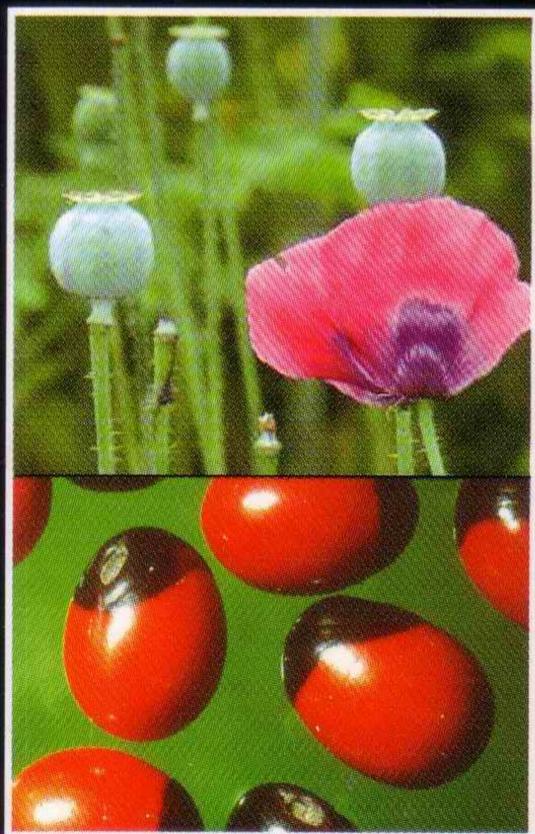
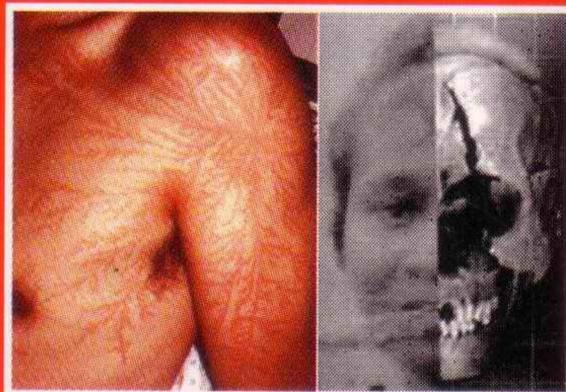


P C Dikshit

**Textbook of
Forensic
Medicine
and
Toxicology**



CURRICULUM BASED

DETAILED COVERAGE

MODERN CONCEPTS

POSSIBLE QUESTIONS

PEEPEE

**Textbook
of
Forensic Medicine
and
Toxicology**

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Introduction and History of Forensic Medicine

The origin of medicine is as old as civilization. In the early stages the mankind used spirituality, herbs, God worship and witchcraft etc. for the cure. Then they felt the need of formulating certain rules and regulations for these practices that led to the origin of medical jurisprudence with advancement of civilization various legal systems came into force and the knowledge of medicine became necessary for the purpose of law and justice and the concept of legal medicine or Forensic Medicine originated. The term Forensic Medicine, Legal medicine and Medical Jurisprudence are all used in different meanings.

Forensic Medicine is an application of medical knowledge for the purpose of law both civil and criminal for example in cases of vehicular accidents. The Forensic expert is able to opinion whether the said injuries are due to vehicular accident or not, duration of injuries, nature of injuries and the cause of death etc. Likewise in case of sexual offences, he is able to give opinion whether the sexual act has been committed or not, the duration and type of injuries etc. The Forensic expert not only expertises in his own field but uses the knowledge of other branches of medical science to solve the problems related to forensic medicine for example while doing postmortem in cases of accidental deaths, criminal abortion and sudden deaths due to medical and surgical causes.

Medical Jurisprudence deals with legal aspects of practice of medicine. The concept of doctor patient relationship, rights and duties of doctors, duties towards the patient and the state in particular the medical negligence etc. are all covered by the term Medical Jurisprudence.

Medical ethics deals with the moral principles that guide the members of medical profession when

they deal with each other, their patients as well as with the State.

Medical etiquette deals with the conventional laws of courtesy observed between the members of the medical profession.

State Medicine is a term, which was suggested by Dr. Stanford Emerson Chaille in the year 1949, but now a days it stands rejected worldwide. In India few States like West Bengal, Manipur and Assam etc. use the word and the subject is known as Forensic and State Medicine.

Forensic medicine can be divided into a number of sub branches. Forensic pathology deals with morbid anatomy, pathology of injury and different aspects of death with their medicolegal significance. Forensic psychiatry is a very interesting branch where legal aspects of mental disorders are studied. Forensic odontology where the ability of dentistry is undertaken when the question of identification and interpretation of bite marks arise. A number of factors are found in the teeth that are utilized for identification purposes. Forensic anthropology, studied of bodily shape and skeletal formation in legal sense and also for the identification purposes.

The branch of forensic science that is a sister discipline of forensic medicine. Now-a-days this is mainly occupied by the non medical scientists mainly a biologist or chemist who deals with the criminal aspects of crime investigations. There is a strong link between the forensic medical experts and forensic scientists as the former collects the evidences during medicolegal autopsy or while examining living persons and the latter carries out the test and gives the opinion. Therefore both these branches are complementary to each other.

HISTORY OF FORENSIC MEDICINE IN INDIA AND ABROAD

The word forensic medicine came from the Latin word 'forensis' meaning marketplace, as during ancient days the justice used to be dispensed from marketplaces or common meeting places. The total period of forensic medicine can be divided into three phases.

1. Ancient Phase

The law and medicine problems were found as written records in China, India, Egypt and Babylon etc. dating back to 5000 BC to 3000 BC. The **Hammurabi code** by King of Babylon is the oldest medicolegal code. Chief justice and Chief physician of the King Zoser of Egypt was the first medicolegal expert. Different techniques were developed for the preservation of the dead bodies (mummification). Indian civilization such as excavation remains of the Indus valley civilisation (3200-2000 BC) gives the evidence of the use of medicines like *shilajit* and use of weapons made of bronze and copper.

Manu (3102 BC) was the first lawgiver in India. His "**Manusmriti**" gives the knowledge about the laws prevalent at that time. Various types of crimes like sexual offences, seduction and adultery have been mentioned. Those who were having mental illness were not allowed to contract. King's permission was required to practice medicine. Definite rules were laid down to practice and to learn medicine. It can be presumed that the origin of medical jurisprudence can be dated back to 3000 BC. At the same time a Chinese materia medica gives information about poisons.

The Vedas of ancient India were composed between 3000 to 1000 BC. The Rig Veda, the oldest one mentions about kings and their administration. Marriage was prohibited in blood relations. The Atharva Veda mentions cures for wounds, snake bite and poisons etc. in the form of mantras or charms while other Vedas mention crimes like incest, adultery, abortion and drunkenness etc. along with the punishment for them. During this time dead bodies were dissected that can be compared to anatomical dissection as well as pathological autopsy today.

At that time Ayurveda was quite advanced. The first treatise was Agnivesh Charaka samhita

supposed to have been composed during 700 BC. Here we find regarding the rules of studying ayurveda, duties and rights of the students, privileges they can avail that can be considered the origin of medical ethics. In addition, there is a mention of poisons, their symptoms with their treatment.

Between third and fourth century BC, the **Arthashastra** of Kautilya was written. In this book the penal laws are defined and the rules regarding the application of medical knowledge to legal matters has been mentioned which can be considered as the origin of legal medicine in India. The physicians were allowed to practice only after king's approval, similar to registration with medical council today. They were liable to be punished if there was any negligence during treatment. The other crimes like sexual offences, violation in marriage laws etc. were also punishable. There were provisions for the examination of dead bodies like post mortem examination of today. The body used to be kept in oil for examination in cases of sudden deaths. If any dead body that was swollen with eyes protruding out, marks on the neck, was taken granted that he has been killed by strangulation. Signs of death due to hanging, strangulation, drowning etc have been mentioned. In cases of poisoning the portions of stomach and heart were put in the fire and the nature of flame and sound were noted to determine the nature of poison. Depending on the seriousness of the crimes different types of punishments like whipping and cutting of body parts were awarded.

Hippocrates the father of western medicine (460 BC to 377 BC) was first to write the code of medical ethics. He practiced in the island of Kos in Greece and discussed the lethality of wounds at large. During 44 BC Antistius had conducted the autopsy on the body of Julius Caesar who was assassinated.

2. Medieval Phase

Justinian court during the 1st to 5th century AD came into existence after the name of Justinian the Roman Emperor who specified the role of medicolegal experts in dealing with medicolegal problems.

During the period of 12th to 15th century AD a Chinese treatise was published named **His-Huan-Lu**. The inquest was made obligatory in all types of unnatural deaths. Different types of injuries were defined and the manner of investigations to be done in these cases was also suggested.

During the period of 1000 AD to 1600 AD India was invaded by several foreign powers like Dutch, the French and East India Company so as to plunder its wealth and establish their colonies. Chaos and uncertainty prevailed in all spheres of life including law and order situation. Lastly the East India Company conquered and ruled over India in the middle of 18th century until it handed over power to the British crown in 1857.

The first medicolegal autopsy was done by Bartoloneo D E Varignana in Bologna (Italy) in the year 1302 AD.

The **Constitutio Criminalis Carolina** was published in Germany in 1532 AD which wrote about different types of homicides that were not punishable for example if a criminal who is deprived of his judgment power was not responsible for the act like a murder committed by an insane.

3. European Phase (1600-1947 AD)

Paulus Zacchias was the principal physician to Pope Innocent X and Alexander VII an expert before the court "rota romana", the court of appeal. His works "medicolegalis" were published in seven volumes during 1621 to 1635 AD. This work remained an authority in medicolegal matters until the start of 19th century. He was considered as the father of forensic psychiatry as well as of legal medicine. An Italian physician **Fortunate Fedele** published the first book in forensic medicine in 1602. In 18th century the post of professorship was created in Germany. **Orphila**, professor of chemistry and legal medicine at Paris is considered as the father of modern toxicology.

The British ruled over India till 1947 and introduced their system in every sphere like culture, education, administration, legal system and crime investigation etc. They introduced the allopathic system of medicine. The present legal system in India is a mixture of the English and Roman system, having been modified to suit the Indian conditions.

The first medical school was established in Calcutta in 1822 that was later on converted into a medical college in the year 1835. Next medical school established in Madras in 1835, which was converted into a college in 1850. In Bombay first medical college was established in the year 1950.

In 1857 AD, the first separate chair in medical jurisprudence was created in Madras medical college. Governor Lord Harris selected Dr Urganhart,

a private practitioner and coroner of Madras as the first professor. Among the earlier professors were Dr Mouat, Dr John Moses of Calcutta medical college, Dr A Porter and Lt. D.G. Roy of Madras medical college and Major Collies Barry of Grant medical college, Bombay. The books that were followed were brought from England. The popular English authors books were Taylor's principles and practice of medical jurisprudence and Caspers medical jurisprudence.

The first book for India was written in 1856 by Dr Norman Chevers the other books written in 1888 by Dr I B Lyon named Medical Jurisprudence for India, outlines of Medical Jurisprudence by Lt. Colonel P. Henir and J D B Gribel in 1889 and Legal Medicine in India and Toxicology by Major Collies Berry in 1902.

Dr Jaising P Modi, lecturer at Agra Medical College and then at Lucknow Medical College since 1918 was a leading medicolegal expert. His first book the Text Book Of Medical Jurisprudence And Toxicology was published in 1920. The book became popular not only with medical students but also among the lawyers and judges.

The British rulers subsequently codified the criminal law in India in 1833. The present Indian penal code (IPC) came into existence in 1860 and superseded all previous laws. The criminal procedure code (CrPC) came into force from 1861. These laws are being followed till today. The procedure for crime investigation was systematized by the introduction of Indian Police Act of 1861 and the coroner system by the Indian Coroners Act 1871. The coroner system was followed in Calcutta, Bombay and Madras whereas the rest of the country followed the police system.

The practice of modern medicine was first regulated by the formation of provincial medical councils in 1912 according to which the medical practitioners were required to register themselves in the respective state medical councils.

In 1916 the Indian medical degrees act was passed to recognize the qualifications from recognized institutions and to penalise persons having false certificates to practice medicine.

The Indian Medical Council Act was passed in the year 1933 and the Indian Medical Council was established to control the state medical councils and to regulate infirmity in medical education throughout the country.

One of the greatest contributions India has made to modern criminology is dactylography or fingerprint

system for personal identification. Towards the later part of 19th century that is in 1860 AD William Herschel, an Indian civil service officer posted at Nadia district of Bengal first used this method for identifying illiterate people while distributing money. After him Edward Richard Henry in 1891 AD developed the method further and published a book entitled "*classification and use of fingerprints*". By his recommendation the government of India also adopted this method of identification and established

a fingerprint bureau in Bengal in 1897 AD. Subsequently this method was introduced in criminal investigation department of Scotland Yard in July 1901 by Henry himself who was posted there as assistant commissioner.

Then the medicolegal work was undertaken and postmortem centers were established at different places. During the time of East India company a post mortem was conducted in Madras in August 1693 by Dr Buckley in an alleged case of arsenic poisoning. This was probably the first medicolegal autopsy in modern India.

MULTIPLE CHOICE QUESTIONS

- The first book in Forensic Medicine was written by a physician of:**
 - U.S.A.
 - U.K.
 - Italy
 - Denmark
- The first medicolegal autopsy was conducted in:**
 - Italy
 - Greece
 - England
 - Portugal
- Who is known as the father of modern toxicology?**
 - Paulus Zacchias
 - Bartolomeo De Varignana
 - Orfila
 - Fortune Fedele
- In India the first book of forensic medicine was written in the year:**
 - 1850
 - 1856
 - 1860
 - 1875
- The first separate chair in medical jurisprudence was created at:**
 - Grants Medical College
 - Calcutta Medical College
 - K.G. Medical College, Lucknow
 - Madras Medical College

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Legal Procedures

The legal procedures should be followed in the cases where death has resulted from some unnatural cause and under suspicious circumstance. The investigating agency should be informed who conducts further investigations. Legal procedures in India are based on the Indian Constitution, Code of Criminal Procedure, Indian Penal Code and the Indian Evidence Act etc.

The Criminal Procedure Code, 1973 (CrPC) deals with the procedure of investigation and trial of offences within the limitations of the Union Territories of India except Jammu and Kashmir and some other tribal areas. It provides for different classes of Courts and defines their powers. It also formulates duties of police in arresting offenders, dealing with absconders, in the production of documents and investigating offences. It defines offences into two categories; cognizable and non cognizable. Some cases are triable by both magistrates as well as Courts of Sessions as indicated in Section 2 of CrPC. However, when tried by a magistrate the punishment must be limited to magistrate's powers. The Code also provides for the period of detention undergone by the accused during investigation, inquiry or trial is to be deducted from the sentenced of imprisonment awarded by the Court and the balance period only is to be undergone. A bar of limitation for taking cognizance ranging from 6 months-3 years has been prescribed in certain cases under Sec.468.

The Indian Penal Code, 1860 (I.P.C) describes various offences and with their punishments in the Courts of Law. It defines offences and several categories of the same offence are created such as House Trespass (Section 448-460) and theft

(Section 379-382 and 401). These offences are categorised under the I.P.C. to keep the workload low for higher Courts as well as in expensive thus reducing the number of such Courts. Besides this the I.P.C. also provides a chapter on general exceptions in which the right of self defence of person or property and extending to causing deaths in certain circumstances is to be found. The IPC describes various punishments such as (i) Death (20 years in prison) (ii) Imprisonment for life including solitary confinement (iii) Simple imprisonment (iv) Forfeiture of property and fine.

The Indian Evidence Act, 1872 (I.E.A.) relates to evidence on which the Courts could come to some conclusion about the facts of the case.

Criminal law relates to the offences that are against the interest of the public namely offences against the person, property, safety of the public as well as security of the state.

Civil law deals with disputes between two individuals or parties called the plaintiff and defendant (accused).

INQUEST

An inquest is investigation in to the cause of death in cases of sudden, unnatural & suspicious deaths conducted by the legal authorities such as the Police Officer, Magistrate or the Coroner etc.

TYPES OF INQUEST

Police Inquest (Sec. 174 Cr.P.C.)

Police to Enquire and Report on Suicide etc.

An officer-in-charge of the police station in whose jurisdiction the death has occurred conducts the

1. Police inquest
2. Magistrate's inquest
3. Coroner's inquest
4. Medical Examination

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police inquest. On receipt of information of an unnatural death, the police officer informs the nearest magistrate and thereby proceeds to the place of death. The police officer then conducts an inquest in the presence of two or more respectable inhabitants of the neighbourhood (*panchas*). After the necessary investigation, a report is drawn up on the apparent cause of death as judged by the state of the body, the injuries present on the body, hearsay evidence and circumstantial evidence etc., this document is called panchnama or inquest report. This report is signed by the police officers and the persons present at the inquest. If any foul play or unnatural death is suspected, the police officer forwards the body for postmortem examination to the nearest government hospital along with the written request for conducting the postmortem examination and a copy of panchnama (inquest papers). If the autopsy report confirms that the death is due to an unnatural cause, further inquiry and trial of the case is conducted in the usual manner by the magistrate concerned to whom the entire records of the case are transferred by the police officer. Police Inquest is done in cases where a person (i) has committed suicide (ii) has been killed by another man (iii) killed by animal (iv) killed by machinery (v) killed in an accident (vi) died in suspicious circumstances raising reasonable suspicion that some other person has committed an offence.

1. In the above situation when the Police Officer gets information, he will immediately inform the nearest Executive Magistrate empowered to hold inquest and then proceeds to the place where the body of such person is found. He makes investigation in the presence of two or more respectable person of the locality, draws up a report regarding the apparent cause of death, describing such wounds, bruises, fractures etc. found on the body and stating the manner and type of weapon that has caused such injuries.
2. The report shall be signed by such Police Officer and other persons or by so many of them who agree with the same opinion and shall be forwarded to District Magistrate/SDM.
3. When (i) The case involves suicide by a woman within seven years of marriage, or (ii) The case

relates the death of woman within seven years of her marriage raising a reasonable suspicion that some other person has committed the offence in relation to such woman or (iii) The case relates the death of a woman within seven years of marriage and any relative of the woman has made the request in this behalf or (iv) There is any doubt regarding the cause of death (iv) The police officer for any other reason considers it suitable to do so sends the body to the nearest civil surgeon or Medical Officer for examination.

4. The following Magistrates will be empowered to hold inquests, namely any District Magistrate or Sub Divisional Magistrate, any other Executive Magistrate empowered by State Govt. or the District Magistrate.

Magistrates Inquest (S.176 CrPC)

Inquiry by Magistrate into Cause of Death

1. The inquest is done by any District Magistrate, sub Divisional magistrate or any other executive Magistrate or any Magistrate empowered by the State Government. Following are the indications of Magistrate's Inquest:
 - i. Custodial deaths
 - ii. Deaths due to Police firing
 - iii. Death inside prison
 - iv. Dowry deaths
 - v. Clause (i) or (ii) of sub Section (3) of Section 174 CrPC. The case involves suicide by a woman within seven years of marriage or the case relates the death of woman within seven years of her marriage raising a reasonable suspicion that some other person has committed the offence in relation to such woman
2. In addition the Magistrate may hold an inquest in the cases done by Police. The proceedings of the Magistrate are not judicial proceedings, they only try to find out the cause of death. After inquest the body is sent for the post-mortem examination. The inquest papers must be handed over to the Autopsy surgeon for information. During the investigation, the Magistrate shall inform the relatives of the deceased and also allow them to stay during investigation.

Coroner’s Inquest

Coroner’s inquest no longer exist in India. However it used to be followed previously in Bombay. It is interesting that in Calcutta still for conducting the inquest, the dual system exists. The presidency town of Calcutta is under the Coroner’s system whereas in rest of Calcutta it is the police inquest. However, the Coroner system is prevalent in countries like U.K and some states of U.S.A. The Coroner’s Court is a Court of inquiry in which the Coroner conducts the inquest in unnatural, sudden and suspicious deaths. In his Court, the jurors are sworn to give a true verdict according to the evidence. Besides this, the Coroner has some judicial powers also.

Medical Examiner’s System

The Medical Examiner’s system for conducting inquest exists in most of the states in the United States. Medical examiner is a medical man who is appointed to perform the duties of a coroner. The medical examiner neither has judicial functions nor does he have authority ordering arrest of any person. In this system, the medical examiner visits the scene of crime and conducts further inquest. This system is superior to that of police or a coroner.

CLASSES OF CRIMINAL COURTS (Sec. 6 CrPC)

Besides the High Courts and the Courts constituted under any law, there shall be in every state: (i) Courts of Sessions (ii) Judicial Magistrates of the first class and, in any Metropolitan area, Metropolitan magistrate (iii) Judicial Magistrate of second class, and (iv) Executive Magistrate.

SENTENCE PASSED BY HIGH COURTS AND SESSIONS JUDGES (Sec. 28 CrPC)

1. A High Court can pass any sentence authorized by law.

2. A Sessions Judge may also pass any sentence authorized by law but any sentence of death passed shall be subject to confirmation by the High Court.
3. An Assistant Sessions Judge may pass any sentence authorized by law except a sentence of death or of imprisonment for life or of imprisonment for a term exceeding ten years.

POWERS OF MAGISTRATES (Sec. 29CrPC)

The Court of Chief Metropolitan Magistrates shall have the powers of the Court of a Chief Judicial Magistrate and that of a Metropolitan magistrate. The powers of the Court of Magistrate are given in Table 2.1.

COGNIZABLE OFFENSE (Sec. 2 CrPC)

Cognizable offence means an offence for which, and cognizable case means a case in which, a police officer may in accordance with the First schedule or any other law for the time being in force, arrest without warrant. These offences include rape, dowry deaths, ragging, deaths due to rash or negligent act etc.

JURISDICTION IN CASE OF JUVENILES (Sec. 27 CrPC)

Any offence not punishable with death or imprisonment for life, committed by any person who at the date when he appears or is brought before the Court is under the age of eighteen years, may be tried by the Court of Chief judicial Magistrate, or by any Court specially empowered under the Children Act, 1960, or any other law for the time being in force providing for the treatment, training and rehabilitation of youthful offenders.

SUMMONS (SUBPOENA)

It is a written document compelling the attendance of the witness in the Court, to depose evidence, at a particular time and purpose under penalty.

Table 2.1: Powers of magistrate			
Magistrates	Imprisonment	Solitary confinement	Fine (Rs)
Chief judicial magistrate	Up to 7years	Yes	Unlimited
First class judicial magistrate	Up to 3years	Yes	5,000
Second class judicial magistrate	Up to 1 year	yes	1000

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This is also called subpoena. Literally *sub* means under and *poena* means penalty. The provisions regarding summons are dealt in the Sections 61 to 69 CrPC.

The summon is issued in duplicate signed by presiding officer of the Court and bears the seal of the Court (Section 61 CrPC). The summons is delivered through a police officer, an officer from the Court or by any other person. The witness signs the carbon copy and returns it to the Court. According to 66 CrPC, if the witness is a government servant the summons can be sent to the head of the office in duplicate and the head in turn serves it to the concerned person. When summons are issued to produce a document or any other article required for investigation or trial (**Section 91 CrPC**) the person in possession of the said document or article may send it instead of attending personally. If the witness fails to attend the Court without any valid reason he will be penalized in the form of paying damages in civil cases and Criminal cases notice will be served under **Section 350 CrPC**. After hearing from the witness if the Court thinks that there was no proper reason for not attending the Court, a bailable/non-bailable warrant may be issued to procure his attendance in the Court (**Section 172-174 IPC** and **Section 87 CrPC**).

If a witness receives two summons on the same date from the same type of Court he will attend the Court from which he received the summon first and will inform the other Court. If he receives two summons, one from Criminal Court and the other from Civil Court, he will attend the criminal Court first with intimation to the Civil Court because Criminal Courts have priority over Civil Courts and also the higher Courts have priority over lower Courts. The witness would not leave the Court without the permission of the presiding officer.

In civil cases certain amount of money is paid to the witness towards his expenses for attending the Court, which is called **conduct money**. This amount is paid to the witness while serving the summons. At times the money is paid in the Court. If the witness thinks that the amount is less according to his status he can pray to the presiding officer in the Court for additional amount. Even he can ignore the summon if no amount is paid.

But in criminal cases no conduct money is paid the witness must attend the Court in the interest of the state or in the interest of justice. The government usually pays TA and DA to attend such Courts. If the witness does not attend the Court he will be charged for contempt of Court.

After getting the summons the witness appears before the Court on the specified date and time with the records called for. Before deposing the evidence the witness has to take the oath.

OATH

The witness stands in the witness box and takes the oath. The main aim is that the witness will tell the truth during the evidence. The provision is incorporated in **Section 51 IPC**. He takes oath as follows. "I swear in the name of God, that what I shall tell, be the truth, the whole truth, nothing but the truth." (Vide **Indian Oath Act 44 1969 Section 4 and 6**). The oath is taken in the case of all Courts other than high Courts and supreme Courts, which is administered by the presiding officer. In cases where the witness appear before a bench of judges / oblique magistrates, one of the judges or the magistrate can administer the oath. (**Indian Oath Act 44 of 1969 Section 2**).

Perjury means willfully giving false evidence under oath or unable to tell what he knows or believes to be the truth (**Section 191 IPC** and **Section 344 CrPC**) he is liable to be prosecuted for perjury under **Section 193 IPC** with imprisonment up to seven years and fine.

EXAMINATION IN CHIEF (Sec. 137 IEA)

After the oath or affirmation the witness will be examined by the lawyer of the party that has called him it is also known as direct examination. In criminal cases the state becomes a party so the burden of proof is always on the prosecution. It is always presumed that the accused is innocent unless otherwise proved. Before giving the evidence the doctor should always see the previously prepared report. If needed he can meet the public prosecutor and go through the files and other records. The doctor should help the public prosecutor to frame the questions so that proper facts are elicited. The medical witness is usually

asked questions as to when he saw the body and started the postmortem examination. The question pertaining to description of the injuries, duration of injuries and possibly about the weapon of offence are also asked during examination in chief.

Normally leading questions are not allowed where the Court is satisfied that the witness is a hostile one (**Section 142 of the IEA**). A leading question is the one, which suggests the answer. (**Section 141 IEA**) for example “did you carry out the postmortem examination”, “did you find a 5 cm laceration on the forehead”. To an ordinary witness, question such as “did you see the accused inside the restaurant” or “have you seen him beating A” are asked. In civil cases also the same procedure is followed.

CROSS EXAMINATION

During the cross examination the lawyer of the opposite party i.e. the lawyer of the accused (defence lawyer) examines the witness. The main objectives are:

- To elicit facts favourable to his client.
- To find out the weak points in the case.
- To verify the accuracy of the facts already told. (**Section 146 IEA**).

The defence lawyer always tries to discredit the witness and tries to prove that the report given is not correct and biased one. It is not necessary that the question should be confined to the facts told in examination in chief. (**Section 143 IEA**). The defence lawyer may even ask questions pertaining to the qualifications, experience etc., especially in the type of the case in question. During this time the Court observes whether a particular question should be answered or not. The Court has power to disallow a question if it is intended to insult or harass the witness but if it is relevant to the case the Court cannot disallow. The witness has to answer the question that relates to the case even if it is harmful to the witness or it exposes his fault or negligence (**Section 146 IEA**). If a witness is forced to answer that goes against him he cannot be arrested or prosecuted or cannot be taken as proof against him for any criminal proceedings. When a question is not audible, the witness can request to repeat the

same and when the question is not understood properly he can again ask to explain. The witness should be clear, direct and precise in his answer.

The doctor is an expert witness to the Court, not to either party even though one party has called him. The Court and the society want him to be impartial and assist the Court to reach a proper conclusion for the sake of justice. This has been well put by the famous French medicolegal authority in 19th century Brovardel. He said, “Where the law has made a physician a witness, he should remain a man of science. He should remember that he has no victim to avenge, no guilty person to convict and no innocent person to save.”

When a witness is harassed and humiliated he becomes angry, hostile and tense. His intellectual faculties are blunted so as to lose the power of giving proper evidence. He may ask the Court for some time to be in proper shape for further evidence. He may volunteer certain facts that are relevant to the case even if not asked by the lawyer. Here the leading questions are allowed and there is no time limit for cross-examination.

RE-EXAMINATION

This is done by the lawyer who has called the witness (**Section 137 IEA**) the main aim is to clarify the points raised in cross examination and correct the mistakes done. However the lawyer should not bring any new points during the examination that has not been raised in the examination in chief. If any new point arises the witness should be cross-examined. Raising a new point should be done after taking permission from the Court.

Questions put by the Court

The judge may ask any question to clarify doubts in the evidences. The Court is also empowered to recall and reexamine any witness already examined if it is essential to arrive at a decision (**Section 165 IEA and Section 311 CrPC**).

The deposition is handed over to the witness after reading carefully and thoroughly he signs at the end of each page with date. Any mistakes in the deposition should be brought to the notice of the Court and then he can correct it with his initials. He should not leave the Court without prior per-

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mission. The TA and DA are usually paid in the Court.

CODE OF CONDUCT OF A DOCTOR INSIDE THE WITNESS BOX

The doctor in the witness box should appear as a man of professional competence and integrity. He should be well dressed and look sober, prepare the facts of the case and take all relevant records / articles in his custody to the Court. The doctor should never be nervous inside the Court and speak loudly and clearly so that the presiding officer can record the deposition.

It is essential that he should use no medical jargon and if medical terms are to be used a short explanation can be added. An expert may refresh his memory by reference to the professional treatise. The writing that is used to refresh the memory can be cross-examined by the opposite party (161 IEA).

The doctor should never be over confident or arrogant. He should always use "Sir" or "Your honour" while addressing the Court. He should never use adjectives or exaggerations like very large bruise severe pain etc. And he should never lose the temper even if provoked by the opposite lawyer.

The doctor should not avoid any question. If the answer is not known, always say, "I don't know". Nobody expects the doctor to know everything. Do not alter your findings to what is said in the statement. The answers should always be brief and to the point.

If the lawyer limits the answer to "yes" or "no" which is not possible at times, the doctor can tell the judge that it requires explanation because you have taken the oath not only to speak truth but the whole truth. Always answer "in my opinion but not as I think or imagine or I am not sure etc". When the answer demands quantity or number, the doctor should not tell the number unless he knows the exact number or quantity such as in cases of age estimation, the answer should be given within certain limits. The doctor should always answer within his own speciality. He should never comment on the points where he has little or no experience at all.

When the lawyer reads a paragraph from the book and asks to comment the doctor should always be careful. He must read the paragraph carefully even the paras before and after it to come to the conclusion. He must see that the book should be recent edition and the views expressed must be latest. He may or may not agree. It's not necessary to agree upon everything written in the book. It should be remembered that views expressed in the book by reputed authors are taken as evidence even if the witness doesn't agree to it.

When attempts are made to influence the witness by one party or the other he must report to the senior police officer or to the Court so that the corrupt person can be penalized and at the same time he and his family members are given protection.

A medical witness cannot claim professional privilege. He has to answer each and every question asked after obtaining the permission of the Court. A witness whether medical or otherwise is completely immune from actions for defamation (libel or slander) for anything he says in the witness box. This is quite important otherwise the witness will not disclose the actual facts because of fear of legal action later on.

Volunteering of statements—At occasions the public prosecutor leaves certain ambiguities in the evidence and the advocates for the defence also put ambiguous questions, as a result the witness gives incomplete answers. The defence lawyer takes the advantage of these during an argument. When certain ambiguity is left or a general statement given by him is not applicable to the facts of the case in hand, the medical witness should come forward and volunteer for a statement making the position clear. When such statements are volunteered the Court writes "as statements volunteered" the statement has the same value as other statements given by the witness in response to questions put to him. The advocates of both the parties have a right to cross-examine such volunteer statements.

MEDICAL EVIDENCE

Section 3 IEA 1972 defines evidence as: All statements, which the Court permits or requires

to be made before it by the witness, in relation to matters of fact under inquiry, are called oral evidence.

All documents produced for the inspection of the Court are called documentary evidence. Before accepting the documents the Court must be satisfied regarding the identification and the place of seizure. Ordinarily the value of medical evidence is only corroborative. It proves that the injuries could have been caused in the manner alleged and nothing more. The defence makes use of the medical evidence to prove that the injuries could not possibly have been caused in the manner alleged and thereby discredit the eyewitness.

Types

Documentary Evidence

It includes all documents produced before Court during trial. According to **Section 29 IPC** the word document means "Any matter expressed or described upon any substance by means of letters, figures, marks or by more than one of these means intended to be used or which may be used as evidence of that matter." The documentary evidences are embodied in the Sections starting from Section 61 to Section 90 of the IEA. The contents of documents may be proved either by primary or secondary evidences (**Section 61 IEA**).

Primary evidence means the document itself produced for inspection of the Court, (**Section 62 IEA**). The secondary evidence means and include the certified copies, copies made from the original by mechanical process, copies made from or compared with the original and oral accounts of the contents of a document given by a person who has himself seen it (**Section 63 IEA**). Tape-recorded statements are admissible as evidence (*K.S. Mohan vs. Sandhya Mohan* AIR 1993 Mad 59). Evidence must confirm to the matters in issue and is admitted on the basis of relevance and admissibility.

Oral Evidence

Section 59 IEA says that all facts, except the contents of documents or electronic records may be proved by oral evidence.

Direct Evidence The oral evidence must be direct (**Section 60 IEA**) for example when it refers to a fact that could be seen/heard/perceived, it must be the evidence of that person who saw/heard/perceived it. It must be direct if it refers to an opinion or to the grounds on which that opinion is held. The evidence must confirm to the matters in issue that is a knife that has caused stab injury, a medical prescription that resulted in the ill effects to the patient.

Indirect Evidence (Circumstantial) At times the other surrounding facts are taken into consideration that are consistent with the direct evidence for example 'A' committed murder of 'B' with knife at a particular place, date and time. Here 'C' will depose that he saw 'A' on that day, date and time with a knife in hand just before the murder. This evidence of 'C' has a bearing to the fact.

Hearsay Evidence Here the persons other than the witness give the statement. For example during the trial 'A' told that 'B' had told him that he has seen 'C' while committing the crime. Here 'B' can give the direct evidence as he has seen 'C' committing the crime.

Varieties of Documentary Evidence

Documentary evidence is of the following:

Medical Certificate Medical certificates are regarded as the simple form of documentary evidence. They are issued by a registered medical practitioner in respect of ill health, age and mental condition. The certificates are also issued in cases of death, a vital document, may be needed during cremation, transportation outside state and certain claims. It is accepted in the Court of law as a piece of evidence as to the facts stated in the medical certificate. Even the doctor who has issued it may be summoned to the Court of law to testify the contents of the certificate on oath and if needed cross examination can be done. Thus the medical practitioner must exercise due care and skill in issuing such certificate. The certificate must state the exact nature of illness and should contain doctor's opinion in clear language. In case of ill health the doctor should write the exact nature of illness, the duration of illness and probable

- Medical certificate
- Death certificate
- Medicolegal reports
- Dying declaration.

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period of absence in cases of employed persons. The signature of the patient should be at the bottom with the date indicating that he attended the clinic and the doctor has examined him while issuing the certificate. The signature is to be verified by the doctor.

Death Certificate It is essential that identification and cause of death is to be determined for the lawful disposal of the dead body. The registration of Birth and Death Act came into force from 1st April 1970. As per the act all the births and deaths are to be registered throughout India. In case of death certificate the doctor must inspect the body and satisfy that the person is dead. He is legally bound to issue the certificate he has attended the patient during his last illness indicating the cause of death. According to the recommendation of Brodrick committee a doctor should not be allowed to issue a death certificate unless he has attended the deceased at least once during seven days preceding death. He cannot charge any fee for issuing a death certificate. He cannot delay or refuse to issue a certificate. He may subsequently sue the legal heirs for his pending dues. He must refuse to give a certificate on the following grounds. (A) if he is not sure about the cause of death. (B) any suspicion of foul play. (C) death by violent and unnatural cause, drugs, poisoning etc. If he is not sure regarding the cause of death and suspects some foul play he should not issue the certificate, instead he should inform the police. Issuing a false certificate under **Section 197 IPC** is punishable under **Section 193 IPC** with imprisonment that may extend to 7 years and also fine. In general in obtaining a certificate the public expectations are great because they think by giving some money anything can be written in a certificate and try to produce false certificates for various purposes.

Death certificate is a predicament in any doctor's practice. The relatives of the deceased may plead, persuade, pressurize, offer a price and at times even threaten the doctor. He may be tempted at times on humanitarian grounds to issue a certificate. The doctor may certify death in doubtful cases but must not give a cause of death and he should inform the police. Failure to do so may result in the doctor being prosecuted under

Section 39 CrPC. Under **Section 17 (1)(b) of RBD Act 1969**, any person may obtain an extract from the registrar relating to any death after paying the requisite fee, though it will not disclose particulars regarding the cause of death as entered in the register. This confidentiality can be overridden in public interest.

International format of death certificate: The death certificate recommended by WHO for international use, is in two parts.

Part I: Records (a) immediate cause: the disease or condition directly leading to death and (b) antecedent causes viz, the morbid conditions, if any, giving rise to the cause mentioned in (a). (c) is the contributory cause to (a) and /or (b). Thus (a) must be due to (b) which must be due to (c), etc. When many conditions are involved, write the full sequence-one condition per line, with the most recent condition (immediate cause) at the top and the earliest (the condition that indicated the sequence of events between normal health and death) the last. The basic pathological condition is the one, which is mentioned on the lower most line, and this is the one that is used for statistical and epidemiological purposes.

Part II: Records other significant conditions contributing to death, but not related to the disease or condition causing it.

International forms are used by various municipal authorities in our country and are available for doctors. Doctors should necessarily use their rubber stamp after affixing signature to death certificate.

Medicolegal Report These are the legal documents prepared by the doctor at the request of investigating officers usually in criminal cases like injury cases, sexual offences, murder and poisoning cases. These reports are made in both living and dead cases. The request usually comes from police officer or magistrate. It usually comes in two parts. The first part consists of observations and findings of the cases while the second part consists of the opinion and conclusion.

The doctor starting the examination should write in detail the name of the individual, date, time and place of examination. The identification marks should be noted and the consent has to be taken because any examination without consent is

regarded an assault in law. The victim should be explained the purpose and outcome of such examination before taking the consent. While examining a female, a lady registered medical practitioner should examine (**Section 53(2) CrPC**). Where the lady doctor is not available a female attendant should be present at the time of examination whose signature should be taken in the report.

The findings are observed in detail and a report is prepared. The opinion or conclusion drawn should not be biased one as it is subjected to cross examination in the Court of law. The opinion must be kept pending till the reports are available in cases where necessary investigations are required. The negative findings are also to be pointed out in the report.

These reports are produced in the Court of law. The doctor who has made the report should attend the Court to testify under oath, and then only they are taken as evidence. If the doctor is not available someone else can attend the Court to testify his signature and handwriting before being taken as evidences.

Dying Declaration This topic is always mentioned in all the textbooks of forensic medicine though it is relatively an unimportant matter. Its major purpose is to render it admissible. Dying declaration is any evidence from a person who dies before the case is heard. Such a dying declaration must have corroborative evidence to support it before it can be accepted (**Section 157 IEA**).

The dying declaration has been incorporated in **Section 32 of IEA** that reads as "a statement 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: (i) When it relates to the cause of death (ii) When a statement made by a person as to the cause of his death or as to any of the circumstances of the transaction which resulted in his death. Such a statement 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 may be the nature of proceeding where his cause of death comes in question.

The above provisions were made on mainly two grounds: (i) At times the victim being the only eyewitness to the incident, exclusion of his or her statement will not be justified to meet the justice. (ii) Impending sense of death that is taken as equal to oath and the person is believed to tell the truth at that time.

Recording dying declaration: If there is time the magistrate should be called to record the declaration in the absence of magistrate the doctor should record it either at the site of accident or when the patient is being taken to the hospital. The police officer can also record it and even anybody like the panchayat head or the relatives can record the declaration depending upon the situation. However the weightage of dying declaration becomes less and less in the descending order.

Procedure: In the hospital admitted cases; the treating physician first tries to stabilize the patient and then informs the police. If the doctor thinks that the patient life is in danger he will arrange for the dying declaration. The police inform the area magistrate for the purpose. The doctor first has to certify that the patient is in perfect mental condition to make a declaration i.e. the patient is in "compos mentis". If the magistrate is not available or there is much delay of his arrival then the doctor will write the declaration. The police should be discouraged to be near the patient so as to avoid the undue influence. The doctor should be there throughout the declaration to check the mental condition. If during declaration the patient becomes unconscious or unfit for a declaration the recordings will be stopped at that time putting the signature of the person who is recording with date and time. The declaration is recorded again when the doctor declares the patient fit. If the dying person is unable to speak but able to make sign in answer to questions this can be recorded and regarded as verbal statement. It can also be recorded in the form of questions and answers. If at any stage a particular point is not clear he can be asked to make it clear. At the end the declaration is read before the patient who puts his signature or thumb impression. It is also signed

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by the recording magistrate, the doctor and the witness mentioning the date and time.

The declaration may take in the form of first information report of a statement before the police (**Section 162 CrPC**) during investigation. There is no prescribed form to record a dying declaration and no rigid rule can be formed.

When a deceased person makes more than one dying declaration then they should be taken as one for the purpose of conviction. If the subject matter differs efforts should be made to reconcile. If at all the differences exist about the facts that should be covered by the explanation to the **Section 161 CrPC** and to be considered as a matter of fact in each case.

If the person survives after making a declaration then he is called to the Court and his evidence is taken. Therefore the dying declaration recorded earlier acts as corroborative evidence.

Dying Deposition There is no provision of dying deposition in the Indian Evidence Act therefore it is not followed in India. Here the magistrate records the evidence after administering oath. The accused or his lawyer is present and they are allowed to cross-examine the victim.

WITNESS

Section 118 IEA states about the person who may testify as witness in Courts of Law and reads as "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 rational answers to those questions as a result of tender age, extreme old age, disease of mind or body or any other cause of the same kind." Even a mentally ill person, lunatic is not incompetent to testify unless he is prevented by his lunacy from understanding the questions put to him.

Types

Common Witness (Ordinary Witness)

Common witness is a person who has seen/observed the facts. It has to be proved that he/she was capable of perceiving the facts or has

actually seen the facts. This principle is known as "first hand knowledge rule". For e.g. 'A' told in the Court that he had seen 'X' and 'Y' fighting in a garden on a particular date and time.

Expert Witness

Section 45 IEA deals with opinion of experts. They are not only medical experts, but also experts in other subjects. It reads as when a Court has to form an opinion upon a point of foreign law or of science or art or identity the handwritings and fingerprints that are relevant facts. Such persons because of their professional training and experience are capable of drawing a conclusion on the facts observed by them or by others and are called experts. They also give expert opinion on hypothetical questions as well as on matters of common knowledge. The answer should be given by an expert witness after understanding the questions clearly and whenever possible in a guarded manner, the examples like the findings are consistent with the alleged history, does not mean that another mechanism cannot produce the same findings. Conclusions must be based on the facts because it is more important than the opinions.

- Common
- Expert

Hostile Witness

Hostile witness is one who purposefully makes statements contrary to the fact or has some interest or motive for concealing truth. **Section 191 IPC** states that whoever, being legally bound by an oath or by an express provision of law to state the truth, or being bound by a 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 belong to be true, is said to give false evidence. The common as well as expert witnesses can be hostile.

Long Questions

1. What is inquest? Discuss different types of inquest prevalent in India and which is the best.
2. Discuss the various types of documentary evidences in medicolegal practices.
3. Discuss the various types of evidences.

Short Questions

1. Cross Examination
2. Dying declaration
3. Powers of magistrates
4. Types of witness
5. Magistrate's inquest
6. Subpoena.
7. Difference between police and magistrate inquest
9. Expert witness
10. Hostile witness

MULTIPLE CHOICE QUESTIONS

1. Section 174 CrPC deals with:
 - A. Inquest
 - B. Murder
 - C. Exhumation
 - D. Inquiry
2. Leading questions are permitted only in:
 - A. Examination –in-Chief
 - B. Cross Examination
 - C. Re Examination
 - D. Dying declaration
3. A Cognizable offence signifies:
 - A. Arrest without warrant
 - B. Imprisonment up to 1 year
 - C. Imprisonment up to 5 years
 - D. Only fine
4. The present Criminal Procedure Code has been enacted in the year
 - A. 1947
 - B. 1963
 - C. 1973
 - D. 1985
5. Code of Civil Procedure which is being followed now a day was amended in the year:
 - A. 1950
 - B. 1962
 - C. 1976
 - D. 1982
6. A second class Magistrate can pass a sentence up to:
 - A. 1 year
 - B. 2 year
 - C. 3 year
 - D. 4 year
7. Chief Judicial Magistrate can pass a sentence up to:
 - A. 5 years
 - B. 7 years
 - C. 10 years
 - D. Life imprisonment
8. Minimum age to be a witness in the court of law is:
 - A. 7 years
 - B. 12 years
 - C. 16 years
 - D. Any age
9. Dying declaration ideally is to be recorded by:
 - A. Police
 - B. Medical Officer
 - C. Magistrate
 - D. Session's Judge
10. In dying deposition which of the following is fulfilled?
 - A. Statement is to be recorded by Magistrate
 - B. Oath is to be taken by the patient
 - C. Cross examination to be done
 - D. All of the above
11. Which of the following document is accepted in the court of law as evidence without being examined the person who has written it?
 - A. Dying declaration
 - B. Expert opinion expressed in a treatise
 - C. Chemical examiners report
 - D. All of the above
12. Conduct money is paid to witness, if he/she comes from a distance of more than:
 - A. 2 kms
 - B. 3 kms
 - C. 5 kms
 - D. Any distance
13. Dying declaration can be recorded by:
 - A. Magistrate
 - B. Police
 - C. Medical officer
 - D. All of the above
14. Before recording the dying declaration, the attending medical officer has to:
 - A. Certify the mental condition
 - B. Make a medicolegal report
 - C. Treat the patient
 - D. All of the above
15. Leading questions are allowed during Examination-in-chief, if the witness:
 - A. Has no previous criminal record
 - B. A hostile one
 - C. Comes to court for 2nd time
 - D. Has a criminal record
16. Summon is a document of the court:
 - A. Compelling attendance of a witness
 - B. Guarantee money of witness
 - C. Providing legal immunity to witness
 - D. That can be attended at will

1 A 2 B 3 A 4 C 5 C 6 A 7 B 8 D 9 C 10 D 11 D
 12 D 13 D 14 A 15 B 16 A

Laws in Relation to Medical Practice

The medical profession is guided by a code of ethics which are formulated by the National Medical Council and State Medical Council alongwith World Medical Association. The ethics are nothing but the moral principles that guide the members of medical profession. The enforcement is done by the Medical Council of India and state medical council.

INDIAN MEDICAL DEGREES ACT 1916

The Act was passed to grant the title in respect of the qualification of western medical science. According to the Act, we have right to grant medical degrees, diplomas and certificates to practice the western medical science but false use of a title is punishable under the Act.

MEDICAL COUNCIL OF INDIA

The Medical Council of India was established by the Indian Medical Council Act of 1956 to maintain a medical register for India and for the matters concerned with it. The act is applicable to the whole of India.

Constitution and Composition of the Council

The council consists of members as follows:

1. One member from each state other than a union territory, to be nominated by the central government in consultation with the state government concerned.
2. One member from each university, to be elected from its medical faculty, by the university senate or court.
3. One member from each state in which a state medical register is maintained, to be elected

from amongst themselves by all the persons enrolled on such register who possess a qualification included in the 1st, 2nd or part II of the third schedule.

4. Seven members to be elected from amongst themselves by persons enrolled on any state medical register and possess a qualification included in part 1 of the third schedule.
5. Eight members to be nominated by the central government.

The president and vice president are elected from amongst the members for a term of 5 years or till their membership expires, This is a provision that they can get re-elected or re-nominated. The council meets at least once in a year and all acts of the council are decided by a majority of the members present and voting. An elected or nominated member is deemed to have vacated his seat if he is absent without excuse, for 3 consecutive ordinary meetings of the council or if he is no longer a member of the medical faculty or he/she ceases to be enrolled in the medical register.

The council constitutes from amongst its members an executive committee (of not less than seven members) and any other committees required. The president and the vice-president of the council are also the president and the vice-president of the executive committee respectively.

It also appoints a registrar who acts as a secretary and if necessary as a treasurer to look after the day to day business of the council.

Functions of The Medical Council of India

The main functions of the Medical Council of India are as follows:

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1. *Maintenance of a Medical Register:* The Council maintains a register for the medical practitioners where the names of all the registered doctors are entered. It also contains the names of the doctors who were registered in the State Medical Council. If due to some reason the name of the doctor is erased from the State Medical Register, it is automatically also erased from the Central medical Register maintained by the Medical Council of India.
2. *Medical Education:* It maintains the uniform standard throughout the country in respect to Undergraduate and Post Graduate courses. There is a Post Graduate Medical Education Committee consisting of nine members to maintain a uniform standard in respect to Post Graduate Qualification imparted by different Universities. They also advise the Universities and give guidance in this respect. Prior approval of M.C.I. is required for starting a PG course in any discipline. If not followed the M.C.I. has power to derecognised the said qualifications. As regards to Under Graduate Medical Education, the Council prescribes minimum standard. It appoints Medical Inspectors to inspect the various colleges before giving sanction either to start a new college or to renew it. Even to increase the numbers of seats in Medical College requires M.C.I.'s prior permission. The inspectors submit the report to the M.C.I. which is considered by the committee formed. Then the committee recommends to the Government to grant permission or not. In cases when the Council is not satisfied, it recommends the Government to withdraw the recognition.
3. *Recognition of foreign medical qualifications* to an Indian National with foreign degree which is not included in the part II of the third schedule applied to central government along with the full information in respect of the syllabus and duration of course etc. The Central Government forwards the applications to Indian Medical Council which has authority to enter in negotiations with any of the Medical Council of the Foreign Countries and can recognize such qualifications. Then the Central Government by official gazette notification may amend the part II and includes such qualifications.
4. *Appeal against disciplinary action:* When name of any doctor is removed from the state medical register, he may appeal to Central Government after exhaustion of all the official remedies under the state medical council. He should apply within 30 days from the date of decisions alongwith all the relevant documents. The Central Government in consultation with the Medical Council of India will decide which is binding on state medical council.
5. *Warning notice:* The Medical Council prescribes standard of professional conduct and code of ethics for the doctors. If the medical practitioner fails to abide the code he can be issued warning notices in respect to professional misconduct.

CODE OF MEDICAL ETHICS

The oldest one is the **Hippocratic Oath** which is being followed till date. It reads as follows:

I swear by Apollo the physician, by Aesculapius, Hygiea and panacea and I take to witness all the Gods, all the Goddesses to keep according to my ability and judgment the following oath:

“To consider dear to me as my parents him who taught me this art; to live in common with him and if necessary to share my goods with him; to look upon his children as my own brothers, to teach them this art if they so desire without fee or written promise; to impart to my sons and the sons of the master who taught me and the disciples who have enrolled themselves and have agreed to the rules of the profession, but to these alone, the precepts and the instruction. I will prescribe regimen for the good of my patients according to my ability and my judgment and never do harm to anyone. To please no one will I prescribe a deadly drug, nor give advice which may cause his death. Nor, will I give a woman a pessary to procure abortion. But I will preserve the purity of my life and my art. I will not cut for stone, even for patients in whom the disease is manifested I will leave this operation to be performed by practitioners (specialists in this art). In every house where I come I will enter only for

the good of my patients, keeping myself far from all intentional ill-doing and all seduction, and especially from the pleasures of love with women or with men, be they free or slaves. All that may come to my knowledge in the exercise of my profession or outside of any profession or in daily commerce with men, which ought not to be spread abroad. I will keep secret and will never reveal. If I keep this oath faithfully, may I enjoy my life and practice my art, respected by all men and in all times; but if I swerve from it or violate it, may the reverse be my lot."

DECLARATION OF GENEVA 1968

It was amended at Sydney in 1968. At the time of being admitted a member of the medical profession:

I will solemnly pledge myself to consecrate my life to the service of humanity;

I will give to my teachers the respect and gratitude which is their due;

I will practice my profession with conscience and dignity;

The health of my patient will be my first consideration;

I will respect the secrets which are confided in me, even after the patient has died;

I will maintain by all means in my power the honour and the noble traditions of the medical profession;

My colleagues will be my brothers;

I will not permit considerations of religion, nationality, race, party politics or social standing to intervene between my duty and my patient;

I will maintain the utmost respect for human life from the time of conception; even under threat, I will not use my medical knowledge contrary to the laws of humanity.

I make these promises solemnly, freely and upon my honour.

Professional Misconduct

It is also known as infamous conduct or disgraceful act. It is the improper conduct of the doctors in the professional aspect. What really constitutes misconduct is controversial. But the M.C.I. has enlisted the acts or behavior that constitutes the

professional misconduct. If the doctor's act falls in any of the enlisted acts, he is liable for disciplinary action. The conduct of the doctor is judged by the professional opinion of good repute and competence. The M.C.I. is also empowered to take action against an act done by the doctor even if it is not included in the list.

The use of Red Cross Emblem by the doctors is prohibited. The Medical as well as paramedical staffs use it, which is wrong. They can be penalized under section 13 of Geneva Convention Act 1960 up to Rs.500/- fine and forfeiture of the property on which it has been used. Section 12 of the above act 1960 prohibits its use and allied emblem without the approval of Central Govt.

Warning Notice

The Medical Practitioner is required to observe certain rules of conduct contained in the code of Medical Ethics prescribed by the M.C.I. and the State Medical Councils. If the doctor fails to observe these, that will be taken as professional misconduct and action can be taken against the doctor. The list of conduct is not a complete one. The doctor can be penalised for a conduct even if not included in the list depending on its nature. He may be issued warning notice or the name may be erased from the register. The following are the few examples for which action can be taken.

1. *Adultery*: If a doctor has voluntary sexual intercourse with a married woman other than his spouse.
2. *Advertisement*. A physician should not exhibit publicly the scale of fees except in the waiting hall of the patients or in his chamber. The doctor can publish in the newspapers or journals regarding his change of address maximum two times. Even he can publish regarding his timings, interruption or restarting of the practice, in the press after a long interval. He can write to the lay press in his name on matters of public interest and even can deliver public lectures or can give talks on the radio. He should not use an unusually large signboard and to write on it anything other than his

name, qualification and the name of his speciality. The same should be contents of his prescription paper, which may in addition contain his address registration number and telephone number. It is unethical to fix a signboard on a chemist shop or in places where he does not reside or work. He should not advertise himself through manufacturing firms directly or indirectly.

3. *Association:* It is unethical to show improper conduct or association with the patient or his family. He should not associate himself with the manufacturing firms in the form of ownership, should not receive any rebates or commission for prescribing medicines. He should not use abbreviations, which can be read in only one particular pharmacy to dispense medicine.
4. *Abortion:* He should not perform an abortion that is not under the cover of MTP Act or he will not assist any unqualified person for doing a criminal abortion.
5. *Alcohol:* He should not practice medicine under the influence of alcohol, which interfere the skilled performances.
6. *Addiction:* He should not use any drugs of addiction that can hamper the practice.
7. *Dichotomy or fee splitting,* receiving or giving commission or other benefits to a colleague or drugs manufacturer.
8. Using touts and agents for fetching patients.
9. Not informing about the notifiable diseases to Health Authority.
10. Disclosing the patient's secrets learnt during examination and treatment without consent.
11. Refusal to treat a patient on religious grounds.
12. To run a chemist shop for the sale of medicine or for dispensing prescriptions of other doctors.
13. Issuing false certificates in connection with sick benefit insurance or passport etc.
14. He should not assist a person who has no qualification to attain or treat a patient.

The Medical Council of India brought into force the new ethical regulations in year 2002 called the Indian Medical Council regulations, 2002. They

are binding on all doctors in India. Its salient points are as follows:

1. The doctors are free to choose their patients but should not arbitrarily refuse treatment and must not refuse to help in an emergency.
2. Doctor should not refer their patients for consultation to other doctors or clinical investigations unless absolutely necessary.
3. Once a case is taken, the doctor must not abandon or neglect the patient without proper reason and appropriate notice to the patient and his family.
4. Doctors must display their fees and other charges clearly and should write clear prescriptions for dispensing medications.
5. Doctors must not provide any endorsement for medications and equipment with or without any financial gains.
6. Drugs should be prescribed with their generic name as far as possible.
7. No sex determination tests should be done with the intention to destroy the female fetus if present.
8. The patients must not be subjected for trials except under ICMR guidelines.
9. Doctors should keep records for a minimum period of 3 years for in patients and must produce the documents if asked for within 72 hours.
10. The doctors should undergo 30 hours of CME training in every five years for renewal of registration.

CONSENT

It is defined as voluntary agreement, compliance, and permission. **Section 13 of Indian Contract Act** defines that "two or more persons are said to consent when they agree upon the same thing in the same sense." According to Section 14 of Indian Contract Act, Consent is said to be free, when it is not caused by (i) coercion, as defined in section 15, or (ii) undue influence, as defined in section 16 or (iii) fraud, as defined in section 17, or (iv) misrepresentation, as defined in section 18, (v) mistake subject to the provisions of sections 20, 21, 22.

Reasons for Obtaining the Consent

To examine, treat or operate upon a patient without consent is regarded as assault in law, even if it does not cause any harm and even if it is beneficial and done in good faith, for which the patient may sue for damages. If the doctor fails to give the requisite information to a patient before asking for his consent for a particular operation / treatment he may be charged for negligence.

Types of Consent

1. Implied
2. Express
 - Oral
 - Written—*informed*.

Implied Consent

This type of consent is specifically stated by the patient and is seen in routine medical practice and is quite adequate. Here consent is implied in the mere fact that patient comes to physician with a problem or when a patient holds out his arm for an injection.

If the procedure of diagnosis is simple and straightforward, the risk is negligible and uncommon and the conduct of the patient implies willingness to undergo treatment. In spite of everything, if there is the slightest fear of complication, the doctor should seek express consent.

Express Consent

May be written or verbal, and should be obtained for any procedure beyond the routine physical examination, like operation, collection of blood, blood transfusion etc. The nature and consequences of the procedure should be explained before getting consent. Oral consent in the presence of a disinterested third party is as good as written one.

Doctrine of Informed Consent

In informed consent, the patient is informed about his/her condition of the disease, nature of the proposed treatment whether surgical or medicinal methods, any alternative procedure available or not, the possible risks and benefits of the proce-

dures and the percentage of chances of success and failure.

The patient should be told in a simple language so that he can understand and give consent in the presence of third party. After that any untoward reaction or harm to the patient cannot lead to charge the doctor for negligence.

The exceptions to the informed consent are:

1. *Emergency*—in an emergency it is assumed that the patient gives consent. So the consent is implied in these cases.
2. *Incompetence*—the incompetent patients may be treated without permission. In general incompetent patients are unable to make rational decisions. In this category the patients of unconscious, delirious, senile or grossly psychotic in nature are included. When patients are in intensive care unit they are treated without informed consent. So it is best either in an emergency or incompetence situation.
3. *Therapeutic privilege*—in this category the physician can be excused of taking informed consent when disclosure of information could have a detrimental effect on the patient.
4. *Waiver*—the last exception allows a patient to waive his right to informed consent. In these cases the patient may delegate the right to the physician or to a third party to make the decision for him.

If one of the exceptions to the informed consent applies, the physician should document the exception under these circumstances how disclosure and consent should occur but in emergency time may not permit disclosure and consent. If patient is incompetent or therapeutic waiver is used, a surrogate should give informed consent for the patient.

Informed refusal: The doctor has a duty to inform the patient that he has a right to refuse the treatment/investigations to be done. If after listening to the doctor, he thinks so, he can refuse to consent.

Rules of Consent

Consent is necessary in every medical examination. Ordinarily, formal consent to medical

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examination is not required because the patient conducts himself/herself in a manner which implies consent.

Written consent is not necessary in every case. However it should be taken for proving the same in the court if necessity arises. In medico legal cases written consent is a must.

Any procedure beyond routine physical examination such as operation, blood transfusion, collection of blood etc. requires express consent. It must be taken before the act and not at the time of admission in to the hospital.

The consent should be free, voluntary, clear, informed, direct and personal. There should be no undue influence, fraud, and misrepresentation of the facts, compulsion and threat of physical injury, death or other consequences.

The doctor should inform the patient that he has the right to refuse to submit to the examination and that the results may go against him / her. If he/she refuses it is an absolute bar for examination except under S 53 CrPC.

Written consent is taken for a specific procedure. It is not same as blanket permission for general procedure where the surgeon can decide in the operation theater itself regarding the type extent of operation.

The doctor should explain the object of the examination to the patient and he should be informed that the findings would be embodied in a medical report.

In criminal cases the victim cannot be examined without consent. The court also cannot compel a person to get medically examined against his will. If the person is arrested being charged of some offence and the examination of his body will provide some evidences toward the commission of the crime he can be examined by a registered medical practitioner even without his consent and if necessary by using reasonable force. (Under Section 53(1) CrPC) and section 53 (2) CrPC mentions that in case of a female the examination should be done by a female medical practitioner or under her supervision.

Under section 54 CrPC and an arrested person can request to be examined by a doctor to detect

any evidences in his favour. Under section 87 IPC a person above 18 years of age can give consent to suffer any harm if the act is not intended and not known to cause death or grievous hurt. Under section 88 IPC a person can give consent to suffer any harm if the act is not intended and not known to cause death or grievous hurt if the act is done in good faith and for the benefit of the person.

Under section 89 IPC for a child under 12 years of age or a person of unsound mind cannot give consent to suffer any harm for an act which may cause grievous hurt or death even in good faith, but the consent has to obtained from the guardian of the child or person. Under section 90 IPC - consent given by an insane person or given under fear of injury, death etc. or due to misunderstanding of a fact is invalid. Under section 92 IPC any harm caused to a person in good faith even without the person's consent is not an offence if the circumstances were such that it was impossible to obtain consent of the person or his lawful guardian in time for the thing to be done for the benefit of the person, but the act should not extend to intentional causing hurt other than for preventing death or hurt or curing of grievous disease or infirmity.

Professional Secrecy

In the Hippocratic Oath there is an affirmation by the doctor that whatever in connection with my professional practice or not in connection with my professional practice or not in connection with what I see or hear in the life of men, which ought not be spoken of abroad, I will not divulge, as reckoning that all such be kept secret'.

Even the places where oath is not formally given in qualifying ceremony, there is an implied acceptance of its spirit and intentions as the ideal standard of his professional behaviour. Therefore, both on ethical grounds and also because he may be used in the civil courts for breach of confidence, the doctor should respect this professional secrecy to the utmost of his ability. The following examples may be kept in mind:

1. The doctor should not discuss any point with others except with the consent of the patient.

2. If the patient is a minor a doctor should not disclose anything about his illness even to his parents without getting the consent.
3. In case of husband and wife the facts relating to the nature of illness of one should not be disclosed to the other without the consent.
4. If the doctor examines a domestic servant at the request of the master who is paying the fees still he should not disclose even to the master without the consent of the servant.
5. Doctors in government service are bound by code of professional secrecy even if they are treating the patients free of cost.
6. The criminals in police custody has right not to allow the treating doctor to disclose about the nature of illness to any person. However once the person is convicted he has lost this right and the doctor can disclose to the proper authorities.
7. While reporting about any case in a medical journal care should be taken not to disclose the identity of the person.
8. When somebody submits for medical examination while taking the life insurance the doctor can disclose the result to the proper authorities because of implied consent.
9. A doctor should not disclose any information about the illness of his patient without consent whenever requested by a statutory body except notifiable diseases.
10. The examination of the dead body at times may reveal certain facts that affect the reputation of the deceased as well as the family members so the doctor should not disclose the fact to others.

Exceptions to Professional Secrecy

Professional secrecy should be broken in certain situations that are known as **privileged communication**

It is a statement made by the doctor to the concerned authority to protect the interest of the community or state. To be privileged it must be made to the concerned person/authority. If the plea of privilege facts is made to more than one

person, then in these situations, the doctor must tell the patient to give the consent. If he fails, then he will tell proper authority to protect the interest of the community. The following are the examples of privilege communications.

Infectious diseases: If anybody having infectious diseases gets an employment, she/he should be persuaded not to undertake the job till he becomes cured. If somebody is found to be suffering from infectious diseases, during his employment, he/she should also be told to go on leave till he/she becomes all right. If they refuse, the doctor can tell the employer about it.

Example: A schoolteacher with tuberculosis, Cook of the hostel with typhoid.

Notifiable diseases: Medical persons has a statutory duties to notify births, deaths and other diseases to the Public Health Authority.

Suspected crimes: The doctor must inform the police (U/s 39 CrPC)

Servants and Employees: When an employee is suffering from a serious disease like colour blindness in case of drivers, Epilepsy in case of cooks, they should be persuaded to change their profession, otherwise the employer should be informed about the illness.

Sexually transmitted diseases: Person having a sexually transmitted disease intending to join in swimming pool or going for marriage, should be persuaded not to join the swimming pool or go for marriage. If he does not listen, then the doctor should inform to the proper person/ authority.

In patient's interest: If the patient having suicidal tendency, the parents/guardians may be informed about, so that, they can take proper care as well as provide proper treatment.

Court of Law: In the court of law the doctor cannot claim the privilege about the illness of his patient. When asked he should appeal to the court that he should submit in writing, so that the public cannot know about the facts. If the court rejects he has to tell before the court. But under section 126 and 129 IEA, a lawyer can claim the privilege in a court of law requesting any communication made to him by his client.

Negligent suits: When a patient files any suit of negligence against a doctor and he employ

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another doctor to examine the patient, the information thus acquired is not privileged and he can divulge in the court of law.

Self-interest: when a patient files a civil or criminal suit, the doctor can disclose evidence.

Rights and Privileges of Registered Medical Practitioner

- Right to practice medicine.
- Right to choose a patient.
- Right to issue a certificate.
- Right to add title to his name.
- Right to recover his fees.
- Right of appointment to public hospitals.
- Right to give expert evidence in the Court of law.
- Right to dispense medicine.

Duties of a Medical Practitioner

The registered Medical Practitioner has multiform duties towards the patient, colleagues and state. Some of them are follows:

Towards Patients

- *Duty to exercise necessary care and skill:* In a patient comes to a doctor and doctor has right to refuse but when starts treating the patient the duty towards the patient starts. For hospitals, the duty arises when the patient gets admitted to it but in cases emergency the doctor cannot refuse the patient but after giving the first aid can refer with proper precaution to the nearest hospitals where facilities are available. He could use his care and skill according to his knowledge and qualification in cases of minors, the doctor has the duty to exercise care and skill even if the fee is being paid by the guardian.
- *To examine the patient properly:* It is the duty of the doctor to examine the patient thoroughly and take proper history from him or from the attendant.
- *To prescribe proper medicines:* The doctor should prescribe proper medicines after carefully evaluating the patient. In cases of Government Hospitals, if the medicines are not available there, the doctor should advice the patient to

purchase from the market. In case of wrong prescriptions causing harm to the patient, the doctor will be liable for the damages.

- *Duty to inform the risks:* The doctor should inform the patient about the disease and possible risks involved. If the patient is not of sound mind the guardian should be informed. After getting the consent the doctor should get his consent for the management. In cases where the doctor thinks that after telling the risks, there will be great psychological upset, he can withhold some of the risks.
- *Duty to give proper instructions:* The doctor should give full instructions to the patient or to the attendants regarding the use and type of diet to be taken. He should also mention the dose and frequency of the medicines to be used. In cases of any untoward reaction the patient should be advised to report immediately.
- *Instructions to those patients who cannot take care of themselves:* In cases of mental ill patients or comatose patients, their attendants or guardians to be properly instructed about the use of medicine.
- *Duty in relation to operations:* After careful diagnosis when the surgeon thinks to operate the patient, he should explain the nature and extent of operation to the patients, the possible risks involved in these cases and any alternative method available. He must obtain consent before operation. He must not experiment on the patient. He should be sure that no swabs, instruments are left inside the body after operation. He must take post-operative care properly to avoid any complication.
- *Inform third parties:* In cases of patient suffering from infectious diseases, the doctor should not only warn the patient but also the third party i.e. relatives, friends and co-workers, so that they can take proper precautions. He also has duty to inform the proper Health Authorities.
- *Poisoning case:* When the doctor attend a suspected poisoning case, he has to do the stomach wash with plain water so that the sample can be sent for chemical analysis. He has to preserve vomitus, blood and urine for chemical analysis. After that he should treat

with suitable anti-dots and take other steps towards treatment.

Toward Colleagues

- *Never criticize:* A medical practitioner should never criticize his colleagues. When a patient comes to him after leaving another doctor, he must not tell to the patient about the ills of that doctor or criticize the medicines prescribed by him earlier in order to gain cheap popularity.
- *Never take fees:* When any specialist examine or treats another doctor, he should not demand any fees from him. It is the professional courtesy he has to extend to his colleagues.
- *Always help:* The doctors should always help his fellow colleagues at the time of need specially any professional matters.
- *Consultation:* It is the duty of a doctor to consult another specialist depending upon the type of disease he is treating. In cases of operation or other emergency situations, he has to take consultation from a specialist in the relevant field. In these situations, the patient must be told that he is being referred to a specialist for consultation. The patient will also be told whether it is only consultation, joint participation or he will be on continuous treatment of the consultant and get proper consent for that. In cases where the patient is completely transferred to another specialist, the liability of the referring doctor ceases and the duty of the consultant starts in relation to the liability towards the patient.

Towards State

- Notifiable diseases.
- Geneva Conventions.

Notifiable Diseases

A doctor is bound to inform the communicable diseases like cholera, plague etc. to the Health Authorities. He is also bound to inform the birth and death to proper authorities. If a doctor fails to inform, a civil suit can be brought against him, but not a criminal suit.

Geneva Convention, 1949

A Medical Practitioner is bound to treat/provide medical aid in cases of person wounded or sick of the armed forces (I Convention), ship wrecked persons (II Convention); prisoner of war (III convention) and civilians of enemy nationalist (IV Convention). All the above categories of person should be treated without any discrimination based on Race, Religion or Political grounds.

MEDICAL NEGLIGENCE (MALPRAXIS)

Introduction: The law of negligence is applicable to the conduct of all individuals whether layman or professional. In addition certain ethical standards are applied to the professionals. Whenever a privilege is bestowed upon an individual, concomitant duties and obligations attach and the physician is no exception.

In present times doctors can not be sure that they will never be threaten with an action for negligence. Even patients are ready to sue who have had excellent treatment with complete cure and without cost. There are three reasons for this:

- 1. Transfer of Hospitals to the State or Central Government.**
- 2. The cost are met out of National funds**
- 3. There is a provision of free legal aid for the patient.**

It is difficult to appreciate that severe disability resulting from an accident during the course of treatment is not necessarily negligence but unfortunately patient may allege negligence and seek compensation.

In Hatcher Vs. Black & Others (1954) Lord Denning compared negligence with a dagger.

The doctors' professional reputation is as much dear to him as his body, perhaps more

so, and an action for negligence could wound his reputation as severely as a dagger to his body.

Professional Negligence is defined as "Want of reasonable care and skill or willful negligence on the part of the medical practitioner while treating a patient resulting in bodily injury, ill health or death."

Negligence was first defined by Justice Baron Alderson in 1856 "The omission to do something which a reasonable man could do or doing something which a prudent and a reasonable man could not do". It could be defined as a failure to perform the duty to exercise a reasonable degree of skill and care in the treatment of patient.

In 1934 Lord Wright defined it as

- . there must be a duty owed
- . Breach of that duty either by omission or commission.
- . Direct causation.
- . Damage

To summarize the doctor owes a duty towards the patient, the doctor was in breach of that duty as a result of which the patient suffered damage. there is a direct relationship between the dereliction of duty and a damage.

Medical negligence is no different in law from any other type of negligence. Negligence medical or otherwise is a civil wrong than as a "tort", rarely the medical negligence may be renewed from a civil action between doctor and patient to the criminal courts, where the state prosecutes the doctor for a severe degree of reckless and dangerous action, amounting to criminal negligence.

Duty of the doctor to choose a patient: The doctor has right to refuse any patient. So any patient who comes to a doctor, he can either refuse or accept for the treatment. Once the doctor accepts the patient, his duty towards the patient starts. The doctor can refuse to accept the patient in the following circumstances (i) Not belonging

to his/her speciality (ii) Facilities are not available at the place. (iii) The doctor is not well or any member of the family is sick. (iv) Busy in important family functions.

Duty of the doctor during an Emergency: During emergency he is morally and ethically bound to provide the best of help he can do to save the life of the patient and see that the patient if not accepted by him, can reach the nearest hospital. Here no patient doctor relationship is established. So if some damage occurs, the patient cannot sue the doctor.

Absence of reasonable care and skill: The duty to exercise reasonable care and skill exists when a doctor-patient relationship is established. This may be true even if the patient is unconscious and quite unaware of the doctor's presence.

A doctor who deals with a patient with the intent to act as a healer establishes a doctor-patient relationship immediately and from that moment he has a legal obligation to exercise due care and skill. Any breach of that duty is a ground for the negligent action.

When a doctor examines a patient for some other purpose than providing advice and treatment, i.e. he is not present as a healer, no relationship is established and there is no duty of care. When a doctor examines a medico legal case like sexual assault, alcoholics, and insurance etc., he is there not as a healer and thus no relationship exists, but there is a duty not to damage, thus if he breaks the needle while taking blood sample, he is liable to pay damages. On the other hand if he reports the findings to the employer and insurance company, no action for damages arises.

Degree of Competence

A doctor must possess reasonable degree of proficiency and he must apply it while treating the patient. A highly qualified doctor will be held negligent if he fails to apply his greater knowledge with sufficient degree of care. Conversely an inexperienced doctor may be negligent if he attempts to do some procedure, which is beyond his capability except in an emergency. The degree of competence is not a fixed quality but varies

according to the status of the doctor.

There is a minimum level of competence for all doctors who have qualified in the examinations and registered in the state medical councils or the Indian medical council. This minimum level is set to protect the public from insufficiently qualified doctors. Beyond this minimum point there is a whole spectrum of proficiency from a newly graduated doctor to a senior specialist. No doctor is expected to possess all up-to-date knowledge nor he can apply all known diagnostic and therapeutic techniques however a doctor of a particular status as regards his grading and experience is expected to have a standard of knowledge and capability corresponding to his position in the profession for example a house surgeon is not expected to possess the same skills as that of a consultant surgeon. A house surgeon volunteering to perform a major surgical operation (not an emergency) might be held guilty of negligence if he causes damage to his patient. This applies to all other specialities such as anaesthesia, medicine and so on. On the other hand if a junior doctor performs any operation or treats a patient in an emergency or where there is no other medical facility available even if there is damage to the patient he will not be held negligent provided that he has acted in good faith.

Breach of Duty of Care

It will not be incorrect to say that categories of negligence are never closed which means that it is impossible to draw a complete list of things that can cause negligence in action because of the advancement of medical technology day by day. Anything a doctor does can be a ground for negligence if the patient is not satisfied and damage occurs.

A doctor is not liable for errors of judgment either in diagnosis or treatment as long as he applied a reasonable standard of skill. Negligence is not a matter of a doctor making a mistake but of not trying hard enough, lack of care and attention for the consequences. A doctor can misdiagnose and mistreat a patient without being negligent even if another practitioner of greater skill and ability

would have had more success. A doctor is not an insurer of as one famous judge said and he doesn't guarantee to provide the best possible care but only care which is reasonably adequate and consistent with his professional status.

Who Sets the Standard?

The court will rely upon the evidence of competent practitioner in the relevant field of medical practice. Approved practice is a criterion for reasonable care. In almost all defence cases it is told that what was done, was done in accordance with general approved practice. Court gives much importance to the expert evidence in the relevant field. A doctor is not negligent if he acts in accordance with general accepted practice. The court pronounces the practice negligent if there are some inherent defects in it. It is not enough to repute negligence by saying that he did it what everybody does. A risky practice may well be held negligent notwithstanding that it is widely followed.

In almost all defence cases it is told that what was done, was done in accordance with general approved practice. Court gives much importance to the expert evidence in the relevant field. A doctor is not negligent if he acts in accordance with general accepted practice.

The court pronounces the practice negligent if there are some inherent defects in it. This is not a usual and normal practice. That the practice was not adopted. The practice adopted is one which no professional man of ordinary skill would adopt.

A medical practitioner when deviates from normal practice it must be established that he is free to exercise his judgment, discretion and the new procedure was done with the consent of the patient for his benefit. Anyone who adopts a noble course of treatment or omits to use some form of diagnosis or treatment, which has gained general approval, must be able to justify his actions if something goes wrong. In these cases success is the best justification.

Duty to Inform the Risks

In a hospital, no matter what care is taken there

is always a risk. Always there is some risk in every operation under general anaesthesia; to what extent the doctor is obliged to inform his patient or relatives about these risks? When there is some non-risk they should be informed before taking the consent. Then there is no room for negligence. For example, in an ECT Therapy the risk of fracture of dorsal vertebrae so the written consent must be taken. The doctor is not expected to inform all possibilities. He is not negligent if he has not informed about remote risks.

Retention of Swabs, Packs, Instruments and Drainage Tube Etc.

Responsibility of recovery of swabs used in operation theatre lies with the surgeon. As he puts the swabs inside he must take them out. He must use degree of care, which is reasonable in the circumstances. That must depends upon the evidences. He cannot say, "I relied upon the nurse". He must ask the nurse if everything is all right. The circumstances in which the nurse's count will be of immense value is that the emergency was so great, the surgeon could not carry out search without endangering the patient's life.

To avoid negligence, the surgeon must satisfy the following: The counting system is efficient and the person working is familiar with it. The system was followed during the operation and the count showed that the number is all right. He must take all reasonable precaution that swabs used were recovered. He must do a visual and manual search of the field. In these cases the plaintiff has only to

prove that he has suffered damage and the onus is upon the surgeon to prove that he is not negligent (Table).

Precautions against Medical Negligence

1. Never guarantee a cure.
2. Obtain informed consent from the patient.
3. The diagnosis to be confirmed by proper investigation depending upon the nature of cases.
4. Reasonable skill and care to be taken
5. Proper, accurate and legible records to be maintained.
6. Immunization to be done specially tetanus in cases of injuries.
7. Sensitivity test to be performed before injecting the drugs known to cause hypersensitivity reactions specially in cases of penicillin, streptomycin and antivenins.
8. The patient or his attendant should be advised that the medicine should be given in the proper dosage and in time and no telephonic consultation should be done.
9. Consultation to be undertaken with appropriate specialist in case of need.
10. Never criticise your colleagues regarding his professional ability.
11. The patients should not be left unattended when she is undergoing labour.
12. In case of absence from practice the patient should be informed or qualified substitute should be arranged.
13. A written informed consent should be

Table 3.1: Differences between civil and criminal negligence

Features	Civil negligence	Criminal negligence
Offence	No specific and clear violation of law	There is specific violation of a particular criminal law in question
Negligence Conduct of physician	Simple absence of care and skill Compared to a generally accepted standard of profesional conduct	Gross negligence, inattention or lack of competency Not compared to a any single test
Consent for act	Good defence in court of law cannot recover damages	Not a defence; can be prosecuted
Trial	Civil court	Criminal court
Evidence	Strong evidence is sufficient	Guilt should be proved beyond reasonable doubt
Punishment	Damages to be paid	Imprisonment

obtained before carrying out surgery and anaesthesia and the detailed surgical procedures and possible risks should be explained to the patient. Anaesthesia should be given by an expert in the field.

14. Before a surgical operation, the surgeon should make sure that all the instrument are in good working condition and proper count of the instruments should be done by the nurse before and after completing the operation.
15. Whenever a death occurs from anaesthesia or during surgical operation the doctor should report to the police.
16. For carrying out any research work on the patient his consent should be taken.
17. No female patient to be examined without the presence of a third person preferably a female or by a female registered medical practitioner.
18. The doctor should be well versed with the advanced medical knowledge in the field.

Contributory Negligence

Contributory Negligence is the absence of reasonable care on the part of the patient or his attendant that combines with the negligent action of the doctor resulting in the damage completed off directly and without which damage should not have occurred. The examples are: Not to provide proper history to the doctor. Failure to follow the line of treatment. Refusal to take up the suggested advice. Leaving the hospital against the doctor's advice. Failure to seek further medical treatment if the signs/symptoms persists.

If the doctor and the patient are negligent simultaneously, the doctor can take a good defence, otherwise the contributory negligence is a partial defence in civil suit only and has no place in criminal negligence. When the patient consents to take the risk of the injuries effects, then he cannot claim damages. When the doctor is not negligent and it is the patient alone who is negligent, it is called "Negligence of the patient".

Limitations of Contributory Negligence

1. Last clear chance doctrine—In this rule the

person who is negligent may recover the damages if the doctor discovered the injury while there was still time to avoid the injury but he failed to do so.

2. Avoidable consequences rule—In these cases the doctor was found to be negligent and he was sued for the same and after that the negligence of the injured person occurs. In these situations the patient is not guilty of contributory negligence since his actions were not the cause of the injury.

Therapeutic Misadventure

A misadventure is a accident or unexpected damage to the patient which the doctor is attending during the treatment is called Therapeutic misadventure. It can be during diagnosis or during experiment.

Almost every therapeutic drug has side effects. So the doctor has to explain the possible side effects while prescribing the drug. When there are more than one drug and there is a choice of treatment for a particular disease, then the doctor should prescribe the drug having lesser side effects. Hypersensitivity to drugs is very common, so when a doctor prescribes a particular drug, he/she has to be very particular to ask about the history of hypersensitivity before starting treatment and at the same time to be equipped with life saving measures if required. The doctor is not liable for the damages caused by administration of a drug unless the element of negligence exists.

Examples: (i) A hot water bottle or heating pads causing burns. (ii) Foetal and neonatal deaths occurring inside uterus while the mother is given some drugs for treatment during pregnancy. (iii) Hypersensitivity reaction caused by administration of Penicillin/streptomycin, tetracycline etc. (iv) Radiological procedures during diagnostic methods can prove fatal. (v) During Barium enema, rupture of rectum causing peritonitis.

Error Of Judgement:

It is a well established fact that a doctor is not expected to do a miracle. The courts do not

condemn an honest exercise of judgement even though other doctors may disagree. To cover up an error of judgement is held to be negligent conduct.

Respondent Superior (Vicarious Liability)

The employer is responsible not only for the wrongful deeds committed by him, but also the wrongful deeds of his employees. The doctor usually employs or supervises other members of staff who may be less qualified persons. He distributes the work and supervises them. If one of the member/s does any negligence, then the principal/doctor is held responsible known as Respondent superior.

Doctrine of Res Ipsa Loquitur

Usually the professional negligence of a doctor must be proved in the Court of Law by the experts in the field. Here the patient has to prove the negligence of the doctor. But in cases of Res Ipsa Loquitur which means "the facts speaks for itself" the patient need not prove the negligence. The rule will be applicable if the following conditions are satisfied:

1. That in the absence of negligence the injury would not have occur.
2. The doctor had exclusive control over the injury producing instrument or treatment
3. There is no contributory negligence by the patient.

Few are the following examples:

1. Breaking of a needle during injection.
2. Failure to remove swabs from the abdomen during operation .
3. Prescribing overdose of medicine.
4. Burns resulting from application of hot water bottle.
5. Failure to give anti-tetanic serum in cases of injury resulting titanus.

This Doctrine is applied to both civil and criminal negligence. It can not be applied against several defendants.

Doctrine of Common Knowledge:

This Doctrine is based on the assumption that the issue of negligence in a particular case is not related to the technical matters which are within the knowledge of medical profession. It is one type of Res Ipsa Loquitur where the patient need not produce evidences regarding the standard of care but in Doctrine of Common Knowledge the patient must prove the causative factor but he need not produce the evidences to establish the standard of care.

Defenses Available in Cases of Negligence

No duty owed to the plaintiff. The duty was discharged according to the prevailing standard. It was a therapeutic misadventure. It was error of judgment. Contributory negligence: It can be taken as defence in cases of civil negligence only. Res judicata: Once the case has been decided it cannot be taken to another court. Only appeal can be made. Limitation: The case should be filed within two years from the date of alleged negligence. If it is alleged that there was a breach of duty to take care under some particular contract between doctor and patient, then the period is three years from the date of breach of contract.

The following points are not good defences:

There was no contractual relationship between the doctor and the patient. Service was rendered free of charge. The medical man had no bad motive.

CONSUMER PROTECTION ACT 1986

The consumer protection act 1986 came into force on 15 April 1987 is a welfare legislation mainly tilting towards the consumer like industrial dispute act that favours the workers.

The act envisages a three tier quasi judicial machinery i.e. District Consumer Disputes Redressal Forum at the District level, State Consumer Disputes Redressal Commission of the State level and National Consumer Disputes Redressal Commission at the National level. The District Forum consists of three members presided by a retired or serving district judge. It entertains claims up to rupees twenty lacs. The State

Commission consists of three members presided a serving or retired high court judge. It entertains claims between rupees twenty lacs to one crore. And the National Commission consists of five members presided by a retired Supreme Court judge, which entertains claims above rupees one crore.

Procedure

The complaint can be lodged at any of the places with or without engaging a lawyer by paying a nominal fee. The complaint may be filed with the district forum by

- The consumer to whom the service has been provided or had been agreed to be provided.
- Any recognised consumer association
- One or more consumers where numerous consumers have the same interest.
- State or central government.

If the complaint relates to any service the district forum shall refer the copy of the complaint to the opposite party directing him to submit a reply within thirty days. Here it is not necessary for the parties to be represented by the lawyers so the disputes are settled on the basis of evidences put before him. The district forum has the same powers as that of a civil court under the code of civil procedure 1908. Every proceeding before the district forum shall be deemed to be a judicial proceeding within the meaning of section 193 and 228 of the IPC 1860.

If the district forum is satisfied that the goods supplied were having some defects specified in the complaints and that any of the allegations contained in the complaint about the services are proved, it will issue an order to the opposite party either to remove the defect, replace the goods or provide adequate compensation. But if the complaint is not proved it will be dismissed and the court directs to pay the costs of the opposite party not exceeding more than rupees ten thousand. But there is a provision that the aggrieved party if not satisfied can appeal in the state commission within thirty days from the date of order. If not satisfied any party can appeal to

the national commission and finally to supreme court within thirty days of the date of order.

Limitation Period

The complaint should be filed in all the above forums within two years from the date on which the cause of action has arisen.

Penalties

For the noncompliance of the order made by district forum, State Commission or National Commission the trader or the person shall be punished with imprisonment ranging from one month to three years and fine from rupees two thousand to ten thousand.

Interpretation of the act by the Supreme Court Since the enactment of the act there was a controversy whether the provisions of the act are applicable to the persons who are rendering medical service to the patients. In this regard there was a difference of opinion between the State Commissions, National Commission and various high courts. The said controversy came to an end by the landmark judgment delivered by the Supreme Court in the case of Indian medical association council vs V P Shantha and others.

The Apex Court arrived at the following conclusions;

Service rendered to patient by medical practitioner except where the doctor renders service free of charge to every patient or under a contract of personal service, by way of consultation diagnosis and treatment both medical and surgical would fall within the ambit of service as defined in section 2(1)(0) of the act.

The fact that the medical practitioners belong to the medical profession and subject to the disciplinary actions of Medical Council of India or state Medical Council constituted under the provisions of the Indian Medical Council act would not exclude the services rendered by them from the ambit of the act.

A 'contract of personal service' has to be distinguished from 'contract for personal service'. In the absence of relationships of master and

servant between the patient and the medical practitioner the service rendered by a medical practitioner to the patient cannot be regarded as service rendered under 'contract of personal service' but such services are regarded as being rendered under a 'contract for personal service' and is not covered by exclusionary clause of definition of service contained in section 2(1)(0) of the act.

The expression 'contract of personal service' in Section 2(1)(0) of the Act cannot be confined to contracts for employment of domestic servants only and the said expression would include the employment of a medical officer for the purpose of rendering medical service to the employer. The service rendered by a medical officer to his employed under contract of employment could be outside the purview of 'service' as defined in Section 2(1)(0) of the Act.

Service rendered free of charge by a medical practitioner attached to a hospital/nursing home or a medical officer employed in a hospital/nursing home where such services are rendered free of charge to everybody, would not be 'service' as defined in Section 2(1)(0) of the Act. The payment of a token amount for registration purpose only at the hospital/nursing home would not alter the position.

Service rendered at a non-government hospital/nursing home where no charge whatsoever is made from any person availing the service and all patients (rich and poor) are given free-service is outside the purview of the expression 'service' as defined in Section 2(1)(0) of the Act. The payment of a token amount for registration purpose only at the hospital/nursing home would not alter the position.

Service rendered at a non-government hospital/nursing home where charges are required to be paid by the persons availing such services falls within the purview of the expression 'service' as defined in Section 2(1)(0) of the Act.

Service rendered at a non-government hospital/nursing home where charges are required to be paid by persons who are in a position to pay, and persons who cannot afford to pay are rendered

service free of charge, would fall within the ambit of expression 'service' as defined in Section 2(1)(0) of the Act irrespective of the fact that the service is rendered free of charge to persons who are not in a position to pay for such services. Free service, would also be 'service' and the recipient a 'consumer' under the Act.

Service rendered at a government hospital/health center/dispensary where no charge whatsoever is made from any person availing the services and all patients (rich and poor) are given free service, is outside the purview of the expression 'service' as defined in Section 2(1)(0) of the Act. The payment of a token amount for registration purpose only at the hospital/nursing home would not alter the position.

Service rendered at a government hospital/health center/dispensary where services are rendered on payment of charges and also rendered free of charge to other persons availing such services would fall within the ambit of the expression 'service' as defined in Section 2(1)(0) of the Act irrespective of the fact that the service was rendered free of charge to persons who do not pay for such service. Free service would also be 'service' and the recipient a 'consumer' under the Act.

Service rendered by a medical practitioner or hospital/nursing home cannot be regarded as service rendered free of charge, if the persons availing service has taken an insurance policy for medical care where under the charges for consultation, diagnosis and medical treatment are borne by the insurance company and such service would fall within the ambit of 'service' as defined in Section 2(1)(0) of the Act.

Similarly, where, as a part of the conditions of service, the employer bears the expenses of medical treatment of an employee and his family members dependent on him, the service rendered to such an employee and his family members by a medical practitioner or a hospital/nursing home would not be free of charge and would constitute 'service' under the Section 2(1)(0) of the Act.

HUMAN EXPERIMENTATION

All research involving human subjects should be conducted in accordance with three basic ethical principles, which have equal moral force and guide the conscientious preparation of proposals for scientific studies. These are:

- **Respect for persons**—It incorporates at least two fundamental ethical considerations
- **Respect for autonomy**, which requires that those who are capable of deliberation about their personal choices should be treated with respect for their capacity for self determination
- **Protection of persons with impaired or diminished autonomy**, which requires that those who are dependent or vulnerable be afforded security against harm or abuse.
- **Beneficence**—Refers to ethical obligation to maximize benefit and minimize harms and wrongs. This principle gives rise to norms requiring that the risks of research be reasonable in the light of the expected benefits and that the research design be sound enough to safe guard the welfare of the research project.
- **Justice**—Refers to the ethical obligation to treat each person in accordance with what is morally right and proper to him/her.

Human experimentation may be:

a. Therapeutic experimentation—Is concerned primarily with improving the condition of a particular patient under treatment

b. Research experimentation—Is concerned primarily with using a human subject as a means of expanding scientific knowledge for the benefit of humanity

c. Innovative experimentation—Are therapeutic procedures that have not yet earned a place in medical practice

The Declaration of Geneva: The Declaration of Geneva of the world Medical Association binds the doctor with the words, “The health of my patient will be my first consideration”

International Code of Medical ethics: It declares, “Any act or advice which could weaken physical or mental resistance of a human being may be used only in his interest”

The purpose of biomedical research involving human subjects must be to improve the

diagnostic, therapeutic and prophylactic procedures and understanding of the etiology and the pathogenesis of the disease. In current medical practices, most of these procedures involve hazards. Medical progress is based on research that ultimately rests in part on human subjects.

Special caution must be exercised in the conduct of research, which may effect the environment, and the welfare of animals used for research must be respected. The results of the laboratory experiments must be applied essentially to human beings to further scientific knowledge and to help the suffering humanity.

World Medical association has prepared the following recommendations as a guide to every doctor in biomedical research involving human subjects. It must be stressed that the standards as drafted are only a guide to physicians all over the world. Doctors are not relieved from the civil, criminal and ethical responsibility under the laws of their own countries. The code of ethics known as Declaration of Helsinki was drawn in 1964 and revised in 1975 as follows.

Basic Principles

Biomedical research involving human subjects must conform to the generally accepted scientific principles and should be based on adequately performed laboratory and animal experimentation. It should be based on a thorough knowledge of scientific literature.

