—spinal, 73.
—umbilical, 377.
Coroner, 2; court of, 2.
Corpus delicti, 23.
Corpus luteum, 322.
Corrosives, 470.
Corrosive, poisons, 491.
—sublimate, 575.
Cotton, fibres, 117.
—root bark, 358.
Coup de Soleil, 196.
Courtesy, tenancy by, 326.
Courts, 9.
—kinds of criminal, 9.
—of Magistrates, 9.
—powers of, 9, 10.
—sessions, 9.
Cow-itch (hage) 672.
Cranium, fracture of, in infanticide, 383.
Creolin, poisoning by, 514.
Creosote, poisoning by, 515.
Cresol, poisoning by, 514.
Cretinism, 396.
Crime, difficulties in detection of, 3.
Criminal abortion, 41, 45, 356.
Criminal Courts and their powers, 9.
Criminal responsibility, in relation to age, 41; in connection with insanity, 424.
Crinum deflexum (Asiaticum), 655.
Crockery, wounds by broken, 228.
Crocus sativus, 359.
Cross-examination, 13.
Crotin, 628.
Croton, 628.
Croton oil, 628.
Croton tiglium, poisoning by, 628.
Crying at birth, 369.
Cryptorchids, 306.
Crystals, hæmin, 96.
—Roussin's, 778.
Cuckoo-pint, 654.
Cucumis trigonus, 359.
Culpable homicide, 243, 244. (See Appendix VII).
Curara (Curare), poisoning by, 819.
—tests for, 820.
Curcin, 652.
Cuscuta reflexa, 359, 652.
—poisoning by, 652.
Cutis anserina, 182.
Cyanhæmochromogen, 99.
Cyanmethæmoglobin, 798, 799.
Cytisine, 649.
Cytisus Laburnum, poisoning by, 649.

D

Daboia, poisoning by bite of, 662.
Daisy powder, 716.
Dakin's solution, 536.
Dangerous Drugs Act, 468. (See Appendix XI).
Darby's fluid, 526.
Darnel, 763.
Daruri, 655.
Data, to ascertain age of injury, 242.
Datura, alba, 733.
Datura, fastuosa, 733.
—illustrative cases, 738.
—metel, 733.
—poisoning by, 734.
—seeds, 736.
—smoke, 736.
—stramonium, 733.
Daturine, 733.
Daucus carota, 359.
Day's test, 92.
Dead body, examination of, 65.
—identification of, 23.
Dead-born, 368.
Deadly agaric, 760.
Deadly nightshade, 739.
Death, cause of, 74.
—definition of, 119.
—manner of, 74.
—modes of, 119.
—molecular, 119.
—presumption of, 151.
—proof of, in life assurance, 441.
—rattle, 122.
—signs of, 123, 124.
—somatic, 119.
—sudden, 122.
—time of, 150, 151.
—time since, 67, 77.
Debility of infant as a cause of death, 378.
Declaration, dying, 14, 16, 17.
Decomposed body, 23, 135.
—age of, 30.
—examination of, 75.
—floatation of, 144.
Decomposition. (See Putrefaction).
Deformities, 24, 55.
—acquired, 55.
—congenital, 55.
Deliriant poisons, 470, 733.
Delirium, 433.
Delirium tremens, 690.
Delivery, during sleep, 381, 382.
—feigned, 328.
—in presumption of survivorship, 153.
—post-mortem, 363.
—signs of recent, in dead, 323.
—signs of recent, in living, 322.
—signs of remote, in living, 324; in dead, 325.
—unconscious, 381.
Delphinium staphisagria, poisoning by, 648.
Delphinine, 648.
Delusion, 389.
Delusional insanity, 404.
Dementia, 396.
—organic, 398.
—naturalis, 394.
—paralytica, 408.
—præcox, 396, 397.
—secondary, 397.
—senile, 398.
Deposition, 17.
Dermatol, 603.
Development, of fœtus, 364.
—of gases in putrefaction, 135, 136.
Dhais, 360.
Dhawal, 780.
Diabetes in life assurance, 439.
Diachylon paste, 358.
Diagnosis of poisoning, 474.
Dial, poisoning by, 715.
Diamond dust, poisoning by, 671.
Diaphanous test, 125, 126.
Diaphragm, 70.
—changes in, 370.
—rupture of, 288.
—wounds of, 288.
Dicodide, 675.
Didial, poisoning by, 715.
Diethyl-barbituric acid, 712.
Diethyl melonyl-urea, 712.
Digestion, degree of, to ascertain time of death, 151.
Digitalis purpurea, poisoning by, 780.
Dilaudide, 675.
Dill seeds, 819.
Dinitrobenzene, poisoning by, 726.
Dinitrobenzol, poisoning by, 726.
Dimethyl-arsenic acid, 544.
Dimethyl-methane-diethyl sulphone, 711.
Dionin, 674.
Diphenylamine-chloroarsine, 817.
Diphenylchloroarsine, 817.
Diphenylcyanarsine, 817.
Diphosgene, 816.
Discharge of lunatics, 417.
Disciplinary control, 445.
Disease, to be distinguished from poison, 474, 475.
Dislocations, 302.
Disputed paternity, 104, 105.
Diuretics, as abortifacients, 358.
Divorce, 303, 311.
Docimasia pulmonaris, 373.
Documentary evidence, 14.
Dolimoola, 751.
Dragendorff's process, 481.
Drowning, 154, 179.
—definition of, 179.
—fatal period in, 180.
—illustrative cases of, 188, 189.
—in infanticide, 383.

Drowning, medico-legal questions in, 186.
—mode of death in, 179.
—post-mortem appearances in, 181, 182.
—stages of, 179.
—symptoms of, 179.
—treatment in, 180.
Drugs used to induce abortion, 358.
Drunkenness, 433.
Dry belly-ache, 595.
Dry method for analysing organic mixtures for mineral poisons, 484.
Dry test, for arsenic, 558.
Duboisia hopwoodii, 776.
Ductus arteriosus, 378.
Ductus venosus, 378.
Dudhia Bish, 790.
Dura mater, examination of, 70.
Duties of patient, 453.
Duties of physician, 448.
Duty of a medical man in suspected poisoning, 484, 485.
Dyer's Spirit, 613.
Dying declaration, 14, 16, 17.
Dynamite, 728.
—bobbins of, 728.
Dysentery, in life assurance, 438.

E

Ear, asylum or insane, 393.
—discharge from, in life assurance, 438.
—in identification, 45.
—injuries of, 276.
Ecbolics, 358.
Ecchymosis, 217.
—colour changes in, 219.
—distinguished from post-mortem staining, 129.
—subpleural, 121.
Echolalia, 407.
Education, in identification, 25, 62.
Effusion of blood in brain, 272.
Elaterium, as an abortifacient, 359.
Electric current, 213.
Electricity, death from, 198, 213, 215.
—effects of, 213.
—medico-legal questions in, 216.
—post-mortem appearances of, 215.
—treatment of, 215.
Electrocution, 214.
Eligibility for employment, 41, 43.
Elimination of poison, 471.
—absorbed into the system, 490.
Elio, 653.
Elwa, 653.
Emerald green, 543.
Emetics in abortion, 358; in poisons, 488.
Emmenagogues, 358.
Encephalopathy, 595.
Epilepsy, 408.
—in life assurance, 439.
Epileptic insanity, 408.
Epsom salts, 620.
—poisoning by, 620.
Epispadias, 305.
Ergot, 632.
—acute poisoning by, 633.
—chemical analysis of, 634.
—chronic poisoning by, 633.
—treatment of, 634.
Ergotism, 633.
Erythroxylum coca, 743.
Eserine, poisoning by, 773.
Essence of bitter almonds, 798.
Ether, poisoning by, 698.
Ethyl alcohol, 688.
Ethylamine, 667.
Ethylic ether, 698.
Ethyl-iodo-acetate, 817.
Ethyl oxide 698.
Eucalyptus oil, poisoning by, 732.
Eukodal, 675.
Euphorbia antiquorum, 652.
—nerifolia, 652.
—resinifera, 652.
—tirucalli, 652.
Euphorbium, 652.
—poisoning by, 652.
Evidence, 14.
—circumstantial in poisoning, 484.
—documentary, 14.
—in insanity, 422.
—medical, 14.
—moral, in poisoning, 484.
—oral, 14, 17.
—rules for giving, 19.
Exalgin, 716.
Examination, in case of exhumation, 85.
—in chief, 13.
—of injured person, 239.
—post-mortem, 65.
Excitement, as a cause of death in injuries, 272.
Exhaustion, 180.
—psychoses, 405.
Exhumation, 85.
—disinfectants in case of, 85, 86.
—of infants, 177.
—report in case of, 86.
—rules for, 85.
—time of, 86.
Expectation of life, 439.
—rules for calculating, 439.
Experts, printed opinions of, 17.
Expert witness, 19.
Exposure of a newly-born child, 387.
Eyes, changes in, 123, 126, 127.
—colour of, in identification, 45.
—decomposition of, 135.
—gouging out of, 274.
—in hanging, 157.
—in identification, 45.
—injuries of, 274.
—in strangulation, 165.
—in suffocation, 175.

F

Face, bones of, 273.
—injuries of, 273.
Factories Act, 43.
False vesicles, 207.
Features, 24, 45.
—decomposition of, 47.
Feeble-mindedness, 395.
Fees to medical witnesses, 11, 12.
Feigned, diseases, 463.
—insanity, 412; distinguishing features of, 412.
—poisoning, 479.
Female, distinguishing points between Hindu and Mahomedan, 26.
—examination of, in rape cases, 335, 336.
Female organs, malformation of, 309.
Femur, ossification in lower epiphysis, 68.
Fencing posture, 204.
Ferric chloride, 612.
Ferrous sulphate, 612.
Fevers, in life assurance, 438.
Finger impressions, 49, 50.
—classification of, 50.
—development of, 53.
—forging of, 53.
Finger, marks, 68; in throttling, 163.
—print bureau, 53.
—prints, 53.
Fire-arm, 62; direction from where fired, 238.
—wounds by, 228.
Fish, poisoning by, 667.
Flaccidity, in muscles after death, 130.
Flame test for barium, 622.
Flash, of light, by fire-arm, 62.
Flash, of lightning, 62.
Fleitmann's test, 558.
Fleming's tincture of aconite, 791.
Floatation of decomposed body, 144.
—circumstances modifying, 144.
—period of, 145.
—power of, 144.
Florence test, 109, 110.
Flies, in decomposition, 137.
Fly, agaric, 760.
—papers, 546.
—powder, 546.
—spanish, 658.
—water, 546.
Fœtal circulation, changes in, 378.
Fœtal heart sounds, 320.
Fœtal lungs, changes in, 370.
Fœtal movements, 320.
Fœtus, 364.
—development of, 364.
—presence of, in pregnancy, 321.
—viability of, 330.
Folie circulaire, 403.
Food poisoning, 666.
Foot mark, 54.
Foot prints, 24, 54.
—casts of, 55.
—of newly born infants, 55.
Foramen ovale, 378.
Foreign bodies, in suffocation, 174.
Forensic medicine, definition of, 1.
Formaldehyde, poisoning by, 696.
Formalin, 696.
Foxglove, poisoning by, 780.
Fractures, 68.
—distinction between ante-mortem and post-mortem, 301.
—of bones, 301.
—of face bones, 273.
—of ribs, 282.
—of skull, 267.
—of spine, 279.
—of sternum, 283.
—process of union in, 243.
Fragilitas ossium, 301.
Fragments, examination of, 77.
Frost bite, 194.
Frost erythems, 194.
Fungi, poisonous, 760.
Fusel oil, poisoning by, 695.

G

Gait, 25, 62.
Gajar, 359.
Galena, 591.
Gall-bladder, examination of, 72.
—injuries of, 293.
Galton system, 48, 49.
Gamboge, 651.
—poisoning by, 651.
Gangrene, in ergot poisoning, 633.
Ganja, 753.
Gardenal, 714.
Gasoline, 729, 730.
Gelsemium nitidum (sempervirens), 774.
—poisoning by, 774.
—tests for, 775.
General diseases, as cause of sterility, 306, 310.
General medical council, 442.
General paralysis of the insane, 408.
Genital organs, condition of, in hanging, 157.
Genitals, in virginity, 311.
—hæmorrhage from, in infants, 379.
Ghagharbel, 359.
Gibson's vermin-killer, 765.
Giesel's test for cocaine, 746.
Girls, development of breasts in, 40.
—kidnapping of, 42.
Glass, powdered, poisoning by, 669.
Glonoin oil, 727.
Gloriosa superba, poisoning by, 654.
Glottis, œdema of, 174.
—spasm of, 492.
Glucosides, separation of, 481.
Gogari lakdi, 764.

Gold, poisoning by, 624.
Gonorrhœa, in rape, 338.
—in unnatural offences, 351, 352.
Goodell's sign, 319.
Goose flesh, 182.
Goose skin, 182.
Gossypium, 358.
Goulard's extract, 590.
Gout, in life assurance, 438.
Grains (poisonous food), 763.
Green vitriol, 612.
Grievous injury, 239.
Group reagents for alkaloids, 483.
Guaiacum test, 92.
Gumchi, 636.
Gunshot wounds, 228.
—appearances of, 229, 231, 234.
Gutzeit's test, 557.

H

Habit, in identity, 25, 62.
—influence of, as regards poison, 473.
Hæmatin, 98.
Hæmatoma, 265.
Hæmin crystals, 96.
—crystal test, 96.
Hæmochromogen, 97, 98.
—crystal test, 97.
Hæmoptysis, in life assurance, 437.
Hæmorrhage, as cause of death, in infants, 379.
—in wounds, 245.
Hair, 24, 40, 48, 117.
—animal, 117.
—change in colour of, 48.
—character of, 118.
—chemical examination of, 48.
—detection of colour of, 48
—examination of, 117.
—fibres, 117.
—nature of, 117.
—source of, 118.
Hallucination, 390.
Hand, blackening of, by fire-arm, 256.
Handwriting, 25, 60.
Hanging, 154.
—and strangulation, 170, 171.
—causes of death in, 155.
—definition of, 154.
—fatal period in, 155.
—illustrative cases, 159, 160.
—judicial, 155.
—ligature mark in, 156.
—medico-legal questions in, 158.
—nature of ligature in, 154.
—post-mortem appearances in, 156.
—symptoms in, 154.
—treatment in, 155.
Hankin's process, 482.
—test for cocaine, 746.
Harrison's test for seminal stains, 112.
Hartshorn, 522.
—spirits of, 522.
Hashish, 753.
Hartal, 544.
Head, injuries of, 265.
—post-mortem examination of, 69.
Health, state of, in modifying action of poison, 474.
Heart, chambers of, 70.
—decomposition of, 142.
—examination of, 70.
—rupture of, 285.
—weight of, 74.
—wounds of, 283.
Heat, 190, 195.
—apoplexy, 196.
—death from, 195.
—exhaustion, 195.
—stiffening, 132.
—stroke, 196.
—treatment, 196.
Hebephrenia, 397, 398, 407.
Hegar's sign, 319.
Height, 34.
—tables of, 34.
Hellebore, 645.
—black, 648.
—green and white, 645.
Helleborous niger, poisoning by, 648.
Hemlock, Spotted, poisoning by, 818.
Hemp, Indian, 752.
Henbane, poisoning by, 750.
Hermaphrodites, 28.
Hermaphroditism, 28.
Hernia, in life assurance, 438.
Heroin, 674.
Hierapicra, 653.
High Court, 9.
Hindu, females, 26.
—males, 25.
Hindus, 25.
Hing, 361.
Hingool, 577.
Hirakashi, 612.
Hirwa, 543.
Homatropine hydrobromide, 743.
Homicidal wounds, 252.
Homicide, in burns, 208.
—drowning, 188.
—in hanging, 159.
—in starvation, 193.
—in strangulation, 168.
—in suffocation, 176.
Hoppe-seyler's test, 810.
Hornets, stings by, 665.
Horoscope, 41.
Human, blood, detection of, 99.
—poisoning, 469.
—spermatozoa, 114.
Hunger strike, 193.
Hunter's vermin-killer, 765.
Husemann's test, 683.
Hydrochloric acid, poisoning by, 502.
Hydrocyanic acid, poisoning by, 797.
Hydrofluoric acid, poisoning by, 503.