The design and the performance of each experimental procedure involving human subjects should be clearly formulated in an experimental protocol which should be transmitted to a specially appointed independent committee for consideration, comment and guidance.

Biomedical research involving human subjects should be conducted only by scientifically qualified person and under the supervision of a clinically competent medical person. The responsibility for the human subject must always be with medically qualified person and never rest on the subject of the research, even though the subject has given his/her consent.

Biomedical research involving human subjects cannot legitimately be carried out unless the importance of the objective is in proportion to the inherent risk to the subject.

Every biomedical research project involving human subjects should be preceded by careful assessment of predictable risks in comparison with foreseeable benefits to the subject or to the others. Concern for the interest of the subject must always prevail over the interest of science and society.

The right of the research subject to safeguard his/her integrity must always be respected. Every precaution should be taken to respect the privacy of the subject, to minimize the impact of the study on the subject's physical and mental integrity and on the personality of subjects.

Doctors should abstain from engaging in research projects involving human subjects unless they are satisfied that the hazards involved are believed to be predictable. Doctor should cease any investigation if the hazards outweigh the potential benefits.

In publication of results of his/her research, the doctor is obliged to preserve the accuracy of his results. Reports of experimentation that are not in accordance with the principles laid down in this declaration should not be accepted for publication.

In any research on human beings, the subject must be adequately informed of the aims, methods, anticipated benefits, potential hazards of the study and discomfort it may entail and that he/she is at liberty to abstain from participation in the study and is also free to withdraw consent to participation at any time.

The doctor should then obtain the subject's freely given informed consent in writing. The doctor should be particularly cautious if the subject is in a dependent relationship to him/her or may consent under duress. In that case, an independent doctor who is not engaged in the investigation and is completely independent of his official relationship should take informed consent.

In case of legal incompetence, informed consent should be obtained from a legal guardian in accordance with national legislation. If the

person is physically or mentally incapacitated and cannot give informed consent or when the subject is minor, permission from the responsible relative replaces that of the subject in accordance with national legislation.

The research protocol should always contain a statement of ethical considerations involved and should indicate that the principles enunciated in the present Declaration are complied with.

Medical research combined with professional care (clinical research)

In the treatment of the sick person, the doctor must be free to use a new diagnostic and therapeutic measure, if in his/her judgment it offers hope of saving life, reestablishing health or alleviate suffering

The potential benefits, hazards and discomfort of a new method should be weighed against the advantages of the best current diagnostic and therapeutic methods. In any medical study, every patient including those of a control group, if any should be assured of the best-proven diagnostic and therapeutic methods.

The refusal of the patient to participate in a study must never interfere with the doctor-patient relationship. If the doctor considers it essential not to obtain informed consent, the specific reasons for this proposal should be stated in the experimental protocol for transmission to the independent committee. The doctor can combine medical research with professional care, the objective being the acquisition of new medical knowledge only to the extent that medical research is justified by its potential diagnostic or therapeutic value for the patient.

Non-therapeutic biomedical research involving human subjects (Non-clinical biomedical research)

In the purely scientific application of medical research carried out on a human being, it is the duty of the doctor to remain protector of the life and health of that person on whom biomedical research is being carried out. The subjects should be volunteers, either healthy persons or patients for whom the experimental design is not related to the patient's illness. The investigator and the

investigating team should discontinue the research if in his/her or their judgment it may, if continued be harmful to the individual. In research on man, the interest of science and society should never take precedence over consideration related to the well being of the subjects

Salient features of Helsinki Declaration

Extensive animal research is an absolute prerequisite to the use of an innovative technique in the treatment of human beings. The most difficult innovative cases will be those in which prior animal research is impossible or would be uninformative. The treatment should be given cautiously and its after effects noted and appreciated.

Experiment on volunteers can only be justified if they do no significant harm to the subject and the results are likely to be beneficial. It would be unethical to do something merely by way of experimentation that is which is not strictly related to cure patient's illness. There must also be no great risk in proposed experimentation even if the patient consents to run the great risk. In considering whether a new treatment is as efficacious as an old one, side effects of the two treatments and their costs should be considered.

ICMR Guidelines for Human Research 1995

The ICMR has laid down certain ethical guidelines for biomedical research on human subjects.

It states that medical and related research using human beings should necessarily ensure that: The **purpose** of such research is directed towards the increase of knowledge about human conditions in relation to its social and natural environment. The research is **conducted** under such conditions that the subjects are dealt with in a manner conducive to and consistent with their dignity and well being. The research must be subjected to a regime of **evaluation** at all stages.

General Principles

Principle of Essentiality

Whereby the research is considered absolutely

essential after a due consideration of all factors and options involved.

Principle of Voluntariness, Informed Consent and Community Agreement

Whereby, the subjects are fully apprised of the research and the impact and risks on him and others; and he retains the right to abstain from further participation in the research irrespective of any obligation they may have entered, subject to only minimal restitutive obligations of any advance consideration received.

Principle of Non Exploitation

Whereby as a general rule the research subjects are remunerated for their involvement in the research or experiment.

Principle of Privacy and Confidentiality

Whereby, the identity and records of the subjects are kept confidential as far as possible.

Principle of Precaution and Risk Minimization

Whereby, due care and caution is taken at all stages of a research to ensure that the subjects are put to minimum risk and generally benefit from the experiment.

Principle of Professional Competence

Whereby, the research is conducted at all times by competent and qualified persons who act with total integrity and impartiality and who have been made aware of the ethical considerations.

Principle of Accountability and Transparency

Whereby, the research experiment will be conducted in a fair, honest, impartial and transparent manner after full disclosure of all aspects of their interest.

Principle of Maximization of Public Interest and of Distributive Justice

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Whereby, the research or experiment and its subsequent applicative use are conducted and used to benefit all human kind.

Principle of Institutional Arrangement

Whereby, there shall be a duty on all persons connected with the research to ensure that all the procedures required to be complied with and all institutional arrangements required to be made in respect of the research and its subsequent use or application are duly made in a transparent manner.

Principle of Public Domain

Whereby, the research is brought into public domain so that its results are generally made known through scientific and other publications.

Principle of Totality of Responsibility

Whereby, the professional and moral responsibility, for the due observance of all the principles, guidelines and prescriptions rests with all those involved in conducting the experiment.

Principle of Compliance

Whereby, there is a general and positive duty on all persons, conducting with any research entailing the use of a human subject to ensure that these guidelines are adhered in letter and spirit.

Long Question

1. What are the functions Indian Medical Council?
2. What are duties of a Registered Medical Practitioners?
3. What is infamous conduct in professional respect? When is warning notice issued to registered Medical Pratitioner?
4. Define negligence and how will you prove it?
5. Describe the precautions one can take to prevent negligence.
6. What is consent? Described the "Doctrine

of informed consent."

7. Discussed the salient features of consumer protection Act.
8. Define consent, the various types of consent, laws in relation to consent and the general rules to be followed for taking consent.
9. Discuss the professional and ethical duties and conduct of a medical practitioner.
10. Discuss medical negligence in detail. Enumerate the various types of negligence.

Short Questions

1. Informed consent.
2. Vicarious liability.
3. Professional secrecy.
4. Privileged communication.
5. Contributory negligence.
6. Human experimentation.
7. Consent.
8. Consumer protection Act.
9. Indian Medical Council Act.
10. State Medical Councils.
11. Functions of Medical Council of India.
12. Differentiate between Civil and Criminal Negligence.

MULTIPLE CHOICE QUESTIONS

1. **In case of an unconscious patient with head injury requiring emergency surgery with no relative to give consent, the doctor should:**
 - A. Not operate at all
 - B. Operate only with consent of police
 - C. Operate only with Medical Superintendent's consent
 - D. Operate without consent
2. **Professional death sentence is given for:**
 - A. Murder
 - B. Rape
 - C. Infamous conduct
 - D. Bestiality
3. **The law does not consider the following doctrine in a charge of criminal negligence:**
 - A. Vicarious Liability
 - B. Novus-actus interveniens
 - C. Contributory negligence
 - D. Res-ipsa-loquitur
4. **Which of the following section of I.P.C. deals with criminal negligence?**
 - A. Sec.302
 - B. Sec.304
 - C. Sec.304-A
 - D. Sec. 306
5. **Paternalism means:**
 - A. Abuse of medical knowledge, so as to deprive the patient to make a choice
 - B. Absence of reasonable care and skill
 - C. To obtain informed consent
 - D. To operate without patient's consent
6. **Indian Medical Degrees Act was passed in the year**
 - A. 1916
 - B. 1920
 - C. 1925
 - D. 1930
7. **The functions of Indian Medical Council is to:**
 - A. Maintain a medical register
 - B. Maintain the standard of medical education
 - C. Recognize the foreign medical education
 - D. All of the above
8. **Warning notice is issued to a medical officer in cases of:**
 - A. Adultery
 - B. Conviction by a Court of law for offences involving moral turpitude
 - C. Dichotomy
 - D. All of the above
9. **The Indian Medical Council Act was passed in the year:**
 - A. 1932
 - B. 1956
 - C. 1960
 - D. 1972
10. **Warning notices is issued to a medical practitioner in cases of:**
 - A. Association with manufacturing firms
 - B. Addiction
 - C. Doing practice under the influence of alcohol
 - D. All of the above
11. **Prescribing an overdose of medicine is an example of:**
 - A. Doctrine of Res-ipsa-loquitur
 - B. Doctrine of common knowledge
 - C. Medical maloccurrence
 - D. None of the above
88. **A patient of head injury has no relatives and requires urgent cranial decompression, doctor should:**
 - A. Operate without formal consent
 - B. Take police consent
 - C. Wait for relatives, to take consent
 - D. Take magistrate's consent
89. **A boy attempts suicide. He is brought to a private doctor and he is successfully cured Doctor should:**
 - A. Inform police
 - B. Reporting not required
 - C. Report to magistrate
 - D. Refer to a psychiatrist
90. **Disciplinary action in professional misconduct of an anesthetist may be taken by:**
 - A. Surgeon
 - B. Medical Superintendent
 - C. Judicial Magistrate
 - D. State Medical Council
91. **Dying declaration can be recorded by:**
 - A. Medical officer

1 D 2 C 3 C 4 C 5 A 6 A 7 D 8 B 9 B 10 D 11 A

- B. Magistrate
 - C. Police officer
 - D. Any of the above
92. **'Dying declaration' is:**
- A. Always made to magistrate
 - B. Cross examination is not permitted
 - C. Has more value
 - D. The person is under oath
93. **Privileged communication is given for all of the following except:**
- A. Negligence suite
 - B. Preventing a person suffering STD from taking a job as a car driver
 - C. Benefit of society
 - D. A doctor for saving himself in court of law
94. **A doctor repeatedly advertising about his professional expertise in newspaper is doing:**
- A. Dichotomy
 - B. Civil negligence
 - C. Infamous conduct
 - D. Privileged communication
95. **All the following can be used as defense in a case of criminal negligence by a doctor except:**
- A. Therapeutic misadventure
 - B. Unforeseeable complication
 - C. Error of judgement
 - D. Contributory negligence

Moment of Death

THANATOLOGY

Thanatology is the branch of science that deals with death in all its aspects.

Death

The definition of death created little or no problem for centuries until 1960 when organ transplantation caused minute scrutiny to be directed at the phenomenon of death itself.

Section 46 I.P.C. states that death denotes the death of a human being unless the contrary appears from the context.

Section 2(b) of the Registration of Births and Deaths Act defines death as ‘permanent disappearance of all evidence of life at any time after live birth has taken place.

Black’s law dictionary (Black 1951) in United States defines death as “The cessation of life, the ceasing to exist”, defined by physicians as total stoppage of circulation and cessation of vital functions, there upon such as respiration and pulsation.

Shapiro (1969) defined death as ‘the irreversible loss of the properties of living matter. However, it is difficult to appreciate his claim that this definition satisfies the practical requirements for death certification.

Calne (1970) gave a more practical definition that states ‘when destruction of the brain has been established, the individual has died no matter what the state of the rest of his body, giving four signs for such a diagnosis:

1. Deep, irreversible coma with fixed, dilated pupils and absent cranial nerve reflexes.
2. No spontaneous respiration
3. Absence of electrical brain activity
4. Cessation of circulation through the retina.

Rentoul and Smith (1973) defined death as ‘complete and persistent cessation of respiration and circulation’

In a case of **Smith v/s Smith**: In a fatal automobile accident both husband and wife sustained injury. Husband died on the spot and the wife was taken to the hospital where she remained unconscious for 17 days and then died. The petitioner argued that the deaths were simultaneous, since there was no evidence of brain activity after accident and the physicians were unsuccessful to revive her by resuscitation. The inference was that resuscitative efforts were maintaining the body in a state of animation although it could not be shown that life existed. The Court did not accept the contention. They quoted Black’s definition of death and stated that breathing though unconsciousness is not dead.

The living body depends upon on the integrity of three principal interdependent systems circulation, respiration and enervation. Failure of one of them will cause failure of the other two. This leads to the death of the individual. There are two phases of death:

1. Extinction of the personality is immediate sign of vital process—Somatic death.
2. Progressive disintegration of the body tissue—Molecular death or cellular death that occurs sometimes later.

Somatic Death

This is the form of death referred to in common parlance. The issue of its occurrence might have considerable legal implications, but the decision when death has occurred is always a medical one. Its onset is usually not difficult to detect even by lay people if the dying person is under observation during the last minute of life. It is said to have occurred when a final expiration is followed by continuous immobility of the chest, loss of pulse, alteration of the features. These changes indicate that respiration and circulation have been stopped and the brain will stop functioning if it has not been so. Residual heart movement is not of practical importance as they are insufficient to maintain the circulation. Somatic death can be detected by ECG that will stop within minutes.

The recent development in medical techniques has posed a number of problems, where as for so many years it was accepted that death has occurred if respiration and circulation have ceased for more then 10 minutes. Now it is possible for methods of cooling the whole body to 15°C or 59°F or less to stop the heart and respiration for an hour or longer and restart them again at will. During this period, ECG and EEG shows no electrical activity and body looks like a corpse. This leads to the view that it is not the cessation

of the respiration or circulation but it is their failure to return that indicates death. Thus we are driven from a positive to negative approach. Therefore, the doctor while taking decision must satisfy himself that not only respiration and circulation has stopped, but their failure has persisted to such a period that under no circumstances it is possible for the person to come to life again.

Finally death can be defined as “Permanent and irreversible stoppage of **tripod of life**”.

The diagnosis of death by auscultation of the chest can be difficult in cases of excessive fat, emphysema, apex beat below the rib, poorly beating heart and shallow diaphragmatic respiration. All these conditions damps the conduction of heart sound with body wall.

Diagnosis of recent death is also very difficult whenever the death of the person has not been observed. Whenever a doctor is called to certify a patient as dead that is brought by ambulance, he must insist that the body must be removed to a well-lit room where he can carry out his examination. Even though the condition is satisfactory, the error can occur in the examination.

The signs of life can be detected by special methods like Oscilloscope, ECG, and EEG etc. It can never be assumed that attempted resuscitation is pointless. On the other hand, resuscitative measures should always be continued for half an hour. In cases of electric shock and drug overdose, treatment should continue until death. In a case of a young women 23 years found lying on a beach at 8:10 AM near Liverpool apparently dead, police was informed. The doctor failed to see any movement for 15 minutes and certified her dead. A pathologist came later examined and agreed with the diagnosis. About two hours later when the postmortem was to start, one of the person noticed flickering of eyelids and formation of tear. She was immediately taken to intensive care unit

where she was survived. On investigation, it was found to be a case of overdose of barbiturate.

This case emphasizes the importance of examination of the body by the doctor before certifying it. If the doctor fails to detect heart sound or respiration after adequate auscultation, he must see rectal temperature. When the rectal temperature is below 75°F (23.9°C) it is to be diagnosed as dead. On Ophthalmoscopic examination, there is segmentation of blood in retinal blood columns. If still there is any doubt, the patient should be taken to intensive care unit for further investigation of heart and brain function.

Major N.C. Kapoor reports a case of resuscitation after cessation of vital functions for 15 minutes. A Hindu 80 years old male was brought to medical college, Calcutta at 10 PM on July 13, 1925 suffering from severe dyspnoea resulting from laryngeal obstruction due to malignant growth. Tracheostomy was done immediately when he suddenly stopped breathing. No heart sounds were audible on auscultation, pupils were dilated and eyes were fixed. Artificial respiration was started and tracheostomy was performed when he was apparently dead, the chest was being flickered by wet towel, there was no pulse at the wrist. After 15 minutes when every body had given up the hope, patient's chest was flicked in a forcible manner and the patient took a shallow breath. The heart sounds were audible and pulse was palpable.

Molecular Death

The occurrence of clinical death implies the failure of the body as an integrated system. For some time afterwards, life continues in the separate tissues, which constitute the body. These only die after varying periods depending upon the ability of the tissue to function without blood supply. This is called molecular or cellular death.

The nervous tissue dies rapidly and the vital centers die in about 5 minutes. The muscle live longer and they will contract to direct electrical stimuli up to 3 hours. The corneal reflex and papillary reflex disappear at the time of death. The pupil reacts to the drugs like atropine that causes dilatation up to 4 hours and esrine that causes contraction up to 1 hour. The cornea can be removed for transplantation up to 6 hours and blood can be transfused for up to 6 hours of death. Therefore, we die in bits and pieces.

The legal definition of death depends upon the diagnosis of somatic death. The distinction between somatic and molecular death becomes important because in order to remove essential tissues and organs for transplantation there is a relatively short time for the biological properties of living matter to persist after somatic death. With somatic death, there is complete generalized anoxia of the tissue and consequently stoppage of metabolic process carried out by the tissue cells. The metabolic process of the ganglionic cells stops in minutes, which are most sensitive where as that of connective tissue stops in hours, which are the least sensitive.

Errors in Diagnosis of Death

Generally, there is no difficulty in the diagnosis of somatic deaths. However, in some cases the heart sounds and breath sounds diminish greatly. Errors occur if proper clinical examination is not done or hurriedly done in patients with kidney failure and Cheyne-stokes respiration etc. Nevertheless, there are occasions when the distinction between death and suspended animation poses a real difficulty.

Suspended Animation

This is a death like state in which vital functions of the body are at such a low pitch that they can not be determined by ordinary methods of clinical

examination. Practitioners of yoga can induce this death like state voluntarily. In addition it can be involuntarily due to (i) Severe shock following accident (ii) Electrical shock (iii) Poisoning—barbiturate (iv) Drowning (v) Epilepsy (vi) Sunstroke (vii) Cholera and (viii) Hysteria

Suspended animation lasts for seconds to hours and by ordinary clinical methods, distinction is not possible.

Importance of Moment of Death

1. The classical concept of so called tripod of life does not hold good now as lungs and heart could be maintained on artificial support system. Any attempt to determine the moment of death in such patients by the help of classical criteria results in a stalemate. Dr Christian Bernard did the first heart transplant on 3rd December, 1967 in Cape town.
2. The second major factor responsible for a fresh appraisal of moment of death is the growing practice of organ transplantation. A beating heart, which is the ideal specimen for transplantation can not be taken out of an individual because of
 - i. If we were to apply to classical criteria, because the heart is beating in the prospective donor, he is obviously not dead,
 - ii. On the contrary, if we wait for his heart to stop to resolve the ethical dilemma, the heart becomes useless for transplantation and chances of such a heart surviving in the recipient are very dim.

Therefore, the people of all over the world were trying to fine out the legal definition of death, as most of the countries had no such definition. In 1968 Bruce Tucker, a labourer fell down from a multistory building. His heart was taken out by the transplantation team while it was beating being declared brain dead on the ground of extensive Cranio-cerebral damage. His brother William

Tucker appealed in the Court in the State of Virginia, USA. The Court verdict was that doctor had not acted lawfully. The jury has directed by the Court decided on the facts of the case and opined that a person having suffered irreparable brain damage and whose bodily function can not be sustained without continuous artificial assistance is dead. Therefore, the loss of brain function was taken into account for declaring a person dead.

Before coming to the modern concept of moment of death, we consider two questions.

1. What is it that is so central to your humanity, that when you loose it you are dead?
2. Whether death is an event or process.

If we consider death to be an event then the end point of existence is very clear and sharp. Nevertheless, the 50 trillion cells of the body do not die at a time. Probably they die turn by turn. Therefore, how many cells whether 10%, 20% or 90% will die to declare an individual dead? Is there any point of no return after which no artificial means will be able to revive the person and will be called dead? Let us clarify the fact with an example:

What we call a person who has just been decapitated but whose heart is still beating. If we go by the concept of point of no return, we would pronounce him dead but if we go by old method, he is not dead. It is absurd to wait for asystole in a decapitated man who has reached the “point of no return”. Therefore, the modern concept of brain death is now taken into account.

Brain Death

Philadelphia Protocol (1969)

It determined death on the basis of:

1. Lack of responsiveness to internal and external environment
2. Absence of spontaneous breathing movements for 3 minutes, in the absence of

- hypocarbia and while breathing room air.
3. No muscular movements with generalized flaccidity and no evidence of postural activity or shivering.
 4. Reflexes and responses:
 - i. Pupils fixed, dilated, and nonreactive to strong stimuli
 - ii. Absence of corneal reflexes
 - iii. Supra-orbital or other pressure response absent (both pain response and decerebrate posturing)
 - iv. Absence of snouting and sucking responses
 - v. No reflex response to upper and lower airway stimulation
 - vi. No ocular response to ice water stimulation of inner ear.
 - vii. No superficial and deep tendon reflexes.
 - viii. No planter responses
 5. Falling arterial pressure without support by drugs or other means
 6. Iso-electric EEG (in the absence of hypothermia, anaesthetic deaths, and drug intoxication) recorded spontaneously and during auditory and tactile stimulation.

Further, it was added that these criteria shall have been present for at least 2 hours and two physicians other than the physician of a potential organ recipient should certify that death. As the protocol insists on a two hour delay, this precludes the use of any organs for transplantation.

The concept of brain death goes back as far as 1959 when two French physicians **Moll-ret and Loudon** described a condition "*Coma depasse*" that is literally a state beyond coma. Again, the adhoc committee of Harvard Medical School revived it. The committee was constituted mainly after Bruce Tucker's case and incorporates surgeons, anesthetists, forensic expert, legal personnel, philosophers etc. It was declared that it is futile to cling to the older concept, as heart and lungs could be maintained indefinitely. It was declared in fact that the brain

was synonymous with the individual, and the death of the brain should be equated with the death of the individual.

Brain was considered superior to heart and lungs because

1. Brain is the seat of consciousness and hence of the individuality of the person.
2. Brain cannot be kept on artificial support, making stoppage of its functioning irreversible.
3. Brain controls, integrates and coordinates the whole body system, making it master of the orchestra, so to say.

Then it was necessary to formulate criteria, which would indicate brain death known as Harvard Report (1968).

Harvard Criteria

1. *Unreceptivity and unresponsivity*: Total unawareness to externally applied stimuli and inner need and complete unresponsiveness to even the most intense painful stimuli.
2. *No movements*: No spontaneous muscular movements in response to stimuli such as pain, touch, sound or light for a period of at least one hour
3. *Apnoea*: Absence of spontaneous breathing for at least one hour and when patient is on ventilator, the total absence of spontaneous breathing may be established by turning off the respirator for 3 minutes and observing whether there is any effort on the part of the subject to breath spontaneously
4. *Absence of elicitable reflexes*: Irreversible coma with abolition of central nervous system activity is evidenced in part by the absence of elicitable reflexes. The pupils are fixed and dilated and does not respond to a direct source of bright light. Ocular movement and blinking are absent. There is no evidence of postural activity. Corneal and pharyngeal reflexes are also absent. Stretch tendon reflexes also

cannot be elicited.

5. *Isoelectric EEG*: Has confirmatory value. All these tests should be repeated after 24 hours with no change. Further it is stressed that the patient be declared dead before any effort is made to take him off the ventilator if he is then on a ventilator. This declaration should not be delayed until he has been taken off the respirator and all artificially stimulated signs have ceased.

By this time all over the world, it was agreed that brain death was indeed equivalent to individual death. However, they did not agree regarding the criteria fixed by Harvard school. There are three distinct schools of diagnosing death:

1. French school that is similar to Harvard
2. English school that is similar to Harvard
3. Austro-German school that includes Harvard criteria and bilateral serial angiography of internal carotid and vertebral artery criteria. A negative angiogram for more than 15 minutes proves death.

Critical Issues Involving Brainstem Death

Since the brain contains 14 billion neurons, how many neurons should be dead to declare a person dead and should one wait until the last neuron is dead? It was argued that even if one neuron is alive that shows electrical activity you cannot pronounce a person dead. Conversations told that yes, even if one neuron is alive, electrical activity could be recorded and the person should not be declared as dead.

However, the argument that remained was that how much human one remains after losing 90% of the brain's tissue. In addition, it was argued that since we do not know where the seat of consciousness lies, it is only reasonable to assume that some consciousness does remain even if a single neuron is left.

Then a new school emerged known as American school, led by Mohandas and Chou in 1971 that summarized the criteria of brain death at the University of Minnesota Health Services Center.

Minnesota Criteria (1971)

The criteria for brainstem death was formulated by **Mohan Dass and Chou** as follows:

1. Known but irreparable intracranial lesion.
2. No spontaneous movement
3. Apnea when tested for a period of 4 minutes at a time
4. Absence of brainstem reflexes:
 - i. Dilated and fixed pupil
 - ii. Absent corneal reflexes
 - iii. Absent Doll's head phenomenon
 - iv. Absent ciliospinal reflexes
 - v. Absent gag reflex
 - vi. Absent vestibular response to caloric stimulation
 - vii. Absent tonic neck reflex
5. EEG not mandatory
6. Spinal reflex not important
7. All the findings above remain unchanged for at least 12 hours

Brain stem death can be pronounced only if the pathological processes responsible for states 1 through 4 above are deemed irreparable with presently available means

The basic teaching of the American school was not the brain, but the brain stem death, after which there is the point of no return that should be equated as death. There were several reasons for this equation:

1. Medullary neurons are most resistant to anoxia, if they are dead, then higher centers are also dead.
2. Brain stem is responsible for the vital functions because the respiratory center and circulatory center are present.
3. Brain stem is necessary for proper

functioning of the cortex as all sensory and motor nerves pass through this gateway.

Immediately after an individual is put on respirator and other life support system, a systemic examination is done to exclude the possibility of brain stem death. If Minnesota criteria are fulfilled, life support systems to be withdrawn and the person is to be declared dead.

Brain Stem Death and Organ Transplantation

For centuries, there has been a dread of being declared dead while still alive. However, it is only since 1959 that there have been conceptual and practical problems with the diagnosis of death following the description of “brain death”. The ability to ventilate brain dead bodies in intensive therapy units preceded the developments of transplant surgery. The concept of brain death is not simply a convenience invented to satisfy the demands of transplant surgeons.

The diagnosis of death is traditionally made using the **triad of Bichat** that states that death is “the failure of the body as an integrated system associated with the irreversible loss of circulation; respiration and enervation”. This is also known as somatic death or clinical death. Death is now accepted as synonymous with brain stem death. The brain stem is a small area of the brain that controls respiration and circulation. If this area is dead, the person will never be able to breathe spontaneously or regain consciousness.

Molecular death may be defined as “the death of individual organs and tissues of the body consequent upon the cessation of circulation”. Different tissues die at different rates depending on their oxygen requirement. Thus, within four minutes of the blood supply to the brain ceasing the central nervous system is irreversibly damaged.

Some authorities recognize a third concept of

apparent death that is also known as a suspended animation. It may occur under certain specialised conditions for example drowning or hypothermia. Severe brain damage which does not involve the brain stem may result in a persistent vegetative state”. These patients breathe spontaneously, open and close their eyes, swallow and make facial grimaces. However, they show no behavioural evidence of awareness. It is in these cases that the moral dilemma of “allowing some one to die” arises.

The above dilemma does not apply to those who are brain stem dead. These patients are dead irrefutably and unequivocally. Switching off a ventilator under these circumstances would not kill the patient but would discontinue ventilating a corpse.

In 1976 the Conference of the Medical Royal Colleges and Faculties of the United Kingdom issued guide lines on the subject of brain stem death. These guide lines are regularly updated and used by doctors in intensive care units throughout the UK.

Diagnosis of Brain Stem Death

Exclusions

1. Where the patient may be under the effects of drugs e.g. therapeutic drugs or overdoses
2. Where the core temperature of the body is below 35°C e.g. exposure.
3. Where the patient is suffering from severe metabolic or endocrine disturbances which may lead to severe but reversible coma e.g. diabetes.

Preconditions of diagnosis

1. The patient must be deeply comatose.
2. The patient must be maintained on a ventilator.
3. The cause of the coma must be known.

Personnel who should perform the

tests

1. The brain stem death tests must be performed by two medical practitioners.
2. The doctors involved should be experts in this field. Under no circumstances are brain stem death tests performed by transplant surgeons.
3. At least one of the doctors should be of consultant status. Junior doctors are not permitted to perform these tests.
4. Each doctor should perform the tests twice.

Tests to be Performed

Before the tests are performed the core temperature of the body is taken to ensure that it is above 35°C. The diagnosis of brain stem death is established by testing the function of the cranial nerves which pass through the brain stem. If there is no response to these tests the brain stem is considered to be irreversibly dead.

1. The pupils are fixed in diameter and do not respond to changes in the intensity of light.
2. There is no corneal reflex.
3. The vestibulo-ocular reflexes are absent, i.e. no eye movement occurs after the installation of cold water into the outer ears.
4. No motor responses within the cranial nerve distribution can be elicited by painful-or other sensory stimuli, that is the patient does not grimace in response to a painful stimulus applied to or to the limbs.
5. There is no gag reflex to bronchial stimulation by a suction catheter passed down the trachea.
6. No respiratory movements occur when the patient is disconnected from the ventilator for long enough to ensure that the carbon dioxide concentration in the blood rises above the threshold for stimulating respiration i.e. after giving the patient 100% oxygen for 5 minutes the ventilator is disconnected for up to 10 minutes. If no spontaneous breathing of any sort occurs within that 10 minutes the brain

stem is incapable of reacting to the presence of the carbon dioxide and is thus dead.

Once two doctors have performed these tests twice with negative results, the patient is pronounced dead and a death certificate can be issued. It is at this stage that a decision concerning the use of organs for transplantation purposes may be raised and the decision made as to the whether the corpse should be maintained on the ventilator until the organs may be harvested.

Organ Transplantation

Advances in medicine have enabled the use of several organs for transplant purposes. It is now possible to harvest from a cadaver the eyes, kidneys, liver, pancreas, small intestine, lungs and heart. From the living donor it is possible to transplant one kidney.

The cornea of the eye may still be suitable for transplant up to 24 hours after death, other organs such as the heart, are very sensitive to low oxygen levels and deteriorate very quickly following the cessation of circulation. It is this, which has led to the controversy regarding the ventilation of corpses prior to harvesting of organs.

To prevent premature declaration of death by the physician eager to obtain organs for his patients, due precautions have been incorporated in The UK Human Tissue Act 1961 and in India, the Anatomy Act 1984 and The Human Organ Transplantation Act 1994 that covers transplantation of human organs.

The UK Human Tissue Act 1961

It states that If a person, either in writing at any time or orally in the presence of two or more witnesses during his lifetime has expressed a request that his body or any specified part of his body might be used after his death for therapeutic

purposes or for purposes of medical education or research, the person lawfully in possession of his body after his death may unless he has reason to believe that the request was subsequently withdrawn, authorize the removal from the body of any part or, as the case may be, the specified part; for use in accordance with the request. British Department of Health has issued cards for donors to carry, indicating their wish to donate organs, if they died.

This Act also provides for obtaining permission for postmortem examination when no medicolegal investigation is being carried out. The person who is legally in charge of the body such as hospital authorities may give permission for autopsy if they are satisfied after reasonable enquiry that there are no surviving close relatives and that the deceased had not objected to such an examination during his lifetime.

Memorandum of British transplantation society

British transplantation society has issued a memorandum that states that Human Tissues Act should be amended to clarify the legal aspects. The society has proposed 'Codes of Practice' for organ transplantation with salient features as:

1. Two doctors who are not members of the transplantation team should do the certification of the death of the donor and of the two one should be qualified for at least five years.
2. As far as decision of switching of the respirator is concerned, it should entirely base on the decision of the treating physicians. This decision should have no connection with the requirements for transplantation.
3. When it is decided to switch off the ventilator, the willingness for organ donation should be discussed with the relatives
4. The surgeon of the transplantation team should ensure that the death has already occurred and the deceased had requested for the

donation during his lifetime and that the relatives now has no objection in removal of the organs for the purpose of transplantation.

5. In situations where relatives object to the decision of the deceased for removal of his organs for transplantation, their decision should be respected.
6. In a medicolegal case, the coroner's permission should be obtained before removing the organs and the surgeon should report any matter of medicolegal significance to the authorities.

The Transplantation of Human Organs Act 1994

This Act came into force on February 4, 1995 and gave a legal recognition to brainstem death.

1. The Act defines brainstem death as a stage at which all the functions of the brainstem have permanently and irreversibly ceased but organ can sustain life on life support systems. The salient features of the Act are
2. Section 3 (6) of the Act provides that the organs from the body of a donor can only be removed after the Board of Doctors certify regarding the brainstem death of the deceased. According to Section 3(6): 'Where any human organ is to be removed from the body of a person in the event of his brainstem death, no such removal shall be undertaken unless such death is certified, in such form and in such manner and on satisfaction of such conditions and requirements as may be prescribed by the Board of medical experts consisting of the following:
 - i. The registered medical practitioner in charge of the hospital in which brainstem death has occurred
 - ii. An independent registered medical

- practitioner, being a specialist, to be nominated by the registered medical practitioner specified in clause (i), from the panel of names approved by the appropriate authority.
- iii. A neurologist or a neurosurgeon to be nominated by the registered medical practitioner specified in clause (i), from the panel of names approved by the appropriate authority, and
 - iv. The registered medical practitioner treating the person whose brainstem death has occurred
3. Section 3 (1) of the Act provides that any donor can authorize for the removal of his organs and utilisation only for therapeutic purposes.
 4. Section 3(2): For donation of organs, the donor at the time of his death should request in writing for removal of organs from his body for transplantation in the presence of two or more witnesses, one of whom should be a near relative
 5. Section 3 (5) the human organs is to be removed from the body of the deceased person when the brainstem death has occurred and has been certified under sub Section 3(6)
 6. The donor who is at least 18 years of age can authorize such removal and parental consent is must in case of a minor.
 7. When any unclaimed or unknown body is lying in the hospital for more than 48 hours, the hospital-in-charge can authorize for such removal.
 8. When a postmortem examination is mandatory either for medicolegal or pathological purposes, the doctor conducting such an examination can authorize for the removal of organs after satisfying himself that the organs to be removed are not required for such examination. The authorization for removal of organs is subject to no objection from the deceased before his death.
 9. The human organs removed from the body of the donor shall only be used for transplantation into the body of his near relative.
 10. For transplantation of organs removed from the body of a donor into the recipient other than the near relative, prior approval of authorization committee should be obtained.
 11. Registration is mandatory for the hospitals that are engaged in removal, storage and transplantation of human organs. In case, they violate any of the conditions specified in this Act, their registration is liable to be cancelled after proper inquiry.
 12. Any person or hospital who are associated in the removal of any human organ without authority shall be liable to punishment with imprisonment up to five years and also fine up to Rs 10,000/-.
 13. If a registered Medical practitioner is convicted for violating the conditions of this Act, his name would be removed from the medical register for a period of two years for first offence and permanently for subsequent offences.
 14. The Act also provide punishment for commercial dealing in human organs that is imprisonment for at least two years but may extend to seven years and fine not less than Rs 10,000/- extending to Rs 20,000/-.

MEDICAL CERTIFICATION OF DEATH

The Registration of Births and Deaths Act, 1970 makes it obligatory for doctors to issue a death certificate certifying the underlying cause of death to the patient whom they have attended during his final illness after examination of the body. In our country, the form of certificate

required by statistical authorities for this purpose based on the nomenclature agreed to by WHO in 1950 that has been in use in U.K. since 1927. According to this medical certification of cause of death is divided into two main parts

Part I: Disease or condition directly leading to death

Part II: Other significant conditions contributing to the death but not related to the disease or condition causing it.

Part 1 is categorized into three subsections (a), (b) and (c) that are interrelated to each other and have a direct chain of causation between each other. That (a) must be due to or be a consequence of (b) which is due to or a consequence of (c)

Part 2 is any condition that is not related to part 1 but which is a significant factor in causation of death.

In this certificate, the doctor should specify in part 1 the underlying pathological condition that led to the death. No clinical manifestations or mode of death should be included in part 1. Part 2 of the form should preferably be left blank unless there is any contributory factor in the death.

The terms such as syncope, heart failure or cardiac failure should not be used on part 1 of the certificate. Most commonly, the physicians use the term cardio respiratory arrest that should not be used as everyone dies of stoppage of circulation and respiration. It is important to specify the underlying pathological cause of the cardiac or respiratory arrest. Similarly the term 'coma' is quite unacceptable as it a clinical symptom and not underlying pathological entity. Also the term 'asphyxia' should be avoided especially when it is not qualified by the underlying cause of death.

Some of the older textbooks give account of the various modes of death but these are irrelevant in legal medicine. These are:

Bishop's Classification of Modes of Death

Death occurs due to the stoppage of the functions of any of the three main organs (Bishop's tripod of life) namely heart, brain and lungs. Depending on the functions of these organs, the mode of death comprises of

1. *Syncope*: Failure of functions of heart with its circulatory system
2. *Asphyxia*: It comprises of failure of the respiratory system
3. *Coma*: Failure of nervous system signifies mode of death as coma

Bishop laid emphasis on the mode of death or the three proximate causes of death. This classification is useful to the law enforcing agencies and nonprofessionals for interpretation of medical evidence as to cause of death.

EUTHANASIA

The word euthanasia is derived from the Greek word 'Eu' meaning good and 'thanatos' meaning death. It means '**good death**' or to '**die well**', with the basic theme of 'mercy killing'.

Euthanasia: is the intentional killing by act or omission of a dependent human being for his or her alleged benefit. (The key word here is "intentional". If death is not intended, it is not an act of euthanasia)

It may be defined as the doctrine or theory that in certain circumstances when owing to disease, senility or the like, a person's life has permanently ceased to be either agreeable or useful, the sufferer should be painlessly killed either by himself or by another. In the last decade, the use of advanced directives or **living wills** has become increasingly common. However, with these also problems can arise. All over the world and especially in North American continent, there is increasing support for euthanasia and its

consequences. The medical profession finds itself caught in a conflict between the Hippocratic Oath and the question “whose life is it anyway?”

Euthanasia evokes the strong feelings in every one including doctors. It can be classified based on the acts of induction as active and passive and based on the will of the patient as voluntary, involuntary and non-voluntary euthanasia

Active Euthanasia (positive euthanasia)

It is a positive merciful act, to end useless suffering or a meaningless existence. It is an act of commission that is when a doctor takes steps to end patient’s life e.g., giving large doses of drugs to hasten death.

Passive Euthanasia (negative euthanasia)

It means discontinuing or not using extraordinary life sustaining measures to prolong life. It includes acts of omission such as failure to resuscitate terminally ill or hopelessly incapacitated patient or a severely defective infant. It is not using measures that would probably delay death and permits natural death to occur.

Voluntary euthanasia

When the person who is killed has requested to be killed with his consent

Non-voluntary

When the person who is killed made no request to be killed such as in persons incapable of making their wishes like persons in irreversible coma.

Involuntary Euthanasia

When the person who is killed made an expressed wish to the contrary.

Assisted Suicide

Someone provides an individual with the information, guidance, and means to take his or her own life with the intention that they will be used for this purpose. When it is a doctor, who helps another person to kill himself or herself it is called “physician assisted suicide.”

Euthanasia by Action: It is intentionally causing a person’s death by performing an action such as by giving a lethal injection.

Euthanasia by Omission: It is intentionally causing death by not providing necessary and ordinary (usual and customary) care or food and water.

What is not Euthanasia: There is no euthanasia unless the death is intentionally caused by what was done or not done. Thus, some medical actions that are often labeled “passive euthanasia” are no form of euthanasia, since the intention to take life is lacking. These acts include not commencing treatment that would not provide a benefit to the patient, withdrawing treatment that has been shown to be ineffective, too burdensome or is unwanted, and the giving of high doses of painkillers that may endanger life, when they have been shown to be necessary. All those are part of good medical practice, endorsed by law, when they are properly carried out.

Views on Euthanasia

There are both protagonist and antagonist views on euthanasia:

1. *Protagonist views:* It advocated euthanasia on the ground that the patient, if willing should be allowed to die a dignified death instead of prolonging the same through the torture of pain and disease. The protagonist plead that it is a tremendous economic and psychological burden on the patient’s relatives to support an incurable patient and have often expressed death wishes for such a patient. They also

argue that medical sciences too have its limitations and it cannot cure all the diseases.

2. Antagonists claim that Medical science has made such a rapid progress and its capabilities are so great that the disease, which seems incurable today, will become curable tomorrow. They remind the doctor of the code of medical ethics and Hippocratic Oath by which the doctor swears to preserve the life and prevent further damage. They remind that euthanasia is against religion and that the crimes against society would increase and would amount to legalizing murder and suicide. If legalized euthanasia could be misused by doctors coming with hand in hand with relatives for material gains or otherwise. It may also encourage people to commit suicides.

History of Euthanasia

1. About 400 B.C, The Hippocratic Oath was given as "I will give no deadly medicine to any one if asked, nor suggest any such counsel"
2. Swift, in his early 19th century treatise on the laws of Connecticut, stated, "If one counsels another to commit suicide, and the other by reason of the advice kills himself, the advisor is guilty of murder as principal." This was the well-established common law view, as was the similar principle that the consent of a homicide victim is wholly immaterial to the guilt of the person who caused his death. The right to life and to personal security is not only sacred in the estimation of the common law, but it is inalienable. Moreover, the prohibitions against assisting suicide never contained exceptions for those who were near death. Rather, the life of those to whom life had become a burden, of those who were hopelessly diseased or fatally wounded and
- even the lives of criminals condemned to death, were under the protection of law, equally as the lives of those who were in the full tide of life's enjoyment, and anxious to continue to live. Even the prisoner who persuaded another to commit suicide could be tried for murder, even though victim was scheduled shortly to be executed.
3. The earliest American statute explicitly to outlaw assisting suicide was enacted in New York in 1828, and many of the new States and Territories followed New York's example.
4. Between 1857 and 1865, a New York commission led by Dudley Field drafted a criminal code that prohibited aiding a suicide and, specifically, furnishing another person with any deadly weapon or poisonous drug, knowing that such person intends to use such weapon or drug in taking his own life.
5. In 1920, the book "Permitting the Destruction of Life not Worthy of Life" was published. In this book, authors Alfred Hoche, M.D., a professor of psychiatry at the University of Freiburg, and Karl Binding, a professor of law from the University of Leipzig, argued that patients who ask for "death assistance" should, under very carefully controlled conditions, be able to obtain it from a physician. This book helped support involuntary euthanasia by Nazi Germany.
6. In 1935 the Euthanasia Society of England was formed to promote euthanasia.
7. In October of 1939 amid the turmoil of the outbreak of war Hitler ordered widespread "mercy killing" of the sick and disabled. The Code named "Aktion T 4," was the Nazi euthanasia program to eliminate "life unworthy of life" and it at first focused on newborns and very young children.

- Midwives and doctors were required to register children up to age three who showed symptoms of mental retardation, physical deformity, or other symptoms included on a questionnaire from the Reich Health Ministry.”
8. The Nazi euthanasia program quickly expanded to include older disabled children and adults. Hitler’s decree of October, 1939, typed on his personal stationery and back dated to September 1, enlarged ‘the authority of certain physicians to be designated by name in such manner that persons who, according to human judgment, are incurable can, upon a most careful diagnosis of their condition of sickness, be accorded a mercy death.’”
 9. In 1973 in America, the Patient Bill of Rights was created that includes informed consent and the right to refuse treatment. In Netherlands, Dr Gertrida Postma received life sentence for giving her dying mother a lethal injection
 10. In 1980, Pope John Paul II issued Declaration in euthanasia opposing mercy killing but permitted the use of painkillers to ease pain and the right to refuse life sustaining means. In California, the Hemlock Society founded by Derek Humphery advocated legal change and also distributed how to die information. Also the campaign for assisted dying was launched in America. Right to die societies was also formed in Germany and Canada.
 11. In 1984, the Netherlands Supreme Court approved voluntary euthanasia under certain conditions.
 12. In 1990, in America, the Medical Association adopted the position that with informed consent, physician can withhold or withdraw treatment from a patient near death. The Hemlock of Oregon introduced the Death with Dignity Act.
 13. In 1995 Australia’s Northern Territory approved a euthanasia bill
 14. The euthanasia bill went into effect in 1996 and was overturned by the Australian Parliament in 1997.
 15. In 1997, the U. S. Supreme Court ruling in the Washington v. Glucksberg was that “More specifically, for over 700 years, the Anglo American common law tradition has punished or otherwise disapproved of both suicide and assisting suicide.”
 16. In 1998 the U.S. State of Oregon legalized assisted suicide. In Switzerland the firm Dignitas was established to offer physician -aided -suicide.
 17. In 1999 Dr. Jack Kevorkian sentenced to a 10-25 year prison term for giving a lethal injection to Thomas Youk whose death was shown on the “60 Minutes” television program in America.
 18. In the year 2000 the Netherlands legalized euthanasia.
 19. In 2002 Belgium legalized euthanasia.

Legal Scenario Around the World

Laws in Australia

World’s first law allowing voluntary euthanasia had come in to effect in Australia on July 1, 1996 under the Northern Territory Rights of the Terminally Ill Act. Northern Territory legislative assembly of Australia passed this Act on the morning of May 24, 1995 by a narrow margin of fifteen votes to ten. The first patient on whom this law was used was former carpenter Bob Dent 66 years old male who was suffering from cancer. In this case, a computer-operated machine was used to administer a lethal dose of drug on September 22, 1996. The bill remained until March 1997 and was overturned by the Australian Parliament. Seven patients have made

formal use of this bill and four died. All these seven patients had cancer and most were in advanced stages with symptoms of depression. The features of this bill were:

1. The patient should be of sound mind, terminally ill and at least 18 years old.
2. The patient must experience unacceptable pain, suffering or distress.
3. Four doctors must be involved in the patient's decision to die
4. The attending physician must believe that patient has come to his decision voluntarily and he must inform the patient about his illness, prognosis and treatment available.
5. The second physician must confirm the diagnosis made by the first physician and the presence of psychiatrist is must.
6. There must be a seven-day cooling off period between the doctor signing off the decision for euthanasia and another two days before euthanasia procedure is performed.
7. A computerized system known as the 'Deliverance programme' enables a fatal injection to be administered by a computer and the doctor stands on one side.

Laws and Practices in Netherlands

In the Netherlands, the word euthanasia has a more limited meaning; it only refers to the deliberate termination of the life of a person on his request by another person that is active voluntary euthanasia. Due to this Netherlands has attracted so much attention and raised so much controversy. According to Dutch Penal code, euthanasia is a crime, however it is not classified as murder that is euthanasia is practically permissible but not legal. Royal Dutch Medical Association after discussion had made certain guidelines to be followed. These are:

1. The request for euthanasia must come from the patient and be entirely free and voluntary,

well considered and patient.

2. The patient must experience intolerant physical and mental suffering, with no prospect of improvement and with no acceptable solutions to alleviate patient's situation.
3. A physician must perform euthanasia after consultation with an independent colleague who has experience in this field.
4. In 1990, a new procedure was agreed upon, according to which a physician informs the local medical examiner by means of an extensive questionnaire, then reports to the district attorney. When he is satisfied that the criteria laid down by the Courts are complied with, the public prosecutor will issue a certificate of no objection to burial or cremation. In other cases, he may order an investigation and decide to prosecute the case.

The medical care of demented patients in the last phase of life is primarily the duty of the physician. Nevertheless, in the year 2000, Netherlands legalized euthanasia

Laws in United Kingdom

The criminal law in U.K. regards as a potential offence of homicide any wrongful act which results in the loss of life. When life is taken deliberately, the person is charged with murder. If a doctor responds to a request from a patient to end his life and administers a lethal injection, the doctor will have acted with the necessary mens rea for murder. It makes no difference from the legal point of view that the patient gave his consent to the doctor's act. Consent is no defence to a charge of murder nor does the doctor's motive make any difference. The fact that there was a case of mercy killing does not affect the status of the act as one of murder. The criminal law does not require doctors to persist in the treatment of a patient when no medical purpose is served by such persistence. All that is required

from legal point of view is that the patient be given appropriate medical treatment.

Scenario in USA

In the year 1994, right to die judgment was given in U.S.A when a New York Court struck down the state law prohibiting doctors for helping dying patients commit suicide. In 1997, the U.S. Supreme Court ruling in the *Washington v. Glucksberg*, opinion written by Chief Justice Rehnquist was 'That suicide remained a grievous, though no felonious, wrong is confirmed by the fact that colonial and early state legislatures and courts did not retreat from prohibiting assisting suicide.

The United States' assisted suicide bans have in recent years been reexamined and, generally, reaffirmed. It seems that because of advances in medicine and technology, the Americans today are increasingly likely to die in institutions, from chronic illnesses. Public concern and democratic action are therefore sharply focused on how best to protect dignity and independence at the end of life, with the result that there have been many significant changes in state laws and in the attitudes, these laws reflect. Many States, for example, now permit "living wills," surrogate health care decision making, and the withdrawal or refusal of life sustaining medical treatment. At the same time, however, voters and legislators continue for the most part to reaffirm their States' prohibitions on assisting suicide

Legal Status in India

India has not come up with any legislation on euthanasia.

Article 21 of the constitution of India has right to live as a natural right embodied in it. Recent Supreme Court judgment has clarified further on that. It says that article 21 guarantees 'right to live' but doesn't in any way imply 'right to die'

and hence extinction of natural life is illegal. As per this verdict suicide, assisted suicide or euthanasia are not permissible.

However, Sec 92 of the Indian Penal Code prevents euthanasia. Sec 92 IPC provides immunity to doctors for any act done in good faith for the benefit of a person without his consent. But under the first and second sub clause of this section, which states that this exception doesn't extend to the intentional causing of death, or attempting to cause death or for an act which is know to cause death. In effect it prohibits euthanasia.

Long Questions

1. Discuss the medicolegal significance of thanatology.
2. Discuss in detail the moment of death with special reference to organ transplantation.
3. Define euthanasia. What are the various types? Discuss the legal status in India.

Short Question

1. Brain death.
2. Brain stem death.
3. Suspended animation.
4. Harvard criteria.
5. The Human Organ Transplants Act 1989.
6. Euthanasia.

MULTIPLE CHOICE QUESTIONS

1. **The first medicolegal autopsy was done in:**
 A. Germany B. Italy
 C. India D. U.K.
2. **The minimum amount of air required for fatal systemic air embolism is**
 A. 50 ml B. 100 ml
 C. 150 ml D. 200 ml
3. **Substance commonly used for preservation of viscera for toxicological purpose:**
 A. 10% formalin
 B. Saturated solution of common salt
 C. 40% formalin
 D. Alcohol
4. **'Triad of Bichat' does not include:**
 A. Respiration
 B. Circulation
 C. Innervation
 D. Reflexes
5. **Harvard criteria doesn't include:**
 A. Unreceptivity and unresponsivity
 B. No movement for 3 hours
 C. Apnoea on 3 minutes off respirator
 D. Iso electric EEG
6. **The Minnesota criteria for brain death were formulated in:**
 A. 1960 B. 1964
 C. 1971 D. 1976
7. **Not a feature of brain death:**
 A. Complete apnea
 B. Absent Pupillary reflex
 C. Absence deep tendon reflex
 D. Heart rate un-responsive to atropine
8. **Suspended animation may be seen with:**
 A. Electrocution
 B. Strangulation / hanging
 C. Drowning
 D. Bum
9. **Surest sign of death due to ante-mortem hanging is:**
 A. Cyanosis of face and lips
 B. Ligature mark around the neck
 C. Protruding tongue
 D. Trickling of saliva, down the angle of the mouth
10. **Molecular death is:**
 A. Complete and irreversible cessation of brain, heart and lungs function
 B. Death of individual tissues and cells after somatic death
 C. Vitals functions are at such a low pitch that can not be detected by routine clinical examination
 D. Total loss of EEG activity but heart is functioning
11. **All are tests for stoppage of circulation except:**
 A. Magnus test
 B. Winslow's test
 C. Icard's test
 D. Diaphanous test
12. **Suspended animation:**
 A. Is apparent death from which person can be aroused
 B. Is true death from which person cannot be aroused
 C. May last for days or weeks
 D. Cannot be produced voluntarily

Identification

Identification means to determine the individuality of a person. It is done by recognizing the physical characteristics. When the person is known by his name with complete address, it is known as **complete identification**. But in certain circumstances only other details like age, sex etc. can be established known as **partial identification**.

The question of identification in living persons arises in criminal cases like assault, murder, rape etc. and even in new born cases when babies get interchanged. In civil cases like marriage, inheritance, insurance and disputed sex etc. identification plays an important role. In the dead especially unknown medicolegal cases, police plays an important role in the matter of identification. He has to investigate and advertise the details in the news media. He has to photograph and also take the fingerprints. He has to find out the relatives who identify the person before bringing the case to the doctors for medicolegal post-mortem. Sometimes, the identification becomes very difficult if the body is destroyed or mutilated in fires, explosions and other mass disasters.

CORPUS DELICTI

Corpus delicti refers to the body of the criminal offence, which is an essential part of the crime. It not only includes the body of the victim but also the weapon of offence, clothing, drawings and photographs of the deceased. The main purpose of corpus delicti is to establish the identity of the person. However, the Court has passed death penalty even though the body has not been recovered and identification not being established.

The following points are taken into consideration for the purpose of identification in a dead person: (i) Age (ii) Sex (iii) Religion (iv) Complexion (v) General development including stature (vi) Anthropometric measurements (vii) Fingerprints and footprints (viii) Superimposition (ix) Teeth (x) DNA fingerprinting and (xi) Personal belongings.

In cases of living person in addition to the above features the following criteria are taken into consideration: (i) Speech and voice (ii) Handwriting (iii) Gait (iv) Habits (v) Memory and education.

While establishing identification, it is advisable to use maximum number of criteria.

There are methods available by which to establish positive undeniable identification that are:

- Fingerprints, footprints, handprints and lip prints.
- Dental patterns.
- Frontal sinus pattern.
- Skull suture patterns and vascular grooves.
- Normal and abnormal bone comparisons.
- Other fortuitous comparisons.

AGE

To establish the identity of an individual, estimation of age is to be done that depends on:

- a. Physical or morphological features.
- b. Laboratory tests.
- c. Dental study.
- d. Ossification activity and growth of bones.

Physical or Morphological Features

Physical feature include height and weight of the person. Starting from the intrauterine life up to

certain periods of extra-uterine life, height and weight of the individual has some relationship with the age.

Determination of Intrauterine Age

Intrauterine age of the foetus can be calculated using the Hasse's rule and Morrison's rule.

Rule of Hasse (1895): According to this rule the age of the foetus is calculated by the square root of the crown heel length in centimeters. For example if the crown heel length is 16 cm, the age of the foetus is about 4 weeks intra uterine life.

Rule of Morrison (1964): On the other hand the rule of Morrison (1964) says that the crown heel length of the foetus in centimeters, divided by five gives the age in months. For e.g. if the length is 40 cm, the age is 8 months. It is applicable after 5 months.

Intrauterine age is that period starting from time of conception up till delivery. The various terms used for the products of conception are:

1. *Ovum*: From fertilization till second week of gestation.
2. *Embryo*: From second week of gestation till third month of intrauterine life.
3. *Fetus*: From third month of gestation till full term.

The age of the fetus can be determined by some of the features as given below:

1. *End of 1st month (4th week)*: At the end of first month the length is 1 cm and the body is attached to chorion by a short chord weight is about 5 gms. 2 dark spots indicate the eyes and mouth is seen as a cleft. The beginning of formation of limbs is by four bud like processes. Fetus of this age cannot be detected in aborted material.
2. *End of 2nd month (8th week)*: the length is about 4-5 cm and is of the size of hen's egg. Weight is about 10 grams. At this age the human appearance can be demarcated from the limbs, mouth and nose. Feet and hands are webbed. Anus appears as a dark spot. Placenta starts forming with a longer

umbilical cord. Sex is indistinct.

3. *End of 3rd month*: the length is about 8 – 10 cm and weight about 30 gms. The head is separated from body by neck formation and there is formation of palate. Placenta is fully developed. Heart is 4 chambered. Alimentary canal is situated in the abdominal cavity. Sex bud appears. Limbs, finger and toes are well developed but nails remain as membranes. The centers of ossification for ischium, 1st, 2nd, 3rd sacral vertebrae are present.
4. *End of 4th month*: length is 15 to 16 cm. weight is 100–120 gms. Sex is distinct skin is covered with fine downy lanugo hair. Ear ossicles are ossified. Centre of ossification for lower segments of sacrum appears. Skull bones are partly ossified. Brain convolutions begin to develop. Gall bladder is formed and meconium is found in duodenum.
5. *End of 5th month*: length is about 25 cms and weight 450 gms. Light hair appear over the scalp and downy lanugo hair cover whole of the body. The nails are distinct and soft. Skin is covered with vernix caseosa. Meconium is present at the beginning of large intestine. Centre of ossification appears for calcaneum and ischium. The germs of permanent teeth are present in jaw.
6. *End of 6th month*: length is about 30 cm and weight is 800gms to 1 kg. Skin is red and wrinkled. Eyelids are adherent but eyebrows and eyelashes begin to form. Gall bladder contains bile and meconium is present in upper part of large intestine which starts showing sacculations. Testicles lie close to kidney. Centre of ossification for lower 4 divisions of sternum are present. Cerebral hemispheres cover the cerebellum.
7. *End of seventh month (28th week)*: Length is about 35 cm and weight is 900-1200 gms. Eyelids are separate and eyelashes can be distinguished. The skin is dusky red in colour covered with vernix caseosa. Nails are distinct but do not reach the finger tips.

Testicles descend in internal inguinal ring. Caecum is in right iliac fossa and meconium is in entire large intestine. Centre of ossification for talus is present. Cerebral convolutions are present.

8. *End of eighth month (32 weeks)*: Length is about 40 cm and weight is 1.5-2 kg. Scalp hair is thick and skin is rosy red in colour and covered with soft lanugo hair except for the face. Nails reach tips of fingers, but not tips of toes. Testicles reach external inguinal rings left testicle may descend in scrotum. Meconium reaches the rectum. Cerebral convolutions are well developed. Centre of ossification for last sacral vertebra appears.
9. *End of ninth month (36 weeks)*: Length is about 45 cm and weight is about 2.5 kg. The scalp is covered with thick dark hair. Downy hairs disappear on the body except on the shoulders. Vernix caseosa may be found in the joint flexures. Scrotum shows wrinkling and contains both the testicles. Meconium is found at the end of rectum. Centre of ossification of lower end of femur appears. Nails grow beyond tips of fingers but only tip of toes.

10. *End of tenth month (40th week)*: Length is about 50-53 cm and crown rump length is 30-33 cms. Weight is about 2.5-4 kgs. Head circumference is 33 cms and scalp is covered with 3.5 cm long hair. Anterior fontanelle is 4 × 2.5 cm wide. Posterior fontanelle gets closed. Face is not wrinkled and lanugo hair is absent except on the shoulders. Cartilages are formed in nose and ear. Umbilicus is situated between pubis and xiphisternum. Scrotum contains testicles the vulva is closed and labia majora cover the labia minora and clitoris. Rectum contains dark greenish or black meconium. Centre of ossification is present for upper end of tibia and cuboid (Table 6.1).

Determination of Extrauterine Age

A full term foetus is 19-20 inches (50cm) in length and 6-7 pounds by weight. At six months of age, it is 24 inches (60cm) and by one year of life it is 27 inches (68cm) in length. At the age of four years, the child becomes 40 inches (100cm) in length. Weight has little relation with the age of the person. The weight of the infant increases one pound every month until five months of age when it doubles and trebles by a year.

Table 6.1: Intrauterine age of fetus

Age (Months)	Length (cms)	Weight	Features
1	1	2.5 gms	Eyes— two dark spots Mouth as a cleft
2	4	10 gms	Anus seen as dark spots; Placenta forms
3	9	30 gms	Pupillary membrane appears; Nails appear
4	16	120 gms	Sex can be differentiated; Meconium in duodenum
5	25	400 gms	Light hairs appear; Meconium beginning of large intestine.
6	30	700 gms	Eyelashes and eyebrows appear; Testes close to kidneys.
7	35	0.9-1.2 kg	Nails thick; Pupillary membrane—disappears; Eyelids—open; Meconium—whole of large intestine; Testis at external inguinal ring. Placenta 350-400 gms; Gallbladder—bile; Caecum—Rt iliac fossa
8	40	1.5-2 kg	Nails—tips of fingers; Left testis—scrotum
9	45	2.5-3 kg	Posterior fontanelle—closed; Scalp hair—4 cm long; Both testis—scrotum.
10	50	2.5-5 kg	Scalp hair 3-5 cm long, dark; male 100 gms more than female. Meconium—rectum; Ossification centers—lower end of femur, upper end of tibia, cuboid.

Development of Secondary Sexual Characteristics

1. **Growth of hair:** The appearance and growth of hair in males and females vary with the attainment of maturity.

Appearance/graying of hair	Males	Females
Pubic hair	14-15 years	13-14 years
Axillary hair	15-16 years	14-15 years
Beard and moustache	16-17 years	Absent
Other body parts	17-20 years	Absent
Graying of scalp hair	40 years	40 years
Graying of pubic hair	55 years	55 years
Balding	45 years	Less common

Hair start developing in the following stages in males and females over genitalia (after Tanner) (Table 6.2).

2. **Development of breasts in girls:** There are different stages of growth of breast by 12 to 20 years depending on the nutrition and sexual practices at an early age. The various stages of development are:
 - Breast and papillae are elevated as a small mound and there is enlargement of areolar diameter at about 11 to 12 years.
 - There is more elevation of breast and papillae and enlargement of areolar diameter at about 13 years.
 - Areola and papillae project over the level of breast at about 14 years.
 - Only papillae project over the level of breast at about 15 years.
 - Only papillae projects over the surface and areola merges with the breast at about 15 to 16 years.
3. Hoarseness (deepening) of voice occurs in males by 15-17 years.

4. Adam's apple is more prominent in males by 16-18 years.
5. Stature starts decreasing after 25 years of age at the rate of 1mm per year.
6. The Arcus senilis is the circular opacity at the limbus of cornea develops by 40 years.
7. Females achieve menopause by 40-45 years.
8. The cataract never occurs in the eyes before 55 years except when the precipitating factors cause it early.
9. The creased or wrinkled skin develops after 55 years.
10. Partial deafness could develop after 65 years of age.

Laboratory Tests

1. Absence of nucleated RBC in the peripheral blood occurs by 24 hours after birth.
2. Fall of reticulocyte count (2%) in the peripheral blood by 10 days after birth.
3. Replacement of foetal hemoglobin by adult hemoglobin is completed by 6 months to two years of age.

Dental Study

The approximate age of the individual can be determined from the examination of teeth in both the jaws. After birth and during developing years, the close estimation of age can be determined by the presence of deciduous dentition and its stages of eruption, the period of mixed dentition, stages of eruption of permanent teeth and loss of deciduous teeth. There are twenty temporary teeth and thirty-two permanent teeth in human jaw. The deciduous and permanent teeth erupt earlier in the lower jaw except the lateral incisors that erupt

Table 6.2: Tanner's stages of growth of pubic hair

Stages	Growth of hair	Age
Stage 1	Prepubertal no pubic hair, fine brown vellus hair	Less than 12 years
Stage 2	Sparse not extending on to mons pubis, light pigmented at the base of penis	12-13 years
Stage 3	Darker, coarse extending on to mons pubis, pigmented and start curling and spreading	13-14 years
Stage 4	Covering most parts but not going up to thighs	14-15 years
Stage 5	Dense hair extending to the inner thighs and dark mature pubic hair	More than 15 years

earlier in the upper jaw. The teeth erupt about a year earlier in the females.

The permanent molar erupts first in the lower and on the left side and then on the right side. The eruption of tooth depends on the hereditary, environmental, nutritional and endocrinal factors. There are 32 permanent teeth comprising of four incisors, two canines, four premolars and six molars in each of the jaw. The temporary teeth are twenty in number comprising of four incisors, two canines, and four molars in each jaw. At 6-7 year of life, first permanent molars erupt behind the second temporary molars. The premolars replace the temporary molars. The amount of wearing in the premolars and molars gives some indication of age after complete dentition. Root formation occur after completion of the crown and as the root becomes longer, the crown erupts through the bone and finally comes out of the jaws. The tooth calcifies from crown to neck to roots. When permanent tooth erupts, the overlying root of its deciduous predecessor undergoes resorption until only the crown remains that falls off. By assessing the stage of eruption of teeth and extent of completion of calcification of root fairly accurate estimate of age of the individual can be made. In old age, either the tooth falls off decayed or the crown is worn away to the sockets.

Dental Anatomy (Fig 6.1)

The development of tooth begins with the formation of cellular tooth germ within the alveolar bone in the shape of the crown. The tooth has a crown,

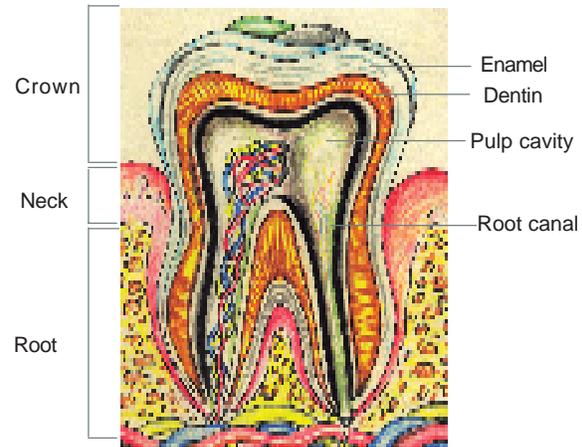


Figure 6.1: Different parts of tooth

neck and root that are embedded in the jaw. There certain differences in the appearance of deciduous and permanent teeth (Table 6.3).

Incisors—The incisors have chisel shaped crown that is convex on labial surface and concave on lingual surface, with a constricted neck and has single root. They have chewing surface and cutting edge.

Canines—The canines are larger compared to the incisors, the crown is conical shaped that is concave on its labial surface but slightly concave on its lingual surface. The masticatory edges tapers and projects beyond the level of other teeth. It has a single root. They have single cusps.

Premolars—The premolars are also known as bicuspids. They are smaller and shorter than the

Table 6.3: Differences between primary and secondary dentition

Features	Temporary teeth	Permanent teeth
Size and weight	Smaller and lighter	Larger and heavier
Colour	China white	Ivory white
Incisors	Vertical	Projected forwards
Neck	More constricted	Less constricted
Presence of ridge	Between neck and body	No ridge
Root of molars	Smaller and more divergent	Longer and less divergent
Replacement by	Permanent teeth	Not replaced
Total number	Twenty	Thirty two
Types	No premolars, 8 molars	8 premolars and 12 molars

canines and the crown is circular. The chewing surface is bicuspid with a groove between them. They have a single root but may have double root in some of the races.

Molars—The molars are the largest of the entire tooth, the crown is cubical that is convex on its lingual and labial surface. In upper jaw, first molar has stable configuration with 4 cusps, second molar is very different individually with 3 cusps and third molar is less mineralized than others and may have chalky spots. Also, upper molar has three roots.

In lower jaw, first molar has got 5 cusps, 3 buccally and 2 lingually whereas second and third molars have 4 cusps each.

Determination of age from dentition In Children

At birth, the rudiments of all temporary teeth and first permanent molars are there. In children and young persons the chronological calcification and eruption of teeth gives their ages.

The age of appearance of the permanent and secondary dentition is as give in the Table 6.4 and Figure 6.2.

Wisdom tooth: The third molar is also known as the wisdom tooth. During the age of 14-20 years, the stage of development of third molar is of particular importance to determine the age. The lateral oblique X-ray of maxilla and mandible will show the development of third molar and root

completion of second molar. The body of jaw grows posteriorly and the ramus is elongated after eruption of second molar and a space is created for third molar.

Mixed dentition: Mixed dentition is the period when both the temporary and permanent are present in the jaw. It is the period starting from 6th year of life when the first molar erupts and till the canine falls that is upto 11 years.

Successional teeth: These are ten in each jaw. The permanent teeth replaces the temporary ones except the permanent premolars that erupt in place of deciduous molars.

Superadded teeth: These do not have deciduous predecessors and erupt behind the temporary teeth. All permanent molars are superadded as the first permanent molar erupts while other deciduous teeth are present.

There are a particular number of teeth in different ages in children that is:

1. At the age of 5 years—There are 20 teeth, all are temporary
2. At the age of 6 years—There is mixed dentition and the number of teeth is 21-24 due to eruption of first permanent molar.
3. At the age of 7-11 years—There are 24 teeth (mixed dentition) with eruption of other permanent teeth replacing the temporary ones.
4. At the age of 12-14 years—The number of teeth is 24-28 due to eruption of second molar.

Table 6.4: Age of appearance of deciduous and permanent teeth

Type of teeth	Age of eruption of temporary teeth	Age of appearance of permanent teeth
Lower central incisor	5-6 months	7-8 year
Upper central incisor	6-7 months	7-8 year
Upper lateral incisor	7-8 months	8-9 year
Lower lateral incisor	8-9 months	8-9 year
Canines	1½ years	11-12 year
First premolar	Absent	9-10 year
Second premolar	Absent	10-11 year
First molar	1 year	6-7 year
Second molar	20-30 months	12-14 year
Third molar	Absent	17-25 year

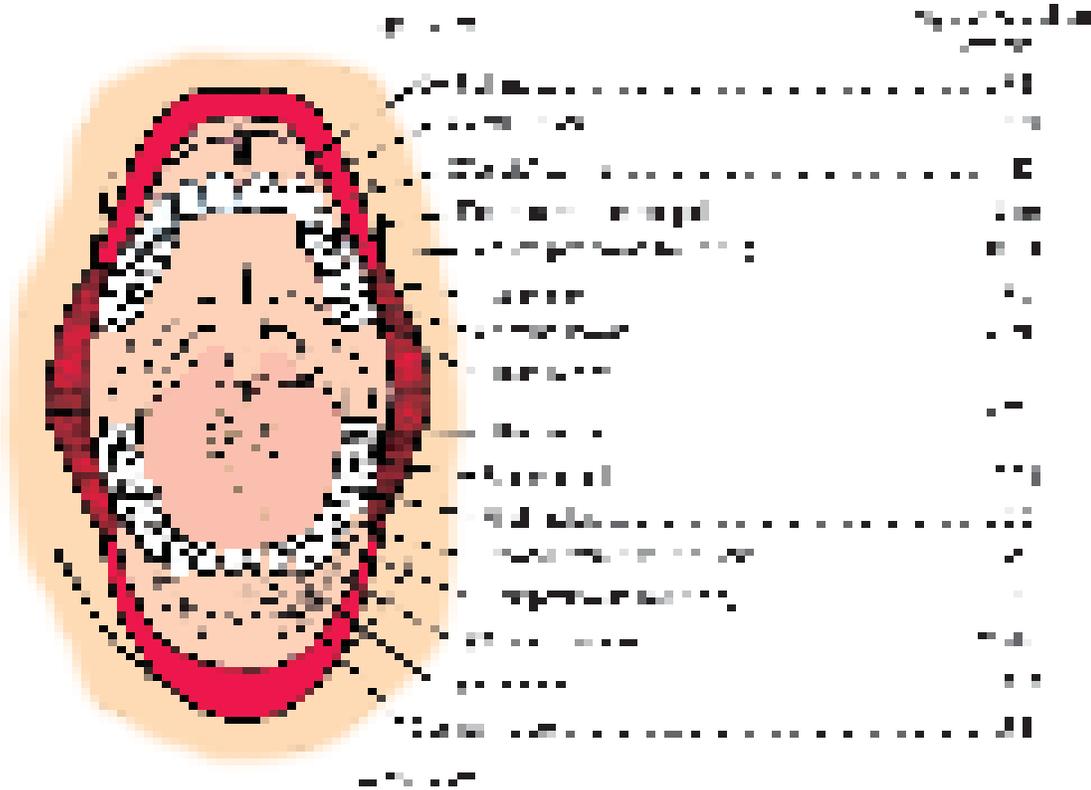


Figure 6.2: Age of eruption of permanent teeth

5. At the age of 14-17 years—The number of teeth is 28 as there is no eruption.
6. At the age of 17-25 years—The number of teeth is 32 due to eruption of last permanent molar or wisdom tooth.
7. At the age of 25 years and above—The number of teeth is thirty-two.

Spacing of the jaw. When the second molar erupts, the ramus of the mandible grows behind and the body of the jaw also increases in length for the eruption of third molar teeth. The eruption of third molar is quite irregular as it can erupt within 17-25 years. The space for third molar should be examined for its hardness, if present whenever dental examination is being carried out. It is to be noted whether the space is half formed or fully

formed by inserting the finger beyond the second molar.

Boyde's formula: Boyde in 1963 suggested a method for determining the age up to a few months after birth from structural features of the enamel. The so-called **cross striations** of the enamel prism are thought to be daily increments of growth. The numbers of cross striations formed from birth (when a particularly prominent incremental line, the neonatal line is formed) until death is counted.

Determination of Age from Dentition in Adults and Older Age

Age changes in mandible: In the mandible the center of ossification of jaw appears at second

Table 6.5: Age changes in mandible

Features	Infancy	Adult	Old age
Angle formed by ramus with body	Obtuse	Right angle	Obtuse
Body	Shallow and small	Thin and elongated	Shallow and big
Ramus	Short and oblique	Stunted	Long and oblique
Condylar process	At lower level than coronoid process	Above the level of coronoid process	Neck of Condylar process is bent backwards
Mental foramen	Placed near to the lower border of ramus and mandible	Midway between upper and lower border	Near the alveolar margin

month of intrauterine life and symphysis menti fuses by 18-20 years. The changes in mandible with age are (Table 6.5 and Fig. 6.3):

When eruption has ended and the person has achieved adult dentition, it is necessary to apply other methods that occur due to the normal wear and tear and age of the person.

Gustafson's method: This method is used for estimation of age in the adults by studying the progressive changes in an individual tooth. Pathological conditions can alter each of the factors separately (Fig. 6.4).

- **Attrition:** It is wearing and tearing on the opposing mastication surface of the teeth of the upper and lower gums due to continuous friction. The four degrees of attrition are:
A0—No attrition

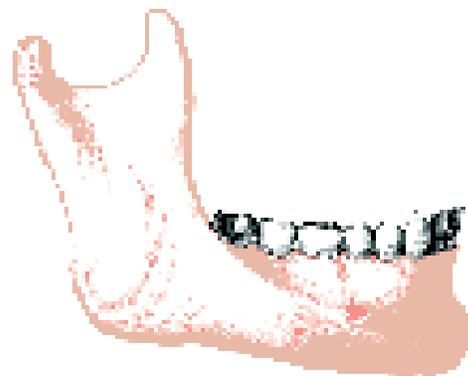


Figure 6.3: Adult mandible

- A1—Attrition within enamel
- A2—Attrition within dentine
- A3—Attrition exposes to soft pulp

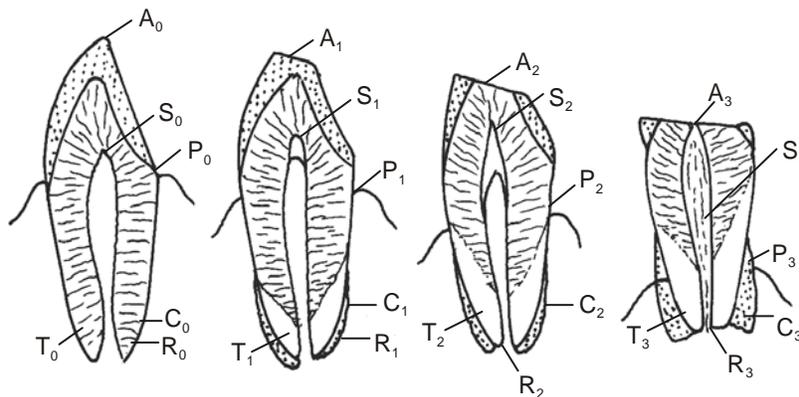


Figure 6.4: Gustafson's method

- Periodontosis:** When the maintenance of teeth and gums is bad, there is regression of gums and periodontal tissue. There is loosening of the teeth, exposure of length of root and deposition of stony hard debris over a long period. According to the length of exposure of root, the periodontosis is of following degrees

P0—No Periodontosis or no root exposure
 P1—Exposure of less than 1/3rd of the part of the root next to crown
 P2—Exposure of more than 1/3rd of the of the root but less than 2/3rd.
 P3—Periodontosis beyond 2/3rd of the length of the root near the crown.
- Secondary dentine:** With advanced age there is deposition of secondary dentine tissue in pulp cavity. The process progress until whole of pulp cavity is replenished and the size of cavity decreases. In mandibular teeth, it starts from above and in maxillary from below. It is due to ageing, pathological conditions, dental caries etc. There are four degrees of secondary dentine formation:

S0—No deposition
 S1—Deposition in the upper part
 S2—Deposition in the half of the pulp cavity from above
 S3—Almost whole of the pulp cavity is involved.
- Root resorption:** It is another decaying change involving both cementum and dentine that shows grooves. First apex is involved then extends upwards.

R0—No root resorption.
 R1—Seen only in some parts.
 R2—Seen over a large area.
 R3—Involves both cementum and dentine.
- Cementum apposition:** Cementum deposition increases due to change in the surface of tooth near the end of roof and forms incremental lines. It occurs on the surface of the root in four degrees:

C0—Only normal layer of cementum is noticed.
 C1—Slightly greater than normal.
 C2—Thick layer has occurred.
 C3—When a heavy layer is present.
- Root transparency:** It occurs in the root from below upwards in the lower jaw and above downwards in the upper jaw due to rarefaction of dentine tissue:

R0—No transparency noticed anywhere
 R1—Transparency noticeable mostly over apical region
 R2—Transparency up to 1/3rd from the apical region
 R3—Transparency noticeable up to 2/3rd of the length of the root from apex.

Out of the six criteria, transparency of the root is the most reliable one for age estimation. The anterior are most suitable as they erupt early and the changes are present earlier than others. This method is applicable only in dead bodies.

The tooth is first examined by naked eye and then sectioned. Before the teeth is extracted, the degree of periodontosis is noted. The tooth is then ground down on a glass slab to about 1 mm size and transparency is noted. Then it is grounded to 1/4 mm for microscopical examination.

The preceding factors are assessed and given a point value according to the degree of departure from the normal. The average is ascertained from an erupted tooth, which has not exhibited any of these factors. When the total points have been allotted for particular teeth, then these values are compared with the points obtained from a series of teeth of accurately known age. This comparison is carried out on a graph by drawing regression lines showing the relation of the point values of teeth of known age. The estimated age is usually found within an error of ± 3.5 years.

Point formula as given by Gustafson is $A_n + P_n + R_n + C_n + T_n + S_n = \text{Total points}$ indicates the Age in years.

Estimation of age from transparency of root: In 1963, Miles stated that transparency of root is a good marker for age and for this purpose anterior tooth are mostly used. The tooth is grounded up to thickness of 1mm and placed on a dotted paper and outline of tooth is made, then the dots visible through transparent root are counted. All dots lying in the area of tooth are counted and the percentage of the two is used to know the age.

Estimation of Age from the Bones

Skeletal age can be determined from the ossification of bones that is helpful in the determination of age. The human bones develop from a number of ossification centers. At 11-12th week of intra-uterine life there are 806 ossification centers that at birth are reduced to about 450. Adult human is made up of 206 bones. The time of appearance of center of ossification and the process of union of the epiphysis with the diaphysis at the metaphysis have a sequence and time that is utilized towards determination of age (Tables 6.6 and 6.7).

Ossification centers are studied up to the age of 20-22 years, thereafter the skull vault sutures, union and activity of sternum; changes in shape of mandible, changes in pubic symphysis are taken in to account.

The ossification begins centrally in an epiphysis that spreads peripherally and gradually takes up the osteological details of the bony part, it is going to constitute e.g., upper end of femur, lower end of radius and ulna etc. the union of epiphysis with diaphysis in long bones is interpreted as united, recently united, uniting and non-united, depending on the stage of union. When the epiphyal lines persist, it will be called as recently united. The anatomical evidence of bony union differs from radiological union by about three years and radiographs give an earlier time of union. In the living subjects while determining the age of subjects to help the Courts of law in cases of disputes relating to age, the radiological examination of the bones is to be undertaken (Figs 6.5

to 6.13). While determining the age of a subject for medico-legal purposes, the standard value obtained from examination of the number of subjects should be the guiding criteria, as individual variations for bony union tend to occur. The degree of growth and development of bones to some extent is affected by the dietary, environmental, hereditary and endocrinal factors in different individuals belonging to different regions and culture. Ossification occurs earlier in tropical climates compared to temperate zones. In females, epiphyseal union occurs 1-2 years earlier than males.

Maturation score for estimation of skeletal age: To minimize the errors of epiphyseal union, Mckern and Stewart in 1957 suggested a scheme of scoring involving seven combinations of various segments. The total score is applied to prediction equation for more accurate age estimation. The scoring is given based on the degree of epiphyseal union that is:

Degree of union	Scoring
No union	1
1/4th union	2
1/2union	3
3/4th union	4
Complete union	5



Figure 6.5: X-ray of shoulder joint showing non union of head and appearance and union of coracoid process (Age = 14-18 years)



Figure 6.6: X-ray of elbow joint showing appearance of medial epicondyle, capitulum, trochlea, heads of radius and ulna and non union of medial epicondyle, conjoint epiphysis and non appearance of lateral epicondyle (Age = 9-11 years)



Figure 6.8: X-ray of wrist joint showing appearance of carpal bones and unossified head of first metacarpal (Age = 12-17 years)



Figure 6.7: X-ray of elbow joint showing appearance of medial epicondyle, lateral epicondyle that are united and ununited heads of radius and ulna (Age = 11-14 years)



Figure 6.9: X-ray of wrist joint showing appearance of carpal bones except pisiform, unossified head of first metacarpal and lower ends of radius and ulna. (Age = < 12 years)



Figure 6.10: X-ray of pelvis showing unobliterated triradiate cartilage, non union of head and greater trochanter of femur and non appearance of lesser trochanter (Age = 11-14 years)



Figure 6.11: X-ray of pelvis showing unobliterated triradiate cartilage and non union of head, greater and lesser trochanter (Age = 14-15 years)

The various segments considered for the purpose of scoring are:

- i. Humerus—Proximal end and medial epicondyle.
- ii. Radius—Distal end.
- iii. Femur—Head and distal end.
- iv. Iliac crest.
- v. Sacrum—Lateral portion and 3,4th segments.



Figure 6.12: X-ray of knee joint showing non union of lower end of femur and upper ends of tibia and fibula (Age = < 17 years)



Figure 6.13: X-ray of sternum—lateral view showing non union of I, II and IIIrd parts of sternum (Age = 15-20 years)

Table 6.6: The important ossification centers and their fusion

Centers of bones	Appearance	Fusion
<i>Clavicle</i> —medial end	15-19 years	20-22 years
<i>Sternum</i>	5 month IUL	60-70 years
Manubrium Body (1st segment)	5 month IUL	14-25 years from below upwards; 3rd and 4th-15 years
IIrd segment	7 month IUL	2nd & 3rd-20 years
IIIrd segment	7 month IUL	1st & 2nd-25 years
IVth segment	10 month IUL	>40 years with the body
Xiphoid process	3 years	
<i>Humerus (Upper end)</i>		
• Head	1 years All three unite at 6 years	18 years
• Greater tubercle	3 years	4-5 years with head
• Lesser tubercle	5 years	5-7 years with greater tubercle
<i>Humerus (Lower end)</i>		
• Medial epicondyle	5-6 years	Capitulum, trochlea & lateral epicondyle form conjoint tendon at 14 years, unites with shaft at 15 years
• Capitulum	1 year	Med. epicondyle unites at 16 years
• Trochlea	9-10 years	
• Lateral epicondyle	10-12 (11) years	
<i>Radius</i>		
• Upper end	5-6 years	15-16 years
• Lower end	1-2 years	18-19 years
<i>Ulna</i>		
• Upper end	8-9 years	16-17 years
• Lower end	5-6 years	18-19 years
• Head 1st <i>metacarpal</i>	2 years	15-17 years
• Head <i>other metacarpals</i>	1½ to 2½ years	15-19 years
<i>Hip bone</i>		
• Triradiate cartilage	11-13 years	14-15 years
• Iliac crest	14-15 years	18-20 years
• Ischial tuberosity	15-16 years	20-22 years
• Sacrum	8 months IUL	25 years
<i>Femur (Upper end)</i>		
• Head	1 year	17-18 years
• Greater trochanter	4 years	17 years
• Lesser trochanter	14 years	15-17 years
<i>Femur (Lower end)</i>	9 month IUL	17-18 years
<i>Tibia</i>		
• Upper end	9 month IUL	16-17 years
• Lower end	1 year	16 years
<i>Scapula</i>		
• Coracoid base	10-11 year	14-15 years
• Acromion process	14-15 year	17-18 years

Table 6.7: Age of ossification of hand and foot bones

Type of bone	Age of ossification
Capitate	2 months
Hamate	3 months
Triquetral	3 months
Lunate	4 months
Scaphoid	4-5 months
Trapezium	4-5 months
Trapezoid	4-5 months
Pisiform	9-12 years
Calcaneum	5 months
Talus	7 months
Cuboid	10 months
Lateral cuneiform	1 year
Medial cuneiform	2 years
Intermediate cuneiform	3 years
Navicular	3 years

Sutural Closure of Skull

Closure of sutures of skull has some bearing with the milestone of life. Absence of any signs of closure of any sutures of the skull, points to the strong possibility of the age having not exceeded thirty years. Evidence of commencing union of the sutures is always noticed first in the endo-

cranial surface, then on the endocranial one (Figs 6.14 and 6.15). As a rule, inner surface closes several years before the outer. The sutural closure occurs earlier in males than females. Estimation of age from closure of sutures of skull can be given within the range of 10 years between 30-60 years and even more in higher age groups. The chronological ages for sutural closure are as follows:

- Lateral and occipital fontanelle closes at 2 months after birth
- Posterior fontanelle closes at 6-8 month of birth
- Anterior fontanelle closes at 1½-2 years of birth
- Metopic suture closes at 2-4 years but may extend upto six years.
- Basisocciput and basi-sphenoid fuses at 18-20 years.
- Sagittal suture is the first to start closing endocranially at about 25 years at its back portion close to parietal eminence. The fusion is complete both ectocranially and endocranially by 35-40 years.
- Coronal suture starts closing endocranially by 25-30 years in its lowest part, close to the junction with the sphenoid. It is completely closed at 40 years.

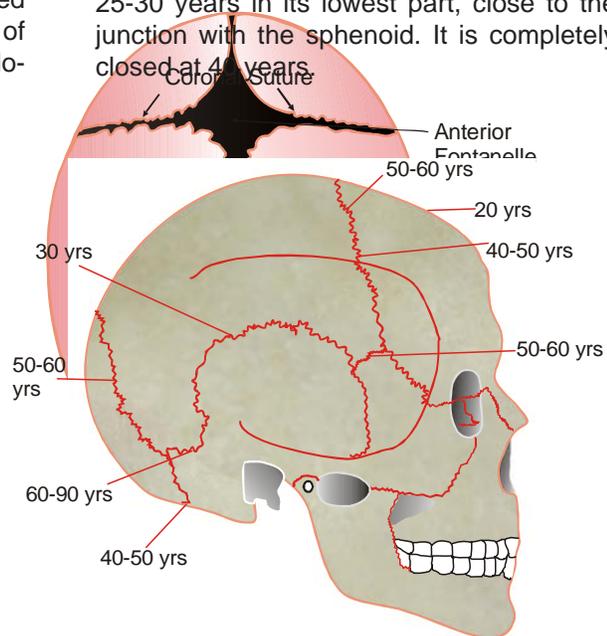


Figure 6.14: Sutures of skull

Figure 6.15: Ages of sutural closure of skull

- Lambdoid suture starts closing endocranially at 25-35 years and terminal closure occurs by 45-50 years.
- Pterion starts closing at 40 years and completely closes by 65 years.
- Mastoido-occipital starts closing at 45 years of age and complete closure occurs by 80 years. Asterion also closes by 80 years and parieto-temporal suture closes by 70 years

Age Changes in Bones in Adults and Old age

Changes in Scapula

Scapula is a delicate bone and is often too damaged to be helpful. It requires experience to interpret and also the changes do not seem to be constant. The changes are not as closely identified with age as desired. The changes in the scapula are divided in to two categories:

- Changes due to ossification after maturity:*
 - i. Lipping of glenoid fossa begins at 30-35 years
 - ii. Lipping of clavicular facet begins at 35-40 years.
 - iii. Appearance of 'plaque; or 'facet' on the underside of acromial process thus prolonging the acromial tip from 2-8 mm at 35-40 years.
 - iv. Increasing demarcation of the triangular area at the base of scapular spine begins at 45-50 years.
 - v. Appearance of crista scapularis that tend to become more broader at the base and prominent at the apices with advancing age.
- Changes due to atrophic process after maturity:*
 - i. Surface vascularity: This is seen as number of fine lines in less than 25 years old. This visibility is slowly diminished and finally disappears with advancing age.
 - ii. Deep vascularity: Deep vascularity is seen as fine lines but only by transillumination by the age of 25-30 years and they tend to diminish with advancing age.
 - iii. Atrophic spots are areas of bone atrophy that starts appearing around 45 years and usually start in infraspinous area.

- iv. 'Buckling' and 'pleating' of infraspinous area is due to irregular atrophy of bone and is the result of diminished vascularity. It begins around the age of 40 years and there is absorption of cancellous tissue.

Changes in Pubic Symphyseal Surface

Ageing is responsible for the fundamental contribution and provides a better guide than skull sutures for age estimation. Todd's system states estimation of age from 10 stages of metamorphosis. Mckern and Stewart that is a more reliable method have replaced Todd's system (Fig. 6.16). In this method symphysis pubis is divided in to three components of which, six stages (0-5) are studies of metamorphic changes. A total score of three components is obtained which in turn is translated in to age provided the individual is male.

Various changes in the three components are:

- Component 1(Dorsal half of joint surface)
 - Stage 0—Dorsal margin absent.
 - Stage 1—Slight margin formation starts in middle 3rd.
 - Stage 2—Dorsal margin extends over the entire border.
 - Stage 3—Formation of plateau (filling of grooves and resorption of ridges) begins in middle third of the dorsal surface.
 - Stage 4—Visible billowing still present and plateau extends over most of the dorsal surface.
 - Stage 5—Billowing disappears completely and the surface becomes flat and slightly granular.
- Component 2 (Ventral half of joint surface):
 - Stage 0—Ventral beveling absent.
 - Stage 1—Ventral beveling present at the superior extremity and ventral border.
 - Stage 2—Beveling extending downwards.
 - Stage 3—Bony extension (ventral rampart) begins from either or both extremities.
 - Stage 4—Ventral rampart extends still further but gaps are still visible.
 - Stage 5—Ventral rampart is complete.
- Component 3 (Combination of both-whole surface):

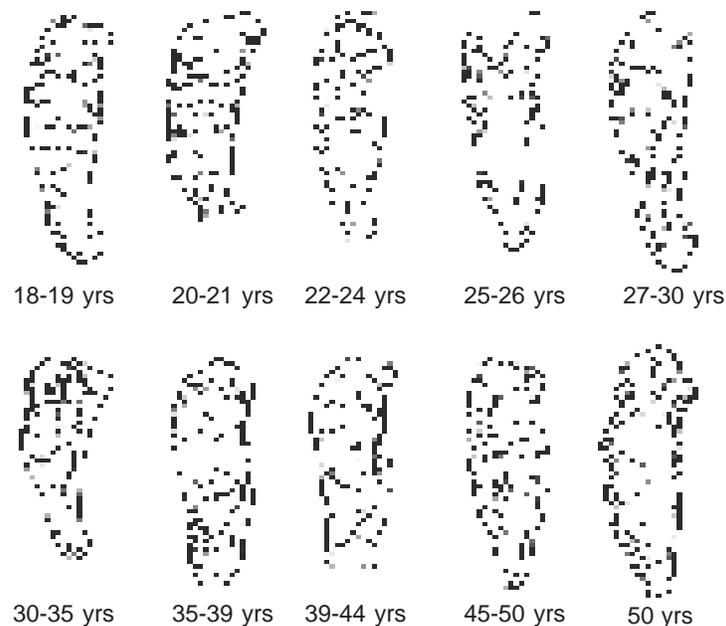


Figure 6.16: Age changes in pubic symphysis (after Todd's)

Stage 0—Symphyseal rim is absent.