Hydrogen cyanide, 797.
—sulphide, poisoning by, 812.
Hydrostatic test, 373.
—method of, 373.
—objections in, 373, 374.
Hymen, 311.
—rupture of, 312, 313.
Hyoscine, 750.
—hydrobromide, 750.
Hyoscyamine, 750.
Hyoscyamus niger, poisoning by, 750.
Hyperæmia in poisoning, 476, 477.
Hypnogen, poisoning by, 712.
Hypnotism, 433.
Hypomania, 399.
Hypospadias, 305.
Hypostasis, 123, 128.
—in internal organs, 129.

I

Icard's test, 126.
Identification, 24.
—in life assurance, 439.
—of a dead body, 24.
—of a living person, 24.
—of bones, 24, 31.
—of fragmentary remains, 24, 77.
Identity, 23.
—mistaken cases of, 63.
Idiocy, 394.
Idiosyncrasy, influencing the action of poison, 473.
Illegitimate child, 326.
Illusion, 390.
Imbecility, 395.
Immaturity, 378.
Impotence, 303.
—causes of, in the female, 307.
—in the male, 304.
—questions relating to, 303.
Impulse, 390.
Impulsive insanity, 430.
Inab-es-Salib, 751.
Incised wounds, 223.
—character of, 224.
Index, cephalic, 27.
Indian, Christians, 27.
—corn, 763.
—hemp, 752.
—ink, 58.
—liquorice, 636.
—Lunacy Act, 414.
—Succession Act, 326, 327.
—tobacco, 780.
—tree sponge, 652.
Indrayan, 631.
Inebriant poisons, 470, 688.
Infamous conduct, 445.
Infant, newly born, 68, 367.
Infanticide, 41, 45, 367.
—appearances of respiration in, 370, 371.
—causes of death in, 378.
Infanticide, definition of, 367.
—evidence of survival in, 377.
—fracture of cranium in, 383.
—hydrostatic test in, 373.
—legal bearing in, 367.
—poisoning in, 386.
—post-mortem examination in, 370.
—static test in, 372.
—wounds in, 384.
Inflation, artificial, of lungs, 375.
Injured person, examination of, 239.
Injured person, neglect of, 246.
Injuries, mechanical, 217.
—of abdomen, 288.
—of bones, 301.
—of chest, 281.
—of eyes, 274.
—of face, 273.
—of head, 265.
—of muscles, 300.
—of neck, 278.
—of nose, 275.
—of spinal cord, 279.
—of spine, 279.
Injury, 239.
—age of, 242.
—grievous, 239.
—nature of, 239.
—in survivorship, 152.
Inorganic poisons, 470, 528.
Inquest, Coroner's 2 ; Police, 2.
Inquisition, 417, 418.
Insane ear, 393.
Insanity, 389.
—associated with nervous diseases, 393, 408.
—causes of, 391.
—civil responsibility in, 419.
—classification of, 393.
—criminal responsibility in, 424.
—definition of, 389.
—diagnosis of, 411.
—feigned, 412.
—indications of, 392.
—in life assurance, 439.
—mental condition in, 411.
—personal history in, 411.
—physical examination in, 411.
—admission into a mental hospital in case of, 413.
—stigmata of degeneration in, 393.
Insolation, 196.
Instantaneous rigor, 132, 133.
—cases of, 133.
Insurance, life, 436.
Intermittent uterine contractions, 319.
Interval, lucid, 391.
Intestines, decomposition of, 141.
—examination of, 72.
—injuries of, 290.
—rupture of, 290.
Intoxication, alcoholic, 433, 434.
Intra-uterine maceration, 368, 369.
Iodide of potassium, 539.

Iodine, acute poisoning by, 538.
—chronic poisoning by, 539.
—tests for, 540.
Iodism, 539.
Iodoform, poisoning by, 706.
—tests for, 707.
Ipecacuanha, as an emetic, 488.
Ipomœa hederacea, 651.
Ipomœa purga, 651.
Ipomœa turpethum, 652.
Iron, 611.
—perchloride, 612.
—poisoning by, 612.
—sulphate, 612.
Irresistible impulse, cases of, 430, 431.
Irrespirable gases, 806.
Irritant poisons, 528.
Irritants, 470.
Izal, poisoning by, 514.

J

Jacquemier's sign, 318.
Jalapin, 651.
Jalap, poisoning by, 651.
Jamalgota, 628.
James's powder, 567.
Jangli, arandi, 653.
—*pyaz,* 654.
Jasat, 603.
—*bhashm,* 604.
Jasmine, yellow (carolina), 774.
Jatropha, curcas, poisoning by, 652.
—multiphida, poisoning by, 653.
—urens, poisoning by, 653.
Javakhar, 523.
Jaw, changes in, from age, 40.
—necrosis of, 533.
—phossy, 533.
Jayfal, 359.
Jequirity, 636.
Jessamine, 774.
Jeye's disinfecting fluid, 514.
Juar kadvi, 804.
Judicial, hanging, 155.
—inquisition, 417, 418.
—proceeding, 17, 18.
—punishment, 41, 44.
Juniperus sabinus, poisoning by, 650.
—oil of, 650.
Jurors, 9.
Jury, 9, 14.
—common, 9.
—special, 9.
Justifiable miscarriage, 355.
—indications for producing, 356.

K

Kachu, 655.
Kafoor, 758.
Kaju, 638.
Kakmachi, 751.
Kakmari, 655.
Kakphal, 655.
Kaladana seeds, 651.
Kala Dhatura, 733.
Kalai, 613.
Kali-katuki, 648.
Kalmi sora, 616.
Kaner, 360, 784.
Karabi, 784.
Karan, 784.
Karela, 359.
Karit, 359.
Kasis, 612.
Kasoomba, 686.
Kastle-Meyer test, 92, 94.
Katai, 751.
Katkar, oil, 655.
Katatonia, 397, 398, 406.
Kavach, 672.
Keller's test, 786.
Kerosene oil, 729.
Kesar, 359.
Kesari Dal, 763.
Khas-khas, 673 ; *-ka tel ;* 674.
Khadya nag, 654.
Kharsivan, 545.
Khorasani Ajwayan, 750.
Kidnapping, 41, 42.
Kidney, decomposition of, 142.
—examination of, 72.
—rupture of, 296.
—size of, 72.
—weight of, 74.
King's yellow, 544.
Kirmani Owa, 757.
Knives, rust stains on, 107.
Kodro, 763.
Kodon, 763.
Krait, bite by, 662.
Kuchila, 764.
—*lata,* 764.
Kunkel's test, 810.
Kurchi, bark, 771.

L

Labarraque's disinfecting fluid, 536.
Labia, majora, 311.
—minora, 311.
Labour, precipitate, 381.
—protracted, 379.
Laburnum, 649.
Lacerated wounds, 227.
Laceration of brain, 270.
Lachrymators, 815.
—poisoning by, 817.
Lal Chitra, 220, 359, 644.
Lal Mirch, 635.
Landolt's test, 512.
Lapis infernalis, 610.
Larynx, decomposition of, 140, 141.
—examination of, 70.
—spasm of, as cause of death of infants, 379.
—wounds of, 278.

Lathyrism, 763.
Lathyrus sativus, poisoning by, 763.
Laughing gas, poisoning by, 814.
Lavang, 359.
Law in relation to medical men, 442.
Lead, 590.
—acetate, 590.
—acute poisoning by, 593.
—carbonate, 591.
—chemical tests for, 597.
—chronic poisoning by, 594.
—compounds of, 590.
—elimination of, 599.
—medico-legal points in, 598.
—oxide, 592.
—subacetate, 590.
—treatment in, 593, 595.
Leading question, 13.
Legal procedure, at an inquest, 2.
Legitimacy, 326.
—Act, 326.
—medico-legal points in, 328.
Legitimate child, 326.
Lesbian love, 352.
Leucomaines, 667.
Leucomalachite green test, 92, 94.
Levant nut, 655.
Lewisite, 816.
Lex's test, 512.
Libermann's test, 513.
Liebig's test, 803.
Life assurance, 436.
—definition of, 436.
—examination of internal organs in, 440.
—family history in, 439.
—general appearance in, 440.
—personal history in, 437.
Ligature, 154.
—in strangulation, 162.
—mark in hanging, 156.
—marks, in a dead body, 68
Lightning, 210.
—illustrative cases of, 211, 212.
—post-mortem appearances in, 210.
—symptoms in, 210.
Lipowitz's test, 534.
Lips, in identification, 45.
—injuries of, 276.
Liquor, ammoniæ fortis, 522.
—amnii, 360.
—potassæ, 522.
—sodiæ, 523.
Litharge, 591.
Live-birth, 369.
—evidence of, 369.
Liver, decomposition of, 141.
—examination of, 72.
—injuries of, 292.
—size of, 72.
—weight of, 74.
Lividity, cadaveric, 123, 128.
Lobelia, 780.
—inflata, 780.
Lobelia, poisoning by, 780.
Lobeline, 780.
Local diseases, in impotence, 306, 310.
Lochia, 323.
—alba, 323.
—rubra, 323.
—serosa, 323.
Loha, 611.
Lolium temulentum, poisoning by, 763.
Loluin, 763.
Lords and ladies, 654.
Lucid interval, 391, 401.
Lucifer matches, poisoning by, 528.
Luminal, poisoning by, 714.
Lunacy (see Insanity), 389.
Lunacy certificates, 414.
Lunar, caustic, 610.
Lunatics, civil responsibility of, 419.
—criminal responsibility of, 424.
—discharge of, 417.
—escape and re-capture of, 418.
—illegal detention of, 419.
Lungs, changes in, after live-birth, 370.
—decomposition of, 142.
—examination of, 70.
—hydrostatic test for, 373.
—weight of, 74.
—wounds of, 283.
Lust murder, 339.
Lynching, 159.
Lysol, poisoning by, 514.

M

Maccai, 763.
Maceration, signs of, 368.
Madak, 686.
Madar, 359, 640.
—juice, 641.
Maggots, in decomposition, 137, 138.
Magistery of bismuth, 608.
Magistrates, 9; classes of, 9.
Magnan's symptom, 745.
Magnesium, poisoning by, 620.
—sulphate, 620.
Magnus's test, 125.
Mahomedan, females, 26.
—males, 25.
Mahomedans, 25.
Main phal, 359.
Maize, 763.
Majority, attainment of, 41, 43.
Majun, 752.
Makoi, 751.
Male organs, injuries of, 298.
—local disease of, 306.
—malformations of, 305.
Malformations, 305, 309.
—as a cause of death in infants, 379.
Malingering, 463.
Malkangani, 359.
Malpraxis, 453.
—cases of, 457, 458.
Malonurea, 712.

Mammary changes, 318.
Mania, 398.
Manic-depressive insanity, 398.
Mansil, 544.
Manslaughter, 454.
Marking ink, 638.
Marking nut, 220. 638.
—poisoning by, 638.
Marquis's test, 683.
Marriage contract, 41, 42.
Marsden's vermin-killer, 765.
Marsh's test, in antimony, 572.
—in arsenic, 556.
Masked epilepsy, 410.
Massicot, 591.
Mayer's reagent, 483.
Mc Naughten case, 426.
Meadow saffron, 646.
Mechanical antidotes, 488.
Mechanical, injuries, 217.
—poisons, 470, 669.
—violence in criminal miscarriage, 359.
—violence in infanticide, 382.
—violence in rape, 336.
Meconic acid, 683.
Meconium, in bowel, 376.
Medical certificates, 14.
—in lunacy, 414.
Medical, evidence, 14.
—Act, 442.
—examiner, 436.
—referee, 441.
—witness, 12, 18, 19.
Medico-legal reports, 14, 15.
Medinal, poisoning by, 714.
Melancholia, 401.
Memory, 25, 62.
Meninges, 70.
Menses, cessation of, in pregnancy, 316.
Menstrual blood, 89, 91, 315, 316.
Mental hospital, 413.
—discharge from, 417.
—escape from, 418.
—illegal detention in, 419.
—restraint in, 413.
Mental power, 25, 62.
Merbaphen, 578.
Mercurial tremors, 582.
Mercuric, chloride, 576.
—cyanide, 576.
—iodide. 576.
—methide, 577.
—oxide, 575.
—nitrate, 577.
—sulphide, 577.
Mercurochrome, 578.
Mercurous chloride, 577.
Mercurous nitrate, 578.
Mercury, 575.
—acute poisoning by, 579.
—chemical tests for, 582.
—chronic poisoning by, 582.
—compounds of, 575.
Mercury, diagnosis of, 579.
—dimethyl, 577.
—medico-legal points in, 583.
—post-mortem appearances of, 581.
—treatment of, 580, 582.
Mesentery, decomposition of, 141.
Mesmerism, 433.
Metallic irritants, 470, 542
Methæmoglobin, 98.
Methi, 359.
Methyl alcohol, poisoning by, 694.
Methylamine, 667.
Methylconiine, 818.
Methylated spirit, 688, 694.
Methyl salicylate, poisoning by, 518.
Metia, sindur, 591.
Microscopic examination, of blood, 95.
—of semen, 113.
Middle ear, change in, for live-birth, 377.
Mild thermic fever, 195, 196.
Milk hedge, 652.
Miller's rat powders, 765.
Millon's reagent, 512.
Millon's test, 512.
Mineral acids, 491.
—poisoning by, 491.
—post-mortem appearances of, 491.
—symptoms of, 491.
—treatment of, 491.
Mineral poisons, analysis of, 484.
Mineral stains, 107.
Minium, 591.
Mirbane, oil of, 724.
Miscarriage, 354.
—artificial, 354, 355.
—causes of, 354.
—classification of. 354.
—criminal, 356, 365.
—definition of, 354.
—evidence of, 362.
—justifiable, 355.
—legal bearing in, 357.
—means to induce, 357.
—natural, 354, 365.
Mistaken identity, cases of, 63.
Mitscherlich's test, 534.
Mitha Zahar, 790.
Mochni, 763.
Modes of death, 119.
—in survivorship, 152.
Momordica charantia, 359.
Monk's hood, 790.
Monorchids, 306.
Monophenylamine, 719.
Monster, 326.
Monte Verde's test, 125.
Morbus cæruleus, 378.
Moringa Pterygosperma, 359.
—poisoning by, 656.
Morison's pills, 653.
Morning sickness, sign of pregnancy, 316, 317.
Morphine, 674.

Morphine, tests for, 683.
Morphinism, 687.
Morphinomania, 687.
Mucuna pruriens, poisoning by, 672.
Mummification, 124, 150.
—artificial, 150.
—in fœtus, 369.
—time of, 150.
Murdasang, 591.
Muriatic acid, poisoning by, 502.
Muscarine, 668, 760.
Muscles, contusions of, 300.
Mushrooms, poisoning by, 760.
Mustard gas, 815.
Muthanol, 608.
Mutilated, bodies, examination of, 77.
—remains, 30.
Mydalein, 668.
Mylabris chicorii, 658.
—pustulata, 658.
Myristica fragrans, 359.
Mytilotoxine, 667.

N

Nœpala, 628.
Nagdown, 655.
Nails, 138.
—arsenic in, 562.
Namak, 623.
Naphtha, 721.
Naphthalene, 721.
Naphthalin, 721.
Narcotic poisons, 673.
Narcotine, 675.
Neck, injuries of, 278.
Necrophilia, 339.
Needles, poisoning by, 671.
Neoarsphenamine, 545.
Neokharsivan, 545.
Neosalvarsan, 545.
Neotrepol, 608.
Nerin, 785.
Nerium odorum, 360, 784.
—poisoning by, 784.
Nervous diseases in life assurance, 439.
Neuraline, 794.
Neural poisons, 818.
Neurin, 667.
Neurosine, 756.
Neurotic poisons, 673.
Neurotics, 470.
Nickel, 626.
—carbonyl, 626.
—chemical tests for, 626.
Nicotiana tabacum, 776.
Nicotine, poisoning by, 776.
—chemical tests for, 778.
Nicotinine, 776.
Nightshade, deadly, 739.
—woody, 751.
Nila tutia, 586.
Nishotar, 652.
Nitrate, of lead, 591.
Nitrate, of mercury, 577, 578.
—of potash, 616.
—of silver, 610.
Nitre, 616.
Nitric acid, poisoning by, 499.
—illustrative cases, 501.
—tests for, 500.
Nitric-oxide-hæmoglobin, 811.
Nitrite of amyl, 696.
Nitrobenzene, poisoning by, 724.
Nitrobenzol, poisoning by, 724.
Nitrogen monoxide, poisoning by, 814.
Nitro-glycerine, poisoning by, 727.
Nitroxyhæmoglobin, 811.
Nitrous oxide, poisoning by, 814.
Nobel's blasting oil, 727.
Non-metallic poisons, 470, 528.
Nose, injury of, 275; in identification of, 45.
Notes in evidence, 20.
Novarsenobenzol, 545.
Novarsenobillon, 545.
Novarsurol, 578.
Novocaine, 743.
Nubile virgins, 337.
Nuts, cashew, 638.
—levant, 655.
—marking, 638.
—soap, 657.
Nux vomica, 764.
—seeds, 764.
Nylofanol, 718.
Nymphæ, 311.