Stage 1—Dorsal rim appears at superior end of the dorsal margin, it is smooth and raised above the symphyseal surface.

Stage 2—Dorsal rim is complete and ventral rim starts forming.

Stage 3—Symphyseal rim is complete and the surface is finely granular.

Stage 4—Rim begins to break down, is sharply defined. The surface becomes smooth and flat with no lipping of ventral edge.

Stage 5—There is further breakdown of rim and rarefaction of symphyseal surface.

Joint lipping begins along the vertebral edge during the last two stage of component 3 at about 35-40 years

Rough Estimate of Age from Pubic Symphysis

1. When the symphyseal surface is even, age is ≤ 20 years.

2. When the surface is markedly ridged and irregular called billowing, the ridges running transversely across the articular surface, the age is 20-25 years

3. Billowing gradually disappears and the articular surface becomes granular with sharp anterior and posterior margins by 25-35 years of age

4. Articular surface is smooth and oval by the age of 35-45 years

5. Narrow and beaded rims develop in and around the margins of articular surface and some erosion of the surface starts by 45-50 years

6. There is varying degree of erosion with breaking down of ventral margin by ≥ 50 -60 years

7. Surface becomes irregularly eroded after about 60 years of age.

Age Changes in the Vertebra

1. By the time the deciduous dentition is completed, the arch unites posteriorly and at the same time the arches and bodies of lowest lumbar vertebra begins to fuse.

2. The posterior parts begins to fuse by 6 years and at the same time the arches of the first cervical vertebra unite posteriorly.
3. The upper and lower surfaces of immature vertebra shows a series of radial furrows up to 10 years.
4. The furrows gradually fades on the upper and lower surfaces between 21-25 years.
5. Lipping of vertebra is seen usually after 45 years.
6. Osteophytic outgrowth from anterior and lateral margins become visible by 40-45 years. The second cervical vertebra up to the age of about three years consists of three pieces that is two lateral portions that constitute the arch, the body and the dens.

Age Changes in Internal Bone Structure

Two structural components are studied that is:

- *Cancellous tissue*: For studying this, humerus and femur are mainly considered and out of the two, humerus is used on priority basis. The methods used are radiological examination of bones and longitudinal section of bones in which resorption pattern of cancellous tissue is examined. The proximal end of the medullary cavity of humerus assumes a cone shape, the tip of which gradually reaches the surgical neck of the bone during the period of 40-50 years and further ascends up to epiphyseal line by 60-75 years.
- *Cortical tissue*: The cortical elements osteon, osteon fragments, lamellar bones and non haversian canals are studied microscopically and the age could be ascertained with in the margin of 5 years. By counting the osteons in cross section of bone, the age can be ascertained but it is not an accurate and reliable method.

Estimation of Age As A Whole from Birth Onwards

To know the age of the person, usually horoscopes, hospital birth certificates, school admission certificates and matriculation certificates are

being used. However, in the Courts horoscopes affidavits about age are not relied upon. In all such cases, the age of the individual is to be determined from those certain parameters are used as a whole. These are

1. *First 15 days after birth*: The changes that occur in the umbilical cord, umbilical vessels and the skin of the newborn helps in determining his age within first 15 days after birth.
2. *First 6 months after birth*: In the first 6 months after birth the age of the infant can be estimated from the following parameters:
 - Posterior fontanelle closes within 6 weeks of birth
 - Pterion and Asterion closes
 - The temporary teeth start erupting
 - Foetal hemoglobin is changed to adult type
3. *Between 6months-2 years*: The following parameters are used
 - All temporary teeth erupt.
 - Ossification centers for heads of humerus, femur, all tarsal and carpal bones appear.
 - Anterior fontanelle closes at 1½ years.
4. *Between 2-6 years*: The parameters that are undertaken for this age group are:
 - The two halves of mandible fuse by second year.
 - The metopic suture close by 2-4 years.
 - Greater and lesser tubercles of head of humerus appear at 3 and 5 years respectively.
 - Condylar part of occipital bone fuse with the squamous part and basi-occiput.
 - Medial epicondyle and upper end of radius appear by 5-6 years.
 - Head of first metacarpal appear.
 - Greater trochanter of femur appear at 4 years.
 - Center for xiphisternum appears at 3 years.
5. *Between 6-12 years*: The various findings are:
 - Ischio-pubic ramus fuses by 6-7 years.
 - Pisiform bone appear between 9-12 years.

- Coracoid process of scapula appear at 10-11 years.
 - Upper end of ulna appear by 8-9 years.
 - Trochlea appear at 9-10 years.
 - Lateral epicondyle appear between 10-12 years.
 - All temporary teeth are shed and permanent teeth erupt except third molars.
 - The secondary sexual characters appear on the body.
6. *Between 12-18 years:* The secondary sexual characters are well developed at this age. The bones around elbow joint, shoulder joint, hip joint, ankle and knee joint ossify during this period of life. The various changes are:
- Upper end of radius fuses with the shaft.
 - Olecranon fuses with the shaft.
 - Third molar teeth erupts between 17-25 years and roots of other erupted teeth calcify.
 - The head, greater and lesser trochanter of femur fuse with the shaft and lower end of femur, upper and lower end of tibia and fibula unite with their respective shafts.
 - Coracoid process fuses with scapular body at 16 years and acromion unites by 18 years.
 - The bony replacement of the cartilage between basi-occiput and basi-sphenoid commences at about 17 years.
 - The third and fourth part of sternum fuses by 15 years.
7. *Between 18-25 years:* By this age long bones epiphysis unites with their shaft
- Lower end of radius and ulna unite with their respective shafts at 18-19 years.
 - Iliac crest unites with the ilium and ischial tuberosity unites with the ischium of hip bone by 20 years
 - The sternal end of clavicle unites with the sternum by 21 years.
 - Secondary centers in articular facet of the rib is complete by 20-25 years.
 - The bony replacement of cartilage between basi-occiput and basi-sphenoid is complete by 20-25 years.
- Second and third part of sternum fuses by 20 years and first and second part unite by 25 years.
 - All parts of sacral vertebra unite with each other.
8. *Between 25-35 years:*
- The coronal, sagittal and lambdoid sutures of the skull start closing endocranially by 25-35 years and sagittal sutures closes usually by 30-35 years.
 - The age changes on symphysis pubis are characteristic after 25 years.
9. *Between 35-50 years:* The following changes are seen:
- The coronal and sagittal suture closes by 35-40 years.
 - Lambdoid suture closes by 50 years.
 - The xyphoid process unites with the body at about 40 years.
 - The age changes in symphyseal surface of pubis are seen in this age group.
 - The articular surfaces of some bones may show changes such as lipping and loss of joint space. The glenoid cavity of scapula first shows such changes whereas bodies of lumbar vertebra and inner border of ischial tuberosities usually show lipping by 40 years and other joints of the body shows lipping 45-50 years.
 - The hyoid and thyroid cartilages ossify.
10. *Above 50 years:*
- Mastoid, occipital, squamous and parieto-mastoid sutures close in very old age. Parietal suture may not close throughout the life.
 - Manubrio-sternal joint fuse by 60 years
 - Laryngeal and costal cartilages calcify
 - The long bones become lighter and brittle whereas skull bones become thinner and lighter as the diploe is absorbed. The fractures are common in old age owing to these changes.
 - Degenerative changes like formation of arcus senilis in the eyes, wrinkling of the skin, silvery white hair deposition of atheromatous plaque in the arteries is

common in old age. The chances of development of osteoporosis in the bone are more with advanced years of life.

Various Ages of Medico-legal Importance

1. Criminal responsibility

- Under S. 82 I.P.C., the child of less than 7 years is not held responsible for the crime.
- Under section 168 of the Indian Railways Act 1989, If the person under 12 years is guilty of offence under (i) S. 150 (maliciously wrecking or attempting to wreck a train—punished with death/imprisonment for life/10 years) (ii) S. 151 (damage to or destruction of certain railway property—punishment with imprisonment for 5 years or fine or both), (iii) S. 152 (maliciously hurting or attempting to hurt persons travelling by railway—punishment with imprisonment for life/10 years) (iv) S. 153 (endangering safety of persons travelling by railway by willful act or omission—punished with imprisonment for 5 years) and (v) S. 154 (endangering safety of persons travelling by railway by rash or negligent act or omission—punished with imprisonment for one year or with fine or both), the court may require father/guardian of such person to execute a bond for such amount and for such period for good conduct of the aforesaid person.
- S. 83 I.P.C.—The child of 7-12 years is presumed to be capable of committing an offence if he has attained sufficient maturity of understanding the nature and consequences of his conduct
- S. 89 I.P.C.—The child of less than 12 years cannot give valid consent to suffer any harm what may occur for an act done in good faith for its benefit
- S. 87 I.P.C.—The adult of more than 18 years can give valid consent to suffer any harm that may result from an act not intended and not known to cause death

2. **Judicial punishment**—According to the “*Juvenile*” Justice Act 2000, juvenile means a boy or a girl who is less than 18 years of age. Delinquent juvenile when commits an offence, no death sentence and no imprisonment is awarded. The juveniles are send to juvenile homes by the Court when they commit an offence.
3. **Infanticide**—If the baby is of less than 6months of intrauterine life, the charge of infanticide cannot be supported.
4. **Criminal abortion**—A woman who has passed the childbearing age cannot be charged of procuring criminal abortion.
5. **Identification**—To identify a person, the age is important.
6. **Impotence and sterility**—Women is sterile after menopause and the boy is sterile before puberty though not impotent. In India there is no legal limit on age of persons who can be declared impotent. There have even been allegations of rape against people more than 80 year old.
7. **Rape**—Sexual intercourse with his own wife below 15 years and other girl below 16 years without her consent is rape (S.375 I.P.C.).
8. **Majority**—A child is major on completion of 18 years but attains majority on completion of 21 years when under guardianship of Courts or wards. (S. 3, Indian Majority Act).
9. **Evidence (witness)**—A child of any age can give evidence if the Court is satisfied about his truthfulness (S.118 I.E.A).
10. **Employment**—A child of less than 14 years cannot be employed in a factory, mine or other hazardous employment. A person who has completed 15 years of age is allowed to work in a factory like an adult if certified by a doctor.
11. **Kidnapping**—It is taking away a person by illegal means. It is an offence to:
 - Kidnap a child with the intention of taking any movable property of a child less than 10 years (S. 369 I.P.C).
 - Kidnap a minor from lawful guardianship of less than 16 years boy and less than 18 years girl (S.361 I.P.C).

- Kidnapping or maiming a minor for purpose of bagging.
- To procure a girl for prostitution, if she is less than 18 years (S.366A I.P.C).
- To import a girl to India from a foreign country for illicit intercourse, if she is less than 21 years (S. 366 I.P.C).
- Kidnapping a minor for begging purposes.

SEX

Criteria for Determination

1. Likely indicators of sex are outline of face, figure, clothing, habits etc.
2. Highly probable indicators are external sexual characteristics, breast, buttock etc.
3. Certain or stronger evidence are penis, testis, prostate etc. in males and vagina, uterus, ovaries etc. in females.

Medicolegal Importance

The question of determination of sex arises in cases of (i) marriage (ii) divorce (iii) legitimacy (iv) impotence (v) rape and (vi) heirship. There are 46 chromosomes in a human being (male being XY and female being XX chromatin pattern).

Histological Examination

1. **Barr and Bertram in 1949 and 1950** demonstrated the differences in nucleolus of the nerve cells. They found in females a body about 1 micron in diameter known as "Barr body" that appears as a small satellite to the large nucleolus in all types of nerve cells while in the males the nuclear satellite is seldom seen. They believed that this nucleolar satellite could be the product of the X chromosome.
2. **Davidson and Smith in 1954** described the presence of drumstick appendage in the white cells of the females. He observed that six nuclear appendages were present in 300 neutrophils examined in case of females but in males no drumstick appendages were found even in 500 neutrophils examined. The former were known as **chromatin positive** and the latter as **chromatin negative**. In puterfied

bodies sex chromatin cannot be made out. The sex determination can be made from examination of specimen of buccal epithelial cells and saliva or hair follicles by the combined treatment of quinacrine dihydrochloride staining for Y-chromosome that is seen as bright fluorescent body in the nuclei of male cells and Fluorescent Feulgen reaction using Acriflavin Schiff reagent for X-chromosomes that is seen as a bright yellow spot in the nuclei. The **quinacrine positive bodies** in males range from 45-80%; 0-4% in females. With Feulgen reaction, **fluorescent bodies** are seen in 50-70% cells in females and 0-20% cells in males. The Y-chromosomes can be detected in dental pulp tissue by using fluorescent dyes for about a year. **F-bodies** can be seen in 0-4% females and 30-70% in males. In addition to these sex can also be determine from bone marrow and amniotic fluid.

3. **Biopsy:** In intersex cases having unpredictable sexual morphological features, biopsy from the primary gonads namely testicles or ovaries can determine definitely as to whether the person is sexually active male or female or neither.

CONCEALED SEX

The person dresses himself/herself similar to that of the opposite sex. The treatment to concealed sex lies in simply undressing the person.

INTERSEX

Intersex means intermingling of features of both sexes in varying degree in one individual. Though these cases are not presently in forensic practice, still one can diagnose them in life. They are intimately connected with sports, service, admission to school, medical science cases, inheritance, succession of property, of marriage, rape etc.

Types

Gonadal Agenesis

There is lack of development of gonads or secondary sexual characters and nuclear sexing is negative. The chromosomes can be demonstrated

by microscopic examination but there is no sex chromosome. In addition morphological features of either sex are not developed during puberty.

Gonadal Dysgenesis

Gonads or sex organs are present but fail to develop during puberty. There are two types of medicolegally important syndromes.

Turner's syndrome Morphology is female, but there is no proper development of feminine features during puberty. Development of primary and secondary sexual features is not observed. Amenorrhoea, sterility, high urinary gonadotropin, short stature and webbed neck etc. are seen. Ovaries do not contain sufficient follicles. Nuclear sexing is positive. Sex chromosome is XO and the Total number of chromosome is 45 instead of 46.

The cause is usually defective Polarization during cell division in the process of development of spermatozoa.

Klinefelter's syndrome In this condition, the anatomical structure is male but the nuclear sexing is female. The sex chromosomal pattern is XXY due to an extra chromosome. It is usually diagnosed when secondary sexual characters fail to develop during puberty. Testicles are small and firm and non-functional. Azoospermia with a normal/small Penis and gynaecomastia are seen. There is scanty pubic hair, moustache and beard. Nuclear sexing is positive for a female.

Sex chromosome pattern is XXY and the total is 47 instead of 46 due to non-polarization of sex during cell division. Sperms contain XY or XX chromosomes and only one of these goes to each sperm but in case of klinefelter's syndrome both the sex chromosomes remain in the same sperm and leads to the XXY pattern.

True Hermaphroditism or bisexuality The individual possesses two testicles or two ovaries or one testicle or one ovary neither of which functions fully and properly. Hence contradictory secondary sexual features are present in the individual. Nuclear sexing is either positive XX type or negative XY (male type).

Pseudo Hermaphroditism Individual has contradictory features of both the sexes. Both groups are either of a male type or female.

In male pseudo-hermaphroditism, the nuclear sex is XY but sex organs and features are that of a female due to testicle feminisation.

In female pseudo hermaphroditism, the nuclear sex is XX but sex organs and features are of a male due to adrenal hyperplasia.

Determination of Sex from Bones

General Features of Bones

The female bones are usually smaller, thinner and lighter than bones of the males in adults whose bones are bigger thicker and massive. The bones in case of males have more prominent ridges and the processes are more marked compared to females. Also in males the shaft of bones have coarser surface and articular surfaces are wider. Compared to females where the shaft of long bones are smooth and articular surfaces are smooth and round. The male skeleton weighs about 4.5 kg while its 2.5 kg in females. While determining the sex from the bones the percentage of accuracy from independent skeletal part (as given by Krogman 1964) is:

- Whole skeleton = 100%
- Pelvis and skull = 98%
- Pelvis alone = 95%
- Skull alone = 92%
- Skull and long bones = 96%
- Pelvis and long bones = 98%
- Long bones alone = 80-85%

Pelvis alone is sufficient for determination of sex especially in adults in more than 90% of cases. In case of children and fetus even from the examination of the pelvis sex can be determined with fair accuracy. Even in adolescents' greater sciatic notch is the single most important criteria from which sex can be determined. The sex differences in pelvis, skull, mandible, sacrum, clavicle, femur, humerus, sternum are given in Tables 6.8 to 6.15 and Figures 6.17 to 6.22.

Table 6.8: Differences between male and female skull

Features	Male Skull	Female Skull
General appearance	Larger, heavier, massive, and surface is rough	Lighter, smaller and surface is smooth
Capacity	More capacious (10 % > females/ 1500-1550 cc)	Less capacious (10% < males/1300-1400cc)
Architecture	Muscle ridges are more marked and prominent over occipital and temporal areas	Less marked
Frontal and parietal eminences	Small	Large
Frontal surface	Irregular	Smooth
Glabella	Well marked and prominent	Less marked and less prominent
Supraorbital ridges	More Prominent	Less Prominent
Zygomatic arches	More Prominent	Less Prominent
Mastoid processes	More Prominent	Less Prominent
Occipital protuberances	More Prominent	Less Prominent
Occipital condyles	Well marked and prominent. Facets are long are large.	Less marked and prominent. Facets are small and broad.
Digastric groove	More deep	Less deep
Fronto-nasal junction	Distinctly angulated	Smoothly curved
Cheek bones	Laterally arched, rough and large	Compressed, smooth and small
Palate	Larger and wider (U shaped) due to relative length of cheek, tooth and jaw	Smaller and narrower (parabola shaped) due to relative length of cheek, tooth and jaw
Orbits	Smaller and square placed low down with round edges	Larger and rounded placed higher up with sharp margins
Forehead	Steeper and less rounded	More Rounded and full
Nasal apertures	Narrow and placed higher up	Broad and placed lower down
Teeth	Larger, lower first molars may have five cusps	Smaller, lower first molars have four cusps



Figure 6.17: Male and female skull

Table 6.9: Differences between male and female mandible

Features	Male mandible	Female mandible
Size	Larger, thicker & heavier	Smaller, thinner & lighter
Chin	U or square & more prominent	V shaped & less prominent
Anatomical angle (between body and ramus)	Less than 125° more prominent and everted	More obtuse, less prominent and inverted
Height of symphysis menti	Greater and Higher up	Smaller and shorter
Condyles	Larger	Smaller
Muscular impressions	More prominent	Less prominent

Table 6.10: Differences between male and female pelvis

Features	Male pelvis	Female pelvis
General framework	Deep, funnel shaped. Massive and rough	Shallow, bowl shaped. Less massive and smooth
True pelvis	Narrow deep and funnel shaped	Wide and shallow
Pelvic brim	Heart shaped	Circular
Pelvic outlet	Smaller	Larger
Ilium	Curve is more prominent, more sloped with less rounded margins	Curve is less prominent, less sloped with more rounded margins
Iliac crest	More prominent	Less prominent
Anterior superior iliac spine	Not widely separated	Widely separated
Pre auricular sulcus	Not prominent, narrow and shallow	Prominent, broad and deep
Acetabulum	Wide and deep, diameter about 52 mm	Small and narrow, diameter about 46 mm
Symphysis pubis	Higher and bigger in depth and narrow in width. Margins of pubic arch are everted and no parturition pits on the dorsal border.	Lower, wider and rounded. Margins of pubic arch are not everted and parturition pits are present on the dorsal border children.
Subpubic angle	70°–75° (acute) and subpubic arch is V shaped	90° (acute) and subpubic arch is U shaped
Greater sciatic notch	Smaller, deeper and narrower and less than right angle	Wider, larger and shallower and forming a right angle
Sciatic notch index = $\frac{\text{Width of sciatic notch}}{\text{Depth of sciatic notch}} \times 100$	4-5	5-6
Obturator foramen	Large, oval shaped with base upwards	Small, triangular with the apex directed forwards
Ischial tuberosity	More or less inverted	Everted
Pubis (body)	Narrow and triangular	Broad and square
Pubis (ramus)	Is continuation of body	Narrow appearance
Pelvic index = $\frac{\text{AP diam. of pelvis}}{\text{Transverse diam. of pelvis}} \times 100$	More	Less
Kell index =	More	Less
$\frac{\text{Surface area of acetabulum}}{\text{Surface area of ilium}} \times 100$		



Figure 6.18: Male and female mandible



Figure 6.19: Male and female pelvis



Figure 6.20: Female pelvis

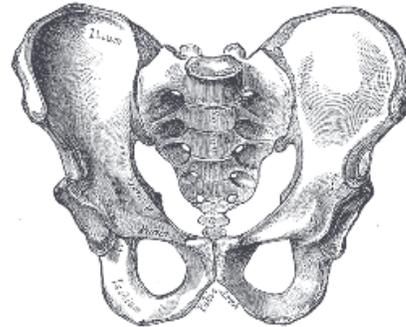


Figure 6.21: Male pelvis

Table 6.11: Differences between male and female sacrum

Features	Male sacrum	Female sacrum
General	Long and narrow, Sacrum promontory is well marked and anterior curvature is equally distributed along its length. curved in lower half.	Shorter and wider with less prominent sacral promontory. Anterior curvature is straight in upper half and sharply curved in lower half.
Sacroiliac articular surface	Large and extends upto 2.5-3 vertebral bodies	Small and extends upto 2-2.5 vertebral bodies
Coccyx	Less movable	More movable
Sacral index = $\frac{\text{Breadth of base}}{\text{Anterior length}} \times 100$	112	116
Ischio pubic index = $\frac{\text{Ischial length in mm}}{\text{Pubic length in mm}} \times 100$	73-94	91-115
Corporo basal index of sacrum = $\frac{\text{Breadth of 1st sacral vertebra}}{\text{Breadth of base of sacrum}} \times 100$	45	40.5



Figure 6.22: Male and female Sacrum

Table 6.12: Differences between male and female clavicle

Features	Male clavicle	Female clavicle
Length	> 175 mm	< 103 mm
Circumference	More	Less

Table 6.13: Differences between male and female femur

Features	Male femur	Female femur
Head	Larger and forms about 2/3rd of a sphere	Small and forms less than 2/3rd of a sphere
Neck	Forms obtuse angle (125° with the shaft)	Almost right angle with the shaft
Vertical diameter of femoral head	47-49 mm	43-45 mm
Bicondylar width	74-89 mm	67-76 mm
Trochanteric oblique length	390 mm	450 mm

Table 6.14: Differences between male and female humerus

Features	Male humerus	Female humerus
Average length	32.5 cm	30 cm
Vertical diameter of head	48.7 mm	41.5 mm
Transverse diameter	44.6 mm	38.9 mm
Feature	Male radius and ulna	Female radius and ulna
General features	Larger, thicker and heavier	Smaller, thinner and lighter
Curvature of radius	Less curved	More curved

Table 6.15: Differences between male and female sternum

Features	Male sternum	Female sternum
Body	Bigger, longer and more than twice the length of manubrium	Shorter and less than twice the length of manubrium
Level of upper border	At the level of lower part of body of second thoracic vertebrae	At the level of lower part of body of third thoracic vertebrae
Ashley's rule of 149	The combined midline length of manubrium and body = 149 mm	The combined midline length of manubrium and body = 149 mm
Manubrium	Smaller	Bigger
Sternal index =	46.2	54.3
$\frac{\text{Length of manubrium}}{\text{Length of body}} \times 100$		

STATURE

There is a progressive diminution in stature as age advances, the annual diminution being 0.06 cm. The stature diminishes during afternoon and evening by 1½-2 cm. Body is said to increase in stature after death by about 2 cm due to loss of muscle tone, relaxation of joints and tensions of intervertebral discs. To calculate stature of an individual, to the total length of entire skeleton 2½-4 cm are added for the thickness of soft parts. In calculating the stature from bones, more than one bone should be taken in to consideration and the long bones of lower limbs give a closer estimate than that of upper limbs. The highest correlation with stature is given by femur ($r = 0.8$) and tibia ($r = 0.8$) followed by humerus ($r = 0.7$) and radius ($r = 0.7$). A combination of bones is more reliable than a single bone in calculation of stature. For measuring the length of bones, sliding calipers can be used but Osteometric Board can be used that measures the length of the bone accurately. After measuring the length, to calculate the stature it is multiplied by **multiplication factors** as given by **Pan (1924)** for East Indians (Hindus). The multiplication factors are as follows: (i) humerus—5.30 (ii) radius 6.90 (iii) ulna 6.30 (iv) femur 3.70 and (v) tibia and fibula (4.48). In case of dismembered or fragmented body, the stature can be calculated as:

1. Stature is equal to the length between the tip of one middle finger to that of the opposite when the arms are fully extended.
2. The length of one arm is taken and twice this length + 30 cm for two clavicle + 4 cm for sternum is equal to the total stature.
3. The length from vertex to symphysis pubis is roughly half of stature.
4. Length from sternal notch to symphysis pubis is measured and multiplied by 3.3, which gives the stature.
5. Length of forearm from tip of olecranon process to tip of middle finger equals 5/19 of the stature.
6. The height of head from top of head to tip of

chin (as verticle length) = 1/7th of the total stature.

7. The length of vertebral column is measured that equals 35/100 of the total height
8. Maximum length of foot is taken and divided by 0.15 that gives stature.

The regression equations given by Trotter and Gleser, Dupurtuis and Hadden can also be used for calculating the stature.

RACE

Race can be determined by:

1. **Complexion:** It has limited value as complexion varies from person to person even in the same race. Skin Colour is brown in Indians, fair in European while Negros are black. It is determined by melanin granules and melanocytes. Also skin colour changes during decomposition and burns etc.
2. **Eyes:** Eyes are dark in Indians though brown eyes are also found. Europeans have blue or grey colour.
3. **Hair:** Black, thin—Indians, Light Brown—reddish Europeans. Straight or weary—Mongolians, Indians and Europeans. Frizzy or wooly (arranged in tight spirals)—Negros. On histological examination, Mongoloid hairs are coarse and dark, circular on cross-section. Dense uniform pigmentation is seen with a dark medulla. Negros have hair that are elongated and oval in cross section with irregular distribution of dense pigment. Caucasian hair are oval or cuneiform with fine or coarse pigment.
4. **Skeleton:** The race can be devised from several indices such as
 1. **Cephalic index or index of breadth:** It is calculated as maximum breadth of the skull divided by maximum length of the skull multiplied by 100. The length and breadth of skull are best measured by Sliding Calipers. In the absence of sliding calipers, the osteometric board can be used. The index varies in different races as shown in the table 6.16.

Table 6.16: Variations in cephalic index in different races

Types of skull	Cephalic index	Race
Dolichocephalic (long headed)	70-75	Aryans, Negroes
Mesati cephalic (medium headed)	75-80	Europeans, Chinese
Brachycephalic (short headed)	80-85	Mangolians

$$2. \text{ Brachial Index (Radio humeral)} = \frac{\text{Length of radius}}{\text{Length of humerus}} \times 100$$

$$3. \text{ Crural Index (Tibio-femoral)} = \frac{\text{Length of tibia}}{\text{Length of femur}} \times 100$$

$$4. \text{ Humero-femoral Index} = \frac{\text{Length of humerus}}{\text{Length of femur}} \times 100$$

SCARS

Identification from Scars

The scar develops whenever there is injury to the dermis. It is a mass of fibrous tissue that is covered by epithelium. Incised, lacerated, penetrating and crushing injuries can cause the scars. The scars can be formed in burns, scalds, corrosive acid injuries, electricity and radiation injuries. All these result in disfiguration of the part. The scars are used to identify the person. The formation of scar and time required for its formation depends on the size, site, and nature of injury, whether the wound was infected, mode of healing, by first or second intention. In an incised wound with healing by first intention the reddish linear scar forms within 10-14 days. The scar may form within 7-8 days when the wound heals with the formation of scab in superficial cuts. In a moderately infected area, the scar formation occurs in 2-3 weeks times. When the healing is by secondary intention, and when there is chronic suppuration with infection, the scar may take 4-6 weeks to form. In cases of burns, chemicals and electricity, the scar may form in several weeks to months even after grafting.

Age of Scars

To ascertain the time elapsed since injury, the assessment of the age of scar is important. The time taken for the scar to develop depends upon the nature, site and size of the injury and also on supervening infection of the wound, the vascularity of the area and the age and sex of the individual. The scars at an early stage are red and sensitive due to blood supply from small capillaries and as blood supply gets obliterated the scar becomes shrunken, depressed, irregular and smaller in size with irregular and insensitive surface. The various stages of scar formation with the passage of time since the infliction of the injury are:

Time since infliction of Injury	Characters of Scar
5-6 days	Reddish, soft, tender and sensitive scar with firm union of wound edges
End of 14 days	The scar is soft, pale and sensitive
2-6 month	Soft, brownish or coppery reddish scar with no corrugation
After 6 months	The scar is white, firm, tough, glistening and corrugated

The age of scar can be determined to be fresh, recent, old or late from the following features.

Fresh scar (Within first few weeks)—Soft and sensitive or pale yellow ecchymosis around contusion or scab formation

Recent (Few weeks-2-3months)—Brownish or copperish red, soft and not irregular

Late (About 3-6months)—Similar to the scar of 2-3 months and usually shows no corrugation

Old (More than 6months)—White, glistening, contracted and tough in consistency

Examination of Scar

While examining the scar, following points are to be noted:

1. The shape, size, direction of the scar.
2. The site of the scar.
3. Colour of the scar, whether reddish, brownish, copperish.

4. Surface whether it is irregular, thick, tough or firm in consistency and whether it is glistening or dull.
5. Whether it is tender or sensitive.
6. Whether it is freely mobile or fixed.
7. Evidence of marks of surgical interference from marks of stitches and incisions.

Disappearance and Erasing of the Scar

Superficial linear scars may disappear within a few years but scars resulting from large wounds, ulcers, skin diseases that involve whole thickness of skin are permanent. The scar if faint can be made prominent by rubbing or applying heat locally. The scar can be examined with a hand lens or under UV light. In the dead, the scar tissue can be examined microscopically by staining for reticuli and elastic tissue. The scar can be erased by excision and skin grafting. This may result in the formation of less obvious scar. If a person wants to efface the scar, he may get the part tattooed or may burn it but that in itself result in further scarring.

Medico-legal Importance of Scar

1. The scar helps in identification of an individual.
2. The age of the scar will tell us the time of infliction of injury.
3. The shape of the scar will give an idea about the nature of weapon or other causative agent. The scars from stab or punctured wounds are usually triangular and smaller than the causative weapon. In incised wounds and surgical incisions that have healed by first intention, the scar is linear and straight but if the wounds are infected, the scar is thick and wide. Scars from infected lacerated wounds, healing by second intention are irregular, thicker, broader and firmer. Scars from severe burns are prominent, irregular, thick and adherent. Vaccination scars are usually circular or oval, and slightly depressed. Scars resulting from corrosives, radiation burns are irregular and thickened.
4. Striae gravidarum, linea alba and old tears in the cervix and posterior commissure indi-

cates previous pregnancy.

5. Disfiguration caused on the face by scars resulting from assault, accident or vitriolage amounts to grievous hurt.

EXAMINATION OF HAIR (TRICOLOGY)

The hair can be examined by naked eye and hand lens as well as by simple microscopy with adequate photomicrography. Certain features like spatial configuration (curl, wave) and physical features such as softness, brittleness etc. are apparent only by naked eye and with hand lens examination. The examination of hair can provide information relating to the identification of the purpose and also it has evidentiary value.

Anatomy of Hair

Hair consists of a bulb or root and a shaft with distal termination of tip (Fig. 16.6).

During the period of growth, the hair follicle lengthens and during its resting phase, the hair follicle shrinks, the hair ceases to grow and the

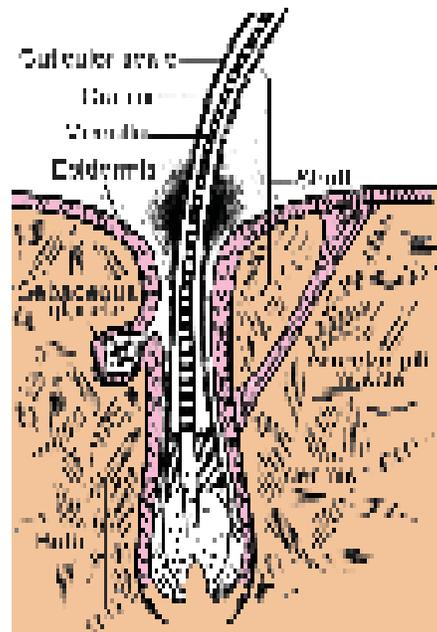


Figure 6.16: Parts of hair

hair bulb alters. This type of hair is readily shed and has shriveled, distorted bulb whereas the bulb of forcibly plucked actively growing hair is rounder and much of the follicle is pulled out of the skin with the bulb.

Hair bulb: The bulb is formed within the follicle in the skin. To pluck a lock of healthily growing hair, considerable force is required.

Hair shaft: The hair shaft is composed of keratinized mass of cortex that forms the bulk of the hair, which has an outer scaly covering or cuticle and a central core, medulla.

Cuticle: The cuticle is a layer of thin, microscopic, non-pigmented scale-like plates, each attached at its lower end to the hair cortex. The thickness of cuticle layer depends upon the thickness of individual scales and the degree of overlapping occurs. Cuticle is usually thin and sometimes thick consisting of ten to twenty layers of scales.

Cortex: The cortex forms most of the substance of a hair. It has abundant keratin that is responsible for the charring and acrid odour when the hair is burned. The cortex is responsible for the strength, the elasticity and the shape of the hair. The cortex is made up of closely set, longitudinally arranged, elongated cells with pointed ends and without nuclei. The fibrils within these cells may have granules on their surface. The cells are held together by cement. The hair when stretched is narrowed and pigment granules are separated in longitudinal direction. Continued stretching causes hair to break and untreated hair is resistant to deforming stresses. The cortex is pigmented, the degree of pigmentation varies from complete absence and sometimes quite intense. A large range of natural colors is present in natural hair of humans. Animal hair may show alternate bands of pigment. Granular pigment of hair is easily seen in hair mounted in an isorefractile medium. Transverse sections of hair enable the distribution of the granules to be determined in relation to the center and periphery of the cortex.

The medulla: Many hairs also have a central, occasionally eccentric core that is hollow called

the medulla. The hair is so darkly pigmented occasionally so that it is difficult to visualize medulla. The medullary width is quite variable along the hair shaft and is expressed in relation to the diameter of the shaft.

The tip: The tip of the hair is at its termination that is pointed and non-medullated. Repeated injuries cause the damage to the cuticle so that the exposed and unprotected cortex splits and frays.

Forensic Importance of Hair

Hair resists the destructive tendencies of adverse conditions such as exposure to gastric juice, prolonged drying, soaking and burial. The hair resists putrefaction for a long time and can be recovered from the graves many years after burial. For most forensic purposes, the durability of hair compare with most of the elastic tissues. The forensic importance of hair is to establish the identity of human remains. In addition, the hair is a source of establishing the association between a suspected person and a particular crime, or between a victim and a weapon or vehicle.

The hair can be collected and fixed for transportation and storage by touching them with a sticky surface of transparent adhesive tape, and then pressing gently this tape to the glass slide. Isolated hair must be kept and labeled separately. When a sample of hair is submitted for examination, the following questions need to be answered from medico-legal point of view.

1. *Is the sample hair or some other fiber?* Few of the fibers can be burnt to note the source. Vegetable fibers burn readily without producing a disagreeable odour and leave sharply bent ends. Animal hair burn with difficulty, with odour of burnt feathers and show curved and rounded bead like ends. Human hair gets curled, blackish, brittle, shriveled, distorted and twisted. Human and animal hair consists of a root, cuticle, cortex and the medulla. Certain chemical tests with sulphuric acid and caustic soda can help in differentiating hair from the fiber. The

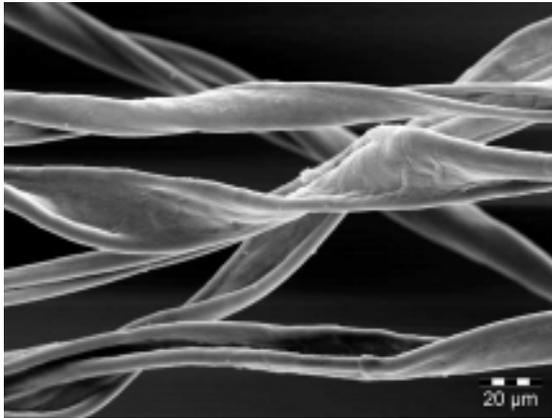


Figure 6.17: Microscopical appearance of cotton fibre

appearance of cotton fibres may be mistaken for hair that appears microscopically as shown in Figure 6.17.

2. *Whether the hair of human or animal origin?* Certain gross and microscopic findings help to differentiate the human and animal hair, are summarized in the Table 6.17. The microscopical appearance of human hair is shown in Figure 6.18.
3. *If the hair is human, part of body, where it is derived from?* The length of the hair, colour



Figure 6.18: Microscopical appearance of human hair

etc are noted to determine that which part of the body does it belong.

- Scalp hairs are long, soft and taper towards the tip. They are circular on cross-section
- Beard and moustache hair are straight, thick and triangular on cross-section
- Axillary and pubic hairs are short, wavy and curly with split ends. They are oval on cross-section

Table 6.17: Differences between human and animal hair

Features	Human hair	Animal hair
General appearance	Thin, fine and usually brown or black coloured	Thick, coarse and may be different
Cuticle and cuticular scales	Short, broad, and irregular margins are placed transversely. The pattern of cuticle is type VII of Moritz	Large with marked step like or wavy projections. The cuticle is other than type VII of Moritz
Cortex	Well striated and broad; 4-10 times broader than medulla	Narrow, rarely more than twice or as broad as medulla
Medulla	Continuous, narrow, fragmented or absent sometimes	Continuous, broader and always present
Medullary index = $\frac{\text{Diameter of the medulla}}{\text{Diameter of hair shaft}}$	Less than 0.3	More than 0.5 (3.5)
Deposition of pigment granules	Concentrated towards periphery of the cortex and is usually light in the medulla and may not be continuous	Heavy deposition in the medulla may be uniform, central or peripheral
Precipitin test	Specific to humans	Specific to animals

- Eyebrow, eyelashes and nostril hair are thick, straight, bristly, stiff and tapering. They are triangular and kidney shaped on cross-section.
 - Body hairs are soft, fine and flexible. They do not show pigment cells in cortex and the medulla is thin.
 - Downy hair of foetus or newborn lack medulla and have no pigment cells.
4. *Is the hair of male or female?* The hairs in male are thick, coarse and dark compared to that in case of females. Beard and moustache are specific to males. Upper boarder of pubic hair is classically almost horizontal in the female and rises towards the umbilicus in the males. Some physiological and pathological conditions cause gross alterations of hair growth such as hirsutism particularly in females. The **Barr bodies** can be detected in the hair bulb in about 70% of females and only about 7% of the males.
 5. *The race of the person to whom hair belongs?* The Indians have dark and fine hair; of Japanese and Chinese are dark, coarse and straight whereas those of Negroes are curly and wooly.
 6. *What is the age of the person to whom the hair belongs?* The age of the person can be deduced from the hair but within certain limits. The lanugo hairs of the newborn are fine, downy, soft and non-pigmented. These hair are non-medullated and cuticular scales have smooth and free edges. The hairs of an adult are coarser, pigmented and medullated having a complex cuticular pattern. Grey hairs are apparent after the third decade of life and are devoid of any pigment. Balding in man may begin in the second or third decade. In females, there is often loss of Axillary hair and increase of facial hair at menopause.
 7. *Has the hair been dyed, bleached or is there any evidence of disease?* The hair colour can be changed artificially by dyeing or bleaching. To bleach dark hair, hydrogen peroxide is used and to render grey hair dark natural colourants such as henna, plant juices or hair dyes are used. People working in certain industries have their hair dyed automatically. Those in copper factories may have greenish hue in the hair, miners working with cobalt exhibit bluish tint. Bleached or coloured hairs are dry, brittle, lusterless and rough. The scalp is also coloured with the hair and the colouring is not uniform. The newly growing hair will be of the original colour. The hair colour is lighter in diseases such as kwashiorkor, malnutrition, and certain vitamin deficiencies. The use of hair dyes produce local dermatitis.
 8. *Was the hair pulled or did it fall naturally?* If the hair has fallen naturally, the hair root is distorted and atrophied and the hair sheath is absent whereas when it forcibly extracted, the hair sheath is ruptured. In case of forcible extraction, the hair bulb is swollen, larger and irregular but when the hair has fallen naturally, the hair bulb has smooth, rounded end.
 9. *If there is presence of any injury to the hair and the nature of injury?* Repeated injury to the tip of the hair damages the cuticle and the exposed cortex splits and frays. Blunt force injury and cutting hair by blunt scissors result in flattening and splitting of hair shaft. The hairs cut by a sharp weapon have a clean uniform cut surface. Hair when singed are swollen, fragile, curled and twisted. They emit a peculiar odour the carbon deposits can be detected.
 10. *Is there any relation with sexual assault?* In rape and sodomy, the hair detected on the victim or undergarments of accused are important corroborative proof.

Medicolegal Importance of Hair

1. Important clue is provided when the similar hair may be detected on the alleged weapon and on the body of alleged assailant.

2. The victim's hair remains identifiable long after the commission of crime and provides valuable physical evidence.
3. In rape and sodomy, the hair detected on the body of the victim and underclothing of the suspected will provide valuable corroboration. In bestiality, the presence of animal hair in and around the genitalia, body and clothing of the accused is important evidence.
4. In road traffic accidents, presence of hair of the victim adhering to the car will provide valuable evidentiary proof.
5. The stains of mud on the hair are suggestive of road traffic accident in some muddy area or struggle in an open field. The pubic hair matted with seminal discharge may be suggestive of sexual offence. In asphyxial deaths, salivary stains are present sticking to beard and moustache. Bloodstains on the body hair suggest assault.
6. Many poisonings such as arsenic, thallium and lead can be detected from hairs.
7. In burns and close firearm injuries, hair shows singeing.
8. Age, sex, race and certain individual characters like bleaching, dyeing and waving of the hair can be deduced from examination that would help in individualization.
9. Nature of weapon whether sharp or blunt can be estimated from examination of hair.
10. Approximate time of death can be calculated from the growth of hair on the face. The growth rate is 0.4 mm per day.
11. Colour of the hair along with evidence of dirt, grease sticking to the hair may give an idea about the occupation of the person.

ANTHROPOMETRY (BERTILLON'S SYSTEM)

In 1883, **Alphonse Bertillon** a French Law enforcement officer and biometric researcher created anthropometry, an identification system based on unchanging physical measurements that was the first scientific system police used to

identify criminals. He found that several physical features and the dimensions of certain bones or bony structures in the body remain practically constant throughout adult life. He concluded that these measurements can be used to distinguish a person from the other. This system is based on fourteen identical measurements, a standardised photographic portrait (frontal and profile), the classification of facial and body characteristics, and the notation of scars and tattoos. These were recorded on cards that were filed according to a complex method, so that they could be matched and retrieved. Bertillon's system (called anthropometry, bertillonage, or signaletics) was adopted by police departments and correction systems in many jurisdictions in Europe and America

Principle

As the body measurements are not expected to change after 21-22 years (except stature). Thus this method is only applicable to adults after 21 years.

Data to be recorded

1. Descriptive data such as hair, eyes, complexion, shape of nose, ears, chin, iris colour etc.
2. Bodily marks like moles, scar, tattoo etc.
3. Bodily measurements.
 - Height.
 - Stretch: Length of body from left shoulder to right middle finger when arm is raised.
 - Bust: Length of torso from head to seat, taken when seated.
 - Length of head: Crown to forehead.
 - Width of head: Temple to temple.
 - Length of right ear.
 - Length of left foot.
 - Length of left middle finger.
 - Length of left cubit: Elbow to tip of middle finger.
 - Width of cheeks.
 - Length of left little finger.
4. Photographs of front and right profile views of the head and face are also taken.

Disadvantages

1. Photographs are not always reliable as there are chances of error when examined by the experts.
2. The measurements requires the use of instruments and trained operators.
3. The method is applicable only to adults.
4. Calculation errors can always be there.

The method has now been replaced by fingerprint system (Dactylography).

DACTYLOGRAPHY

Dactylography is also known as **Henry Galton system** of identification. It is the study of fingerprints as a method of identification. The ultimate positive identification of the dead body presented in an unidentified state is a major goal of the medico-legal expert. There are many reasons to establish effective positive identification namely.

To resolve the anxiety of the next of kin of a person missing and presumed dead. To settle the estate of the person missing and assumed dead. To establish the Corpus delicti. To allow the medicolegal official to release the body to the next of kin for burial.

Since the turn of the century, fingerprints have been used as a very effective means of establishing identity of the individual.

Introduction

The skin on the palmer and planter surface is certainly wrinkled with narrow minute ridges that are known as friction ridges and is also completely free from hair and sebaceous glands. However there is profusion of sweat glands. The dermal carvings or fingerprints appear for the first time from the 12-16 week of intrauterine life and their formation gets completed by 24th week i.e., 6th month I.U. life. The ridge thus formed during the foetal period does not change the course of alignment throughout the life of the individual until destroyed by mutilation.

Fundamental Principles of Fingerprinting

First principle: Fingerprinting is an individual characteristic; no two fingers have been observed to possess identical ridge characteristic.

Second principle: A fingerprint will remain unchanged during an individual's lifetime. Though it is not possible to change one's fingerprint, there has been no lack of effort on the part of criminals to obliterate them. If an injury reaches deeply enough in to the skin and damages dermal papillae, a permanent scar will form. For this to happen such a wound would have to penetrate 1-2mm beneath the skin's surface. Presence of such permanent scars would serve as a new and additional characteristic for the purpose of identification.

Third principle: Fingerprinting has general ridge characteristics that permit them to be systemically classified.

History of Fingerprinting

In the BC era, fingerprints were used on pottery to indicate the make and brand. Clay slabs with fingerprints nearly 3000 years old have been found in Intan khanan and tomb in Egypt. Thumbprints were used as official seals or documents by Chinese emperors since 240BC. The work of **Grew (1684) and Biloo (1685)** are among the earliest scientific descriptions of dermatoglyphics.

Purkinje's contribution in **1823** is an important landmark because he classified the varieties of finger patterns systematically for the first. He pursued a treatise commenting upon diversity of ridge pattern on the tips of fingers and distinguished a variety of patterns of fingerprints.

W.J. Herschel in 1858 began the first known official use of fingerprints in India on a large scale. He used it to prevent fraudulent collection of army pay accounts and for purposes of identity on other documents. **Henry Faulds in 1880** wrote a comment on practical use of fingerprints for identification of criminals.

Francis Galton began his observations around 1890 and published his book on fingerprints in

1892. He established the individuality and permanence of fingerprints and devised the first scientific method of classifying fingerprints.

Juan Vucetich in 1891 installed fingerprint files as an official means of criminal identification. He based his system on the patterns identified by Galton.

The first fingerprint bureau in the world was officially established in Calcutta in June 1897. In 1901, fingerprinting for criminal identification was officially introduced in England and Wales. Edward Richard Henry, IG of police in lower Bengal did extensive study on fingerprints to develop a register for classification of fingerprints. The system of **Henry and Vucetich** forms the basis of modern ten digit fingerprint identification.

Importance of Fingerprinting

1. Identification of criminals whose fingerprints are found at the scene of crime.
2. Identification of fugitive through fingerprinting comparisons.
3. Assistance to prosecutors in presenting their cases in the light of defendants' previous records.
4. Exchange of criminal identifying information with identification bureau of foreign countries in cases of mutual interest.
5. Assistance to probation or parole officers and to parole board for their enlightenment in decision making.
6. Imposition of more equitable sentences by the Courts.
7. Identification of persons and maintenance of identity records (service or criminal).
8. Identification of unknown deceased persons.
9. Identification of persons suffering from amnesia.
10. Recognition by the government of honored dead.
11. Identification of disaster work.
12. Prevention of hospital mistakes in the identification of infants.
13. Identification of missing persons.

14. Identification of licensing procedures for automobiles, firearms.
15. Identification of unconscious person
16. Problems of mistaken identity.
17. Establishing correct identity in cases of kidnapping.
18. Detection of bank forgeries.

Classes of Fingerprints

All fingerprints are divided into three major classes on the basis of their general pattern (Fig. 6.19). These are:

- **Loops 60-70% (67%):** Ridges start on one side then run parallelly and end on the same side. May be medial (ulnar) loop or lateral (radial) loop.
- **Arches 5-10% (6-7%):** Ridges start on one side and after a backward course end on the opposite side.
- **Whorls 30-35% (25%):** Multiple circular or oval ridges one around another.
- **Composite 1-2%:** Combination of more than one pattern.
- **Accidental:** No specific ridge pattern.

Characteristics of Fingerprints

Each fingerprint being different from the other but all fingerprints have common characteristics among themselves, (Fig. 6.20) which are:

1. **Pattern area:** It is that part of a loop or whorl in which appears the core, deltas and ridges with which we are concerned in identifying
2. **Type line:** Are ridges that determine the pattern area of loops and whorls. Arches lack presence of type line
3. **Delta or triradius:** It is formed when a ridge bifurcates and the two arms of the bifurcating ridge diverge or when two adjacent ridges running side by side diverge causing an interspace within which the pattern lies.
4. **Core:** Core is the central portion of the pattern and the type of core varies with the type of pattern.

The three basic pattern types arch, loop and whorl are subdivided into nine subtypes for the purpose of classification.

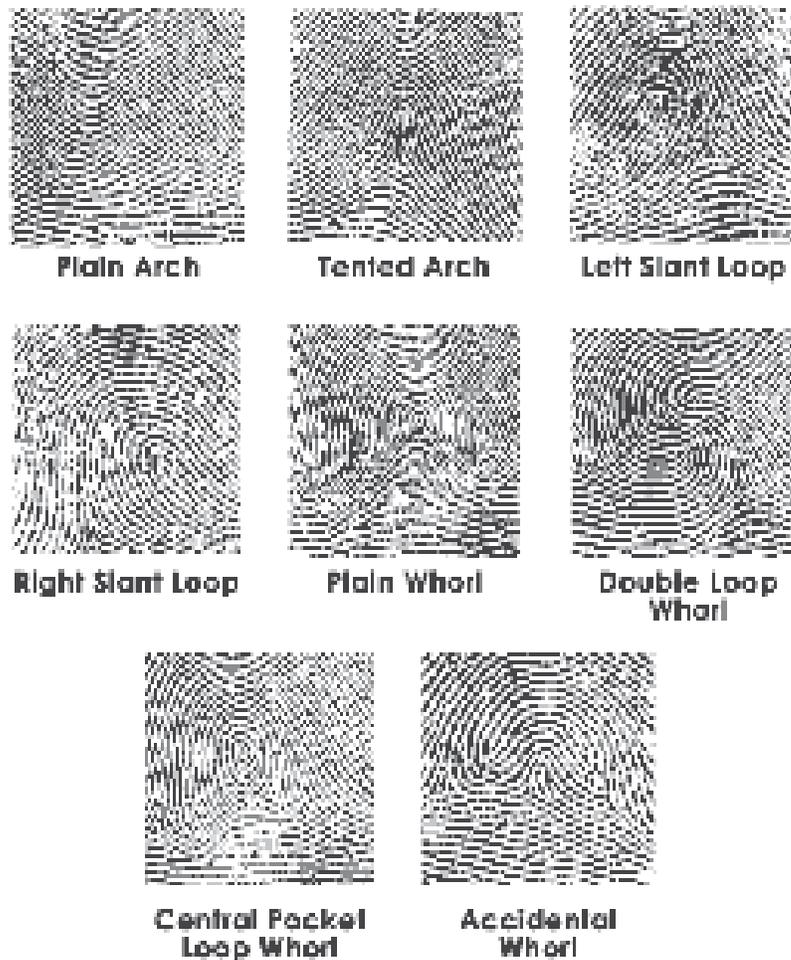


Figure 6.19: Various classes of fingerprints

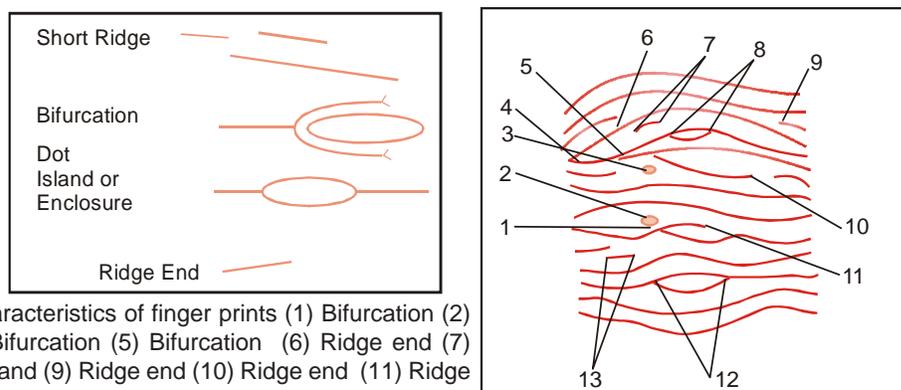


Figure 6.20: Characteristics of finger prints (1) Bifurcation (2) Dot (3) Dot (4) Bifurcation (5) Bifurcation (6) Ridge end (7) Short ridge (8) Island (9) Ridge end (10) Ridge end (11) Ridge end (12) Island (13) Short ridge

Basic pattern	Pattern type	Symbol	No. of deltas present	
Arch	i. Plain	A	Nil	
	ii. Tented	T	Nil	
Loop	i. Radial	R	One	
	ii. Ulnar	U	One	
Whorl	i. Plain	W	Two	
	ii. Central	C	Two	
	iii. Double loop	pocketed loop		
		-Lateral pocket loop	S	Two
		-Twinned loop	S	Two
	iv. Accidentals	X	Two or more than two	

Types of Fingerprints Obtained at The Scene

The form of fingerprints obtained at a scene of crime may be classified under the following categories.

- **Visible prints:** These are visible to the naked eye, they may be formed when a visible or coloured contaminants are present on the fingers of the perpetrator, it will leave a visible print, for example a blood print, an inked print, a colour print, a paint print, a dirt print. These visible prints are photographed directly for the purpose of comparison and maintenance of records.
- **Plastic prints:** Formed when the fingers or the palm comes in contact with soft surface such as soap, butter, wax, soft putty, tar, grease or freshly painted surface. Plastic prints should not be dusted with fingerprint powder and should not be photographed with the help of direct or side lighting.
- **Latent prints:** Are such impressions not visible to the naked eye. Whenever the fingers or palm come in contact with any polished or smooth surface, they leave an invisible image on that surface of contact due to perspiration. This invisible image of fingers or palm is known as latent fingerprint. Sweat glands become more active when a person is nervous or excited and the hands perspire more than normal during this

state of mind. Thus a perpetrator of crime leaves behind invisible print impressions on whatever surface he touches during the act. The latent finger impressions then detected are converted to visible prints through fingerprint powders and chemical sprayer solutions so that they may be photographed, preserved and used for identification.

Printing of Fingerprints

Fingerprint impressions thus obtained are a reverse of actual pattern on the skin surface. The essential material needed for obtained fingerprints include Printer's ink, Paper or card, Inking slab, Inking rubber or cotton pad, Benzene, Alcohol, Kerosene or gasoline and Soap. Fingerprint impressions can be obtained as:

1. **Rolled impressions:** By nail to nail rolling of fingers
2. **Plain or dab prints:** These are unrolled impressions obtained by direct contact

For rolled prints, following precautions should be taken. (i) Hands should be properly wiped. (ii) Arm should be kept relaxed and not try to help in rolling the fingers. (iii) The rolling of the fingers and thumb should be in the following manner, the thumb should be rolled towards the subject's body and the fingers are rolled away from the body that is thumb in and fingers out. (iv) Not too much ink should be used or excessive pressure applied on the fingers. (v) Unglazed papers are better for fingerprints. (vi) If a particular finger is missing or is so defective or deformed that it is not possible to obtain an impression, this fact should be specified in ink on the space allotted for that. (vii) Deformities on fingers like cut marks, scars shall be fully described and it should be noted whether they are temporary or permanent in nature.

Fingerprints Classification System

There are over fifty classification systems in use in different countries that are based on Galton, Henry and Vucetish systems. The famous ten digit classification systems and the single digit classification involve less famous features. Of

these systems the best known is the Battley system. The classification of fingerprint is a method through which the fingerprint impressions are transformed in to a formula to facilitate the recording and searching of the fingerprints. In the ten-digit system, it is analyzed under the following seven systems:

1. *Primary classification system:* Scores are allotted for presence of whorl pattern in different fingers of each hand as follows

- Whorl in right thumb or right index finger = 16 scores
- Whorl in right middle or ring finger = 8 scores for each place
- Whorl in right little finger or left thumb = 4 scores
- Whorl in left index or middle finger = 2 scores
- Whorl in left ring or left little finger = 1 score
- No scores for fingers where no whorls are present

Scores are then $R.T.+R.R.+L.T.+L.M.+L.L.+1$
arranged as $R.T.+R.M.+R.L.+L.I.+L.R.+1$

- 1 is added for the purpose of calculation.
 - The total score of numerator is multiplied by denominator.
2. Major division system
 3. Secondary classification system
 4. Sub-secondary classification system
 5. Second sub-secondary classification system
 6. Final classification system
 7. Key classification system (For the right thumb only).

After the complete analysis of the seven systems, the complete Ten digit formula may be represented in the following manner for each fingerprint step for maintenance of records—Key, Major, Primary, Secondary, Sub-secondary, Second sub-secondary, and final

LATENT FINGERPRINTS

Powdering and Lifting Latent Impressions

A black or grey fingerprint powder is adequate in most places. Black powder should be used for light coloured and white objects and grey powder for black or dark coloured backgrounds. Among other powders, aluminium powder is used as a substitute for grey powder. The Dragon's blood (a natural powder) may be employed on both dark and light coloured surfaces. A fluorescent type of powder is used for developing latent prints on multicoloured surfaces

Magnetic brush and powders are used to increase the efficiency in development of latent prints. The magnetic brush marks with magnetic powders only, which are available in many colours (grey, black, red, yellow etc.). The two types of lifting medium employed in latent fingerprint work are: (i) Transparent cellulose tape in 1.5 or 2.0-inch width rolls (ii) Opaque rubber lifter.

Chemical Methods of Developing Latent Prints

Perspiration consists of about 98% water with traces of salts like sodium, chloride, sulphates, phosphates, carbamates, lactic acid, fatty acid, glucose and urea. Chemical methods make use of the presence of chlorides, which will react with chemicals to produce visible prints. The following methods are used:

- **The iodine method:** Made visible by the absorption of iodine fumes that react with fatty acids and ridges of the print appear yellowish brown or brownish against the background, it is used for old prints.
- **Silver nitrate method:** Sodium chloride reacts with silver nitrate to form silver chloride, which is an unstable white substance that darkens when exposed to light breaking in to silver and chlorine that appear reddish brown against background.

- **Ninhydrin method:** Reacts to amino acids and gives purple reddish brown stains.
- **Osmium tetra-oxide method:** It is used for recent prints. In the presence of fatty substance, osmium tetra-oxide is reduced to free osmium that is dark in colour.
- **Chem. Print method:** Trade name in aerosol type container, used as spray.
- **Hydrofluoric method:** On glass, waxy substance of the print itself repels the liquid.
- **Tannic acid method:** Sets the albumin making the latent prints visible.
- **Osmic acid method:** Osmic acid spray when exposed to light turns black.
- **Mercuric iodide method:** Fumes of mercuric iodide are used.
- **Bromine method:** Bromine vapours are used.
- **Reducing agents for developing prints:** Organic reducing agents like amidol, pyrogallol or hydroquinone is used.
- **Fleming's reagent method:** Mixture of osmic acid, chromic acid and glacial acetic acid is exposed to diazine fast yellow dye that fluoresces in UV light.

Factors making Satisfactory Fingerprinting of Dead Body Difficult

Wrinkled skin of the finger pads resulting from immersion of the body in water; injection of air or various liquids in to fingerpads has been advocated

If immersion is prolonged or decomposition is advanced or in some instances of radiant burns, the skin of the hands and fingers can be peeled away from the underlying tissue. Fingerprints can be made by inserting the technicians finger in to the 'skin glove' inking the area to be printed and rolling. Where the epidermis is absent, skin can be used for producing useful fingerprints.

In dead bodies with contractures and with decomposing tissues, removal of the fingers from hands may permit more satisfactory printing.

Points of Comparison between Two Fingerprints

(i) Ridge pattern, (ii) Ridge endings, (iii) Missing ridge, (iv) Gap in two ridges, (v) Ridge breaking, (vi) Ridge bifurcation, (vii) Ridge re-union, (viii) A dot in ridge gap, (ix) A stud in ridge, (x) An island formation, (xi) A lake formation, (xii) A dot in the lake, (xiii) Union of two ridges, (xiv) Starting of a new ridge in between two ridges, (xv) Presence of a scar, (xvi) Distance between delta and core, (xvii) Number of ridges between delta and core (xviii) Direction of any particular ridge.

Points Necessary for Establishing Complete Identity

Different countries have developed their **minimum number of points** for establishing complete identity of the fingerprints based on their personal practical experience for presentation before the Court. These are:

France 16, Australia 12, Japan 12-14, Canada 10-16, Interpol 12, US 7-12, Spain 10-12, New Zealand 8-12, Israel 10-12.

In India, Maharashtra 12, Assam 7-12, Kerala 7, West Bengal 6, Madras 8-10, Bihar 7, Orissa 5-7, Central fingerprints Bureau (Calcutta) 9.

In 1973, the first All India Forensic Science Congress held at Srinagar adopted a resolution stating that the minimum number of points for establishing the identity beyond doubt in case of fingerprint examination should be fixed at 8. However, if there are 6 or 7 points of identity a qualified opinion can be offered by the expert on his responsibility.

The fingerprint experts of **New Scotland Yard**, London however recommended that at least 16 identical points should be established. Certain American experts are of the opinion that at least 12 identical points should be established. **US, FBI** is of the opinion that if there are 12 points of similarities in the two impressions, the identity is absolute.

Certain Indian experts are of the view that in partially blurred or smudged impressions, 3 identical points are sufficient to establish the identity. Supreme Court in 1978 upheld the opinion on 8 points. Under section 293 of Cr.P.C., the report of the Director of a fingerprint bureau is admissible as evidence without his physical presence in the Court.

Maintenance of Fingerprint Records

Federal Bureau of Investigation (F.B.I.) USA maintains more than two crores of fingerprints. There is systemic maintenance of separate files for different types of prints. In 60% of the world population there is no whorl in any finger, so according to primary classification, the score is 1. On the basis of this scoring, 1,024 divisions are made called **pigeon holes**.

Poroscopy

Poroscopy was described by Locard. The ridges on the fingers and hands are studded with microscopic pores, formed by mouths of ducts of subepidermal sweat glands. Each millimeter of a ridge contains 9-18 pores. These are permanent and immutable during life and vary in size, shape, position, extent and number over a given length of ridge in each individual. This method of examining pores is called Poroscopy and is useful when only fragments of fingerprints are available.

ELECTRONOGRAPHY

X-rays propagate themselves by a waveform motion just as does visible light. It was discovered by German physicist Plank that in some instances X-rays behave more as bullets or bundles called quanta. This means that X-rays are able to cause the ejection of electrons from their orbits round the nucleus of the atom and impart to these electrons a velocity which carries them away from the parent atom to become photoelectrons. This particular characteristic of X-rays can be used for a technique known as Electronography. Electronography is a method whereby metallic elements can be made to produce a recordable emission

when irradiated by a high kilovoltage monochromatic X-ray beam. For the proper functioning of electronographic technique, primary beam should be capable of causing the ejection of electrons and it must be so homogenous and of such a short wavelength that it does not itself have a photographic effect.

Emission Electronography

Here X-ray beam is first passed through a copper filter of 1 cm thickness. This was found to be the optimum thickness of copper necessary to remove all the soft wavelength components and render the beam practically homogenous. The use of the emission electronographic method for the visualization of routine fingerprints is more advantageous.

This method substitutes for the multiplicity of factors involved in the other techniques such as lights, lenses, focusing, positioning, black and grey powders, fluorescent substances, UV light etc. This is a routine procedure, which results in a well-defined print against a clear background.

The pure metallic powders are admittedly, not so adherent to the fingerprint deposit as are the grey and black powders. They can, if necessary be brushed off and the normal police powders reapplied to the fingerprint deposit. The demonstration then carried out by the present methods. Thus there is never any danger or detriment to the fingerprint deposit by trying the electronographic method. If the normal police practice fails to satisfy in a particular case, the metallic element can be sieved over the already treated print. It will adhere to it and the electronographic method can be used.

Method: To the fingerprint deposit, metallic powder is to be applied by sieving it through a 400-mesh sieve so that it drops in a free fall on to the suspected area. The excess powder is removed by blowing or tapping leaving the fingerprinting visualized against its background. Recording film should be mounted on the fingerprint and in intimate contact with it. If the object can be enclosed in a Cassette, it is placed in the cassette containing a layer of clear film.

The recording film is then placed with its emulsion surface in contact with the treated print. This is again covered with a layer of clear film. The cassette is then closed with cardboard packing to ensure intimate film object contact. The cassette with suitably selected filters on top is placed under the X-ray tube. An X-ray exposure is given and backgrounds are eliminated entirely and the palm prints stand out quite clearly.

This technique can be applied to metallic objects also. In case of metallic objects, extremely differential results can be obtained. This is because finely powdered lead dust emits much more readily under the action of the primary beam than does the lead sheet.

GRENZ RAYS

Grenz ray unit is also extremely useful in many fingerprint investigations, especially when used in conjunction with Polaroid papers. It is simpler to use than Electronography and provides an immediate result. Its only application is to objects that can be penetrated by the beam and are of a fairly homogenous structure.

FOOTPRINTS

The offender at the scene of crime may also leave footprints. It can be of barefooted person or of the shoes. Small portion of footprint or shoe is sufficient for comparison. In case of shoe print, primary marking are caused by the make of shoes, soles and secondary markings are caused by changes due to usage. The footprints are used for identification of offenders and as well as of newborn children in hospitals.

LIP PRINTS (CHEILOSCOPY)

Lip prints can be found over wine glass, love letters, on private parts etc. **Le moyne sunder** (1950) pointed out that wrinkles and cracks of lips have certain individualistic characteristics like fingerprints. Japanese authors **Kazuo suzuki** and **Yasuo tsuchihashi** 1970 classified lipprints in the following ways:

1. Type I clear cut grooves running vertically.
2. Type I' vertical grooves but incomplete length.
3. Type II branching.
4. Type III intersected grooves.
5. Type IV reticular.
6. Type V other types or non-classifiable irregular prints.

NAIL STRIATIONS

Striations over nails are said to be permanent but diseases destroying the nails can affect them.

PALATOPRINTS

Rugae present over the palate are also considered permanent and can be used for identification.

FRONTAL SINUSES

Frontal sinuses can be found in over 95% of the population and they can be compared with finger prints. Like finger prints, sinus patterns are unique for the person, the structure is permanent and fixed. Unlike the fingerprints and the bony sinuses can very rarely be altered by infection or injury. The frontal sinuses are classified based on:

1. Size of each frontal sinus.
2. Comparison between right and left frontal sinuses
3. Polar coordinate dimensions from frontal sinus prints.

X-ray of the skull is taken in occipito-mental plane and the comparison is made to the line of frontal septum, upper border of the sinuses, partial septa and supraorbital.

TATTOO MARKS

Tattoo marks are fairly good for identification of both living and dead. It gives wide range of identification information about the persons. It tells us identity, mental makeup, social status, desires and inclinations of the person.

Also they provide information about identification, nationality, religion, God, Goddess, occupation, name of person/relative/lovers, language, habits such as drug addiction etc. Flower,

obscene figure or an idol that is tattooed tells about the mental makeup of the person.

Faded tattoo marks can be made visible by rubbing or by UV light and by burning. In dead bodies tattoo pigment may be found in regional lymph glands at autopsy. Artificial obliteration strongly suggests that the person is trying to conceal his identity. Tattoo in few cases may cause infection, sepsis, ulcers and keloid formation.

Tattooing can be involuntary like by gunpowder or voluntary when they are produced by imprinting pigments of different colours in the dermis, by multiple puncture method mechanically or by electrical device. The pigment used in tattooing are: Carbon dust, Indian ink, Prussian blue, Vermillion/lead tetroxide etc. The duration of tattoo is fairly permanent and can even stay for life if the pigment is put in the dermis or the covered parts of the body. They may fade if the pigments are placed in the subepithelial level or exposed parts due to sunlight, on those parts which undergo constant friction 8-10 years.

They can be removed by surgical operation, local use of corrosive agents, electrolysis by dissolving the pigments and washing them away, by applying carbon dioxide snow and by inflicting deep burns over the area.

FORENSIC ODONTOLOGY

It is the application of dentistry to forensic problems. It is important for: (i) Interpretation of bite marks (ii) Personal identification for establishing the individuality of the person especially in mass disasters.

INTERPRETATION OF BITE MARKS

Police may sometimes bring objects for examination like apple or piece of cheese. The unusual bite mark on the objects may be due to some dental abnormality. 'Plastic' marks on objects such as butter, cheese, lard, wax or chocolates are to be stored in refrigerator to prevent them from melting or flowing; they are not deep frozen as they may brittle and crack.

Fruits especially Apples are preserved in Campden solution (Meta bisulphate fluid) used for fruit bottling. Photographs are taken with film plane at right angles to the bite. Salivary traces should be swabbed. Bites are commonly seen in cases of :

1. Child abuse: In child abuse, the marks are seen anywhere on the body such as arms, hands, shoulders, cheeks, buttocks and trunk. Bites are examined whether the size of the mark is same as of adult dentition; if it is of small size, could be due to bite by the sibling and when it is of different size it could be an animal bite.
2. Sexual assault: Sexually oriented bites can be caused at any parts of the body namely Breasts, neck, shoulders, thighs, abdomen, pubis or vulva.
3. Police officers by the resisting offenders.
4. In sporting events like Football and wrestling.
5. In assaults anywhere on the body.

Medicolegal Aspects of Bite Marks

1. Self inflicted: Self inflicted bite marks seen at accessible parts of the body such as shoulders or arms etc, usually seen in psychiatrics, teenage girls and old children.
2. Accidental at times resulting from falls on to the face and during fits; biting of tongue and lips will also be there.

Nature of Bite Marks

Bite mark comprise of a crop of punctate haemorrhages varying from small petechiae to large Ecchymoses merging in to a confluent central bruise.

Front teeth cause bite marks from canine to canine with an invariable gap at either side representing the separation of upper and lower jaw. A circular or shallow oval is human bite and deep parabolic arch or U shaped is characteristic of an animal bite. Teeth may cause clear separate marks that run in to each other as continuous, intermittently broken lines. There may be abrasions, bruises and lacerations or a combination of all these.

Suction petechiae without teeth marks in sexual assault are caused by human bites, have a sucking effect and due to the rupture of small venules petechial haemorrhages result.

Identification from bite marks is possible if incisors and canines has some characteristic features.

Bite Mark Investigation

1. Bite mark is photographed from different angles; from a directly perpendicular viewpoint with a plane of film at right angles to that of the lesion, with an accurate scale
2. Swabbing of saliva: To identify or exclude assailant; 80% of the people are 'secretors' who exude blood group substances in the saliva. Plain cotton wool swabs are rubbed on to the bite after moistening with water or saliva. These are then deep frozen and sent to serology laboratory
3. Impression of bite mark: Plastic substance is laid over the bite mark that hardens and produces permanent negative cast of the lesion. Plastic substance is made with a rubber or silicone based medium containing a catalytic hardener or Plaster of Paris (water based plaster).
4. Skin carrying the bite is removed and preserved in formalin for future examination; formalin causes shrinkage and distortion and makes tooth matching difficult.

Matching the bite mark with the suspect's dentition:

1. Full informed consent before examination of the suspect in writing to be taken
2. Oral consent with at least one witness, if not written to be taken
3. Dentition examined and points determined and recorded by diagram and writing
4. Photographs can be taken.

The points to be noted in bite marks are:

1. Presence of full or partial denture; were they worn at the time of incidence?
2. Number of teeth in the upper and lower jaw.

3. Charting of missing teeth.
4. Estimate of bite overhang; whether there is an edge-to-edge occlusion or an undershoot projection of lower teeth.
5. Recording of any broken teeth or teeth with significant individual abnormalities are charted and described.
6. Any irregularity or marked variation in cutting edge profile of any front teeth.
7. Evaluation of size and prominence of any teeth especially canines and incisors.
8. Any developmental abnormalities are to be noted.
9. Recording of any abnormality in orientation of any tooth such as twisting (rotation) of anteroposterior tilting or double row of teeth; any gap or irregular spacing: (i) Six upper and six lower front teeth give the most information. (ii) Canines may provide particular help. (iii) Premolars and molars are rarely useful due to being posteriorly positioned in the jaw.

IDENTIFICATION OF THE DEAD FROM DENTITION

The teeth helps in identification of the individual as no two sets of teeth are identical. This is particularly important in:

1. Disasters such as aviation accidents, marine accidents and air crashes in cases of mutilated or burned bodies.
2. To identify the unknown in accidents, suicides or homicides.

Identification in mass disasters includes the:

- *General or reconstructive identity:* This includes classification of the unknown by age, sex and race.
- *Comparative method:* To confirm or exclude personal identity against antemortem dental records.

General/Reconstructive Identity

In fragmented bodies or skeletonised remains from jaw remnants and teeth, the following feature are easily recognized.

Sex of the Person

There are certain differences in male and female dentition as given in Table 6.18.

Findings	Males	Females
Size of upper central and lateral incisors	Different	Same
Size and shape of the teeth	Less pointed and bigger, more in mandible	Mandibular teeth are more pointed and smaller
Mandibular first molar	Has a fifth cusp	Fifth cusp is absent
Pulp tissue from tooth (Fluorescent staining for intranuclear F body)	Absent	Present

Race of the Person

In civilized societies, first molar is the longest and third molar is smallest where in primitive races, first molar was smallest and third molar was longest in size.

1. **'Shovel'** shaped upper central incisors is seen in Mongoloids. Posterior surface have a depression centrally with two marginal bars, causing the back of tooth to appear like a coal shovel with turned up edges. This is found in 91% of Chinese, Japanese and Tibetans; 84% Eskimos; 46% Palestinian Arabs, 90% Finns and rarely in Negroes and Australians aborigines. In White races, lateral incisors in upper jaw are smaller than the central especially in females; is less marked or absent in Negroid and mongoloids.
2. Long pointed canine roots are not seen in the mongoloids
3. **Enamel pearls** Are small nodules of enamel on the tooth surface, are frequent in mongoloids
4. **Carbelli's cusp** Small nodules on lingual surface of maxillary molar is common in white races and in rare in others.

5. **Taurodontism** (Bull tooth) Are common in mongoloids; the pulp cavity of molars is wide and deep and the roots are fused and bent.
6. Congenital lack of third upper molar is common in Mongoloids.
7. Large teeth with more cusps in their molars even up to eight with two lingual cusps on Mandibular first premolars are common in Negroid races.

Age of the Person

1. By eruption of deciduous and permanent teeth.
2. Gustafson's criteria:
 - Occlusion attrition of the tip of the tooth
 - Secondary dentine deposition in the apex of pulp cavity.
 - Apical migration of attachment of periodontal membrane.
 - Increase in root transparency—Is best single criterion.
 - Root resorption.
 - Accumulation of cementum around the root. This method was later modified by Johanson; age can be calculated within ± 5 years.
3. Comparative identification from the teeth by charting.

Charting the Teeth: On the charts following are recorded:

1. Any extractions, recent or old from the condition of the socket.
2. Any fillings, number, position and composition.
3. Artificial teeth whether of gold, porcelain or stainless stain.
4. Prosthetic work in mouth such as bridge work or braces.
5. Any crowned teeth.
6. Any broken teeth.
7. Pathological conditions in teeth, jaws or gums
8. Congenital defects such as enamel pearls, carabelli's cusps or ectopic teeth.
9. Malpositioned teeth that are rotated or tilted.

- 10. General state of care and hygiene like caries, plaque, tobacco staining, gingivitis etc.
- 11. Racial pointers such as shovel shaped upper central incisors and multi-cusped molars.

Chart designation of teeth: In the middle of nineteenth century, the designation of teeth was based upon anatomical names such as upper and lower jaws, left and right in relation to midline. Latin names were used depending on the function of the teeth.

There are various systems for charting of the teeth such as:

1. **Zigmond system:** First system was given by Zigmond in 1861. He numbered the permanent teeth with arabic numerals 1 to 8 from midline backwards. the quadrants were denoted by a line drawn between the central incisors bisected by a line separating the upper and lower teeth. The deciduous teeth were given Roman numerals I to V, individual teeth been denoted by the quadrant sign.

8 7 6 5 4 3 2 1	1 2 3 4 5 6 7 8	Permanent
8 7 6 5 4 3 2 1	1 2 3 4 5 6 7 8	
V IV III II I	I II III IV V	Deciduous
V IV III II I	I II III IV V	

Every type of tooth had same designation so the angle of mandible was used to differentiate and the following numbers were given such as:

- 6 = upper right first molar; 3 = upper left canine and 2 = lower right lateral incisors etc.

Disadvantages: This method could be not be easily adopted in the typewriters. As far as hand written system is concerned this system is very simple to use.

2. **Palmer's notation:** This method was devised by dentist Palmer in 1870 and 1891 as

Right	8 7 6 5 4 3 2 1	1 2 3 4 5 6 7 8	Left
	8 7 6 5 4 3 2 1	1 2 3 4 5 6 7 8	

3. **Universal (Cunningham) system:**

Cunningham in 1883 proposed that it would be simpler to avoid the quadrant symbols by using a straightforward numbering notation. Cunningham's notation involved numbering the teeth 1 to 32 and lettering the deciduous teeth A to T, starting at the posterior upper right and continuing in a clock wise direction. In this system, the teeth are numbered from 1-16 from right to left in the upper jaw and 17-32 from left to right in lower jaw. The universal system followed by the international society of Forensic Odontology. But it underwent a number of modifications in USA.

Right	1 2 3 4 5 6 7 8	9 10 11 12 13 14 15 16	left
	32 31 30 29 28 27 26 25	24 23 22 21 20 19 18 17	

It is the best method of charting but the disadvantages are that it is difficult to learn and the risk of selecting the wrong number is great.

4. **Haderup system:** Haderup devised a notation published in 1902 which made use of the plus and minus sign and retaining the anatomical classification of single teeth as devised by Zsigmond. To differentiate between the left and right sides the '+' for the maxilla and '-' for the mandible were placed in front of the number for the left side and behind for the right side, the sign thus being towards the midnight. For the deciduous teeth a zero was additionally placed in front of the number. This notation was adopted by the Scandinavian countries

For permanent teeth,

8+ 7+ 6+ 5+ 4+ 3+ 2+ 1+	+1 +2 +3 +4 +5 +6 +7 +8
8- 7- 6- 5- 4- 3- 2- 1-	-1 -2 -3 -4 -5 -6 -7 -8

For deciduous teeth,

05+	04+	03+	02+	01+	+01	+02	+03	+04	+05
05-	04-	03-	02-	01-	-01	-02	-03	-04	-05

5. FDI Notation: A two digit notation capable of indicating tooth and quadrant was developed. Each permanent and deciduous quadrant was designated by a separate number in a clock wise direction followed by Zsigmondy's number at a meeting of the Federation Dentaire Internationale in Bucharest in 1971 this was adopted as the International Standard and was adopted subsequently by the American Dental Association, the International Standard Organizations, The International Association for Dental Research and the World Health Organization. From the Forensic Odontologist's point of view the acceptance of this notation by Interpole for identification purposes was a significant step in International communication the the system is known as the FDI notation.

For permanent teeth

18	17	16	15	14	13	12	11	21	22	23	24	25	26	27	28
48	47	46	45	44	43	42	41	31	32	33	34	35	36	37	38

For deciduous teeth

56	55	54	53	52	51	61	62	63	64	65	66
86	85	84	83	82	81	71	72	73	74	75	76

The disadvantage of this method are that teeth could be confused in the change over from one 2-digit notation to another.

Advantages of Dental Charting: The dental charting is better for forensic purposes involving telegrams or telephones. However in such cases, good photography is also necessary and in no

case should telegrams or telephone calls alone be relied upon.

Medicolegal Importance of Dental Study

1. Identification of the individual: From the dental study, age, sex and race of the person can be identified as described before. Other factors for identification include:
 - The habits such as chewing tobacco are also helpful for identification.
 - Peculiarity in setting of the teeth may be familial and may help in identification.
 - The teeth resist decomposition so they are important for identification even for a long time after death as peculiarity of setting of teeth, evidence of any missing tooth or any artificial dentures help in proving the identity of the person.
 - Occupation can be known if there is an notch under tooth e.g. in case of tailors.
2. *Grievous hurt:* Fracture, Dislocation of tooth amounts to grievous hurt according to section 320 (7) I.P.C..
3. *Cause of death:* The teeth resists putrefaction and the amount of deposition of heavy metals can be detected for a considerable period after death, special helpful in poisoning cases as well as in mass disaster.
4. In chronic phosphorus poisoning, evidence of phossy jaw and affection of teeth and gums are characteristic.
5. Bite mark over the articles or food help towards detection of the criminal.
6. Bite mark on private parts, breasts, cheeks, neck of a girl are suggestive of some sexual instinct.
7. *Artificial dentures* when dislodged can sometimes cause choking in the elderly.
8. *Pink teeth:* In putrefied bodies, near gum line, teeth are of pink colour. It is due to dentine being stained by haemoglobin products

SUPERIMPOSITION

It is the technique by which a particular skull recovered can be matched with the photograph. It

is very helpful for identification. More recent photograph is better. Even photograph of lateral view can be used. Two negatives are prepared one of the person and the other of the skull. Appropriate magnification is done to achieve maximum alignment and where interpupillary distance of both exactly matches. It is a known fact that the interpupillary distance of a person never changes regardless of his age. The two negatives are then superimposed by keeping over each other and various points are compared.

The canthus, the nasion, the nasal spine and lower border of nose and upper jaw, the supra-orbital ridges, angle of jaw, external auditory meatus and the teeth etc. are compared and a photograph is taken which is analysed for similarities and dissimilarity to arrive at a conclusion.

This test has more of negative value as it can definitely rule out a combination of a photo and a skull though only a possibility of a match can be established.

Long Questions

1. What is identification? Discuss various points to establish the identity of a person.
2. Enumerate the points to identify a living person. Discuss in detail Dactylography.
3. How you will proceed to determine the age of a person of 16 years? What is its medicolegal significance.
4. Discuss the medicolegal importance of age.

5. Discuss the role of dentition in age determination.
6. Discuss in detail the various aspects of forensic odontology and its application in identification of a person.
7. Discuss in detail the medico legal significance of dactylography.
8. What is the medicolegal importance of hair?

Short Questions

1. Inter sex.
2. Tattoo marks.
3. Super imposition.
4. Scar.
5. Sutures of skull.
9. Dactylography.
10. Latent fingerprints.
11. Poroscopy.
12. Electronography.
13. Tattoo marks.
14. Klienfelter's syndrome.
15. Turner's syndrome.
16. Difference between temporary and permanent teeth.
17. Difference between male and females skull.
18. Difference between male and female mandible.
19. Difference between male and female sacrum.
20. Differentiate between animal and human hair.

MULTIPLE CHOICE QUESTIONS

1. Identification means to determine the:
 - A. Age of person
 - B. Sex of person
 - C. Height of person
 - D. All of the above
2. Which of the following is the main part of corpus delicti:
 - A. Identification of the dead body
 - B. Identification of the weapon of offence
 - C. Identification of clothing
 - D. All of the above
3. Rule of Haase was formulated to calculate the:
 - A. Age
 - B. Sex
 - C. Stature
 - D. None of the above
4. Which of the following feature is not correct in a foetus of 7 months of intra uterine life:
 - A. Testes are found close to kidneys
 - B. Length is 35 cm
 - C. Caecum is found in right iliac fossa
 - D. Meconium is seen in whole of large intestine
5. The average length of a full term new born is:
 - A. 40-45 cm
 - B. 45-50 cm
 - C. 50-53 cm
 - D. 55-60 cm
6. In a child, first permanent tooth to erupt is:
 - A. Central incisor
 - B. First molar
 - C. Canine
 - D. Lateral incisor
7. Total number of teeth at the age of 8 years in females is:
 - A. 12
 - B. 16
 - C. 20
 - D. 24
8. The anterior fontanelle usually closes between:
 - A. 6-9 months
 - B. 9-12 months
 - C. 12-18 months
 - D. 4-5 years
9. Maximum number of milk teeth in a child is:
 - A. 16
 - B. 20
 - C. 24
 - D. 28
10. Temporary molars are replaced by permanent:
 - A. Molars
 - B. Premolars
 - C. Canines
 - D. Incisors
11. The permanent canine tooth erupts at:
 - A. 9-11 years
 - B. 10-12 years
 - C. 11-12 years
 - D. 12-14 years
12. Total number of teeth in females at age of 4 years:
 - A. 16
 - B. 18
 - C. 20
 - D. 24
13. A boy has 20 permanent teeth and 8 temporary teeth. His age is likely to be:
 - A. 9 yrs
 - B. 10 yrs
 - C. 11 yrs
 - D. 12 yrs
14. Permanent second molar erupts at around....of age:
 - A. 6 years
 - B. 12 years
 - C. 18 years
 - D. 24 years
15. First permanent molar appears at age of:
 - A. 6-7 yrs
 - B. 7-9 yrs
 - C. 9-11 yrs
 - D. 11-15 yrs
16. To estimate the age of 16 years, following X-rays are advised:
 - A. Elbow, hand, oblique view of jaw
 - B. Wrist, pelvis, oblique views of jaw
 - C. Knee, pelvis, skull
 - D. Ankle, shoulder, pelvis
17. The appearance of center of ossification in case of olecranon is:
 - A. 5 years
 - B. 7 years
 - C. 9 years
 - D. 11 years
18. The different pieces of sternal body becomes a single bone at the age of:
 - A. 16 years
 - B. 20 years
 - C. 25 years
 - D. 30 years
19. The normal male skeleton weighs about:
 - A. 2.5 kg
 - B. 4.5 kg
 - C. 6.5 kg
 - D. 8.5 kg
20. The appearance of center of ossification in case of ischial tuberosity is:
 - A. 12 years
 - B. 16 years
 - C. 18 years
 - D. 20 years
21. X-ray of which of the following region is useful for the determination of age below 16 years of age:
 - A. Lower end of femur
 - B. Lower end of humerus
 - C. Upper end of humerus
 - D. Upper end of tibia

1 A 2 D 3 A 4 C 5 C 6 B 7 D 8 C 9 B 10 B 11 C
 12 C 13 D 14 B 15 A 16 A 17 C 18 C 19 B 20 B 21 B

22. Age determination of an 18-year old on X-ray can be done from:
 A. Wrist and elbow
 B. Ankle and knee
 C. Knee and shoulder
 D. Shoulder alone
23. Angle of mandible is obtuse in:
 A. Edentulous person
 B. Females
 C. Person above 40 years of age
 D. Person above 60 years of age
24. Age estimation is best done by examination of:
 A. Pubic hair
 B. Dental X-ray
 C. X-ray of maxilla
 D. Eyes
25. Calcification in costochondral cartilage occurs at an age (years) of:
 A. 20-25 B. 35-50
 C. 50-60 D. 70-80
26. Calcification in costal cartilage occurs at age of:
 A. 20-25 years B. 35-50 years
 C. 50-60 years D. 70-80 years
27. Xiphoid process unites with sternum at the age of:
 A. 25-30 years B. 40 years
 C. 16-24 years D. 60 years
28. Medial end of clavicle fuses at the age of:
 A. 12 years B. 14 years
 C. 16-18 years D. 18-22 years
29. Attainment of 16 years of age is diagnosed on X-ray of:
 A. Medial end of clavicle
 B. Knee
 C. Wrist
 D. Ankle
 E. Elbow
30. Angle of mandible is more obtuse in all except:
 A. Males
 B. Female
 C. Infancy
 D. Old age
31. Age of 21 years is important medico-legally in all except:
 A. To import a girl for illicit intercourse
 B. Marriage contract in boys
 C. If person is under protection of court
 D. To procure a girl for prostitution
32. In old age, the mental foramen opens:
 A. Near the lower margin
 B. Near the alveolar margin
 C. Midway between upper and lower margins
 D. None of the above
33. Best bone to assess age between 20-50 years:
 A. Skull
 B. Ribs
 C. Sternum
 D. Symphysis pubis
34. Hair usually start appearing at the pubic region in females at the age of:
 A. 11 years
 B. 13 years
 C. 15 years
 D. 16 years
35. Beard hair grows at the rate of:
 A. 0.3 mm per day
 B. 0.4 mm per day
 C. 0.5 mm per day
 D. 0.6 mm per day
36. The suture between the parietal bones is known as:
 A. Lambdoid suture B. Sagittal suture
 C. Coronal suture D. Metopic suture
37. Coronal suture completely fuses by the age of:
 A. 20 yrs B. 30 yrs
 C. 40 yrs. D. 45 yrs
38. Closure of all skull sutures indicates age more than:
 A. 40 years B. 50 years
 C. 60 years D. 70 years
39. Number of carpal bones ossified by 3 years of age are:
 A. 2 B. 3
 C. 5 D. 7

22 23 A 4 C 5 C 6 B 7 D 8 C 9 B 10 B 11 C 12
 C 13 D 14 B 15 A 16 A 17 C 18 C 19 B 20 B 21 B

40. Which of the following is the most reliable criteria to find out age by Gustafson's method?
 A. Periodontosis
 B. Attrition
 C. Secondary dentine
 D. Root resorption
 E. Transparency
41. Age estimation from the physiological changes of teeth after 25 years is known as:
 A. Galton's method
 B. Bertillon's method
 C. Pearson's formula
 D. Gustafson's method
42. In Turner's syndrome, the chromosomal pattern is:
 A. 46 XY B. 45 XO
 C. 47 XXY D. 48 XXY
43. The chromatin pattern in a female is:
 A. XX B. XO
 C. XY D. XXY
44. Sex Chromatin can be detected in:
 A. Monocytes
 B. Lymphocytes
 C. Neutrophils
 D. All of the above
45. Masculine or feminine characteristics most directly depends on:
 A. Genotype of somatic tissue
 B. Genotype of gonadal tissue
 C. Gonadotropin output
 D. Level of circulating sex hormones
46. In the buccal smears of females, the percentage of nuclei containing chromatin body ranges from:
 A. 0-4
 B. 6-10
 C. 12-16
 D. More than 20
47. Karyotyping of foetus may be done from all of the following except:
 A. Lymphocyte
 B. Monocyte
 C. Amniocyte
 D. Fibroblast
48. For determination of sex, smear is taken from:
 A. Buccal mucosa B. Vagina
 C. Skin D. Nose
49. Sex can be determined by:
 A. Urinary epithelial cells
 B. Buccal mucosa epithelial cells and RBCs
 C. Dentine
 D. Bone marrow
50. Chromosomal defect is a feature amongst all of the following except:
 A. Inter Sex
 B. Concealed Sex
 C. Pseudo hermaphrodite
 D. True hermaphrodite
51. The chin in cases of male looks:
 A. Triangular B. Square
 C. Rounded D. Flattened
52. Skull of a male differs from that of a female by all of the following except:
 A. Capacity greater than 1500 c.c.
 B. Muscular markings over occiput are less marked
 C. Orbits are square
 D. Frontal eminence small
53. The cranial cavity volume of male skull as compared to female skull is more by:
 A. 100 ml B. 150 ml
 C. 200 ml D. 250 ml
54. The degree of accuracy in sexing adult skeletal remains from skull alone is:
 A. 50 percent B. 60 percent
 C. 70 percent D. 90 percent
55. All of the following are features of female pelvis which distinguish it from male pelvis except:
 A. Sub pubic angle 80° or more
 B. Wider greater sciatic notch
 C. Anterior part of iliac crest turned inwards
 D. Sacrum is short and wide
56. The obturator foramen in cases of females looks:
 A. Oval B. Rounded
 C. Triangular D. Square

1 A 2 D 3 A 4 C 5 C 6 B 7 D 8 C 9 B 10 B 11 C
 12 C 13 D 14 B 15 A 16 A 17 C 18 C 19 B 20 B 21 B

57. The difference between a female pelvis and a male pelvis are all **except**:
- Subpubic angle 90° or more
 - Wider greater sciatic notch
 - Large obturator foramen
 - Well marked preauricular sulcus
58. In male pelvis:
- Sub pubic angle is more than 90°
 - Obturator foramen is oval
 - Greater sciatic notch is larger and shallower
 - Preauricular sulcus is well marked
 - Ischiopubic rami is everted
59. Difference between female pelvis from a male are all **except**:
- Sub pubic angle 90° or more
 - Wider greater sciatic notch
 - Larger Obturator foramen
 - Well marked Preauricular sulcus
 - Female pelvis is lighter
60. The degree of accuracy is sexing adult skeletal remains from Pelvis alone is:
- 80%
 - 85%
 - 95%
 - 98%
61. 'Preauricular Sulcus' is useful for:
- Age determination
 - Sex determination
 - Race determination
 - Mode of death
62. A male clavicle differs from female clavicle by all **except**:
- Longer
 - Broader
 - Heavier
 - More curved
63. Single best criteria to distinguish the female from male pelvis is:
- Shape of Obturator foramen
 - Shape of ischial tuberosities
 - Shape of acetabulum
 - Shape of greater sciatic notch
64. Which of the following adult bone is most useful in determining sex:
- Skull
 - Pelvis
 - Femur
 - Clavicle
65. 99% sex determination is by:
- Sacropubic angle
 - Presacral sulcus
 - Iliosacral notch
 - Obturator foramen
 - Ischiopubic rami
66. Ischiopubic index is used to determine:
- Age
 - Sex
 - Race
 - Stature
67. Height of the body can be deduced from the length of humerus by following equation:
- Humerus is 10 percent of height
 - Humerus length is 50 percent of height
 - Humerus length is 20 percent of height
 - Humerus length is 2 percent of height
68. Stature of a person is more at:
- Evening
 - Night
 - Mid-day
 - Morning
69. Cheiloscopy is used to identify the person from:
- Palato prints
 - Lip prints
 - Foot prints
 - Finger prints
70. The surest method of identification is:
- Cephalic Index
 - Finger prints
 - Handwriting
 - All of the above
71. The most common type of finger print is:
- Arch
 - Whorl
 - Loop
 - Composite
72. For the matching of fingerprints, the minimum points required is:
- 5
 - 10
 - 15
 - 20
73. Which of the following is the surest method of identification:
- Handwriting
 - Speech
 - Fingerprint
 - Complexion and feature
74. Majority of finger prints in Indians are:
- Loop
 - Whorl
 - Arch
 - Composite
75. Best method for identification of a person is:
- Dactylography
 - Anthropometry
 - Personal appearance after death
 - Colour change of hair

1 A 2 D 3 A 4 C 5 C 6 B 7 D 8 C 9 B 10 B 11 C
12 C 13 D 14 B 15 A 16 A 17 C 18 C 19 B 20 B 21 B

76. **Most specific and sensitive for identification is:**
A. Anthropometry
B. Dactylography
C. Skull
D. Pelvis
77. **Cephalic index of 80-85 is seen in:**
A. Pure Aryans
B. Negroes
C. Aborigines
D. Mongolians
78. **What would be the race of individual with the skull bone having following features—rounded nasal opening, horseshoe shaped palate, rounded orbit and cephalic index above 80:**
A. Negro
B. Mongol
C. European
D. Aryans
79. **Cephalic Index helps to determine the:**
A. Age
B. Race
C. Weight
D. Sex
80. **Human and animal hair are differentiated by:**
A. Scales and cuticle
B. Medulla absent
C. Both of the above
D. None
81. **Absent or fragmented medulla of hair is seen in all except:**
A. Mongols
B. Negroid
C. Caucasians
D. Servocratarians
82. **Which of the following feature of human hair distinguishes it from animal hair:**
A. Medulla is 1/3 of the shaft diameter
B. Medulla and cortex have equal diameter
C. Heavy continuous pigment in human hair
D. Medulla has more than half of shaft diameter
83. **Feature of human hair are all except:**
A. Cuticular scales are short and flattened
B. Medulla is continuous and wide
C. Cortex is thick and medulla is one third of shaft diameter
D. Pigment in medulla is light and broken
84. **Male and female hair can be differentiated by that female hair has:**
A. No medulla
B. No bulb
C. Is longer
D. Sex chromatin is positive
85. **Scar usually becomes pale at the age of:**
A. 1 week
B. 2 week
C. 3 week
D. 4 week
86. **For the erasure of tattoo marks, which of the following method is used:**
A. Laser beam
B. Electrolysis
C. Surgical method
D. All of the above
87. **Identification of the face using a skull X-ray is known as:**
A. Galton's system
B. Gustafson's method
C. Superimposition
D. Locard's Principle

Artefacts

Dorland's Illustrated Medical Dictionary defines an artefact as any artificial product; any structure or feature that is not natural, but has been altered by processing. In Forensic Pathology, the artefact is any change caused or a feature introduced into a body after death that is likely to lead to misinterpretation of medicolegally significant antemortem findings.

It is the duty of the pathologist to interpret the artefacts correctly. The misinterpretation may lead to: (i) wrong cause of death (ii) wrong manner of death (iii) undue suspicion of criminal offense (iv) halt in the investigation of a homicide and non-detection of murder (v) miscarriage of justice in civil suits (vi) unnecessary spending of time and effort as a result of misleading findings. The artefacts are divided into two groups:

1. Artefacts introduced between death and autopsy.
2. Artefacts introduced during autopsy.

ARTEFACTS INTRODUCED BETWEEN DEATH AND AUTOPSY

Agonal Artefacts

Regurgitation and aspiration of the gastric contents is a very common artefact seen in forensic practice. This may occur in natural deaths as a terminal event, from handling of the body or during resuscitation, this may result in physicians in wrongly giving aspiration of gastric contents as

cause of death.

Resuscitation Artefacts

The body may reveal injection marks, bruises and fractures etc. when the body is subjected to resuscitative attempts. The injection marks are usually seen in the cardiac region or on the extremities. The intracardiac injection may be associated with bruising of the heart and collection of some blood in the pericardium. The injection prick marks produced in the agonal period need to be differentiated from the marks of drug abuse.

The defibrillator applied over the body produces ring like bruises over the chest. The ribs may sometimes be fractured from external cardiac massage but they do not show effusion of blood at the fracture site or in the intercostal muscles. Sometimes recent hemorrhages may be present in post mortem fractures, in such cases careful history of resuscitative measures is to be obtained. Evaluation of such fractures is of immense importance in deaths from violence particularly beatings and battered babies. Fat or bone marrow embolization is frequently associated with or without fractures of ribs or sternum with external cardiac massage. The positive pressure breathing apparatus used for resuscitation tend to leave the evidence of acute emphysema, occasionally with subpleural blebs, air in the mediastinum and even

tension pneumothorax.

Embalming Artefacts

The dimensions of the homicidal stab or gunshot wounds may be modified by the embalmer while trying to introduce the trocar through them. The multiple trocar holes may in a case of gunshot injuries may cause difficulties for the pathologist. Also the track of the weapon or the bullet may be disturbed, creating false tracks. The surgical alteration or suturing of gunshot wounds may complicate the situation. The embalming fluids may cause confusion for the forensic scientists when analyzing the viscera for drugs or chemicals. Embalming fluids generally contain formaldehyde, methyl alcohol and sometimes other interfering compounds.

Internment and Exhumation Artefacts

In bodies interned for sometime and later exhumed, fungus growth is common at body orifices, eyes and site of open injuries. The underlying skin is discoloured simulating bruising. While performing autopsy on exhumed bodies, the agonal, resuscitative, embalming artefacts and those resulting from handling of the body after death should be borne in mind. Internment leads to the exposure of the body to the contaminants of the soil and draining water.

Artefacts Related to Rigor Mortis and Hypostasis

While handling the body the rigor may break resulting in false estimate of time of death. The onset or progress of rigor may be significantly affected by postmortem circumstances e.g. extreme heat or cold or antemortem conditions e.g. extreme hyperthermia due to widespread infections. Rigor of the heart causes the stiffening of the muscles and may simulate its concentric hypertrophy.

The colour of the post mortem staining is usually purplish. Cherry red colouration may sometimes be seen in carbon monoxide poisoning. Pink hypostasis is a common finding in bodies exposed

to cold temperature in an open environment or in bodies refrigerated for sometime. Localized areas of hypostasis may resemble bruises.

Decomposition Artefacts

The decomposition of the body produces certain changes that may be mistakenly interpreted as pathological lesions. Advanced decomposition causes swelling of the lips, nose and eyelids, protrusion of the eyes, distension of the chest and abdomen and swelling of the extremities. These changes may lead to the false impression of the antemortem obesity. In decomposed bodies the presence of body fluids in the mouth and nose is a frequent finding. In the presence of pulmonary oedema, large quantities of such fluid may escape from the mouth and nose. This may lead to an erroneous conclusion that the person died of massive haemorrhage. Decomposition blebs are occasionally misinterpreted as vesications from burns. In a decomposed body, a deep groove may be seen around the neck if the deceased has been wearing a buttoned shirt or blouse at the time of death. These grooves often simulate ligature marks seen in strangulation cases. Even in nondecomposed bodies, bands or grooves of contact flattening in the skin of the neck of obese individual or of well nourished infants may be seen. The decomposed blood acquires a dark colour and produces appearance of congestion in the brain, lungs, right side of the heart and other parts of the body so that it becomes difficult to form the conclusion of death from asphyxia. The marks on the neck should be carefully interpreted. In some cases marked bluish discoloration of the loops of intestine is seen. This finding may be labeled by some pathologist as that of infarcted bowel. Correct interpretation of the finding of the air in the large veins and in the right side of the heart is of great importance in medicolegal autopsies. During life air may enter the systemic venous circulation during criminal abortions, craniotomy, urethroscopy, fallopian tube insufflation, pneumoperitoneum, aortography and intravenous

infusions. In cases of air embolism following abortion, air bubbles are usually found in the cavity of the uterus and its wall, the uterine and adnexal veins, the inferior vena cava and right heart, the pulmonary arteries, the left heart and the systemic arteries. The presence of gas in the right side of the heart resulting from decomposition may resemble the findings in air embolism and it may be difficult to distinguish one from the other. The analysis of the gas that is collected from the heart and brought into contact with an alkaline pyrogallol solution turns brown in the presence of free oxygen, gives support to the diagnosis of antemortem air embolism. Gas produced by decomposition will not produce this colour change.

Gunshot Wounds Artefacts

During the hospital stay the victim of gunshot wound may sustain additional pseudogunshot wounds. The drainage holes in the chest or abdomen, such as embalmer's trochar holes may be confused with the gunshot wounds. Surgical alteration or suturing of the wounds may lead to difficulty in evaluating the wound known as **Kennedy phenomenon**.

In decomposed bodies these wounds may be greatly modified.

Toxicological Artefacts

Decomposition of the biological material often causes apparent alterations in the constituents of the tissue. It is generally known that the alcohol is produced postmortem because of putrefaction (enzymatic decomposition, especially of proteins, with the production of foul smelling compounds) of the tissues and the fact that many of the bacteria can produce alcohol. However it must be emphasized that the levels of alcohol generated by putrefaction are usually less than 100mg percent. If the levels are more than 100mg percent it should arouse the suspicion of alcohol ingestion prior to death. Decomposition is also known to cause an increase in the concentration of carboxyhaemoglobin in the blood; the increase

has been reported of 19-25% by various authors. Cyanide is produced by decomposition in toxicologically significant amounts; levels up to 10mg% have been found in three month old blood.

Many substituted phenols (extremely poisonous compounds generally from coal tar) have been found in the decomposing tissues. The one that cause disturbing interference in the analysis of weak acid fractions are related to p-hydroxyphenyl derivatives.

Combustion of the body tissues and other materials associated with the burned body produces several gases. Oslen and his colleagues investigated gases generated in experimental fires and demonstrated the presence of ammonia, carbon monoxide, hydrogen cyanide, hydrogen sulphide, nitrogen, oxides of nitrogen and sulphur dioxide. Combustible material like silk and wool produces high concentrations of several of these gases because their nitrogen content is high. Some of these gases may be detected in the body tissues and the results of toxicological analysis may raise false alarms. Cyanide levels in the blood of the burn victims were reported between 17-220mg per 100ml.

Miscellaneous Artefacts

Fauna bites: The animal bites produce confusing changes but bears characteristic teeth marks. The rodent bites are commonly seen in bodies that are stored in a cold room. Drowned bodies may bear the marks of injuries produced by water creatures such as fishes, crabs and may simulate stab wounds.

Postmortem haemorrhage: The finding of the extravasation of blood in the tissues or even pools of blood in the body cavities does not necessarily mean that the haemorrhage was from antemortem trauma. Under normal circumstances blood in the body remains in a fluid state for a time. During this time the postmortem injury may open a blood vessel and lead to postmortem haemorrhage. The significance of blood in the pleural cavities after death has been explained to be substantial

bleeding from wounds inflicted on the chest wall and the lung tissue.

Flattening of the convolutions of brain:

Flattening of the cerebral hemisphere is invariably seen in cases of oedema of the brain caused by pathological lesions. This flattening is generalized. However, regional flattening of the cerebral convolutions is a common postmortem artefact. The parts of the brain in contact with the cranium, particularly the dependent parts show flattening, the occipital lobes showing such flattening more frequently. The longer the body remains in such position and longer the time after death, the more marked is the flattening. To prevent additional artefactual flattening from contact with the container, the brains in order to be fixed should be suspended in 10 % formalin by a hook passed around the basilar arteries for a period of 3 weeks before dissection.

Grooving of unci: It is widely accepted concept that the grooving of the unci is an important accompaniment of genuine oedema of the brain from cerebral lesions. But this finding is an extremely common in normal brains at autopsy, and this artefact should not lead the pathologist to a misinterpretation.

Discolouration of the liver: Blackish brown discolouration of the liver at the site of contact with the large bowel is fairly common. This is a postmortem change, the result of substances passing from the bowel to the liver and depositing sulfides in the adjacent liver tissue. Similarly bile also produces staining of the liver surface.

ARTEFACTS INTRODUCED DURING AUTOPSY

In the head one of the commonest artefacts is the introduction of air bubbles into the vessels at the top of the brain. In the majority of cases while the dura is being pulled in the sagittal line, air gains access to the blood vessels. Likewise postmortem introduction of air into the veins of the neck during the reflection of the skin is also very common. In cases of suspected air embolism following

attempts at criminal abortion, artefactual presence of air bubbles in the vessels of the brain and neck may initiate confusion; the diagnosis of air embolism may be erroneously entertained.

While sawing of the vault of skull during the autopsy, partial sawing and the forceful pull of the cap sometimes leads to the fractures of the skull. In the already fractured skull, the antemortem fractures may be extended or additional fractures created. Postmortem tears of the midbrain may be caused during the removal of the brain from the cranium. Handling of the organs and incisions of the vessels during routine postmortem examinations often result in the extravasation of the blood into the tissues, particularly into the neck. Recognition of autopsy artefacts in cervical tissues is of special importance because they simulate antemortem bruises seen in cases of throttling and strangulation. Therefore dissection of the neck should be meticulous avoiding the artefacts from seepage of blood from the neck vessels.

Toxicological Artefacts

Some of the toxicological artefacts may be introduced during the autopsy caused by:

Faulty Technique in Collecting Samples:

Blood samples are sometimes drawn from the heart with a long needle and this may result in contamination of sample with gastric contents and tests particularly for alcohol may be misleading. In traumatic deaths such as those due to automobile accidents, the stomach may rupture and lead to the added likelihood of contamination. Also, alcohol is known to diffuse in significant amounts from the stomach to the pleural and pericardial cavities. During an attempt to draw blood from the heart, pericardial and pleural fluid may be withdrawn giving unreliable results.

Faulty techniques in storage: Artefacts may be introduced by cutting the organs with instruments contaminated by stomach contents or by putting the samples of tissue in contaminated containers. It is a common practice to store

portions of several organs in one container. Such a practice is apt to cause alterations in true levels of the poisons because the diffusion or drainage of the blood or fluid from one organ to another may lead to an altered concentration of the poison.

Use of preservatives: The use of the preservatives can also add to the artefacts. It should be emphasized that preservatives such as EDTA (ethylene-diamine tetra-acetic acid), formalin, heparin and methanamine give a positive test for

methanol and these should not be used as anticoagulants for blood if an analysis for methanol is required.

Long Questions

1. What do you mean by artefacts. Discuss the different artefacts that can be found during autopsy.
2. Discuss the artefacts produced between death and autopsy.

Autopsy Procedures and Exhumation

AUTOPSY

Autopsy means examination of a body after death. In India, there is provision for a complete postmortem examination. Every cavity of the body should be examined. Even if the cause of death is evident, other areas and organs of the body to be examined to find out any contributory cause of death. Complete autopsy is necessary to corroborate the evidence of the eyewitnesses and the investigations done by investigating office since a poor autopsy can lead to miscarriage of justice.

A qualified Registered Medical Practitioner should carry out the autopsy. He should himself dissect the body and examine the organs to find out the queries asked by the Investigation Officer, most frequently the cause of death. The Post-mortem attendants and sweepers should be there to assist in the procedure. The term is synonymous with necropsy, thanatopsy and postmortem.

The autopsy is of two types: (i) Medico legal autopsy (ii) Pathological autopsy.

The Medicolegal autopsy is conducted in the Department of forensic medicine. The request comes either from the police or SDM who is the Investigation Officer of the case. The consent of the relatives is not required. The doctor conducts a full and complete postmortem to find out the cause of death and other relevant questions asked by the Investigating Officer.

The **Pathological autopsy** is done by the Department of Pathology and request comes from the relatives, who are the consenting party. In these cases the doctor conducts a partial autopsy of the organ or cavities where the pathology is suspected to find out the cause of death.

The forensic autopsy differs from the pathological autopsy in its objectives as well as relevance. The forensic pathologist conducts the autopsy to determine the cause of death, manner of death (natural, accidental, suicidal or homicidal), the identity of the deceased if unknown and the time of death and/ or injury. The forensic autopsy may also involve collection of evidence from the body that can be subsequently be used to prove or disprove an individual's guilt and confirm or deny his account of how the death occurred.

The forensic autopsy involves not only the actual examination of the body at the autopsy table, but consideration of other aspects that the general pathologist does not consider part of the autopsy such as the crime scene, clothing and toxicological. The forensic pathologist should know the circumstances leading up to and surrounding the death prior to the autopsy. The history of the case should be obtained from the investigating police officer or the Sub-divisional magistrate. At times the relatives or the eye witness may give history.

The scene of crime should when an opportunity arises be examined and documented either with

diagrams or photographs or with both. The statements of the relatives and the witnesses at the scene should be recorded by the police and should be given to the pathologist. The body should be handled a very little at the scene. In cases of violent deaths, paper bags should be secured around the hands so that no trace evidence will be lost. Plastic bags should not be used to cover the hands to avoid condensation in cold room and possible loss of trace evidence when it is moved to warm environment. The body should be wrapped in a clean, white sheet or plastic bag.

The examination of clothing for blood stains and trace evidence and correlating the tears/cuts on the clothing with bodily injuries is an important part of the autopsy. When fingerprints are to be taken for identification purposes, it should be done only after the examination of the hands.

In all gunshot deaths and severely burnt bodies, X-rays should be undertaken. This is especially important in gunshot wounds in which the bullet appears to have exited. This is because the bullet may not have exited but rather only a piece of bullet or bone may have exited. This is common with the semi-jacketed ammunition in which the jacket may remain inside the body and lead core may exit.

Aim and Objectives

The aims and objectives of the postmortem are to:

1. Determine the identification in cases of unknown body.
2. Find out the cause of death.
3. Find out the manner of death whether suicidal, accidental or homicidal.
4. Find out the time since death.
5. In new born babies, determination of viability and cause of death.
6. Preserve the trace evidences and viscera when needed.
7. Reconstruct the accident scene from the examination of injuries as to nature and duration etc.

Permission for Autopsy

A written request must come from the Police Officer, who is the investigation officer. At least the Police Officer of the rank of ASI should be the investigating officer. In cases of murder the Inspector is the investigating officer. In circumstances where the SHO/Inspector is busy and not available, he should get permission from the Deputy Commissioner to depute one junior officer for the same. The inspector should take the charge of the case once he is free. There are certain types of cases where the Magistrate holds an inquest and the request for autopsy should come from the Magistrate.

Preparation for Autopsy

1. The Investigating Officer should fill the performa 'Request for Postmortem' and alongwith other inquest papers should submit the body in the mortuary.
2. Autopsy should be done without delay.
3. The I.O. and relatives whose names are written on inquest papers should identify the body. In cases of unknown bodies, the I.O. is directed to take the photographs and fingerprints of the body and the doctor should note at least five identification marks on the report.
4. Autopsy is conducted in a in the mortuary of a government hospital. At times the autopsy is carried out in the open space, or a covered area in emergency such as at the accident site in cases of mass disasters; air crash or train accident etc.
5. The mortuary should have good lightening, equipments, facility of cold storage and proper drainage system alongwith the manpower.
7. The Forensic Pathologist should take the history from I.O. and should carefully go through the inquest papers.
8. The autopsy is done during the daytime, because of difficulty in appreciating certain

colour changes in injuries and in jaundice etc. in artificial light.

9. No unauthorized persons should be present in the autopsy room. The I.O. may be allowed if required.
10. The autopsy is also carried out in a decomposed, mutilated, fragmented and skeletonized bodies as certain important information still may be found after examination.

Procedure of Autopsy

External Examination

The external description should include the age, sex, race, physique, height, weight and nourishment. Congenital malformations, if present should be noted. A simple listing of the articles found on the body should be noted initially such as a short-sleeve white shirt, or a long-sleeve white shirt, unbuttoned down the front, or a blood stained white T-shirt. In a case of traumatic death with significant alterations of the garments due to trauma, the clothing should be described in further detail. One should then describe the degree and distribution of rigor mortis and postmortem staining. The colour of the eye, the hair and the appearance of the eyes should be noted. Any unusual appearance of the ears, nose, or face for example congenital malformations, scarring or severe acne should be recorded. Note the presence of teeth and or presence of any dental plates. The presence of vomitus in the nostrils and mouth should be noted. Any significant scars, tattoos, moles should be noted for the purpose of identification. If there is any evidence of disease, it should be recorded. All the old injuries should be noted. Evidence of recent medical and/ or surgical intervention should also be recorded.

Evidence of injuries: All recent external as well as internal injuries, whether minor or major should be described along with the age of the lesions in this section, if possible. Gun shot wounds and to a degree stab wounds should be described under the separate sections of external and internal injuries.

In gunshot wounds, if possible each individual wound should be described in its entirety before going on to a second wound. The gunshot wound should be described on the body in inches or centimeters in relation to the top of the head or the sole of the foot and to the right or left of midline. It should then be described in regard to a local landmark, such as the nipple or the umbilicus. The features of the wound that make it an entrance and that determine at what range it was fired should be described, for example abrasion collar, soot or tattooing. Pertinent negatives should also be noted. Then, the course of the bullet through the body should be described. All organs perforated or penetrated by the missile should be noted. It is useful to give an overall description of the missile path through the body in relation to the planes of the body. If the bullet exits, the exit wound should be described in relation to the entrance.

If the bullet is found, one should state where it was found, whether it is intact, deformed, or fragmented, whether it is jacketed, and the approximate caliber. A letter or number should be inscribed on the bullet. The bullet then should be placed in an envelop with the name of the victim, the date, postmortem, the location from which the bullet was recovered, the letter or number assigned to it and the name of the police station.

To describe stab wounds, one should indicate whether the weapon was single or double edged, which edge of the wound is produced by the cutting edge of the knife, the exact dimensions, and an estimation of the depth of the wound track produced. In dozens of knife wounds, it may not be possible to handle each wound separately but it should be described in groups.

Examination of clothing is to be done to locate the defects, whether they correspond to the injuries, the presence of trace evidence such as powder, soot, grease or car paint etc.

Demonstration of Pneumothorax

The various methods to demonstrate pneumothorax are:

1. The skin and subcutaneous tissue are first reflected from the chest wall, by sweeping cuts

through the subcutaneous tissue over the chest wall angling the blade down towards the ribs, being careful not to open the pleural cavity. The intercostal soft tissues should not be punctured and neither the pleural space penetrated as this releases air from underlying pneumothorax. When reflection is completed to mid-axillary line, water is poured into the angle between subcutaneous tissue and the chest wall, and the intercostal tissue below the water line are pierced with the blade to detect an underlying pneumothorax, which may occur following trauma, in patients with chronic obstructive airway disease or asthma. If present, air bubbles will be seen rising through the water

2. Before giving any incisions over the body, a wide bore needle attached to a 50 ml syringe minus the plunger is introduced into the subcutaneous tissue over an intercostal space and the syringe is then filled with water. The needle is pushed slightly deeper to enter the pleural space and the water is observed for the presence of any air bubbles. The process can be repeated on the other side also.
3. The chest X-ray of the body to demonstrate pneumothorax can be undertaken.

Demonstration of Air Embolism

Whenever there is possibility of venous air embolism, the following steps should be performed

1. A plane X-ray of chest should be performed before eviscerating to demonstrate air embolism
2. The retina should be thoroughly examined with an ophthalmoscope for intravascular bubbles by moistening the cornea with isotonic saline to prevent interference from corneal opacity.
3. During dissection of the neck, the large neck veins should be carefully exposed but not opened before the heart is dissected *in situ* to avoid the confusion of air introduction during evisceration.
4. The abdomen is opened in the usual way to closely inspect the inferior vena cava for air bubbles in the lumen through its transparent wall.

5. The sternum is removed by dividing the ribs, being careful not to puncture the pericardial sac, and cutting through the sternum distal to the sternoclavicular joint. The internal mammary vessels should be clamped. The anterior pericardial sac is opened and external epicardial veins are inspected for evidence of intraluminal bubbles. The water is then introduced to fill the pericardial space. The right atrium and ventricle is incised covered in water and inspected for any air bubbles that escape. Alternatively, a water-filled syringe without the plunger is connected to a needle that is inserted into the right ventricle and the chamber of syringe is observed for air bubbles. When the presence of an air embolus is established the vena cava should be clamped and the thoracic and abdominal cavities flooded with water in an attempt to localize the source of embolism. Sometimes the intracardiac gas produced by postmortem bacterial activity may produce the appearance of a counterfeit air embolus.

6. *Pyrogallol test*: 4 ml of a 2% freshly prepared pyrogallol solution is collected into two 10 ml syringes. Four drops of 0.5M sodium hydroxide solution are added to the first syringe and the mixture should turn yellow in the presence of air. Gas is aspirated from the right side of the heart; the needle is removed and replaced with a stopper. The syringe is then shaken and the mixture should turn brown, if air is present. In the absence of air, the solution will remain clear, indicating gas production by bacteria. The second syringe is used as a positive control by including a volume of air with the introduction of sodium hydroxide. This should also turn brown.

The arterial air emboli are even more unusual and usually the result of traumatic injury such as thoracic trauma involving pulmonary veins or following air introduction during cardiopulmonary bypass. A much smaller volume of air is associated with such emboli and are much more difficult to demonstrate. Systemic emboli may be demonstrated by inspecting the intracranial

vessels of the meninges and circle of Willis and then examining under water after clamping the internal carotid and basilar arteries.

Internal Examination

In India due to provision of complete autopsy, the whole body is to be examined. The following types of incision are usually given depending upon the nature of the case, to open the body. The incisions given for autopsy (Fig. 8.1):

1. *I shaped incision*: It extends from the chin straight down to the symphysis passing either on the left or right side of the umbilicus. The umbilicus is spared because of its dense fibrous tissue, which is difficult to stitch.

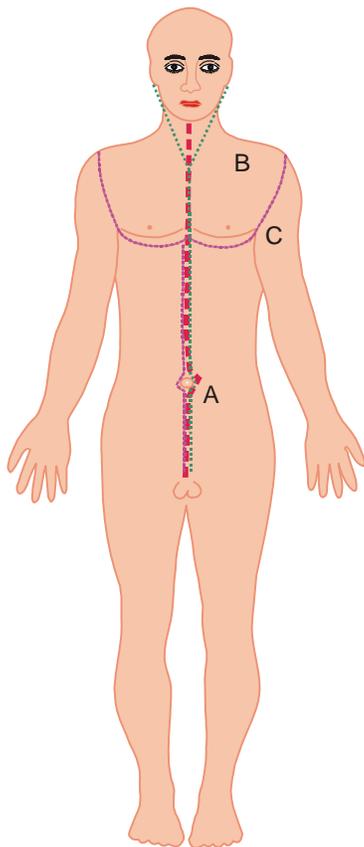


Figure 8.1: Various incisions over the body: A = I shaped, B = modified Y shaped and C = Y shaped

2. *'Y' shaped incision*: The incision starts at a point close to the acromial process on both sides. It extends down below the mammary line up to the xiphoid process from where it extends down up to symphysis pubis on either side of umbilicus.
3. *Modified 'Y' shaped*: An incision is made from suprasternal notch in midline up to symphysis pubis by the side of umbilicus. It extends from suprasternal notch to the middle of clavicle on both sides extending up to side of neck behind the ear till mastoid process.

The choice of opening the skull or other body cavity depends upon the doctor doing the autopsy.

The major organ systems as well as organ cavities are systemically described in this section. The various organ systems and body cavities to be described are the head, neck, thoracic, abdominal and pelvic cavity as well as the biliary tract, pancreas, adrenals, urinary tract, reproductive tract and musculoskeletal system when the need arises.

Principles of Autopsy Dissection Techniques

The autopsy techniques used in various centres and hospitals differ very little. Therefore the pathologist can depend on the method he masters best. Autopsy techniques may vary in the order in which the organs are removed, in the planes and lines of sectioning and most importantly in the method of removal of organs in the body.

1. *R. Virchow's technique*: In this method organs are removed one by one. First the cranial cavity is exposed and from the back, the spinal cord, followed by the thoracic, cervical and abdominal organs. This method has been used most widely. The advantages of this technique that each organ can be studied in detail. However, the anatomico-pathological relationships of organs are not preserved thus cannot be studied.
2. *C. Rokitansky's technique*: This technique involves *in situ* dissection in part combined with *en block* removal. This technique is commonly preferred whenever the pathologist wants to limit

the spread of infection such as HIV, hepatitis B etc. The disadvantage of this method is that the organs cannot be studied in detail.

3. *M. Letulle's technique*: In this method thoracic, cervical, abdominal and pelvic organs are removed en masse. But subsequently dissected as organ block. This technique is the best technique for routine inspection and preservation of connections between organ and organ systems. Also the body can be made available to the undertaker in less than 30 minutes without having to rush the dissection. The organ blocks can then be studied in detail. But the organ mass is often awkward to handle.
4. *A. Ghon's technique*: In this method the thoracic, cervical, abdominal organs and the urogenital system are removed as organ blocks. But now a days the modification of this technique is used where the organs are removed enblock.

The method of evisceration should be the one with which the pathologist is most comfortable that is removing thoracic and abdominal organ blocks prior to dissection or removing each organ in sequence. Many crucial observations can be made only during evisceration.

All the organs should be weighed and a brief description of the organs with pertinent negatives should be recorded. With the pancreas, adrenals, and spleen, if there are no positive findings, use of the term 'unremarkable' as the sole description is acceptable. The term 'normal' should not be used as organs are rarely 'normal' whatever that is. The microscopic slides should be made when indicated. Samples of tissue from all major organs should be saved, but microscopic slides are often not needed in forensic cases, especially in trauma cases.

The samples and viscera preserved for toxicological analyses should be noted. Finally, the opinion should be given describing briefly the cause of death in language as simple as possible.

Detailed Examination of the Neck

In asphyxial deaths, especially in hanging and strangulation, it is advisable to open the skull first

and brain is taken out to drain out the blood from the neck so that the neck structures can be examined in a relatively bloodless field thus avoiding artefactual haemorrhages. The chest organs can be removed prior to neck dissection avoiding injuring the neck veins.

The modified Y-shaped incision will permit a thorough examination of the anterior neck organs and removal of the tongue. The primary midline (I-shaped) incision can be extended superiorly in one of the following ways; (i) a straight incision in front of the trachea, (ii) a bilateral extension of the primary midline incision along the anterior border of the clavicles to the skin in front of the acromion process and (iii) also a bilateral incision extending the primary again along the anterior border of the clavicles but moving superiorly towards the tragus along the lateral side of the neck, ending just behind the ears. If air embolism is a possibility, the neck dissection should be altered, being careful not to injure the large neck veins. The skin and superficial tissues of the neck should be reflected to expose the underlying structures by grasping and retracting the cut border of the skin using the fingers or non-toothed forceps and making gentle horizontal slices along the dermo-subcutaneous tissue plane.

After retracting skin and muscles of the anterior chest, a pleural window should be created to detect pneumothorax by scraping the intercostals muscle off the external aspect of the parietal pleura. This should be done on both sides of the anterior aspect of the chest, usually near the third ribs. In cases of third and fourth degree burns, it is usually necessary to make a midline incision to the chin because the tissue is contracted and indurated.

Dissection of anterior neck structures:

Layerwise examination of the anterior neck structures is desirable in all cases, and is accompanied by sequential *in situ* photography in cases of suspected strangulation. The platysma muscle is inspected for evidence of bruising. The sternocleidomastoid muscles are left intact and the external jugular veins are then examined. The underlying neck muscles are then reflected in

layers. First the sternal head of the sternocleidomastoid muscles is divided from the manubrium and then the more lateral clavicular head is detached. These are reflected laterally and the suprahyoid and infrahyoid muscle groups are then examined being careful not to damage any adjacent vessels and produce a false impression of significant haemorrhage. The carotid sheath containing carotid arteries, internal jugular veins and the vagus nerve is exposed after the omohyoid is reflected. The contained structures are gently mobilized and inspected for evidence of haemorrhage. All the anterior neck tissues can now be separated dividing all the structures at the thoracic inlet or further dissection can follow removal of these structures with the thoracic contents.

Dissection of posterior neck structures: Posterior neck dissection is necessary to rule out craniocervical derangement in cases of suspected suffocation in traffic accidents and is recommended in all infant deaths occurring outside the hospital. Layerwise examination of posterior neck structures is required for traffic fatalities in which there is sufficient trauma elsewhere to account for the death, or in which there is unexplained laceration of brainstem, or haemorrhage in the paravertebral fascia. The prevertebral fascia can be examined for evidence of traumatic injury, such as presence of crepitus. The fascia is then reflected from the underlying bone. The body is turned over and the superficial tissues including ligamentum nuchae is reflected from the occipital region inferiorly to the base of the neck, in order to expose the underlying soft tissue.

Examination of the Heart

The sternum is removed and then the pericardial blood clots, if present should be weighed. Normal hearts and most hearts with acquired disease can be excised separately.

After removal of the blood clots, the heart is weighed. The heart may be of normal size or enlarged (cardiomegaly) due to hypertrophy or

dilatation, or both and can involve one or more chambers. The heart is normally conical shaped but it may be globoid or irregular, as with ventricular aneurysm and one or more chambers may be abnormal in shape. The endocardium is examined for evidence of fibrosis and any evidence of premortem thrombus. The colour of the subepicardial myocardium may be gray with an old infarct, pale with chronic anaemia, and mottled or haemorrhagic, with an acute infarct or rupture. Left ventricular consistency can be firm due to hypertrophy, fibrosis, amyloidosis, calcification or rigor mortis; or soft due to acute myocardial infarction, myocarditis, dilated cardiomyopathy or decomposition.

Examination of myocardium: The myocardium is examined for fibrosis or recent infarct. The myocardial infarct is easily identifiable when it is of more than 12 hours. Between 12-24 hours, the infarct is dusky and shows hemorrhagic discoloration. At 1-4 days, the infarct is more obvious and shows a mottled yellow and red pattern. The infarct is more homogenous and is at first yellow than white in colour after the passage of five days. The size of the infarct, its location, the extent whether it is sub-endocardial or transmural and its correlation with any arterial disease is to be noted.

Evidence of ventricular hypertrophy:

1. Thickness of both ventricular walls at a point about one cm below atrio-ventricular valve is measured. However, any dilatation will mask the hypertrophy. The upper limits for thickness of ventricular wall is 1.5 cm for left ventricle, 0.5 cm for right ventricle and 0.2 cm for the atrial muscle.
2. Left ventricle and septal wall are separated from the right ventricle and then weighed and the ratio is obtained.

Examination of valve: The circumference of the valve is measured with a ruler or the length of the string. The circumference of mitral valve is 8-10.5 (10 cm) and admits two fingers; tricuspid valve is 10-12.5 (12 cm); aortic valve is 6-8 cm (7.5 cm)

and pulmonary valve is 7-9 cm (8.5 cm). The decrease in circumference denotes stenosis whereas increased circumference could be due to regurgitation and when the valves are incompetent. The abnormality in the measurement should always be accompanied by scarring, fusion or calcification of the valves otherwise little importance should be given to abnormal measurement.

Evaluation of coronary arteries: Coronary arteries should be inspected for calcification and tortuosity. In subjects younger than 30 year, in which the cause of death is non-cardiac, the coronary arteries may be opened longitudinally. Otherwise, the vessels should be cut in cross-section at 3-5mm intervals. Calcified vessels that cannot be readily cut with a scalpel should be stripped off the heart and decalcified for at least 24 hour before cutting. To grade coronary obstruction, a four-point system is applied, by 25% increments of narrowing in cross sectional area. A grade-4 lesion indicates stenosis of at least 75% of the coronary artery and is considered as severe, whereas a grade-4 lesion of 90% represents **critical stenosis**. As a rule, grade-4 lesions should be documented microscopically. Depending on the number of major epicardial vessels with grade-4 lesions, a heart may have severe 1-vessel, 2-vessel, or 3-vessel disease, and its coexistence with grade-4 disease in the other three coronary arteries represents severe 4-vessel disease.

Dissection of the Heart: Only two methods are practically used for routine diagnostic pathology, these are the inflow-outflow method and the short axis technique.

1. *Inflow-outflow method or following the direction of blood flow:* The technique is suitable primarily for normal hearts and the atrium is opened first on each of the side and then the ventricle is opened along its inflow and outflow tracts. The valves are cut between their commissures. Using scissors, the initial cut is made from the inferior vena cava to the right atrial appendage, sparing the superior vena cava with the region

of the sinus node. The right ventricular inflow tract is opened with a knife or scissors from the right atrium, through the posterior tricuspid leaflet, running parallel to and about 1 cm from the posterior ventricular septum. The outflow tract is opened in a similar fashion, about 1 cm from the anterior ventricular septum extending through the pulmonary cusp and into the main pulmonary artery. The left atrium is opened with scissors between the right and left upper pulmonary veins and then between the upper and lower veins on each side. The incision can be extended into the left atrial appendage to assess for mural thrombus. The left ventricular inflow tract is opened with a long knife along the inferolateral aspect through the left atrial wall near its appendage, through the mid-portion of posterior mitral leaflet, between the two mitral papillary muscles and then through the apex. The outflow cut travels parallel to the anterior ventricular septum and about one cm from it. This curved cut is best accomplished with a scalpel, taking care not to cut into either the anterior mitral leaflet or the ventricular septum. Scissors can be used to extend the cut across the left aortic cusp and into the ascending aorta, to one side or other of the left coronary ostium.

2. *Short axis or ventricular slicing method:* This method is of choice to evaluate ischemic heart disease as well as any other cardiac condition as the slices expose the largest surface area of myocardium. They correspond to the short-axis plane produced clinically by the two-dimensional echocardiography. The flat diaphragmatic surface of the heart is placed on a paper towel to prevent slippage and then cuts 1.0-1.5 cm thick are made with a sharp knife, parallel to the atrioventricular groove. One firm slice should be used, or two slices in the same direction, avoiding sawing motions that leave hesitation marks. Each slice is viewed from the apex towards the base. The basal third of the ventricle is left attached to the atria. The basal portion is then opened according to the inflow-outflow method.

3. *Other tomographic methods of dissection of heart:* For teaching purposes, the short-axis, long-axis and four chamber planes are ideally suited for demonstrating cardiac pathology.

- i. *Long axis method:* The plane for long-axis method is best demarcated with three straight pins before making the cut. The first pin is placed in the cardiac apex, the second in the right aortic sinus adjacent to the coronary ostium and the third near the mitral valve annulus, between the right and left pulmonary veins. The heart can then be cut along this plane, from the apex towards the base, passing through both the mitral and aortic valves.
- ii. *Four-chamber method:* In this method, the heart is divided into two portions, both of which show all four chambers. Using a long knife and beginning at the cardiac apex, a cut is extended through the acute margin of the right ventricle, the obtuse margin of the left ventricle and the ventricular septum. The upper half is then opened along the ventricular outflow tracts according to the inflow-outflow method.
- iii. *Base of heart method:* This method displays all four valves intact at the cardiac base and is ideal for demonstrating the relationships between the valves themselves and between the valves and adjacent coronary arteries and the atrioventricular conduction system.
- iv. *The window method:* This method is used for the preparation of dry museum specimens using paraffin or other material or plastination. Heart should be perfusion fixed. Windows of various sizes can be removed from the chambers or great vessels with scalpel. The tissue blocks can then be used for histologic study.
- v. *Unrolling method:* The technique is used for demonstrating opacified coronary arteries in a single plane. Following postmortem coronary angiography, the ventricular septum and free walls are unrolled by any

of the three techniques; Schlesinger technique, Lumb and Hardey technique and the Rodriguez and Reiner technique. But the latter is the simplest technique of the three. The unrolling method results in considerable mutilation of the heart and is best reserved for research purposes.

- vi. *Partitioning method:* Each ventricle is weighed separately for detailed assessment of ventricular hypertrophy. The epicardial fat and coronary vessels are stripped from the specimen then atria and great arteries are removed. Finally ventricular free walls are separated from the ventricular septum, the weight of each segment is now compared with the normal values. Biventricular hypertrophy may produce an abnormal ratio.

Normal ratio of LV + S: RV is 2.3-3.3:1

In left ventricular hypertrophy, this ratio is more than 3.6:1 or left ventricle with septum is more than 225 gm whereas in right ventricular hypertrophy, the ratio is less than 2:1 or right ventricle is more than 80 Gms.

- vii. *Examination of the conduction system:* This conduction system is examined if pathology within the conduction system is suspected. It is largely a histological process and three tissue blocks containing SA node, AV node and AV bundles are made.

Examination of the Lungs

The lung is weighed prior to the dissection. The normal lung weighs 250-400 grams each but there is a considerable variation depending on the built and sex of the individual. The weight of the lung increases with severe cardiac failure or with diffuse lung pathology. The internal examination of lung can be done by:

1. *Dissection of the airways:* Airways are dissected using a pair of Scissors from the medial to the lateral including all the lobes and segments

opening along the branches as they are documented. The airway caliber, mucosal surface and parenchymal appearance is examined for fibrosis, consolidation, tumours, scars or cavities. Old tuberculous cavities and fungal balls can be assessed. Chronic obstructive airway disease can be assessed by how peripherally airways can be opened. The examination of airways is of considerable importance in drowning, choking and burn deaths.

2. The pulmonary vessels can be dissected to look for the emboli and atheroma.
3. By horizontal slicing of the lung through each lobe with brain knife and to inspect the parenchyma for haemorrhages, abscesses and consolidations.

Examination of Liver

The liver varies in weight from 1300-1500 gms. The liver may be enlarged with inflammations and neoplasms and shrunken with cirrhosis. A series of parallel vertical slices 1-2 cm apart from one side to the other are made to examine the liver parenchyma for haemorrhages, lacerations or abscesses

Examination of Brain in Adults

Incision of scalp: First the head should be elevated using the head rest. The hair is then parted with a comb along an imaginary coronal plane connecting one mastoid over the other over the convexity. The incision is then given on one side just behind the ear lobe, as low as possible and is extended to a comparable level on the other side cutting whole thickness of the scalp using the scalpel blade (Fig. 8.2). The anterior and posterior halves of the scalp are then reflected forwards and backwards respectively by short undercutting of the scalp and using a dry towel draped over the scalp edges to facilitate further reflection. The anterior flap is reflected to above 12 cm above the supraorbital ridge.

Removal of skull cap: The cranium is best opened using an oscillating saw within a protective device

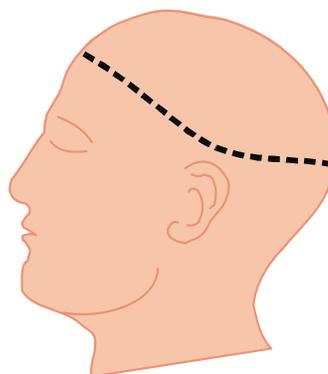


Figure 8.2: Lines for removal of scalp

such as plastic bag for collection of dust. The temporalis muscle should be cut with a sharp knife clearing it from the path of blade of the saw. The frontal point of sawing should start approximately two finger breadths above the supraorbital ridge and then the head is turned to the opposite side and sawing extended to the temporal point that is at the top of ear in its natural position. Finally the occipital area that is approximately two finger breadths above the external occipital protuberance is sawed. The oscillating blade should be moved from side to side during cutting to avoid deep penetration in a given area. Ideally sawing should be stopped just short of cutting through the inner table of skull that can be further detached with mallet and chisel. A hand inserted between the skull and the dura helps the blunt separation of these while the other hand is pulling the skullcap. If the dura adheres too firmly to the skull, it can be incised along the lines of the sawing and the anterior attachment of the falx to the skull can be cut between the frontal lobes. The posterior portion of the falx can be cut from the inside after the skullcap is fully reflected. Leaving the dura and underlying leptomeninges intact allows viewing the brain with overlying cerebrospinal fluid still in subarachnoid space. To obtain this view, after removal of the skullcap, the dura must be cut with a pair of scissors along the line of sawing and then reflected. The dura is then peeled off the

skullcap. The superior sagittal sinus may be opened with a pair of scissors at this time. The dorsal dural flaps on both the sides can be removed easily from the brain by severing the bridging veins but in the presence of epidural or subdural haemorrhage or neoplasia, the dural flaps are left attached to the dorsal vein and can then be sectioned together (Fig. 8.3 and 8.4).

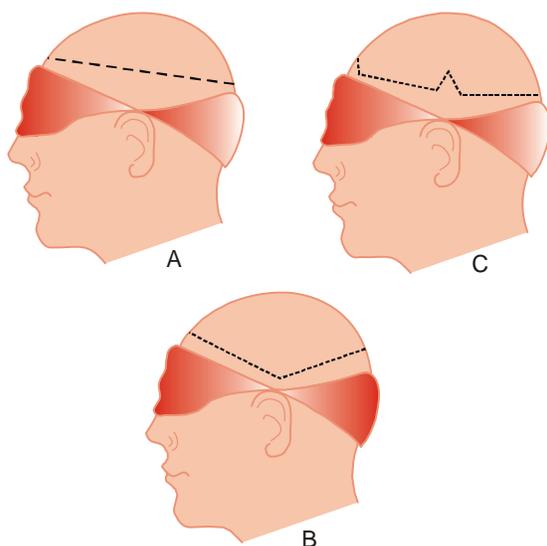


Figure 8.3: Methods of Removal of Skull cap

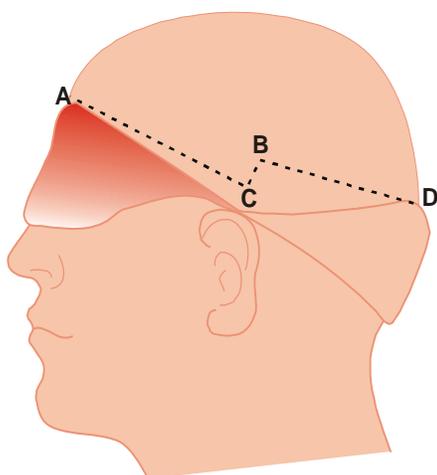


Figure 8.4: Lines along which saw cuts are made for skull cap removal

Removal of Brain in Adults

The brain is removed by raising the frontal lobes gently and tearing the olfactory bulbs and tracts away from the cribriform plates. The optic nerves are then cut as they enter the optic foramina. Under its own weight, the brain is allowed to fall away from the floor of the anterior fossa, supported by the palm of the other hand. The pituitary stalk and internal carotid arteries are then cut as they enter the cranial cavity. Third, fourth, fifth and sixth cranial nerves and subdural communicating veins are also severed close to the base of skull. The attachment of tentorium along the petrous ridge is cut on either side with curved scissors while the brain is prevented from dropping backwards and causing stretch tears in cerebral peduncles. Now the VII, VIII, IX, X, XI and XII cranial nerves are cut and the vertebral arteries are severed with scissors as they emerge in cranial cavities. The cervical part of spinal cord is cut using curved scissors as caudally as possible and avoiding too oblique plane. The brain is reflected back further by using the support of hand to deliver the brain stem and cerebellum from the posterior fossa without causing excessive stretching at the rostral brain stem level. The brain is pulled away from the base of skull after cutting the lateral attachment of the tentorium to the petrous bone along with the pineal body.

Fixation of the Brain

The best routine fixative that allows the widest choice of stains for the nervous tissue is formalin, usually as a 10% solution. In fetuses and infants, the addition of acetic acid to the fixative solution appears to be helpful. It increases the specific gravity of the fixative and allows the brain to float in the solution; it also makes the tissue firmer without altering its histological characteristics. Fixation can be done by immersion and perfusion methods. For detailed anatomic studies, it is best to fix the brain specimen in a large amount of freshly prepared 10% formalin solution. The plastic buckets for holding 8 L can be used to suspend

the brain to prevent distortion during fixation by passing a thread underneath the basilar artery in front of the pons. A thread can be passed under the internal carotid artery or middle cerebral arteries on both the sides, provided that no pathological lesions are suspected in these regions. Alternatively, the dorsal dura can be used as an anchoring point. However, suspension from blood vessels deforms the parenchyma less than dural suspension. The ends of the threads are tied to the attachments of the bucket handle, care being taken not to allow the specimen to touch the bottom or sides of the bucket. Formalin solution should be replaced within the first 24 hours but this is not mandatory if a large amount of fixative is used. If the fixative becomes bloody, is to be replaced with fresh solution that prevents undue discolouration of the specimen. About 10-14 days are required for satisfactory fixation. If the brain is dissected earlier, the central portion may remain pink.

The brain can be perfused with fixative through the arterial stumps before further fixation by immersion. This shortens the fixation time and ensures adequate fixation of deeper portions of the brain. When it is necessary to dissect the brain at the time of autopsy, this preliminary fixation makes tissue further, facilitates dissection and decreases tissue wrinkling. For the perfusion, injection of 150ml of isotonic solution followed by 10% formalin manually with a syringe connected to a simple tubing system causes least problems.

Dissection of Fresh Brains

In adults the examination of brain lesions in terms of anatomic correlations with clinical symptoms can be achieved when the brain is sectioned after adequate fixation. At times the brain must be dissected in fresh state when microbiological and chemical investigations are important or when immediate diagnosis is necessary. Three to four coronal cuts through cerebral hemispheres are given leaving anatomic structure basal ganglia and upper brainstem undisturbed (Fig. 8.5A). This reveals the presence of large lesions directly or indirectly by distortion of the ventricular system.

The sections can then be made into primary slices of brain tissue to expose hidden but suspected lesions. The brainstem can be severed and cut into infratentorial structure and one horizontal cut through these structures is sufficient for preliminary examination. With fresh dissection of the brain, small lesions are easily missed and subtle lesions such as an early infarct can be overlooked. Every brain should be reexamined with new dissection after adequate fixation. Preliminary perfusion or cooling of the brain in a contoured support in refrigerator in about 30 minutes will increase the consistency of the brain and make regular dissection of the brain easier. The brain suspected of harbouring diffuse, roughly symmetric lesions as in cases of metabolic diseases or widespread infections may be bisected along the sagittal plane. One half may be submitted for chemical analysis or for microbiological examination while the other half is to be kept and later sections are taken for histological examination (Fig. 8.5).

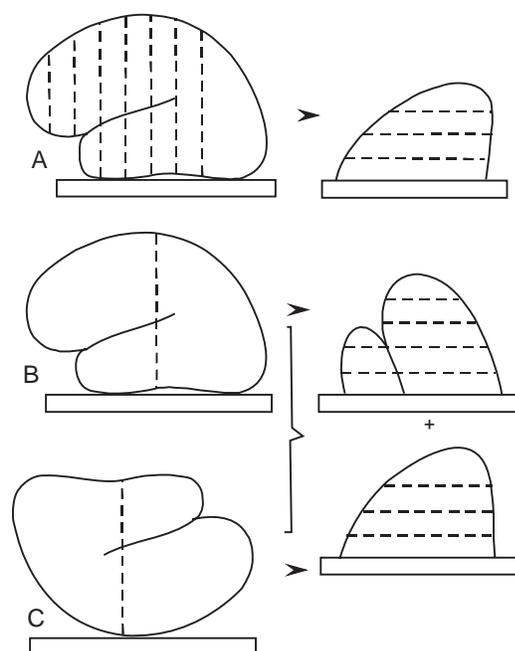


Figure 8.5: Coronal dissection methods for the cerebrum

Dissection of Fixed Brain

The external surface of the brain is carefully examined and the arteries at the base may be exposed through the tears made into arachnoid membrane. The pathologic conditions like thrombosis, embolism or aneurysm are observed.

After adequate external examination the next step is to separate the cerebellum and brain stem from the cerebral hemisphere.

The brain is placed right side up that gives more stability to the specimen. The dissection may be carried out with the frontal lobes by slicing method. The preferred thickness is about 1 cm and each new cut surface is examined before a new cut is made. The slices are displayed on the autopsy table on the right side of the doctor. This corresponds to viewing one's own brain from behind. There should be no overlapping of the slides displayed.

The cerebellum can be dissected on the horizontal plane on planes perpendicular to the folial orientation, the latter method gives the best histological orientation of the structures. The brain stem is sectioned perpendicular to its axis (Fig. 8.6).

Pediatric Autopsy

There are various pediatric autopsy techniques being used by the pathologists. The external examination particularly of the newborn is important. The pathologist should concentrate on the search for malformations such as cleft palate, choanal atresia, stenosis and atresia of the anus and vagina. Face, ears and hands should be properly examined to look for characteristic changes in Down's syndrome and renal agenesis. The placenta and umbilical cord must be studied in all these autopsies. The esophageal hiatus should be examined *in situ*. In this age group, the skull is often difficult to separate from the dura as in adults. **Beneke's technique** is used to open the skull when the sutures are not closed and the skull bones are soft. After the scalp is reflected as in adults, the cranium and dura on both the sides are cut with blunt scissors starting at the lateral edge of the anterior fontanelle extending the incisions as shown in Figure 8.7 along the midline and the lateral sides of the skull. The midline strip about 1 cm wide containing the superior sagittal sinus and the falx is left and also an intact area in the temporal squama on either side, which serves as a hinge when the bone is reflected. This method usually results in damage to the brain. The damage can be minimized by

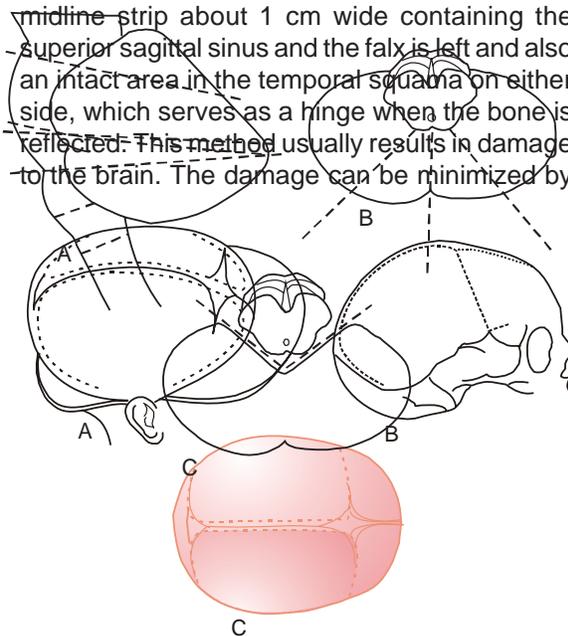


Figure 8.6: Dissection lines for brainstem and cerebellum

Figure 8.7: Method for opening skull in fetus and neonates

infusion of 10% formalin in 70% alcohol through the neck arteries in an early stage of autopsy. In a modification of Beneke's method, the skull is incised lightly along the sutures and at the fontanelles. The scalpel is then reversed and passed under the bones to separate it from underlying dura. The bone flaps are reflected after a small nick is made at the base in each of the bones. The dura is then cut as close to the base of skull as possible. This method protects the friable brain of the infant during its removal. The damage to the brain can further be minimized if the scalp and skull are opened and the falx sectioned with the body in a sitting position and infant's head being supported by the assistant. The tentorium and vein of Galen are transected in this position by separating the parieto-occipital lobes. The tentorium is separated and the body is suspended upside down by the assistant, the brain being cut away from the base of the skull on both the sides in this position thus minimizing the movements of the brain and hence the damage. The brain is not touched during these procedures and when all the attachment are severed, it is allowed to fall free preferably in to the body of water, not on hard surface.

In infants the whole chest cavity can be opened under water in order to demonstrate a pneumothorax. In infants and foetuses **Letulle's technique of en masse** removal is the preferred technique in most cases so that certain rare malformations can be properly preserved for example pulmonary venous connections.

Histological sections should be taken from lungs, liver, kidney, brain, thymus, placenta, umbilical cord and the free margin of falciform ligament.

Routine Sampling of Toxicological Material

The highest concentration of toxicological substances is found in dialysis and lavage fluids. The gastrointestinal tract is not the only route of entry of poisons in the body. Furthermore, if any significant period of 4-6 hours elapses from the time of ingestion until death or treatment, the

poison will have passed out of the stomach. The poison also could have been introduced to mask a homicide. Thus in all the cases a variety of tissues and fluids should be sampled for toxicological analysis. The knowledge of postmortem distribution of a toxic agent in different body fluids and tissues enables the forensic pathologist to distinguish inhalation poisoning from ingestion poisoning. Generally the viscera are preserved in rectified spirit except in poisoning by carbolic acid, alcohol, phosphorus, paraldehyde and acetic acid where it is preserved in saturated solution of common salt. Rectified spirit being costly and not easily available, all the viscera are routinely preserved in saturated solution of salt. Various types of samples that have to be preserved are:

Tissue or fluid	Sample
Stomach	Stomach with all its contents (vomitus and gastric lavage fluid if available)
Intestine	Separately tied portions of intestinal tract with contents (about 30 cm of first part of intestine)
Liver	500 gms or whole organ with gallbladder
Kidneys	One half of each kidney
Spleen	Half or whole organ
Blood	100 ml with preservative and 100 ml without preservative

Blood, bile and urine should be kept refrigerated. Sodium fluoride may be added as a preservative when alcoholic intoxication is suspected (250mg/30ml of fluid). For longer storage periods toxicological material is best kept in deep freeze.

Methods of preservation of various samples:

Blood: After death the serous membrane of the stomach and intestine break down allowing the substance to migrate to the organs of the chest and abdomen as there is a false rise of the substance in the blood level. Ideally before autopsy about 50 ml of sterile blood should be collected from the peripheral (femoral, inguinal or subclavian) veins. After removal of the viscera again 10-15 ml of blood should be collected from these areas. No blood to be collected from the pleural and the peritoneal cavities. If the heart blood is

clotted, the vena cava or the aorta should be punctured. If peripheral blood cannot be obtained samples from the right and left sides of the heart should be collected separately. In cases of drowning 10 ml of blood is removed from each side of the heart for separate sodium, chloride or magnesium determinations. In barbiturate and ethanol poisoning, toxicological examination of portal vein blood may be of interest. Any anticoagulant other than heparin may be used. The blood should be preserved by adding 10 mg of sodium or potassium fluoride and 3 mg of potassium oxalate.

CSF: It is usually collected by lumbar puncture or from the cisterna magna by inserting a long needle. This is to be done during internal examination. More than one blood free sample may be needed. Direct aspiration can be done from the lateral or third ventricle.

Vitreous: Vitreous humour can be used for determination of alcohol and for other toxicological studies, particularly when other body fluids are not readily available. It can be withdrawn using fine needles through the outer canthus of the eye. 1-2 ml can be taken from either eye. The tip of the needle should lie near the centre of the eyeball. Forceful aspiration must be avoided because it may detach retinal cells, which cloud the specimen and give spuriously high potassium values. The vitreous humour may be stored in a refrigerator at 4°C for upto 48 hours.

Bile: Bile is best removed by puncturing the gallbladder in situ.

Gastrointestinal tract: The stomach can be saved with its contents by placing ligatures around the lower end of esophagus and the upper end of duodenum. The organ is then opened to inspect the contents of the stomach and its walls. In poisoning with yellow phosphorus, the stomach has to be opened under nitrogen just before analysis. Several portions of the small and large intestine each about 60 cm long are tied by double ligatures. The separate portions are labeled and submitted unopened for toxicological examination.

Hair: Hair should be pulled from the scalp, not cut. A large sample of about 10 gm should be collected and tied in locks with cotton. This should be done in a manner that identifies the ends where the hair roots are.

Skin: In suspected injections of poisonous substances the skin around the suspected site is excised at a radius of 2-4 cm from the injection site. If a poisonous substance might have been taken up by absorption, the skin is excised in an area where the absorption is thought to have occurred, and a distant preferably a contralateral area.

Bone: In cases of subacute or chronic poisoning by heavy metals mostly arsenic, antimony and thallium about 10 cm of the shaft of femur to be preserved for analysis.

Urine: Urine is best collected in a sterile needle and a syringe through the dome of the diaphragm. The sterilized wall is stretched between two hemostats and then punctured. Urine is of utmost importance for toxicological analysis. Every effort must be made to collect it. The urethra should be tied or clamped and the dome of bladder widely incised between hemostats, which then are used to hold the bladder open. Urine should be preserved in 50 ml bottles.

Uterus: In deaths due to criminal abortion, uterus and appendages along with upper part of vagina are preserved with any chemicals or foreign bodies found in the genital tract to be preserved separately.

Heart: Heart should be preserved in cases of poisoning by cardiac poisons like strychnine and digitalis.

Brain: Brain has to be preserved in poisoning from alkaloids, organophosphorus compounds and volatile organic poisons. Spinal cord should be preserved in strychnine poisoning.

As a routine practice the stomach and intestine are preserved in one glass jar filled with saturated solution of common salt up to 3/4th of its volume. The second jar is used to preserve the liver, spleen and kidney. Blood is kept separately in a bottle.

In another bottle the sample of the preservative is supplied. All the jars and bottles are sealed and kept in a wooden box which is finally sealed and handed over to the I.O. for onward transmission to the laboratory along with the sample seal. The polyethylene bags or containers can be used but the volatile poisons may diffuse through it. In cases where lungs have to be preserved for analysis like that of volatile poisons, nylon bags should be used and the blood should be kept in plastic capped tubes.

Laboratory Investigations

Samples for histopathological examination:

The internal organs like liver, lung, brain etc. are taken for histological examination. It is not being done routinely but if after gross examination some pathology is suspected then 1-2 mm thick tissue can be removed along with normal area. It is preserved in 10% formalin or 95% alcohol. The amount of preservative should be enough not to dry the tissues inside the container.

Bacteriological examination: Blood should be kept in sterile containers using sterile syringe to collect the blood from the right ventricle. The samples from other organs must be collected by using sterile methods for culture of specific tissue. The surface is seared with a hot spatula and the area is punctured with a sterile instrument and the pulp is scraped.

Smears: In deaths due to suspected malaria, the smears from cerebral cortex, spleen and liver may be stained and examined for malarial parasites. Bone marrow and ribs or sternum is used in cases of blood dyscrasias.

Virological examination: A piece of the tissue is collected under sterile conditions and the sample is preserved in 80% glycerol in buffered saline or frozen for further examination.

Second Autopsy

There is no provision in Indian law to have a second autopsy. However, nowadays it is being conducted

either by the order of a district magistrate/senior police officer or by a order from the Court of law, though the instances are rare.

Aim: (i) To find out the cause of death, when it can not be opined by the doctor who had conducted the First autopsy. (ii) When the relatives are not satisfied with the cause of death, they request to higher authorities for a second autopsy done.

Procedure: Second autopsy usually done by a board of doctors, mostly three in number, from different institutions. In these circumstances, the body is usually buried which is to be taken out from the grave. If the body has been kept in the mortuary after the first postmortem, it should be removed to the place of the second postmortem. The board should obtain a request letter/ order of the competent authority along with the inquest papers. If possible the doctor who had conducted the first postmortem should be in the theatre, so that necessary clarification or collaborations can be obtained. The interpretation of the findings of an exhumed body, that has undergone the first autopsy is very difficult because of putrefactive changes and artefacts caused during burial/exhumation, as well as the alterations made during the first autopsy. The findings should be written in detail irrespective of the fact that it correlates or contradicts with the findings during first autopsy.

During the second autopsy some new facts may come up, that may help further investigations. If no new facts are discovered, the second autopsy will correlate the findings of the first autopsy and further doubt and allegations will come to an end.

Negative Autopsy

One of the important objectives of autopsy is to find out the cause of death as well as any contributory cause of death. Nevertheless, when the gross and microscopic findings fail to reveal any apparent cause of death, along with the examination of chemical analysis report and other relevant laboratory investigations, it is termed as negative autopsy. The rate of negative autopsy is

about 5-8% of all the autopsies. Even the best equipped centres of the World have more than 5% negative autopsy rate. The possible reasons are as follows:

Inadequate history: In medicolegal autopsies, the doctors should take adequate history about the incidence. Particular references should be made about the signs and symptoms prior to death as deaths from vagal inhibition, epilepsy, laryngeal spasm, electrocution in water as well as hypersensitivity reactions may not show any external findings.

Lack of proper external examination: Electrical burn marks should be looked especially for the exit wounds as they are most often missed by the pathologist due to lack of experience. In addition, the needle pricks in anaphylaxis are often missed. The concealed wounds may also be missed often.

Improper internal examination: Faulty laboratory examination along with faulty toxicological analysis is also responsible for negative autopsies. The experience and capability of the doctors can be responsible for this.

Insufficient histological examinations: Various microscopic lesions account for death, and are missed due to insufficient histological examination.

Inadequate Pathologist's training: Sudden, unexpected deaths with atherosclerotic narrowing of the coronary arteries without thrombosis or infarction may be a cause of death which is often missed by a hospital pathologist.

Obscure Autopsy

The autopsy is termed as obscure, as there is no definite or obscure cause of death. This type of autopsy is create a lot of confusion to the forensic experts. The causes of obscure autopsy are:

1. **Concealed trauma:** Concussion is a state of transient unconsciousness as a result of blunt force injury to head followed by amnesia and spontaneous recovery. Autopsy may not show

any change, but in some cases petechial hemorrhages are found. Even microscopic examination does not reveal any axonal injury up to 12 hours.

Injury to the cervical spine causing fracture dislocation and injury to the spinal cord can cause instantaneous death without any obvious external injuries.

2. **Cardiac lesions**
 - a. In blunt force injury to heart it may stop functioning without any visible signs.
 - b. Cardiac arrest can occur during or immediately following heavy exercise in which there is increase in heart rate and systolic pressure with progressive ischemia and cardiac arrest.
 - c. Cardiac arrhythmias which, may be precipitated by emotional excitement can cause physiological asystole and may lead to death.
3. **Reflex vagal inhibition:** which can be due to pressure on neck following blows on the larynx, chest, abdomen and genital organs. When death is due to reflex vagal inhibition, there is hardly any findings at autopsy. It may occur during drowning, when the body is subjected to very cold temperature. Any manipulation of external auditory meatus may also lead to death due to vagal inhibition.

Exhumation

It is the lawful digging out of an already buried body from the grave. It is done both in civil and criminal cases. Also, if there is an allegation that the proper autopsy has not been done or the body has been disposed off without any autopsy and foul play is suspected, exhumation can be carried.

Civil Cases (i) Accidental death claim (ii) Indurance (iii) Liability for medical negligence (iv) Workmans compensation claim (v) Inheritance and disputed identity cases.

Criminal Cases (i) Deaths due to criminal abortion (ii) Homicide or suspected homicide when death

has been labeled as otherwise (iii) Suspicious poisoning (ii) Criminal negligence

Rules of exhumation: The written order is obtained from an Executive Magistrate. There is no time limit for exhumation in India. The magistrate along with police officer and medical officer should report at the spot. The spot of burial is to be identified correctly. This can be done with the help of relatives and other persons related with the disposal. Even the person who had made the coffin can be called to identify the coffin. Besides, the location of the grave from some fixed objects like road, tree etc. to be noted. If the map is available, it will be of help. The entries in the register should also be seen. The relatives are allowed to stay throughout the investigations (**S.176(4) CrPC**).

Procedure: After correct identification of the spot, the area should be enclosed from the public. The exhumation is carried out in the early hours so that there is less public as well as the digging out work can be completed during day time.

The identified grave then should be dug carefully up to the depth of 10-15 meters and condition of soil, water content and vegetable grown should be noted down. The grave is further dug up to coffin or corpse and photographs are taken. A sketch may be drawn about the position of coffin and body. At this point it should be identified by the relatives. The plastic sheet or a plank may be lowered down to the level of the corpse then it (corpse) is shifted to plank and taken out carefully to avoid artefacts. No disinfectant should be sprinkled on the body.

In case of skeletonisation of the body, the grave is further dug and the skeleton is lifted to sheet/plank. The soil to be searched for smaller objects like bullets, teeth, hyoid bone, metallic objects. If the body can not be transported or the mortuary is very far off then autopsy can be carried out at the spot. Any fluid and debris in the coffin should be collected.

Injuries if present should be carefully noted. The injuries on soft parts may get distorted or disfigured due to decomposition thus care should

be taken to interpret them correctly. The possibility of artefacts during the process of digging should be excluded.

In suspected poisoning, viscera should be kept for analysis. If organs are converted into mass, then loose masses to be preserved. If nothing is found then hair, soil, teeth and bones should be preserved. About ½ kg of earth to be collected from the top, sides, bottom of the body and kept in dry clean bottles for chemical analysis.

A portion of coffin and burial cloths must be removed in order to exclude any possibility of contamination from external sources.

Though, there is no time limit for exhumation in India. The period is restricted to 10 years in France whereas in Germany it is 30 years.

BUNDLE OF BONES

At times some skeletal remains may be recovered from an open land, ditches, rubbish dumps etc., or a skeleton may exhumed from a temporary grave, a burial ground, or even while new constructions. The bones should be listed and the photographs are preferably taken. The bones should be arranged in an anatomical position of articulation. If some earth, sand, dust etc is sticking to the bones, it is to be cleaned by brushing.

General Examination of Bones: The bones are thoroughly examined as to whether the bones are dry, clean, brittle and whitish in colour with cartilages attached or whether they are moist and humid, yellowish or yellowish brown, with soft tissues still attached and cartilages adhering. The stage of putrefaction of soft tissues should also be noted (Fig. 8.8).

Cleaning the Bones: The soft tissues can be separated by boiling in water containing sodium bicarbonate for about 6-12 hours and then brushing gently.

Examination Proper: Whenever a skeletal remains or a single bone is brought for examination to the forensic expert, the following questions need to be answered:



Figure 8.8: Skeletalized remains

1. **Whether they are actually bones:** Sometimes pieces of stones may be mistaken by the Police officer for bones. For this, proper examination, looking for the protuberances, surfaces, borders etc. is necessary.
2. **Whether the bones are human or animal?** Human skull is commonly mistaken for that of the Chimpanzee, Gorilla or Monkey. The bones can be differentiated from that of an animal from the anatomical configurations. In an animal skull, glabella is more prominent, nasion is deeper, jaw is protruded and the cranium is small in size. In Human pelvis, iliac crest and upper border of symphysis pubis lie in the same plane whereas in animals they lie in different planes due to different postures while walking. Precipitin test being species specific would be helpful when the remnants of blood are still attached to the surface of bones.
3. **Whether they belong to one or more than individuals:** The bones are arranged in anatomical position and if all the bones fit properly and anatomically and there being no disparity between the bones of contralateral side or duplication and all bones belong to same age and sex it suggests that the bones are of same individual.
4. **The race of the person to whom the bones belong?** Race of the individual can be

known from **cephalic index** that is 70-75 when the skull is dolicocephalic in pure Aryans, 76-79 in mesaticephalic skull of Europeans and 80-84 in brachycephalic skull of mongoloids. The other indices used to determine race are (i) Brachial Index (ii) Crural Index (iii) Humero-femoral Index.

5. **The sexing of bones** gives definite results only after puberty. The sex of the person can be determined from the subjective and objective parameters. The subjective parameters include the examination of different bones determining the sex such as the pelvis, skull, long bones, mandible, sacrum, sternum, clavicle etc. When the bones are strong having rough surface, marked muscular marking and well marked prominences and tubercles they belong to males. The average weight of Indian male skeleton is about 4.5kg and in an average Indian female it is about 2.5kg. Children attain half the adult weight by about 12 years in case of boys and under 11 years in case of girls. The pelvis is the best bone used for the purposes of sexing; the accuracy for sexing from different bones according to Krogman is from pelvis (95-96%), skull (90-92%), skull and pelvis (98-99%), long bones (80%).

The objective indices for determining sex include: (i) Sciatic notch index (ii) Ischium pubic index (iii) Pelvic index (iv) Kell index

6. **The age of the person to whom it belongs?** The age of the person can be determined from the ossification and union of the bones state of dentition, closing of sutures, state of calcification of laryngeal cartilages, sternum and hyoid bone, condition of the symphyseal surfaces of the pubic bone, changes in the sacrum and mandible, extent of wear and tear in both the jaws with ageing and changes like bony lipping, osteoporotic and osteoarthritic changes. Epiphyseal union is about two years earlier in females compared to males. The age

changes after 25 years and in old age can be ascertained from the changes in the mandible, vertebra, pubic symphysis and internal bone structure.

7. **The stature of the individual:** The stature is determined fairly accurately using the long bones such as femur, tibia, humerus or radius. The tibia being the best bone used for the purpose. Hepburn's osteometric board is used for the purpose of measuring the lengths of long bones accurately. The length is then divided by the multiplication factors as devised by Pan and Nat. The stature can also be devised by using regression formulae devised by researchers such as Dupertuis and Hadden, Karl Pearson, Trotter and Gleser.
8. **Identifying features:** The person identity can be established from the teeth, any congenital peculiarities, any bony disease or deformities such as caries, osteoarthritis, mal-united fracture, spinal deformities, supernumerary ribs and cervical ribs etc. when the skull is available, the superimposition technique can be used by comparing the skull with the photograph of the individual.
9. **Nature of injury:** The bones should be examined as to whether they are any sharp cuts, or any fractures implying use of blunt weapon or from a vehicle or sometimes they are gnawed by animals when the soft tissues are attached. The charred and blackened bones are suggestive of burns. But in case of intense heat of fires, the bones turn to ashes and are so brittle as to turn to powder when touched. Superficial bones when burnt will show evidence of heat fractures, charring, and cracking, splintering and calcining whereas bones lying embedded amidst thick soft tissues will show molten or guttered condition. A bone when burnt in open fire will become white but when burnt in close fire is black or ash-grey.
10. **Time since death:** The nature and circumstances of burial of the body modifies

the rate of decaying of the bone. If the bones are wet and humid, and have an offensive odour, they are recent. Bodies when exposed to open air gets skeletanised within seven to fourteen days. But when the bones loose their soft tissues, the decaying odour will be lost. Because of ground water seepage, the buried bones may show increase of decrease of mineral contents e.g. calcium phosphate, calcium carbonate etc. depending upon mineral rich content of soil. X-ray defraction studies may give an idea about the mineral content of the bone.

Following putrefaction, the bones loose their organic constituents and thus become light and brittle, dark or dark brown in colour, such changes depend upon manner of burial (with or without coffin), the nature of soil of the grave, age of the individual etc.; usually the time taken for these to occur varies between 3 to 10 years. In case of burial in mass graves of in shallow graves without any coffin, putrefaction will occur very rapidly. Long buried bones may have chalky texture. Bone marrow and periosteum may persist in the bones for several months often after burial. Superficially buried bones may expand or crack within few years by repeated freezing or thawing. When burial is old, the cancellous bone at the metaphysis and epiphysis may get eroded by effects of weather.

In case of fracture, the time may be judged with some accuracy by examining the callus by cutting it longitudinally. As globulin disappears rapidly, precipitin tests on 10 years old samples become negative.

The Dating of Human Bones: Examination of the bones rarely, if ever, permits a precise estimate of the time interval since deposition in the ground. At the same time it is possible and important to decide whether they are 'ancient' or 'modern' bones that the interval is greater or lesser than 50 years. In recent years, a considerable amount of research

has been carried out in an attempt to increase the accuracy of dating skeletal remains. Some of these depend on sophisticated laboratory techniques, such as radio-carbon analysis, which are difficult and expensive to perform. Radio-carbon is essentially a tool for archeologists and its forensic use is limited because of the insignificant fall in the C-14 content of bones during the first century after death. This is the stumbling block for many physical and chemical methods for bone dating, as although old samples (in excess of 100-200 years) can fairly readily be differentiated from recent bones, discrimination between the dates of skeletal remains recent enough to be of interest to law enforcement agencies and coroners, is too poor to be of much practical use.

The environmental conditions are more potent than age in causing progressive degeneration of the bone; even different parts of the same skeleton (and even opposite ends of the same long bone) may be quite different in their chemical and physical properties, if local changes in inhumation such as drainage, are marked. Bones in wet peaty soil may be decalcified and crumble within two decades, yet bones in dry gravel or sand may remain almost pristine for millennia.

Naked eye appearances are very deceptive, but bones with remnants of periosteum, tags of ligament or soft tissue other than adipocere are likely to be less than 5 years old, unless kept in a dry protected place. A 'soapy' texture of the surface, from residual fat, also indicates a date of less than a few decades.

The various laboratory test used for the purpose of dating human remains include the following but results of each test should be interpreted in the light of the others and with due regard to the macroscopic appearances and the fullest information of the place of concealment and any circumstantial evidence.

- i. *Nitrogen content:* New bones contain 4.0-4.5 gm% of nitrogen, derived mostly from the collagenous stroma. After a variable interval following death, usually longer than 60-100 years, this declines. A value of 2.5gm% usually indicates an age of at least 350 years.
- ii. *Amino-acid content:* Amino acid content is estimated by autoanalyser after acid hydrolysis of the residual protein. Up to 20 acids may be found in bones less than a century old. They then decline in number and concentration. Earlier work using thin-layer chromatography suggested that praline and hydroxyproline (constituent of collagen) vanished by about 50 years, but the more sensitive modern methods of analysis do not confirm this.
- iii. *Blood pigments:* Blood remnants may be found up to a century using the most sensitive, though non-specific tests. As benzidine have carcinogenic activity, other tests such as phenolphthalein and leucomalachite green can detect blood only up to 5-10 years, using either bone dust or the periosteal surface as the test area.
- iv. *Fluorescence:* Fluorescence can be seen across the whole freshly sawn surface of a long bone under ultra-violet light for more than a century, but beyond this time, declining fluorescence is seen advancing from both the outer surface and the marrow cavity. The 'sandwich' of fluorescence progressively narrows during the first 50 years and may vanish with 300-500 years.
- v. *Immunological activity:* Eluted extracts of bone when tested against animal antihuman serum gives a visible antibody-antigen reaction, either in cross-over electrophoresis or by passing diffusion in gel. Early work suggested that this persisted for 5-10 years, but recent repetition of the tests indicated that it ceases within months of death.

11. **Cause of death:** Presence of depressed comminuted fracture, cut injury or bullet wounds in the skull, fracture-dislocation of the vertebral column, fracture of ribs, hyoid or any other limb bones etc. will be informative, pointing towards the cause and nature of death.

Metallic poisons like Arsenic, Lead, Antimony, Mercury etc. can be detected in the bones long after death. Arsenic can be recovered even from examination of charred bony fragments. From the type and depth of cut in the bone the nature of the offending weapon can be made out.

Examination of Mutilated Bodies and Fragmentary Remains

Sometimes the mutilated bodies and fragmentary remains of the body such as one limb, part of trunk or only the head, a mass of soft tissue etc. is brought for examination by the investigating police officer. In such cases, not only the body parts mutilated but there are chances of disfiguring the face and dismembering the limbs to destroy the identity in order to hide the crime. The body may also get mutilated by the dogs, rats, jackals, vermins specially when left exposed in open places and also by the fishes when the body is recovered from the water, to such an extent that the body remains unrecognizable even by the near-relatives. The assailants are also likely to also destroy finger tips for fingerprints, tattoo marks, scars, moles and also remove jewellery, clothing and other personal belongings to further make it difficult to identify the person. The body may be burnt or incinerated. Also because of advanced decomposition, the soft tissue and viscera putrefy leaving behind the bare skeleton. The body parts may be deliberately dismembered after death and thrown off in rivers, canals, sewage holes. The body may be put in a vat of corrosive acids or alkalis to get it chemically destroyed as was in Acid bath murder case. The body parts may get mutilated into fragmentary remains beyond recognition in bomb explosions. The questions

listed below should be answered in detail while examining the fragmentary and mutilated body parts.

1. *Whether the remains of human or animal origin?* When the head, trunk or limbs are available, it is easy to specify them to be human or animal but when only soft tissue with no visceral organs is to be examined. The mass of soft tissue without any preservative should be sent to the forensic science laboratory for the precipitin or antiglobulin inhibition test that is a species specific test.
2. *Whether the parts belong to one or different individuals?* When the mutilated parts fit to each other snugly and anatomically with no disparity or duplication, bearing more or less the same skin colour etc, it can be said that all the parts are of the same individual. Bone from different skeletons can be differentiated, when they show different colour emissions on exposing them to short wave ultraviolet lamp.
3. *Do the fragments belong to male or female?* If the head or trunk is available, it may be easy to determine sex from the scalp hairs, beard, moustache, pubic hairs, external genitalia, breasts etc. it is known that prostrate and non-gravid resist putrefaction for a long period. In case of skeletal remains, the characteristic sex features in skull, pelvis, sacrum, femur etc. will be helpful. General configuration, remnants of cosmetics, jewellery, clothing etc. will speak of characteristic feminine features.
4. *The age of the person:* The age can be ascertained from general development, teeth, ossification of bones, union of sutures, calcification of laryngeal cartilages, union of pieces of sternum and sacrum, changes in the symphyseal surface of pubic bone, osteoarthritic changes in the joints, colour of scalp, moustache and beard hair
5. *Stature:* The stature can be determined from the long bones, if any
6. *Race:* Race can be determined from the scalp hair, skin complexion, cephalic index

and other anthropological features in the skull, vertebral column or other body parts.

7. *Establishing identity:* The identity can be established from the finger print, tattoo marks, scars, moles, deformities, congenital abnormalities if any, dental study, recent or old fractures, personal belongings of the deceased if available.
8. *Any marks of habit or occupation:* Occupational marks can help in determining the profession of the deceased.
9. *Manner of separation of parts:* This can be determined by close examination of the margins of the dismembered parts to see, whether these had been cleanly cut, sawn, disarticulated by several strikes at different levels done haphazardly or gnawed through by animals. At times the face and other body parts may be defaced and disfigured to remove the identifying features purposefully. Meticulous examination can only help to differentiate the cut in bones by sharp weapons, or sawing of long bones by sharp weapons, for surgical amputation, from gnawing of dead bones by wild animals, showing no evidence of antemortem reaction.
10. *Time since death:* The probable time since death can be ascertained from the conditions of the soft parts in relation to the stage of putrefaction
11. *Cause of death:* It is easily determined in case of antemortem fatal injuries affecting large blood vessels, vital organs or body parts are there. Other causes are extensive antemortem burns, head injury with fracture of skull, fracture dislocation of cervical vertebra, fracture of hyoid bone or several bones. Sometimes bullet or its fragments and part of the weapon used are recovered from the body that points towards the nature of death. As the soft tissues decompose quickly, the injuries involving these tissues may be easily missed as in the case of neck

tissues when death has been caused by compression of neck.

EMBALMING

The treatment of the dead body with antiseptics and preservatives to prevent putrefaction is called embalming. Embalming causes coagulation of tissue proteins and they get fixed and hardened. It should be done as early as possible after death and for satisfactory results, preferably within 6 hours.

The main constituents of embalming fluid are formalin, sodium borate, sodium citrate, sodium chloride, glycerin etc. dissolved in water. The fluid has to be forced into the arteries for diffusion to occur into the cells and tissues through the capillaries. It can be done by hand/foot pump, stirrup pump, bulb syringe, gravity injector and motorized injectors.

The injection can be continuous or interrupted with a continuous or interrupted drainage. Embalming is arterial when the fluid is injected in the arteries and then cavity embalming is done in which the fluid is injected in the body cavities.

The vessel to be chosen for the arterial embalming should be preferably nearer to the heart for better results. A common method is the 'six point' injection system in which the carotids, axillary and the femoral arteries of both the sides are used for pumping the fluid for the different body regions.

Long Questions

1. Discuss the general procedures to be followed for a medico legal autopsy.
2. What are the aims and objectives of doing the various types of autopsy?

Short Question

1. Negative autopsy
2. Obscure autopsy
4. Preservation for viscera
5. Exhumation.

MULTIPLE CHOICE QUESTIONS

1. **The fluidity of blood after death is due to:**
 - A. Fat embolism
 - B. Fibrinolytic enzymes
 - C. Air embolism
 - D. All of the above
2. **The time limit for exhumation in India is:**
 - A. 10 years
 - B. 20 years
 - C. 30 years
 - D. Unlimited
3. **Which of the following is not a common incision technique for conducting autopsy?**
 - A. 'I' incision
 - B. 'Y' incision
 - C. Modified 'Y' incision
 - D. 'H' incision
4. **Which of the following methods involves removal of individual organs during autopsy?**
 - A. R. Virchow's technique
 - B. C. Rokitansky's technique
 - C. M. Letulle's technique
 - D. A. Ghon's technique
5. **After postmortem the body should be handed over to:**
 - A. Magistrate
 - B. Patient's relatives
 - C. Local Inspector
 - D. Coroner
 - E. Investigating officer
6. **During postmortem doctor is supposed to examine:**
 - A. All parts
 - B. injured parts
 - C. Parts requested by victims' relatives
 - D. Parts mentioned in the initial report
7. **A sample to look for uric crystal (Gouty tophus) would be submitted to the pathology laboratory in:**
 - A. Formalin
 - B. Distilled water
 - C. Alcohol
 - D. Normal saline
8. **Sodium fluoride may be used for preservation of:**
 - A. Cyanide
 - B. Arsenic
 - C. Alcohol
 - D. Urine
9. **Most commonly used preservative is:**
 - A. Formalin
 - B. Distilled water
 - C. H_2SO_4
 - D. Oxalic acid
10. **To preserve specimen, concentration of formalin used is:**
 - A. 1%
 - B. 5%
 - C. 10%
 - D. 40%
11. **Preservative used for toxicological specimen:**
 - A. 20% formalin
 - B. Saturated sodium chloride
 - C. 20% alcohol
 - D. 10% alcohol
12. **Urine containing Bile salts, is preserved in:**
 - A. Formalin
 - B. Rectified spirit
 - C. Saturated sodium chloride solution
 - D. Ethyl alcohol
13. **'Rectified spirit' is contraindicated as preservative in poisoning with:**
 - A. Phenol
 - B. Paraldehyde
 - C. Both of the above
 - D. None of the above

1 B 2 D 3 D 4 A 5 B 6 A 7 A 8 C 9 A 10 C
11 B 12 C 13 B

Crime Scene Investigation

The objectives of crime scene investigation are:

1. To reconstruct the accident.
2. To ascertain the sequence of events.
3. To determine the mode of operation.
4. To disclose the motive.
5. To uncover the property that was stolen.
6. To find out what all the criminals may have done.
7. To recover physical evidence from the scene of crime.

EVIDENCE

Webster defined 'evidence' as something legally submitted to a competent authority as a means of ascertaining the truth of any alleged matter of fact under investigation before it. According to **Section 3 I.E.A.**, 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 enquiry.
2. All documents produced for inspection by the courts.

Types of Evidence

Evidence is of two types: (i) Testimonial and (ii) Physical evidence that may vary from fiber to a building.

1. Physical evidence is very valuable as it can prove that a crime has been committed or establish key elements of a crime, e.g. in cases of alleged rape the victim's torn clothing and

contusion over inner side of thigh are sufficient to prove a case of rape.

2. The evidence can place the suspect in contact with the victim or with the crime scene, such as in case of alleged bestiality. If the animal hair is found on the suspect's clothes that can prove the contact with the animal.
3. The physical evidence can establish the identity of persons associated with the crime, e.g. the finger prints at the crime scene or over any articles present in the crime scene can pinpoint the identity of the criminals.
4. The evidence can exonerate the innocent, can corroborate the victims testimony, such as in a case of alleged rape, if the victim narrates the story alleging that the person lured her to an unknown place and after mixing some sedatives in the cold-drink which she drank and became unconscious was raped. On examination and subsequent chemical analysis of the blood and urine, it has found that she was not given any sedatives. On repeated interrogation she told that she had given a false history as she was trying to falsely implicate the person.
5. A suspect confronted with physical evidence may make admissions or confess such as in a case of murder blood stained clothes were found in the accused possession. On grouping of the blood it has found to be matched with that of the victim after which the accused admitted the crime.

6. It is more reliable than the eye witnesses. After several months of the crime when the eye witnesses are asked in the court of law to narrate the events, they give different versions to the same event but the physical evidences remains the same.
7. The Court's decisions have made physical evidence more important and is expected in criminal cases by courts. As you know, the Court decisions depends more upon the physical evidences than that of the eye witnesses. In the recent years, more and more eye witnesses are turning hostile resulting in injustice during the trial.

Collection and Preservation of Physical Evidences

In any case of trial, the Court will require the proof that the evidence is collected at the crime scene is the same that are being produced in the Court for which the chain of custody is very important. Usually the evidences are kept in a container, tagged and identifying information are written over it such as:

1. Victim's or suspect's name, date and time of collection, case number and type of crime, brief description of the item, name of the person collecting the evidences and the name who has the custody of the item.
2. Storage of the physical evidences has a legal implications. It must be kept in a secured area prior to the transportation to the Court of law and no unauthorised person should have access to it. As a general rule, sufficient samples should be collected; as much as samples are available are to be collected. Control samples are also needed for the comparative study. The physical evidence should be handled as little as possible because too much handling may arbitrate the fingerprints, dislodge the minute evidences like hair, fibers etc. At times forceps, gloves and other instruments may be necessary to handle the evidences. Contamination is a great concern and to be avoided at any cost.

First Officer at the Scene

Crime scenes are dynamic, rapidly changing and the first officer must be concerned with myriad of details. His actions or steps taken lead to a successful investigation /solution of crimes. The first officer's most important duty is to prevent the destruction or diminished utility of potential evidence that may lead to the apprehension of criminal and solution of the crime. Nothing should be changed, destroyed, added or remove to or from the crime scene which may mislead investigators, though the general rule of protecting crime scene can not be applied to every case. It may not be possible to preserve the crime scene in certain crimes because of their locations e.g. when accident occurs on the road with heavy traffic on. Detailed and meticulous note taking should be adhered to. All movements should be calm and deliberate Errors committed at the scene of crime can never be rectified. Every effort should be undertaken to preserve the crime scene as **Locard's exchange principle** states that whenever a body comes in contact with another there is an exchange of material between them.

Recording the Time

The time of examining the scene of crime should be recorded. Precise noting of the time is very important. The time at which the crime was committed, the first officer was called, his arrival at the scene etc. are very important. Track of time spent at the scene indicates the systemic way the things were done.

Entering the Scene Proper

Efforts should be made to observe the details and make notes on such points as doors, windows, lights, shades or shutters. Any odour like that of cigarette, smoke, gas, gun powder or perfume etc should be noted. Signs of activity like meal preparation, dishes in sink etc. date and time indicators like mail, newspaper, stopped clock, spoiled food and position of the articles to be noted

Nothing should be removed unless essential. Position of the body should be noted, along with sketches, photographs and videography. No one should use toilets, eat or drink anything at the scene.

At times, it happens that the victim or relatives attempt to clear up the scene. Such persons would like to conceal something or want that the things should be in proper order. It is often possible to recover physical evidence on damaged items that have been thrown in the trash.

Protecting the Integrity of Scene

The scene should be protected from the public, other officers, press, family members and any other unauthorized access to be banned. For indoor crime scene, the door should be locked or the gate of the premises should be closed. While for the outdoor scene, one stringing rope or a commercially available tape should be used to cordon off the area. Vehicles and police barricades can act as physical barriers to intrusion by unauthorized people. Officers have to play a very active role in protecting the crime scene and extent of measures depends upon case to case. It is important to protect crime scene proper and paths of entry and exit by criminals. If it is a very large area then local trusted citizens can be of help to protect the area. The protective measures at the scene should be taken at the earliest thus preventing the destruction of valuable evidence.

Injured persons at the scene have to be attended immediately. First aid has to be given even if valuable evidence is lost / destroyed. If first aid is not essential then detailed position, photographs and sketches should be made. Hands must be examined for hairs/fibers. Dying declaration should be obtained from the injured while on the way to the hospital. In the hospital clothing to be taken charge of.