O

Oath, 12.
Obsession, 390.
Occupation, in life assurance, 437.
—marks, 25, 60.
Œsophagus, decomposition of, 142.
—examination of, 70, 71.
—wounds of, 278.
Oil, castor, 627.
—croton, 628.
—kerosene, 729.
—of bitter almonds, 724, 798.
—of eucalyptus, 732.
—of mirbane, 724.
—of poppy seed, 674.
—of savin, 358.
—of turpentine, 730.
—of vitriol, 493.
Oleander, sweet-scented (white), 784.
—yellow, 787.
Omentum, decomposition of, 141.
Ophidia, 660.
Ophitoxæmia, 662.
Opium, 673.
—diagnosis of, 679.
—habit (eating), 686.
—malwa, 674.
—medico-legal points in, 684
—official preparations of, 675.

Opium, Patna, 674.
—poisoning by, 677.
—proprietary medicines of, 676.
—tests for, 683.
—treatment for, 681.
Oral evidence, 14, 17.
Ordeal bean, 773.
Organic acids, poisoning by, 504.
Organic poisons, 470, 627.
Ornaments, in identification, 25, 61.
Orpiment, 544.
Osmic acid, 626.
Osmium tetroxide, 626.
—poisoning by, 626.
Ossification, 35.
—table of, 36, 37, 38, 39.
Os uteri, 323, 325.
Otto's process, 481.
Ovum, 321.
—presence of, 321.
Oxalates, 509.
Oxalic acid, 504.
—chemical analysis of, 507.
—poisoning by, 504.
—post-mortem appearances of, 506.
—treatment of, 506.
Oxydimorphine, 637.
Oxyhæmoglobin, 97, 98.

P

Pæderastia, 347.
Pancreas, injuries of, 291.
Papadkhara, 523.
Papaverine, 675.
Papaya seed, 359.
Papita, 359, 764.
Para, 575.
Paraffin oil, 729.
Paraffins, 729.
Paraldehyde, poisoning by, 710.
Paralysis, general of the insane, 408.
—in lead poisoning, 595.
Paranoia, 406.
Paris green, 543.
Parole, 14.
Parsi, males, 27.
—women, 27.
Parson in the pulpit, 654.
Paspalam scrobiculatum, 763.
Past diseases, in life assurance, 437.
Paternity, 328, 332; cases of disputed, 104.
Pattinson's white lead, 592.
Pearl ash, 523.
Pearl white, 608.
Pellagra, 763.
Pelvis, sex from, 31.
Penal Code, Indian (See Appendix VII).
Penetrating wounds, 225.
Penge murder case, 193.
Penis, wounds of, 298.
Perforation, in poisoning, 478.
Peripheral poisons, 470, 818.
Peritoneum, examination of, 71.
Permanganate of potassium, 526.
—in opium poisoning, 681.
—poisoning by, 526.
Peronin, 674.
Persian insect powder, 649.
Personal, habits, in life assurance, 437.
—identity, 23.
Petrol, poisoning by, 728.
Petroleum, poisoning by, 728.
Pharaoh's serpents, 576.
Pharbitisin, 651.
Pharbitis seeds, 651.
Phenacetin, poisoning by, 716.
Phenazone, poisoning by, 716.
Phenic acid, poisoning by, 510.
Phenobarbital, 714.
Phenobarbitonum, 714.
Phenolphthalein, 94.
—reagent, 94.
Phenol, poisoning by, 510.
Phenoquin, 718.
Phenyl alcohol, poisoning by, 510.
Phenyl-ethyl-barbituric acid, 714.
Phitkari, 619.
Phosgene, 807, 816.
Phosphine, 535.
Phosphine test, 534.
Phosphorus, 528.
—acute poisoning by, 529.
—chronic poisoning by, 533.
—tests for, 534.
—treatment of, 530, 533.
—trihydride, 535.
—varieties of, 528.
Phosphuretted hydrogen, 535.
Phossy-jaw, 533.
Photographers, 60.
Photograph, for identification, 45.
Phthisis, in life assurance, 437.
Physic nut, 652.
Physiological antidotes, 489.
Physostigmatis semina, 773.
Physostigma venenosum, 773.
Physostigmine, poisoning by, 773.
Picric acid, 516; poisoning by, 516.
—reagent, 484.
Picrotoxin, 655.
Pigmentation of skin, as a sign of pregnancy, 318.
Pila kaner, 787.
Pithing, 281.
Pithori, 652.
Placenta, disease of, as cause of death of infants, 379.
—prævia, 379.
Plantinic chloride, reagent, 484.
Platinum, poisoning by, 625.
Pleurisy, in life assurance, 437.
Plouquet's test, 373.
Plumbagin, 644.
Plumbago rosea, 220, 359, 644.
—poisoning by, 644.

Plumbago zeylanica, 220, 644.
Plumbism, 594.
Pneumonia, in life assurance, 437.
Poisoning by, abrus precatorius, 636.
—acetic acid, 519.
—aconite, 790.
—alcohol, 688.
—alkalies, 522.
—allantiasis, 667.
—almonds, bitter, 800.
—aloes, 653.
—aluminium, 619.
—amanita, muscaria, 760; phalloides, 760.
—ammonia, 522, 523.
—ammonium carbonate, 523.
—amyl alcohol, 695.
—amyl nitrite, 696.
—aniline, 719.
—antifebrin, 716.
—antimony, 567.
—antipyrin, 716.
—ants, 665.
—argemone mexicana, 655.
—arsenic, 542.
—artemisia maritima, 757.
—arum maculatum, 654.
—aspirin, 519.
—atropine, 739.
—barium, 621.
—bees, 665.
—belladonna, 739.
—benzene (benzol), 722.
—binoxalate of potash, 509.
—bismuth, 608.
—bisulphide of carbon, 812.
—boracic acid, 540.
—borax, 540.
—boron, 540.
—botulism, 667.
—bromine, 537.
—bromoform, 706.
—brucine, 764.
—cadmium, 623.
—calabar bean, 773.
—calotropis gigantea (procera), 640.
—camphor, 758.
—cannabis indica, 752.
—cantharides, 658.
—capsicum seeds, 635.
—carbazotic acid, 516.
—carbolic acid, 510.
—carbon dioxide, 806.
—carbon disulphide, 812.
—carbon monoxide, 807.
—carbon tetrachloride, 705.
—castor oil, 627.
—cerbera thevetia, 787.
—chloral hydrate, 707.
—chlorine, 536.
—chloroform, 700.
—chopped animal hair, 671.
—chromium, 614.
Poisoning by, cinchophen, 718.
—citric acid, 522.
—coal gas, 807.
—coal-tar naphtha, 721.
—cobalt, 626.
—cocaine, 743.
—cocculus indicus, 655.
—cocculus suberosus, 655.
—colchicum, 646.
—colocynth, 631.
—conium maculatum, 818.
—copper, 586.
—corrosive sublimate, 579.
—creolin, 514.
—creosote, 515.
—cresol, 514.
—crinum deflexun, 655.
—croton oil, 628.
—curara, 818.
—cuscuta reflexa, 652.
—cyanide of potassium, 799.
—cytisus laburnum, 649.
—datura, 733.
—delphinium staphisagria, 648.
—diamond dust, 671.
—digitalis, 780.
—dinitrobenzene, 726.
—ergot, 632.
—eserine, 773.
—ether, 698.
—ethyl alcohol, 688.
—eucalyptus oil, 732.
—euphorbium, 652.
—fish, 667.
—food, 666.
—formaldehyde, 696.
—fungi, 760.
—gamboge, 651.
—gelsemium, 774.
—glass, 669.
—gloriosa superba, 654.
—gold, 624.
—grains, 763.
—helleborus niger, 648.
—hemlock, 818.
—henbane, 750.
—hornets, 665.
—hydrochloric acid, 502.
—hydrocyanic acid, 797.
—hydrofluoric acid, 503.
—hydrogen sulphide, 813.
—hyoscyamus, 750.
—Indian hemp, 751.
—Indian tobacco, 780.
—iodine, 538.
—iodoform, 706.
—ipomœa turpethum, 652.
—iron, 611.
—izal, 514.
—jalap, 651.
—jatropha curcas, 652.
—jatropha multiphida, 653.
—jatropha urens, 653.
—juniperus sabinus, 650.