ROLE OF FORENSIC PATHOLOGISTS

At this point, the forensic pathologist should be called to the crime scene by the investigating

agencies but it partly depends upon the local custom. At most of the places, it is better for the investigating officer to arrive first and later the doctor should be called to the crime scene. Once the doctor is at the scene, he should first diagnose that the victim is dead. He along with the investigating team will collect the evidences, see the position of the body before transferring them to the mortuary. Examination of the scene is very important for the doctor as while doing the postmortem that can correlate between the two.

Diagnosis of Dead Persons

Diagnosis of dead at the scene is important. The body should be touched and tested for circulation, respiration, light reflex, pupillary reaction and corneal reflex. Sketch or photograph of the stains etc should be taken and then the body is transferred to mortuary with identification tags. Each body should be sent in a separate bag.

When a Suspect is Found at the Scene

When a suspect is found at the scene then the duty of the officer is to protect the scene. When this is not possible leave the scene in charge of a civilian till other officers arrive. The suspect should be searched and removed from the location as he may try to destroy / change the evidence.

Role of the First Officer before the Investigating Team Arrives

1. Writedown the name of the witnesses and other person who has entered the scene and who were at the scene before the arrival of the officer.
2. The basic facts to be established.
3. The suspect and the witnesses should be kept separated
4. The witnesses are instructed not to discuss anything with anybody.
- 5 The crime scene must be protected.

Crime Scene Search

After collecting information about the crime scene, a quick survey should be done and actual crime



Figure 9.1: Body found at the scene with smashing of head with heavy stone



Figure 9.2: Bleeding from the smashed head and face same as in figure

scene search should be done. It can be done in the following ways (Fig.9.3):

- a. Spiral search method:** In this method the search is started from a central point in the scene and then the single investigator walks in an outward spiral covering the whole area.
- b. Grid method:** In this method the whole crime scene is divided into a grid and each of the grids are carefully searched for evidence. Multiple investigators can search the scene.
- c. Strip or line search:** In this method a single investigator starts searching the scene from one end and covers the whole scene in straight lines returning from side to side.
- d. Quadrant or zone search:** In this method the whole area is divided into quadrants and each of the quadrants are searched by one or more investigators. The quadrants can be further subdivided into more quadrants as per convenience.

SPECIFIC CRIME SCENES

Traffic Accidents

Evaluation should be done with a request for ambulance, fire service, forensic experts and traffic control assistance. Tow truck service should be availed to remove the damaged vehicles. First aid must be provided to all the injured.

Protecting the Scene The scene has to be protected by a patrol unit with flashing light warning and traffic flow should be arranged properly.

Witness There are three ways to find out witnesses. One can ask "Did anyone see what happened" Or can say "I like to have witnesses to remain here." One should watch the crowd while giving first aid. Teenagers and ladies are usually the best witnesses

Evidences A sketch should be prepared or a photograph may be taken of the scene. Vehicle debris and broken glass etc should be collected.

Vehicle Removal Tow truck driver is an important member. Vehicles should be removed one by one.

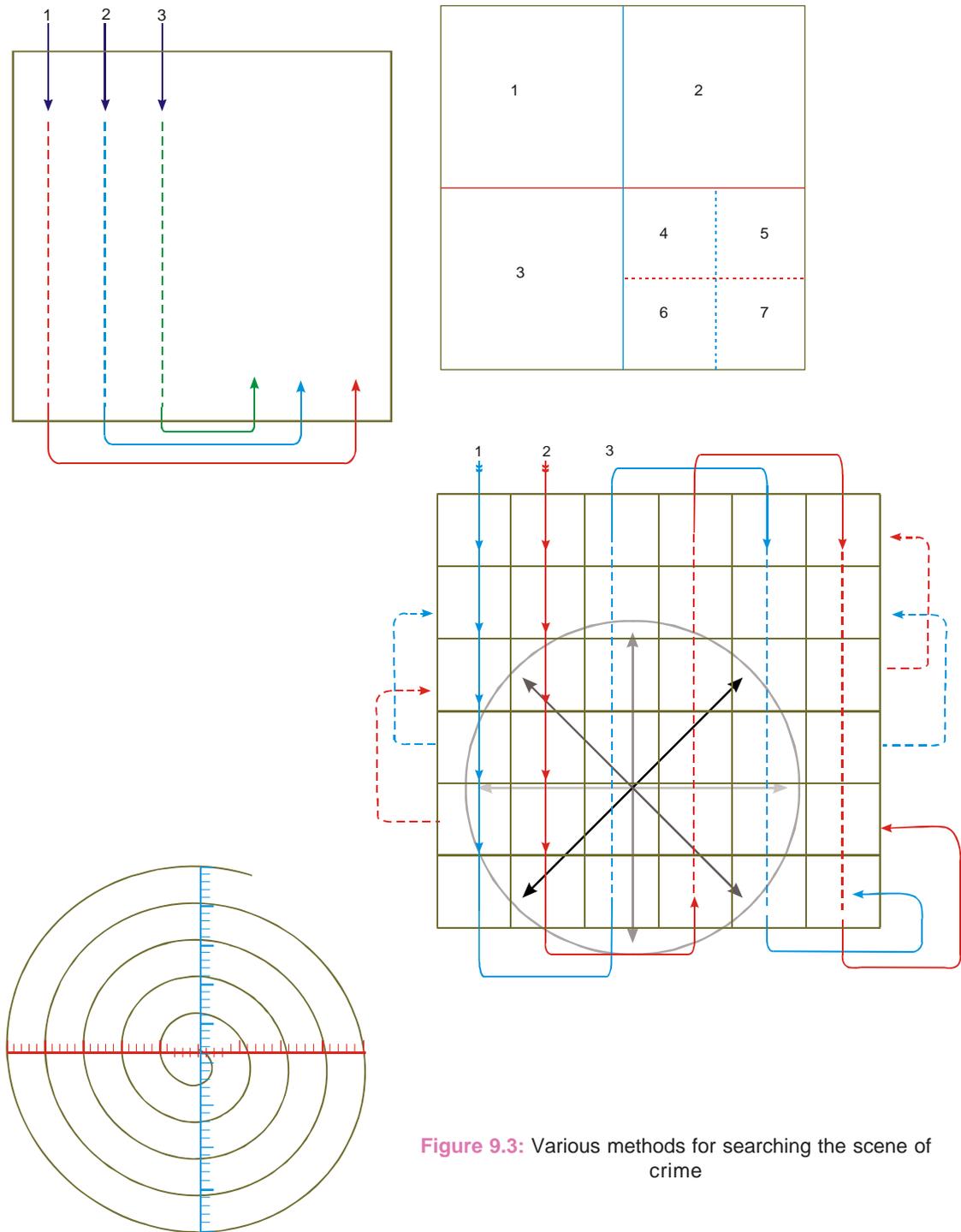


Figure 9.3: Various methods for searching the scene of crime



Figure 9.4: Body found at the site showing decomposition changes



Figure 9.5: Body found lying on the bed early in the morning with no external injuries

Storage Report Drivers / passengers should be taken to hospital. Inventory of the vehicle should be maintained. Only engine key should go to avoid possible allegation of theft. Money and suitcase etc. should be sent with the victim. The roadways should be opened.

Statements The passengers, drivers and public should be interviewed. Questions relevant to the accident must be asked. Statements of witnesses should be recorded. Identity of the witness should be recorded for future reference. To interview a child witness simple words and plain dress should be used rather than police dress.

Arrests Drunken drivers should be arrested. Alcoholic testing should be done.

Reporting Prepare a report and send it to higher authorities.

Collection of Physical Evidence

a. **Skid marks:** It should be identified by the type of mark, origin and termination. Measure with a steel tape and not by pacing. Attempt to

identify the skid marks with tyre of the vehicle. Measure the gaps, skips and deviations as they assist in locating the point of impact. If no skid marks are found write none.

- b. **Graze marks:** The length of grazing is to be measured and direction can be determined. Try to locate the point, grease, oil, water and residue around graze mark. These help in identifying the vehicle.
- c. **Debris:** Identify the type of debris and the location whether over the head lamp glass, radiator water and brake fluid etc.
- d. **Vehicle parts:** The bumpers, fenders, door handles, radio antennas etc. should be examined.

Other Physical Evidences Examination of other physical evidence such as a suitcase falling from the top of the vehicle or fall and breaking of the baggage while a pedestrian is crossing the road when hit by the car, all help in all these establishing the victim and reconstructing the scene of crime.

Burns

The burns can occur at any place. The room may be closed or open. The fire may be outside or any other place. Smell, cooking material, hair condition and sometimes bangles may be broken or weapons recovered from the scene. Pugilistic attitude and heat ruptures should be looked for.

Hanging and Strangulation

The points to be noted are height of suspension, the platform height, mark on resting points/ suspension / removal of the body. The pattern of knots and noose of certain type should be noted and whether the person can hang himself with it or not. Diagrams/sketches/photographs/ videography etc. should be done. Noose should be examined immediately and if it is cut, the pieces are to be recovered and analysed. The ground under and around the hanged person must be examined immediately. Knots in hanging case should never be undone/cut except while life saving



Figure 9.6: Body found at the scene in a case of hanging showing ligature around the neck

measures are undertaken. A case of suicide at times gives a picture of homicide or a murder. On the other hand, there are cases of murder when the body has been hanged to simulate a case of suicidal hanging. In all such cases, it is important that the neck should be carefully examined along with other bodily injuries. The examination of circumstances where the crime has occurred is important such as noting whether the room was bolted from inside or not, other evidences of struggle at the scene.



Figure 9.7: Body found at the site of crime with ligature *in situ*—ligature strangulation

Firearm

In firearm cases, injuries and surroundings must be protected. Finding such as tattooing, blackening, singeing, condition of the clothes should be protected from rain while moving the body from the scene. Hands should be protected by wrapping in polythene and paper bags should be used for collection of evidence.

Deaths by Poisoning

In poisoning deaths, it is very rare for the investigating officer to find the presence of physical evidence at the crime scene that indicates the death was caused due to ingestion of some poisonous substance. Physical evidence located at the crime scene and will be noted on examination of the deceased may sometimes indicate poisoning. In case of death from poisoning the investigation of the scene and examination of witnesses will decide whether it is suicidal, homicidal or accidental. At the scene any vital clue in the form of tablets, powder, residues in bottles of medicines, wrappings, boxes, tubes, ampoules, vials and other containers if found should be preserved. All medicine bottles, tubes, syringes and needles including the empty ones should be preserved and sent for analyses to the forensic science lab. When food poisoning is suspected, the dishes should be collected and packed. Besides, food dishes and remains of food are packed in a clean glass jar that is well sealed. In cases of drug abuse when syringes are found at the scene they should be recovered preventing their contamination. The needle of the syringe is conveniently stuck in a cork to prevent it from breaking. The scene should also be searched for ampoules and vials.

TEAMWORK

Final element in crime scene investigation is teamwork. System was purposefully designed, so that, nobody can operate independently. No person is more important. Each person has vital role to play. Each must feel empowered to do what needs to be done for the sake of justice.

Long Questions

1. What are the objectives and usefulness of crime scene investigations?
2. Discuss the role of first officer at the crime scene.
3. What are the different findings in a crime scene?

Short Questions

1. Methods of crime scene search
2. Role of Forensic pathologist in crime scene.
3. Collection of evidences in crime scene.

MULTIPLE CHOICE QUESTIONS

1. Various methods of crime scene search are:
- | | |
|-------------------------|---------------------|
| A. Spiral search method | C. Strip method |
| B. Grid method | D. Zone method |
| | E. All of the above |

Anaesthetic and Surgical Deaths

Anaesthetics are used for the management of pain in various surgical and operative procedures. Anaesthetics may in themselves result in various adverse effects and sometimes death of the patient. Various surgical procedures carry the risk of life for some of the patients. Anaesthesia may be the sole cause of death in itself and may be a contributory factor in some of the deaths. Every new technique can potentially form a new hazard that is why the lists of mishap that can occur through medical practice are unlimited.

Deaths Associated with Surgical Operation

1. *Those directly caused by the disease or injury for which the operation or anaesthetic was necessitated:* In the advanced stage of some disease, the intervening surgical procedures may be undertaken to save the life of the patient. This procedure causes death of the patient but the disease from which the patient was suffering is given as cause of death. In cases when the patient dies on the operation table itself and is brought out dead from the theatre, the relatives may demand investigations into cause of death. In those cases where the patient is of trauma, and surgical operation is done, it is not possible to separate the relative contribution of the two to the cause of death.
2. *Those caused by a disease or abnormality other than that for which the procedure was being carried out:* When the death was caused by a disease other than that for which procedure was carried out and that was known to exist

before carrying out the surgical procedure, it is for the Pathologist to evaluate whether an operation is necessary. However, if clinicians did not suspect the disease and neither the facility of specialized diagnosis was available, the death is excusable. When common conditions such as hypertension, ischemic heart diseases etc. are present, they should be properly diagnosed and treated. In the aged and debilitated patients, who may be a poor risk for operation, account must be taken of the condition of their myocardium and lungs.

3. *Surgical or diagnostic procedure mishap:* Failure of surgical technique might be:
 - i. Inadvertent in some unusually difficult operative circumstances or some anatomical abnormality.
 - ii. Accidental due to failure of equipment
 - iii. The result of error or incompetence on the part of the surgeon for which the surgeon can be sued.

Deaths Associated with Anaesthesia

Most fatalities are not related to the actual procedure but due to other factors. The fact that the death occurred during or soon after an anaesthetic by no means indicates that the anaesthetic played a significant part in the death. Anaesthetic deaths can be classified as:

1. *Those occurring under anaesthesia, but not related to anaesthetic agent:* These types of deaths can be due to:
 - i. Death due to injury and disease that made the operation and anaesthetic necessary

- ii. Deaths due to a disease other than that for which the operation was performed but about which the medical attendants knew before death. In this category, there is some contraindication to anaesthesia that surgeon and anaesthetist is aware of but there is presence of some other urgent injury or disease for which the operation is vital. In these cases, the risk of anaesthetic must be balanced against the risk of leaving the patient untreated.
 - iii. Deaths due to a disease that is unknown at the time of operation. In an elective operation, a full clinical examination together with ancillary investigation should be made to detect any significant disease that might affect the patient during the operation.
 - iv. Deaths may be due to surgical shock and exhaustion in cases where there is no preexisting disease. A poor risk patient because of the condition for which operation is being carried out is more likely to be affected.
 - v. A surgical accident may occur under anaesthesia rarely by the inexperienced doctors under emergency situations.
2. Deaths as a result of administration of an anesthetic:
- i. *Overdosage of an anaesthetic agent:* The deaths that arise out of the overdosage of an anaesthetic agent are rare and usually result from ill-advised polypharmacy. To achieve Anaesthesia, the current trend is to achieve a light plane of unconsciousness with a barbiturate or a hypnotic, accompanied by the inhalation of nitrous oxide and oxygen along with small quantities of a liquid agent such as halothane. To achieve muscle relaxation, long acting muscle relaxants such as gallamine, tubocurarine or pancuronium that compete with acetylcholine at the motor end plate or short acting muscle relaxants such as succinylcholine, decamethonium iodide, which depolarize the motor end plate rendering it unexcitable to acetylcholine, are used. Both the categories result in muscle paralysis. Overdosage with long acting muscle relaxants can be treated with neostigmine while overdosage with short acting agents such as succinyl choline can be overcome by the use of prolonged artificial respiration. Anaesthetic gases cause physical and psychological dependence and are commonly used by the medical personnel.
 - ii. *Anaesthetic misadventure:* An anaesthetic misadventure or a critical incident is a human error or equipment failure that could have led, or did lead to an undesirable outcome, ranging from increased length of hospital stay to death when it is not discovered in time.
The anaesthetic morbidity and mortality occur from the complications of anaesthetic procedure, failure of equipment and due to some human error such as emergencies, failure to perform a normal check, lack of skilled assistance, restricted access to patient, and inadequate supervision of the patient due to distraction, fatigue, inattention, boredom or anxiety.
3. The deaths that are the direct result of the administration of an anaesthetic can be due to
- i. *Acute cardiovascular failure:* This is the most common cause of sudden unexpected death due to the administration of general anaesthetic. Cardiac arrest is the usual way in which the mishap presents and it is often impossible to discover either clinically or at postmortem examination. Most cases occur under relatively light anaesthesia rather than deep unconsciousness and thus tend to occur either at the beginning or at the end of surgery. This can occur even in the hands of most experienced and careful anaesthetist.

Cardiac arrest appears to be mainly neurogenic in origin and can result from any form of irritation of the respiratory tract such as intubation or laryngoscopy.

- ii. *Deaths due to respiratory failure:* Hypoxia occurring during anaesthesia may be due to defects in the supply of oxygen from the anaesthetic machines, either from mechanical failure or from the inexpert manipulation by the anaesthetist. Respiratory failure can also occur due to an overdose of an anaesthetic depressing the respiratory center. Excess of premedication and the use of muscle relaxants also predisposes to respiratory failure, unless the appropriate anaesthetic procedures such as assisted respiration are carried out. Mechanical blockage in the air passage may lead to hypoxic respiratory failures due to laryngeal spasm. Regurgitation of stomach contents and other mechanical defects in the apparatus may cause hypoxia and respiratory failure. Laryngeal spasm, swabs, dentures or teeth in the airways and haemorrhage from the naso-pharynx may cause or contribute to hypoxia and unless appropriate posture is maintained after an anaesthetic, the tongue may obstruct the airway in the anaesthetized patient.
- iii. *Recovery phase:* During the recovery phase, hypoxia may follow anaesthetic hyperventilation. After a period of hyperventilation, the tissue carbon dioxide store being depleted, the body attempts to replenish them. The need for carbon dioxide elimination is diminished, and hyperventilation results which may prove fatal. The cause of impairment to ventilation may lie at the level of CNS, peripheral nerves, the myoneural junction or even at the level of upper or lower airways.
- iv. *Postoperative problems:* Adequate postoperative care must be undertaken as medical practitioners will be responsible for any danger that befalls a patient due to his negligence
 - a. *Respiratory insufficiency:* Respiratory insufficiency that occurs within 72 hours of the surgery is usually due to the preoperative pulmonary diseases. If it occurs after four to five days, it is due to atelectasis or pneumonia, peritonitis, pulmonary embolism or increase in energy required for breathing because of exertion.
 - b. *Aspiration:* Aspiration of foreign material such as secretions, gastric contents, blood and food can contaminate the tracheobronchial tree. Aspiration of pus leads to multiple abscesses and that of gastric content may lead to irritation. As a result acute pulmonary oedema and chemical peritonitis may occur.
 - c. *Atelectasis:* Atelectasis frequently occurs due to the misplacement of tracheal tube in to one of the bronchi or due to respiratory insufficiency.
 - d. *Pulmonary oedema:* Pulmonary oedema may be the result of circulatory overload due to administration of excess blood, fluids or plasma expanders.
 - e. *Pneumothorax:* Spontaneous pneumothorax may occur during operative procedures with inhalation of anaesthesia or due to rupture of an emphysematous bleb. Tension pneumothorax is dangerous as it displaces mediastinum and has a deleterious effect on the heart and blood vessels.
 - f. *Air embolism:* Massive air embolism causes circulatory collapse by producing a frothy mixture of blood and air-bubbles within the heart chamber and causes immediate cessation of peripheral circulation.

- v. *Systemic toxicity of local anaesthetics:* These are those adverse effects of the absorbed drug that affect the whole body. The blood shows high blood levels of these agents. There may be allergic reactions ranging from anaphylactic shock to dermatitis. They also depress medullary centers leading to apnoea and vascular collapse. Local anaesthetics may depress the myocardium directly resulting in hypotension.
- vi. *Spinal anaesthesia:* Spinal anesthesia causes two major types of neurologic complications namely myelopathy and arachnoiditis.
- vii. *Deaths due to physical fault in the anaesthetic procedure:* The failure of equipment leading to anaesthetic mishaps usually occurs in a small number of cases. Explosions are particularly common with cyclo-propane or ether. Flammable liquids can cause fires. The fatalities can occur from electric shock due to defects in monitoring equipments, defibrillators or diathermies in the operation theatre.

Preventive Strategies to Combat Anaesthetic Complications

1. *Training and supervision:* The inexperience, inadequate training, inadequate knowledge about a new equipment or technique and even inadequate supervision by the seniors has been cited as a cause of error. To rectify these errors, regular in-house training of the anaesthetists should be carried on.
2. *Specific protocol development, more complete preoperative assessment and equipment and apparatus inspection:* Preoperative assessment of patients and preoperative inspection of equipment and apparatus will prevent a number of critical incidents.
3. *Additional monitoring instrumentation and equipment and human factors improvements:*

Improved standardization or arrangement of drugs in the Anaesthesia workspace, and improvement in design of gas flow control knobs and breathing circuit scavenging connections are now mandated by the national standards for new apparatus.

American Society of Anaesthesiologists has recommended the standards for basic patient monitoring such as electrocardiography, arterial blood pressure recorder, ventilator disconnection alarm and an oxygen analyzer. Besides these, the monitors that are encouraged but not mandatory include pulse oximetry, spirometry and capnography.

Medicolegal Problems associated with Anaesthetic and Operative Deaths

The main precipitating factors of negligence suits are failure of empathy and communication towards the patient and his relatives. To minimize the possibility of malpractice actions, the anaesthetists and surgeons should ensure the following

1. A proper physician patient relationship should be established.
2. In surgical procedures, the patient should be properly identified, the type of surgery to be performed, the side on which the surgery is to be performed is important.
3. Before carrying out surgery, an informed consent should be obtained from the patient in his own language, properly stating the detail of the procedure, the risks and disabilities associated with it. For anaesthesia, separate consent should be obtained.
4. Pre-anaesthetic check-up of the patient should be properly documented.
5. It is imperative to ascertain whether the anaesthetist has acquired the necessary skill to operate it by formal training.
6. Any preexisting natural disease such as the heart or lung disease should be properly evaluated before undertaking surgical procedures.

Autopsy Procedure in Anaesthetic and Operative Deaths

Whenever deaths occur during or shortly after the performance of a surgical operation and general anaesthetic the pathologist must answer the following questions

1. Was the death due to the effects of the surgical procedure or anaesthetic or is it due to the natural disease for which operation was being carried out?
2. Would the patient have died at the time he had died, if he has not undergone through the anaesthesia or operation?
3. Was the surgery and anaesthesia vital to save the life of the patient?
4. Was there any defect in anaesthetic or surgical technique?
5. Was the patient suffering from any predisposing condition relating to the condition for which he was operated that made him more susceptible to death from anaesthetic or operative procedure?
6. Was the death due to some unsuspected natural disease directly unrelated to the disease for which surgery was being performed?
 - i. Detailed hospital record of the patient, including full clinical and pre-anaesthetic check-up details are essential especially in anaesthetic deaths.
 - ii. Surgical intervention and its sequelae like sepsis, haemorrhage, oedema etc. should be noted that frequently make examination of the operative site difficult. It is sometimes difficult to distinguish defects due to post-mortem changes from abnormalities existing during life. It is common for the inexperienced pathologist to misdiagnose resuscitative artefacts. The finding of gastric contents in the airway at the autopsy should be cautiously interpreted. The agonal regurgitation of gastric contents in the air passages should not be misinterpreted.
- iii. There are rare instances of surgical mishap, which may not be negligence if the operating conditions were difficult, include ligation of arteries and veins, ureters, bile ducts, perforation of large blood vessels.
- iv. A full clinical history along with the consultation with the surgeon, anaesthetist and other medical staff is necessary to arrive at the best possible opinion. Sometimes the cause of death is obscure even after the autopsy.
- v. Presence of pre-existing natural disease such as the heart disease, respiratory insufficiency due to the lung disease and their contribution to the cause of death must be evaluated.
- vi. In deaths occurring due to surgical shock and exhaustion, the pathologist must exclude or confirm surgical incompetence and also evaluate as far as possible the pre-existing state of the patient.
- vii. If pneumothorax, air embolism or surgical emphysema is present, it should be clearly evaluated.
- viii. Surgical and anaesthetic devices such as airways, endotracheal tubes, needles, catheters etc. should not be removed prior to autopsy.
- ix. A radiograph that will show a ring of oedema of oesophageal mucosa at the level of the tube along with distention of stomach and intestines, in oesophageal intubation, can check position of the endotracheal tube.
- x. All the organs should be dissected and surgical sutures should be inspected.
- xi. Chloroform and halothane are hepatotoxic and chloroform may cause ventricular fibrillation sometimes. Halogenated hydrocarbons cause cardiac irritability.
- xii. The blood samples obtained prior to the death of the patient should be sent for analytical tests.
- xiii. *Toxicological examination:* For toxicological examination following samples should be collected:

- One lung sealed in nylon bag
- Two grams of fat from mesentery
- 100 grams of cerebrum
- 100 grams of liver
- 100 grams of kidney
- 10 grams of skeletal muscle
- Urine.

In case of inhaled anesthetic specimens should be kept in containers of appropriate size to avoid empty space, are sealed and refrigerated or frozen. Alveolar air should be collected with needle and

syringe by puncturing the lung under water before the chest is opened.

Long Questions

1. What are the causes of death on the operation table? What precautions you should take to avoid such deaths.
2. Discuss in detail the autopsy procedures in anaesthetic and operative death. And how do you preserve viscera in such cases.

Sudden and Unexpected Deaths

The medicolegal autopsy is conducted in cases of sudden and unexpected deaths, including apparently accidental death, primarily to establish cause of death. The definition of sudden death varies according to authority and convention. WHO defines sudden death as 'death that is unknown or sudden and occurring within 24 hours from onset of symptoms'. However, some pathologists and clinician will only accept sudden death as the one occurring within one hour from the onset of illness. The unexpected nature of death is more important than its suddenness. Many unexpected deaths will be sudden those occurring immediately upon collapse or within minutes and there may be a delay of hours or even days without a diagnosis being clinically evident.

In sudden deaths, the immediate cause is usually in the cardiovascular system.

Causes of Sudden Death

1. Cardiovascular system	45-50%
2. Respiratory system	10-15%
3. CNS	10-18%
4. Alimentary system	6-8%
5. Genito-urinary system	3-5%
6. Miscellaneous	5-10%

CARDIOVASCULAR SYSTEM

In the cardiovascular, the causes of sudden death are: (i) Coronary atherosclerosis (ii) Hypertensive heart disease (iii) Aortic valve disease (iv) Coronary circulation anomalies (v) Coronary artery diseases-

polyarteritis (vi) Cardiomyopathic enlargement (vii) Congenital heart disease.

Ischaemic heart disease is by far the most common cause of sudden death in Western Nations. The normal anatomy of heart and coronaries can be summarized. The normal weight of an adult heart varies between 275-300 gm and that of female between 225-250 gm. The atrial walls are 1-2 mm thick whereas right and left ventricular walls are 3-5 mm and 10-15 mm thick respectively.

Blood Supply of Heart

- The left coronary artery** arises from the left aortic sinus and the main trunk after a short course of 1 cm or less is divided into (Fig. 11.1):
 - Left interventricular (Anterior descending) branch:* This courses down the interventricular septum to reach the apex of the heart and supplies anterior aspects of right and left ventricles and the anterior 2/3rds of I.V. septum at the apex of the heart.
 - Left circumflex branch:* It follows left A-V sulcus to reach to back of heart. It supplies small portion of the lateral aspect of left ventricle extending slightly anteriorly and posteriorly.
- The right coronary artery** arises from right aortic sinus, passes behind the pulmonary trunk and follows the right A-V sulcus
 - Right marginal branch:* This branch is given off at the right border of heart and supplies the remaining anterior surface of right ventricle and posterior third of interventricular

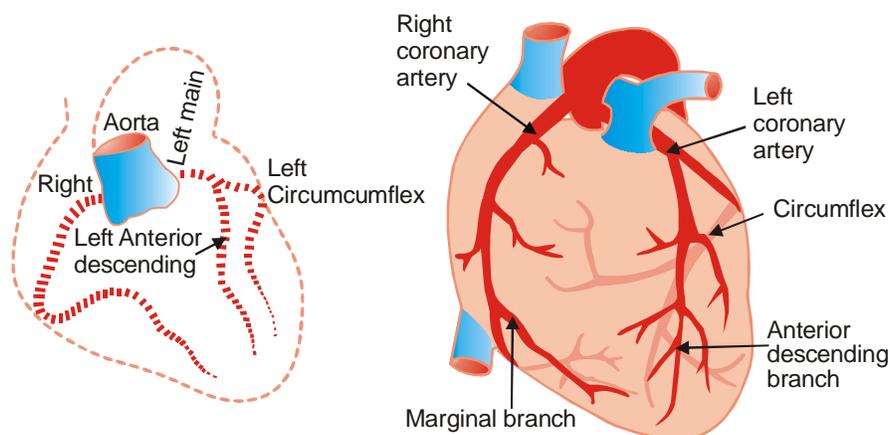


Figure 11.1: Blood supply of heart

septum.

- ii. *Posterior descending (interventricular) branch*: The main part of right coronary artery continues at the back of the heart as posterior descending branch.

Congenital Anomalies of Coronary Artery:

Minor anomalies that usually involves the proximal coronary artery but the distal blood flow is normal.

1. "High take off"—location of the coronary ostia is 1 cm above the level of cusp margin.
2. Multiple coronary ostia.
3. Separate ostium in the right coronary sinus.
4. Third coronary artery arises from the non-coronary sinus.

Frequency of Site of Fatal Coronary Stenosis with or without Thrombosis

Coronary stenosis with or without thrombosis commonly involves first two cm of the origin of the vessel. The incidence of involvement of the branches is:

1. Anterior interventricular→45-64%.
2. Right main coronary→24-46%.
3. Left circumflex→3-10%.
4. Left main coronary→0-10%.
5. Right marginal and posterior interventricular branches are particularly immune.

Coronary Atherosclerosis

Coronary atheroma may be focal, with irregular plaques. Every part of the major vessels must be examined at autopsy, with transverse cuts at no more than 3 mm intervals since few and localized atheromas may be present.

Types of Occlusion

1. *Simple atheroma*: Plaques are often eccentric with a central pin hole leaving a crescentic or concentric residual lumen.
2. *Ulcerative atheroma*: This results due to breaking down of endothelium over atheromatous plaques. This is conducive to thrombus formation.
3. *Subintimal haemorrhage*: Sudden haemorrhage resulting from rupture of tiny vessels in the wall of a coronary artery. The hematoma thus formed forces the plaque inwards to further narrow or occlude the vessel lumina.
4. *Coronary thrombosis*: This occurs on an already stenosed plaque of atheroma with damaged endothelium.
5. *Periarteritis Nodosa*: This is pan inflammatory disease of arteries of unknown etiology that affects males around fourth decade involving mainly muscular arteries of heart and kidneys.

Sequelae of Coronary Occlusion

1. *Sudden death*: May occur at the time of occlusion or subsequently as a result of ventricular fibrillation due to damage to nodal tissue or conducting system or any other complication.
2. *Myocardial infarction*: Myocardial infarction occurs in the myocardium distal to a complete occlusion of coronary artery in the absence of an adequate collateral circulation
3. *Rupture of the myocardium*: The rupture of myocardium occurs in people over the age of 60 yrs. It usually occurs on the 2nd or 3rd day after infarction because at this time muscle wall is soft and necrotic. The cardiac rupture results in haemopericardium leading to **cardiac tamponade** and death. The rupture almost always involves left ventricular wall.
4. *Myocardial fibrosis*: Localized patches result from healed myocardial infarcts and are seen in areas of predilection of infarcts like distal interventricular septum, apex and the posterior wall. Diffuse fibrosis may arise from many causes like healed rheumatic carditis and are also seen in cases of long standing coronary stenosis, which are sufficient to produce focal infarcts. The fibrotic patches are also seen in hypertensive heart disease or hypertrophy due to aortic incompetence
5. *Aneurysms*: Aneurysm of the left ventricle may occur if a large full thickness plaque of fibrosis becomes stretched. Rupture of aneurysm is rare.
6. *Mural thrombosis*: Thrombosis very often develops over an infarct, which reaches the endocardial surface of ventricle due to adhesion of platelets and fibrin deposition. Thrombus always poses danger of emboli.
7. *Pericarditis*: Occurs in transmural infarcts involving epicardium. Ischemic heart disease is the most common cause of pericarditis.

Myocardial Infarction

An infarct is a localized area of ischaemic necrosis in an organ or tissue resulting from sudden reduction of the arterial supply or occasionally

venous drainage. Myocardial infarction occurs with factors predisposing to atherosclerosis like hypertension, cigarette smokers, Diabetes mellitus and familial hypercholesterolemia.

Pathogenesis of Myocardial Infarction

Myocardial infarction will occur in myocardium distal to complete occlusion of a coronary artery in the absence of an adequate collateral circulation. These are of the following types

1. *Subendocardial infarction* involves layers of muscle adjacent to lumen of infarction.
2. *Intramural infarction*: is less common and is usually seen as satellite areas to a more extensive infarct.
3. *Transmural or full thickness infarction* is of large extent and gives rise to both mural thrombi and pericarditis, involves most commonly lower part of I.V septum, apex and posterior wall of left ventricle. All transmural or full thickness infarcts involve:
 - Left ventricle with I/V septum—67-80%.
 - Right and left ventricular wall—15-30% of cases.
 - Isolated right ventricle—1-3% cases.
 - Isolated left atrial—Rarest of all.
4. *Papillary muscle infarction*: Involvement of papillary muscle of left ventricle are common especially posterior muscles which are susceptible to ischemia.

Postmortem Demonstration of M.I.

The autopsy demonstration of an early myocardial infarct have profound medicolegal implication. The age of an infarct is difficult to establish as the onset of clinical symptoms however dramatic, are often much later than the onset of the pathological lesion precipitated by a coronary occlusion.

Gross Appearances

It is generally impossible to detect MI by naked eye examination within the first 8 hours after coronary occlusion and in many cases, little or no change may be apparent up to 18 hours.

On autopsy, the infarcted of the heart on cut section looks granular and dull instead of normal moist luster when cut by sharp knife. The infarcted area shows following features:

1. *Oedema*: There is swelling of infarcted area and coarsely fibrillar appearance of ventricle on cutting. The muscles seem separated with a grayish opaque sheen.
2. *Colour*: The colour of infarct is more pale than normal with a grayish opaque sheen. This changes to brownish purple darkening to reddish bluish Yellow after 24-48 hours of occlusion.
3. *Tigroid appearance*: The tigroid appearance that is alternate bands of red and pale areas is seen after 24 hours.
4. *Fully developed infarct*: A fully developed infarct is yellowish with reddish marginal zones of hyperemia and is gelatinous and than fibrous.

Histological Methods

This conventional method is very much helpful for the detection of early myocardial infarction. However, the main disadvantage is the time required for the preparation.

Haematoxylin and Eosin Staining

The paraffin embedded sections are stained with haematoxylin and eosin and the following features are noted under microscope:

1. *Eosinophilia*: It is one of the most important features in the early stages not less than 6 hours of infarction. The cells are reddened and most of the cells are eosinophilic in nature along with the swelling as well as increased granularity at the same time. Eosinophilia is easily seen by the application of green filter.
2. *Swelling of the Muscle fibres*: The intercellular spaces are reduced due to edema of the cells. It may occur in the large masses and at times it may be patchy in nature. It is usually associated with some degree of increased eosinophilia.
3. *Granularity of Cytoplasm*: This may be found easily in both transverse and longitudinal

sections of muscle fibers known as cloudy swelling. At times it is a reversible phenomena.

4. *Corrugation*: Dead muscle fibers may be corrugated and they may be sharply angulated in the areas where there is severe bending of the fibers.
5. *Granularity of the cell membrane*: Granularity occurs at an early stage before the cells begin to disintegrate as a result of necrosis. Granularity is also seen as artefact in frozen sections but when found in paraffin sections it has got some significance unless there is advanced putrefaction.
6. *Increase in Interstitial cells*: Increase in numbers of cells occur quite early but may also appear late. Some time it does not appear at all. The increase is due to wandering macrophages and the cells which cannot be distinguished from fibroblast. Polymorphs never appear in an infarction before 24 hours.

The above findings of early infarction goes on increasing as the time passes. There is increase in the necrosis of the cells with disintegration, heavy polymorph infiltration, filling of the inter-spaces with basophilic nuclear debris and marked vascular congestion and dilatation.

Periodic Acid-Schiff Stain Technique

The main purpose of this technique is to detect glycogen and stains the necrotic myocardium purplish pink. The substance responsible is diastase-resistant and by histochemical analysis has been shown to be either a muco- or glyco-protein. It does not appear until 12 hours of onset of infarction. Thus it has very little value for the diagnosis.

Phosphotungstic Acid-Haematoxylin Technique

The above technique does not allow to detect the myocardial infarction earlier than the stains by haematoxylin and eosin but it can demonstrate the cell death in a more clear way. The characteristic lesion is a break-up of the muscles striations, starting with a "sandy" appearance that progresses to a bizarre clumping and aggregation

of the muscle. This technique is superior to haematoxylin or eosin as the infarcted areas appear in a better way.

Macro-enzyme Techniques

During infarction, the metabolic changes in the cells occur before any morphological changes are demonstrable. One of these changes is loss of enzymes from the cells as a result the damaged fibers are enzymatically less active. The main enzyme **dehydrogenases** may be visualized by tracing their action in a suitable substrate by means of a colour indicator.

The methods like tellurite reduction by Malic Acid Dehydrogenase and Nitro-BT method are found to be unsatisfactory because of the technical unreliability and the expense of reagent but the TTC method is of considerable practical use.

Triphenyl Tetrazolium Chloride (TTC)

Transverse slices of ventricles about 5 mm thick are cut with a knife during postmortem and incubated for one hour in the staining solution of 1% Triphenyl tetrazolium chloride in a phosphate buffer at pH 8. The maintenance of this pH is very important. If the solution is on the acid side of the neutrality, no deposition of the dye will form. Moreover, if the PH rises above 8.5 the reaction will be too rapid and formazan will be deposited over the whole surface of the heart slice. Normal heart muscle with active dehydrogenases acts on the TTC to produce a **bright red formazan** compound. After the reaction has been developed with 30 minutes, the slice may be fixed in formalin which retains its colors for several weeks when kept in the dark. The real contrast appears after proper fixation. The slice so obtained may be used at the direct evidence of infarction. The technique may be very simple but needs careful supervision with occasional agitation of the solution while incubating. Postmortem interval upto two days appears to have no deleterious affect on the reaction but the slices should not be exposed to the atmosphere before incubation.

Appearance of Myocardial Infarction with Appearance of MI with Time

MI less than 6-12 hours old:

- Unapparent on gross examination
- Oedema
- Slight pallor
- Eosinophilia within 4-6 hours
- Within 3-6 hours, histochemical techniques highlight necrotic area.
- Increased cellularity (macrophages and lymphocytes) within 12 hours.

MI > 18-24 hours old:

- Inter fibrillar oedema deepens and cellular oedema subsides
- Better demarcated pallor lesion
- Eosinophilia with red line.
- Tigroid appearance.

MI 36 hours to 2-4 days:

- Nuclei are hollow with marked polymorphic activity in 4 days.

MI by 1st week:

- Disintegrated muscle fibres
- New capillaries and fibroblasts.

By 2nd week:

- Disintegrated macrophagic activity

By 3rd week:

- Removal of dead cells is complete
- Collagen formation starts

By 4-6 weeks:

- Marked collagen formation (Table 11.1).

Lesions Involving Myocardium

1. *Hypertrophy of myocardium:* Hypertrophy of myocardium may be associated with coronary ischemia. It can occur with hypertension, valvular disease, shunts and long standing pulmonary disease. Depending on the underlying cause either one or both the sides of the heart may be hypertrophied. It may be familial or sporadic. Interventricular septum is enlarged and bulges into both the ventricles producing a ridge beneath the aortic valve. Sudden death is common in the familial variety.

Table 11.1: Gross and light microscopical findings in a myocardial infarct

Age	Gross microscopy	Light microscopy
<4 hours	No change	No change
4-12 hours	Slight mottling with areas of dark discolouration	Intense sarcoplasmic eosinophilia, nuclear pyknosis with contraction bands
12-24 hours	Mottled and mildly oedematous and bulging out surface	Sarcoplasmic eosinophilia with early interstitial oedema and neutrophilic infiltrate.
2-4 days	Soft yellow-tan core with mottled border.	Maximum neutrophilic infiltrate, nuclear loss and sarcoplasmic coagulation
5-7 days	Yellow-tan core and irregular hyperemic red-brown border	Basophilic interstitial debris, early macrophage infiltration, dilated capillaries at border.
8-10 days	Yellow gray core and red brown border, depressed cut surface.	Numerous macrophages with active phagocytosis, pigmented macrophages filled with lipofuscin.
11-14 days	Yellow gray core and red gray border, depressed cut surface	Granulation tissue along border, ongoing phagocytosis at core
2-4 weeks	Core becoming smaller, border becoming larger, grayer, firmer and less gelatinous, less depressed cut surface.	Ongoing scar formation dense at outer border dilated peripheral blood vessels, central core of necrotic tissue.
>1 month	Firm gray-white or red-gray scar, with scar retraction, variable wall thinning.	Mature scar (dense collagen, focal elastin, and hypercellularity) focal lymphocyte

2. *Neoplasm*: Primary neoplasm such as rhabdomyoma are occasionally encountered. Secondary neoplasm are very rare.
3. *Myocarditis*: Myocarditis is inflammation of the heart muscle evidenced by exudative and proliferative cellular reaction. Acute purulent myocarditis can occur as a complication of septicemia and myocardial abscesses may be produced. This is seen sometime in death following severe burns with supervening infection with *Pseudomonas aeruginosa*. A pyogenic myocardial abscess as a cause of sudden unexpected death is quite unusual.
4. *Alcoholic cardiomyopathy*: Alcoholic cardiomyopathy may result from direct toxic effect on the myocardium, general nutritional deficiencies including vitamin deficiencies and Beri Beri heart disease. The toxic alcoholic cardiomyopathy is the most common. In alcoholic toxic cardiomyopathy, the heart is over weight with hypertrophy of both ventricles. Coronary arteries are free from atheroma. On microscopic examination left ventricular wall shows scattered fibrosis.
5. *Miscellaneous conditions*: Cardiac arrest may occur with no organic disease of the heart

apparent at the autopsy. A careful examination of deceased and family history can provide an evidence of preexisting disease or abnormality. A variety of minor and scarcely detectable insults to the myocardium can cause faults in the normal transmission of the heart and can cause sudden death. Sudden death can also sometimes occur in patients without any gross or microscopic abnormality of the heart who have a history of paroxysmal or sustained arrhythmia. Also in an apparently healthy young individual over exertion can cause sudden death but preexisting cardiac conditions should be excluded for the diagnosis.

6. Primary neurogenic shock or reflex vagal inhibition can cause cardiac arrest in the absence of any demonstrable cardiac lesion.

Lesions Involving Pericardium

Obliterated pericarditis following acute inflammatory changes can occur. Fibrinous pericarditis with blood stained fluid may result from infarction, rheumatic heart disease or uremia. These secondary effects may cause death.

Lesions Involving Endocardium

Fibro-elastosis is commonly found as congenital abnormality causing death in infancy. The secondary form is associated with other congenital heart diseases. In the primary form the endocardium is thick and the changes are more diffuse. Death occurs from heart failure in first week of life in about 1/4th of the infants. Subendocardial hemorrhages are seen in cases of obstetric shock, arsenic poisoning, head injury and from infected blood transfusion. There may be severe drop in blood pressure accompanied by severe blood lesions.

Valvular Lesions

Congenital valvular lesions are most commonly associated with fusion of aortic or pulmonary cusps. This may be a site of valvulitis or calcification. Valvular disease may be accompanied by long-standing medical history, Mitral valve disease can be present undetected in life. It may be associated with ball valve thrombus in left auricle and some emboli may be found in kidney or spleen.

RESPIRATORY SYSTEM

Sudden death due to respiratory tract disease in a healthy individual is rare. The main cause is hemoptysis in such cases. Death mainly results from haemorrhage or from inhalation of blood in to the lungs. Hemorrhages may result from neoplasm or inflammatory lesions of the nasopharynx, carcinoma of bronchus or esophagus infiltrating adjacent tissues. Inflammatory lesions such as tuberculous cavitation, lung abscess or bronchiectasis are less common causes. An aortic aneurysm may rupture into bronchus or esophagus. Spontaneous pneumothorax rarely causes death. It most commonly results from rupture of an emphysematous bulla. Respiratory tract infections may also result in sudden and unexpected death. However usually there is a period of some hours or a day or two preceding death. Death may result in respiratory tract

infection from hypoxia due to mechanical obstruction of air passage by pus in bronchopneumonia or acute purulent bronchitis superimposed upon chronic bronchitis. Acute respiratory obstruction of the larynx may be due to local neoplasm but may occur due to angioneurotic edema or inflammation arising locally.

CENTRAL NERVOUS SYSTEM

Subarachnoid hemorrhage

Sudden and unexpected death due to diseases of central nervous system result from subarachnoid or intracerebral hemorrhage. Subarachnoid hemorrhage may be due to rupture of Berry aneurysm usually located on the **circle of willis** (Fig. 11.2) and sometimes on other cerebral arteries. These aneurysms are most commonly developmental resulting from congenital weakness of the vessel wall. Rupture of the vessel may be the result of atheromatous degeneration. Mycotic cerebral arterial aneurysm are due to damaged vessel wall by infected emboli as may arise in bacterial endocarditis. Subarachnoid hemorrhage

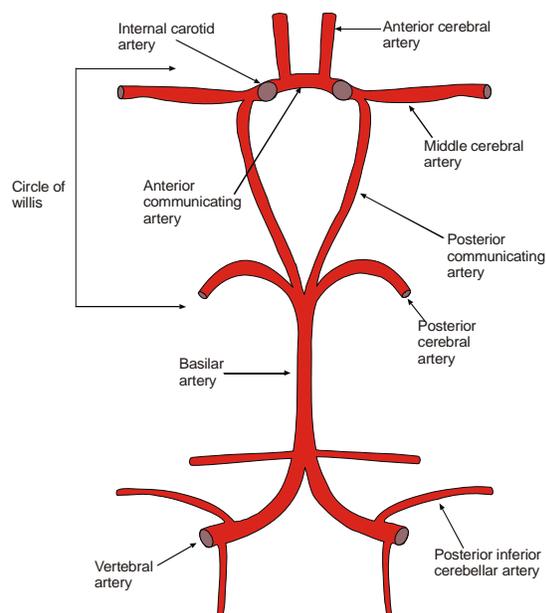


Figure 11.2: Circle of willis

may produce sudden collapse and rapid death. A transient rise in blood pressure for some relatively minor injury might contribute by causing damage in the already damaged tissue and by initiating the first split in the arterial wall resulting some time later in rupture. At autopsy, there is a massive collection of blood at the base of brain that is why it is difficult to locate the aneurysm. Multiple aneurysm may be present but are uncommon.

Intracerebral Hemorrhage

Intracerebral hemorrhage may be located in either internal capsule or in brain substance, in cerebellum or pons. It is commonly seen in capsular region and rarely immediately fatal. Death may occur after a few hours or the patient may partially recover and may later on collapse due to rupture into lateral ventricle. Sometimes intracerebral hemorrhage in capsular region resulting from cerebral artery aneurysm bursts and tears through the cerebral tissue and floods the subarachnoid tissue in the base of brain.

Pontine Hemorrhages

Primary pontine hemorrhages are usually single and involve hypothalamus. Secondary pontine hemorrhages may be associated with head injury and are of considerable medicolegal importance.

Cerebral Thrombosis and Embolism

Cerebral thrombosis and embolism may cause loss of consciousness and paresis. In cerebral embolism it is sudden and source of embolus is usually the left auricle in cases of auricular fibrillation, mitral stenosis, a valve or a mural thrombus. The embolus can originate from varying sites and the position of lesions are those supplied by cerebral arteries, basilar arteries and rarely carotid arteries.

Spinal Cord Hemorrhages

Spinal cord hemorrhages are uncommon and may be missed if spinal cord is not routinely examined.

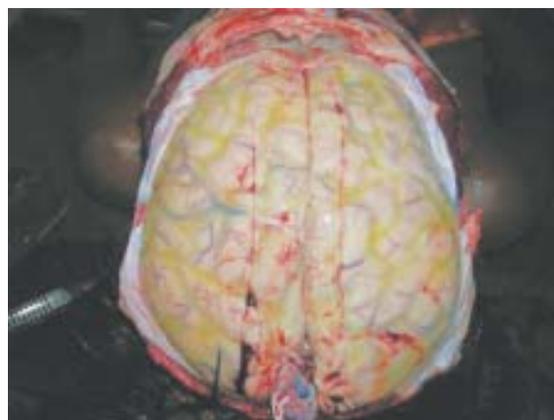


Figure 11.3: Cerebral meningitis

Cerebral Tumours

Cerebral tumours are incidental findings in small number of medicolegal autopsies. Meningiomas are the most common cerebral tumours. Rarely sudden death can occur from hemorrhage into neoplasm. The common tumour to cause hemorrhage is glioblastoma. Vascular tumours like hemangiomas may rarely bleed. Tumours themselves except meningiomas are not traumatic in origin.

Sudden death is rarely of infective origin when occasionally cerebral abscess may rupture. Rapid death has been associated with meningitis (Fig. 11.3) – meningococcal, pneumococcal, influenza and tuberculous. Acute poliomyelitis and encephalitis may be fulminant, specially when brain stem is involved. Sudden death within hours of onset of symptoms may occur in malaria. Postmortem diagnosis depends on demonstration of malarial pigment in brain, spleen liver and kidney. The pigment must be distinguished from formalin pigment in tissues fixed for days or longer. Sudden death can also result from trypanosomiasis in which postmortem diagnosis is difficult as organisms are difficult to find in tissues. Nevertheless, the inflammation of brain and meninges with perivascular inflammatory reaction and large histiocytic cells (morula cells) are prominent. Sudden death can also occur in

an epileptic seizure frequently in status epilepticus from heart failure due to myocardial ischemia. Examination of brain may fail to reveal any lesion in idiopathic epilepsy. However, some evidence may be obtained from bite marks on the tongue and from the clothing for evidence of incontinence of urine and faeces. Organic diseases may precipitate epilepsy and evidence of cerebral tumours and cysticercosis should be looked for.

ALIMENTARY SYSTEM

The causes involving the gastrointestinal system that can be result in sudden death are: (i) haemorrhage into gastrointestinal tract resulting from peptic ulcer, esophageal varices (ii) perforation of ulcers such as peptic, typhoid (iii) strangulated hernia (iv) rupture of liver abscess (v) rupture of enlarged spleen as in malaria (vi) intestinal obstruction (vii) Twisting of the intestinal.

GENITOURINARY SYSTEM

The conditions involving genitourinary system that can be the cause of sudden death are (i) chronic

nephritis (ii) nephrolithiasis (iii) carcinoma of kidney and urinary bladder (iv) uterine haemorrhages (v) carcinoma of female genital tract eroding femoral vessels (vi) twisting of ovary, ovarian cyst or fibroid tumours.

MISCELLANEOUS

Many other conditions can be the cause of sudden and unexpected death such as (i) diabetes mellitus (ii) cerebral malaria (iii) vagal inhibition (iv) drug anaphylaxis (v) shock due to emotional excitement (vi) mismatched blood transfusions.

Long Questions

1. Define sudden death. Discuss in detail the causes of sudden death due to cardiovascular disease.
2. Describe the anatomy of Heart and the diseases of heart that cause sudden death.
3. Draw a sketch of Circle of Willis. Describe in detail the disorders of central nervous system that cause sudden death.

MULTIPLE CHOICE QUESTIONS

1. The most common cause in sudden death involves:
 - A. Cardiovascular system
 - B. Respiratory system
 - C. Nervous system
 - D. Idiopathic
2. The most common site of fatal coronary stenosis is:
 - A. Anterior interventricular
 - B. Right main coronary
 - C. Left circumflex
 - D. Left main coronary

Road Traffic Accidents

Accidents occurring on the road between the vehicles or a vehicle with pedestrians. It accounts about 60 % of the total deaths. The most common form of accident is between a vehicle with a pedestrian.

MECHANISM OF INJURY

The mechanism of road traffic accident is thoroughly studied by De'Haven. He has found that the severity of injury to a vehicle occupant is directly proportional to the degree of acceleration or deceleration force. The change of speed is the main culprit, rather than its absolute speed. The G formula is used to calculate the main force involved in an accident impact of deceleration force may be calculated from the following formula.

$$G = \frac{V^2 \times 0.034}{D}$$

Where G = equal to force exerted by gravity

V = velocity of the vehicle in miles / hour

D = stopping distance in feet

0.034 is a mathematical conversion factor

The injuries are caused to the occupants while they are inside the vehicle (Non ejection crash injury) or when thrown out of the vehicle (ejection crash injury) or both types of cases the nature, severity of injury vary.

Primary Factors in Accidents

These factors are the most important ones in the causation of the accident. The factors can be both human as well as environmental factors.

Human Factors

Age, sex, education, medical conditions, fatigue, psychological factors, lack of body protection.

Environmental Factors

Relating to road: Defective narrow roads, defective layout of cross roads and speed breakers, poor lighting, lack of familiarity.

Relating to vehicle: Excessive speed, old, poorly maintained, large number of two or three wheelers, overcrowded buses, low driving standards. Bad weather, inadequate enforcement of existing laws, mixed traffic slow and fast moving, pedestrians, animals etc.

Both the above factors lead to increased vulnerability and risk situation and lead to accidents. Prevention of accidents, measures to be devised. If accident is a disease, education is a vaccine. Safety education Belief that accidents are inevitable must be curbed. Safety education must begin with students, driver need to be trained for safe driving and proper maintenance of vehicles. Young people must be educated regarding risk factors, traffic rules, safety precautions and first aid.

Non-ejection Crash Injuries

Frontal impact is more common in road accidents, the occupant may also be exposed to lateral and rear impact collisions. During lateral impact, the force is of larger magnitude, for there is very little of the car structure to cushion the impact. In

general there is little difference in the frequency and locations of injury between rear seat and front seat passengers.

Ejection Crash Injury

Ejection is largely dependent on whether the door springs open at the time of crash. This occurs most commonly in roll over accidents. In ejection fatalities, one frequently sees severe multiple injuries, any one of which may be responsible for death. Head alone or in combination of chest and abdomen is injured in 2 out of 3 cases. Chest and abdomen alone or in combination is seen in more than half the cases. Fracture of ribs is seen in 2/3rd of cases.

Non-ejection Crash Injuries

Sitting position

Front seat

The front seat passengers are injured by the primary impact. The head, abdomen, knee and chest are injured in this impact. The head, knee and the chest are injured as they hit the steering wheel and the dashboard of the car while the abdomen is injured by the seat belt.

Seat belt injuries

The injuries caused by the seat belt depend upon the type of belt present. Perforation of ileum, caecum and sigmoid colon is common due to compression of intra-abdominal viscera. Presence of abdominal wall contusion associated with paralytic ileus should raise a suspicion of significant intra-abdominal injury.

Lap Belt

Injury to the lumbar spine, Spleen, pancreas, uterus, urethra and iliac artery.

Shoulder Restraints

If shoulder restraints are used then various injuries occur such as fracture of the ribs, cervical spine, lumbar spine and sternum. Deep organ lesions of larynx, liver, spleen and kidney can be found..

Three Point Belts

If three point seat belts are used it causes a pattern of injuries which includes fracture of ribs, sternum and clavicle. Abdominal injury leading to jejunal or duodenal perforation is seen. Contusions and abrasion in the chest, shoulder, neck and back may also be seen underlying the position of the seat belt..

Ejection Crash Injury

Ejection is 2nd only to the steering wheel as a major cause of injury. It depends upon whether the doors spring open at the time of crash. There are multiple injuries, the head alone or in combination with chest and abdomen alone or in combination with each other or with group.

Fracture rib	66.6 %
Laceration in liver	33 %
Soft tissues and other abdominal viscera	50 %

Injuries caused by Vehicular Accidents

- Primary impact injuries are the injuries caused to the first body part struck by the vehicle.
- Secondary impact injuries are the ones caused by further impact by the vehicle.
- Secondary injuries are caused by the impact with the ground.

Injury to Pedestrians

Both primary and secondary impact injury occur as the person is thrown to ground or may be crushed and run over. Bumper of the car causes injuries below the knee; the radiator hits the pelvis and the abdomen is hit by the fender. It has been found that the pedestrians sustain about 25 % cases fracture of legs while 40 % cases there is injury to chest and abdomen. The fracture of ribs and injury to heart and lungs are the commonest form of injuries.

Bicycle

When a person travels by a bicycle and is hit by a vehicle, fracture bones, severe soft tissue injuries and bicycle spoke injury, particularly in children.

Run over Accidents

When a victim is run over the tire marks and grinding abrasion may be seen. Burning of the skin due to heat produced by sliding of the body, deep crushing of the body with rupture of internal organs may be found.

Motorcyclist

Usually occurs by running in front of a vehicle from one side to other. The cycle is hit and the person is thrown violently on the ground. At times they are thrown beneath a vehicle and crushed like any pedestrian.

Helmet

Egg shelled helmet may lead to fracture of spine or strangulation by the strap. Classical injury in motor cyclist and pillion passengers is fracture of skull usually from secondary impact on the ground.

Crush Helmet

This reduces friction of the head against the ground. It makes deceleration force less by allowing the protected head to skin across the ground rather than come to an abrupt halt. Temporal bone and parietal bone fracture, fracture of base of skull and contra coup injury to the brain are seen.

Car Drivers

Driver may impact the chest on the steering wheel or the upper abdomen on the wheel rim leading to fracture of the sternum, laceration to heart and splitting of the aorta. Lung roots may be pressed against the wheel rim and may crush the spine, liver, spleen or the kidney. Crush the throat by top of the steering wheel, split the head or face on a shattering windscreen. Asphyxia may result by inhalation of blood. Thrust the whole skull down on to cranial spine. Drivers or passengers may sustain 3 or 4 sliding forward off in seat and may also split one or both patellae or crush the knee joint. Fracture of femur or pelvis. If car overturns the person may be pinned to ground or crushed

leading to traumatic asphyxia. He may be burnt or get asphyxiated.

Injuries to Vehicle Occupants

These are important in identifying their positions in the vehicle at the time of accident including the driver and the persons in the front seat Dash board injuries Grazes or lacerations of the skin of knees from the impact with dashboard occurs. Also there may be fracture of leg bones.

Investigation of Road Accidents

Pattern of injury, role played by a concurrent natural disease, the crash as a possible method of self destruction (suicide) should be looked into. The crash as a possible method of making a homicide, role played by intoxicating drugs. The incidence of accidents caused by the natural death of a sober driver has been estimated to be less than 6 per 100,000 motor vehicle collision. The possibility must be kept in mind where bodily injury is minimal or absent and damage to the vehicle is limited.

Hit and Run

These cases requires very thorough investigation of the scene as well as clothing worn by the victim. Detection of patterned injuries, paint or metal fragments, grease, dirt or glass particles may provide the only evidence capable of leading to the identification of the responsible vehicle.

Role of Alcohol and Drugs

Driving under the influence of drugs or alcohol or both remains a major socio-economic problem in modern society. Drug use should be considered when drivers behavior is suggestive of alcohol consumption but tests of his blood or breath for alcohol are negative. This particular category of driver should be tested by standard methods of drug screening for barbiturates, amphetamines or other drugs that might explain the disorderly behavior. The alcohol intoxication contributes to a traffic fatality both by impairing judgment, coordination and vision before the accident and by interfering with the evaluation and interfere with

the treatment of injuries sustained at the time of crash. The most important investigation in road accidents is the determination of the degree of intoxication present. The concentration of blood alcohol should always be determined in the driver of a vehicle, in the pedestrian injured or killed in the accident in the passenger of the vehicle involved with accident.

Suicide

The imprint of the gas pedal pattern on the driver's shoe sole and the absence of skid marks leading to the site of collision are believed by some to be suggestive of suicide, because they indicate that the driver was accelerating his vehicle at the time of impact.

Purpose of Accident Injury Investigation

To determine directly by detailed examination of the results of actual road accidents, the effects of different types and severities of vehicle collision upon the living human body. More accurate information relating to mechanisms of injury will be obtained resulting from impact of human body upon the vehicle interior (or in case of pedestrians, the vehicle exterior).

Factors to be Investigated Include

The pattern of injury and of death in various groups of road users. The time interval between accidents and death. The cause of injury in relation to specific parts of the vehicle. The direction in which force acts on the human body to produce injury. The levels of human tolerance to dynamic loads applied to different parts of the body. The effectiveness of various safety design features in terms of reduction in frequency and or severity of injury.

Procedure of Accident Injury Investigation

It is the same for all classes of road users whether they be vehicle occupants or pedestrians or motorcyclists. Research team is interdisciplinary and consists of medical scientists, engineers, professional photographers etc. and it requires co-operation of hospitals, police, garages, motoring

public etc.

The standard procedure for investigation consists of:

Medical interview and medical examination of injured persons. Follow up study of vehicles. Interview of police officers concerned with the accidents and other witnesses. Writing up the accident in detail. Analyses of results when number of accidents reaches an adequate level.

Medical examination of casualties:

- Undertaken usually in hospital
- Patient's informed consent is obtained
- clinical and radiological findings are obtained
- all injuries are classified according to their severity as minor, moderate, severe as per the road research laboratory leaflet issued in 1969
- exact site of injuries and nature recorded on diagrams.
- Color photographic record made if possible
- In case of fatalities, post mortem examination with details of all injuries, time elapsed between injury and death and an assessment of the cause or causes of death are recorded.
- Enquiry into accident and examination of the vehicles it is necessary to reconstruct with reasonable accuracy the events of accident in relation to both the vehicle and occupant movement.
- Whether using on the spot techniques or retrospective techniques, a three phase investigation of the accident circumstances is undertaken.
- Reporting police officer and other witnesses are interviewed.
- Accident site is examined.
- Vehicles are subjected to careful scrutiny.

Using Retrospective Techniques

Certain basic data can be obtained from reporting officers regarding the accident which will include Directions in which various vehicles were traveling, Final positions of vehicle after the accident, Rough estimate of pre-accident speeds, Evidence of ejection or Trapping of occupants.

Examination of Scene

It is useful for reconstruction of accident and for interpretation of wounds and the mode of dying. Dragging marks, which are superficial and extensive, suggest that the victim had been dragged and not ejected. Measurements of the leg fracture and other body injuries from the heel of the feet may help in identification of suspect vehicle in hit and run cases.

The bumper fracture may be compared with the suspected car by measuring the height of bumper from the road. Diagrammatic representation of injuries is quite useful. All injuries cannot be interpreted but patterned injuries should primarily be considered. Specimens should be collected for alcohol, barbiturates, antihistaminic drugs, any foreign material like glass, paint or oil found on clothing, skin or the wound should be collected. The vehicle itself may give evidence of contact in the form of blood, flesh, hair, fragments of clothing, damage and signs of attempt to clean or repair.

Reconstruction of the Crash

Over interpretation of the post mortem findings is undesirable, since it must be remembered that it is usually impossible to attribute a specific causative mechanism to each and every injury on the victim's body. Patterned injuries by virtue of their location are vital to the accident reconstruction for example the circular abrasion on the driver's chest or face from the impact on the steering column or steering wheel or the impact of the head light on the thigh of a pedestrian struck by an automobile. It is advisable to collect blood and urine samples for alcohol in vehicular accidents. A driver who is not a property controlled diabetic or epileptic or suffers from cardiac disease is a potentially dangerous on the road. He is liable to unexpected impairment of consciousness. Also a driver who smokes more than 20 cigarettes in a day is considered to be living at a physiologic altitude of 8000 feet and impaired oxidation has striking effects on visual acuity at low levels of illumination.

$$PS = \frac{1}{2} MV^2$$

P = deceleration force—this is the force acting on the person when brakes are applied and the speed of the vehicle starts decreasing.

S = linear deceleration distance—this is the distance between the point when the brakes were first applied and the vehicle comes to a complete stop.

M = mass of car

V = velocity of the car at the time of applying the brakes.

Promotion of Safety Measures

Seat belts reduce the number of fatalities and non-fatal injuries by about 50%. It has been made compulsory for cars light trucks and similar vehicles. Safety helmets reduce the risk of head injury by about 30%. This reduces fatalities by about 40%. They prevent damage to scalp and skull fracture to a great extent. Full-face helmets are very popular among two wheeler riders.

Leather Clothing and Boots

Leather clothing reduces risk of extensive superficial soft tissue injury. Leather belts protect lower legs and feet. Children should remain seated when they are in vehicle. They should be prohibited to take front seats of cars. '**Child locks**' should be present in the cars.

Supreme court guidelines for school buses as safety measures are;

- Foot boards to enter bus at lower levels.
- Drivers with minimum of 5 years driving experience and with driving license.
- Doors should be locked.
- Parallel bars on windows.
- A school teacher and a conductor is mandatory.
- The bus should be painted clearly as school bus and in case of put buses should be clearly written as bus on school duty.
- Spaces for bags behind seats.
- Phone no. of school, name of school should be maintained in the bus.
- First aid boxes.

Other features are use of door locks, proper vehicle design and use of laminated high penetration resistance windscreen glass.

Alcohol and other drugs: Alcohol impairs driving ability and increases the risk of an accident as well as severity of its consequences. Alcohol is the direct cause of 30=50 % of road traffic accidents. Though the legal limit is 80 mg % but impairment can result at blood level of 50 mg % and accident risk increases in 50-80 mg % level. Drugs like barbiturate, amphetamines and cannabis impair driving ability.

Primary Care

Primary care includes planning, organization and management of trauma treatment and emergency care services for the trauma patient. Emergency care should begin at the accident site and anytime during transportation and should conclude in hospital emergency department.

Elimination of Causative Factors

Improvement of roads, imposition of speed limits, marking of danger points, reduction of electric voltage, provision of fireguards, increase of safety equipment in industries, safe storage of drugs, weapons, poisons. Accidentology deals with accident research and involves gathering precise information about extent, type and other causes of accidents, seeking ways to make environment-evaluating efficiency of content. Investigating new and better methods of altering human behavior.

Accidents are a matter of chance, safety isn't. So proper safety measure must be devised to minimize the chances of road traffic accidents. These can be broadly of three types.

Primary preventive measures

Primary preventive measures are devised to improve the driving condition and also to reduce the driving fatigue to avoid accidents.

User Friendly Systems

Electronic Power Steering: It uses less than half the power consumed by an ordinary power

assisted steering. It doesn't suffer from losses of power assistance due to leakage. Electronic sensors in these provide varying levels of power assistance at different speeds for , better maneuverability and greater control. Electronically controlled and incorporates self diagnosis in case of any problems. Power brakes, roll control device, anti-lock brakes, brake assist, acceleration skid control, strengthened body shell, antiglare wind-screens and glasses and mirrors, large vertical headlamps for enhanced visibility, good quality headlights for improved visibility and also high quality wide windscreen. Improved design of tail lights. Automatic transmission streamlined care structure and improved engine design for smooth and effortless pickup.

Collapsible steering column: For total comfort full flat reclining front seats as well as reclining rear seats. Upright design, low side steps, high seats and wide opening doors for easy entry and exit. Better air-conditioning to reduce the fatigue of driving in temperate climates to minimize irritability arising out of heat and carbon monoxide emanating out. Various safety measure like child locks indoors, central locking of all 5 doors, Bernouli's flaps on the undersurface of cars makes it to be driven sticking to the undersurface. Block shock absorbers, wide diameter shockers struts allow to drive smoothly over the worst roads. Wide tread tires ensure surest road grip and the safest drives. Better road designs for better safety and proper road lights to minimize accidents. Better traffic safety traffic lights and traffic signs should be visible from a distance and not be hidden behind a tree. Self diagnosing on board computers. The driver while driving should avoid talking on a cell phone. High volume music may divert attention. He should also avoid talking to the other occupants.

New safety features being introduced include:

- **Lane departure warning system:** This system warn drivers if they are about to leave a lane unintentionally
- **Adaptive cruise control:** This system prevents the driver from getting too close to

the vehicle in front of them by applying brakes automatically if the distance is too little.

- **Pre crash warning:** This system detects a possible collision and warns the driver. If the driver fails to react, the advanced versions of the system can activate the brakes and even turn the steering wheel.

Secondary Preventive Measures

These are to reduce fatalities if accident has occurred.

Safety belts 1 or 2 point safety belt. It has a disadvantage of slipping out from the hold and causes whiplash injuries. *3 point safety belt.* It is for a better hold. They avoid the impact of driver or other occupant to the front in case of sudden deceleration. The cars should have airbags and window bags. Injury over the dashboard on driver's side reduces the injury in case of sudden impact. Collapsible steering column and energy absorbing steering column. High penetration resistant windscreen glass. Sturdy design of vehicle to absorb front and rear impact through crumple zones. Side impact beams and bars in front and rear door to take up the impact. Anti roll bars are net like pillar or strongholds in case of rolls during accidents and prevent the collapse of whole structure. Laminated windshields are designed to prevent the shredding of glass in case of impact and if it displaces then it gets stick to the place only. Fire proof paints, upholstery prevents fire from spreading. Helmets in case of two wheelers minimizes the impact to head in case of an accident.

Tertiary Measures

Enable speedy salvage, rescue in case of accidents:

- Cellular.
- Audio system.
- Global positioning system.
- Computers.

Seat Belt Syndrome

With abrupt deceleration, the set belt compresses the contents of the abdominal cavity the mus-

culature of abdominal cavity ruptures and often of left hemi diaphragm. Also shear forces lead to the bowel injury. Applied against adjacent structures, such forces cause bowel transection, particularly near points of fixation, such as iliocaecal valve and the ligaments of trietz. Also fracture of lumbar spine results.

Road Traffic Injuries

Large varieties of injuries are sustained by persons involved in traffic accident and by detailed examination of these injuries it is possible to reconstruct the accident these may help in confirming or refuting the evidence of eyewitness. Further it may be possible to identify the hit and run accidents. In RTA, the following categories of people may receive/ sustain injuries.

Injuries to Pedestrians

Often the following pattern of injuries are seen:

Primary Impact Injuries

In these injuries, the part of body involved i.e. right or left side, front or back, will depend upon the position of person in relation to vehicles when struck i.e. whether crossing the road or waling with or against traffic. The injuries will be further modified as to whether both feet are on the ground or one is raised and the firmness or otherwise of either or both feet. The relative heights of various parts of the vehicle i.e. bumper, radiator, door handles etc. and the pattern of these parts of the vehicle if found on the body of person in the form of injuries, help in identification of vehicle. For reconstruction of accident, it is better to discuss injuries only in relation to one type of motor vehicle as the models keep on varying all over the world and from year to year (Figs 12.1 and 12.2).

Secondary Impact Injuries

Injuries caused if the victim strikes other objects such as the ground after falling from the vehicle. If the person is struck from behind with the feet fixed, primary impact will be on the back of legs and



Figure 12.1: Multiple abrasions over the shoulder in a case of road traffic accident



Figure 12.2: Abrasions over the forearm in a case of road traffic accident

fracture occurs if the feet are fixed. At the same time the buttocks and back will come in contact with the car and the person is pushed forward. It may cause fracture dislocation of lumbar or thoracic spine. If the person is struck from behind and feet slide forwards, the whole body may fall forwards and have a secondary impact of the head against the windshield, causing shattering of the windshield and head injuries. Alternatively, the body may be thrown in to air or to one side with resultant secondary injuries from striking the ground i.e. on head and other parts of the body. If the person is facing the vehicle, he may receive intra abdominal injuries and injuries to the chest wall and thoracic

cage. The victim may get injured from the bumper over the front of legs resulting in fracture of both tibia (bumper fracture). Frequently bumper injuries are present at different levels on the two legs or absent on one leg which suggest that the person was walking or running when struck. An impact against a mudguard or headlamp may result in a fracture of pelvis or fracture dislocation of sacroiliac joint.

If the person is struck on hip on its side, The initial impact will be against leg and produce bruises the other injuries will depend if the foot was fixed or sliding forwards. Additional injuries like bruising of elbow and fracture of ribs may occur from striking the different parts of the vehicle. Victim will then receive secondary injuries on striking the ground. In a glancing blow from the side of a car in cases where driver attempts to avoid a direct impact, injuries to projecting parts of body such as elbow, shoulder or hips may take place. Penetrating/ tearing wounds caused by protruding objects of the vehicle such as door handle etc may occur. When the vehicle travels at a greater speed, the pedestrian may be thrown height and fall on the vehicle roof and sometimes on the road behind the vehicle. In them the head is injured by direct impact with vehicle and by striking the road. Both impacts produce fracture involving almost all parts of the skull specially those with underlying extensive injuries of the brain.

If the person is run over, Crush injuries will occur and its severity will depend on the weight of the vehicle and its clearance from the ground. The findings would be Tire marks and grinding abrasions. Skin avulsion, Burning of the skin due to heat produced by sliding of the body, Deep crushing of the trunk or limbs with rupture of internal organs Flattening deformities of the head, chest or pelvis. When a child is run over by a vehicle, fracture may not be produced due to elasticity of partly cartilaginous skeleton (Fig. 12.3).

Injuries to the Driver

Driver has the advantage of a momentary appreciation that an accident on steering wheel



Figure 12.3: Crush injury over the leg

and thus avoids full impact on the chest or of the head against windshield. He may sustain sprains or fractures of the wrist or if he attempts to apply brakes. Fracture of femur or the pelvis takes place. Steering wheel impact type of injuries. Movement of the seat forwards and the steering wheel backwards may cause crush injuries i.e. fracture of ribs and sternum, splitting of aorta, crushing of heart, liver, spleen, kidneys etc. Imprints of brake or accelerator pedal designs on the sole of driver's shoe may be found.

Whiplash Injuries

The driver and front seat passengers may split the head or face on a shattering wind screen and may even sustain a fracture dislocation of cervical vertebrae due to impact or jerk resulting in hyperextension / hyper flexion of neck causing fracture of the cervical vertebra at the level of C2-C3 and C3-C4.

Injuries from flying glasses are less common due to use of safety by laminated glasses but face may be cut or damaged by impact. The risk of front seat passengers is more to drivers or back seat passengers.

Injuries to back seat passengers. They receive the injuries due to striking the side, or top of the vehicle or back of the front seat. Ejection injuries. The door may open sometimes and person may fall out. Traumatic asphyxia when vehicle over-

turns the occupants may be pinned and crushed or these may be serious injuries to heart lungs, liver, spleen or kidneys. Rarely occupants may be burnt or asphyxiated while in the interior of the car.

Injuries to Motorcyclists or Scooter Riders

These in most of the cases are from being struck with a motor vehicle pattern of injuries will be of

- Primary impact injuries of the leg.
- Secondary impact injuries resulting from the contact with the vehicle.

Secondary injuries will occur from striking the ground and on occasions being dragged with the vehicle. The same type of injuries are found in pillion passengers. Cervical fatal injury is fracture of skull usually from secondary impact with the ground. Temporoparietal fractures are common which usually extends across the base of skull through pituitary fossa. Crash helmet reduces friction of head against the ground and although the head injuries may be less, there will be other effects such as broken neck or fractured ribs or limbs with internal visceral injuries. Occasionally when helmet is pinned or crushed, the chin strap may be drawn upwards and cause strangulation. One type of injury peculiar to motorcyclists and scooter rider is collision with the rear of the truck when the vehicle will pass underneath the back but the head of rider impacts against the tailboard. Injuries sustained by pedal cyclists If caused by an automobile Injuries may be similar to those sustained by a pedestrian except that the impact will be lower on the body or only against to some part of bicycle itself. The secondary injuries may be more severe due to greater distance to fall. Injuries due to running over may be present.

Medicolegal Importance of Road Traffic Accident

Most of the accidental deaths are caused by automobiles but it may be occasionally used to commit crime like Planned killing, Accident faked to conceal crime, victim killed by other means and thrown on the road, victim killed by other

means and dead body burnt in a car, Suicide with automobile is difficult to prove but following events may be useful.

History of previous suicidal attempts and recent threat to commit suicide. History of depression recently or in the past or history of domestic quarrels or financial crisis, Evidence of speeding, Impact with a tree or bridge usually on the front of vehicle in its center, Single occupancy of the car, straightening of road, Absence of evidence of applying brakes, Suicidal note.

Natural death at the wheel versus death from injuries. Natural disease like coronary artery disease may lead to an accident and sudden unexpected death of a driver. Driver suffering from uncontrolled diabetes or epilepsy is a potential danger on the road. In case of pedestrians. Cardiovascular diseases, cerebrovascular diseases, poor vision or impaired hearing may all lead to

accidents. In these cases only a complete autopsy can provide a right answer as to whether the persons died of accidental injuries or naturally. Sometimes death occurs after a considerable time lapse after seeing injuries and it is important to give opinion in such cases if the death was entirely natural or directly / indirectly related to injuries specially if injuries were minor.

Long Question

1. What are the different patterns of injury seen in a pedestrian when hit by a car.

Short Questions

1. Whiplash injury
2. Seat belt syndrome
3. Tail gating injury
4. Rolling injuries

Mechanical Injuries

A wound is a break in natural continuity of any of the tissues of the living body. The question of examination of wounds arises in the Court of law. Mechanical injuries are the injuries that are caused to the body by physical violence.

Classification of Injuries

1. **Mechanical Injuries:** (i) *Those produced by blunt weapon:* (a) Contusion (b) Abrasion (c) Laceration (ii) *Those produced by sharp weapon:* (a) Incised wound (b) Stab wound (iii) Firearm wounds (iv) Fractures.
2. **Thermal Injuries:** (i) *Produced due to cold:* (a) Local hypothermia (b) Frost bite (c) Trench foot (d) Immersion foot (e) General hypothermia (ii) *Produced by local application of heat:* (a) Burns (b) Scalds (iii) *Produced by general application of heat:* (a) Heat Hyperpyrexia (heat stroke) (b) Heat Exhaustion (c) Heat cramps
3. **Chemical Injuries:** (i) Corrosives (acids and alkalis) (ii) Irritants (weak acids and alkalis).
4. **Injuries due to:** (i) Electricity (ii) Lightning (iii) X-rays (iv) Radioactive substances.
5. **Legally:** (i) Simple injury (ii) Grievous hurt.
6. **Depending on manner of death:** (i) Suicidal (ii) Homicidal (iii) Accidental
7. **Wounds can be:** (i) Defense wounds (ii) Fabricated wounds
8. **Wounds in relation to death:** (i) Antemortem (ii) Postmortem.

Mechanism of Wound Production

Moritz has described in detail the mechanism of production of wound. According to him, wound is caused by a mechanical force, which may be

either: (i) *Moving weapon* (counterforce is provided by the inertia of the body) or (ii) *Moving body* (rigidity of some stationary object). Due to impact between the propelling force and counterforce, energy is transferred to the tissues of the body that causes a change in their state of rest or motion. Human body contains many complex tissues such as solidity, fluidity, elasticity, density etc that vary in their physical properties. Therefore, there is no uniform change or effect on the body. All the body tissues except the ones that contain gases are resistant to compression forces. Mechanical forces does not cause compression of the tissues but causes their displacement and deformation with the result that traction strains are produced in the affected tissues. Such strains may usually be due to forces causing simple elongation of the tissues. In addition, they may be due to other mechanisms such as bending, torsion or shearing that are more complex.

Shear Strain: Shear strain is a strain, which is produced in a body by the forceful alteration of its shape but not volume. It causes stretch in direction parallel to the plane of contact. When tenacity of an organ exceeds, it ruptures and the bones fractures.

Factors influencing nature and extent of wounds

1. *Nature of the objects/instruments:*
 - i. *Blow from pin pointed/sharp edged weapon:* The force is concentrated over limited area resulting in incision or deep penetration. The hardness and friction of divided tissues resist passage of the instrument.

- ii. *Blow from blunt objects:* The force is dissipated over relatively large area so the damage caused to a unit of mass is less than when force is applied over a small area. A rapid transfer of energy over this small area is more likely to produce a wound than an equal amount over large area. The rapid transfer of energy is fatal than the slow transfer.
2. *Amount of energy discharged:* Kinetic energy is measured in a moving object by: $K.E = \frac{1}{2}mv^2$, where m = mass and v = velocity.
An object with definite velocity and definite weight have a definite amount of energy. When the mass doubles, kinetic energy is also doubled. However, when velocity is doubled kinetic energy increases four times. This indicates that the velocity has far more influence on the energy compared to the mass of the object.
3. *Conditions under which energy is discharged:* Moving objects have energy due to their movement that is directly proportional to their mass and to the square of their velocity as explained above. When moving objects are stopped suddenly on an encounter with some stationary object, the energy is dissipated at the sites of resistance, which causes various types of injuries like abrasion, contusion, laceration or incised wounds. On the other hand, if the human body is moving and encounters a stationary object, similar injuries are produced.
4. *Nature of the tissue affected:* The tissue that encounters the resistance suffers damage depending on its type as well as nature. Bones being tough undergo fractures on infliction of injury while muscles develop hemorrhages due to rupture of blood vessels in them. Soft tissues also tend to have hemorrhages or may rupture on infliction of severe force especially in cases of large organs like liver. Even, mesenteries get torn by blunt injury to the abdomen.

BLUNT FORCE INJURIES

CONTUSIONS

Contusions are a collection of blood into the tissues due to rupture of blood vessels, caused by blunt trauma. It can be caused in the skin or inner organs like liver, brain, kidney etc. It is a

painful swelling and is caused by crushing or tearing of subcutaneous tissue. There is no destruction of external surface in a contusion. Due to rupture of blood vessels, blood extravasates in to subcutaneous tissues and the margins are blurred. The size of a bruise varies from small pinhead sized to large collection of blood in the subcutaneous tissues. The more the force is used to inflict the injury, the more is the collection of blood and the bigger is the size of the resulting bruise (Figs 13.1 to 13.4).



Figure 13.1: Bluish bruise over the front of forearm



Figure 13.2: Bruise over the thigh



Figure 13.3: Black eye—bluish black bruising



Figure 13.4: Multiple abrasions and bruises over the thigh and abdomen

Factors modifying size and shape of bruise:

1. **Condition and type of tissues:** The softer the tissues, more easily they are likely to bruise.
2. **Age:** The persons who are old aged tend to bruise easily because of the loss of flesh and the age related cardiovascular changes in them. The children having a softer skin and delicate tissues also get easily bruised.
3. **Sex:** Women bruise more easily because of the delicate tissues and more of subcutaneous fat.
4. **Colour of skin:** Bruising is easily visible in fair skinned people compared to the dark skinned ones though the bruised area can be felt with palpation. In an embalmed body, the bruise becomes more prominent as the embalming fluid makes the skin lighter in colour and more of blood is forced into the damaged tissues and the embalming fluid makes a dark pigment complex. Photographs are quite helpful as they can highlight even those contusions, which are not clearly visible to the naked eye.
5. **Natural disease:** The appearance of a bruise varies with the various natural diseases such as: (i) Arteriosclerosis—Bruising may occur very easily even from slight coughing or exercise. (ii) Whooping cough—In children whooping cough causes violent bouts of cough, which may cause bruising without any direct external injury (iii) Conditions such as purpura, leukemia, hemophilia, vitamin C & K deficiency, phosphorous poisoning etc. lead to easy bruising and in purpura colour change is discernable without any swelling.

Antemortem and postmortem contusions:

Signs of inflammation such as redness, pain, loss of function are the hallmark of antemortem bruising and in addition, extravasation of blood is present in case of antemortem injury. Colour changes can be seen only in case of antemortem injury. On microscopic examination, white blood cells are present in antemortem bruises and after about 12 hours rupture of red blood cells and the release of iron pigments occurs. A bruise has to be distinguished from postmortem lividity, congestion of organs and artificial bruise (Tables 13.1 to 13.3)

Table 13.1: Differences between bruise and P.M. lividity

Findings	Bruise	P.M. Lividity
Cause	Blunt force	Engorged vessels
Cuticle	Damaged	Not damaged
Site	Any part	Dependent part
Appearance	Elevated	Not so
Edges	Ill-defined	Well defined
Cut Section	Clot cannot be washed	Blood inside vessel, easily washed
Colour	Change of colours	Bluish, no change
Pressure	Lighter	No effect

Table 13.2: Differences between artificial and true bruise

Findings	Artificial bruise	True bruise
Cause	Juice of plant	Blunt force
Situation	Accessible parts	Any part
Colour	Dark brown	Change of colours
Margins	Well defined	Ill defined
Shape	Irregular	Shape of the weapon
Swelling	Not present	Slight swelling
Contents	Serum	Blood
Itching	Present	Absent
Chemical test	Positive	Negative

Table 13.3: Differences between bruise and congestion

Findings	Bruise	Congestion
Cause	Force	Pathological condition
Colour	Change	No change.
Margins	Diffuse, ill-defined	Well-defined
On dissection	Extravasation of blood	Engorged vessels, blood inside

Medicolegal significance of bruise:

1. A bruise can be a simple injury.
2. When any vital organs are involved it may be a grievous hurt

3. Multiple bruises can also cause death
4. When the force is applied over the body by a weapon having definite patterns like belt, chain etc., it leaves a pattern simulating the size and shape of the object. This is called **patterned bruise** and is important from medicolegal point of view as from its examination, the type of weapon that has caused the injury can be known. This may connect the victim and the weapon and can indicate the nature of weapon.
5. *Nature of weapon*: The characteristics of bruise produced by different types of weapons/objects are: (i) Hammer, closed fist produces round bruise (ii) Rod, stick, and whip produces bruise in the form of two parallel linear hemorrhages (iii) In a broad and flat weapon like plank, the edges of plank may cause parallel lines separated by apparently normal tissue (iv) Ligature mark also forms a definite patterned bruise showing the pattern of woven, spiral ligature (v) Elliptical patterned bruise indicates teeth bite (vi) The tip of the shoe will also produce a definite patterned bruise (vii) Tyre tread marks in a road traffic accident produce the pattern of the tyre.
6. *Age of a bruise*: The age can be estimated by the characteristic colour changes that the bruise undergoes with the passage of time. These colour changes are due to breakdown of RBC's and release of different pigments. These colour changes in a bruise are:

Colour of bruise	Pigment	Time
Red	Haemoglobin	Immediate (24 hours)
Blue	Deoxygenated hemoglobin	1-3 days
Bluish black to brown	haemosiderin	4 days
Greenish	haemotoidin	5-6 days
Yellow	Bilirubin	7-12 days
Completely disappears	—	2 weeks

However, it is very difficult to estimate the exact age from the colour changes. Sub-conjunctival hemorrhages do not undergo the usual colour changes and becomes yellow from red. In old age and diseased it takes a longer time for the various colour changes

- to appear. The colour changes are sustained during CO poisoning and remain bright red.
- III. From the examination of bruises, the degree of violence can be estimated and the character and manner of production of injuries can be ascertained as different types of violence produce different types of bruises. In deaths due to manual strangulation thumb and finger impression in the form of bruises are seen over the neck. In sexual assault cases, the bruises may be present over breasts, inner sides of thigh and genitalia. From the pattern of bruises over arms, it can be known if the arms were grasped by the assailant and also the relative position of the assailant; front or behind can be known. If bruising is present over shoulder blades, it indicates pressure on the body against ground or some other hard surface.
 - IV. *Delayed Bruising*: The bruising at times does not appear immediately after the impact. This is due to the fact that the blood comes to the superficial layers after a delay of few hours or even days. This is known as delayed bruising and is commonly seen in abdomen, thigh etc. A deep bruise takes several hours to appear on the surface and very deep extravasations never appear on the surface. In casualty any injured person with no visible bruise should be advised to come after 24 hours to find delayed bruising.
 - V. *Shifting of the bruise*: Bruising may not necessarily appear at the site of impact. The blood gravitates through the loose areolar tissue to appear at a different place at the lower area. A few examples of bruising at sites distant from the point of injury are as given below.
 - Anterior cranial fossa fracture causes bruising of the eyes and the eyelids become black.
 - Fracture of the base of skull leads to accumulation of blood behind the ears.
 - In case of an injury on calf muscle blood shifts in between the muscle planes to the ankles.

ABRASION

In an abrasion, there is damage of the superficial epithelial covering of skin/mucus membrane due to impact of hard surface or weapon.

Mode of Production

An impact causing friction between weapon and body surface is the commonest mode of production of an abrasion. Direction of force may be horizontal or tangential causing scratches or graze abrasions or it may be perpendicular when it causes an imprint or pressure abrasion.

Features

These are superficial injuries with denudation of the epithelium. Later on a scab of dried blood is formed at the site. Gradually the granulation tissue forms beneath the scab and wound healing occurs.

Types

1. *Linear or Scratches:* These are caused by horizontal friction produced by pointed end of weapon. It is wider at the start and shows heaping of epithelium at the end. The features of linear abrasion depend on: (i) type of weapon (ii) direction of force and (iii) the relative position of victim and assailant.
2. *Graze or sliding:* These are produced by rubbing against a rough surface of an object or ground etc. Grazed abrasions are seen most commonly in vehicular accidents. It is wider and deeper at the beginning and there is thinning at the end. Heaping of epithelium is present and indicates the direction of the force. Its size and shape have no relation to the weapon.
3. *Pressure:* In this type of abrasion, the force is more or less perpendicular and its size and shape depends on the part of the weapon in contact. Examples include the ligature mark in hanging or strangulation and in throttling. Teeth bite marks are also pressure abrasions.
4. *Imprint or patterned:* They reproduce the pattern of weapon causing e.g, in vehicular accidents

patterned abrasion is caused by the impact by radiator grill, headlamp. In run over accidents, the tyre marks may also produce patterned abrasions (Figs 13.5 to 13.10).

MedicolEgal Importance

1. They are usually simple injuries, as they do not leave a scar and usually abrasions are not dangerous to vital organs.
2. They can be self-inflicted for leveling false charges against enemies.
3. The type of weapon can be identified from the examining the abrasion
4. Time of assault can be estimated by estimating the age of an abrasion
 - Fresh – No scab
 - 8-12 hours – Reddish scab
 - 2-3 days – Brown scab
 - 4-5 days – Dark brown scab
 - 6 days – Black scab, starts falling
5. Nature of offence: In sexual offences, abrasion are found around breast, inner side of thighs whereas in asphyxial deaths such as throttling there are abrasions over the neck.
6. Postmortem abrasions are yellowish parchment like and may be caused during transport.
7. Type of weapon can be identified and matched with the injuries found on the person.

LACERATED WOUND

When the tissues gets crushed or stretched beyond the limits of their elasticity thus tearing off leading to the formation of lacerated wound. The lacerated wound is caused by a blunt trauma. Thus, it may be caused in falls, in accidents, and in blows to chest, abdomen, head etc. by blunt object. The lacerations can be divided into split, stretch, avulsion and cut lacerations.

Split Lacerations

This is the type of laceration, which occurs due to crushing of the skin between two hard objects and leading to splitting of the skin. It is usually found on the areas of the body, which have subcutaneous bones like scalp, shin of tibia and

ulnar border of forearm. If these areas are hit with a hard object then it may crush the skin and the thin subcutaneous tissue and cause a split laceration. In these areas, the lacerated wounds may also appear clean cut and may simulate an incised wound. Such wounds are known as **incised looking lacerated** wounds. Even though they look like incised wounds but they are caused by blunt force on the areas where skin is closely adherent to bone and subcutaneous tissues is scanty like on the scalp, Jaw, pelvic bones (iliac crest) and eyebrows (Table 13.1).

Stretch Lacerations

This type of lacerations are caused by excessive stretching of the skin beyond which it causes tear of the skin. It is usually seen in run over accidents or in cases where a body part may be caught in the some moving machinery. These type of lacerations if very extensive can cause a flap of loose skin and tissue. The flap can depict the direction of the force applied on the skin and can



Figure13.5: Multiple abrasions over the face

be useful in establishing the manner of the injury.

Avulsion

This is the type of laceration in which there is separation of the skin from the underlying tissue. This is also caused in a similar manner as a



Figure13.6: Glazed abrasions



Figure13.7: Abrasions over the forearm



Figure13.8: Abrasions over the shoulder

stretch laceration but the force involved is much more severe and the area involved is comparatively larger. It causes flaying of the tissue, which is a type of crush injury. The shearing or grinding force



Figure13.9: Abrasions over the heel



Figure13.10: Multiple abrasion over the thigh

produced by a weight is the reason behind this type of injury.

Cut Laceration

This is caused by a heavy sharp edged instrument, which causes a deep and wide cut over the body tissues.

Characters of Lacerated Wound

The margins of a lacerated wound are irregular with bruising present all around. The deeper tissues are unevenly divided and the hair bulbs are crushed rather than being cut. The lacerated

wound is less hemorrhagic as compared to an incised wound. Foreign matter may be present in the wound. Direction of the force can be established but the shape and size of the wound may not correspond to the instrument (Fig. 13.11).

INCISED WOUNDS OR SLASH WOUNDS

The incised wounds are produced by sharp cutting weapons like knives, dagger or a razor and blade etc. Some authors restrict the term incised wounds to wounds that are longer than they are deep but some include stab wounds under this category. An incised wound that is longer than its depth is referred to 'cut' or 'slash'. The knife, razor blade, sword, cleaver, panga, bottle or broken glass pieces may inflict such cuts that are typically seen in fights when the assailant strikes out with a sweeping action rather than the thrusting (Fig. 13.12).

The force is delivered over a narrow area of the skin by a sharp edged weapon, which causes cutting of the skin. The edges are clear, well-defined and there is no bruising. Due to the elasticity of the skin, the width of the wound is greater than the edge of the weapon and the length is greater than the width and the depth. The length of an incised wound has got no relation to the cutting edge of the weapon. These wounds are spindle shaped as the skin retracts due to its elasticity more so in the middle of the wound. Gaping is more if underlying fibers are cut transversely or obliquely. Haemorrhage in an incised wound is more compared to a laceration or crush injury. The wound is deeper at the start as there is more pressure at that point than at the termination where it narrows down leaving only a superficial scratch which is known as tailing of the wound. The **tailing of the wound** can be evaluated for estimating the direction of the injury. When the weapon enters obliquely, then an oblique wound is created in which one flap is visible while the other one is undermined. This type of cut is known as a **beveling cut**.

Chop Wounds

Table 13.1: Difference between incised wound and lacerated wound

S. No.	<i>Incised wound</i>	<i>Lacerated wound</i>
1.	Any where in the body	Over bony prominences
2.	Edges are clean cut	Lacerated
3.	No bruising of margins	Bruising present
4.	Blood vessels, nerves clean cut	Crushed
5.	Hair bulbs clean cut	Crushed
6.	Bleeding more	Bleeding less

These types of wounds are produced by a blow with a sharp cutting edge of a heavy weapon like an axe, basola etc. In this type of injury, the dimensions of the wound correspond to cross-sections of penetrating blade. The margins are sharp with lacerated edges.

Hesitational Cuts

These are the cuts made by person with suicidal tendency. While attempting suicide these persons try to cut them to bleed to death. However, they hesitatingly make many superficial cuts before attempting a more deep cut. These hesitational cuts are typically multiple, superficial, parallel, uniform in depth and present on the accessible parts of the body.

Incised wounds are less dangerous than stab wounds as the relative shallowness of the wounds is less likely to affect a vital organ. The arms and the face are the common targets. Since the brain and chest organs are well protected by the ribs and skull, it is only the abdomen that is the target for a swung blade. A broken drinking glass is also used as a weapon, it may be broken prior to the attack to produce exquisitely sharp edges. The glass can inflict deep wounds almost akin to a stab wound. Where a glass or bottle is used as a blunt object, it may shatter on impact with the victim's head and the resulting injuries may be both blunt and incised.

CUT THROAT WOUNDS

Suicidal Cut Throat Wounds

Cut throat wounds are commonly suicidal in



Figure 13.11: Lacerated wound

manner and have a number of tentative cuts. If successful, there will be one or more deep incisions superimposed, which may destroy some of the previous shallow cuts. The classical description of the cut throat is of incisions starting high on the left side of the neck below the angle of



Figure 13.12: Multiple incised over the forearm

jaw, which pass obliquely across the front of the neck to end at a lower level on the right. This assumes that the victim is right handed, the obliquity being reversed in a left-handed person. The cuts are said to be deeper at their origin, becoming shallower as they cross the throat, tailing off into surface cuts at the extremity but commonly the cuts show no variation at the either end. Most suicides raise their chin to provide a better access to the throat, so that the skin is stretched out when cut. This tends to cause straight-edged incisions, rather than the jagged cut that is seen when a knife is drawn over the loose skin. Throwing back the head moves the carotid bundle under the protection of the sternomastoid muscles and if cuts are confined to the centre of the front of the neck, only the larynx or trachea may be damaged, rather than large blood vessels.

Homicidal Cut Throat Wounds

Cut throat wounds are also homicidal in manner when there are no tentative cuts. The wound is more deep and is may be found any where on the neck. Usually there are findings of presence of ligature material and underlying ligature mark over the neck (Fig. 13.13).

Cause of Death in Cut Throat Wounds

Death from a cut throat deepens on the nature and extent of local damage to the neck. Sever haemorrhages from the jugular veins, or less often the carotid arteries, may lead to death from exsanguinations. If the larynx or trachea is opened, then even relatively minor haemorrhage from local vessels may cause blockage of airways by blood and clot, though many slashed air-passage victims survive. A rare cause of death is air-embolism, caused by aspiration into cut jugular veins while standing or sitting with the neck at a higher level than the thorax. Many suicides either use multiple methods of suicide or may sometimes abandon the pain of cut throat for some other mode such as poison, burns or fall from height.

STAB OR PUNCTURED WOUNDS

A stab or punctured wound is a wound that is deeper than it is wide. Sharp pointed objects such as a knife, dagger, nails, needle, spear, arrow, screwdriver etc cause it. A stab wound can be produced by (i).Driving the object into the body or (ii) Falling against the object. A punctured wound is called *penetrating wound* when it enters the body cavity and once it passes through and through it is called a *Perforating wound* In a perforated wound, the entry is larger with inverted edges while the exit is smaller with everted edges. Though a stab wound may penetrate only the skin and subcutaneous tissue, more commonly fatal wounds are encountered in the practice of forensic medicine.

Nature of Stabbing Weapon

Most commonly, the knives are employed in producing a stab wound and their physical features are quite important in shaping the wound. The examination of the knife should include following:

1. Length, breadth and thickness of the blade of knife
2. Whether the knife is single edged or double edged
3. Whether the blade especially its extreme tip is sharp
4. In a single edged knife, the nature of the back edge
5. The face of the hilt guard adjacent to the blade
6. Is there any grooving, forking or serration of the blade
7. Note whether any foreign material sticking to the blade such as blood stains

Besides the knife, certain knife like weapons are used for stabbing including scissors, chisels, razor blades, swords and even sharpened screwdrivers. In addition, larger cutting instruments such as axes, choppers, *parangs*, machetes, shears, hayknives have been used as stabbing weapons. Spiked instruments such as ice-picks, hayforks, case-openers cause a typical looking wound. Some of these weapons are exquisitely sharp and the wounds made by them exhibit extremely fine



Figure 13.13: Cut throat injury

divisions of the tissues. To razor and razor blades may be added surgical and craft knives, often with a disposable blade. Equally sharp is broken glass and sometimes porcelain. Both sheet glass and smashed glass utensils can provide edges that equal or exceed surgical scalpels in their cutting ability. Smashed china cups and mugs can also provide sharp cutting edges.

Characteristics of Stab Wound

By examining the characteristics of stab wound, the forensic expert can provide opinion on (i) Dimensions of weapon (ii) Type of weapon (iii) The taper of the blade (iv) Movement of knife within the wound (v) Depth of the thrust (vi) Direction of thrust (vii) Amount of force used.

Dimensions of the Weapon in Relation to Characteristic of Stab Wound

The dimensions of the knife may be a vital part of investigation of a homicide when the weapon has been removed from the scene by the assailant. The examination of the suspected weapons recovered from the site, may indicate whether the dimensions are consistent with the injuries found on the body. For this purpose, the injuries found on the person of the victim should be thoroughly



Figure 13.13: Stab wounds over chest



Figure 13.13: Body showing multiple stab wounds on chest

examined under the following headings:

1. **Length:** Length of a stab wound is slightly less than width of the weapon due to elasticity of skin. It is measured to the nearest of a centimeters. In most instances, the wound will have gaped across the centre, to form a long ellipse. The extent of gaping will depend upon the anatomical situation, for example over joints or in the axilla or groin, and whether the axis of the stab is in line with or across the tension of Langer's lines or underlying muscle bundles.

The length of the wound should be again measured after opposing the edges that more accurately approximates the length when the blade was *in situ*. The length will be more if lateral movement of the weapon or the body occurs. When the knife is withdrawn the elasticity of the skin causes it to retract, making the wound smaller than when the blade was inside the body.

2. *Breadth*: The breadth is the second dimension on the surface of the body.
3. *Depth*: It is the length of the track and is the greatest dimension of the injury. In a patient, the injury should not be probed as there is danger of dislodging a clot causing more hemorrhage during life or it may cause a false track to be formed. Depth is equal to or less than the length of the blade producing it. Depth is greater on yielding surface such as anterior abdominal wall, rib cage and chest in young individuals. The depth of the wound and the force required depends on
 - i. *Nature of the weapon*: A sharp, slender weapon will pass more easily through the body while a blunt pointed weapon will require more force to penetrate the body.
 - ii. *Part of the body*: Soft body parts like abdomen can be pierced easily while parts with underlying bone like skull cannot be pierced and sustain only superficial cuts.
 - iii. *Age and bodily built of the injured*: Healthy adults with muscular bodies are less likely to sustain deeper injuries while children who have softer skin can sustain deep penetrating injuries with small amount of force.
To know the track of the wound it should not be probed with a metal probe, instead a pliable tubing should be used or a Radio opaque dye should be injected and then X-ray is taken to reveal the complete track. A hard and stiff metallic probe may dislodge a loosely formed clot and cause excessive bleeding during life.
4. *Margins*: The margins of a stab wound are clean cut when produced with a sharper weapon. The

margins are irregular in case a blunt pointed weapon such as a screw driver is used. A hilt mark of the knife may be seen at the angles of the wound indicating that the full length of the weapon has entered

5. *Shape*: If both the angles of the wound are acute, it has been caused by a double-edged weapon. While if one angle is more acute than the other, the weapon is a single-edged. A stab wound that cuts the cleavage lines of Langer transversely or obliquely will gape. A stab wound that runs parallel to the cleavage lines will be slit like. Shape of the wound usually corresponds to the weapon used but it varies at different parts of the body. Single edged weapon may give a triangular or wedge shaped wound in which one angle is sharp and the other angle is blunt. But when the weapon enters parallel to the cleavage lines, even though single edged it can be produce wound with two acute angles. Because of initial penetration by the knife point, it first produces a dermal defect with sharp angles at each end. Double-edged weapon can cause elliptical, slit like wounds in which both angles are acute. Round object or a spear may cause a circular wound. A round blunt pointed object may cause a circular wound with ragged margins whereas square weapon causes cruciform injuries. Ice-pick or screw driver causes injuries like small bullet holes and two "A" or "V" shaped stab wounds can also be seen. In a homicidal stab wound, the knife may be pulled upwards and downwards during insertion or withdrawal and the length is more than the width of the blade.
5. *Direction*: In solid organs the track is better seen. The principal direction is noted first and then the others backwards and to the right. One external wound and two or more internal wounds may be caused. In a perforating wound entrance, track and exit indicates the direction of the wound.
As far as the direction of the wound is concerned the pathologist should take care of the differences in the position of internal organs as measured at autopsy in a supine body

compared with their position in the living body that is often in an upright position. In the chest and abdomen the relationship of the various organs varies with the stage of respiration.

6. *Tapering of the blade:* The wound size depends on the taper of the blade. If the taper blade is inserted to 4 cm, the length of the wound is that of the width of the blade whereas in case of blade showing tapering at 8 cm level, the wound will be correspondingly longer. When the blade edges become parallel the wound size will remain constant for further penetration of the knife.
7. *Movement of the knife within the wound:* When the knife is stabbed directly into the body and withdrawn along the same track then the size of the wound will indicate the minimum width of the blade at the maximum depth of penetration. A knife that is thrust into the body may itself move or the body may move against it before it is withdrawn. This relative movement may extend the wound in a linear fashion or there may be twisting of the blade in the wound. In the latter case, the resulting skin incision may be V-shaped or is totally irregular. A single stab can also produce multiple skin wounds such as stab over the arm that enters the superficial tissues and then reenters the chest wall.
8. *Depth of the thrust:* To assess the length of the weapon the depth of the stab wound is important. Certain points should be observed
 - i. When the knife is driven inside the body up to the hilt, depth of the wound when measured at autopsy may be greater than the true length of the blade. This is quite common in the injuries on abdomen and rarely chest because the impact of stab may indent the chest or abdominal wall so that the tip of the knife penetrates tissues that apparently should have been out of the reach. This particularly happens when the hiltguard of the weapon impinges on the skin.
 - ii. *Direction of thrust:* Determination of the direction depends on the appearance of the skin wound and the track in deep tissues.

Where a knife penetrates the skin with the plane of the blade at appreciably less than a right angle to the surface, the wound is often undercut being shelved so that subcutaneous tissue is visible below one edge of the wound. Where the knife is plunged in obliquely but the plane of the blade remains perpendicular to the surface, no such shelving can be seen at the site of the wound though it may just be visible at the end of the wound. The track of the stab wound can further inform on to the direction. Attempts have been made to delineate the track of the stab wound before dissection either by filling by the defects with the radio-opaque fluid before taking X-rays or by filling by the plastic or even metallic substance that will harden to form a cast. These methods have a very little advantage over routine dissection methods.

9. Estimation of the degree of force used in stabbing: The assessment of force used in the stabbing is subjective and cannot be quantified in any satisfactory way that is meaningful to the court of law.
 - i. This tissue most resistant to the knife penetration is the skin followed by muscle apart from the bone or calcified cartilage.
 - ii. For skin penetration, the sharpness of extreme of knife is the most important factor. The cutting edge of the knife once the knife is penetrated has no significance.
 - iii. In achieving penetration the speed of approach of knife is important as when a knife held against a skin and steadily pushed requires more force than the same knife launched against the skin like a dart.
 - iv. It is difficult for the knife to penetrate a lax skin compared to a stretched skin. The chest wall is easy to penetrate to sharp knife as there is stretching of intercostal spaces.
 - v. The thick of the palms and soles is tough to penetrate than the rest of the body.
 - vi. When the knife penetrates skin rapidly as when the body falls or runs on to the blade,

the knife does not need to be held rigidly in order to prevent it being pushed backwards.

- vii. Uncalcified cartilages such as costal cartilages are easily penetrated by sharp knife. Calcified rib and bone provide a resistant barrier but a forceful stab from strong, sharp knife can easily penetrate sternum, skull or rib.

MedicoLegal Importance

1. The shape of the wound may indicate the type of weapon
2. The depth will indicate force of penetration as in homicidal injuries more force is used compared to suicide.
3. By the direction and dimensions the relative position of the victim and assailant can be known.
4. The age of injury can be evaluated.
5. The position and number of injuries can indicate whether it is homicidal, suicidal or accidental.
6. If broken fragment of the weapon is available it can identify the weapon (Tables 13.2 and 13.3).

RAILWAY INJURIES

Railway injuries are quite common in countries with many 'level crossings' where a public road crosses a railway track with either no barrier at all or with only a flimsy lifting pole. Few rail passengers are killed or injured in moving trains compared with accidents on railway property. Track workers may be run down and some may die from electrocution from overhead cables. The person to commit suicide lays himself in front of an approaching train. Decapitation is the most common injury with features of local tissue destruction, usually with grease, rust or other dirt soiling of the damaged area. In railway accidents the viscera should be analysed for alcohol and other drugs as suicides often employ multiple methods for committing suicide. Sometimes the individual is killed and the body is thrown over the railway track to mimic an accident but in such circumstances, the presence of coexistent injuries such as strangulation mark over the neck will be evident (Fig.

13.14).

INJURY REPORT

The patient in a medicolegal case should be examined by the doctor for preparation of medicolegal case report after receiving a written requisition from the investigating police officer. The preliminary details of the patient should be noted such as:

1. Name
2. Age, sex
3. Address
4. History
5. Brought by
6. Identified by
7. *Identification marks*: Two permanent identification marks preferably on exposed parts of the body that would help in identification of the victim in Court of law should be noted.
8. *Consent*: An informed consent of the patient should be recorded describing the procedure along with its risks and consequences. The doctor can be charged of assault if examination is attempted without prior consent. The consent should then be signed or the thumb impression applied by the patient in presence of a witness preferably third party. The signature of the witness should also be recorded.

The injury report should describe the following in detail:

1. General physical examination noting the vitals of the patient as well as his systemic examination.
2. Examination proper that includes:
 - i. Nature of each injury.
 - ii. Size, shape and direction of the injury.
 - iii. Part of the body on which injury is present (site).
 - iv. Whether injury is simple or grievous.
 - v. The type of weapon used.
 - vi. Whether weapon is dangerous or not.
3. Opinion: The opinion should include the nature of injury whether it is simple or grievous, the time passed since the infliction of the injuries, the type of weapon producing it. In addition, if any investigations are mandatory such as radio-

Tables 13.2		
<i>Findings</i>	<i>Suicide</i>	<i>Homicide</i>
Position of wound	Accessible parts	No fixed site, vital parts
Grouping	Arranged	Irregular
No. of wounds	One	Multiple
Direction	In right handed persons, left to right	Any direction
Severity	Mostly superficial, 1-2 fatal	Mostly severe an extensive
Defense wounds	Absent	Present
Secondary injuries	Absent	May be connected with fight
Weapon	Present	Absent
Clothes	Not damaged	Damaged
Motive	Domestic worries, disappointment in love	Revenge, robbery, sexual assault

Tables 13.3			
<i>Sl. No.</i>	<i>Findings</i>	<i>Antemortem Injuries</i>	<i>Postmortem Injuries</i>
1.	Edge	Swollen, everted, retracted, gaping	Absent
2.	Hemorrhage	Profuse	Slight, venous
3.	Spurting	Arterial flow, over clothing	No spurting
4.	Extravasation of blood	Staining of the edges of wound, by washing cannot be removed	Not deeply stained, removed by washing
5.	Coagulation	Firm and difficult to wash	Soft clot easily removed
6.	Vital reaction	Swelling, reddening, and repair	No such process
7.	Enzyme	- Esterase, acid phosphates	No such reaction

logical examination in firearm cases, preservation of blood for grouping, examination of clothing should be mentioned in the report itself. Finally the report bears the signature of the doctor along with the date.

Long Questions

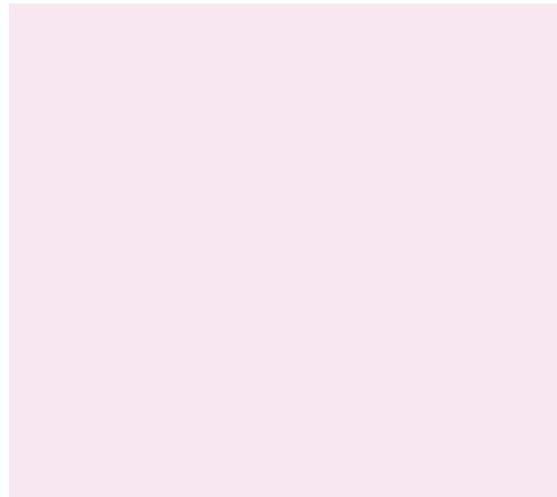
1. Discuss in detail the injuries caused by a blunt weapon. Enumerate the differences between the various types of injuries and the age of the injuries.
4. How can you classify injuries? Discuss grievous hurt. How can you estimate the time of injuries?



Figure 13.14: Decapitation of head

Short Questions

1. Abrasion
2. Contusion
3. Laceration
4. Stab wounds
7. Ageing of a injury



MULTIPLE CHOICE QUESTIONS

1. **Colour of subconjunctival haemorrhages changes from red to:**
 - A. Blue
 - B. Green
 - C. Yellow
 - D. Black
2. **Tentative cuts are seen in:**
 - A. Homicide
 - B. Fall from height
 - C. Suicides
 - D. Accidents
3. **Sites notorious while looking for incised looking lacerated wounds are all except:**
 - A. Zygoma
 - B. Skin
 - C. Iliac chest
 - D. Chest
4. **The colour of postmortem abrasion is:**
 - A. Red
 - B. Yellow
 - C. Black
 - D. Reddish-brown

5. The exact age of the abrasion can be assessed by:
A. Histological method
B. Histochemical method
C. Naked eye examination
D. None of the above
6. In cases of stab wounds:
A. Depth is more than length and breadth
B. Length is more than depth
C. Breadth is more than length
D. All dimensions are equal
7. The colour of the bruise becomes green on:
A. 2nd day B. 3rd day
C. 4th day D. 6th day
8. The depth of an incised wound is:
A. Equal throughout
B. Deeper at the beginning
C. Deeper at the end
D. Uneven throughout
9. Which of the following is not an example of pressure abrasion?
A. Ligature mark in hanging
B. Ligature mark in strangulation
C. Finger nail marks
D. Teeth bite
10. A reddish scab is formed in an abrasion in about:
A. 2-3 hours
B. 6-8 hours
C. 8-12 hours
D. 12-24 hours
11. In an incised wound:
A. Breadth is more than length
B. Length is more than depth and breadth
C. All are equal
D. Depth is more than breadth
12. Latent bruises can be detected by:
A. Colour photography
B. U-V light
C. Oblique light
D. Infra Red light
13. The direction of a stab wound indicates:
A. Type of weapon
B. Force of penetration
C. Relative position of assailant and victim
D. Stretching of the tissues
14. Blunt force injury will produce incised looking wounds over:
A. Forehead B. Mandible
C. Skin of Tibia D. All of the above
15. Stretch laceration are caused due to:
A. Run over vehicular accident
B. Grinding force by a weight
C. Injury of axe
D. Pieces of glass
16. Blunt objects may cause:
A. Contusion B. Laceration
C. Abrasion D. All of the above
17. Defence wounds are found in cases of:
A. Accidental injuries
B. Homicidal injuries
C. Suicidal injuries
D. Self inflicted injuries
18. Grievous injury is:
A. Disfiguration of face due to injury
B. 14 days hospital stay
C. 20 days hospital stay
D. Permanent impairment of hearing.
19. Lacerated wound looks like incised wound at:
A. Scrotum
B. Thigh
C. Bone with thin overlying tissue
D. Abdomen
20. At 4th day, the colour of the bruise will be:
A. Bluish-black B. Blue
C. Green D. Yellow
21. Margins of a clear incised wound adhere in:
A. 6 hours B. 12 hours
C. 18 hours D. 24 hours
22. Lacerated wound resembles incised wound on:
A. Scalp
B. Thigh
C. Gluteal region

- D. Anterior abdominal wall
23. Which of the following feature does not differentiates antemortem bruise from postmortem hypostasis?
 A. Variegated in colour
 B. Clearly defined margins
 C. Present anywhere in body
 D. Extravasated blood in surrounding tissues which cannot be easily washed away
24. Lacerated wound looks like incised wound at:
 A. Forehead B. Scrotum
 C. Abdomen D. Thigh
25. Antemortem fractures differ from postmortem fracture by:
 A. Effusion of blood
 B. Comminuted type
 C. No sign of injury over skin
 D. Lack of oedema
26. Postmortem wound best differs from the antemortem wound by:
 A. Gapes on incising
 B. No clots
 C. Absence of erythema and cellular changes
 D. Stain can be removed by washing
27. Tailing of wound indicates:
 A. Nature of weapon used
 B. Direction of force applied
 C. Whether suicidal or homicidal
 D. Is of no consequence
28. Greenish discolouration of bruise is due to:
 A. Staphylococcus
 B. Streptococcus
 C. Klebsiella
 D. Hematoidin pigment
29. Which of the following statement is true about stab wound?
 A. Has entry and exit wound
 B. Breadth is maximum
 C. Depth is more than length & breadth
 D. Has irregular margins and broader than edge of weapon
30. Antemortem bruise is identified best by:
 A. Colour changes B. Bleeding
 C. Histology D. None of the above
31. Most dangerous wound is:
 A. Gun shot in head
 B. Stab wound abdomen
 C. Sucking wound in chest
 D. Fracture of femur
32. Greenish appearance of bruise is seen on:
 A. 3rd day B. 5th day
 C. 9th day D. 15th day
33. The wound is antemortem if:
 A. Blood is clotted
 B. Spouting present
 C. Extravasation is present in edges and surrounding areas
 D. All of the above
34. Avulsion is a type of:
 A. Chop wound B. Incised wound
 C. Laceration D. None of the above
35. 'True bruise' is distinguished from 'artificial bruise' by:
 A. Itching is present
 B. Present at accessible site
 C. Irregular shape with ill-defined margins
 D. Inflammatory reaction present at site of bruise
36. If there is bluish colour of a bruise, then the age of injury is:
 A. 1 day B. 2 days
 C. 5-6 days D. 14-28 days
37. Postmortem fractures differ from antemortem fractures by all except:
 A. Absence of bleeding
 B. Absence of callus
 C. Absence of granulation at fracture site
 D. Laceration over skin
38. Colour of subconjunctival haemorrhages changes from red to:
 A. Blue B. Green
 C. Yellow D. Black
39. Tentative cuts are seen in:
 A. Homicide A. Fall from height

- A. Suicides A. Accidents
40. Sites notorious while looking for incised looking lacerated wounds are all except:
A. Zygoma B. Skin
C. Iliac chest D. Chest
41. The colour of postmortem abrasion is:
A. Red B. Yellow
C. Black D. Reddish-brown
42. In cases of stab wounds:
A. Depth is more than length and breadth
B. Length is more than depth
C. Breadth is more than length
D. All dimensions are equal
43. The colour of the bruise becomes green on:
A. 2nd day B. 3rd day
C. 4th day D. 6th day
44. The depth of an incised wound is:
A. Equal throughout
B. Deeper at the beginning
C. Deeper at the end
D. Uneven throughout
49. Which of the following is not an example of pressure abrasion?
A. Ligature mark in hanging
B. Ligature mark in strangulation
C. Finger nail marks
D. Teeth bite
50. A reddish scab is formed in an abrasion in about:
A. 2-3 hours B. 6-8 hours
C. 8-12 hours D. 12-24 hours
60. In an incised wound:
A. Breadth is more than length
B. Length is more than depth and breadth
C. All are equal
D. Depth is more than breadth
61. Latent bruises can be detected by:
A. Colour photography
B. U-V light
C. Oblique light
D. Infra Red light
62. The direction of a stab wound indicates:
A. Type of weapon
B. Force of penetration
C. Relative position of assailant and victim
D. Stretching of the tissues
63. Blunt force injury will produce incised looking wounds over:
A. Forehead B. Mandible
C. Skin of Tibia D. All of the above
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A. Run over vehicular accident
B. Grinding force by a weight
C. Injury of axe
D. Pieces of glass
65. Blunt objects may cause:
A. Contusion B. Laceration
C. Abrasion D. All of the above
66. Defence wounds are found in cases of:
A. Accidental injuries
B. Homicidal injuries
C. Suicidal injuries
D. Self inflicted injuries

Regional Injuries

HEAD INJURIES

Anatomy of Scalp

The scalp extends from the top of forehead in front to the superior nuchal line behind. Laterally it projects down to the zygomatic arch and external acoustic meatus. It consists of five layers: (i) skin (ii) subcutaneous tissue (iii) occipitofrontalis and its aponeurosis (iv) subaponeurotic areolar tissue and (v) pericranium (Figure 15.1).

INJURIES TO SCALP

The usual range of abrasion, contusions and laceration may be inflicted though a modifying

factor that is presence of brain, which may deflect a tangential blow or partly cushion a direct impact. In hair-covered areas, care must always be taken at autopsy to palpate the scalp in any case in which there is possibility of injury that otherwise be missed. When a lesion is found or suspected, the hair must be carefully shaved away to expose the scalp for further examination and photography.

Bruising of Scalp

Bruising may be difficult to detect until the hair has been removed. Marked swelling is a common feature of extensive bruising, as the liberated blood cannot extend downward because of the rigidity

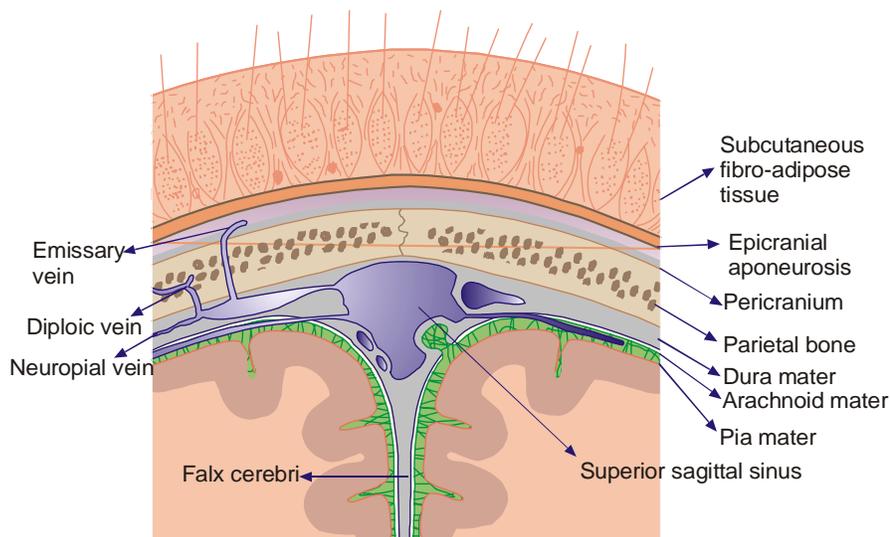


Figure 15.1: Coronal section through scalp, skull and brain

of underlying skull. In addition to frank bleeding beneath the scalp, marked oedema may occur after injury and the layers of the scalp may be greatly swollen and thickened by jellylike infiltration of tissue fluid. Bleeding under the scalp (Figure 15.2) may be mobile. A bruise under anterior scalp may slide downwards to appear in orbit. Similarly, a temporal bruise may later appear behind the ear. As with bruises elsewhere, those under the scalp may be obvious immediately after infliction or their appearance may be delayed either during life or as a postmortem appearance.



Figure 15.2: Effusion of blood underneath the scalp with diastatic fracture of skull

Laceration of Scalp

Laceration of scalp bleeds profusely and dangerously and even fatal blood loss can occur from an extensive scalp injury. The injury that is most gross is avulsion of a large area of scalp. This may happen when hair is entangled in machinery while working in industries.

Scalp injuries may bleed profusely even after death, especially if head is in dependent position. Severe blows from shaped object such as hammers or heavy tools may reproduce the profile of weapon totally or in part. A circular faced hammer may punch a circle but more often only an arc of circle is seen. In such cases, position of edges that digs in most deeply may give an

indication of the angle of blow. A major problem in scalp injuries is differentiation between incised wound and laceration. Close examination using a lens if necessary will show that this blunt laceration has (i) bruised margins, even though this zone may be narrow (ii) head hair crossing the wound are not cut (iii) fascial strands, hair bulb and perhaps small nerve and vessels are in the depth of wound.

Scalp Injuries from Fall

From the fall, the injuries usually involve the occipital protuberance, forehead or the parieto-temporal area and are not possible on the vertex even from a considerable height. Falls from standing position can occur if the person is drunk, in an assault and during some illness (fit/faint).

Complications of Scalp Injuries

1. Deep bruise may take a longer time to appear and heal.
2. The infected lacerated wound may cause infection of superior sagittal sinus through the emissary veins.

Black Eyes (Figs 15.3 and 15.4)

Black eye is also called periorbital hematoma and may be the result of

1. *Direct violence:* It may be associated with abrasion or laceration of upper cheek, eyebrow, nose or other part of the face.
2. *Gravitational seepage:* An injury to front of scalp and also on or above the eyebrow leads to gravitational seepage of blood beneath the scalp in to the eyelids.
3. *Percolation of blood:* This results from fracture of anterior cranial fossa of skull, is often a contrecoup injury caused by fall on to back of head leading to secondary fracture of paper-thin bone of orbital roof.

Anatomy of Skull

Adult skull consists of two parallel, thin tables of compact bones; outer being twice thicker than



Figure 15.3: Black eye in a case of head injury



Figure 15.4: Black eye with abrasion over the forehead

inner, separated by soft cancellous (spongy) bone containing bone marrow. The term 'diploe' is used to describe this cancellous bone within the flat bones of skull. The diploe starts forming at 4 years of age. Before this age, it is a thin compact and elastic bone. This zone is interrupted at suture lines and vanishes where the bone is particularly thin especially in floor of skull. Greater wing of sphenoid, petrous temporal, sagittal ridge, occipital protuberance and glabella are the strong parts of cranium. The thin areas are parieto-temporal, lateral frontal and lateral occipital zones. The temporal bone is the thinnest with a thickness of 4 mm and occipital is thickest, in midline may

be more than or equal to 15 mm. The frontal and parietal bones are about 6-10 mm in thickness.

FRACTURES OF SKULL

Fatal brain damage can occur even with an intact skull. Thin skull is less likely to be fractured as it is flexible and can return to normal after distortion like that of infants. It is rare for the skull fracture itself to be dangerous but the concomitant effect of transmitted force upon cranial contents makes it dangerous. The presence of skull fracture is an indication of the severity of force applied to the head. Postmortem and antemortem differentiation is difficult in the absence of soft tissue. The fracture of skull can occur either by direct or indirect violence.

Direct violence: The forces act directly on the bone to produce a fracture such as:

1. Compression of foetus during forceps delivery or clinical use of a cranioclast.
2. Moving object like stone/bullet strikes the head.
3. Head crushed under wheels of a moving vehicle in road traffic accident.
4. **Punch drunk**—Due to repeated blows by boxers, the victim presents with findings that may be mistaken for drunkenness.

Indirect violence: Here the forces act indirectly on the skull through some other structure, which receives primary impact such as

1. Fall on buttock from height transmits force through the vertebral column to occipital bone producing ring fracture
2. Blast explosions of face from below
3. Heavy blows on chin when the force is transmitted to skull with resulting fracture of base of skull.

Mechanics of Skull Fracture

Rowbotham has described direct and indirect injuries to skull.

Direct injury to skull: Direct injuries can be due to local or general deformation such as:

1. *Fractures due to local deformation (Struck hoop analogy):* When skull receives a focal

impact, there is momentary distortion of the shape of cranium. Infants skulls are more pliable and flexible and more liable for which more distortion occurs than the more rigid adult skulls. The area under the point of impact bends inwards, contents of skull being virtually incompressible, a compensatory distortion or bulging of other areas occurs called the **struck hoop analogy**. Skull is more susceptible to traction forces than the compression, so convexities fracture during distortion of 'struck hoop'. When skull is deformed, compression occurs on the concavity of the curved bone and tension (tearing) forces on the convexity. If traction forces exceed the elastic threshold, fracture occurs. Thus inner table will fracture where skull is indented and outer table will fracture at the margins of deformed area. If forces are great enough, depressed comminuted fracture results.

2. **Fractures due to general deformation:** Skull is compressed like elastic sphere. When skull is deformed, compression occurs on the concavity of curved bone and tension (tearing) force on convexity. If tearing force exceeds the limit of elasticity, fracture results. The inner table will fracture where skull is indented and outer table will fracture at the margins of deformed area. If forces are great enough, comminuted fracture occurs.

Indirect injury to skull: Here, the forces act away from the body that is chin, feet and buttocks resulting in skull fracture. This could result from:

1. **Gurdijian** gives the hypothesis for the purpose of linear fracture, which develops, in the areas of stress. When force is applied to the skull at a particular point, in bending of skull occurs at a point of impact and stress areas develop at places away from the point of impact and result in linear fracture, which may extend to join the point of impact. These stress areas may be primary, secondary or tertiary.
2. A heavy impact on the side of the top of head often leads to fracture of the vault running into base of skull usually across the floor of middle cranial fossa along the anterior margins of petrous temporal to enter the pituitary fossa.
3. Sometimes, fractures are initiated at a distance away from the point of impact due to compensatory deformation but running usually back towards the impact site. In major injuries, this fracture may often cross the floor of skull to form a hinge fracture.
4. A blow or fall onto the occiput may produce a fracture passing typically vertically or obliquely downward just to the side of midline of posterior fossa commonly reaching foramen magnum and causing ring fracture.
5. When severe local impact causes focal and general deformation, combination of depressed fracture and radial fracture lines may form a 'spider web' pattern.
6. **Puppe's rule**—When two or more separate fractures occur from successive impacts and meet each other the later fracture (Y) will terminate at, that is not cross, the earlier fracture line (X) which naturally interrupts the cranial distortion which proceeds fracturing (Figure 15.5).

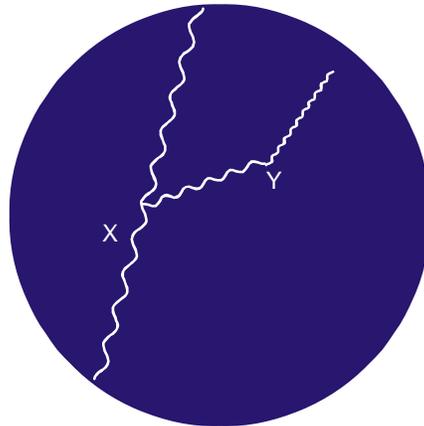


Figure 15.5: Puppe's rule

Types of Skull Fracture

- I. *Linear (fissured fracture)*: Linear fracture is commonly missed radiographically and is found at autopsy. The fracture is straight or curved lines often of considerable length. They either radiate out from a depressed zone or arise under or at a certain distance from impact area from bulging deformation. It usually involves either table of skull, sometimes both inner and outer. The fractures are common in weak unsupported areas like temporal, frontal, parietal and occipital. The fracture line may extend towards foramen magnum to the base of skull, across the supraorbital ridges or in to the floor of skull. In children and young adults a linear fracture may pass in to a suture line and causes a 'diastasis' or opening fracture of the weaker seam between the bones. The fracture is direct or 'stepped'. It is also called 'motorcyclist's fracture' (Figs 15.6 to 15.7).
- II. *Diastasis (sutural fracture)*: It is a linear fracture, which in children and adults passes in to a suture line and cause diastasis or opening of weaker seam between sutures. The most commonly involved suture is sagittal and even the metopic suture may reopen. This type of fracture is common in child abuse syndrome.
- III. *Ring fracture*: Occurs in posterior cranial fossa and foramen magnum. It is most often caused by fall from height on to the feet. If kinetic energy of fall is not absorbed by fracture of legs, pelvis or spine, impact is transmitted up the cervical spine. This may be rammed in to the skull, carrying a circle of occipital bone with it.
- IV. *Pond fracture*: The pond fracture is shallow depressed fracture forming a concave 'pond'. The fracture is more common in pliable bones of infants. Depression occurs in absence of fracture akin to distortion produced by squeezing a table tennis ball.
- V. *Mosaic (spider web fracture, Fig. 15.8)*: A comminuted depressed fracture may also



Figure 15.6: Linear fracture



Figure 15.7: Linear fracture of skull



Figure 15.8: Comminuted fracture of base of skull showing spiderweb pattern

have fissures radiating from it forming a spider web or mosaic pattern. This results when severe local impact causes focal and general deformation of the skull.

- VI. *Depressed fracture (signature fracture)*: When the focal impact is severe, it causes outer table to be driven inwards and unless absorbed in the diploe, inner table will be intruded in to cranial cavity with all the dangers of direct damage to the contents. Sharp-edged weapons such as heavy knives and axes may cause this type of fracture. The deepest part of the depression will indicate where the weapon first struck, there may be 'terracing' of the margin (Fig. 15.9).
- VII. The presence of hair and scalp markedly cushions the effect of impact over the vault so that a far heavier impact is required to cause the same damage compared to base of skull.



Figure 15.9: Vault of skull showing depressed fracture

Force required for causing skull fracture

1. Tensile strength of adult skull is 100-150 psi and compressive strength varies from 5000 to 31000 psi. A force of about five foot-pounds can cause a simple fissured fracture of the skull. Falling to the ground from an erect posture develops at least 60 ft./lb and can easily produce skull fractures. A small stone or golf

ball weighing about 100 gms may cause a linear fracture or when thrown with moderate force against the temporal bone.

2. The adult head weighs 3-6 kgs averaging 4.5 kgs, when falling through about one meter so frontal area strikes hard surface, impact energy of about 35 ft/lb develops that can cause one to two linear or mosaic fracture. Great variations occur in this, the area of skull struck, the thickness of the skull, scalp and hair, direction of impact all affect the outcome.

Complications of Fracture of Skull

1. *Injury to brain*: The fracture of the skull causes the injury to the brain that is dangerous to life.
2. *Haemorrhage*: If fracture passes through embedded meningeal artery haemorrhage results that is fatal.
3. *Traumatic epilepsy*: Late effect of depressed fracture of skull is traumatic epilepsy that usually manifests as tonic or clonic fits, which may be difficult to differentiate from idiopathic epilepsy. Usually when fit starts one or two year after major head injury in otherwise fit individual then the diagnosis is easy. There is usually a depressed fracture impinging on underlying cortex often in parieto-temporal area. It is more common in open head injuries with infection.
4. *Infections*: Infections following skull fractures can be by (i) direct spread through compound fracture (ii) by spread from nasal cavity when fracture of cribriform plate is allowed communication to anterior fossa and (iii) by spread from fracture that involves a paranasal sinus such as frontal or ethmoid or from the mastoid air cells or middle ear cavity.

TRAUMATIC INTRACRANIAL HAEMORRHAGES

The intracranial haemorrhages are of the following types:

Extradural haemorrhage

Bleeding between inner surface of skull and duramater is least common of three types of brain

membrane haemorrhage. It is also known as epidural haemorrhage. According to Rowbotham, only 3% of head injuries have epidural haemorrhage. The dura is closely applied to the interior of the skull except in the posterior fossa, that extradural bleeding does not occur over the skull floor. Most extradural haemorrhage is associated with fractures of the skull. About 10% of extradural haemorrhage is associated with subdural haemorrhage. The usual site is unilateral in a parieto-temporal area caused by the rupture of middle meningeal artery that run between the dura and the skull (Figs 15.10 and 15.12).



Figure 15.10: Extradural haemorrhage



Figure 15.11: Extradural haemorrhage underneath the depressed fracture of skull



Figure 15.12: Extradural hematoma in a case of road traffic accident

Hume Adams suggests that a minimum volume of 35 ml is needed before clinical signs are apparent though other writers suggest 100 ml is usually the minimum associated with fatalities. Leakage of high-pressure arterial blood strips back the underlying dura with progressive accumulation of a hematoma. When bleeding is venous, the hematoma rarely reach a large size as pressure is insufficient to tear back much of dura. The clinical signs of an epidural haemorrhage are clinically those of a **lucid or latent interval**, as there may be recovery from the initial phase of concussion before. Typically the patient recovers consciousness from the initial trauma only to slip back into a progressively deepening coma. Sufficient blood accumulates to cause raised intracranial pressure and consequent relapse into unconsciousness. In Rowbotham's series, the range of latent interval was from two hours to seven days, but most were apparent after 4 hours. Extradural hematomas are surgical emergencies that if not drained will produce uncaline herniation, tonsillar herniation, medullary compression, respiratory arrest and death rapidly.

Heat Hematoma: This artefact mimics an extradural haemorrhage, when head has been exposed to severe external heat sufficient to burn the scalp and perhaps skull. Blood may be

extruded from diploe and venous sinuses into the extradural space to produce a 'heat hematoma'. It contains about 100-120 ml of blood. This hematoma is brown and friable and adjacent brain shows hardening and discolouration from heat. The hematoma shows honey-comb appearance and is pink in colour due to high levels of carboxyhaemoglobin. Level of carboxyhaemoglobin in the hematoma in cases of fire can give us the idea about whether the bleed is true extradural hematoma due to blunt force/surface impact or is heat hematoma. If level of carboxyhaemoglobin is same in both hematoma and blood, then it is probably heat hematoma. If there is, little or no carboxyhaemoglobin the bleed is due to head injury.

Subdural Haemorrhage

Bleeding into relatively wide space between the dura and arachnoid membrane is much more common than extradural haemorrhage. It results from the rupture of the bridging veins that connect the venous system of the brain to the large intradural venous sinuses. Because the brain, in its bath of CSF, can move within the skull but the venous sinuses are fixed, the displacement of brain that occurs in trauma can tear some of these delicate veins at the point where they penetrate the dura. It is less often associated with fracture of skull. As subdural hematomas depend for their origin on movement of the brain rather than the direct application of force, they are the reflection of inertial rather than impact forces. Consequently, they occur most commonly where the freedom of movement is the greatest, that is over the convexities of the hemispheres (Figs 15.13 and 15.14). They are relatively uncommon where little relative movement is possible, such as the posterior fossa.

Subdural haemorrhage can occur at any age but is common at extremes of lives. Subdural haemorrhage with long bone fracture is typical of child abuse as seen by Caffey. In old people, these haemorrhages exist in chronic form. The condition



Figure 15.13: Subdural haemorrhage



Figure 15.14: Subdural haemorrhage with fracture of skull

is always due to trauma. In alcoholics and people with bleeding diathesis, minimal trauma can start the bleed. The lesion is traditionally classified into acute, the subacute and the chronic.

Acute subdural haemorrhage: The classical feature is presence of lucid (latent) interval. Since the bleeding in acute subdural haemorrhage is of venous origin and at lower pressure, onset of symptoms is delayed. The lesion is manifested by gradual decline in level of consciousness and focal signs rarely.

Chronic subdural haemorrhage: Older people and chronic alcoholics are commonly the victims of chronic subdural haemorrhage. In these patients, there is usually some atrophy of brain and consequent upon this, an increased range of movement of the brain within the skull. Thus there is a higher risk of rupturing the bridging veins and bilateral subdural hematomas may develop following slowly following an insignificant or unnoticed trauma. As they develop relatively slowly, the brain is able to accommodate to their mass effect. Thus these hematomas produce much greater cerebral distortion and herniation than acute subdural hematomas before symptoms and signs develop.

Gross appearance of subdural haemorrhage varies with age. Recent lesions up to several weeks old are tan or brown with gelatinous membrane covering the surface. The contents are thick but liquid and may have area of redder more recent bleeding. An older hematoma up to months or even a year old is firmer with a tough membrane around both surfaces, resembling a rubber hot water filled with jelly or oil. The contents are liquid and may be brown or even straw coloured. Some-time variegation in colour is seen due to bleeds of different ages. Many subdural bleeds in old people are small and give rise to no neurological defects. Hence, the mere finding of such small lesion at autopsy should not be used to provide cause of death.

The chronic hematoma may become large and press down on cerebral hemisphere sufficiently to dent or distort the surface. This may progress and give rise to sign of hippocampal and cerebellar tonsillar herniation and dangers to vital center in the brainstem. The chronic hematoma arises from the center lesion, which after an interval becomes sheathed in a capsule of connective tissue. The mechanism of enlargement is controversial; (i) one theory is repeated bleeding from new blood vessels that enter the mass as part of healing process and (ii) other theory involves osmosis, in which because the center of haemorrhage commonly liquefies forming haemorrhagic fluid that

osmotically attracts into it the CSF from outside the capsule.

Dating of Subdural Haemorrhage

Gross appearance:

1. Within few hours: Reaction to subdural bleeding begins within a few hours of onset, when cellular infiltration begins from dural surface.
2. A subdural hematoma gradually changes colour from dark red to brownish colour, first being apparent not before five days and sometimes not obvious for 10-12 days.
3. Discrete surface membrane becomes obvious by 2nd week.
4. Liquefaction of contents occurs after more than 3 weeks.
5. After one month, a firm capsule forming a cystic cavity containing a dark brown watery fluid is formed.

Histological appearance: A delicate 'neo-membrane' histologically composed of thin-walled capillaries and fibroblastic granulation tissue, grows from periphery to cover outer surface of the clot during next few days and weeks. If no further enlargement occurs, this capsule becomes more and more fibrous. According to Crompton the presence of a membrane firm, enough to be picked off with forceps makes subdural haemorrhage at least 12 days old. There are claims made for accurate dating of subdural hematomas by histological criteria given by Monro & Meritt. They are briefly summarized under Table 15.1.

Subarachnoid Haemorrhage

The third type of brain haemorrhage bleeding is even more common than subdural haemorrhage. It is usually associated with extradural or subdural haemorrhage, sometimes it can occur as pure lesions. It sometimes occurs as a result of natural disease especially ruptures of vascular malformation of several types.

Appearance and mechanism of formation: The appearance of subdural haemorrhage caused by

Table 15.1: Histological dating of subdural hematoma

<i>Time</i>	<i>Histological appearance</i>
With 36 hours	Fibroblasts appear at margin of clot
By 4 days	Neo-membrane adjacent to dura is few cell thick
5-8 days	Membrane well established; fibroblast migrate from it into the clot. After 5 days, hemosiderin laden phagocytes are present which may be stained with Perl's reaction
By 8 days	Membrane 12 to 14 cell thick, visible to the naked eye after few days
By 11 days	Strands of fibroblast subdivide the clot
By 15 days	Membrane also present on the under surface of clot and outer neo-membrane is half to one third the thickness of dura. Inner membrane is still only half thick.
1-3 months	Membrane has lost many fibroblastic nuclei and becomes hyaline
6-12 months	The membrane thick and fibrous resembling the dura

trauma varies greatly according to the nature and extent of injury. When it is secondary to laceration of the brain or extensive cortical contusions then its localization and severity depends upon primary injury.

When it arises from a blunt impact, then position is not a good localizing sign. Blood in the subarachnoid space mixes with CSF, which dilutes it and makes it less ready to clot and allows more mobility. Hemolysis turns CSF into xanthochromic yellow and within weeks, the blood is gone. There may be some residual brown or yellow staining of pia or arachnoid (Figs 15.15 and 15.16).



Figure 15.15: Subarachnoid haemorrhage of brain with petechial haemorrhages



Figure 15.16: Subarachnoid haemorrhage

Traumatic Intracerebral Haemorrhage

Traumatic intracerebral haemorrhage is primarily occurring at the time of impact or soon afterwards; others are secondary and caused by changes in intracranial pressure. In the cerebral hemisphere, deep haemorrhage can be caused by coup or contrecoup mechanisms and may be situated anywhere in the hemisphere. They may rupture into ventricular system or through overlying cortex with resulting intracerebral haemorrhage. Whether a scalp injury (such as fall) was responsible for cerebral haemorrhage or whether sudden stroke caused by a natural cerebral haemorrhage resulted



Figure 15.17: Fourth ventricle bleed in a case of road traffic accident

in fall is to be answered during autopsy examination (Fig. 15.17).

NON-TRAUMATIC INTRACRANIAL HAEMORRHAGE

Spontaneous Subarachnoid Haemorrhage

The hypertensive haemorrhages result from the rupture of microaneurysms called Charcot-Bouchard aneurysms that form at the bifurcations of small intraparenchymal arteries. Bursting destroys the aneurysm responsible for the haemorrhage and the adjacent brain tissue. These aneurysms are seen in increasing number in the arteries of the brain with age and length of history of hypertension. The major sites of hypertensive haemorrhages are the putamen (55%), lobar white matter (15%), thalamus (10%), pons (10%) and cerebellar cortex (10%).

Bleeding into subarachnoid space is usually the result of rupture of an aneurysm or rarely an arteriovenous malformation. Aneurysms can be (i) Developmental (Berry, congenital) (ii) Arteriosclerotic (fusiform) (iii) Inflammatory (mycotic) and (iv) traumatic.

Berry aneurysms are the most common variety accounting for 95% of the aneurysms that rupture.

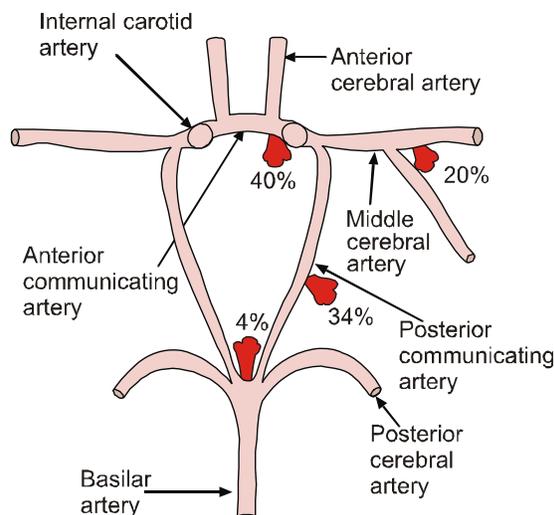


Figure 15.18: Common sites of berry aneurysms in the circle of willis

They occur at the bifurcations of major cerebral arteries. The most common sites that account for about 85% of all ruptured berry aneurysms are (i) the junction of carotid and posterior communicating arteries (ii) the anterior communicating artery (iii) the major bifurcation of the middle cerebral artery in the Sylvian fissure. Remaining of the ruptures are in the posterior circulation. In about 20-30% of cases, there are multiple berry aneurysmal rupture (Fig. 15.18).

Berry aneurysms are also called congenital aneurysms but are not present at birth. They develop because there is a discontinuity in the smooth muscle of the media of the carina of arterial bifurcations. The arterial wall bulges out through the muscular defect to form a thin-walled sacular fundus, composed of only fibrous tissue in which there may be additional local degeneration and calcification. Laminated blood clot and fibrin may be deposited on this attenuated wall. This insubstantial structure is like an inflated balloon, and ruptures in activities causing increase in intravascular pressure such as straining while defecation, lifting heavy weights and sexual

intercourse.

Mixed Subarachnoid and CNS Haemorrhages

These mixed haemorrhages result from (i) arterio-venous malformations (ii) cavernous angiomas (iii) capillary telangiectasis.

Medicolegal Importance of Brain Haemorrhage

Ruptured berry aneurysm and trauma: Several variations of this exist. Most commonly, an assault is rapidly followed by signs of subarachnoid bleeding and subsequently death. When the blow to the head is hard then it is easy to prove that trauma led to the rupture of berry aneurysm, but when the blow is not so heavy then the evidence is not so convincing.

The complicating factor is that most assault occurs in conditions when both the aggressor and victims are physically and emotionally active, so that adrenal response is likely to be present. Muscle tone, heart rate and blood pressure increase and it is likely that raised internal blood pressure in a weak aneurysm is a far more potent reason for rupture than a blow on the head. A second possibility is that already leaking aneurysm may have a rapidly developing neurological even behavioral abnormality that led him to conflict with another person or into dangerous physical position, such as fall or traffic accident. However, at autopsy, it may not be able to distinguish the sequence of events and the aneurysmal rupture may be blamed on the trauma instead of the reverse.

Subarachnoid Haemorrhage and Alcohol: A high blood alcohol is said to facilitate bursting of an aneurysm because it dilates cerebral blood vessels and increases blood flow. The fibrous wall of an aneurysm is incapable of dilating; neither can major basal arteries do so to appreciate degree, as they possess little muscle. However, there is no evidence that alcohol is associated with completely natural subarachnoid haemorrhage

from a ruptured aneurysm, though intense physical activity such as sports or coitus certainly does predispose to rupture. Moreover alcohol increases the chance of fight producing trauma and leading to subarachnoid haemorrhage. Due to unsteady gait, there are always chances of fall.

VERTEBROBASILAR ARTERY INJURY

It has been recognized that blows to the side of the neck can give rise to fatal subarachnoid bleeding because of tearing or dissection of intrathecal course of vertebral artery allowing blood to track along the upper part of the vessel and enter the cranial cavity where the artery penetrates the dural membrane of the foramen magnum. Basilar artery and internal carotid artery can also give rise to the syndrome. The injury is manifested as skin bruising and bleeding into the deep neck muscles.

Mechanism of vertebral artery trauma: The vertebral artery is injured due to (i) overstretching of atlanto-occipital membrane and (ii) impact on muscles overlying the transverse process of upper cervical vertebrae. The damage to the vertebral artery occurs at the following places:

1. In the canal within the first cervical vertebrae.
2. Just below the axis, in the space between transverse process of Axis and Atlas.
3. As it emerges from the exit of the canal in the Atlas.
4. Within the subarachnoid space in the foramen magnum.

The most vulnerable points for trauma are (i) at the penetration of dura (ii) at exit from Atlas and transverse process and (iii) in osseous canal in Atlas.

The demonstration of intracranial bleeding from the vertebrobasilar system is difficult since while removing the skull and brain at autopsy, the damage to vessels occurs causing artefactual bleeding.

Autopsy findings:

1. When an external bruise is seen on the side of the neck in a case of fatal assault such as

- from a fist, shoe or blunt weapon, the possibility of vertebral artery injury should be borne in mind.
- When there are circumstantial evidences suggesting subarachnoid haemorrhage, vertebral artery damage should be suspected.
 - Usually an external injury is absent but on dissection of neck, there is bruising of subcutaneous or deep tissues of neck.
 - Radiographs of the anteroposterior and lateral view of the upper cervical region should be taken. These would reveal fracture of transverse process of the atlas vertebra, if rarely present.
 - Before search for vertebral artery damage is made, brain should be examined for berry aneurysms or other vascular haemorrhages in cases of substantial subarachnoid haemorrhages.
 - Postmortem angiograms can be taken, if facilities exist.
 - When the facilities for radiography and angiogram do not exist, upper cervical region is dissected to search for vertebral artery damage.
 - Upper cervical spine is exposed by a posterior approach by making spine free of muscles. The spine should be then sawn till fourth cervical vertebra. The block of bone is cut out of the floor of the posterior cranial fossa. This block is decalcified by prolonged immersion in 10% formic acid. After a week, lateral parts of transverse process can be shaved. The block is again decalcified for further one week. The artery can now be exposed by shaving transverse processes.

HEAD INJURIES IN BOXERS

Persons who indulge in boxing are at the risk of both acute and chronic damage to their brain. The acute injuries are less common but occur during or soon after fight itself. The most common lesion is a subdural haemorrhage. Extradural bleeding almost never occurs.

Most attention has been paid to the chronic changes in boxer's brain, which is very common and give rise to what is generally known as **punch-**

drunk syndrome. The length of time a boxer is involved in fighting is more important than number of serious traumatic events he has suffered. There are both anatomical and microscopic lesions. Grossly affected brain may show some cortical atrophy and slight hydrocephalus. Throughout the brain substance, there may be scars and patches of gliosis. These persons also suffer from development of an Alzheimers like condition, with neuro-fibrillary tangles throughout cerebral cortex and brainstem though no senile plaques are present.

CEREBRAL INJURIES

The brain may be injured:

- By direct intrusion by foreign object such as a penetrating weapon, either bullet or fragments of skull in a compound fracture.
- By deformation of the brain in closed head injury. Rotatory movement is the main culprit in causing brain damage. No actual blow or fall need be suffered by the head to cause severe and even fatal brain damage. It is the change in velocity, either acceleration or deceleration with a rotational rather than solely axial element that leads to damage. Among the competing theories of brain damage are (i) rotational shear force theory (ii) pressure gradient theory (iii) the vibration theory (iv) transmitted wave force theory (v) brain displacement theory (vi) skull deformation theory (vii) theory of bony irregularities.

Shear strain: Strain produced by applied forces, which causes or tend to cause adjoining parts of body to slide relatively or tend to cause adjoining parts of the body to slide relatively to each other in a direction parallel to their point of contact.

Cerebral Lacerations and Contusions

They are most often found in those areas of the brain where cortex is most likely to be exposed to irregularities in the internal profile of skull. The undersurface of the temporal lobes and the orbital surface of the frontal lobes suffer most often.

Cerebral contusions: They occur where blunt trauma crushes or bruises brain tissue without rupturing the pia. The most common sites of contusions are either (i) directly related to the trauma, in which case they may be coup or contrecoup lesions or (ii) indirectly related, occurring at points where the brain as it moves, can strike the irregularities on the inner surface of the skull such as the lesser wing of the sphenoid, and the orbital ridges that produces contusions at the frontal and temporal poles and on the orbitofrontal gyri. Contusions usually affect only the crowns of the gyri, leaving the depths of the gyri intact, but there is often substantial attenuation of the underlying white matter that extends under adjacent intact cortex. In ischemia, cortical lesion is usually more extensive than the damage to white matter. Histologically, acute contusions show foci of haemorrhagic gray matter that is subsequently removed by macrophages leaving an irregular yellow-brown crater with a floor of reactive glial tissue and often some leptomeningeal fibrosis.

Cerebral lacerations: These are tears in the brain tissue produced by more severe blunt trauma that is often accompanied by other damage such as fractures and local haemorrhage and necrosis. Resolution of lacerations is similar to that of contusions, except that it results in an irregular, yellow-brown gliotic scar that involves not only the cortex and meninges but also deeper structures.

Coup and Contrecoup Damage

Coup (blow impact) means injury that is located beneath the area of impact and results directly from impacting force. **Contrecoup** means that the lesion is present in an area opposite to the side of impact.

The following practical points should be considered:

1. Sometimes only contrecoup lesion is there with no coup damage at all.
2. There need not be any fracture of skull, even in presence of coup and contrecoup lesion.
3. The most common sites for contrecoup injury is in the frontal lobes. It is often at the tips of

frontal poles and may be symmetrical, if a fall on occiput has occurred.

4. In temporal and parietal impacts, the contrecoup lesions are likely to be diametrically opposite on the contralateral surface of the brain.
5. It is virtually unknown for a fall on the frontal region to produce occipital contrecoup.
6. In a temporal impact, the contrecoup damage may not be on contralateral hemisphere but on opposite side of ipsilateral hemisphere from impact against the falx cerebri.
7. The degree of contrecoup damage may be severe, sufficient to cause blood-filled cavitations in the deep cortex and underlying white matter especially in frontal lobes and tips of temporal lobes.
8. With severe frontal contrecoup from a fall on occiput, the transmitted force may be sufficient to fracture the thin bone of the floor of anterior fossa. Such cracks in the roof of orbits may allow meningeal haemorrhage to seep into orbits and appear as **black eyes**.
9. Though contrecoup contusion is classically caused by deceleration of a falling head, it can also occur when a fixed head is struck.
10. Contrecoup lesions occur in the form of cortical contusions or lacerations.

Theories of Contre Coup Lesions

The various theories for such type of injuries are:

1. **Struck Hoop theory:** Due to the elasticity of skull, the flattening of the skull result at the point of impact resulting in compression of skull so that skull assumes an ovoid shape shortly and thus damage is caused to the opposite side of the impact on brain.
2. **Russell's theory:** Sudden displacement of brain towards impact site as the brain reacts as jelly mass and potential space is developed on opposite side, injuring the vessels and resulting in subdural and cortical injuries.
3. **Gaggio's pressure gradient theory:** At the moment of impact, there is positive pressure

on the side of impact and negative pressure on opposite side; this bursts the vessels on the other side.

4. **Holbourn shear strain theory** (*Rotational force theory*): Contrecoup lesions are chiefly due to local distortion of blow that causes shear strains due to pulling apart of constituent particle of brain.
5. When the moving head is suddenly decelerated by hitting a firm surface, contrecoup injury results e.g. striking head on ground directing fall. The sudden arrest of head results in brain, which is still in motion striking the stationary skull.
6. **Rawling's theory of bony irregularities**: Irregular bony prominences particularly orbital and cribriform plate, lesser wings of sphenoid contuse or lacerate base of frontal lobes and tips of temporal lobe or lacerate base of frontal lobes and tips of temporal lobe sometimes with fracture of orbital plate. A blow at the front of head may damage inner and lower parts of back of brain by contact with the edges of tentorium leading to brain stem injury and pontine haemorrhage.
7. Fall on the side of head producing contusion on the opposite side of the brain is due to formation of cavity or vacuum in cranial cavity on the opposite side of impact as brain lags behind the moving skull. The vacuum exerts a suction effect that damages the brain.
9. **Moritz radiating wave theory**: Energy of impact in a hollow organ propagates by radiating waves along the meridional lines that damages as they leave the site of impact and converges as they approach the opposite side.

CONCUSSION

It is a transient paralytic state due to head injury, which is of instantaneous onset, does not show any evidence of structural cerebral injury and is always followed by amnesia from actual moment of accident. There may be evidence of depressed medullary function. Rate of change of velocity of

head is important in producing concussion. It is extremely common and true concussion lasts for seconds or minutes. At autopsy, there is no macroscopic damage; though sometimes there is slight cerebral oedema and scattered nonspecific, petechial haemorrhages. Shear stresses are instrumental in causing neuronal damage. Concussion is followed by 'post concussion' state characterized by headache, unsteadiness and anxiety. Retrograde amnesia is almost inevitably associated with concussion and it may also be transient. Though commonly only of 'minutes' duration, it can extend to several days before head injury. However, there is often a later recovery of much the lost period, the memory of events immediately before the incident rarely return. The most acceptable hypothesis is 'diffuse neuronal injury'. Histologically, extensive diffuse lesions are demonstrated as disruption of axon from shear and tensile strain, leading to bulbous and clubbed appearance.

CEREBRAL OEDEMA

Cerebral oedema leads to death because it compresses vital centers of brain. It may result from a local phenomenon around any laceration, contusion and infarct. Generalized cerebral oedema is common after head injury in children. Oedema is most common cause of raised intracranial pressure seen more often than localized space occupying such as hematoma and tumour. In cerebral oedema the amount of fluid in the brain mainly in the white matter increases and total weight of brain increases by 100 gms .

Autopsy features of cerebral oedema: The weight of the brain is increased to more than 1450 gms. The dura is stretched and tense and the brain is bulging. The gyri are pale and flattened and there is thinning of grey matter. The sulci are filled and as a result, the cerebral surface is smooth. The cut surface is pale. In children, ventricles may be reduced to slits. Severe oedema causes large volumes of cerebral hemispheres to press down upon tentorium that herniates through midbrain opening. In addition, there are findings of uncal herniation.

CHEST INJURIES

The thoracic and abdominal viscera are most vulnerable to the penetrating injuries by the knife to the lower lateral wall of the thorax that may enter the pleural as well as the peritoneal cavity. The stab wounds of the heart may also involve the diaphragmatic and upper abdominal injuries. The stomach may often be penetrated.

Injuries to the Chest Wall

The integrity of the chest wall can be compromised either by severe mechanical failure of the rib cage or by the penetration of the pleural cavities. Rib fractures commonly occur but may embarrass respiration when they are multiple and thus prevent expansion of the thorax; their broken ends penetrate pleura and lungs or when the pleural and muscular pain limits respiratory effort.

Flail Chest

The flail chest occurs when there are multiple bilateral fractures of most of the ribs and also of sternum. The rigidity of rib cage is lost and attempts at expanding the thoracic volume during inspiration are impaired. The loose section is sucked inwards during inspiration, is called paradoxical respiration. In this condition dyspnoea, cyanosis and progressive hypoxia results that is incompatible with life. The flail chest is caused by:

1. Frontal impact in motor vehicular accidents where the victim is thrown against the steering wheel or fascia
2. In stamping accidents where the shod foot is applied violently to the supine body.

In any substantial chest injury, broken rib ends may be displaced inwards, the jagged tips ripping the parietal and visceral pleura. This may cause a pneumothorax or haemothorax, or both with formation of bronchopleural fistula.

Rib Fractures

Rib fractures are most often seen in the anterior or posterior axillary lines caused by falls on to the

sides. The upper ribs are less often fractured, except by direct violence from kicking, heavy punching or traffic accidents. The fracture sites almost always show bleeding beneath the periosteum or the parietal pleura if the fractures occurred during life. Attempts at resuscitation especially external cardiac massage cause extensive rib fractures in about 40% of the cases. In infants, especially victims of child abuse, rib fractures are common. When an infant is squeezed from side to side, as when adult hands are clamped in each axilla or lower on the lateral sides of the chest, the hyperflexion can easily break ribs in their posterior segments usually near their necks. The ribs are levered against the transverse processes of the vertebrae by excessive anterior flexion, which explains the tendency to fracture in paravertebral gutter. Fresh fractures will be obvious both on radiography and autopsy.

The sternum may be fractured by stamping or other frontal impacts, but far more force is necessary than with ribs. If posterior displacement of a fragment occurs, the underlying heart or great vessels may be severely damaged.

Haemorrhage in the Chest

Any injury to the chest wall or lung surface that breaches blood vessels and the pleura can lead to hemothorax. Intercostals and less commonly, mammary arteries can bleed into the pleural cavities but most massive haemorrhage comes from large vessels in the lung or mediastinum. The lung hilum can be torn or penetrated by stab wounds. Several litres of blood may accumulate in the chest, either as liquid or clot, or both (Fig.15.18). Death may occur from loss of circulating blood volume even if there is relatively little external bleeding. Many intrathoracic haemorrhages may be fatal with virtually no external blood loss. A knife that passes obliquely into the chest through intercostals muscles may puncture a great vessel or heart chamber allowing a fatal cardiac tamponade or hemothorax, yet the valve-like overlap of the tissues after withdrawal of the blade may seal up the external wound almost



Figure 15.18: Effusion of blood underneath the rib cage in injuries to chest wall

completely and prevent significant bleeding, especially as the blood inside the chest is not under any appreciable pressure. In a gunshot wound also, if the wound is uppermost after death, the bleeding can be virtually absent.

Infections in the Chest

Infection following a chest wound is uncommon in forensic practice as most deaths occur from haemorrhage within a relatively short time before infection sets in. Cellulitis, pleural inflammation and empyema may supervene due to use of some dirty weapon or clothing or some foreign material being carried in to the wound.

Pneumothorax

Pneumothorax are of three types:

1. **Simple pneumothorax:** Where a leakage through the pleura allows air to enter the pleural cavity but where the communication rapidly closes. The lung partly closes and the air is soon absorbed. If the communication remains open, then a bronchopleural fistula ensues with air in the pleural cavity, but is not under pressure. It will bubble out at autopsy when 'water test' is attempted. Radiology is the best to demonstrate this type of pneumothorax.
2. **Tension pneumothorax:** When the leak in the pleura or rarely the chest wall has valve-like

action, air is sucked into the pleural cavity at each inspiration, but cannot escape on expiration. This pumping action leads to a tension pneumothorax, which causes complete collapse of lung onto its hilum and a mediastinal shift to opposite side. This type can be demonstrated at autopsy by penetrating an intercostals space under water and also by radiological examination.

3. When an injury of the chest wall communicates with the pleural cavity, a sucking wound may form with direct passage of air from the exterior. This type is seen in military surgery and is complicated by haemorrhage and infection.

A common traumatic cause of pneumothorax is a stab wound of the chest that allows direct communication with the exterior, though usually the layered skin and intercostals muscles closes the track when the weapon is withdrawn. Natural diseases such a ruptured emphysematous bulla, a tuberculous lesion at lung periphery or a tear at the site of fibrous pleural adhesion can also cause pneumothorax.

Injuries to the Lungs

The lung injuries can be of following types:

Bruising of the Lungs: Bruising of the lungs can occur in open as well as closed injuries to the chest. The lungs can be contused at the site or opposite the site in any substantial impact to the chest. Deceleration injuries are seen in falls and traffic accidents commonly along the postero-lateral surfaces where a vertical line of subpleural bruising occurs. The outline of the ribs may be imprinted in lines of contusion on the pleural surface of lungs. Severe bruising may cause subpleural blood blisters. In all severe chest injuries the central parts of the lungs may show bleeding with actual hematoma formation.

Laceration of the lungs: Lacerations of the lungs can result in blunt injuries with detachment of lobes or parts of a lobe. The hilum may tear and the pulmonary ligament below the hilum is a frequent site of haemorrhage. Vessels in the hilum espe-

cially pulmonary veins or those more peripherally, may be ripped, causing severe intrapleural or mediastinal haemorrhages.

Penetrating Injuries of the Lungs: Penetrating injuries of the lungs are commonly produced by the knives. The wounds may penetrate lung parenchyma, large vessels or through and through causing severe damage to heart and great vessels.

Blast Injuries to Lungs: Lungs are the most vulnerable organ to blast injuries.

Injuries to the Heart

The heart is vulnerable to both blunt and penetrating injuries.

Penetrating Injuries to the Heart: A stab wound of chest that penetrates the heart is a common homicidal wound. Sometimes the sternum is penetrated by a forceful blow that reaches the underlying heart but most stab wounds enter via the intercostals spaces, or through a rib or costal cartilage. Rarely, an upward stab from the abdomen reaches under the costal margin to penetrate the diaphragm. The right ventricle is often injured by a stab wound as it presents the largest frontal area, but the anterior interventricular septum and the left ventricle are also vulnerable. A shallow stab wound may enter the myocardium and not reach the lumen of the ventricle. In such a case there may be little disability unless a coronary vessel is severed, which may either cause death from myocardial insufficiency, if a major artery is transected or cardiac tamponade.

In the thin right ventricle the knife passes more often leading to copious bleeding in the pericardial sac even though the intraventricular pressure is relatively low because of the thin wall is unable to close the defect by muscle overlap and contraction. Whereas in the left ventricle, the contraction of the layered thick wall may partly or wholly seal the wound and there is slight bleeding. Cardiac tamponade can result if the drainage from the pericardial wound is less than the leakage from the ventricle. The wounds in the right ventricle are more dangerous than that in the left ventricle due

to absence of muscular self-sealing effect. The stab wounds of the heart are mainly through and through with knife entering one wall and emerging from the other.

Blunt Injuries to the heart: The blunt injuries to the heart result in road traffic accidents fall from height and in stamping assaults. Multiple rib and sternal fractures result with or without flail chest. In a child due to pliable thorax, heart can be damaged in an intact rib cage. Fatal blunt damage to the heart may occur, without a mark on the skin or thorax or damage to rib cage. The cardiac injuries are usually seen on the front of the organ' especially right ventricle. Rarely posterior bruising and laceration can occur if the heart is compressed against the thoracic spine as in stamping assaults and steering wheel impacts. In gross injuries such as aircraft crashes the whole heart may be avulsed from its root.

Haemopericardium and Cardiac Tamponade

Haemopericardium

Haemopericardium or bleeding in to the pericardial sac may occur from the surface or the cavities of the heart, or from the intrapericardial segments of the roots of the great vessels' particularly the aorta and the pulmonary artery. Haemopericardium may result from (i) ruptured myocardial infarct (ii) ruptured dissecting aneurysm of the aorta (iii) chest injury such as stab injury

When the damage has been caused by the stab injury that has perforated the pericardial sac, bleeding can escape into the pleural cavities, mediastinum or even the abdomen when diaphragm has been perforated. Death usually occurs from haemorrhage.

Cardiac Tamponade

Cardiac tamponade is the condition when blood accumulates in the pericardial sac faster than it can escape, either because the bleeding rate exceeds the drainage or because the exit hole in the pericardium becomes blocked by blood clot. In cases where the bleeding is from a contusion

or laceration of heart, there is no escape route from the sac. When sufficient blood accumulates, the pressure in the pericardial sac increases and begins to prevent the passive filling of the atria during the diastole. The cardiac output falls, as does the systemic blood pressure and the venous pressure rises. If unrelieved the death will occur with collection of about 400-500 mL of blood

Injuries to Great Vessels

Injuries to Aorta

Aorta is the most vulnerable vessel that suffers in injury in deceleration trauma from both road and air accidents and falls from height. When the thorax is suddenly decelerated, the heart being relatively mobile in the chest, attempts to continue in the origin direction. This causes severe traction on the root of the heart with complete or partial rupture of the aorta in the descending part of its arch. In falls from height, the lesion is the result of abdominal and thoracic viscera being forced caudally by the abrupt deceleration while landing on the feet or buttocks. Rupture commonly occurs almost constantly at a point 1.5 cm distal to the attachment of the ligamentum arteriosum. The lower thoracic aorta is closely bound to the anterior longitudinal ligament on the front of the dorsal spine, until it reaches the termination of the arch, where it curves forwards. This appears to be the weak point and transaction occurs at this level, sometimes so cleanly that it looks like a surgical incision. The tear is annular and at right angles to the axis of the aorta. Sometimes, there may be multiple parallel intimal tears near the main transaction, called the 'ladder-rung tears'. In deceleration trauma, these incomplete tears, that affect the intima and media only, may be found without major transactions.

Injuries to Pulmonary Artery

The pulmonary artery may be damaged by depressed rib cage and sternal fractures in stamping assaults and steering wheel impact in vehicular accidents. The pulmonary artery and vein branches may also be damaged in the root of the

lung, where hilar tears are not uncommon. The great vessels are often involved in penetrating injuries, notably stab wounds. Stab of the upper part of the chest may pass directly into the arch of the aorta, especially on the right side of the sternum. Stabs that are too high or are directed too laterally puncture the chambers of the heart may penetrate the ascending aorta or pulmonary artery. If the wound is below the reflection of the pericardium, a haemopericardium and cardiac tamponade may result.

ABDOMINAL INJURIES

Abdominal injuries can be of two types:

1. Open or penetrating injuries
2. Closed, non penetrating or blunt injuries.

Closed/blunt injuries: These types of injuries usually result from increased abdominal pressure and cause dysfunction of chest and abdominal organs. They can occur in:

- Road traffic vehicular accidents due to the impact of steering wheels was quite common when the use of safety belts or air bags was not popular or due to a carriage wheel passing over the abdomen.
- Crush accidents that result from crushing between the two vehicles, crushing between a vehicle and a wall, railway accidents and industrial accidents.
- Blow by blunt weapons or by kicking, stamping, heavy punching occurs in homicides, assaults and child abuse. The liver, spleen, intestine and mesentery are most vulnerable internal organs to be affected.
- Fall from heights.

Open/penetrating injuries: They may result from cutting or stabbing weapons, firearms, horns, and claws of animals or fall over sharp projecting object. These are dangerous injuries and death may occur without any injury to abdominal viscera sometimes. Sharp pointed weapon injuries to peritoneum, intestines or soft viscera may penetrate with slight force. The injuries may be fabricated for charge of assault such as superficial incised wounds. Homicidal injuries are the most common whereas suicidal stab wounds are

rare, may result from the **Japanese Hara-kiri method** when the victim falls forwards on ceremonial sword and tries to disembowel himself. Intestines and mesentery are the major targets and are often involved in multiple wounds. Liver and spleen bleed extensively with resulting haemoperitoneum. Stomach is more commonly involved in chest stabs passing downwards through the diaphragm.

Features of Abdominal Injuries

1. **Bruising of abdominal wall:** Bruising in muscle tissues without any external violence can occur in tetanus and strychnine poisoning whereas discrete bruising on abdominal skin may result from kicks that produces scuffed bruised abrasion if tangential unless protected by clothing and in cases of child abuse where fingertip/knuckle bruises from holding, forcibly gripping or lifting the child, on the side of the abdomen or along the maxillary lines of chest. Usually abdominal wall escapes gross injury by transmitting force of violence to more resistant organs inside the abdominal cavity, which gets injured due to protective clothing and owing to the large surface area of abdomen. Laceration of abdominal wall with protrusion of coils of intestine can result in run over accidents under the wheels. Blow on upper/central (epigastria region) of abdomen has an inhibitory action on the heart through reflex action on solar plexus. Bleeding may be subcutaneous from initial area of impact to more diffuse involving large area of abdomen. Injury to epigastric artery causes severe haemorrhage. Extensive bleeding into peritoneal cavity may result from rupture of the solid viscera or from bleeding from the mesenteric vessels.
2. **Bruising or rupture of stomach:** The stomach is less vulnerable than the intestines and are lacerated by heavy blows in upper abdomen when full of fluid or food or the penetrating injuries.
3. **Intestines and mesentery:** External bruising of the gut and vascular mesentery being crushed against the prominent lumbar vertebra

in the midline. Mesenteric lacerations occur in traffic accidents and assault. Bruising or tearing of central part of mesentery occurs while compression against the lumbar spine occurring towards the intestinal margins. Duodenum or jejunum is more vulnerable to transaction compressed against the spine especially in children from heavy blows to central or upper abdomen cutting through the third part of duodenum. Colon is rarely injured. The injuries to intestine and mesentery cause intractable bleeding.

4. **Injuries of Liver:** The rupture of liver results from the penetrating as well as non-penetrating injuries. The non-penetrating type of injuries may occur in abdominal trauma, fall from height, crush from wagon, and road traffic accidents from the impact of driver on rim/center of steering wheel or by unrestrained passenger being thrown against the face. The pedestrian can receive such injuries as primary impact from the vehicle or secondary damage by being thrown to the ground, external cardiac massage, sudden contraction of abdominal muscles such as during epileptic fits, lacerations by the fractured ends of ribs may perforate the diaphragm. In infants abdominal injuries may be found in child abuse syndrome and during breech delivery

Penetrating injuries are usually stab injuries of the abdominal walls over the front, back or sides of lower part of chest. The liver is frequently injured due to its large size and weight, friable consistency and fixed and exposed position. The liver may be injured by forcible impact over the lower parts of right side of the chest, upper parts of right side of abdomen from the front, back and the sides. The right lobe of liver is injured five times more frequently than the left due to its size, and position. The convex surface and anterior margin gets involved. Anterior surface and inferior border is more commonly involved. Ruptures can vary from superficial linear tears on convex upper surface to oblique to anteroposterior and rarely transverse. The

tears are 1-2 inches deep and may pass through the entire substance of organs. There is complete transection of organs when greater force is applied. There may be sub capsular tears or sub capsular dermatome. Liver is involved in 40% of multiple injuries. Lacerations may range from shallow splits confined to capsule to deep laceration separating right and left lobe of liver. In children, one lobe of liver may lie in peritoneal cavity, as it is more prone to being run over in road traffic accidents. According to *Kindling et al*, the lacerations along the line of falciform ligament occurs in drivers and passengers of automobiles due to severe compression causing bursting of organs; due to upward or downward displacement of liver. The satellite lacerations or fractures occur in pedestrians. The bruising of liver is usually localized to sub capsular dermatome and is common in newborn due to complications of delivery. Penetrating injuries are caused with the help of knives by multiple thrusts; limited bleeding occurs and complete transpiration of organ may occur.

The puncture of liver by needles while taking liver biopsy causes haemorrhages in diseased liver. The liver is more easily lacerated if enlarged and fatty as in alcoholics. The rupture of the liver cause haemorrhage and shock and immediate death occurs especially if portal vein or vena cava is injured. The liver abscess may rupture causing septic infection and if patient survives for long time, infarction results. The lacerations of liver can be of the following types:

- Transcapsular over convex surface under the site of impact, the portal vein or inferior vena cava may be involved and profuse haemorrhage results.
- Subcapsular over the convex surface under the site of impact.
- Distortion can cause laceration over the superior or inferior surface.
- Contracoup involving the posterior surface
- Whole organ ruptures in vehicular accidents and gunshot injuries.

- Large subcapsular dermatome rupture after several days following muscular exertion or violence.
- Fatal pulmonary embolus may result from liver tissue emboli.

5. **Rupture of Spleen:** Spleen is most vulnerable to injuries by blunt force and considerable crushing and grinding force is required like passing of carriage or motor car over the body; railway accident crushing, fall from height, foreign body piercing accidentally the pulp and by stabbing or cutting. Normal spleen may rupture by the broken ends of fractured ribs, severe kicks or blows by blunt weapon over lower part of right side of chest and in mechanical or industrial accidents. Enlarged spleen may weigh up to 2kgs, is 32.5 × 17.5 × 6.0cm, and is liable to rupture from fall or slight violence. Spontaneous rupture of spleen can occur during bouts of coughing, sneezing, vomiting or straining particularly if the spleen is abnormally mobile. When the spleen is diseased from malaria, infectious mononucleosis, kala azar and glandular fever it enlarges and is more friable and the incidence of rupture increases. It may also rupture spontaneously in pregnancy. The spleen can rupture due to compressive force resulting from laceration of the surface or whole of it and traction force that tearing spleen from pedicle and penetrating injuries. Concave or inner surface is the commonest site of rupture and death occurs from haemorrhage. The injuries may vary from the large lacerations of capsule most often on lateral surface running up to the hilum to multiple lacerations traversing the surface almost bisecting the organ; complete detachment from the hilum is uncommon. Subcapsular hematoma if capsule is intact and substance ruptured causes delayed death as capsule prevents excessive bleeding and is disrupted with sudden muscular exertion.

PELVIC INJURIES

In severe trauma, the pelvis undergoes various

fractures as well as dislocations such as:

1. When there is application of great pressure to the front of abdomen or pubic area such as in run over by the wheel, the pelvis is splayed open, symphysis pubis separates and one or both sacroiliac joints also dislocate.
2. When an impact occurs from the side, superior and inferior pubic ramus are fractured with dislocation of sacroiliac joint on the side of impact.
3. In circumstances of fall from height on to the feet, due to transmission of force up the legs, both the sacroiliac joints may dislocate and even one or both femoral head also may be driven in to acetabulum. When the hip joints remain intact, the pelvic girdle may fracture and sacroiliac joints may dislocate.
4. Due to a kick or heavy fall on to the base of spine, fracture of sacrum or coccyx may result.
5. Empty bladder is rarely injured in trauma but a full bladder gets injured from blows, kicks and other blunt trauma. Other pelvic organs are quite protected from blunt injuries.
6. Male urethra may be injured as a result of direct trauma such as falling astride a solid object like a gate or being kicked in the crutch, due to being compressed against the undersurface of

the pubis.

7. External genitalia may suffer injuries especially scrotum is quite vulnerable to severe bruising resulting from kicks. Scrotum and vulva may suffer injuries from falling astride on objects and in vehicular accidents.

Long Question

1. Discuss injuries caused by a blunt weapon impact to the head. Enumerate the differences between the various types of injuries and the age of the injuries in intracranial haemorrhages.

Short Questions

1. Extra dural haemorrhage.
2. Sub dural haemorrhage.
3. Sub arachnoid haemorrhage.
4. Theories of head injury.
5. Contre coup injury.
6. Signature fracture.
7. Lucid interval.
8. Flail chest.
9. Injuries to spleen.

MULTIPLE CHOICE QUESTIONS

1. Pott's fracture are seen in:

- A. Children
- B. Adolescents
- C. Young man
- D. Elderly

2. Contrecoup injury is seen in:

- A. Heart
- B. Lung
- C. Brain
- D. Kidney

Explosion Injuries

Explosions are uncommon and few doctors have much experience of them. Usually explosions occurs during work time but few notable explosions has occurred in the past affecting the civilians. For example in the Hulton disasters in 1911, 343 people died. Another disasters occurred in Taxas City during 1947 in which a ship loaded with ammonium nitrate fertilizers exploded at the dock filling 561 people and injuring 3000 people. But things have changed a lot now a days as terrorist activity in many parts of the world particularly the Ireland has added a new dimensions to the civilians explosions like that of bomb, latter bomb and car bombs. They have become a daily potential hazards in many countries.

During explosion, explosive material is converted in to large gas volume and there is release of tremendous amount of energy. Pressures of 1000 tons/inch² (150,000 atm) and the temperature of 3,000°C can be generated in an explosion.

Effects of Explosion on the Individual

1. Those very near the seat of explosion are blown to pieces and scattered by force of explosion gases.
2. In those who are near enough for the skin to be in contact with explosion flame, flame burns are produced.
3. At greater distances, exposure to momentary heat radiation produces flash burns.
4. Persons can be injured by:
 - Shock wave that is the pressure wave spreading concentrically from the blast center.

- Air blast is the shock wave produced by the explosion in the air.
 - Post-blast wave also causes the damage.
 - Underwater blast is the pressure change occurring from explosion in water.
 - Solid blast: In underwater blast solid material like ship deck is forced against the body.
5. Person can be struck by the flying missiles propelled by the explosion.
 6. The debris of building demolished by explosion can cause injuries.
 7. The poisoning can occur by the fumes of the explosive material.

Distruptive Affects of Blasts

The persons in the immediate vicinity of the blasts are laterally blown into pieces. The small parts of the body may be scattered over a wide area. If someone is carrying the bomb or in cases of land mine explosions the victims is blown up. The body parts of the individuals who died as a results explosion should be collected by the trained persons otherwise mistakes are liable to be committed. The internal organs are less frequently found at the scene of explosion. In cases of small explosions, the limited body parts are blown up and in all cases the mangled remains are heavily soiled.

When there is a blast in the civilian, the number of victims should be counted. Within 200 yards the areas must be searched for the parts that are usually scattered due to explosions. The parts thus recovered must be worst in water and carefully examined. The non human tissue must be identified and discarded, if it is not possible to

identify them tissue by naked eye examination then precipitation test can be done.

The segregated parts will help to establish the number of victims. Pieces of scalp, skin, jaw, joints and hands etc. are put in separate heap. Thus skin must be kept according to the colour, likewise the hair to be segregated according to the colour length and texture. The joints to be marked whether they belong to left or the right side of the body. The sex can be determined from the genitalia, hair, and breast tissue. At times the help of the microscopic study will be required. From the above data the minimum number of the body will be counted but there is always is chance for more number of bodies.

The identification of the bodies can be done by visual inspection after washing the parts recovered. The evidence of the lay people must be expected with caution because of the deformity of the skull or facial injuries may be a misleading factor. The clothing are to be identified by the relatives. The most important thing is taking the finger prints and matching with the records available specially in cases of the terrorist and criminals. In other cases the fingerprints will be matched with some article of use at home. The dentism and the denture found at the seen may also be recovered and matching to be done. Other points like the tattoo marks, scars and old fractures are also helpful to identify the victims.

Effects of Flame and Radiant Heat

The temperature rises to 3000°C and momentary flame contact causes burns (Figs 16.1 to 16.3). Radiated heat burns people outside this range. The burning effect of radiated heat depends on the total thermal energy received and the rate at which the energy is supplied. The rate of heat transfer depends on the duration of explosion. Thermal explosion received increases directly with the weight of the explosive and inversely with the square of distance from it. The flash burns produced effect an area perhaps the face or one side of limb and spares the area that are protected by the clothing and solid objects. Areas are dry



Figure 16.1: Burns over the back in a case of bomb explosion in a cinema hall



Figure 16.2: Burns over the back in a case of bomb explosion in a cinema hall



Figure 16.3: Burns over the sides in a case of bomb explosion in a cinema hall

and reddish brown or parchment like. The individual has to be with in feet of an ordinary bomb to sustain heat burns.

Effects of Air Blast

Shock wave spreads concentrically from the blast center in a round wave of very high pressure, the velocity of this shock wave soon decreases to about the speed of sound in air (1120 fps). It is many times faster near the explosion and as the distance from the explosion is increased the pressure component lasts longer. Blast wave can knock a person down, move objects and cause demolition of buildings. On the heels of the blast wave there are winds that can displace objects at considerable distances. Post blast wave can knock buildings down. Ground shock is a complex train of shock waves that is similar to earthquake.

Biophysics of Air Blast Injury: Shock wave strikes the body surface and the part of it is deflected, a part reflected and the rest of it is absorbed. The shock wave passes through the homogenous organs like heart and thigh and solid organs but does not damage them. The organs that contain air are very vulnerable e.g. in its passage through inhomogeneous lungs, there is considerable damping of wave achieved by the movements of lung tissue. Acceleration of parts of different density and compression and expansion of alveoli as pressure wave passes through them are thought to be among the causes of lung damage. Also the shredding and bursting effect that occurs when a pressure wave breaks out of liquid medium of lung tissue in to gaseous medium of the alveolus. Structural damage due to shock wave is confined to air/gas containing organs such as ears, lungs and segments of large bowel. Effects of air blast diminishes rapidly as the distance from the explosion increases.

Tissue Damage Due to Air Blast

The blast wave being a sound wave damages the ear and the damage depends on: (i) The intensity of the blast (ii) Its proximity to the source (iii) Orientation of axis of external auditory canal to

wave front (iv) Duration of exposure (v) Awareness of explosion (vi) Protection by any object.

Effects of Air Blast on Ear

1. Hyperemia of intact tympanic membrane.
2. Punctate bruising of intact tympanic membrane.
3. Rupture of tympanic membrane at an overpressure of 5 psi, results in bleeding into middle ear.
4. Damage to the cochlea that is more frequent than tympanic membrane rupture.
5. Sudden severe deafness with period of recovery during which acuity for all frequencies is depressed.
6. Slight haemorrhage into scala tympani near round window.
7. Haemorrhage into basal coil of cochlea and in fundus of internal meatus.
8. Prominent symptoms are tinnitus, sensorineural and conductive deafness.

Effects of Air Blast on Lung

Lung lesions of the air blast are due to direct impact on the body wall of pressure component of shock wave. The severity of lung lesions vary from the size of explosive charge and becoming greater nearer the victim is to the explosion. The alveolar haemorrhages are due to tearing of alveolar septa and they arise in each lung immediately after explosion, if patient survives for few hours.

Autopsy examination reveals Sub pleural haemorrhagic patches of purple red colour and of varying size. These haemorrhages are scattered at random, cross the lung surface in line of ribs and are grouped near anterior and costophrenic margins. Lung sections reveal discrete haemorrhages that are more central than peripheral and the rest of the lung tissue is patchily oedematous. On microscopic examination, alveoli contain haemorrhages and oedema fluid, the alveolar septa are not discernible and there is emphysematous dilatation of alveolar ducts. The haemorrhages are seen in the bronchioles and interstitial tissue. The pink staining fluid and red cells may be found

between epithelium and lamina propria is infiltrated by neutrophils. Direct blows by bomb fragments debris on chest bruise the lungs.

Effects of Air Blast on Gastrointestinal Tract

The injuries to GIT are inconstant. The haemorrhages beneath the peritoneum of gut, mesentery and omentum vary in size. They are usually seen as small punctate spots, over extensive areas of intestine. They extend circumstantially as annular bands occurring mainly over the caecum, colon, small intestine and stomach. The stomach and small intestine are less frequently affected. There is no correlation between the abdominal haemorrhages and peak pressure or impulse of blast wave.

Effects on Other Organs

Other organs such as liver, kidney and spleen are uncommonly injured showing haemorrhages or rarely a laceration. No specific changes are seen in the brain. **Zuckerman** described haemorrhages around nerve roots of thoracic spine and posterior column of spinal cord and oedema around central canal.

Clinical Effects of Air Blast

The person receiving the blast is shocked, is pale, cyanotic and restless. The blood pressure falls, pulse is rapid and thready, there is chest pain, widespread crepts and haemoptysis.