Poisoning by, *kala dana* seeds, 651.
—kerosene oil, 729.
—lathyrus sativus, 763.
—laughing gas, 814.
—lead, 590.
—lewisite, 816.
—lobelia, 780.
—lolium temulentum, 763.
—luminal, 714.
—lysol, 514.
—magnesium, 620.
—medinal, 714.
—mercury, 575.
—methyl alcohol, 694.
—methyl salicylate, 518.
—mineral acids, 491.
—moringa pterygosperma, 656.
—mushrooms, 760.
—mustard gas, 815.
—naphtha, 721.
—naphthalene, 721.
—needles, 671.
—nerium odorum, 784.
—nickel, 626.
—nicotine, 776.
—nightshade, deadly, 739.
—nitric acid, 499.
—nitrobenzene, 724.
—nitrogen monoxide, 814.
—nitroglycerine, 727.
—nux vomica, 764.
—oleander, 784.
—opium, 673.
—osmium, 626.
—oxalic acid, 504.
—paraldehyde, 710.
—paspalam scrobiculatum, 763.
—petrol, 728.
—petroleum, 728.
—phenacetin, 716.
—phenol, 510.
—phosgene, 816.
—phosphorus, 528.
—physostigmatis semina, 773.
—picric acid, 516.
—picrotoxin, 655.
—platinum, 625.
—plumbago rosea and zeylanica, 644.
—potassium, 616.
—p. permanganate, 526.
—prontosil, 718.
—prussic acid, 797.
—quinine, 783.
—ricinus communis, 627.
—ruta graveolens, 657.
—salicylic acid, 517.
—salvarsan, 560.
—santonin, 757.
—sapindas trifoliatus, 657.
—scammony, 651.
—scorpions, 665.
—semecarpus anacardium, 638.
—silver, 610.
Poisoning by, snakes, 660.
—sodium, 623.
—stigmata maides, 763.
—strychnine, 764.
—sulphanilamide, 718.
—sulphonal, 711.
—sulphur dioxide, 814.
—sulphuretted hydrogen, 813.
—sulphuric acid, 493.
—tartar emetic, 567.
—tartaric acid, 521.
—taxus baccata, 650.
—terminalia Bellerica (Belleric myrobalans, *Bahera*), 657.
—thallium, 600.
—tin, 613.
—tobacco, 776.
—trinitrotoluene, 726.
—turpentine, 730.
—urginea scilla, 653.
—vegetable hairs, 672.
—veratrine, 646.
—veronal, 712.
—wasps, 665.
—yew, 650.
—zinc, 603.
—war gases, 815.
Poisonous insects, 665.
Poisons, 466.
—Act, 467.
—action of, 471.
—causes modifying the action of, 471.
—channels of elimination in, 471.
—chemical analysis of, 478.
—classification of, 470.
—definition of, 466.
—diagnosis of, 474.
—duty of medical man in suspected cases of, 484.
—law relating to, 466.
—methods of administering, 470.
—post-mortem appearances of, 476.
—sale of, 466.
—treatment in cases of, 486.
Police, inquest, 2.
—surgeon, 2.
Policy, in life assurance, 436.
Pomum Adami, in age, 40.
Poppy, capsules, 673.
—seeds, 673.
—seed oil, 674.
Poroscopy, 50.
Porphyroxine test, 683.
Post-epileptic insanity, 410.
Post-ka-doda, 673.
Post-mortem, calorieity, 128.
—delivery, 363.
—examination, 65.
—instruments for, 66.
—object of, 65; rules of, 65.
—report, 74.
—rigidity, 124.
—staining, 123, 128; time of, 128, 129.

Posture, fencing (pugilistic), 133.
Potassio-tartrate of antimony, 567.
Potassium, poisoning by, 616.
—arsenate, 543.
—arsenite, 543.
—binoxalate, 509.
—bromide, 537.
—carbonate, 523.
—chlorate, 618.
—chromate, 614.
—cyanide, 798.
—dichromate, 614.
—ferrocyanide, 107, 798; test, 815.
—hydrate, 522.
—hydroxide, 522.
—iodide, 539.
—nitrate, 616.
—permanganate, 526.
—sulphate, 618.
—sulphide, 619.
—sulpho-cyanide, 107.
Precipitin, 99.
Precipitin test, for blood, 99; for semen, 116.
Precipitate labour, 381.
Pre-epileptic insanity, 410.
Pregnancy, 315.
—average duration of, 328.
—legal questions in, 315.
—maximum period of, 329.
—minimum period of, 330.
—objective signs of, 317.
—signs of, in the dead, 321.
—subjective signs of, 316.
—unconscious, 386, 387.
Premature labour, 354.
Presumption, of death, 152.
—of survivorship, 152.
Preternatural combustion, 209.
Primary dementia, 396.
Primary relaxation, 130.
Professional secrets, 20, 450, 451.
Prontosil, poisoning by, 718.
Prosecuting Inspector, 13.
Protargol, 610.
Protracted labour, 379.
Prussian blue test, 803.
Prussic acid, 797.
Psychical influences, in impotence, 307, 310.
Psychic epilepsy, 410.
Ptomaines, 667
Puberty, in female, 303.
—in male, 304.
Pugilistic attitude (posture), 133.
Punctured wounds, 223, 225.
Purgatives, in criminal abortion, 358.
Putrefaction, 124, 135.
—circumstances modifying, 145.
—external phenomena of, 135.
—internal phenomena of, 140
—in water, 143.
—tables of, 139, 140.
Putty powder, 614.
Pyrogallic acid, poisoning by, 516.
Pyrogallol, poisoning by, 516.
Pyroxylic spirit, 694.

Q

Quickening, 317.
Quicksilver, 575.
Quick with child, 317.
Quinina, 783.
Quinine, as abortifacient, 358.
—poisoning by, 783.
Quotations, in medical evidence, 20.

R

Race, 24, 25.
—Caucasian, 27.
—in skeleton, 27.
—Mongolian, 27.
—Negro, 27.
Railway spine, 281.
Randia dumentorum, 359.
Rape, 41, 43, 333.
—accidents following, 339.
—age of the male in, 334.
—age of the victim in, 335.
—consent in, 333.
—definition of, 333.
—examination of accused in, 339.
—examination of clothes in, 336.
—examination of genitals in, 336.
—examination of victim in, 335.
—illustrative cases, 342.
—marks of violence in, 336.
—medico-legal questions in, 341.
Rasakapoor, 577.
Ras sindoor, 577.
Rati, 636.
Reaction, Weppen's, 646.
Reagent, Mayer's, 483.
—Scheibler's, 484.
—Sonnenchein's, 483.
—Wagner's, 483.
Realgar, 544.
Reception order, 415.
Rectum, injuries of, 290.
Red arsenic, 544.
—chromate, 614.
—lead, 591.
—pepper, 635.
—phosphorus, 528, 529.
—spirit of nitre, 499.
Re-examination, 14,
Regional injuries, 265.
Reinsche's test, 554, 572, 583.
Relaxation, primary, 130.
—secondary, 130, 133.
Reports, medico-legal, 14, 15.
Residence, in life assurance, 437
Respiration, artificial, 180, 181.
—cessation of, 123, 124.
—tests for cessation of, 126.
Responsibility, civil, 419.
—criminal, 424.

Responsibility, of a medical man for negligent acts of nurses or students, 455.
—of a physician in criminal matters, 450.
—of managers of a charitable hospital for negligent acts of its medical staff, 456.
Restraint of the insane, 413.
Revenchino shero, 651.
Rheumatism, acute, in life assurance, 439.
Rhitha, 657.
Ribs, 31.
—examination of, 70.
—fracture of, 282.
Ricin, 627.
Ricinus communis, poisoning by, 627.
Rigidity, cadaveric, 130, 131.
Rigor mortis, 130, 131.
—circumstances modifying, 132.
—conditions simulating, 132.
—duration of, 131.
—onset of, 131.
Rock oil, 728.
Rough on rats, 545.
Roussin's crystals, 778.
Russian fleas, poisoning by, 672.
Rust-stains, 107.
Ruta graveolens, poisoning by, 657.