Blast (Shell) shock: The blast shock is psychogenic war neurosis and the symptoms of anxiety are produced. There is confusion, muscular and mental weakness, stammering and jerking, hysterical blindness, numbness and paralysis.

Causes of Death

1. *Suffocation:* Death from suffocation occurs from extensive pulmonary haemorrhages in an hour of explosion and there is copious bloody froth in air passages.
2. *Systemic air embolism* is the result of air gaining the pulmonary veins after blast damage

to the lungs. In animals, air has been found in lethal quantities in coronary arteries, left chamber of heart and basal arteries of brain. Air embolism might account for those deaths that occur soon after explosion and no obvious pathologic changes can account for death.

3. Blast shock.

Immersion Blast

Immersion blast refers to blast effects of an explosion in water experienced in wartime by those who abandon the sinking ship. Injury can be expected within 150 feet of a 300 lb charge of TNT exploding at a depth of 50 ft. The physical changes are similar to those accompanying an explosion in air. Explosion converts the charge in to gas of a very high temperature and pressure, which expands pushing the surrounding water radically. Pressure of underwater explosion might be 1000 psi. The pressure waves pass through the solid tissues unimpeded. Injuries are found predominantly in GIT and less constantly in the lungs. Most of the lung injury is due to pressure transmitted from abdomen through the diaphragm. There is pain abdomen, abdominal rigidity and tenderness, occasional testicular pain, nausea, vomiting with blood and bloody diarrhoea. No external injury marks are seen over the trunk.

Autopsy Findings

1. *Haemorrhage:* There is haemorrhage beneath the peritoneum, mesentery and intestinal mucosa. The submucosal haemorrhages account for blood found in the lumen of the gut, haematemesis and malena. The haemorrhages are multiple and in majority are up to 3 cm in diameter and the intestinal walls are oedematous.
2. *Perforations:* The perforations are found in the small and large intestine are circular, half to one inch in diameter with prolapsed mucous membrane. They can occur anywhere but the anterior mesenteric border of ileum and outer side of caecum are the sites of predilection. The gas filled bowel segments are vulnerable to pressure of more than 250 psi.

Solid Blast

Solid blast refers to a wave of energy that spreads through a rigid structure when an explosion is detonated near it and people in contact with structure can be injured. Steel construction of tanks and warships conducts shock wave well and causes solid blast injury. An explosion beneath tank/ship causes initial movement of small displacement by very swift acceleration. Person standing in deck of ship could blow, bones can break and skin and subcutaneous tissue may burst causing compound fracture. The solid blast injuries are predominantly skeletal with fracture of calcaneum, lower end of tibia and fibula. The situation depends on subject's position; those standing receive injuries to lower limbs and those seated in chair suffer from fracture of vertebral column. The skin of ankles and feet are bruised and lacerations of ankle and weight bearing surface of heel occurs. The knee joint may be dislocated and rarely fractured. The fractures of femur and pelvis occur rarely. Fracture of spine is more common and 9th thoracic or 4th lumbar vertebra are involved.

Flying Missiles

Explosions injure and kill by means of solid material, which is propelled in all the directions. The material such as the fragmented containers of explosive, metal case of shell, hand grenade that is meant to expel missiles, missile casing designed to fragment into lethal shrapnel. In a nail bomb, large nails are bound round the stick. Other materials propelled by explosion are remains of explosive material and debris of surroundings namely earth, brick, plaster, wood and dust. Larger fragment penetrate deeply. The fragments accelerate more slowly than associated gases, lag behind blast in early stages and tend to be more damaging at a distance with kinetic energy of $\frac{1}{2}mv^2$.

Injuries Resulting from Blast

Common basic injury due to fragments of explosion is a triad of small lesions; bruises, abrasions and punctate lacerations intimately mixed on skin giving it a purplish colour (Figs 16.4 to 16.7).



Figure 16.4: Triad of injuries in a case of AC blast of a compressor of AC



Figure 16.5: Blast injury over the neck due to blast of a compressor of AC



Figure 16.6: Blast injury over the neck and chest due to blast of a compressor of AC



Figure 16.7: A triad of laceration, contusion and abrasion in a case of bomb blast

Abrasions and bruises are small 1-10 mm in diameter and punctate lacerations are up to 3 cm in diameter surrounded by an irregular zone of bruising and abrasion. Large lacerations due to larger fragments can perforate neck damaging carotid arteries or airways. Perforation of chest and abdomen results in internal organ injury. The dust propelled from the explosion is large in amount and when driven in to skin produces uniform tattooing and skin darkening. Fragments of minute size can penetrate soft tissues of the body quite deeply. The distribution and pattern of bruises, abrasion and punctate lacerations helps to determine relationship of victim to explosion.

The severity, distribution and pattern of injuries can be compared. Those carrying a bomb have their arms, and midriff blown; the one carrying the

bomb in his lap or car receive injuries on the pelvis. The explosion at ground level produces more of leg injuries whereas explosion at the side have more of side injuries than other types of explosion. The fragments and debris of explosion may cause an eye injury.

Injuries from Falling Masonry

The demolished building by an explosion may lead to injuries caused by collapsing masonry. The body and clothes are soiled by dust from brick, cement or plaster. The cause of death in these cases is traumatic asphyxia and the findings are of purple discolouration of upper parts of body, patchily haemorrhages of skin and conjunctiva, oedema of conjunctiva and congestive haemorrhages of nose and ear.

Poisoning by Fumes

Toxic fumes are harmful in an enclosed space and mining accidents greater damp.

Long Question

1. Describe the postmortem findings in cases of blast injuries.

Short Questions

1. Blast lung.
2. Effects of air blast on ear.
3. Immersion blast.

MULTIPLE CHOICE QUESTIONS

1. Which of the following is most likely to be affected by shock waves in cases of explosion injuries?
 - A. Liver
 - B. Lungs
 - C. Heart
 - D. Kidneys
2. Triad of injuries are characteristic of:
 - A. Vehicle accident
 - B. Burns
 - C. Explosions injuries
 - D. Air craft accidents

Fall from Height

Fall is dropping down from a height of relatively high position by the force of gravity. International classification of diseases (ICD 9) gives a range of heights like slipping or stumbling through to the falls from the cliff. During fall the potential energy due to height is converted to kinetic energy under the influence of gravity. At impact some of the energy is imparted to the body resulting in injuries. Falls may occur at the ground level or from some height.

FALLS AT GROUND LEVEL

Causes of Fall

1. Mechanical: Due to slipping on polished surface or rugs or due to some faulty footwear.
2. Cardiovascular accidents.
3. Postural hypotension.
4. Intake of alcohol or hypotensive drugs.

Injuries Resulting from Fall on Ground Level

1. Fractures: Femur (In old people, commonly), Cole's fracture, Pot's fracture, humeral fracture, pelvic fractures.
2. Internal organ injuries retroperitoneal haemorrhage resulting from pelvic fracture, rupture of liver and spleen, bruising of intestines.
3. Injury to head leading to subdural haemorrhage. Speculation arises as to whether femoral fracture has antedated the fall as this fracture can occur spontaneously due to bone rarefaction; poor blood supply.

FALL FROM HEIGHT

The fall from height may vary from high rising building to ladder, chairs, tables or staircases. The falls are common in the middle aged from the windows or accidentally in children or suicidal in adults.

Epidemiology of the Fall

Age and sex distribution: Falls are more common in extremes of ages. The range extends from children falling from the cots through to elderly slipping on ice or at home. The falls in middle age persons are usually due to accidents related to work or suicidal. Females are more vulnerable to suicidal falls whereas males are more prone to falls in their late adulthood.

Geographic distribution: Places near high rise buildings and high bridges are the preferred sites for suicidal attempts. For these sites considerable distances may be traveled to their chosen sites.

Coexisting psychiatric conditions: Suicidal jumping makes a considerable proportion of those who die from fall in whom psychiatric conditions are common. More than 50% people have some preexisting psychiatric illness or heavy drug over dosage. In the elderly, coexisting medical problems also contribute to increased incidence of fall.

Velocity of Fall

For a fall on to the earth from a height, the acceleration is due to gravity 'g' and velocity 'v' after a fall from height 'h' is given $V = \frac{1}{2}gh$

It is true for a fall in vacuum but in actual position, air drag, clothing, body surface area, human body flexibility and non-homogenous mass distribution come in to play. The error may vary from 5-7% for jumps up to height of 23 meters.

Factors influencing the pattern of injury

1. *Height:* A direct relationship exists between height fallen and survival. It is also correlated with the extent of injury as measured by Injury severity score. The height of fall of about 11 meters, results in pelvic damage and when the height is more than 15 meters, multiple rib fractures result. When a person falls or jumps from height, trajectory is downwards and outwards; the distance the body strikes the ground is variable.
2. *Orientation:* Orientation of body at point of impact is important in determining injury pattern and mortality. In majority of falls, vertical landing with feet first and the next common were with head first. In all falls, lower limbs were almost exclusively injured. In side impacts and in feet first impact, mainly upper limbs were injured due to secondary impact.
3. *Surface impact:* The surface on to which a body falls determines the pattern of deceleration and energy exchange. On a relatively yielding surface, such as a safety net, the energy is given up slowly, but on a relatively unyielding surface such as concrete, time of deceleration is shorter and hence the forces on the body are much great.
4. *Deceleration:* Quicker the body brought to rest, greater will be forces acting on it. It depends upon the weight of the body, surface of impact and energy dissipation i.e. bouncing back. Duration of deceleration is extremely short, in milliseconds. A fall onto concrete results in a very rapid onset of deceleration or a high jolt factor. At impact body comes to a halt, in doing this, it must give up the kinetic energy. The magnitude of kinetic energy and the way in which it is dissipated determines the changes

to body tissue. If certain tolerance limits are exceeded, injury results. The body turns and twists in an unpredictable manner during the fall.

A person falling 15 meters, attains a speed of 17m/s. When falling from high building, the displaced air tends to act as the cushion, which drives the body from the wall. A simple fall can result in a body impact some distance from the foot of the building, not an evidence of push or deliberate jump.

Injury Pattern from Fall

The injuries due to fall from height can be direct or indirect.

Direct Injuries

These are immediately anatomically related to the point of impact. As a result of the injury resulting from direct impact due to impact on head, head is suddenly decelerated; brain tends to continue falling because of its momentum. Brain is suddenly pressured against the skull at the site of impact; a corresponding negative pressure is present on the opposite surface of brain. In more severe falls, the head is accelerated prior to impact and deformation of skull. It results in massive fracture often with laceration of scalp with extrusion of brain and vault and base fracture. Other injuries on various parts of head can be:

1. *The top of the head:* Injuries are severe and the character and extent will depend upon the surface or object against which impact takes place and they depend on the distance fallen. Other injuries such as fracture of spine, limbs or chest will usually be present. The point of impact against a hard flat surface e.g. concrete will be indicated by a bruised laceration overlying fissure fractures of skull following direction of lines of force. Impact against blunt protruding object such as sharp corner of furniture will produce a localized depressed fracture of skull with comminuted or radiating fissure.

2. *The side, back and front of head:* These may occur from simple falls from erect position, the part of the head striking the ground depending upon the direction of fall. The usual injury is bruise situated just above and behind either ear with or without abrasion or laceration of scalp.

On a flat surface, fissure fracture occurs parallel with the line of impact and usually run through the middle fossa in to pituitary fossa, sometimes to opposite side, anterior and posterior fossa may also be involved.

Ring fracture of base of skull separates rim of foramen magnum from the rest of the base. It occurs in fall from height landing on feet or buttocks thus driving vertebral column in to skull. The leg bones (tibia) are sometimes driven through the side of the feet. The major blood vessels near their junction with the heart are severed. The inner lining of carotid arteries shows small horizontal tears. Heart may be driven through the pericardial sac and the diaphragm, may be burst and expose ventricle or may be torn from its pedicle.

3. *Fall on the back of head:* The inertia of brain produces a positive acceleration pressure over the fronto-orbital region and negative acceleration pressure over the occipital and cerebellar regions of brain. This pressure lasts until the deformation of skull at the site of impact reaches its maximum. Since with the onset of this deformation the brain becomes displaced towards opposite side, two positive forces act on the fronto-orbital cortex.

At the site of impact, the positive force of deformation is diminished by the existing negative acceleration pressure that is why contrecoup contusions are formed in falls on head. The degree of acceleration does not depend on the height of fall. It may be greater and produce more damage in a fall from sitting position than from standing position. In fall from high buildings, brain and skull accelerate at the same rate; no acceleration pressures develop within the skull prior to the impact i.e.

why skull may be totally fractured with minimal coup and contrecoup contusions. In a study, the variations of impact due to fall on the back of head were:

- Fall on right parieto-occipital area resulted in contrecoup contusions on left temporal lobe.
- Fall on cerebellar region with force of impact traveled forward causing contrecoup contusions in pons and in gyri.
- Fall on lower surface of occipital region showed intermediary coup contusions in tegmentum of pons.

Indirect Injuries

From the forces transmitted through the body or from its deceleration. As a result of fall on feet, axial skeleton may break up at number of points and ring fracture results.

Fractures of Skull

Goonetilleke classified the pattern of skull fracture in fatalities into five groups:

1. Fracture of vault, including depressed fracture
2. Fracture at the level of brim of a hat
3. Fracture of the base only
4. Extensive fracture of the vault and base
5. Ring fracture at the rim of foramen magnum.

The first four patterns of skull fracture are attributed to direct head impact, either primary or secondary. The ring fracture is associated with feet first and is attributed to indirect fracture, the deceleration force being transmitted up the spine to atlanto-occipital joint.

The scalp can absorb 20 J of energy and 18-55 J forces can cause skull fracture. The extensive damage could be caused due to small increase in energy beyond that needed to produce linear fracture

Injury to Brain

A range of intracranial injury from the extradural, subdural and subdural bleed to brain laceration or contusion. It is clear from many reports that brain

injury may be present without skull and vice versa. Brain and skull injury offer the poorest correlation with height fallen but the severity of brain injury correlates well with probability of mortality.

Other Injuries Resulting from Fall

Rib fractures: The ribs are prone to fracture through direct force and break at the point of impact. Anterior impact produces indirect stress concentration at posterolateral angle of the rib, and laterally because of posterior curvature and caudal angle of ribs.

Cardiac and pulmonary contusion and laceration: Cardiac contusion and laceration are frequently involved in extreme falls. Any chamber may be affected, the atria is more commonly injured in indirect injury without rib fracture. Pneumothorax and haemothorax are commonly associated with thoracic trauma. Pneumothorax in presence of rib fracture is explained by rupture of visceral pleura.

Diaphragmatic injury: Diaphragmatic injuries are infrequent in falls related to deceleration. The mechanism involved is the relative upward movement of the abdominal contents, forced up against the diaphragm as a result of impact to abdomen or deceleration (feet first or head first) as abdominal contents oscillate up and down on impact.

Abdominal viscera: Damage to solid viscera; the liver and spleen are common from fall from height in excess of 12-15 meters. Liver is more commonly involved than spleen with multiple laceration. These injuries are usually coincident with severe overlying trauma to bony thorax but in most cases indirect shear mechanism are involved.

Medicolegal Aspects

The death that occurs due to fall from height must be carefully evaluated. Majority of the cases are

either suicidal or accidental in nature but there may be allegations that the person was thrown/pushed from high rise buildings. Therefore injuries over the body must be evaluated thoroughly while the intervening object to be kept in mind.

1. *Suicidal:* When the person commits suicide by jumping from the building, the body is usually found at a distance from the base line of the building. In these cases, usually the person lands on foot and he is expected to get injury involving both the feet alongwith the fractures, fracture of the pelvic bones that may be pushed upwards. Even at times, there may be fractures of base of skull (Ring fracture) due to transmission of force.
2. *Accidental:* In cases of accidental falls the body is found very close to base line unless there is rolling of the body on landing and usually there is injuries to the head, chest and abdomen.
3. *Homicidal:* On the other hand, if the person has been pushed or thrown from the building the body may be found a little bit away than that of the accidental fall though the injuries may be more or less similar to accidental fall. However, there may be defence wounds on the body during quarell or fight while the person is pushed down. The position of the body after the fall also depends on the weight of the body as well as the surface of landing.

Autopsy Findings

1. Before deciding the nature of crime one has to visit the scene of crime and examine the location and height of the building, presence of any intervening objects, presence of boundry wall at the edge of the roof and the site and nature of injuries over the body.
2. While interpreting injuries over the body following factors should be considered: (i) Body orientation on forces of impact (ii) Distribution of forces of impact (iii) The age of the patient.

Dysbarism and Barotrauma

DYSBARISM

Dysbarism consists of those disturbances in the body exclusive of hypoxia and air sickness that result from existence of a pressure differential between the total ambient barometric pressure and the total pressure of dissolved and free gases within the body tissues, fluids and cavities.

Classification of Dysbarism

Dysbarism has been classified by **Adler** as:

1. *Those due to hyperbarism:* Result from an excess gas pressure over that with in the body fluids, tissues and cavities are noted during the descent.
2. *Hypobaric effects:* Result from excess of gas pressure with in the body fluids, tissues and cavities over ambient gas pressure, are noted during ascent.

Injuries From Hyperbarism (Barotrauma)

Physical effects related to increase in atmospheric pressure are referred to as barotraumas. It occurs most frequently under water. The pressure of underwater environment is always greater than that of atmosphere. When pressure of air or gas in body cavity differs from surrounding tissues, problems of dysbarism arises. The failure to maintain equality of pressure between body and surroundings may be due to the: (i) Weakness of the individual himself (ii) Inadequacy of the protection available to him or his equipment (iii) Mishandling of the equipment by other people.

When an individual inhales a lungful of air and plunges in to aquatic environment, as he descends in to water, chest pressure increases with increasing depth and the volume of air in lungs is decreased as air pressure increases. At about 100 ft, the total respiratory volume may be reduced to residual volume and with increasing descent air in the lungs is at constant volume and pressure but pressure of pulmonary circulation and tissue wall continues to increase. Thus a pressure difference is created resulting in pulmonary oedema by fluid transfer from the alveolar capillaries into alveolar spaces leading to vessel rupture and haemorrhage, the air pressure increases than that of tissues that results in pulmonary tissue rupture and air escapes into interstitial tissue or pleural cavity or be drawn into circulation.

High altitude decompression sickness has symptoms similar to those of Caisson disease but the two differs in circumstances of development and progression of the process.

Syndromes of Barotraumas

Pulmonary Barotraumas

This can manifest as pneumothorax, interstitial emphysema and air embolism causing chest and abdominal pain, nose bleeding, haemoptysis, coughing and cyanosis. Immediate recompression is mandatory to shrink the embolism bubble mass. The clinical effects of air embolism depend upon the actual site namely, convulsions, visual changes, spastic/ flaccid paralysis, tingling in the

limbs and vertigo.

Barotalgia

The second location where injury from barotraumas can occur is in middle ear where air pressure differs from that in the external ear and nasopharynx. If air pressure in middle ear cannot be kept in equilibrium with that of nasopharynx for example because of inflammation of eustachian tube, eardrum may bulge inwards resulting in stretching, pain, haemorrhage, and ultimately perforations. This occurs in sudden descents from high places such as mountains or in landing of an aircraft, and in diving. In diving this is more of a problem but acute pain prevents diver to go deep enough to produce the perforation in his ear. If an individual is wearing a tight fitting cap, air may be trapped in external canal.

Barosinusitis and Barodontalgia

Divers experience vertigo or dizziness under water and those with sinusitis or polyps of nasopharynx may experience a painful condition owing to pressure variations between surrounding tissue and sinuses. Small pockets of gas resulting from fermentation in the roots of teeth may shrink during compression and the space becomes occupied by blood or fluid with increased pressure and pain when the diver returns to the surface.

Intestinal Complications

As a result of increased volume of gaseous content of intestinal tract, continued fermentation may produce additional gas which will cause abdominal distension, discomfort and embarrassing flatulence upon return to the surface.

Nitrogen Narcosis and Oxygen Poisoning

Despite the chemically unreactive nature of some noble gases at certain pressures they display all typical properties of anesthetic agents.

Nitrogen makes up about 79% of the air we breathe, produces euphoria followed by narcosis. With alcohol, the effect of nitrogen is greatly

enhanced. Usually upon release of pressure, excess nitrogen leaves tissues and recovery is complete. Helium is used in place of nitrogen in breathing mixtures which allows for deeper diving potentials.

Hyperemia (Oxygen poisoning)

The effects may result from prolonged exposure to low oxygen concentration or brief exposure to very high tensions (1500 mmHg).

Acute oxygen poisoning is much more common danger in diving. Convulsions are reported if divers have breathed pure oxygen at a pressure of 4 atm. At depths greater than 25 ft pure oxygen is not currently used. Mechanism of oxygen poisoning includes an excessively high partial pressure of oxygen.

In *chronic oxygen poisoning*, irritant effect on lung tissue from smaller excess pressure for a considerable time may be there.

SCUBA Diving Fatalities

SCUBA is a Self Contained Underwater Breathing Apparatus. The diver is supplied by one or more tanks strapped to his back, which enable him to remain submerged for indefinite periods of time

Snorkeling: The hazards are similar to that of swimming. It refers to diving apparatus that has mouth piece at one end and the other end of the tube remains above the surface of water unless the diver submerges, when the spherical float attached to it keeps the water from entering it. The diver holds his breath for the length of time he remains underwater. These fatalities are contributed by four basic groups of factors:

1. *Panic in minor emergencies such as*: (i) Loss of a mouth piece or face mask (ii) Entrapment in kelp or gill net (iii) Poor judgment in estimating distance from safety.
2. *Improper use or maintenance of equipment*: (i) Insufficient servicing (ii) Use of defective components (iii) Occasionally regulators are defective (iv) Water may be inhaled instead of air.

3. *Acute alcoholism*: Due to alcoholism, judgments are impaired and inhibitions are lost.
4. *Organic diseases*: Cardiovascular diseases especially severe coronary sclerosis.

Hypoxia is associated with increased susceptibility to the effects of alcohol, impairment of coordination of muscular activity and decreased perception of depth and colour.

Hazards of Scuba diving include drowning, barotraumas, bends (Caisson's disease), acute pulmonary oedema, emphysema, pneumothorax and air embolism. In Scuba diving, frequently there is entrapment of air with in the lungs on rising from depths producing fatal or non-fatal extra-alveolar air syndrome. Air escapes from alveoli and may result in interstitial emphysema, pneumothorax and air embolism. It is caused by disproportionate expansion of air containing alveoli compared to the adjacent fluid filled vascular changes during too rapid an ascent.

Water Accident Investigation Team (WAIT)

The medicolegal investigation technique for water accidents is developed by Noguchi. It consists of forensic pathologist and his medical or paramedical support team, diving expert (may be a medical officer who has sufficient experience in hyper baric medicine). The work of this team is coordinated by means of a liaison officer. Every scuba fatality should be thoroughly documented and investigated as to what happened, circumstances surrounding the death and what recommendations should be made to prevent such incidents.

The training should be improved and licensing and maintenance of diving gear is important to prevent such fatalities. A coordinated effort should be made with active sports diving equipment manufacturers.

Medicolegal Autopsy in Fatal Water Accidents including Scuba Deaths

1. Do not remove any mask or gear from the divers, as equipment should be examined for flaws such as absence of a weight belt.

2. Photograph the tank and send it to proper testing facility.
3. Leave wetsuit undisturbed until the autopsy begins.
4. Document the time of notification, arrival time and diving time.
5. Conversations with witnesses such as skipper, other divers and diving instructions are important.
6. Photograph the body in position in which it was found underwater (face down, face up) as soon as possible.
7. Note the condition of the body especially rigor mortis.
8. There should be minimum disturbance of the position of the remains, diving gear, wetsuit and safety equipments.
9. Transfer the body to the examining room without delay to prevent artefacts being increased by putrefaction.

External examination: The external examination should be conducted from top of head to tips of toe without missing any surface of skin as:

1. Evidence of squeezing or pressure of skin.
2. Eyelid, conjunctiva, sclera, neck and thoracic wall should be examined for subcutaneous emphysema.
3. Radiological examination: X-ray of chest in standing position will indicate a fluid level in chest cavity or presence of pneumothorax or pneumomediastinum. Radiographs of knees will show nitrogen bubbles in the joint, indicating a long diving period.
4. In case of air embolism, air samples should be removed with a syringe lubricated by oil for gas chromatography and electron mass spectrometer analysis of gas components. >95% nitrogen indicates nitrogen narcosis (In case of massive air embolism from rupture of lungs, O₂ to N₂ ratio would be 20 : 80%).
 - While giving skin incisions, never overlook small air bubbles coming out from subcutaneous tissue.
 - Blood is generally fluid, not coagulated.
 - Heart need not be opened underwater.

- It is more important to measure total amount of air in heart chambers to find out air embolism.
5. Stomach contents must be examined for presence of food, drugs or alcohol.

Injuries Relating to Low Atmospheric Pressure (Hypobarism)

The barometric or atmospheric pressure at sea level is above 760 mm Hg. This pressure is referred to as standard or 1atm if temperature is 0°C. The effects of hypobarism are related to its causation by: (i) Evolved gas types (ii) Trapped gas types.

Evolved Gas Types

Gases are evolved and form bubbles when with a rapid decrease in ambient pressure, tissues saturated with a dissolved gas may become supersaturated. Nitrogen is implicated in most of the cases but oxygen and carbon dioxide may also form and cause dysbarism. In the body nitrogen is transported in simple solution according to Henry's law. It enters into and is removed from the body by establishment of equilibrium between nitrogen pressure in the alveolar air and venous blood and another between tissues and arterial blood. During an ascent, ambient nitrogen pressure is decreased and nitrogen transport is directed towards its removal from the lungs. Nitrogen in tissue, then being higher in pressure than that of arterial blood, is transported to blood then to alveolar air and is expired.

As only a small amount of nitrogen can be transported by a given amount of blood, the rapid reduction in ambient pressure results in super saturation of the tissues with the resultant bubble formation. Nitrogen bubbles can form in any tissue including blood and are characterized by bends and chokes.

Bends are joint pains caused by evolved gas bubbles with in the joints and

Chokes are characterized by cough, chest pain and dyspnoea due to pulmonary evolved gas as

well as mediastinal emphysema from evolved gas bubbles.

CNS symptoms are caused by evolved gas pressure on or in the brain as well as embolic phenomenon. CNS disturbances are produced due to evolved gas bubbles under the skin. *Micro-circulatory phenomenon* are produced due to bubble pressure on nerves and blood vessels.

Trapped Gas Types

Gases may be trapped in hollow viscera, teeth or body cavities with resultant pain and possibility of ruptures. Gases may be trapped in sinuses, middle ear, teeth, joints, beneath skin, and intestinal tract.

Factor Effecting Clinical Manifestations of Hypobarism

1. Age.
2. *Obesity*: Fatty tissue contains less blood than muscular tissues and nitrogen is five times as soluble in fat as in muscle.
3. Exercise, because of concomitant increase of tissue metabolic carbon dioxide.
4. Rate of ascent.
5. Attained altitude during ascent.
6. Increased tissue pressure of nitrogen prior to ascent.
7. *Recent scuba diving*: United States has adopted the 'Rule of 12' to cover the circumstances in which hypobarism occurs at lower altitudes; if individual has been Scuba diving for 12 hours. 'No flight above 12,000 ft for 12 hours following a Scuba dive of 12 ft or more'. Physiological reason for this rule is that enough time must be allowed at sea level, breathing air to complete respiratory wash out of the excess nitrogen stored in the tissues during the dive.
8. *Lower ambient temperature*: Occurs with great frequency because of decreased nitrogen transport associated with peripheral vasoconstriction due to cold.
9. Total dissolved gas tension.

10. *Previous injury*: Bubbles tend to form at the sites of recent injuries
11. *Repeated exposure*: Having one such attack of dysbarism is prone to another episode

Clinical Features

1. *The Bends*: Onset follows the ascent with a variable lag period. It includes joint symptoms, pain in muscles, long bones. The areas involved are first the knee then shoulder, elbow, wrist, hand and finger in decreasing order. The features are not serious and may be relieved in descent
2. *The Chokes*: Are characterized by substantial distress, nonproductive cough, respiratory distress, sense of apprehension and suffocation. The chokes are more serious than the bends. They result from bubble formation and accumulation in the pulmonary capillaries or from the effects of extravascular mediastinal bubbles exerting pressures on mediastinal contents and adjacent pulmonary tissue. It may be solitary manifestation of hypobarism and may accompany the bends. Chokes are found later than bends in the flight and become progressively disabling. Cough and substantial distress are markedly aggravated when an attempt is made to take deep breath resulting in decreased pulmonary ventilation, then hypoxia, syncope and collapse. Onset of chokes demands: (i) Prompt attention (ii) Return to normal pressure by way of descent to lower altitude (iii) Termination of

flight of aircraft (iv) Removal from low pressure chambers.

3. *CNS involvement*: Vision is disturbed and there is disturbance in orientation. This is related to bubble size and location of lodging in the arterioles and capillaries of the brain, indicate mandatory hospitalization.
4. *Skin manifestation*: Are due to pressure on dermal nerve endings and sub dermal emphysema revealed by crepitation on palpation.
5. *Rapid and explosive decompression*: They result from mechanical failure of aircraft pressurization system.
6. *Explosive decompression*: Loss of pressure is less than a second in explosive decompression and more than a second in rapid decompression. The eardrums are ruptured in the absence of basal skull fractures. Microscopic examination of lungs will reveal presence of bone marrow emboli resulting from nonfatal injuries sustained before ground impact.

Long Question

1. Discuss injuries caused by exposure of humans to high pressure environment.

Short Questions

1. Barotalgia
2. Barodontalgia
3. SCUBA
4. Dysbarism.

Thermal Injuries

HYPERTHERMIC INJURIES

Hyperthermia can cause systemic as well as localized injuries to the body. A delicate balance between heat loss and heat dissipation maintains thermal homeostasis. To maintain it both environmental and internal heat of the body are involved. Internal heat is generated by the oxidation of heat produced during rest that is the basal metabolism of the body. Environmental heat comes from the sun and its effects are influenced by:

- i. Moisture content of the air
- ii. By its wavelength
- iii. Spectral distribution
- iv. Absorption by ozone and dust
- v. Reflection and absorption by skin
- vi. Clothing worn by the individual

Both the internal and environmental heat is measured in calories. **A calorie** represents the quantity of heat needed to raise a kilogram of water from 0° C to 1°C.

Exogenous or No Febrile Hyperthermia

The human body reacts to an environmental elevation of temperature by reducing thermo genesis and by increasing heat dispersion. Thermo genesis is decreased by reduction of metabolic rate to the minimal required for the vital functions and thermo dispersion is achieved by: (i) peripheral vasodilatation (ii) profuse sweating (iii) evaporation converting sweat to water vapour. All these factors are affected by the high humidity and lack of air movement.

The diseased states due to the effect of excessive heat are divided in to four categories in progressive order of severity:

- Heat cramps
- Heat exhaustion
- Thermogenic anhydrous
- Heat stroke.

Heat Cramps (Boilers room, Miners cane cutters)

The painful spasm of the voluntary muscles especially of the extremities and abdominal wall is produced during or after physical activity in a hot environment. The predisposing factors such as ill health and intake of alcohol result in depletion of salt that are the main causes of heat cramps. The cramps are severe, paroxysmal and affect the muscles of legs, arms and abdominal wall. Flushing of face and dilatation of pupil occurs. Administration of physiologic saline brings about dramatic relief. Prophylactically, salt tablets can be used to combat heat cramps.

Heat Syncope

Heat syncope is a circulatory phenomenon induced by posture and/or exercises.

Heat Exhaustion

Heat exhaustion results from dehydration and salt depletion producing the following symptoms: (i) intense thirst (ii) nausea, vomiting and headache. (iii) mental fogginess (iv) dizziness (v) irritability

(vi) incapability to work (vii) fainting (viii) peripheral vascular collapse (ix) sweating (x) temperature may or may not rise (xi) skin is not dry (xii) oliguria (xiii) death results from heart failure.

Thermogenic Anhydrous

Thermogenic anhydrous is also known as **Desert syndrome or Anhydrite heat exhaustion**. It occurred in some soldiers who were separated during the summer in American desert. The features are most likely that of heat exhaustion. After profuse generalised sweating for several days perspiration ceases suddenly in all parts of the body below the neck region and persists in face and neck. Eruption of prickly heat rash is seen known as *miliria profunda*. The condition is not uncommon in Bengali children, tanker personnel reforming from Persian Gulf. There is no suitable explanation for this. There is hyperkeratotic plugging of sweat glands leading to functional failure of sweat apparatus or a combination of both.

Heat Stroke

In heat stroke, there is complete absence of sweating and the body temperature is raised to 106°F or 41°C. In the environmental heat stress, absence of any other cause of pyrexia constitutes heat hyperpyrexia. Several workers have pointed out that heat stroke can occur at lower temperatures also. At a temperature of 32°C with 100% humidity may lead to heat stroke. The features of heat stroke include hot and dry skin, increased depth of respiration, pulse is raised to 160-180/min and there is severe asthenia. Cerebral deficit produces convulsions, delirium, stupor and coma. When there is hypotension, increased urea concentration and raised potassium levels then prognosis is bad. Urine shows proteins and casts. Serum iron and bilirubin levels are raised due to liver damage. SGOT is raised due to liver and muscle damage. The first 24 hours are critical and death usually occurs within a week.

Autopsy Findings of Hyperthermic Deaths

1. The autopsy findings in hyperthermic deaths are similar to those in cardiac failure.

2. Visceral haemorrhages and congestion in heart, liver, lungs and kidneys are present due to thrombocytopenia, decreased plasma fibrinogen concentration and decreased prothrombin levels causing hepatic damage.
3. Haemorrhage also occurs due to increased capillary fragility due to anoxia and circulatory collapse.
4. Patients who survive for more than 24 hours show lobar pneumonia, acute tubular necrosis, centrilobular necrosis, adrenal necrosis and haemorrhages.
5. Heart shows subendocardial haemorrhages, changes in the muscle fibers and the chambers are dilated.
6. Brain is congested and oedematous; convolutions are flattened and scattered patches are found in the walls of the third ventricle and floor of the fourth ventricle.
7. The cerebellar changes are more rapid and in the form of oedema of purkinje layer and swelling, degeneration and reduction of purkinje cells.
8. Those who survive longer show rarefaction of granular layer along with oedema of hypothalamic nuclei.
9. In the brain, the heat dissipation center (pre-optic and supra-optic area) and the heat conservation and production center (occipitolateral portion of hypothalamus) are usually affected.

Medicolegal Aspects of hyperthermia

1. All hyperthermic deaths are accidental
2. Autopsy examination is done to rule out any other cause of death or contributory cause of death.

HYPOTHERMIC INJURIES

The term systemic hypothermia is used to indicate cooling of the human body below 35°C (95°F). This occurs when the loss of heat exceeds the production.

Causes of Hypothermia

1. Hypothermia caused by exposure to severe cold possibly with exhaustion called environmental

hypothermia depending on environmental factors.

2. Hypothermia under the influence of drugs and alcohol affecting the generation or conservation of body heat.
3. Hypothermia caused by climatic conditions with some disease affecting the body resistance.
4. Hypothermia ending fatally in connection with therapeutic procedure that has some medico-legal importance.

Types of Hypothermia

1. Dry cold hypothermia
2. Immersion hypothermia

Accidental Hypothermia

It is produced during (i) hiking or skiing expeditions (ii) in ship wrecked, (iii) people falling in ice water or (iv) when there is insufficient heating facility in the room.

Immersion Hypothermia

In immersion hypothermia, the loss of heat is three times more than that due to exposure to dry cold air. Water conducts heat 25 times faster than dry air. Insulating air between skin and water is 1/10th than that of skin and air. Women endure cold water better than males due to body fat. Human body is able to retain body heat in water up to 22°C and below 16°C, survival time decreases sharply. Immersion hypothermia is more fatal as there is rapid loss of heat.

Diagnosis of Hypothermia

I Clinical signs and symptoms

Body temperature preferably oesophageal (rectal temperature is also satisfactory) should be taken. Clinically three phases are produced:

1. Body temperature is 37°C-32°C with shivering and vasoconstriction.
2. Body temperature is 32°C-24°C and body response to production of heat grossly disappears.
3. Body temperature is below 24°C, body cannot withstand the heat loss and general paralysis is produced.

Clinical features in children: In children features produced are cold skin, redness of face, subcutaneous oedema, scleroma, muscular stiffness, feeble cry, unable to suck, cutaneous and mucosal lesions are uncommonly present. Convulsions are also seen in some cases.

Clinical features in adults: In adults the features produced are red patches over the skin, pallor of skin, subcutaneous oedema of the face, stiffness of neck muscles, mental disorders, pulse is decreased and irregular with hypotension and respiration is slow.

II. Laboratory Findings

1. E.C.G. is mandatory and following findings are discerned (i) occurrence of 'J' wave (ii) widening of QRS complex (iii) prolongation of PQ interval (iv) Inversion of 'T' wave.
2. Hyperglycemia.
3. Raised blood urea and lactic acid levels.
4. Haemoconcentration.
5. Serum amylase levels are increased.

Causes of Death

The mechanism of death in hypothermia is:

1. Cessation of circulation preceded by ventricular fibrillation; the dangerous level of temperature for ventricular fibrillation is 26°C-30°C though people have survived at a much lower temperature.
2. There is decreased supply of oxygen to the central nervous system with resulting hypoxia of the brain cells.

Autopsy Findings

1. Postmortem staining is red or pink due to antemortem binding of oxygen to haemoglobin and its postmortem diffusion through the skin.
2. Rarely the skin is white that is why hypothermic deaths are called the '**white deaths**'.
3. Spots of bluish discoloration are seen on the hands, elbows, and knees. They are minor frostbite lesions.

4. In the heart, there is dilatation of right atrium and ventricle and congestion of vena cava.
 5. Trachea contains frothy and sanguineous fluid.
 6. Lungs are congested, oedematous and shows haemorrhages.
 7. Bronchopneumonia is a frequent complication.
 8. Liver and spleen are congested.
 9. Gastric mucosal erosions occur in 72% of cases.
 10. Kidney shows acute tubular necrosis.
 11. Fat deposition occurs in the cardiac and skeletal muscles and there is focal myocardial necrosis.
 12. Viscera should be submitted for blood alcohol and toxicological analysis.
 13. Histological examination of skin shows oedema and hyperemia of the dermis with occasional inflammatory cell infiltration.
3. *Cold panniculitis*: This can be produced with local application of ice. There is inflammation and necrosis of the underlying adipose tissue. The children are most commonly affected because the fat of the children contains more saturated fatty acids than that of adults. The finding could be of interest in a battered baby syndrome as deliberate exposure of an infant to cold develops panniculitis.
 4. *Immersion foot and trench foot*: Immersion foot and trench foot usually occurs at a temperature above 0°C most commonly when the temperature is between 5-8°C. Wetness is an important contributory factor. There is necrosis of fingers and toes. Obliterating angiitis causes severe gangrene and loss of tissue.
 5. *Frostbite*: Frostbite occurs due to exposure to dry cold as well as following immersion at temperature below 0°C (usually -2.5°C). It is commonly seen in mountaineering and polar expeditions. It is of superficial type when only the skin is affected and deep type when subcutaneous fat, muscles, nerves and bones are affected. Degrees of frostbite and their manifestations are given in table 19.1.

Treatment

1. Re-warming by warm water (40-42°C); but never rewarm the patient by exercise or intense dry heat or fire as it leads to gangrene.
2. The blisters should not be pricked.
3. Antibiotics should be given to combat infection.
4. Alcohol and tobacco is to be avoided as they hamper the peripheral blood flow.

Local Cold Injuries

1. *Chilblains (Acute perineo)*: Chilblains can be categorized as a first-degree frostbite. The skin is red or bluish as there is capillary congestion and mild inflammation. As a rule, complete recovery occurs without residual deficit.
2. *Chronic perineo*: Chronic perineo presents with reddish flat nodules in the skin, seen commonly in the women involving the lower limbs. There is ulceration, haemorrhages and scarring. These lesions become activated in the cold weather. The microscopic findings include skin atrophy, dermal fibrosis, panniculitis, angiitis, and sclerosis of arterial walls.

Table 19.1: Degree of frostbite and their manifestations

Degrees of frostbite	Features
First degree	Redness and oedema of skin
Second degree	Only epidermis is affected, there is blister formation
Third degree	Skin and subcutaneous tissue is necrosed
Fourth degree	Total loss of tissue in the exposed area including muscles and bone

ELECTRICAL INJURIES

Factors Influencing Electrical Injuries

The factors are that affect production of electrical injuries and shock are:

1. *Voltage or tension*: Voltage is less important than the amperage or intensity.
Low voltage (<50 volts)—It is used for therapeutic purposes and rarely causes

accidents. Low tensions of A.C. can cause fatalities. Tension as low as 25 volts passes through heart and can cause deaths.

Medium voltage (< 500 volts)—This voltage predisposes to prolonged contact because the victim grips and holds on to the conductor. It really depends upon amperage rather than voltage. The typical 'Joule burn' cases are related to "Hold on" cases. The agents commonly responsible are hand tools, overhead lines, switchgears and lamps etc. Most fatalities occur at 220-250 volts that is the usual household supply.

High voltage—It is the voltage in thousands and the death does not occur necessarily. Life can be saved depending upon the time of exposure to this voltage.

2. *Amperage (Intensity)*: It is the most important factor in electrocution. It is the cause of "hold on" to conductors that is the cause of serious local damage and fatal shock. It is given by $A=V/R$; Where V =voltage, R = Resistance of the conductor.

Contact with the current at 1mA causes tingling sensation. As amperage rises, it is a more painful experience. Normal healthy man can tolerate up to 21.6 mA but 'hold on' occurs at 8-9 mA. Women are less tolerant, the maximum level is 14 mA and 'hold on' occurs at 6 mA. Danger increases when the amperage is between 100 mA to 4 A. Above 4 A, it arrests cardiac fibrillation; this is the principle of treatment with a defibrillator. High voltage with low amperage is less dangerous than moderate voltage with high amperage.

3. *Density of the current*: .Density of the current depends upon the following
 - Dry or moist skin: Dry skin of the palm have the resistance of 1-2 million Ohms. Sweating reduces the resistance from 30,000 ohm to 2500 ohm.
 - When moistened with water or saline the density of current further drops between 1200-1500 ohms.

- The charges in skin due to current is less than 380 ohms but once it is burnt, resistance of the skin rises and that may cause a break in the circuit.

4. *Resistance of the body*: It plays an important part causing effect of elctrocution. Effects will be modified according to the part of the body that comes in contact with the current. Resistance is greatest on the palms (of the labourers) and least on the inner sides of thighs.Average resistance of various body areas and tissues is:

- Skin is 500-10,000 ohms
- Mucosa is 1500-2000 ohms
- Bone is 9,00,000 ohms
- Vascular areas like cheek are better conductors and most of the current passes through the blood vessels.

5. *Insulation*: Stout rubber gloves and rubber boots in good condition gives considerable protection . Therefore, all the tools used in electricity should be adequately insulated.

6. *Contact with electric supply*: Electrocution is normally the result of direct contact with 240 volts A.C. and closer the contact, greater is the danger especially when it results in 'hold on', glancing touch or fall followed by a break in the circuit. High tension currents throw or repel violently and the death may be due to the fall. Broad and good contact reduces the skin resistance from 100,000 ohm to 1000 ohm. Indirect contact with high-tension current results in arcing and direct flow of current in an indirect fashion such as while urinating on a high-tension conductor. The area of contact modifies external appearances such as:

- Conductor involving small surface area such as the end of a rod or wire produces circular hole in the tissue and simulates bullet injury.
- A wire wound around wrist produces linear grooves.
- A broad conductor closely applied may not leave any mark.

*In electrocution in a bath, flow of current occurs without generation of heat and no external injuries are produced.

7. *Duration of contact:* Longer the contact, greater is the damage. There is formation of electric mark, blister formation, charring of the tissues, Joule burn and destruction of tissues with exposure of muscles and bones.
8. *Site of contact:* Electrical injuries on the face and arms are serious whereas those on the palms are less serious. When the path taken by the current is from left hand to leg, 2-3 times more current is passed to the heart compared to the path taken from right hand to leg. Various paths taken by the current are shown in Figure. 19.1

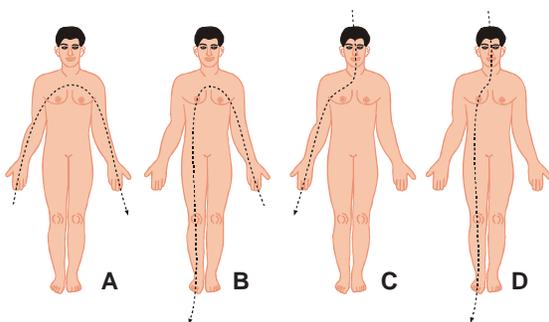


Figure 19.1: Various paths taken by the current: A-Hand to hand, B-Hand to feet, C-Head to hand, D-Head to feet

9. *The kind of electrode:* **Jellineck** found that area of contact did not determine the severity of shock but others believe that it is important.
10. *Kind of current:* A.C. is more dangerous than D.C and the numbers of cycles in A.C are important. The rate of: (i) 40-150 cycles/second is dangerous (ii) 50-60 cps is the critical frequency (iii) At frequencies more than 1700 cps, heart becomes 20 times more tolerant.
11. *Personal idiosyncrasies:* Individual personality, physical condition and existence

of mental or bodily distress influence the effect of shock.

12. *Presence of diseases:* Cardiac diseases may predispose to electrocution even at low tension current.
13. *Anticipation of shock:* In anticipation of shock, the individuals can sustain current better
14. *Sleep:* Sleep increases the resistance to an electrical current
15. *Important factor:* The effect = Voltage \times Amperage \times Duration—tolerance and alertness.

Mechanism of Electrocution

Ventricular fibrillation: **Prevost and Botelike** first described ventricular fibrillation in 1899. It is an important factor in low and medium voltage currents. Ventricular fibrillation is not always associated with loss of consciousness or respiration. Some individuals can also walk about before they die. Cyanosis is not seen in these cases.

Titanic asphyxia: Low or medium voltage current passing through the chest of 20-30 mA can induce titanic contraction of the extrinsic muscles of respiration and death from mechanical asphyxia. Cyanosis is present in these cases. Production of Joule burn at the point of contact may lower the resistance thus increasing the amperage above the critical level at which the ventricular fibrillation occurs.

Respiratory arrest: Route of current passes through the respiratory center and is most likely when the head is involved or current passes through arm to arm.

Cerebral anoxia: Prolonged ventricular fibrillation may cause brain damage due to inadequate blood supply. In cases of survival, permanent cerebral damage occurs.

Neurological damage: Current may cause tearing of nervous tissue, shrinkage around the smaller blood vessels and rupture of layer of large blood vessels and elastic membrane.

Injuries Produced in Electrocutation

Low and Medium Tension Injuries

The electric mark: The electric mark is found at the point of entry mostly associated with the thermal burns. The typical mark is usually absent. The mark may be trivial at times. Rarely there may not be any mark, but it only indicates inadequate search by the pathologist rather than the absence of mark. The electric mark is specific for contact with electricity but not a proof of electrocution as an electrocution mark may be produced after death. However, when the mark is present, there is strong presumption of death due to electrocution.

Features: The mark is in the form of round or oval, shallow crater bordered by a ridge of skin 1-3 mm high, partly or fully around its circumference (Figs 19.2 to 19.4). The floor is lined by pale, flattened skin. Ridge pattern is preserved but due to flattening ridges are broad and scarcely above the general level. At times, there is breach in the continuity of skin that should be differentiated from broken blister. The skin is pale and hyperemia is present beyond the mark. The shape of the conductor or part of it determines shape of the electric mark. When the conductor is rod like wire or flex, round or oval mark is produced. Contact with the long axis of wire produces linear mark or groove whereas contact with the end of the wire produces a deep hole in to muscles or bone that needs to be differentiated from the bullet injury.

The Joule Burn (Endogenous burn): When contact is more prolonged, skin shows biscuit or brown tint that on further contact produces charring due to burning that is why it is called Joule burn. Flash burn is the term that distinguishes it from the changes caused by exogenous heat following contact with high voltage. Electric mark and Joule burn are usually found on the exposed parts of the body and more commonly the palmer aspect of hand especially the tips of index finger and thumb in the hands of electriciticians. It can be present anywhere on the body and can be produced beneath the intact clothing that may



Figure 19.2: Electrical entry wound over the palm in a case of electrocution



Figure 19.3: Electrical entry wound due to fall of high tension wire



Figure 19.4: Electrical entry wound in a case of electrocution

be undamaged by thermal heat such as shoulder or thigh. The Joule burn is produced by the conversion of electricity in to heat within the tissues, the so-called **endogenous burns**. Heat generated in the skin (corium) and subcutaneous tissue causes the fluid to boil to produce blisters by prolonged process and the steam thus generated bursts through the skin.

High-tension Injuries (Exogenous Burns)

High-tension injuries are either due to direct contact or indirectly by arcing of the flash. As much heat is generated in the arcing of the flash and there is knock down by sudden increase of local atmospheric pressure, multiple discrete lesions due to arcing are produced with the blast effect. These are called **crocodile burns** (Fig. 19.5). When someone climbs on to an electric pole for suicide or theft of cable, there are chances of arcing when the person comes nearer to the conducting wire. Distance between person and cable leading to arching is related to voltage. At the voltage of 5000 voltages, the distance between the individual and the person should be 1mm and at 1,00,000 voltages there should 35 mm distance for arching (**Mueller** cited by Simonin, 1955).

Although flash burns are extensive and severe and they may be exaggerated from ignited

clothing, direct contact causes gross destruction of soft tissues, charring of bones or fusion of bone in to pearl like bodies and sequestration of dead bones that are seen radiologically as '**bone pearls**' or '**wax drippings**'. In flash burns due to revitalization of tissue, healing may take a long time even up to 6 years

Exit mark: Exit mark is variable in appearance and has some of the features of electrical entrance mark. Exit mark causes more disruption of tissues and is seen as splits in the skin where the skin has been raised in to ridges by the passage of current. Splitting may be continuous or interrupted. There is honeycomb appearance of damaged tissue, which is seen microscopically (Fig. 19.6).

Metallisation: Metallisation is a specific feature of electrocution and lightning stroke, seen more commonly and completely in the latter conditions. The following features are encountered:

- The face of the victim may become darkened brown and black.
- The colour of the skin varies with the nature of the conductor; if the conductor is iron, the skin is black or brown and when the conductor is copper; the skin is yellow brown or blue. It is due to volatilization of the metal particles that are driven in to the skin.
- Small gray, green areas are present in the floor of the electric mark and the face of the



Figure 19.5: Crocodile burns in a case of electrocution



Figure 19.6: Electrical exit wound over the sole

brass becomes pitted and dull. It occurs in low and medium voltages.

Metallisation is detected by low magnifications on histological examination and chemical examination. Metallic deposit is invariably present and for this acroreaction test should be done. It is useful even in advanced putrefaction. A positive test is a proof of electrocution. Depending on the composition of the conductor, metallic deposits are mainly in the superficial layers of the skin and hair follicles seen on histological examination.

Acroreaction test: A micro-chemical test for metals devised by **Skalos** in 1944 for the identification of electric marks. It is based on the solubility of metals either in the hydrochloric acid or nitric acid. Iron, aluminum, zinc and nickel are soluble in hydrochloric acid except copper whereas all of the above except iron are soluble in nitric acid. Metal of the electric mark is dissolved with the acid and this solution is kept on a filter paper with the reagent, the colour change will occur. When iron is the metal, over the filter paper add a solution of potassium ferrocyanide and the blue colouration will indicate iron.

Microscopic examination: The epidermis is flattened and there is distortion of the cells but no disintegration of cells occurs on microscopical examination.

Description of Electrocution Injuries

Confusion has arisen because the forensic pathologists constantly describe the effects of electric shock as burns. It is better to call the mark of electricity as electric mark and distinguish it from the effects of a flash that produces burns akin to flame burns. Joule burn is produced by same mechanism that causes electric mark but prolonged contact is needed. Visible damage is usually the result of high-tension current or prolonged contact with low or medium tension current.

Delayed Effects of Electrocution

1. Gangrene and haemorrhage due to arterial damage and necrosis of the tissue.

2. Damage to spinal cord
3. Aseptic necrosis and resultant depression of the bone marrow
4. Angina pectoris or myocardial necrosis
5. Eye injuries resulting in cataract and optic atrophy.

Autopsy Findings

1. Electric mark and Joule burn are the pathognomic features when low and medium voltage current is involved. Proof of electric mark is obtained by histological and photochemical (Acroreaction) examination.
 2. High-tension currents cause gross thermal injuries that are the result of direct contact, flash over, or thermal burns.
 3. The circumstantial findings are corroborative in cases of electrocution.
 4. In electrocution in a bath, the following findings are present:
 - No external injuries are present over the body
 - On examination of the scene, live wire is recovered.
 - Exclusion of other causes of death such as natural disease and poisoning etc. is to be ruled out.
 5. Other non-specific findings may be present such as cyanosis of the face, petechial haemorrhages over the face, pleura and pericardium.
 6. The viscera is congested and lungs are intensely congested and oedematous.
 7. Heart is dilated due to fibrillation
 8. Froth is present over the mouth and nostrils
 9. *ostmortem electric mark:* Electric mark unaccompanied by hyperemia can be produced even after death. It is possible to produce changes in the skin, which resemble electric mark by applying Bunsen burner or a glowing wire. In electric mark, acroreaction is positive but this is negative in thermal burns.
- Judicial electrocution:** Death penalty is carried out in an electric chair in USA. The condemned man is strapped to a wooden chair and one cap like electrode is put on the shaven scalp,

moistened with the conducting paste and the other is applied to the right lower leg. A current of 2000 volts and 7 amperes is passed through the body. After titanic spasm and loss of consciousness, the same current is passed for a second time for one minute.

Medicolegal Aspects

Electrical burns are usually accidental resulting from defective appliances or negligence in the equipment. They may be produced from application of live wire during convulsive therapy of mentally diseased patients. Homicidal electric burns may occur during theft and Suicidal electric burns are rare.

THERMAL BURNS

Burns are injuries that are produced by the application of dry heat such as flame, radiant heat, and some heated solid substance like metal or glass to the surface of the body resulting in tissue destruction. Injuries caused by friction, lightning, electricity, UV rays, infra red light, X-rays and corrosive chemical substances are also classified as burns for medicolegal purposes.

Scalds are moist heat injuries produced by the application to the body of a liquid at or near its boiling point or in its gaseous form steam. Water at 60°C for 10 seconds at vulnerable parts produces scalds.

Microscopically, the epithelial cells are elongated and flattened that are deeply stained with haematoxylin and eosin. There are small haemorrhages in the deeper layers and vacuoles are also seen.

Varieties of Burns

The burns are produced at a minimum temperature of 44°C for 5-6 hours and at 65°C for two seconds. External appearances vary with the nature of the substance used to produce the burns such as:

Highly heated solid body or molten metal: It produces blisters and reddening of the corresponding size. When kept in contact with the part, the destruction and charring occurs.

Cuticle is found blackened and dry with shriveled appearance. Hair is seinged and distorted.

Burns by flame: These burns may or may not produce vesication but the singeing and blackening is always present. The hairs are curled, twisted, blackish, breaks off or are totally distorted.

Kerosene oil and petrol: Produces severe and sooty blackening of the parts, and have a characteristic odour.

Explosion in coal mine and gun powder: Causes extensive burns and tattooing due to deposition of particles and splinters or fragments are present in the tissues.

X-rays and radiation: The injuries vary from redness to dermatitis with shedding of hair and epidermis with pigmentation of the surrounding skin. Nails show degenerative changes and wart like growth. Severe exposure may produce vesicles and pustules. The scars radiate in shape with surrounding pigmentation.

U-V rays: Erythematic and eczematous dermatitis and vesication are seen.

Corrosive substances: The corrosive substances produce ulcerated patches, line of redness is absent and there is no singeing of hair

Electrical: The characteristic electrical entry and exit mark is present

Lightning stroke: The lightning injuries are linear in shape present in moist creases or skin folds. Arborescent markings like branches of tree on the chest are present due to staining of the tissues by haemoglobin from lyses of red cells. Lightning is always accidental and there is history of rain thunders. True burns occur beneath the metallic objects that the person is wearing or carrying.

The differences between the burns due to dry heat, moist heat and chemical are as given in Table 19.2.

Preternatural Combustibility: During life gases from anus can cause partial burning but during putrefaction, inflammable gases are ignited if a flame is nearby. These gases are hydrogen, hydrogen sulphide and methane. It is very rare and complete combustion does not occur.

Classification of Burns

Table 19.2: Differences between the burns caused by dry heat, moist heat and chemical

Features	Burns	Scalds	Chemical burns
Cause	Flame, heated solid	Steam or liquid	Corrosive chemicals
Site	At and above	At and below	At and below
Splashing	Absent	Present	Present
Skin	Dry, shriveled and charred	Sodden and bleached	May be destroyed
Vesicles	At the circumference of the burnt area	Over burnt area	Very rare
Red line	Present	Present	Absent
Color	Black	Bleached	Distinctive
Charring	Present	Absent	Present or absent
Singeing	Present	Absent	Absent
Ulceration	Absent	Absent	Present
Scar	Thick and contracted	Thick and less contracted	Thick and contracted
Clothes	Burnt	Wet not burnt	May be burnt, color on the clothes

The burns are classified based on two classifications:

I. **Dupuytren's classification:**

Dupuytren divided burns in to six degrees that were later on merged by **Wilson** in to three degrees.

- First degree and second degree—Wilson's epidermal.
- Third degree and fourth degree—Wilson's dermo-epidermal.
- Fifth degree and sixth degree—Wilson's deep.

II. **Heba classification:**

- Superficial—Less than full thickness burns.
- Deep—Full thickness burns.

Epidermal Burns

First degree: Erythema and redness is present that disappears in few minutes. Signs of superficial inflammation are present. Mild irritants can also produce the features of first degree burn. It may last for several days when cuticle peels off but leaves no scar. They disappear after death due to gravitation of blood to the dependent parts.

Second degree: This type of burns consists of acute inflammation and blister formation. These blisters are to be distinguished from blisters produced by strong vesicants like cantharides.

Blisters can also be due to decomposing fluid and urine and faeces when subjected to warmth in old bed ridden patients. In deeply comatose patient, bullous formation may be there. Blackening and singeing of the hair is present in second degree burns. On healing, no scar formation occurs but only some light staining of the skin remains.

Dermo-epidermal Burns

Third degree: There is destruction of cuticle and part of true skin that appears horny and dark. There is exposure of nerve endings and much pain is produced. Scar formation occurs but no contraction is seen as scar contains all the elements of a true skin.

Fourth degree: In fourth degree burns, whole skin is destroyed. Slough is yellowish brown and parchment like that separates on the 5th or 6th day. The burns are not very painful.

Deep Burns

Fifth degree: The penetration of the deep fascia and muscles occur and great scarring and deformity results.

Sixth degree: The bones and nearby organs are involved in sixth degree burns. These burns can still be compatible to life, if the initial shock is overcome.

Factors influencing the effects produced by the burns

1. *Degree of heat:* House fires at temperatures more than 650°C do not completely burn the adults. For cremation, incineration is done at 900-1000°C and only ashes about 1-1½ kg and bone fragments remain after incineration.
2. *Duration of exposure:* Duration of exposure influences the burns produced.
3. *Body surface area involved in the burns:* The estimation of the burn surface area in adult is calculated by the **Wallace rule of nine**. In children the burn surface area can be calculated by the **Lund and Browder chart**. If the burn surface area is more than 15% in an adult and 10% in a child, the loss of circulating blood volume must be replaced else hypovolemic shock may develop. Patients with superficial burns involving 50% of the body surface area can survive.

Wallace Rule of Nine

- 9% for head and neck
- 9% for each upper limb
- 9% for front of chest
- 9% for back of chest
- 9% for front of abdomen
- 9% for back of abdomen
- 9% for front of right lower limb
- 9% for front of left lower limb
- 9% for front of right lower limb
- 9% for back of left lower limb
- 1% for perineum.

In children, the burn surface area is calculated using Lund and Browder chart. (Table 19.3 and Fig. 19.7).

4. *Age:* The fatality caused by the burns depends upon the age of the victim; in children about 50-60% burns is fatal, in a person of about 40 years of age, about 40% is fatal whereas in old age only 10% can be fatal.

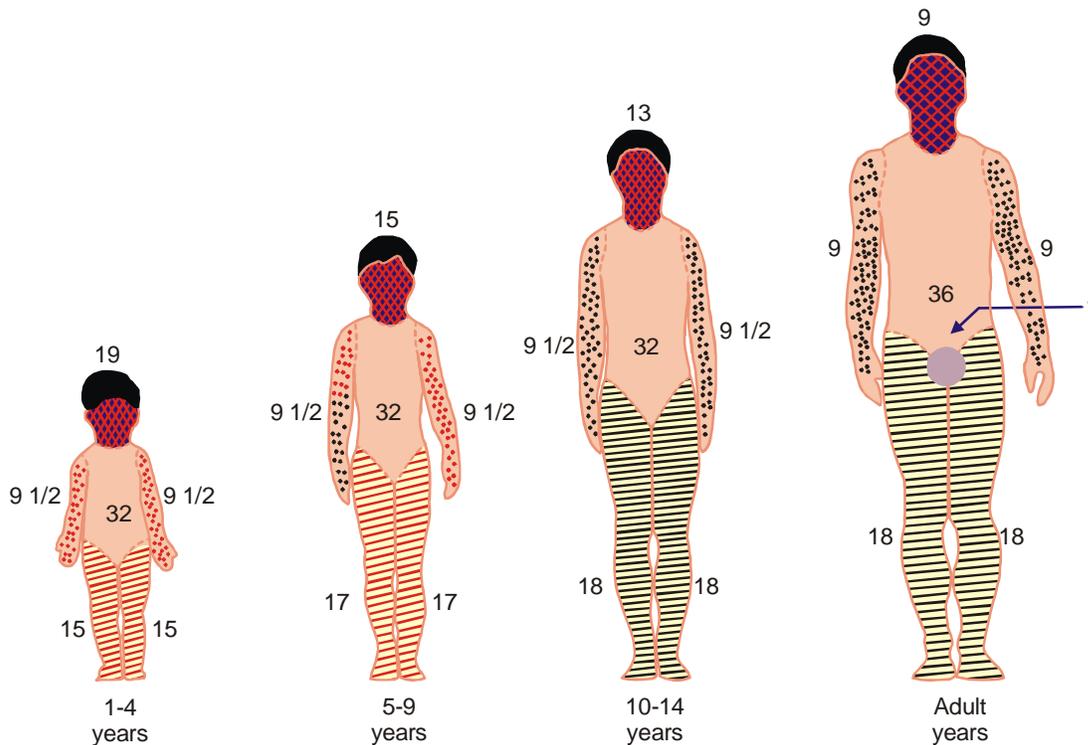


Figure 19.7: Estimation the body surface area in burns in children (Lund and Browder chart) and adults (Rule of nine)

Table 19.3: Estimation of body surface area of burns

Burnt area	Age group			
	1-4 years	5-9 years	10-14 years	Adults
Head and neck	19%	15%	13%	9%
Upper limbs	19%	19%	19%	18%
Lower limbs	15 × 2	17 × 2	18 × 2	18 × 2
Front of chest	8%	8%	8%	9%
Back and Chest	8%	8%	8%	9%
Front of abdomen	8%	8%	8%	9%
Back of abdomen	8%	8%	8%	9%

5. *Site of the body involved:* The burns are more dangerous when involving the face, chest or genitals.
6. *Age of the person:* Children and old people are more susceptible to the burns.
7. *Sex of the person:* Women are more susceptible than men (Fig.19.8).



Figure 19.8: Burn injuries over the face

Causes of Death in Burns

1. *Primary shock:* Due to the severe pain caused by burns.
2. *Secondary shock:* From hypovolemia due to fluid loss from the surface of burns within first 48 hours.
3. *Toxemia:* Within 3-4 days due to absorption of toxic metabolites from the burnt tissue.
4. *Septicemia:* Sepsis of infected burns results in death within 5-6 days (Fig. 19.9).
5. *Biochemical disturbances:* This also occurs due to loss of fluid and tissue destruction e.g. hypokalemia
6. *Acute renal failure:* This develops on 3-4th day due to necrosis of lower nephron.
7. *Glottis and pulmonary oedema:* This occurs in massive house fires or fires in buildings when the person dies at the spot itself. Oedema of glottis and lungs results from inhalation of CO and CO₂.
8. *Suffocation:* Inhalation of smoke containing carbon monoxide, cyanide gas, lack of oxygen, and free radicals and nonspecific toxic substances.
9. *Accidents:* These may occur while attempting to escape from a burning house or by injuries



Figure 19.9: Infected burns over the upper part of the body

resulting from falling masonry, timber or other objects over the body.

Determination of Cause of Death

To determine the cause of death one has to establish whether the victim was alive at the time of fire.

1 **Differences between antemortem and postmortem burns:** The differences are shown in Table 19.4.

- i. Presence of CO in the blood is suggestive that the person was alive at the time of burns. However, its absence does not exclude it. It is absent in flash fires such as explosions, chemical plant fires or warfare etc.
- ii. As a rule, flames that hit the face are liable to cause death rapidly due to oedema of airways and suffocation.
- iii. Toxic effects of inhalation of combustion products of some synthetic material like plastic furniture, upholstery cause rapid death.
- iv. Phosgene, chlorine, cyanides and nitrogen oxides aggravate the anoxic effects of carboxyhaemoglobin.
- v. Carbon monoxide is generated by the combustion of household material. Carbon monoxide level varies in a dead body depending upon age and health of the victim.
- vi. The fatal level is usually 50-70% carbon monoxide saturation but in the presence of

lung and cardiac diseases death occurs at a much lower level.

- vii. Infants and children build up the carbon monoxide level quickly due to increased respiratory rate. As a result, the children die before the parents when sleeping in same room.
 - viii. In a foetus, diffusion of CO across the placental barrier has conflicting opinion.
 - ix. Black soot particles get deposited over the mouth and nostrils. Soot frequently extends to the lungs and at times may be found in the esophagus. The soot can be spread as a thin film on a white paper and examined with the naked eye and then under the microscope. The trachea and other airways show congestion.
 - x. There is blister formation over the joints and any other parts of the body in second degree burns. These blisters are to be differentiated from those formed in CO and barbiturate poisoning, deep coma.
2. **Fat embolism:** Presence of fat droplets in blood vessels of the lungs must be interpreted with care, occurs due to fat embolism. This is to be differentiated from the similar finding in blunt force injury and physiochemical alterations of fat in the blood.
3. **Heat fractures:** Fractures of extremities due to shrinkage of muscles occurs. Skull fractures are seen on either side of skull above the

Table 19.4: Differences between antemortem and postmortem burns

Features	Antemortem burns	Postmortem burns
Line of redness redness	Present; absent if death is immediate Base is inflamed and swollen; contains protein and chloride; inflammatory reaction is present	Absent Base is dry and yellow; contains air and no protein or chloride but at times traces may be seen; inflammatory reaction is absent
Vital reaction	Positive	Negative
Enzymes	SH group increased, mucopolysaccharides are present. The following enzymes show an increase in the peripheral zone Nonspecific esterases—1hr Lucien amino-peptidase's—2hr Acid phosphates—3hr Alkaline phosphates—6hr	Mucopolysaccharides are absent. There is no increase in any of the enzyme groups

temples and are sometimes bilateral and no displacement of the outer table occurs. It consists of several lines radiating from a common center. It is commonly by bursting effects with brain tissue coming out due to steam pressure within the skull. The cracks are limited to the outer table. The skull fractures due to burns should be differentiated from the fractures due to blunt force.

Heat fractures do not involve the sutures of the skull even in young individuals with open sutures. The bone becomes brittle due to direct effect of heat. Blood and marrow are expressed from the skull bones by heat that accumulates between dura and the bone that clots and formation of heat hematoma occurs.

4. **Heat hematoma:** Heat hematoma is soft, friable clot that is pink in colour and shows honeycomb appearance. It is 2-15 mm thick and contains 100-120 ml of blood. There is charring of the skull. The distribution of heat hematoma is related to the charring of skull. The diploic veins and longitudinal sinus is involved. The brain is shrunken, firm and yellow to brown due to cooking and the dura is leathery. Heat hematoma should be differentiated from extradural hematoma. The extradural hematoma is found over the temporo-parietal region of due to rupture of middle meningeal artery. It is always traumatic in origin and does not have a cherry red colour or honey comb appearance.
5. **Subdural hematoma:** Subdural hematoma in burn victims develop due to the injuries and not by the effects of the flame. This clot should not be submitted for chemical analysis as positive alcohol level in subtotal clot indicates that the victim is habitual to alcohol because the peripheral blood alcohol may have metabolized but subtotal clot still contains it.
6. Due to the effects of heat, blood is pressed out of the lung tissue in to the airways, mouth and nostrils suggesting injury.
7. Splitting of the skin should be differentiated from antemortem cuts and slashes by the absence

of bleeding, intact nerves and vessels, absence of bruising and irregular margins.

8. **Curling ulcer:** Curling ulcer due to inflammation and ulceration of Payer's patches develops on the 7-10th day of the burns most probably due to thrombosis of the vessels. Anus is occasionally dilated due to shrinkage of peripheral tissue by heat that could be mistaken for sodomy or homosexuality. In putrefaction also due to formation of gases the abdominal pressure is increased and dilatation of anus occurs that may be mistaken for sodomy.

Postmortem Findings

The aim of the postmortem examination is to establish identification of the victim and cause of death, whether the person was alive at the time of incidence that is whether the burns are antemortem or postmortem, time since death, age of the burns, whether the burns are accidental, suicidal or homicidal are also to be established. The postmortem examination in deaths due to burns in cases of women is to be conducted by the two doctors when the period of marriage is less than seven years or the woman is less than thirty years of age. (vide Govt. of NCT)

Establishment of Identity

1. The identification of the victim is carried out according to the general outline but certain principles are peculiar to the victims of fire.
2. The length and weight of the charred body are unreliable as dimensions are altered due to desiccation of the tissues resulting in skull fractures and pulverization of the intervertebral discs. Body length may be shortened by several inches and weight is reduced to about 60%.
3. Body features are changed and skin is tightened and contracted. An ear is reduced to 2/3rd of its original size. Eyes show cataract formation due to coagulation of proteins in lens even at 150°F.

4. Identification marks on the body like scars, tattoos, moles etc may be destroyed. Removal of superficial layers of skin by wiping or rubbing makes tattoo better.
5. Degloving occurs due to deposition of fluid between the layers of skin that is used to obtain fingerprints. This is to be differentiated from deaths due to decomposition or immersion.
6. Surgical scars such as missing appendix and mastectomy can be diagnosed at autopsy.
7. Bones, pelvis and teeth can help in identifying the age, sex and race of the deceased.
8. *Sex of the victim*: It is identified from the presence of genitalia and breast tissue. Internally, sex can be identified from the organs such as prostate and uterus.
9. *Age of the victim*: Estimation of age from breast tissue is misleading as heat causes fluid loss that leads to shrinkage, erection and firmness regardless of the age of the person.
10. *Race of the victim*: The skin is brown or mottled in Negroes. There dark gray deposit of melanin in the arachnoid membrane in the medulla oblongata, base of frontal lobe, olfactory bulbs etc. is seen on microscopical examination.
11. *Complete X-ray of the body*: Is done to detect old fractures, bony abnormalities, foreign bodies etc.
12. *Dental examination*: X-ray of the teeth to identify metal pins in root canal treatment and fillings. Teeth examination is difficult when the teeth are fragmented or there is deposition of smoke and soot. When heat contractures have tightly locked the jaws; in such cases the teeth can be examined after disarticulating the mandible and excising the upper jaws.
13. Pathologist should look for the patches of intact skin under tight clothing, belts, brassiere or buttoned collar. On the ankle and wrist as the victim may be tied with strings or ropes before burning. Ligature



Figure 19.10: Body showing pugilistic attitude and extensive burns covering 100% of the body surface area

marks may be present on the neck. At times, skin and hair in the armpits are spared and gums are also preserved.

External findings

The buttocks and armpits are usually spared from burns.

Pugilistic attitude: Pugilistic attitude results due to the coagulation of muscle proteins and flexors muscles are more affected (Fig.19.10). Pugilistic attitude is also known as Boxer's attitude. It is not an antemortem phenomenon

Hair changes due to burns: Light hair changes at different temperature; gray hair at 250°C attains brassy blonde and brown hair at 400°F for 10-15 minutes attain slight reddish hue.

Heat ruptures: These are produced by splitting of soft parts at the joints. They may be confused with the incised or lacerated wounds. Can be differentiated from them by the absence of bleeding; presence of intact vessels and nerves; irregular margins and absence of bruising and other vital reactions.

Internal Findings

The internal findings such as heat hematoma can be recognized by the presence of soft, friable clot;

pink in colour due to the presence of Carboxy-haemoglobin and honeycomb appearance.

Age of the Burns

The question arises as to when the burns were caused and whether several burns were caused simultaneously. The following features help in finding out the time of burn:

- Redness—Immediate.
- Vesication—1-2 hours.
- Exudates begins to dry—12-24 hours.
- Dry brown crust formation—48-72 hours.
- Pus formation— 48-72 hours but not before 36 hours.
- Superficial slough separates—4th-6th day
- Deep slough separates—15 days.
- Granulation tissue begins to cover in more than 15 days.
- Formation of cicatrix and deformity takes several weeks or months, depending on the amount and extent of infection.

MedicoLegal Aspects

Suicidal Burns

Suicidal burns are very rare among men but common in women. The victim may commit suicide by burning due to disappointment in love, being tired of domestic worries or chronic diseases. At times, suicidal note is recovered and the room is locked from inside. Dowry deaths are caused using this method commonly that occurs in young brides who are unable to bring dowry or due to harassment by the in-laws leading to abetment to suicide. Self-immolation is also employed sometimes to protest like Mandal commission, *Sati* system.

Homicidal Burns

Homicidal burns occur in the following circumstances. Bride burning by the in-laws is common. The master and sometimes stepmother or stepfather applies hot *chimta* over the private parts, hands or thighs etc. of the servants and children. As a punishment for infidelity, cigarettes are

applied to produce burns that are be circular, or triangular in shape. The victim may be burned after he is murdered by strangulation or by any other manner to conceal the crime. Homicidal burns are also produced to torture a criminal to extort information or as a punishment for infidelity. In addition, burns, scalds or electrical injuries may be produced in child abuse cases. Acids or old bulbs may be thrown on the face out of jealousy or disgruntled behaviour. At 1000-1100°C, complete burns are produced but house fires can occur at 700°C causing charring of the body.

Accidental Burns

Accidental burns result while the person is drugged, diseased or drunk, the house is on fire, while sleeping in winter, cooking, during epileptic fits, or children may fall in to fire while playing. Scalds are usually accidental due to application of hot water bottles or when water from the boiling kettle falls on the victim. Lamps or stoves may explode and cause burns.

Self Inflicted Burns

Self-inflicted burns are caused for the false accusation.

Dowry Deaths

Dowry death is also known as bride burning. For burning a bride, fire accelerants such as kerosene and petrol can be used by the husband and in-laws.

A new prescribing punishment for dowry deaths (**S. 304B I.P.C**) has been recently enacted. A criminal charge may also be brought under other related sections of the Indian Penal Code, viz, **S. 498A I.P.C.** (causing cruelty to a married woman), and **S. 34 I.P.C** (more than one person acting with common intent).

Dowry Prohibition Act 1961 was amended in 1983, 1984 and 1986. According to Section 3 of Dowry Prohibition Act as amended in 1986, taking dowry is an offence and Section 4 implies demanding dowry is an offence.

Section 304 B I.P.C—Dowry death: Where death of the woman is caused by any burns or bodily injuries or occurs otherwise than under normal circumstances within 7 years of her marriage and it is shown that soon before her death she was subjected to cruelty or harassment by her husband or any relative of her husband for, or in connection with any demand for dowry, such death shall be called “dowry death”, and such husband or relative shall be deemed to have caused her death.

Section 498 A I.P.C—Husband or relative of husband of woman subjecting her to cruelty :Whoever, being the husband or the relative of the husband of a woman subjects such woman to cruelty shall be punished with imprisonment for a term which may extend to 3 years and shall also be liable to fine.

Section 113 B I.E.A—If cruelty or harassment before the death of woman is related to ‘dowry demand’, dowry death shall be presumed.

Section 176 Cr.P.C.—Magistrate inquiry shall be conducted in dowry death cases.

Postmortem will be conducted by the two doctors where the period marriage is within 7 years. Duty of the public is to inform the police who will take the necessary action.

Section 201 Cr.P.C—Causing disappearance of evidence of offence or giving false information to screen offender. In cases of Capital offence, punishment with imprisonment for 7 years or fine.

Section 202 Cr.P.C—Intentional omission to give information of offence by person bound to inform. Punishment with imprisonment for 6 months, fine, or both.

Section 203 Cr.P.C—Giving false information respecting an offence committed. Punishment with imprisonment for 2 years or with fine or with both.

LIGHTNING STROKE

Lightning stroke is a flash of lightning due to an electrical discharge from the cloud to the earth. The electric current is direct with a potential of 1000 million volts or more. At the track of current much energy is liberated most of which is

converted in to light. The highest points attract the lightning stroke. Dry skin and dry clothes are bad conductors whereas wet clothes and wet skin are good conductors.

Signs and Symptoms

1. Unconsciousness.
2. In non fatal cases, giddiness, ringing in the ears and headache develops.
3. Conjunctivitis, clouding of cornea, cataract and retinal haemorrhage.
4. Loss of memory.
5. Paralysis and titanic convulsions.
6. Deafness or dumbness etc.

Postmortem findings: The following effects are produced

1. Direct effect from electrical discharge passing to the earth surface produces ‘**flash burns**’, mechanical effect due to force of displaced air and compression effect due to air movement in its return wave produces the changes.
2. Clothes are usually torn or burnt at the point of entry or exit. At times, clothes are thrown at a distance. There can be exceptions when clothing is not damaged even if the person has been killed. Conversely clothing may be damaged without any damage to the person.
3. Blast like lesions are produced comprising of a triad of contusions, lacerations and rupture of organs.

Burns Due to Lightning

These are linear burns 3-30 cm or more in length and 0.3-2.5 cm in breadth involving the moist creases and folds of the skin called **filigree burns**. The **arborescent markings** (Fig. 19.11) are superficial, thin, irregular and tortuous markings in the skin like a tree due to minute deposits of copper in the skin. They indicate the path taken by discharge and are due to breakdown of RBC within the capillaries of the skin and escape of haemoglobin. They disappear in 1-2 days if person survives.



Figure 19.11: Filligree burns in lightning

The surface burns in case of lightning are true burns and occur beneath the metallic objects worn or carried by the person that are fused by the flash. Internal findings are not very characteristic and the cause of death is due to paralysis of heart and respiratory center.

MedicoLegal Importance

1. Death is accidental.
2. The appearance left on the body by lightning

stroke sometimes closely resembles those produced by criminal violence.

3. When the body is found in an open field, history is characteristic and there is fusion or magnetization of the metallic substance.

Long Questions

1. Discuss burn injuries caused by a hot liquids. Enumerate the differences between the various types of injuries caused by flames and hot liquids.
2. Define and classify burns. Discuss the various medicolegal issues in deaths due to burns.
3. Write in detail the various legal issues in cases of burns.

Short Questions

1. Hypothermia.
2. Hyperthermia.
3. Filigree burns.
4. Joule burns.
5. Rule of nine.
6. Pugilistic attitude.
7. Differences between antemortem and postmortem burns.
8. Heat fractures.

MULTIPLE CHOICE QUESTIONS

1. **Pugilistic attitude is seen in cases of:**
 - A. Hanging
 - B. Lightning stroke
 - C. Heat stroke
 - D. Burns
2. **Arborescent marks seen in:**
 - A. Firearm wounds
 - B. Burns
 - C. Lightning injury
 - D. Head injury
3. **Heat hematoma to be differentiated from:**
 - A. Extradural
 - B. Subdural
 - C. Subarachnoid
 - D. None of the above
4. **Frost bite occurs when the body is exposed to a temperature of:**
 - A. -10°C
 - B. -5°C
 - C. -2.5°C
 - D. $+4^{\circ}\text{C}$

1 D 2 C 3 A 4 C

5. **Vesicles in an antemortem burn are differentiated from a postmortem burns by the presence of :**
 A. Chloride and albuminous fluid
 B. Chloride
 C. Plasma and WBC in fluid
 D. Air
6. **Heat haematoma is present within:**
 A. Skull bone and galea aponeurotica
 B. Duramater and arachnoid mater
 C. Below piamater
 D. Duramater and skull bone
7. **Antemortem burns differ from postmortem burns by:**
 A. Charring
 B. Cyanosis
 C. Scab has plasma and albumin
 D. Carboxyhaemoglobin
8. **Which of the following is not true about fracture in burn?**
 A. Seen in the skull
 B. There is wide separation of sutures
 C. Usually located above the temples
 D. Intracranial haematoma is not closely related to fracture
9. **A burnt body of driver is found near a burnt car, which of the following suggests that the person was alive at the time of burning:**
 A. Black soot in upper airway
 B. Paltauf's haemorrhages
 C. Blisters on skin are present
 D. Dermis contains abundant fat globules
10. **A bum is antemortem if:**
 A. Blisters are present
 B. Blisters are absent
 C. Line of redness present
 D. Dull white appearance of skin
11. **All are true about pugilistic attitude except:**
 A. Occurs due to coagulation of proteins
 B. Seen only in antemortem burns
 C. Seen both in antemortem and postmortem burns
 D. Also known as Boxer's attitude
12. **Antemortem burns differ from postmortem burns by:**
 A. Vital reaction
 B. Presence of fluid
 C. Presence of chloride in blister
 D. Presence of cyanmethaemoglobin
13. **'Pugilistic attitude' is found in death due to:**
 A. Asphyxia
 B. Burns
 C. CO poisoning
 D. Cardiac failure
14. **'Filigree burns' are caused by:**
 A. Electrocution
 B. lightning injury
 C. Chemical burns
 D. Fire accidents
15. **'Joule bum' is produced by:**
 A. Electricity
 B. Lightning
 C. Gas stove
 D. Burning matches
16. **Pugilistic attitude is due to**
 A. Coagulation of proteins
 B. Cadaveric spasm
 C. Rigor mortis
 D. Coagulation of fats
17. **Arborescent marks seen in**
 A. Firearm wounds
 B. Burns
 C. Lightning injury
 D. Head injury
18. **In a Postmortem burn:**
 A. Line of redness is present
 B. Blisters contain serous fluid
 C. In superficial zone of burnt area there is no acid mucopolysaccharide
 D. The peripheral zone shows an increase in enzyme reaction.

Mass Disaster Injuries

Mass disaster is commonly defined as the death of more than 12 victims in a single event. It is the number of deaths that exceeds the capacity of the local death investigation system to handle it. In certain parts of the world, nature is frequently responsible for mass disaster with flood, hurricanes, avalanches or earthquakes killing hundred to thousand to more of the people. In these nearly enormous disasters, when whole communities are wiped out, there can be very little detailed investigations. The task would be far too great and the most that could be attempted would be identification of some of the dead bodies. Examination of the bodies of the victims by pathologists is necessary for two main reasons:

1. To identify each dead body and to establish the cause of death for legal purposes so that a death certificate can be issued.
2. To discover evidence relating to the investigations of the disaster itself like obtaining sample for toxicological analysis where appropriate
3. To find out the cause of the disaster itself e.g. bomb or detonator fragments that may be embedded in the bodies of the victims

In many disasters, the extent of damage to bodies and their clothing by traumatic forces or by fire is such that specialized examination of the bodily remains is necessary to establish the identity. It is therefore considered highly desirable that from the outset very close liaison could be established between the police and the pathologist, to carry out postmortem examination.

Scandinavian practice provides for the appointment by a Prefect of Police of an identification commission including a Policeman, a doctor usually a pathologist) and a dentist, whenever the need arises by reason of mass disaster or crime. This commission collects evidence of identity of the bodies recovered, each member working in his own field of competence and jointly it reviews all the evidence and determines whether or not in each the evidence warrants a particular identity being assigned to a particular body. In India, no such practice is followed and collection of evidence at the site as well as shifting the bodies to the morgue is the role of the police officials. The cooperation of pathologist and the police can be conveniently considered in three phases.

First Phase (At the Accident Site)

Priorities in the initial stages of the post-disaster situation will generally be self-evident. There may be survivor to be rescued; all possible means will be used to save life without unduly endangering rescuers from the various continuing dangers that may exist e.g., fire. When no further lives can be saved, situation should be taken stock of.

Evidence should be preserved that may contribute both to their identification and to the investigation of the accident itself in the widest sense. The first task should be to:

1. Locate bodies.
2. Label them with a number.
3. Photographing them *in situ*

4. Preparing a plan of the disaster site: A map of the site showing the main pieces of wreckage and position of the bodies is clearly more appropriate in the content of an aircraft accident than, say a hotel fire, for in the latter a written record of which numbered bodies were found in which rooms might be considered sufficient.
5. The exact location of a body at a disaster site may prove to be very important, whatever the disaster.
6. Work of identification of the bodies.
7. For accident reconstruction from medical evidence, bodies are labeled, photographed and a record made of their precise position.
8. Record each body and place it in a suitable container for transfer to the mortuary. Make sure that things belonging to one body are not assigned to another. Polythene sheeting can be used for covering it, It should be semi-transparent to see the numbered label attached to a body without need for further labeling to facilitate carrying.
9. Investigations of mass disaster is expensive, therefore there is a strong case for planning and executing disaster investigation in liberal fashion with sufficient manpower, but properly supervised for search and rescue procedures, with the quickest possible transportation to the scene for the specialist investigators and with the provision of a sufficient quantity of efficient modern equipment.

Second Phase (In the Mortuary)

In the mortuary accommodation and in particular refrigeration are likely to be problems following any major disaster. Bodies should be dispersed to numerous small mortuaries but preferably a building of suitable size to be designated a temporary mortuary, for a coordinated investigation, where there is sufficient space and light.

In certain types of mass disaster, where identification is not difficult and evidence in the bodies cannot contribute in anyway to a reconstruction or explanation of the accident, only external examination may be required.

When as is the rule in aircraft accidents, a full autopsy is required from the point of view of accident investigation itself, then better facilities such as a properly equipped autopsy room becomes essential.

It is ideal to have in close proximity an autopsy room, refrigerated mortuary accommodation, an area for embalming and casketing room for inter-viewing relatives and viewing bodies, if necessary and a suitable room for use as a communication headquarters. The ideal is likely to be most closely approached when there has been pre-planning by the appropriate local authorities (in particular the police) before a disaster situation of medium to considerable magnitude occurs, with decisions having been taken in advance about what accommodation could be brought in to use in the event of a disaster of particular size.

The procedures in the mortuary should be a matter for team work involving at least a pathologist and a police officer, usually a forensic odontologist and occasionally other specialists such as a radiologist, when appropriate to the particular circumstances and conditions of the bodies.

It is always important to count the number of bodies recovered. This is easy enough when there has been little or no mutilation and fragmentation, but when the traumatic forces of the accident have been severe, the total number of items of human remains recovered may far exceed the number of persons killed.

1. *Primary identification*: A term that could apply either to the first clue to identify discovered in a given instance, whether it be strong or insubstantial evidence, or to the most valuable clue, judged in retrospect, when all evidence has been adduced.

The categorization of pieces of evidence as primary or secondary is probably of little practical consequence that is to seek routinely from the outset to establish identity by all or several of the different means and not to rest content unless identity has been established and confirmed by at least two different means if at all possible.

- i. *Visual identification*: It is the standard method used by police to establish the identity of a dead body. Visual identification of facial features can be reliable when a body is intact or little damaged externally; the effect of burns in a disaster reduces its value considerably.
 - ii. Photographs of distinctive clothing, personal possessions or physical characteristics may be recognized if facial features are damaged. Initial and subsequent photography is essential to make a photographic record made to relate, body, site and cadaver numbers. After initial photography clothing and jewelry must be removed from the body, examined and catalogued. This is primary for identification purposes.
 - iii. *Examination of clothing*: Evidence of vomit, blood or stains upon clothing may prove useful. Any marks on clothing like manufacturer's label or laundry marks also prove valuable. Examination of clothing is necessary; find out any damage to clothing due to the accident sequence or bomb explosion.
3. *Careful external examination* to determine:
(i) Sex (ii) Height (iii) Weight (iv) Hair colour (v) Location and abundance of hair (vi) Colour of eyes/ skin (vii) Any anatomical, surgical and traumatic scars. (viii) Tattoos or birth marks. (ix) Abnormalities such as congenital or acquired.
 4. *Injury sustained* during the accident related to cause of death.
 5. In case of blast injuries, trace evidences may be preserved
 6. *Radiographic examination*:
 - Ideally, complete set of skeletal x-rays should be taken for each victim.
 - In case of suspicion of sabotage, full skeletal radiography is required.
 - Radiographs are helpful in identification in case of extremely burnt body.
 - They are also helpful in revealing the presence of articles embedded deeply in charred muscle, may be overlooked otherwise.
 - A permanent record of bone injuries are obtained that reduces the time spent in assessing and recording these during autopsy.
 7. *Full internal autopsy*: Evidence relevant to identification will be collected such as:
 - Surgical absence of internal organs.
 - Presence of post-surgical states like gastroenterostomy.
 - Evidence of pre-existing disease.
 - Any internal injury.
 - Precise cause of death.
 8. *Histological examination*: During autopsy, specimens of certain organs and tissue will be collected for routine histological examination in 10% formal saline and 1% sodium fluoride for fluids like blood or urine. Tissue specimens containing metal fragments for chemical analysis should be deep-frozen and for histological examination preserved in 10% formalin. Histological examination may reveal a disease relevant to consideration of impaired function if the body is that of operating crew or expectation of life, should medicolegal problems for compensation arise.
 9. *Fingerprints*: They should be taken after the autopsy when all possible evidence has been collected from examination of the body. Fingerprints are of limited value in identification of victims of aircraft accidents due to nationality of most of passengers being in question.
 10. *Dental examination*: At the end of the autopsy, dentist should make an examination of the jaws and teeth; value of dental evidence in identification has become widely

recognized after the work of Scandinavian forensic odontologist such as Strom, Keiser Nielson and Gustafson.

- A positive identification can be made when there are a number of points of similarity as between teeth missing and points of matching in restorations by a particular tooth and surface and when there are no incompatible inconsistencies.
- The minimum number of points of similarity (7 or 8) are required for a confident identification, but each case must be judged on its merits by forensic odontologist.
- A tooth present in a cadaver recorded as extracted in a known person, or an intact tooth in the body recorded as having been filled in the known person's chart, is an incompatible inconsistency; it can only be disregarded in the face of overwhelming forceful evidence that the inconsistency is due to miscasting of some other artifact perhaps in the telegraphed transmission of the dental notes or chart.
- In the context of mass disaster in which traumatic forces are severe the value of the dental charts is reduced and the value of radiographs is enhanced.
- It is quite common for a body to have retained but one or two fragments of jaw, perhaps with one or two teeth. These teeth may have a restoration or two, but they may be commonly filled teeth and a chart may be quite inadequate in its detail of the size of the restorations for the evidence to be strong enough to form the basis of identification. If an antemortem radiograph of the relevant part of the dentition is available, however it may prove to be of greatest possible value for comparison with the postmortem radiograph of the fragments recovered.

- The above references to dental evidence as a means of identification have been primarily in connection with the comparative approach of this work, this is most appropriate in the field of aircraft accidents when the names of all the persons involved in the accident are nearly always known with a considerable degree of certainty.
- When a completely unknown body is found, or when mass disaster involves quite unknown persons, is usually the case in a department store fire or railway disaster, the reconstructive value of dental evidence is more often utilized. The teeth may reveal much about its owner, his/her country of origin, perhaps from a characteristic restorative work, his/her occupation such as notching of upper incisors may occur in hair dressers, for example and the teeth may be a good guide to age.

One of the main problems in the field of international forensic odontology has been the lack of a common dental nomenclature.

Freehold and Lysol reviewed the various systems used by dentists in various countries in 1962. These authors emphasized the possibilities of combustion after a mass disaster involving people of many nationalities, when dental information is received in telegraphed form.

At the international dental Federation meeting at Bucharest in 1970, new **two-digit system** was proposed and adopted for recommendation for international use.

11. Other means of identification such as: (i) Multivariate analysis of race in cranium (Stewart 1970) and cephalic index (ii) Identification of scars of parturition and other pubic symphyseal changes in skeletal remains of female (iii) Neutron activation analysis of hair are also used.

Last task to be performed in the mortuary is the examination of any fragments of bodies, with, so far as possible, matching of those fragments with the bodies from which they originated.

Not only many clues to identity be found in separated fragmented remains, but occasionally important evidence about the cause of the accident can be ascertained.