S

Sabadilla, 645.
Sabji, 752.
Sadists, 339.
Safeda, 591.
Safed dhatura, 733.
Safed tutia, 604.
Sajjikhara, 523.
Sal ammoniac, 359.
Sal, de duobus, 618.
—polychreest, 618.
—prunelle, 616.
Sale of poisons, 466.
Salicylic acid, poisoning by, 517.
Salt of saturn, 590.
Salts, of sorrel, 509.
—essential, of lemon, 509.
Saltpetre, 615.
Salvarsan, 545.
—poisoning by, 560.
Sal volatile, 523.
Sanguineous mole, 321.
Sankhya, 542.
Sanocrysin, poisoning by, 624.
Santonin, poisoning by, 757.
Sapindas trifoliatus, 657.
Saponin, 657.
Saragwa, 359.
Satap, 657.
Saturnine poisoning, 594.
Sausage, poisoning by, 667.
Savin, 650.
Scalds, 198, 204.
Scalp, injuries of, 265.
Scammony, poisoning by, 651.
Scars, 24, 55.
—age of, 57.
—appearance of, 56.
—character of, 56.
—disappearance of, 56.
—growth of, 57.
—illustrative case, 57.
Schaffer's method of artificial respiration, 180.
Scheele's green, 543.
—acid, 798.
Scheibler's reagent, 484.
Scherer's test, 534.
Schonbein's test, 92.
Schultze's method, 375.
Schweinfurt green, 543.
Scopolamine, 751.
Scorpions, stings by, 665.
Scotch form of oath, 12.
Secondary relaxation, 130, 133.
Secrets, professional, 20, 450.
Sehund, 652.
Self-inflicted wounds, 261.
—illustrative cases of, 262.
Semecarpus anacardium, poisoning by, 638.
Semen, 109.
Seminal stains, 108.
—biological examination of, 108, 116.
—chemical examination of, 108, 109.
—microscopical examination of, 108, 113.
—physical examination of, 109.
Semisomnolence, 432.
Sessions, Court, 9.
—Judge, 10.
Sex, 24, 28.
—concealed, 30.
—doubtful, 30.
—in survivorship, 152.
—of decomposed bodies, 30.
—of skeleton, 31.
Sexlessness, 30.
Shah Daula's *chuha*, 395.
Shajna, 359, 656.
Sharagava, 656.
Sheep-dip, 546.
Shingarf, 577.
Shisha, 590.
Shock, 119, 245.
Shohaga, 540.
Sialkanta, 655.
Siddhi, 752.
Signs, of death, 123.
—of delivery, 322.
—of pregnancy, 316.
—of virginity, 311.
Silver, acute poisoning by, 610.
—arsphenamine, 545.
—chronic poisoning by, 611.

Silver, nitrate, 610.
—salvarsan, 545.
Simple injury, 239.
Sindur, 591.
Sirka, 519.
Skeleton, 27, 31.
Skiagraphy, 35.
Skin, changes in, 123, 127 ; in a newly-born infant, 377.
—diseases, in life assurance, 438.
—pigmentation of, 318.
Skull, 267.
—base of, 269.
—fracture of, 267.
Sleep, delivery during, 383.
—rape during, 341.
Smegma, 341.
Smothering, 173.
Snakes, 660.
—poisoning by, 662.
—treatment of, 662.
—varieties of, 660.
Snake, venom, 661.
—wood, 764.
Soamin, 545.
Soap lye, 523.
Soap nuts, 657.
Sodium, poisoning by, 623.
—arsenate, 543.
—arsenite, 543.
—aurothiosulphate, 624.
—barbitone, 714.
—borate (biborate), 540.
—cacodylate, 544.
—carbonate, 523.
—chloride, poisoning by, 623.
—fluoride, 503.
—hydrate, 522.
—hydroxide, 522.
—salicylate, 517.
Sodomy, 347.
—examination of the active agent in, 352.
—examination of the passive agent in, 349.
Softening, in poisoning, 477.
Solanine, 751.
Solanum dulcamara, 751.
—indicum, 751.
—jacuinii, 751.
—nigrum, 751.
—tuberosum, 751.
Soluble barbitone, 714.
Somalkhar, 542.
Somatic death, 119.
Somnambulism, 432.
Somniferous poisons, 470, 673.
Somnolentia, 432.
Sona, 624.
Sonnenschein's reagent, 483.
Sorakhar, 615.
Sorrel, salt of, 509.
Sowa, 359.
Spanish fly, 658.
Spasm, cadaveric, 131, 132.
Spectroscopic, examination of blood, 97.
—test, for carbon monoxide, 810.
Speech, 25, 61.
Spermatozoa, 114, 338.
Spinal cord, examination of, 73.
—injury of, 279.
Spinal poisons, 470, 764.
Spine, concussion of, 281.
—examination of, 73.
—fracture of, 279.
—injuries of, 279.
Spirit, of salts, 502.
—of turpentine, 730.
Spleen, decomposition of, 141.
—examination of, 72.
—illustrative cases, 295.
—rupture of, 293, 294.
—size of, 72.
—weight of, 74.
—wounds of, 295.
Spontaneous combustion, 209.
Spotted hemlock, poisoning by, 818.
Squill, 653.
Staining, post-mortem, 123, 128.
Stains, blood, 89.
—dye, 107.
—mineral, 107.
—rust, 107.
—seminal, 108.
—vegetable, 107.
Stannic chloride, 613.
Stannous chloride, 613.
Staphisagrine, 648.
Starvation, 190.
—acute, 190.
—chronic, 190.
—conditions influencing, 191.
—fatal period of, 190.
—medico-legal question in, 192.
—post-mortem appearances in death from, 191.
—symptoms of, 190.
—treatment of, 191.
Stas's process, 481.
Static test, 372.
Stature, 81, 82.
Stave sacre, poisoning by, 648.
Sterility, 303.
—in females, 307.
—in males, 304.
—questions relating to, 303.
Sternum, fracture of, 283.
Sternutators, 815.
—poisoning by, 817.
Stibamine, 568.
Stibenyl, 568.
Stibin, 568.
Stibosan, 568.
Stigmata maides, poisoning by, 763.
Stigmata of degeneration, 393.
St. Ignatius beans, 764.
Still-births, 368.

Still-born child, 368.
Stomach, decomposition of, 141.
—examination of, 71.
—post-mortem digestion in, 151.
—effects of poison on, 476.
—rupture of, 289.
—wounds of, 289.
Stomach tube, use of, in poisoning, 487.
Stramonium, 733 ; seeds, 735.
Strangulation, 160.
—appearances on the neck, 162.
—causes of death in, 161.
—definition of, 160.
—feigned, 170.
—illustrative cases, 171.
—in infanticide, 382.
—ligature mark in, 162, 166.
—medico-legal questions in, 166.
—post-mortem appearances of, 161, 162.
—symptoms of, 161.
—treatment of, 162.
Stricture of urethra in life assurance, 438.
Strychnine, 764.
—poisoning by, 765.
—tests for, 769.
Strychnos colubrina, 764.
—ignatii, 764.
—nux vomica, 764.
—tieute, 764.
Stupor in katatonia, 497.
Subpœna, 11.
Subsulphate of mercury, 578.
Sudden death, 122.
—causes of, 122, 123.
Suffocation, 154, 173.
—causes of, 173.
—definition of, 173.
—fatal period of, 175.
—illustrative cases, 177.
—in infanticide, 381, 382.
—medico-legal questions in, 176.
—mode of death in, 174.
—post-mortem appearances from, 175.
Sugar of lead, 590.
Suggilation, 123, 128.
Suicidal wounds, 252, 257.
Suicide, in burns, 207.
—in drowning, 187.
—in hanging, 159.
—in starvation, 193.
—in strangulation, 167.
—in suffocation, 176.
Suis, poisoning by, 636.
Sukhadarshan, 655.
Sulphanilamide, poisoning by, 718.
Sulpharsenobenzene, 545.
Sulpharsphenamina, 545.
Sulphate of indigo, poisoning by, 494.
Sulph-methæmoglobin, 135, 813.
Sulphocyanide of mercury, 576.
Sulphonal, poisoning by, 711.
Sulphonmethane, 711.
Sulphur dioxide, poisoning by, 814.
Sulphuretted hydrogen, poisoning by, 813.
Sulphuric acid, poisoning by, 493.
—post-mortem appearances in, 495.
—properties of, 493.
—tests for, 497.
Sulphuric ether, 698.
Sulphurous anhydride, poisoning by, 814.
Sunstroke, 195, 196.
Superbine, 654.
Superfecundation, 328, 331.
Superfœtation, 328, 331.
Supposititious children, 328.
Suran, 655.
Surma, 568.
Survivorship, 152.
—conditions in determining, 152, 153, 154.
Suspended animation, 124.
—cases of, 124, 125.
Sympathetic disturbances, as a sign of pregnancy, 316, 317.
Syncope, 119.
—cause of, 119.
—post-mortem appearances of, 119, 120.
—symptoms of, 119.
Synthetic dye stains, 107.
Syphilis, in rape, 337.
—in life assurance, 438.
—in unnatural offences, 350.
Syphon tube, 483.

T

Table salt, poisoning by, 623.
Tailors, 60.
Takayama, reagent, 97.
Talispatra, 650.
Tamba, 586.
Tambaku, 776.
Tankankhar, 540.
Tannin, reagent, 484.
Tar camphor, 721.
Tardieu's spots, 70, 121, 216.
—in suffocation, 175.
Tartar emetic, 567.
Tartaric acid, poisoning by, 521.
Tattoo marks, 25, 57.
—artificial removal of, 58.
—designs of, 57.
—disappearance of, 57.
Taxus bacata, poisoning by, 650.
Taxine, 650.
Tear gases, 815.
Technique for determining blood groups, 103.
Teeth, artificial, 47.
—deciduous or milk, 32.
—general characteristics of, 32.

Teeth, Hutchinson's, 33.
—in identification, 47.
—injuries of, 276.
—natural, 47.
—permanent, 32, 33.
—temporary, 32, 33.
Teichmann's, crystals, 96.
—test, 96.
Tenancy by courtesy of England, 326.
Terminalia Bellerica, 657.
Telyabish, 792.
Teora, 763.
Testamentary capacity, 422.
Testicles, contusion of, 299.
—squeezing of, 299.
Tests for alkaloids, 483.
Tetrachlor-methane, poisoning by, 705.
Tetra-ethyl lead, 592.
Tetronal, 712.
Thallium, 600.
—chemical analysis of, 602.
—medico-legal points in, 603.
—poisoning by, 601.
—salts, 600.
Thebaine, 675.
Thevetia neriifolia, poisoning by, 787.
Thevetin, 787.
Thohar, 652.
Thorax, examination of, 70.
Thorn apple, 733.
Throat, wounds of, 278.
Throttling, 160, 163, 383.
Thumb-mark impression, 50.
Tichborne case, 63.
Tidhara, 652.
Tinned fruits, 614.
Tin, poisoning by, 613.
Tobacco, 776.
—camphor, 776.
—poisoning by, 776.
Toxicology, 466.
Trachea, decomposition of, 140, 141.
—examination of, 70.
—wounds of, 278.
Traumatic asphyxia, 281.
Tribadism, 352.
Trichloromethane, 700.
Tricks of manner and habit, 25, 62.
Trigonella fœnum-græcum, 359.
Tri-iodomethane, poisoning by, 706.
Trinitrin, poisoning by, 727.
Trinitroglycerine, 727.
Trinitrophen, poisoning by, 516.
Trinitro-phenol, poisoning by, 516.
Trinitrotoluene, poisoning by, 726.
Trional, 712.
Triphala, 657.
Trotyl, 726.
True vesicles, 207.
Turpentine, 730.
—old, 92.
Turpeth, black, 652.
Turpeth, mineral, 578.
—white, 652.

U

Ulceration, in poisoning, 478.
Umbilical, arteries, 377.
—cord, 377.
—changes in, 377.
Umbilicus, hæmorrhage from, 379.
—knots of, 380.
—mummification of, 377, 378.
—pressure on, 380.
—prolapse of, 380.
—vein, 378.
—vesicle, 364.
Unconscious, delivery, 381.
—pregnancy, 386, 387.
Unnatural offences, 347.
Unsoundness of mind, 389.
Upas tree, 764.
Urari, poisoning by, 819.
Urea stibamine, 568.
Urethra, injury of, 298.
rupture of, 298.
stricture of, in life assurance, 438.
Urginea indica, 654.
—scilla, 653.
Urotropine test, 683.
Uterine changes, 319, 321.
—contractions, 320.
—souffle, 320.
Uterus, 73, 323.
—decomposition of, 142.
—examination of, 73.
—rupture of, 297.
—size of, 73.
—weight of, 73.

V

Vagina, 311.
—changes in, 319, 323, 324.
—laceration of, 299.
Vagitus, uterinus, 369.
—vaginalis, 369.
Van Deen's test, 92.
Varicose veins, in life assurance, 438.
Variot's method, of removing tattoo-marks, 60.
Vault, fractures of, 268.
Vegetable, hairs, 672; poisoning by, 672.
—chemical analysis of, 480.
—poisons, 470, 627.
—stains, 107.
Veins, Tamassia's identification of, 53.
Veratrine, poisoning by, 646.
Veratrum, 645.
—album, 645.
—officinale, 645.
—viride, 645.
Verbigeration, 407.
Verdigris, 586.
Vermine killers, 765.

Veronal, poisoning by, 712.
Vertebral column, examination of, 73.
Vesicants, 815.
Vesication, in burns, 207.
Vesicular mole, 321.
Viability, 330.
Vibert's fluid, 95.
Vibices, 128.
Vinegar, 519.
Violence, 272.
—marks of, 67 ; in rape, 336.
Violet powders, 543.
Viperine snakes, 660.
—bites by, 662.
Virginity, 311.
—signs of, 311.
Vitali's test, 741.
Vitriolage, 498.
Vitriol, blue, 585.
—green, 612.
—oil of, 493.
—throwing, 498.
—white, 604.
Voice, 25, 61.
Volatile poisons, chemical analysis of, 480.
Volitional act, in wounding, 249.
Volunteering a statement, 20.
Vulnerant poisons, 669.
Vulva, wounds of, 299.

W

Wagner's reagent, 483.
Wake-Robin, 654.
Walford's formula, 439.
War gases, poisoning by, 815.
Washerwoman's hand, 184.
Washing soda, 523.
Wasps, stings by, 665.
Water gas, 807.
Water test, 608.
Weapons, 240.
—dangerous, 242.
—kind of, 240.
Weed-killer, 546.
Weighman, 60.
Weight in determining age, 34.
Weppen's reaction, 646.
Wet method for analysing organic mixtures, 484.
—for mineral poisons, 484.
Whipping, 10.
White, arsenic, 542.
—copperas, 604.
—lead, 591.
—turpeth, 652.
—vitriol, 604.
Will, 422, 464.
Willich's formula, 439.
Winslow's test, 126.
Witness, 19.
—box, 12.
Witness, common, 19.
—examination of, 13.
—expert, 19.
—hostile, 13.
—medical, 12, 17, 18.
Wolfsbane, 790.
Woody nightshade, 751.
Wood, naphtha, 694.
—spirit, 694.
Workmen's Compensation Act, 460.
Worm wood, 757.
—oil of, 758.
Wounds, 217, 223.
—ante-mortem and post-mortem, 251.
—causes of death from, 243.
—classification of, 223.
—gunshot, 223, 228.
—illustrative cases, 249, 257, 258.
—incised, 223.
—in infanticide, 384.
—in putrefaction, 137.
—lacerated, 223, 227.
—on a dead body, 68.
—penetrating, 225.
—punctured, 223, 225.
—suicidal, accidental and homicidal, 252.
Wourali (Wourara), poisoning by, 815.
Wredin's test, 377.

X

Xanthopsia, 757.
Xanthoproteic acid, 499.
X-ray, burns caused by, 198.
X-ray examination of bones in age, 35.
—in pregnancy, 320.

Y

Yellow, arsenic, 544.
—jasmine, 774.
—oleander, 787.
—phosphorus, 528, 529.
Yew, 650.

Z

Zangal, 587.
Zafran, 359.
Zelio-grains (corn), 600.
—paste, 600.
Zinc, 603.
—acute poisoning by, 604.
—chloride, 604.
—oxide, 605.
—paste, 604.
—stearate, 604.
—sulphate, 604.
—tests for, 606.
—white, 604.