Third Phase (Comparison of Records and Identification)

Identification of bodies depends upon accurate information about those who are believed to have been involved. When the persons killed are of the country in which the disaster occurs, it is probable that normal police communication system is the best for collection of required information. During recent years, the need for practical record forms in the context of a mass disaster has come to be appreciated, Australian Police, Interpol has produced a disaster victim identification form.

The forms, which have evolved from 15 years of practical experience by those pathologists who have worked in the Royal Air Force Aviation Pathology, Department of Haltom consists of four different coloured forms and a white one. Two of the coloured forms are for the males, other two for the females; white form is for missing persons. One of the coloured forms is for external feature of the body and the other is for autopsy findings. These are kept in transparent envelop upon completion and since they are coloured, on single sheets of paper, the subsequent exercise of matching records and 'proving', the identification of bodies is much more simplified. In India no such

forms are available and all the information related to identification should be recorded in a post-mortem report.

Review of Evidence and Accident Reconstruction

In some disaster the pathological examination of victims of bodies, can achieve beyond proving evidence leading to identification and confirmation of an obvious cause of death. Comprehensive pathological investigation can help a great deal to reconstruct the accident, sometimes to the extent of revealing the cause, though more often helping to encode possible causes. Sometimes a serious disease is revealed as a likely cause of an accident.

Routine toxicological tests on the tissues of human bodies also reveal a cause of death (like CO poisoning in disaster due to navigational error). Injuries in passengers of and their correlation with damage to seat structures and other solid structures is also helpful in knowing the relative safety of different parts of aircrafts/ other vehicles and how to improve them to prevent such disasters.

Long Question

1. How will you investigate a case of mass disaster? What measures will you undertake for proper identification of persons involved.

Short Questions

1. Preservation of trace evidences in mass disaster.
2. Methods of identification in burnt bodies.
3. Autopsy procedures in mass disaster.

Trauma and Disease

The relationship between trauma, work stress and disease assumes importance mainly for two reasons:

1. *Compensation:* Under the Workman's compensation Act, provision is made for disabilities suffered as a result of occupationally acquired diseases or industrial accidents while at work, provided the worker himself was not negligent or responsible for the injury. In cases of death without any medical attention and when the possibility of disease seems remote, a question arises whether the ill effects of work conditions or previous injury suffered while at work could be responsible for death.
2. *Insurance:* A person who has insured himself for accident only. Some life insurance policies include a double indemnity policy; the sum payable is doubled if death is due to accident.

When these matters become subject of investigation, a medical officer is called upon to examine the person:

1. To assess disability or degree of physical damage: A very thorough examination is essential; if necessary the worker may be kept under observation to assess degree of disablement either temporary or permanent. Most important aspect is evaluation of whether the disabilities claimed and observed are consistent with the injury received. Malingering means a deliberate attempt on the part of the patient to deceive the doctor.

The person may malingering and make false claims or he may have developed neurosis after the accident.

2. To perform an autopsy to interpret the relationship of disease or trauma to death. The medical officer should analyze the data carefully and opine on the relative role of trauma and disease to death. Just because a given episode of stress, exertion or trauma was antecedent, the assumption of cause and effect relationship is not necessarily warranted.

The association of trauma and preexisting disease is most often met in fatal cases, where an injury or alleged injury has been sustained by a person with substantial natural disease. The problem then is to evaluate whether:

1. Death was due entirely to injury and would have occurred whether or not the disease was present.
2. Whether the death was due entirely to disease and would have occurred at that time irrespective of the injury.
3. Whether the two processes in combination has caused death.

Trauma and Infection

Bacteria being the normal inhabitants of external environment gain entry to the injured tissue and set up local infection. The bacteria may also be present on the object producing injury.

Wounds associated with extensive crushing of tissue are more likely to be infected. Penetrating

wounds involving deeper tissues are more susceptible to contamination than superficial wounds. Pathogenic microorganisms are commonly present in the blood of normal individual (benign bactericidal). Usually these bacteria are quickly disposed off by the host defenses and do not elicit any signs or symptoms of disease. Concurrently to the stress of trauma and lowered resistance, these transient bacteria can easily migrate in to the wound, setting up localized infection. Since the time elapsing between the contamination of the wound and the reaction of the host is variable the question may arise (i) Whether the infection occurred in coincidence with the original trauma (**primary infection**) (ii) Whether the infection occurred subsequently to original trauma as a result of accidental implant of bacteria e.g. following application of contaminated dressings (**secondary infection**).

The concept of aggravation applies well to the situation in which trauma in association with accident or assault affects a preexisting infection, disturbing the local defense mechanism and thus transforming a relatively innocuous process into a life threatening situation e.g. dislocation of an infected teeth resulting from blow to the face or to the abdomen leading to the development of peritonitis from perforation of gastric or duodenal ulcer.

Trauma and Heart Disease

Injury to any part of the body may predispose to myocardial infarction with or without coronary thrombosis, if the injury is sufficiently severe to cause hypovolaemic shock and coronary arteries are previously narrowed by atherosclerosis. In such a case, the injury causes circulatory failure and myocardial infarction occurs during the period of shock.

Diagnosis is to be confirmed by clinical and laboratory tests in the living and appropriate findings at autopsy in the dead.

If the disease is present months before trauma, only a direct blow to the chest will damage heart and dislodge an athermanous plaque leading to

subliminal haemorrhage proving that the trauma has caused death from myocardial dysfunction. Actual death may not occur soon after trauma because period of hypertension will precipitate MI and death. Even threatening the person can cause cardiovascular changes, sudden rise in blood pressure that can rupture the athermanous plaque.

Two basic situations are most likely to find access to the Courts of Law:

1. *Direct injury*: When the heart has been perforated or lacerated by a penetrating object (flying missile or sharp object) or has been bruised or crushed by a violent impact against the chest, the ensuing death or disability can reasonably be attributed to injury.

Contusion of heart is a common complication of steering wheel injuries in car collisions. As a general rule, the cause and effect relationship is substantiated by the absence of a symptom free period, and if the victim dies, unmistakable evidence of cardiac damage is found at autopsy.

Blunt trauma to chest can cause a broken rib to bruise the myocardium or one of the coronary arteries, causing a blood clot (thrombus) to form in the lumen of the damaged blood vessel. In this situation, the casual relationship between the injury and the ensuing heart failure may remain a matter of speculation unless the sequence of events can be proved at postmortem examination.

2. *Indirect heart injury*: It is more difficult to prove as after injury to heart, ECG changes occur due to arrhythmias, over activity, increased sympathetic tone or excessive release of catecholamine occurs.

A severe injury to any part of the body is usually associated with loss of blood and destruction of the tissue with resulting low blood pressure, decreased blood volume and slowing of blood flow that results in shock leading to clotting of blood (thrombus formation) that may result in thrombosis of coronary artery and myocardial ischaemia and death.

Mechanical injury causes increased coagulability of circulatory blood that results in coronary artery thrombosis, cardiac instability and death.

A casual relationship between the trauma and post traumatic increase in blood coagulability and resulting coronary thrombosis can be accepted if laboratory evidence can be presented that following trauma, blood coagulability was greater than normal and that the manifestations of coronary thrombosis developed a few hours or at the most a day or two after the injury.

More difficult to assess is heart failure or any other cardiac disorder occurring in an individual with antecedent history of heart disease as an indirect effect of trauma. Anxiety, pain, anger or fear associated with severe injury impose an extra load on heart leading to heart failure.

Whenever the coronary circulation is impaired the extra work imposed on the heart by condition of anxiety may contribute to an angina attack as well to a host of acute changes in heart rhythm such as paroxysmal tachycardia, arrhythmias, flutter and fibrillation. These are regarded as evidence of vasomotor instability or autonomic imbalance.

Any attempt to recognize a casual relationship between the aggravation of the cardiac disorder and the trauma includes the following criteria:

1. The trauma must be of some significance: This is indicated by the disappointing but realistic observation that death does not always find a suitable explanation in detectable lesions at autopsy. This occurrence is particularly well documented in the case of blunt head trauma.
2. The symptoms must develop shortly after the event that precipitated the episode of cardiac distress.

The weakness of the concept in this case rests in the fact that some time may elapse between the establishment of the disorder and the ability of the physician to demonstrate the existence by clinical and laboratory methods.

Since heart failure occurs in the natural course of events of any cardiac condition and this may be sudden and unexpected, not infrequently the situation arises that the critical event occurs while the patient is walking or driving a vehicle.

Injury concurrent to heart failure raises the question whether the fall and the ensuing injury precipitated the cardiac condition or whether the sudden failing of the heart was responsible for the loss of consciousness, the fall and the injury. As a general rule; (i) If the injury was sufficiently severe to be considered incompatible with life and it can be shown to occur before death, the death can be attributed to the injury. (ii) Conversely, if the injury was of minor nature, and the heart condition at autopsy provides an adequate explanation at death, the death can be attributed to the cardiac condition. Following injury bacteria may gain entry to blood stream, lodge and grow in heart valves and give rise to acute bacterial endocarditis.

Occupational Trauma

As defined by effort at work, occupational trauma can precipitate death by giving rise to circulatory overloading or by aggravating previously existing disease such as coronary atherosclerosis, or aortic aneurysm. Narrowing is sufficient to explain death and a clot or acute obstruction is not necessary. Death from partial coronary stenosis is commonly precipitated by some exertion resulting in a greater demand of blood, the pumping action of the heart and oxygen demand of the muscle of the left ventricle. Climbing stairs, working a hand pump, and lifting a weight may precipitate such deaths.

If the deceased at the time of such deaths was engaged in some such work as a part of his employment, then a claim of compensation may succeed. Cardiac conditions which predispose to acute heart failure following physical exertion or excitement and which frequently result in sudden death include syphilitic arteritis with or without aortic valvular insufficiency and hypertensive heart disease usually with coronary atherosclerosis.

Trauma and Post Traumatic Psychosis

In the absence of acute brain damage, nearly all post traumatic mental disturbances represent both an aggravation of preexisting latent psychosis, primarily dementia praecox (schizophrenia) and MDP. In a general sense, injury comes to play the role of a precipitating factor.

It is generally accepted that the relation between injury and true post-traumatic psychosis rests on the demonstration that:

1. The patient had been a mentally well adjusted person prior to injury
2. The injury has produced organic damage to CNS
3. The injury regardless of the location was of such magnitude as to reasonably threaten the very existence of the victim of the injury
4. The injury was of a nature to affect the structure or function of certain parts of the body to which emotional importance is attached such as genital organs, breasts, eyes, face and hands etc
5. The victim was obsessed about the outcome from the time the injury was sustained.
6. The psychosis developed within a reasonable time after the occurrence of the injury
7. The psychotic thoughts had a positive relationship to the assault or accident that was responsible for the injury.

Trauma and Nervous System

It is difficult to refute completely the direct cause and effect relationship between: (i) Head injury and meningitis (ii) Head injury and epilepsy (iii) Head injury and psychosis (iv) Head injury including occupational stress and rupture of a congenital cerebral aneurysm.

Intracerebral haemorrhage can occur due to violence alone without any evidence of disease or blood vessels. Head injury can cause death due to subarachnoid haemorrhage due to rupture of Berry aneurysm in the Circle of Willis that can rupture spontaneously also. A ruptured aneurysm

can occur after a fight or assault also. Very small aneurysm may be destroyed by bleeding, which may not be recognized at autopsy. Following trauma, there may be absence of rupture of Berry aneurysm due to rupture of vertebral arteries in the transverse process of atlas vertebra by the kick or blow over the side of the neck.

To attribute trauma as a cause of death for disease or disability, following criteria should be satisfied:

1. The disease must not have been present before the injury but developed after injury was sustained e.g. meningitis following violence to the head with or without physical damage.
2. The disease is such as to be compatible with the nature of trauma ascribed to it e.g. meningitis following violence to the head with or without physical damage.
3. The length of interval (latent period) that elapsed between injury and development of disease is compatible, e.g. tetanus developing 3-10 days after injury; sometimes 2-3 weeks after the injury.
4. The disease does not develop spontaneously e.g. tetanus

Trauma and Pulmonary Embolism

It is almost always due to impaction of thrombus, which has been shifted from thrombosis of leg veins, pelvic thrombosis and post puerperal embolus. It is a well known complication of trauma. When leg is the site of injury, it occurs more commonly and takes two weeks after the injury.

Main aim of the doctor is to tell that initiation of leg vein thrombus occurred after the injury. Histological dating to correlate with injury can be done. There can be pulmonary infection leading to death after injury. Pulmonary infection occurs within the first few weeks after injury. During intervening period when the patient is immobilized and respiratory distress occurs, a connection is established.

Trauma and Alimentary System

Whenever a history is obtained of blow to the abdomen, the patient should be kept under observation until all likelihood of a ruptured abdominal viscous can be dismissed. A blow to the epigastric region can cause death by inhibition of heart through reflex action on the celiac plexus without leaving a mark externally or to the viscera. The result of the trauma to bowel may be very serious. Perforation of peptic ulcer leads to rapid death; this gives rise to compensation claims when death occurs due to some abdominal injury at work. Perforation of lower small intestine and large intestine are usually not followed by intense shock similar to the shock of peptic ulcer perforation and may not cause death if timely surgical aid is available.

The liver is easily lacerated and ruptured especially if it is fatty, congested, enlarged or diseased. However, death may not occur immediately. Sometimes bleeding occurs between the liver and its capsule that is the subcapsular hematoma. Serious symptoms become apparent only when the capsule ruptures. The damage is often fatal due to bleeding from the organ into peritoneal cavity. A kick or punch on the upper abdomen may injure the duodenum and pancreas and cause death within a few days from resultant chemical peritonitis. External injury to the abdominal wall may not always be visible in such cases.

Trauma and Malignancy

Trauma may occasionally cause cancer. The occurrence of estrogenic sarcoma and development malignancy in a scar tissue after injury has been documented. Cancer on rare occasions develop at the site of long standing unhealed wound of the skin or in the scarred skin provided the trauma that was responsible for scar formation was definitely something more than a single mechanical injury e.g. severe burns, X-rays burns and chemically inflamed lesions like varicose ulcer and chronic sinus tracts e.g. draining of a chronic osteomyelitis sinus, which may cause skin cancer

adjacent to the site of continued irritation. In accepting trauma as a cause of cancer, the following criteria based on **Ewing's postulates** must be satisfied:

1. The tumor site prior to injury was normal.
2. The injury was sufficiently severity to disrupt continuity of tissues at the site of injury and so initiate a reparative proliferation of cells.
3. The tumor followed injury within a reasonable length of time.
4. The tumor is of same histological type that might reasonably develop as a result of regeneration and repair of specific tissues that received the injury.
5. The exact nature of the tumor can be established by clinical, X-ray and microscopic examination.
6. The tumor must have originated in the part of the body that has sustained the injury.
7. It must develop within a certain period of time between a minimum of 3-4 weeks and maximum of 3 years after receipt of injury. Some investigators have increased upper limit to 10-20 years.

No organ in the human body is there in which the development of a cancer has not been attributed to trauma. Most frequently implicated are breasts, testes and bones.

Occupational Cancer

Exposure to arsenic, asbestos, chrome, nickel and other metals, aromatic amines, azo-compounds and an endless list of other organic substances for which the ability to produce cancer is substantiated by controlled experiments. Industrial or occupational cancer rests on the concept that exposure to chronic irritants may provide in the targeted tissues a background that is favorable to the development of a tumor. Mechanical trauma can provoke such a situation but among the irritants, it represents a statistically minor factor far surpassed by the chemical, physical and biologic agents that crowd the environment.

Medicolegal Aspects of Injuries

Injury (S. 44 I.P.C.): Any harm whatever illegally caused to a person in body, mind, reputation or property.'

Hurt (S. 319 I.P.C.): Bodily pain, disease or infirmity caused to any person. In English Law, hurt is defined same as battery. Infirmity is the disease or weakness due to administration of any poison or even alcohol.

Assault (S. 351 I.P.C.): An attempt, offer or threat to apply force to the body of another in a hostile manner. It is basically an attempt to offer with force and violence, a corporal hurt to another.

Battery: Battery is an assault brought to execution. It is more than an attempt to do a corporal hurt to another. It is an actual injury to a person. Every battery includes the assault, the distinction is only of the degree.

Simple Injury

Simple injury is defined as which is neither extensive nor serious and heals without leaving a permanent scar.

Grievous Hurt (S. 320 I.P.C.)

1. Emasculation: It means loss of masculine power and the term is only applicable to males. The loss of power can be due to direct trauma to genitalia leading to amputation of the organ. However, even if one testis with intact male organ is there, it cannot be called emasculation. Trauma to lumbosacral region leads to loss of masculine power indirectly.

2. Permanent privation of sight of either eye: Causing the permanent loss or impairment of vision in either eye is considered as grievous hurt. Examples include corneal scarring, retinal detachment etc.

3. Permanent privation of hearing of either ear: Causing loss or impairment of hearing of either ear is considered as grievous hurt. Rupture of the tympanic membrane due to a blow to the head will be considered as grievous even if there is partial hearing loss.

4. Privation of any member of any joint: Causing loss of function or anatomical separation of any body part is considered grievous hurt. A **member** means any organ or tissue capable of performing a distinct function in the body.

5. Destruction or permanent impairing of power of any member or joint: If any joint is destroyed so as to cause loss of its function or there is damage to any other organ or part of the organ, causing loss of its function is grievous hurt. The damage should be permanent.

6. Permanent disfiguration of head or face: Permanent disfiguration of face or head region is considered as grievous hurt. This depends on the nature of impact of the injury on the person involved. Even a small scar on a young girl may be considered as grievous while a large scar or even cutting the nose and ear on an old aged man may not be considered as grievous hurt.

- 7. Fracture dislocation of a bone or tooth:** Even dislocation of a tooth by a blow is taken as grievous hurt. All fractures including hairline fracture and even of the outer table of skull is sufficient for the purpose of law to be labelled as grievous.
- 8. Any hurt which endangers life, which causes its sufferer severe bodily pain or makes him unable to follow his ordinary pursuits for a period of 20 days:** Under this clause any injury which is dangerous to life and also causing great pain to the person is considered as grievous. If the injured is not able to carry on with his ordinary pursuits for a minimum period of 20 days, it is considered as grievous. Whether an injury is dangerous or not, it is to be assessed from the clinical condition at that moment. It is the prerogative of the examining doctor to label an injury as 'dangerous to life' depending on the vitals of the patient. Thus it can be said that all dangerous injuries are grievous but not vice versa. It is very difficult to tell that the patient is suffering from severe bodily pain for a period of 20 days. For this, the attending physician has to evaluate the nature and site of injuries; method of treatment and presence of infection. For this clause 'ordinary/daily pursuits' means acts which are a daily routine in every human being's day to day life like eating food, taking bath, going to the toilet etc. A simple inability to attend the office or work even for period longer than 20 days is not considered as enough for this clause to categorize the injury as grievous.

Homicide

The word "homicide" is derived from the Latin word *homo* and *cide*. *Homo* means 'man' and *cide* means 'I cut'. Thus homicide is the causing of death of a human being by a human being, i.e. death through human agency. Death from an accident such as falling or slipping is not homicide but if such fall or slip is caused by human agency it will amount to homicide. Homicide may be lawful or unlawful. Lawful homicide may again be

classified as (1) Excusable homicide and (2) Justified homicide.

Unlawful homicide may be classified as: (i) culpable homicide not amounting to murder (ii) murder (iii) homicide by a rash and negligent act that is not culpable.

Culpable Homicide (S. 299 I.P.C.)

Whoever causes death by doing an act (i) with the intention of causing death, or (ii) with the intention of causing such bodily injury that is likely to cause death, or (iii) with knowledge that he is likely by such act to cause death, conducts the act of culpable homicide. This section defines culpable homicide which is a wider offence than that of murder. The offence of culpable homicide as defined in this section involves the doing of an act (including illegal omission) (A) with the intention of causing death; or (B) with the intention of causing such bodily injury as is likely to cause death. or (C) with the knowledge that it is likely to cause death. and if death results from any of them the offence of culpable homicide is committed. Thus the guilty intention in (A) and (B) above and the knowledge that the act is likely to cause death as in (C) are the main ingredients of the offence. Further this section enacts that the doing of an act which causes death is culpable homicide if it is (A) with the intention of causing either (i) death or (ii) such bodily injury as is likely to cause death. or (B) with the knowledge that death is likely to be caused by such act. Thus (A) and (B) being alternative it is seen that culpable homicide may be committed without any intentions on part of the accused if he has knowledge that death is likely to result from the act. 'Knowledge' implies consciousness—a mental act, a condition of the mind which is incapable of direct proof. The word 'likely' means probably. The knowledge required by this clause is the knowledge that the act is likely to cause death. The word likely implies higher and lower degrees of probabilities of risk. On the question of knowledge much depends on the intellectual capacity of the actor. Even an illiterate person will realise that a blow with an

axe in the region of abdomen can cause death or injury resulting in death. On the question of intention, it is stated that the man in law must be held to intend the natural and ordinary consequences of his acts irrespective of object at the time of doing such act.

Explanation 1

The first explanation reproduces the English rule that an injury which accelerates the death of a dying man is deemed to be the cause of death, if he knows that the condition of the deceased was such that his act was likely to cause death. When the injury inflicted is such as to cause death but that results from rupture of organs such as spleen or liver and the accused had no knowledge of his ailment, the offence is not culpable homicide.

Explanation 2

It deals with refusal of medical treatment for injury inflicted which could have averted death as being a defence to the charge cannot be sustained. That is because the accused is responsible for the natural consequences of his conduct and the fact that the natural consequences could have been averted by artificial means is no answer. The fact that if an operation had been conducted within half an hour after the infliction of the injury and that consequently his life could have been saved could not be valid defence to the charge of culpable homicide.

The *mens rea* or mental element in culpable homicide is intention or knowledge towards the consequences of his conduct. There are three kinds of *mens rea*:

1. An intention to cause death.
2. An intention to cause dangerous injury.
3. The knowledge that death is likely to result.

The prosecution is not bound to establish motive for a crime, but evidence of motive may be adduced and considered. The mere absence of motive can be of no assistance to the accused when the offence could be proved by the prosecution otherwise in the absence of intention or knowledge as

envisaged by section 299, I.P.C. the offence committed may be one of grievous or simple hurt.

Murder (S.300 I.P.C.)

Whoever causes death by doing an act (i) with the intention of causing death or (ii) 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 (iii) with the intention of causing the bodily injury and such bodily injury intended to be inflicted is sufficient to cause death in ordinary course of nature or (iv) with the knowledge that the act is imminently dangerous that it must in all probability cause death.

Differences between culpable homicide and murder: There is real distinction between culpable homicide and murder. Broadly speaking, the offence is culpable homicide if the bodily injury intended to be inflicted is likely to cause death. It will be murder if such injury is sufficient in the ordinary course of the nature to cause death. When the question of intention arises, such intention has to be gathered from what he does. Where the injury deliberately inflicted is more than its being merely likely to cause death but sufficient in the ordinary course of nature to cause death, the higher degree of guilt is presumed.

Clause (1): In order to attract the provisions of clause (1) of Section 300, the prosecution has to prove that the very act, that was done by the accused, was done with the intention to cause the death of the victim. The intention also includes the foresight of certainty. A question of intention is always a matter of fact. Once intention of causing death is proved, culpable homicide amounts to murder unless any of the exceptions applied. Intention can be rarely proved by direct evidence, when facts are so intervened determining whether it is culpable homicide and then finding out separately whether it amounts to murder may not be convenient.

Intention can also be inferred from the acts of the accused, namely nature of the weapon used,

the part of the body where injury is inflicted, the force of the blood etc.

Clause (2): This clause deals with cases where the intention is to kill the person even though the injury is not fatal in the ordinary course of nature but is fatal in the case of that particular person, by reason of the knowledge of accused as regards the physical infirmity, disease of the person's subnormal state of health and his special physical condition. It requires not the intention to cause death but only the intention of causing such bodily injury as is likely to cause death of the person to whom the harm is caused. Here both the elements of 'intention' and 'knowledge' are required to be proved. The intention to injure and the knowledge about the consequences of injury relating to a particular victim. The 'knowledge' imparts a certainty and not merely probability.

Clause (3): The third clause of section 300 speaks of an intention to cause bodily injury which is sufficient in the ordinary course of nature to cause death. Emphasis here is on the sufficiency of injury to cause death. Sufficiency is the high probability of death, depending upon the nature of weapon used, or the part of the body where the injury is inflicted or both. If the injury is not such as would be sufficient to cause death i.e. if the probability is not so high, the offence will not fall under section 300 but will fall under section 299 or even a lesser offence.

Supreme court in the case of **Virsa Singh vs. State of Punjab**, observed that to bring the case under this part of the section the prosecution must establish objectively:

1. That a bodily injury is present,
2. That the nature of injury must be proved.
3. It must be proved that there was an intention to inflict that particular bodily injury.
4. That the injury inflicted is sufficient to cause death in the ordinary course of the nature (this is purely objective).

Once these four elements are established by the prosecution, then the offence is under section 300. It does not matter that there was no intention to cause death. It does not

even matter that there is no knowledge that an act of that kind will be likely to cause death. Once the intention to cause the bodily injury actually found to be present is proved, the rest of the inquiry is purely objective, and the only question is whether as a matter of purely objective inference, the injury is sufficient in the ordinary course of the nature to cause death. Thus where no evidence or explanation is given about why the accused thrust a spear into the abdomen of the deceased with such force that it penetrated the bowels and the coils of the intestines came out of the wound and that digested food oozed out from cuts in three places, it would be perverse to conclude that he did not intend to inflict injury that he did.

Clause (4): Clause (4) of section 300 is usually applied to cases where the act of the offender is not directed against any particular person. There may even be no intention to cause harm or injury to any particular individual, but it is the result of a general disregard for human life and safety. What this clause contemplates is the imminently dangerous act which must in all probabilities cause death. This clause deals with cases where an act which is dangerous is done, without any intention to kill a particular person but with knowledge that death is very likely and that such act is done without any excuse.

1. Number of persons set upon one and hit him on his head with *lathi*, it is murder and necessary intention must be presumed.
2. Several persons take part in assaulting a man who dies from cumulative effect of their assault.

S.302 I.P.C.—Punishment for murder: Whoever commits murder shall be punished with death or imprisonment for life and shall also be liable for fine.

S.307 I.P.C.—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 and shall be punished with imprisonment for a period

of 10 years and also fine. If only hurt is caused life imprisonment or like before attempts by life convicts shall be punished with death.

Essentials for the criminal attempt:

1. An existence of an intent on the part of the accused to commit a particular offence.
2. Some steps taken towards it after completion of preparation.
3. The steps must be apparently though not necessarily adopted to the purpose destined
4. It must come dangerously near to success
5. It must fall short of completion of ultimate design

S.321 I.P.C.—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”.

S.322 I.P.C.—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 staid 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.

Example: A, intending or knowing himself to be likely, permanently to disfigure Z’s face, gives Z a blow which does not permanently disfigure Z’s face, but which cause Z to suffer severe bodily pain for the space of twenty days. A has voluntarily caused grievous hurt.

S.323 I.P.C.—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.

S. 324 I.P.C.—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 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.

S.325 I.P.C.—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.

S.326 I.P.C. 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 human body to inhale, to swallow, or to receive into the blood, or by means of any animal, shall be punished with, imprisonment for life, or with imprisonment of either description for a term which may extend to ten years, and shall also be liable to fine.

S. 304A I.P.C.—Causing death by negligence:

Whoever causes death of any person by doing any rash and negligent act not amounting to

culpable homicide shall be punished with imprisonment up to 2 years or with fine.

To impose criminal liability under section 304 A I.P.C., it is necessary that the death should have been the direct result of a rash or negligent act of the accused and that the act must be the proximate (not remote) and efficient cause without intervention of another's negligence and with no intention or knowledge

Distinction between rash act and negligent act:

Rash act: The criminality lies in running the risk of doing such an act with recklessness or indifference as to the consequence.

Criminal negligence: Criminal negligence is the gross and culpable neglect or failure to exercise that reasonable and proper care and precaution to guard against injury either to the public generally or to an individual.

COMPLICATIONS OF INJURIES

Serious bodily injuries, from whatever cause, may either lead to virtually instantaneous death from destruction of vital organs and structures or may cause delayed death from complications of the origin injury. This delay may be short, as in torrential haemorrhage or acute respiratory failure or it may be progressively longer stretching for hours, days, weeks or even years. Some of the most difficult problems in forensic pathology and some of the most arduous testimony and cross examination in the Court of law concerns deaths from which post-traumatic complications are disputed as being causative factors. The various complications of injury resulting in death are discussed in this chapter.

SHOCK

Shock commonly called circulatory collapse may develop following any serious assault on the body's hemostasis such as profuse haemorrhage, severe trauma or burns, extensive myocardial infarction, massive pulmonary embolism, or uncontrolled bacterial sepsis. Shock constitutes widespread hypoperfusion of cells and tissues due

to reduction in the blood volume or cardiac output, or redistribution of blood resulting in an inadequate effective circulating volume. Incident to the perfusion deficit, there is insufficient delivery of oxygen and nutrients to the cells and tissues and inadequate clearance of metabolites. The cellular hypoxia induces a shift from aerobic to anaerobic metabolism, resulting in increase in lactate production and sometimes lactic acidosis. While at the outset the hemodynamic and metabolic derangements are correctable and induce reversible injury to cells, persistence of worsening of the shock state leads to irreversible injury and death of the cells. The shock is classified in to the following types (Table 22.1):

Primary or Neurogenic Shock

Primary or neurogenic shock is a reflex neurovascular disturbance immediately after an injury. It results from exciting factors such as pain, grief, anxiety, emotions, stress etc. Even a site of blood may cause this type of shock and is common after massive haemorrhages. The reactions are due to symapthetico-adrenal stimulation with its resulting systematic effects. There is sudden reduction of venous return to the heart due to pooling of blood in the splanchnic and peripheral vascular bed from neurogenic vasodilatation. The sudden rise of blood pressure can precipitate complications like intracerebral haemorrhage from rupture of arterio-sclerotic cerebral vessels or Berry aneurysm and rupture of dissecting aneurysm of aorta. Minor stimuli or injury over the receptive spots having receptor nerve endings forming the afferent pathway for reflex action by inhibitory vagus nerve causing vagal inhibition result in sudden death from neurogenic shock. Rapid death can occur especially in persons with chronic cardiac lesions.

Clinical features of neurogenic shock: (i) Tremors and anxiety (ii) Pallor (iii) Cold clammy extremities (iv) Sighing respiration and repeated yawning (v) Dilatation of pupils (vi) Rapid pulse and slightly raised blood pressure.

Table 22.1: Classification of shock

<i>Types</i>	<i>Clinical examples</i>	<i>Principal mechanisms</i>
Cardiogenic shock from interference of heart function	Myocardial infarction, heart rupture, arrhythmias, cardiac tamponade, massive pulmonary embolism	It is due to the failure of myocardial pump resulting from intrinsic myocardial damage, extrinsic pressure or obstruction outflow
Hypovolaemic, also known as haemorrhagic or traumatic shock	Massive haemorrhage (10-20% of total blood volume) and loss of fluid in as in vomiting, diarrhoea or burns	The blood or plasma volume is inadequate
Septic	Gram negative bacteremia-endotoxic shock or gram positive septicemia	There is peripheral vasodilatation with pooling of blood and cell membrane injury with DIC
Primary or neurogenic shock	Anaesthesia and spinal cord injury	Parasympathetic inhibition or sympathetico-adrenal stimulation of the circulation—peripheral vasodilatation with pooling of blood

Diagnosis at autopsy

1. There is history of sudden death resulting from blow or injury over the receptor areas.
2. The person is usually of emotional temperament, severely ill, feeble, an old person or young child.
3. Findings of fatal injuries, poisoning and natural diseases should be ruled out.

Secondary or Haematogenic Shock

The circulatory disturbances develops gradually after infliction of injuries. It is more serious and fatal.

Pathogenesis: The reduction in effective circulatory volume can result from loss of blood or plasma at the site of injury and peripheral capillary dilatation or increased capillary permeability. Due to changes in this hemodynamic mechanism, there is diminution of effective circulatory volume, diminished venous return to the heart, decreased cardiac output and diminished supply of oxygen to the tissues. This tissue anoxia results in the disturbance of intracellular and anaerobic glucose metabolism leading to increased production of lactic and other acids that causes disruption of lysosomes and release of lytic enzymes with resulting cell death. The reduced production of

ATP causes reduced protein synthesis, disturbance in transport mechanism and cell membrane permeability. The continuing tissue anoxia causes interference with enzyme systems leading to metabolic acidosis, water and electrolyte imbalance and renal insufficiency. If there is superadded gram negative endotoxaemia circulatory failure may be enhanced by increased capillary permeability or widespread peripheral vasodilatation.

Reversible or compensated shock: The compensatory body mechanism try to fight this by producing arteriolar vasoconstriction and increased peripheral resistance by increasing levels of circulating catecholamine, cortisol, aldosterone and angiotensin and secretion of ADH hormone. As a result, arteriolar constriction may try to improve the venous return to heart and circulating blood volume. With appropriate treatment the compensatory mechanism reestablish the circulation and the patient is recovered. This type of shock is reversible or compensated shock.

Decompensated or irreversible shock: In the decompensated or irreversible shock, the compensatory mechanisms fail to prevent deterioration of hemodynamic mechanisms as a result the condition of patient worsens with ensuing death. This irreversible shock occurs in massive

myocardial infarction, bacterial sepsis and repeated haemorrhage and is due to many factors

1. Due to persistent reflex vasoconstriction, cellular hypoxia and acidosis occurs
2. The generalized vasoconstriction may injure gut mucosa helping in the absorption of proteins and bacterial products with resulting bacterial endotoxaemia that causes motor atony and peripheral pooling of blood.
3. Due to falling circulation, tissue anoxia is produced that disrupts lysosomes in the cells and release of hydrolases and thus further tissue damage.
4. Due to vasoconstriction after injury, the renal anoxia, splanchnic anoxia is produced that results in the production of vaso-depressor material in the liver, spleen and kidneys. This causes peripheral vessels to become refractory to adrenaline. This is followed by vascular atony with capillary dilatation and peripheral pooling.
5. Due to formation of minute capillary thrombosis of the injured capillaries from hypercoagulability.

Hypovolaemic or Oligaemic Shock

It results from diminished blood volume caused by:

1. Loss of blood or plasma resulting from traumatic injuries, burns or surgical operations
2. Loss of fluid as in diarrhoea, vomiting and adrenal insufficiency.
3. Due to generalized vascular permeability and peripheral vasodilatation there is escape of fluid in tissue spaces resulting in withdrawal of blood in to these vessels from the general circulation.

Clinical features: (i) Generalized weakness (ii) Profuse respiration with cold clammy skin (iii) Dilated pupils (iv) Tachycardia and tachypnoea (v) Diminished pulse pressure.

Autopsy findings: The findings are non-specific with generalized capillo-venous engorgement and widespread Petechial haemorrhages in different areas of body. There are findings of tissue oedema and necrosis of visceral organs.

THROMBOEMBOLISM

Thrombosis: It is the formation of clotted mass of blood within the non-interrupted cardiovascular system is termed thrombosis and the mass itself a **thrombus**. Thrombi may diminish or obstruct vascular flow causing ischemic injuries. Thrombi may diminish or obstruct vascular flow causing ischemic injuries to tissue and organs, or become dislodged or fragmented to create the emboli.

Emboli: These are defined as a detached intravascular physical mass that is carried by the blood to a site distant from its point of origin. 95% of all emboli consist of mass of blood clot dislodged from an intravascular thrombus. Other less common form are globules of fat, bubbles of air, debris of amniotic fluid, particles of normal tissue such as bone marrow and brain, some abnormal tissue such as malignant brain cells and aggregates of bacteria or even parasites. Embolic phenomenon can be an added risk associated with surgical operation procedure, trauma and non-traumatic natural disease.

External embolism: External embolism can be due to a:

1. Foreign body (bullet from firearm) or
2. Foreign material (I.V) that is:
 - Oxygen (by accident)
 - Air (by accident/ injection)
 - Solid particles (drug addicts from I.V. injections)
 - Solid particles (suicidal and therapeutic injections).

Internal embolism: Most emboli are derived from the thrombi. Thrombosis may be due to vegetations or clusters of bacteria (septic thromboembolism). It can be: (i) Fat embolism (ii) Bone marrow embolism (iii) Amniotic fluid embolism.

Thrombus may be of arterial or venous origin.

Arterial thrombi may arise from:

1. Left auricle and appendage especially in mitral stenosis and atrial fibrillation.
2. Mural thrombus mainly from left ventricle.
3. Thrombus dislodged from aorta or from aneurysms.

4. Vegetations from valves due to subacute bacterial endocarditis or ulcerative endocarditis.

Venous emboli may result from sudden immobilization for even a short period of time may result in bilateral phlebothrombosis in calf veins.

Most common sites of thrombosis are in deep femoral vein, posterior tibial vein and popliteal vein. The factors predisposing to leg vein thrombosis after an injury that is traumatic lesions of lower extremity especially fractures of long bones are:

1. Local tissue damage causing injury to the veins.
2. An increase in clotting time of blood which is maximum at two weeks after the injury.
3. Immobility and bed rest.
4. General debility especially in the old leading to poor general circulation and cardiac output.

Thrombus is more common in veins than the arteries and usually develops in thigh and leg veins. Thrombus usually develops in 10-20 days (usually a week after the trauma). It may be seen in pelvic vessels and rarely in jugular veins. The thrombus is detached in part or whole, and travels to pulmonary and other veins such as venous plexus of the dura mater, subclavian veins, axillary veins, external & internal iliac veins, prostatic veins, uterine veins and ovarian veins. All these veins develop bland thrombi after traumatic injury. Bullet wounds and stab wounds of carotid arteries may injure intima and a thrombus may develop at the site. The thrombus is to be differentiated from an postmortem clot. The thrombus is pale gray in colour and tangled fibrin strands are present. It is often attached to the underlying vessel wall. It shows the perfect casts of the vessels in which they are located. **Postmortem clots** are rubbery, gelatinous coagulum that lacks fibrin strands and attachment to the underlying wall. It is uniformly dark red, slippery, soft, lumpy moist clot (**black currant jelly**) or pale, bright yellow (**chicken fat**).

As a result of thromboembolism, massive infarcts may result but may not be seen if death has occurred rapidly.

When a large embolus obstructs the bifurcation of pulmonary artery or one of its branches, an immediate acute dilatation and failure of right side of heart occurs (**Acute Cor pulmonale**). Unusual types of pulmonary embolism are sometimes produced by trauma.

1. Fragments of bone marrow may be found as emboli in smaller branches of pulmonary artery, usually follow fracture of bone containing marrow.
2. Fragments of liver parenchyma may enter torn hepatic veins after blunt force injury to liver and may be carried as emboli.
3. Laceration of brain accompanied by tearing of venous sinuses may lead to pulmonary embolus.
4. Foreign bodies may enter veins or arteries and are transported as emboli e.g. a bullet.

Pulmonary Air Embolism

Pulmonary embolism is a condition in which thrombi are formed in the walls of the pelvic and leg veins and such thrombi break away and embolise to the lungs. Although the thrombosis is the primary event, the embolus itself usually consists of a tube of thrombus with a central core of clotted blood. When it reaches the lung, its effect depends on its size, small ones are carried to the periphery of the lung and causes **pulmonary infarcts**. Large emboli reach the bifurcation of pulmonary artery completely blocking the blood circulation. Most common sites of thrombosis are in deep femoral vein, posterior tibial vein and popliteal vein.

Intravenous route is the most common recognized source of entry especially during therapeutic injection. For a serious result, two main factors are required; either positive pressure or negative pressures usually at a site near the heart. In any event there must be an adequate amount of air, at least 60 ml or more. Systemic air embolism occurs when an air in sufficient quantity enters a vein of the pulmonary system and is carried through the left side of the heart to block arterioles and capillaries in different parts of the body especially

brain and heart. One to two ml of air may be enough to produce death because air travels directly from the lungs to the left side of the heart, from which it can be forced into coronary or cerebral arteries. Death from air embolism occurs within a few minutes, not delayed beyond 45 minutes. Air entering the venous system is carried to the heart and pulmonary arteries, which causes mechanical obstruction of the pulmonary arterial vasculature. This causes churning of blood and air producing froth. The churning can result in development of complexes of air bubbles, fibrin, platelet aggregates, erythrocytes and fat globules thus further occluding the vasculature, which causes death. This occurs most commonly in penetrating wounds of chest, crush injuries of the chest and surgical procedures upon thorax. Other circumstances under which such accidents occur are:

1. During transfusion when positive pressure is being used
2. During surgical operations especially during those on neck and chest when negative pressure will produce a 'suck-in' effect.
3. During craniotomy in the sitting up position
4. Operations on nasal air sinuses
5. Incised and penetrating wounds of the neck and chest involving jugular and subclavian veins. Fatal accidental oxygen embolus occurred on one occasion during an operative procedure.
6. Intravenous drug addicts inject foreign bodies at the time or following subsequent surgical procedures e.g. metallic mercury embolism.
7. A wound of sagittal sinus inside the skull.
8. Injection of air under pressure in fallopian tube to test the patency
9. Injection of air or fluid mixed with air or soap water in the pregnant uterus for procuring abortion.
10. Caesarean section, version or manual extraction of placenta may introduce air into the uterus.
11. Crush injuries of chest
12. Subclavian vein catheterization
13. Positive pressure ventilation in newborn infant
14. Artificial Pneumothorax and pneumoperitoneum
15. Air encephalography
16. Caisson's disease.

Autopsy Diagnosis

Thromboembolism should specially be suspected when death occurs after a week of trauma. This is seen in upto 65% of such patients who die after a week of trauma. The thrombus can be demonstrated by inserting a flexible wire with blunt terminal knob inserted in femoral vein.

1. If air embolism is suspected, head should be opened first and surface vessels of brain examined for gas bubbles, which must be prominent and definite and not the segmental break up of blood in the vessels with collapsed segments in between.
2. Avoid pulling the sternum and ribs to avoid creating negative pressure in the tissues, which may result in aspiration of air into vessels.
3. Before handling thoracic organs, the pericardium is opened, heart is lifted upwards and the apex is cut with a knife.
4. The left ventricle is filled with frothy blood, if air is in sufficient quantity to cause death.
5. If the right ventricle contains air, the heart will float in water.
6. Air embolism can also be demonstrated by cutting the pericardium anteriorly and grasping the edges with hemostat on each side.
7. The pericardial sac is filled with water and heart is punctured with the scalpel and twisted a few times. Bubbles of air will escape if air is present.
8. The amount of air can be measured by placing inverted water filled graduated glass cylinder, with the mouth of cylinder in pericardial sac.
9. Oxygen in heart indicates air embolism because it is not present in appreciable

quantity if gases were those of decomposition. Frothy blood resembling air embolism can be found especially in right ventricle due to handling of heart before it is opened at autopsy, putrefaction, as a postmortem event and artificial respiration in dying or recently dead.

10. Skull vault be removed without puncturing the meninges.
11. Internal carotid and basilar arteries are ligated before the brain is removed.
12. Meningeal vessels should be examined for visible air bubbles. In acute cases, gas bubbles will be visible within the cerebral arteries but not in the cortical veins.
13. Brain should be submerged in water and ligature should be released. Then the vessels are cut and slightly compressed to watch for air bubbles.
14. An air tight, water filled glass syringe with a needle can be used to collect gas from blood vessels, heart or cavities.

Dating of Pulmonary Embolus

For dating an embolus, veins along with the thrombi and the muscles around it are excised as a sample for routine. Histopathological examination. This tissue can be processed and fibrin can be demonstrated by phototungstic acid-haematoxylin stain and Martius Scarlet Blue stain. With phototungstic acid-haematoxylin acid stain the changes are:

- First day—Purplish strands of fibrin.
- Four days—Thicker strands and sheets of fibrin.
- Two weeks—Fibrin strands are dark purple.
- By twenty-fifth days—Fibrin begins to be absorbed.

With, Scarlet Blue stain, initially the fibrin is seen as pink coloured that becomes scarlet in about a week.

Diagnosis of Air Embolism

Radiological examination of the whole body will detect large quantities of air. Ophthalmoscope will

detect air bubbles in retinal arteries. The gas collected from heart on addition of alkaline pyrogallol solution will become brown in the presence of free oxygen and indicates antemortem air embolism.

Bone Marrow Embolism

Emboli can always be found in the lungs in cases of death from severe multiple injuries such as that seen in air craft disasters, their presence may be of value in determining the rapidity of death. Fat will also be present in the lungs.

Amniotic Fluid Embolism

It is one of the leading causes of sudden death during labour and during abortion. Vernix and fat can be demonstrated in the lungs and cardiac vessels.

Fat Embolism

Fat embolism is caused by:

- Fracture of a long bone namely, femur and pelvic bones
- An injury to adipose tissue which forces liquid fat into damaged blood vessels
- Injecting oil into circulation e.g. in criminal abortion
- Occasionally due to natural disease without trauma as in sickle cell anemia, diabetes, following blood transfusion, fatty change seen in liver as seen in chronic alcoholics, septicemia, steroid therapy, acute pancreatitis, osteomyelitis, decompression sickness (Caisson's disease), in too rapid ascent to high altitudes without functional pressurized cabin or under simulated flight conditions in decompression chambers and in burns.

Two factors favours pulmonary fat embolism:

1. Fixation of walls of veins e.g. in clavicular and pelvic regions, the upper dorsal spine and dural membranes.
2. The suction effect of the respiratory movements and heart's actions on such veins as the jugular, subclavian and vertebral has a tendency to create a negative pressure in the vessels during phase of inspiration.

Presence of fat droplets in the blood stream indicates that the injury was produced during life, except in case of burning, advanced putrefaction and charring after death. Large amounts of liquefied fat are derived from fat cells. When cardiac massage is done, fat enters the blood vessels even if the circulation has stopped. If during resuscitation, the sternum or ribs are fractured bone marrow embolism is seen in the lungs.

Clinically: The phenomenon presents as sudden loss of consciousness usually after a period following the traumatic accident, which need not in itself be serious such as a fractured tibia and fibula.

Autopsy diagnosis: Droplets of fat should be demonstrable in the peripheral viscera notably vessels of kidneys and brain. There are petechial haemorrhages in organs and punctate haemorrhages in the white matter of brain.

HAEMORRHAGE

Bleeding may occur externally through a lacerated or incised wound or through trachea, bronchi, or nasal passages, ears, vagina, urethra or rectum. The contusions result in internal bleeding from rupture of vessels and bleeding into body cavities; pericardium, peritoneum, pleura and cranium. The amount of total blood volume, the rapidity and site of bleeding governs its fatality.

1. When the leakage is slow the body can compensate for the loss of far greater volume, by adjustment of vascular bed and by restoration of blood volume by transfer from other aqueous compartments.
2. As far as site is concerned, the small quantity of bleeding into the brainstem is likely to be fatal whereas the same volume exuded into the pleural cavity would be of little consequence.
3. Bleeding usually begins at the time of injury with a momentary delay at the instant of infliction. Due to transient spasm of local vessels from the stimulus of the injuring object, blood may hesitate for a second or so before welling out in sharp cuts of skin. Then the bleeding continues until normal hemostasis plugs the vessels.

4. In some injuries involving arteries, the musculo-elastic vessel may retract and its wall invaginates, so that an immediate seal prevents copious haemorrhage.
5. Sometimes gross injuries such as amputation of limb by railway wheel may be devoid of significant bleeding because the crushing effect may combine with arterial wall retraction to seal the cut vessel.
6. Failure of hemostasis is a hematological matter but it has medicolegal significance if a surgery is performed on someone who has a haemorrhagic diathesis either from natural disease or from anticoagulant treatment.

Delayed bleeding is seen in

1. In road traffic accidents, liver, spleen or lung may be injured within their capsules or covering membranes that may initially remain intact. A subcapsular hematoma of the liver may grow even larger due to continued bleeding stripping more capsule from the parenchyma. Eventually, a blister may rupture with bleeding into peritoneal cavity.
2. Trauma can weaken the walls of an artery or vein leading to a false aneurysm that can rupture later on. An arteriovenous fistula may also rupture.
3. Infections at the site of trauma can involve vessels in the vicinity so that an abscess or cellulitis can lead to secondary haemorrhage when the vessel wall is eroded.
4. It is sometimes difficult to know that how much of the haemorrhage found at autopsy may be accounted for by post-mortem bleeding. Except in serous cavities such as pleura or peritoneum, or externally from the body surface, in most cases this is a small proportion of that which leaked under arterial pressure during life, due to the tissue pressures opposing passive bleeding.
5. A hemothorax from a ruptured aorta may amount to several litres and much of this is in the form of a large clot. In this case any postmortem addition will not affect its interpretation.

INFECTION

Infection used to be common after wound infection before the advent of antibiotics. In criminally inflicted wound that were not dangerous to life may become fatally infected so that an assault becomes a homicide. Purulent wound infection from Gram positive cocci, Gram negative bacilli, anaerobes such as *Clostridium perfringens* and the other more common organisms is the most frequent. Tetanus and anthrax are also dangerous. The forensic relevance is to prove the chain of causation between the original injuries and death from intercurrent infection. There may be medico-legal issues such as failure to give or delay in giving antibiotic cover which have both civil and criminal consequences. A criminal assault that ends in death because of a neglected infection does not exonerate the perpetrator from all responsibility, even though there has been a *novus actus interveniens* in the form of defective medical treatment.

ADULT RESPIRATORY DISTRESS SYNDROME

Adult respiratory distress syndrome develops following severe lung injury, such as gross impact upon the thorax or blast injury from explosion, or from aspiration of gastric contents, infections, toxins, systemic shock, irritant gases, near drowning and many other causes. As a result, the lung epithelium may suffer 'diffuse alveolar damage'.

Clinical features: (i) Marked dyspnoea. (ii) Progressive respiratory failure, leading to hypoxaemia.

Autopsy findings: At autopsy the lungs are hard and retain their shape after removal, having a 'dry-oedema' appearance, being almost double in weight in some instances.

Histopathological findings: Pathologically the lungs show a stiff oedema that progress to a rigid, infiltrated lung if survival is long enough. Histologically the initial changes are shedding of type I pneumocytes with intra-alveolar exudates, the formation of hyaline membrane and patchy alveolar haemorrhage. This destructive phase is then

replaced after a few days with a proliferative stage, when Type II pneumocytes begin to fill the alveoli and a mononuclear response infiltrates the interstitial areas. The alveolar proliferation organizes, if survival continues, and eventually pulmonary fibrosis develops.

RESPIRATOR LUNG

This condition is similar to adult respiratory distress syndrome developing when a patient dies after an appreciable period of mechanical ventilation. It may appear within a few days, but usually supervenes after many days or weeks on a ventilator. The lungs are stiff and rigid which though heavy does not usually appear obviously oedematous. Microscopically, proteinaceous fluid and a mixture of proliferative cells may be seen in the alveoli.

RENAL FAILURE

Renal failure is a common sequel to extensive muscle damage or to burns affecting considerable areas of skin. When the muscle is crushed, severely torn or otherwise rendered ischemic, where there are excessive burns, or where poisons such as mercuric salts or carbon tetrachloride are given, destruction of the renal tubular epithelium may be seen. A similar change occurs during post-mortem autolysis. In burns and muscle damage, the tubules may also be blocked with brown casts of myoglobin and these two changes of distal tubular necrosis and the casts were thought to cause profound oliguria and anuria often associated with trauma and burns. In biopsy as opposed to autopsy material, the tubular damage has been found to be minimal and the casts may be a sequel to reduced infiltration rather than to tubular damage, so the validity of acute tubular necrosis as an explanation of renal failure following trauma has been questioned.

DISSEMINATED INTRAVASCULAR COAGULATION

DIC is a consumption coagulopathy associated with the blood clotting mechanism. It follows a

whole range of traumatic, infective and other acute events. There is an abnormal activation of the coagulation process within the blood vessels caused by many factors related to the blood itself, the vessel walls and to blood flow. Damaged tissue from trauma and burns can trigger thromboplastin initiated coagulation-entry of tissue cell elements, especially from erythrocytes; brain and placenta are particularly potent. Particulate matter such as microorganisms or microemboli of all types (including fat and air emboli, especially those from decompression) can precipitate coagulation via Factor XII. Vascular endothelial damage and stasis of flow can have a similar effect. Whatever the causes, the fibrinogen is consumed and fibrin precipitated in vessels, leading to both vascular obstructive effects and to haemorrhagic diatheses from depletion of the coagulation system. Platelets are also consumed, adhering to the fibrin thrombi. Fibrinolysis is activated and there is a dynamic contest between intravascular coagulation and its removal.

After death there can be postmortem fibrinolysis, which lessens the ease with which fibrin deposits can be detected; staining techniques are also an imperfect method of visualizing the fibrin so a careful search must be made in autopsy histology for fibrin remnants. Lungs, liver, kidney and adrenals are most likely organs to yield positive results. Microvascular obstruction leading sometimes to frank infarction as well as reduced function together with bleeding forms major dangers of DIC in forensic context.

SUBENDOCARDIAL HAEMORRHAGE

Subendocardial haemorrhages were specifically studied in the 1930 by **Sheehan** in cases of abortion and acute haemorrhages associated with pregnancy. They were formerly known as 'Sheehan's haemorrhages' and are a striking feature of many autopsies especially on victims of severe trauma:

1. These are well marked haemorrhages seen under the endocardium of left ventricle, on the interventricular septum and on the opposing

papillary muscles and adjacent columae carnae of the free wall of the ventricle.

2. These are flame shaped, confluent haemorrhages, not petechial, and tend to occur in one continuous sheet rather than patches. The bleeding is in a thin subendocardial layer, but when severe may actually raise the endocardium into a flat blister that can be palpable on the smooth septum.
3. The mechanism of production is obscure, but they are commonly seen:
 - After sudden, profound hypotension from shock resulting from severe haemorrhage.
 - When there is intracranial damage from head injuries, cerebral oedema, surgical craniotomy or large intracranial tumours and sudden intracranial decompression.
 - In obstetric conditions such as deaths from antepartum or postpartum haemorrhage, ruptured ectopic gestation, abortions and ruptured uterus often reveal these lesions.
 - Poisonings especially heavy metal poisoning, particularly arsenic

The common factor seems to be sudden hypotension and if the intraventricular pressure drops precipitously, the existing blood pressure in the coronary system is then unsupported across the endocardium by an equal pressure within the ventricular lumen, so that rupture of superficial vessels occurs. These being flaws in this theory, one being the common incidence in intracranial lesions. These haemorrhages are known to be a part of Virchow's triad of 'pulmonary oedema, gastric erosions and subendocardial haemorrhages' seen in head injuries and cases of raised intracranial pressure.

NON-FATAL SELF INFLICTED INJURIES

These types of injuries exist in persons who have some abnormality of mind leading the victim to mutilate his body and in whom the injuries are inflicted for motives of gain. Some mentally disordered persons may inflict many small wounds upon themselves, which may be additional to the

actual cause of death. The arms may be covered with scores of parallel superficial incisions from a knife, razor or broken glass. They may cross each other in groups and are more frequent on the non-dominant side, usually the left. Another well recognized injury is self mutilation of the genitals, almost invariably by men who are paranoid schizophrenics. The penis, testis and scrotum may be removed and death occurs from intractable haemorrhage in such cases.

Self-inflicted injuries, usually non-fatal unless some complication has arisen, may be motivated by some form of gain. The most common is fabrication of injuries to simulate an assault, either to divert attention from the person's own theft or to arouse sympathy. Fabricated accidental injuries are usually a form of malingering, as in armed forces, or in fraudulent attempts to obtain compensation. In other cases the injured may claim that a specific person assaulted him.

Munchausen's Syndrome

It consists of repeated simulations of illnesses or the infliction of repeated minor injuries, with the object of gaining admission to hospital, or obtaining medical care and attention.

Features of Self-inflicted Incised Wounds

1. The cuts are usually superficial but occasionally may penetrate the full thickness of the skin, but not in sensitive areas such as face. The injury is rarely dangerous to life unless infected.
2. The incised wounds are regular with an equal depth at origin and termination unlike serious wounds that tend to be deeper at the start and tail off to the surface.
3. The cuts are multiple and parallel (Fig. 22.1) and avoid the sensitive areas like the eyes, lips, nose and ears, usually being drawn on the cheeks and the jaws, temples and forehead, sides of neck, chest, shoulders, front and back of arms and forearms, back of hands and thighs. This is inconsistent with an attack by another person, as the victim is unlikely to stand still to allow these multiple delicate and uniform injuries to be carefully executed.



Figure 22.1: Multiple self inflicted injuries

4. In the right handed person, most of these injuries are on the left side, especially side of face and left hand.
5. When the incisions are on the covered area, the relevant garments may show either no cuts or if there are cuts that do not match the injuries in position or direction.

SURVIVAL PERIOD AFTER WOUNDING

During an investigation, a forensic pathologist is often asked the probable time interval between the fatal injuries and death? The pathologist should never give a dogmatic answer unless the nature and severity of injuries are obviously incompatible with continued life or activity. The doctor should always give opinion with caution as victims sometimes survive for long than expected. A person whose brain stem is destroyed by a penetrating injury or whose aortic arch is completely transected, will be inactive and clinically dead almost immediately. But extraordinary instances of survival have been recorded when the frontal lobes are damaged or the abdominal aorta was crushed by a railway wheel. In the more usual stab injury, head injury, cut throat or firearm wounds, the forensic pathologist must try to assess the nature of severity of the physical damage and relate that to the age, health and environment of the victim. Other than injuries to the brain or to a large blood vessel, most other injuries can rarely be declared to have caused sudden death or rapid loss of

function. A senile old lady, or someone with severe cardiac or respiratory disease is less likely to survive multiple injuries for as long as the robust young person, though if the nature of injury is grossly life-threatening, these factors make little difference. In criminal cases, it may be a point of some importance to decide whether the victim could have continued fighting, have resisted, ran away or even have inflicted injuries on someone else before collapsing and dying. In the adrenaline response of 'fight or flight', the shock element of pain is greatly suppressed in an assault and only the sheer physical and hemodynamic sequelae of injury will eventually lead to a slowing down, then collapse and death

1. A wound in the left ventricle can partly seal itself by the contraction of muscle around the defect and victim will collapse only when sufficient blood has leaked into the pericardium to form cardiac tamponade.
2. Wounds of the right ventricle are often more rapidly fatal as, although the pressure of contained blood is less, the thinner wall is not so effective in preventing the leak.
3. Head injuries present many paradoxical instances of prolonged survival, depending on the part of brain injured. Frontal lobes are remarkably resistant to damage as it is generalized impact rather than focal damage to this area that does the most harm.
4. A cut artery will lose blood faster than a vein of the same general size, especially if it is only partly severed so that it cannot retract. Other factors such as possibility of air embolism in cut jugular veins might alter the situation. The heart is less vulnerable than great thoracic vessels in many stab wounds. Where a wound has transected a major coronary artery, prolonged survival is unlikely, as is one that interrupts a major branch of the conducting system.

Most issues of such nature revolve around periods of a few minutes, the perimortem or agonal time when few morphological signs are available. The accepted wisdom of wound dating is that polymorph leucocytes begin to appear in the

wounded tissue within minutes, but can happen even several hours after death as all leucocytes do not become immobile with cardiac arrest. The death being a process is conventionally taken to be the moment of cardiac arrest and thus collapse of blood pressure and cerebral circulation, the cells of the body are still alive and remain so for variable time—only minutes in the case of neurons, but leucocytes and muscle cells survive for many hours and connective tissues such as fibroblast for days. Thus it is unreasonable to expect dramatic changes within minutes in skin wounds, until progressive hypoxia alters biochemical processes and enzyme activity. Leucocytes may be motile for more than 12 hours and can aggregate around chemotactically active material such as gastric content around the air passages. This makes the vital reaction a dubiously valid phenomenon in perimortal period. If the survival is longer, then both gross and histological changes of 'vital reaction' such as thrombosis, inflammation, infection and healing may be useful, but they usually require hours or even days of survival to appear.

TIMING AND HISTOPATHOLOGY OF WOUNDS

Clinical Timing of Wounds

It is not possible to determine the exact age of a wound by naked eye examination because the intensity of local inflammatory reaction varies. Under normal conditions,

After 12 hours—Edges of the wound are red and swollen

After 24 hours—Small wound may show scab

After 36 hours—Epithelium around the edge of the wound begins to grow

4-5 day—Epithelization of small clean wounds may be complete.

Timing of Bruise

- Initially—Red in colour.
- After 18-24 hours—Blue or livid margins become lighter acquire a violet tint.

- 3-4 days—Reddish brown.
- 4-5 days—Green.
- 7-10 days—Yellow.
- It heals in about a week.

A small wound may show scab formation after about 24 hours and pus formation appears after about 36-48 hours, if septic and does not heal for days or weeks of sloughing of surrounding tissue. Vascular endothelium shows distinct proliferate changes. During 24 hours, vascular buds are given off from minute vessels at periphery and in 36 hours a complete network of new capillary vessels seen. In the living body the damaged tissues imbibe the extravasated blood. The wound edges gape and become swollen after a lapse of about 12 hours. Later on signs of inflammation and of healing dominate the picture. Haemorrhages even with abundant fibrin formation are no longer reliable indicators of antemortem origin of wound.

Microscopically Examination of the Wounds

Because of the uncertain and variable results of the naked eye examination, it is important to study injuries microscopically.

- Leucocytic reaction is the earliest histological sign of inflammation
- The appearance of a few leucocytes in the wound periphery does not justify the diagnosis of a vital reaction because these cells are regular component of the connective tissue.
- Margination and limited migration of leucocytes may occur in tissues in response to injury even after somatic death.
- The first leucocytes to pass in to the tissues are polymorphonuclear neutrophils.
- Monocytes appear before a lapse of about 12 hours but later than neutrophils.
- Exudation reaches its maximum intensity within 48 hours of the injury.
- Epithelial growth is clearly visible on 3rd day.
- Fibroblasts show reactive changes within a few hours. They begin to undergo division within about 15 hours after injury and granulation tissue develops within at least three days.
- New collagen fibrils appear with 4-5 days of injury.

- A fibrous tissue scar may appear with in a week in small wounds.

Wound Healing

Wound healing is described as an epimorphic process by **Needham** namely a form of regeneration in which there is development in situ of portion lost. It shows two main phases.

Regressive phase: It is subdivided in order of occurrence of wound closure, demolition of damaged cell and differentiation of cells to provide new tissue for the process of repair.

Progressive phase: It is divided in to three phases; the formation of repair tissue, growth of repair tissue and differentiation of repair tissue.

All these stages overlap and vary in different organisms and even in different parts of the same organism. The series of events in response to the initial injury generally follow a definite order and the two main phases described above are generally subdivided in descriptions of wounds in human tissues in to four periods (**Douglas, 1963**).

1. *The phase of traumatic inflammation:* It lasts from one to three days after wounding when fibrin and dilated capillaries appear histologically.
2. *Destructive phase:* It varies from 4-6days and is characterized by numerous leucocytes and macrophages.
3. *Proliferation phase:* It lasts from 4-14days and is characterized by the presence of fibroblasts around capillaries and some metachromasia of tissue ground substance.
4. *Maturation phase:* This phase is gradually reached over many months. Collagen formation is accompanied by decrease in number of fibroblasts and there is progressive increase in tensile strength of the wound.

Phase 1 & 2 are lag phase in which no proper repair occurs.

Histochemical examination of Wounds (Fig. 22.2)

Enzyme histochemistry methods are applied for the study of wound healing. Enzymes are proteins

Age of wound	ATP-ase	Esterase	Amino-peptidase	Acid phosphatase	Alkaline phosphatase	Mitraging polymorpho-nuclears	Cells mono-nucleas
1 hr	[Low enzyme activity]						
2 hrs	[Moderate enzyme activity]						
4 hrs	[High enzyme activity]						
8 hrs	[Very high enzyme activity]						
16 hrs	[Maximum enzyme activity]						

Figure 22.2: Changes in enzymes as seen histochemically in the first hours after wounding

that catalyze biologic reactions. Thus demonstration of enzymes could reveal earlier reaction than could visualization of the resulting morphologically demonstrable changes. Healing is an example of enzymatically-catalyzed processes in the organism on which its life depends. Histological examination is based on the production of microscopically visible reactions by applying chemical tests to tissue sections prepared for microscopic study. For histological and histochemical examination:

- The wounds or their parts are excised with their surroundings about $\frac{1}{2}$ an inch in each direction.
- One half of the tissue block is fixed overnight in neutral 10% formalin at 4°C for demonstration of esterase and phosphatase activity and for histological examination.
- The other half is fresh frozen with isopentane chilled with dry ice; the frozen specimen is subjected to histochemical methods for detecting adenosine phosphatases and amino peptidases.

The demonstration of enzymes in situ depends on their action on a specific substrate in the

presence of other substances with which one of the decomposition product resulting from enzyme activity will form an insoluble deposit at the site of enzyme action. If this deposit is not already coloured, it is rendered visible by the use of suitable chemicals. That is, an increase in enzyme activity appears as an intensified colour and a decrease in enzyme activity as a declining stainability.

To summarize the results, two zones can be demonstrated around antemortem wounds (Fig. 22.3).

1. *Central (superficial) zone*: It is in the immediate vicinity of the wound edge and is 200-500 μ deep, shows decreasing enzyme activity. It should be considered as an early sign of imminent necrosis. The regressive phenomenon in this zone may be called **negative vital reactions**, since no such decrease in enzyme activity is observed in the wounds inflicted after death.
2. *Peripheral zone*: It surrounds the central zone, is 100-300 μ deep and has increasing enzyme activity. It represents adaptive

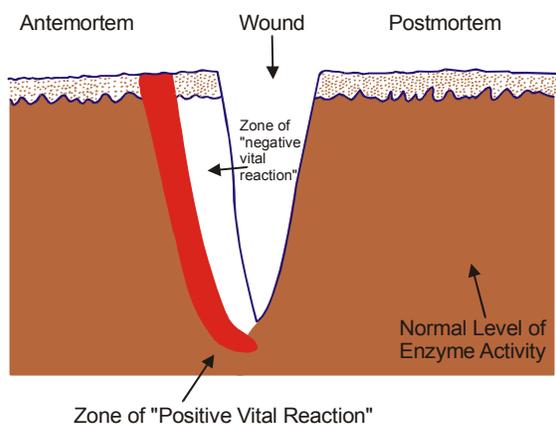


Figure 22.3: Difference between the enzyme activity in antemortem and postmortem wound

defense mechanisms of the local connective tissue cells as an enzymatic response to injury. The increase in enzyme activity in this zone is called **positive vital reaction**, since there are no such changes in post mortem wounds.

Histochemistry of An Antemortem Wound

Activity of adenosine triphosphatases and esterases increases as early as about one hour after injuries and the activity of aminopeptidases increases in about two hours whereas activity of acid and alkaline phosphatases increases in about four and eight hours respectively (Fig. 22.2). Histochemical vital reactions are recognizable several days after death.

Mucopolysaccharides in Wound Healing

Acid mucopolysaccharides disappear from vital wounds and reappear during healing process. They also disappear from vital bruises, abrasions and electric marks but remained present in the antemortem hanging and strangulation marks. This suggests that the alteration in vital wounds and bruises are mainly the consequence of haemorrhage into the connective tissue

Distribution of Histochemically Identified Substance in Two Main Components of Skin

RNA: By pyronin methyl green/ribonuclease method, in the epidermis, RNA is moderately intense in basal cells and becomes less so in the squamous cells showing inverse relationship to degree of keratinization. In skin wounds, a great increase in concentration of this substance is noted when there is active regeneration at the edges of a healing wound. It generally reaches the peak of activity when cells have bridged the central gap.

DNA: By Fielgen method it is shown in lower squamous and basal cells to comprise faint purple granules of uniform size normally and confine it to the nucleus.

Glycogen: By Periodic acid Schiff/diastase method, an inverse relationship is shown to the degree of maturity of the epidermal cells being aggregated in basal cell layer and virtually absent in keratinized epithelium.

Sulphydryl and disulphide group shows an inverse relationship to each other, the presence of S-S groups being noted when keratinization is almost complete.

The study of distribution of these components has not proved to be of any practical assistance in evaluation of wounds for forensic purposes. An increased reaction for RNA, glycogen and sulphydryl groups is noted in actively regenerating epithelium.

Connective Tissue Histochemistry

Fibroblasts

Fibroblasts show increased RNA content in the cytoplasm and prominent glycogen and metachromatic granules showing some alkaline phosphatase activity.

Mucopolysaccharides: Mucopolysaccharides disappear from vital bruises, abrasions and electric marks but remain in the antemortem hanging and strangulation marks that were suggested to be due to the consequence of haemorrhage into

connective tissue in case of vital wounds and bruises.

Fibrin: With Mallory's phototungstic acid haematoxylin and Lendrum's acid picric Mallory method, haemorrhages in human tissue are shown as:

- 4-12 hours—Network of fine fibrils.
- >24 hours—Coarse fibrils.
- >4 days—Small concentrated areas appeared.
- >2 weeks—Solid areas predominated.
- >1 month—Granular areas appeared among solid areas.
- At 4 months—Only granular appearance.

Platelets: Thrombosis has been recognized as an essential reaction to injury and was regarded by earlier writers as an unequivocal sign of antemortem infliction. Four stages are distinguished as platelet adherence, aggregation, degranulation and disruption. They are associated with formation of fibrin and platelet plug that is mixed fibrin red cell leukocyte mass and extension of thrombus. Agglutinated platelets arrest haemorrhage in normal skin by rapidly sealing the mouths of all cut vessels larger than capillaries.

Elastic tissue: Haematoxylin and eosin stains the elastic tissue; normal elastic fibers are pink and others are blue gray with pink granules. Toluidine stains as deep blue green and normal elastic fibers as colourless. From a microscopic examination of elastic fibers in skin, it is possible to differentiate between antemortem wounds where these are wave like and post mortem wounds when they are straight.

Pigments: Phagocytosis of red corpuscles is well marked after 48 hours; formation of hemosiderin after 24 hours and formation hematoidin with in some 80 days.

Disc electrophoresis: Distribution of esterases was compared using disc electrophoresis in the cathode area. Two of these fractions showed up more intensely in antemortem skin wounds compared with undamaged controls. No difference in the esterase pattern was distinguished in undamaged skin and postmortem wounds.

Biochemical Examination of Wounds

Although enzyme histochemical methods have considerably shortened the indeterminable period after wounding but the study of last hour before death is very challenging. Histamine and serotonin (5-hydroxytryptamine) are vasoactive amines known to participate in an acute inflammatory process especially in the earliest phase after injury. The minimal increase of free histamine content occurs within in 20-30 minutes and of serotonin in 10 minutes of injury. These additives have been conducted in experimental investigations of skin injuries as well as autopsy materials. About 2 gm of injured skin sample is removed and an equal size of control sample is taken from neighbouring intact skin of the corpse. The subcutaneous fat is removed from the samples before further processing and the free histamine and serotonin in the wound samples are compared with those of control samples.

Immunofluorescence Techniques

Fibrin in certain tissue lesions does not stain by standard techniques in the generally accepted manner. Sections from these fibrin clots were shown to react specifically with fluorescent-labeled rabbit antihuman fibrin antibodies. Also fluorescent antibody method was used to demonstrate fibrin in leucocytes in areas of fibrin accumulation. Acute inflammatory cellular response to fibrin at ultra structural level by electron microscopy and by fluorescent microscopy using rhodamine antifibrinogen in experimental work on dogs was studied and was noted that fibrin is either a network of heavy interweaving strands with orange fluorescence or less intensely stained delicate fibers. Postmortem fibrin cannot be distinguished with any certainty on morphological grounds from vital fibrin. A large proportion of postmortem subcutaneous haemorrhages undergo fibrinolysis, which destroys the formed fibrin networks with a day of the haemorrhage. Therefore well preserved fibrin networks found at autopsy performed two to

three days after death appear to point to the vital or agonal origin of a subcutaneous haemorrhage against its postmortem origin.

TORTURE

The Webster dictionary defines torture as 'anguish of body or mind; something that causes agony or pain, to punish, coerce or afford sadistic pleasure.

The oppression of the Nazi regimen during the second World War prompted the UNO to come out with Universal declaration of Human Rights (1948), followed by an International agreement of civil and political rights (1966) and a declaration on the protection of all persons from torture and other cruel, inhuman or degrading treatment or punishment (1975).

In 1975, World Medical Association adopted a declaration called **Declaration of Tokyo** that defined torture as, 'A deliberate, systemic or wanton infliction of physical or mental suffering by one or more persons acting alone or on the orders of any authority to force another person to yield information, to make a confession or for any other reason. This declaration clearly expressed that a doctor must in no way, for any reason take part in the practice of torture or other forms of cruel, inhuman or degrading procedures as the doctor's role is to alleviate the distress of his fellow persons and no motive whether personal, collective or political shall prevail against this higher purpose. Though this definition is considered by many as the most authoritative as far as doctors are concerned, it has few drawbacks, namely:

1. It is applicable only in content of detention and imprisonment and not in other situations where torture could take place.
2. There is no distinction as far as severity or aim of torture is concerned.
3. Unethical experimentation with human subjects has not been covered under this declaration.
4. It is implied that torture can be perpetrated only by others and not by self. No guidelines have been offered for self torture.

Classification of Torture

1. That done on the person by his consent.
2. That done by others (without consent) that can be further sub classified as that committed on an innocent person or on a person who is not innocent (criminals or terrorists).

Reasons for Torture

The reason for torturing a person varies from country to country and time to time depending on the availability of weapons, circumstances and the motive for the crime. In the earlier days, the basic purpose of the torture was to get information or confession; to spread terror in the society or a country; to destroy a personality; to take revenge and to get a testimony incriminating others.

Types of Torture

Torture can be physical or psychological.

Types of Injuries in Torture

Though virtually any type of injury can be inflicted deliberately to extract information, punish or degrade the victims, but the repetition of a particular injury, such as **tram line bruises** from beating, is suspicious. The following types of injuries are commonly produced in the victims of torture:

1. **Beating** is one of the most common forms of torture and can take various forms, varying both with the weapon used and the part of the body injured. The blows may be inflicted with a fist, foot, shoe or some weapons such as whip, a *lathi*, metal or wooden bars, clubs, batons, rifle butts or belts may be used. Many of these weapons produce a characteristic single or double edged linear bruise. The bruise may be line of confluent petechial haemorrhages or a continuous mark of red skin. The 'tram line' bruise is a double line of parallel marks with a pale unbruised zone between them, caused by the impact of a rectangular or circular-sectioned object. The bruising may be intradermal, when it reproduces the pattern

of the weapon well. If a leather whip with, for example, plaited thongs is struck against the skin, the pattern may be imprinted clearly on the skin. Buckles on belts and other recognizable artefacts may occasionally be useful in identifying the weapon. Where the bruising is deeper, no such pattern is likely. The skin may be broken causing abrasions or lacerations and if full thickness is breached, healing will cause scars, which may even be recognizable as 'tram lines'.

Repeated beating leaves multiple overlapping and criss-crossing marks that have a generally similar orientation that indicates that the attacker stood in a relatively fixed position to the victim. The use of multi-thonged whip will leave a series of marks with same orientation as above.

Back is the most common target but whipping and beating may be applied to the buttocks, thighs, front of chest, breasts and abdomen, lower legs, soles of feet and even perineum and genitals.

Beating of the soles of feet with canes or rods (**falanga**) is extremely painful and debilitating a torture rooted in antiquity. The tough tissues and thick fascial planes of the foot do not readily reveal bruising, though it may be found on deep dissection in victims who have been killed by some other means. If injuries were inflicted months before the examination, little or nothing may be found unless the skin was broken, when scarring will have taken place. Sometimes however, faint red lines will be seen in pale skinned people and hyperpigmentation along the lines of injury in the dark-skinned.

2. **Burns** are also commonly caused as a means of torture and they may either be the actual cause of death or visible as recent or scarred evidence of previous torture. All kinds of burns may be suffered such as from molten rubber dripped onto victims from motor tyres suspended overhead, hot irons applied to the skin, ignited kerosene soaked

rag wrapped around the limbs and numerous burns from cigarettes passed on to the skin. The findings of burning **motor tyre necklace** and shadow areas indicating direction of contact where molten liquids have been dripped from above onto a bound victim may be seen on examination of the victim.

3. **Cutting and stabbing wounds** are also quite common and may be caused by a variety of weapon but knives and bayonets are commonly used. The chest and upper arms are the favourite sites for infliction of injury. The old stab wounds from knives and bayonets may be recognizable as elliptical scars many months or years after the infliction.
4. **Blunt injuries** and clubbing are quite common, either to cause death or as form of abuse. The head is the most common target, but the legs and knees are also often struck. Blows on the back and sides are neck may be from due to damage to vertebrobasilar artery.
5. **Suffocation and drowning** are not commonly employed practices but non-fatal practices such as **submarining** (repeated dipping of the victim's head under water or even in foul liquid such as sewage water) is a well-known method of abuse. Such practices may cause death from drowning or if the victim survives, pneumonia may develop. Partial suffocation by enveloping the head in an opaque plastic bag is more a means of disorientation than physical torture but may eventually prove fatal.
6. **Electrical torture** is also quite commonly employed method of abuse. Either mains voltage of 110 or 240 volt is used, that can cause fatal cardiac arrhythmias and local burning. A magneto delivering high voltage is painful but not lethal and will not leave any significant marks over the skin owing to its low amperage. Electrical current may be applied anywhere on the body but the

genitals especially the penis and scrotum and the female nipples are the favoured sites.

7. **Injuries to ears** may occur as a result of **telefono** (repeated slapping of the sides of head and ears by open palms of the assailant). Very rarely injuries to the inner ear and rupture of tympanic membrane may be demonstrated on autopsy.
8. **Suspension:** In such cases abrasions, bruises and chafing marks may be found as ligature sites usually on legs, arms and sometimes genitals. It is a common method of torture.
9. **Shooting** is a common means of execution or non-fatal punishment but not a method of torture. **Knee-capping** where opponents and suspected traitors are shot either through knee joint or lower thigh, perpetrated in Northern Ireland.
10. **Sexual abuse** is commonly seen even multiple rapes in women that will leave some of the physical signs

Management of Torture Victims

First and foremost is the diagnosis of torture by taking a proper history, complete examination including the general physical examination, examination of the injuries and proper investigation including the radiological examination and bone scintigraphy. The treatment of the victim is an important aspect by providing the physical and psychological treatment simultaneously and treating the victim and his family. The victim should not be reminded of torture and proper rehabilitation is necessary.

Medico-legal and Ethical Aspects of Torture

1. Freedom from torture is among the human rights contained in the United Nations, Universal Declaration of Human Rights
2. Doctors are obliged by the Hippocratic Oath, not to use their professional knowledge in order to harm their patients.
3. Geneva Declaration of the World Medical Association reconfirmed this obligation and precludes any medical involvement in the

practice of torture.

In spite of this the doctors are at the risk of being involved in the practice of torture directly or indirectly;

1. As a humanist citizen.
2. As a participant in the process of torture by coercion by society, government or terrorists, or sometimes unknowingly becoming a part of it and with his own desire.
3. As victims of torture themselves e.g., Chile, Turkey.

Participation of Doctors in Torture

The doctors can participate in torture in various ways, namely:

1. Evaluating the victim's capacity to withstand torture
2. Supervising torture through provision of medical treatment, if complication occurs.
3. Providing professional knowledge and skills to the torturer.
4. Falsifying or deliberately omitting medical information when issuing health certificates, or autopsy reports.
5. Administering torture by directly participating in it.
6. Remaining silent in spite of the knowledge that abuse have taken place.

The most vulnerable doctors are military doctors, police and prison doctors, those in Government jobs and forensic medicine specialists. These experiences led to the adoption of a special declaration on the doctor's in torture at the World Medical Assembly in Tokyo in 1975, known as the '**Tokyo Declaration**'. These are

1. The doctor shall not countenance, condone or participate in the practice of torture or other forms of cruel in human or degrading procedures, whatever the offence of which the victim of such procedures is suspected, accused or guilty and whatever the victims' belief or motives and in all situations including armed conflict and civil strife.
2. The doctor shall not provide any premises, instruments, substances or knowledge to

facilitate the practice of torture or other forms of cruel, inhuman or degrading treatment or to diminish the ability of the victim to resist such treatment

3. The doctor shall not be present during any procedure in which torture or other forms of cruel, inhuman or degrading treatment is used or threatened.
4. A doctor must have completed clinical independence in deciding upon the case of a person for which he or she is medically responsible.
5. The doctor shall in all circumstances be bound to alleviate the distress of the fellowmen and no motive whether personal, collective or political shall prevail against this higher purpose.
6. Where a prisoner refuses nourishment and is considered by the doctor as capable of forming an unimpaired and rational judgment. Concerning the consequences of such voluntary refusal of nourishment he or she shall not be fed artificially. The decision as to capacity of the prisoner to form such a judgment should be confirmed by at least one other independent doctor, the consequences of the refusal of nourishment shall be explained by the doctor to the prisoner.
7. The WMA will support and should encourage the international community, the national medical associations and the fellow doctors to support the doctor and his/her family in the face of threats or reprisals resulting from a refusal to condone the use of torture or other forms of cruel inhuman or degrading.

Statement by the International Union of Psychological Science (1976)

'No psychologist in the exercise of his/her professional functions should accept instructions or motivations that are inspired by considerations that are foreign to the profession'. It requests each member of society to make certain that it has enacted a code of ethics and to take those actions

required by its code against any member guilty of such abuses against human rights.

Principles of Medical Ethics (United Nations 1982) relevant to the role of health personnel particularly physicians in the protection of prisoners and detainees against torture and other cruel, in human or degrading treatment and punishment as given by United Nations in 1982 are the following:

1. Health personnel particularly charged with the medical cases of prisoners and detainees have a duty to provide them with protection of their physical and mental health and standard as is afforded to those who are not detained or imprisoned.
2. It is a gross contravention of medical ethics as well as an offence under applicable international instruments for health personnel particularly physicians to engage actively or passively in acts which constitute participation in complicity in incitement to or attempts to commit torture or other cruel, in human or degrading treatment or punishment.
3. It is contravention of medical ethics for health personnel particularly physicians to be involved in any professional relationship with prisoners or detainees, the purpose of which is not solely to evaluate, protect or improve their physical and mental health
4. It is a contravention of medical ethics for health personnel particularly physicians
 - i. To apply their knowledge and skills in order to assist in the interrogation of prisoners and detainees in a manner that may adversely affect the physical or mental health or condition of such prisoners or detainees and which is not in accordance with the relevant international instruments
 - ii. To certify, or to participate in the certification of fitness of prisoners or detainees for any form of treatment or punishment that may adversely affect their physical or mental health and which is not in accordance with the relevant international

- instruments, or to participate in any way in the infliction of any such treatment or punishment which is not in accordance with the relevant international instruments
5. It is a contravention of medical ethics for health personnel particularly physicians to participate in any procedure for restraining a person or detainee unless such a procedure is determined in accordance with purely medical criteria as being necessary and for the protection of the physical or mental health or the safety of the prisoners or detainee himself or his fellow prisoners or detainees or of his guardians and it presents no hazard to his mental or physical health.
 6. There may be no derogation from the foregoing principle on any ground whatsoever including public emergency.

Declaration of Hawaii World Psychiatric Association (1983)

The declaration states that “The psychiatrist must never use his professional possibilities to violate the dignity or human rights of any individual group”. If the patient or some third party demands actions contrary to scientific knowledge or ethical principles the psychiatrist must refuse to cooperate.

World Conference on Human Rights (1993)

The conference stated that “Freedom from torture is a right which must be protected under all circumstances, including in times of internal or international disturbance or armed conflicts”.

The Human Rights Act (1993)

In the United States, the Human Rights Act has the principal aim to curb violation of any of the rights, including safety from torture of the individuals of this country.

The victim of torture may either suffer from injuries or may die because of injuries and therefore such cases should be carefully and thoroughly

examined, reported to the police or the magistrate and treated. A proper examination may help in establishing or disproving the charge of torture both during the examination of injury and autopsy.

S. 330 & 331 of the Indian Penal Code deals with crime and punishments of voluntarily causing hurt and grievous hurt for the purpose of extorting confession or any information which may lead to the detection of an offence or misconduct.

The following are some of the steps taken to prevent torture

1. To abolish torture a campaign amongst the medical community against torture must be devised and how it is contrary to medical ethics.
2. Doctors should document evidence of torture and conduct autopsies in a fair manner and help the victims to obtain redress. It is the ethical obligation of all doctors to report cases of torture.
3. The medical association should issue official directive condemning and banning these practices and prescribe strict ethical rules and guidelines in this regard and strict measures should be taken for their violation
4. The law should be amended so that every accused person, on arrest should be medically examined and injuries of any on his person should be entered in to the record.
5. Training about torture, its consequences, treatment and rehabilitation of torture victims and medical ethics should form part of the medical curriculum especially at the undergraduate level.
6. Doctors involved in the cover-up operations should be made personally liable and proceeded against **section 166 of I.P.C.**. The responsibility of filing such charges should be taken up by the Indian Medical Association.
7. State medical councils should be properly activated to enforce ethics within the profession.

CUSTODIAL DEATHS

Custodial deaths are those occurring while a person is either in the custody of police or is an inmate of a prison. A meticulous autopsy is needed to confirm or dispel the allegations that an act of commission or omission on the part of custodians has led to, or contributed to, the death. It is sometimes helpful to the pathologist to visit the scene of the death, especially if it was caused by hanging or some form of mechanical trauma.

Situations under which death may occur in custody: The death may occur during or soon after the arrest by the police officers during physical struggle, especially when the police officers are attempting to control or overpower the resisting offender. The offender may also threaten the police officers with a knife, gun or blunt weapon and the police have to subdue him either by sheer physical force, truncheons or riot sticks or by the use of firearms. The arrest of a drunken offender poses many problems.

Causes of Deaths During Arrest

1. *Traumatic asphyxia:* When several policemen fall upon a resisting offender to overpower him.
2. *Arm-locks or neck holds:* Applied by police officers to resisting persons are other causes of deaths during arrest. The arm lock is applied either from behind or with the head of the offender tucked under the police officer's arm against the waist. The dangers are compression of the front and sides of the neck and death can occur either from reflex cardiac arrest or cerebral ischemia during carotid compression or asphyxia from airway obstruction, though the latter is unlikely to be the sole mechanism.
3. *Blunt injury* may occur from the use of fist, arm or leg and by use of a weapon such as a truncheon, riot stick or pistol butt. Any type of blunt injury may be received. Head injuries may occur during a scuffle from falls either against the ground, or against a wall or other obstruction. A heavy punch in the face may cause nasopharyngeal bleeding that can block the air-

passages, especially in a person affected by alcohol. A blow on the sides of the neck can cause reflex cardiac arrest or a subarachnoid haemorrhage from vertebrobasilar vascular damage. A backward blow from the point of an elbow can be damaging, if it strikes the face, neck or abdomen. Kicking and stamping are unusual, but not uncommon in custodial deaths. A blow in the abdomen can be fatal if delivered with sufficient force. Though in an adult a fist blow is not very likely to cause serious damage, it can in a very younger person. The use of the elbow, knee or a head butt can deliver extreme force, especially from a fit, muscular police officer as well as from the offender.

4. *Alcohol* is a frequent cause of death in custody and also it is major factor in provoking aggression and violent resistance. When blood alcohol levels rise to above 350mg/100mL there is an increasing risk of coma and central respiratory depression. At lower blood alcohol levels there is still the risk of aspiration of vomitus and choking on gastric contents. At autopsy, this aspiration should be distinguished from the common agonal phenomenon. Where an otherwise healthy person dies with a high blood alcohol concentration in these circumstances, then gross blocking of the trachea and bronchi with vomit can be accepted as the cause of death. Alcohol also attributes to accidents during custody, especially head injuries resulting from falls on the ground, falls down steps and stairs. Falls onto a hard surface are often on the occiput and the frequent finding of frontal and temporal contrecoup brain damage at autopsy is good evidence of a deceleration injury rather than an assault with a weapon. Some falls may occur during custody or in transit from the site of arrest to the police station and the police is often blamed for allowing or causing the injury. Drugs such as amphetamine, cocaine or hallucinogen may also lead to physical damage. Drugs are commonly available within prisons and overdose and hypersensitivity deaths are occasionally seen.

5. *Suicides* in custody are quite common. Most police forces deprive the prisoner of any objects that could be used to hang himself in the cells such as belts, braces, cord or even bootlaces. In addition, the police cell may be specifically designed to avoid any convenient suspension points, such as bars, hooks, internal door handles. Nevertheless, prisoners regularly manage to find some means of killing themselves as hanging can be successfully accomplished by traction on the neck at low levels and need not occur at high suspension points, so prisoners have killed themselves by attaching ligatures to bed-heads, chairs and other unlikely objects in the cell. At autopsy homicidal hanging should be ruled out by the absence of signs of struggle and other injuries resulting in death. The person at times may be hanged after killing to simulate suicide. The situation is fully assessed preferably by a visit to the scene with the body at the site.
6. Deaths may occur from purely natural causes, usually of cardiovascular origin and no objective proof can be provided when the emotional and physical upset of being arrested and confinement may have affected the blood pressure and heart rate sufficiently, by an adrenaline response, to have precipitated an acute cardiac crisis in the presence of severe preexisting disease. The presence of diabetes, epilepsy, asthma or other diseases that can potentially cause sudden or unexpected death should be sought for by medical history and autopsy appearances.
4. Such types of death are usually involved with the non-cooperating attitude of the authorities that are hostile to impartial inquiry.
5. The cases are handed over to the responsible authorities such as Red cross, Amnesty International, Physicians for human rights.
6. There is usually delay in the process before a victim is being brought for examination, the injuries may have healed and body may have decomposed. Body is sometimes disposed off early.
7. The causes of deaths in torture or custodial deaths may be sepsis, haemorrhage, internal organ injury and even sheer exhaustion.

Recommendations of the National Human Rights Commission in custodial deaths and torture

1. The commission has required all District Magistrates/ Superintendents of the Police to report any instance of custodial death or rape directly to the commission within 24 hours of occurrence. Failure to send such reports, it has been emphasized would lead to a presumption by the commission that an effort was being made to suppress the occurrence.
 2. The commission believes that the country must act deliberately and decisively to end custodial violence and signify to itself and to the World that it will not countenance brutality in custody.
 3. The chairperson made clear that a permissive approach to the use of third degree methods in investigation led to serious violations of the right of the citizens of India and that it was wrong to believe that such practices were necessary for the successful investigation of cases and the matter continues to be pursued.
 4. The commission is of the view that a recommendation should be made for the insertion of a Section 114(B) in the I.E.A.1872 to introduce a rebuttable presumption that
1. All deaths in custody should be examined with care to ensure that the guardians are cleared of any suspicion of ill-treatment.
 2. The torture and custodial deaths are confirmed by circumstantial and corroborative evidence.
 3. The victim is usually in detention in or some form of custody.

Investigations of Torture and Custodial Deaths

injuries sustained by a person in police custody may be presumed to have been caused by a police officer.

5. The commission supports the recommendation of the Indian Law commission that section 197 Cr.P.C. to be amended to obviate the necessity of governmental sanction for the prosecution of police officer where a prima facie case has been established in an enquiry conducted by a session's judge, of the commission of a custodial offence.
6. The commission also endorses the view that there should be a mandatory enquiry by a session's judge, in each case of custodial death, rape or grievous hurt.
7. Commission believes that the arrested person being held in the custody is entitled, if he so requests to have a friend, relative or any other person who is known to him, or likely to take an interest in his welfare, to be informed that he has been arrested and told of where he has been detained.
8. The compensation due to the next of kin of those who have died in custody should be the liability of not just the state government but of the offending police officials themselves.
9. The commission gathered detailed information from all the states regarding the manner in which postmortem examination were being conducted, it was found to be substantial time gap between the PM examination and writing of the report; facilities in many mortuaries were abysmal and there is lack of trained and qualified personnel.
10. The chairperson of the commission recommended **all postmortem exami-**

nation in respect of deaths in police or jail custody to be video-filmed and the cassettes sent to the commission together with the written reports of postmortem examination.

Delhi Declaration on torture (Courtesy Justice MR V.S.Malinath NHRC, 1999)

The symposium was held in Delhi that noted with concern that torture continues to be prevalent in many countries. Wars and situations of armed conflicts within and between states have in recent years given rise to the gravest human rights violations including torture. Health professional in many countries still find themselves under duress to condone or cover up torture in violation of their codes of ethics and the UN principles of medical ethics. The symposium urged the action to be taken at national level and the action to be taken by United Nations against torture.

Long Questions

1. How can you classify injuries? Discuss grievous hurt. How can you estimate the time of injuries?
2. Discuss the autopsy findings in a case of death due to torture.

Short Questions

1. Torture
2. NHRC guidelines for custodial deaths
3. Grievous hurt.
4. Simple injury
5. Section 44 I.P.C.
6. Falanga
7. Thromboembolism.

MULTIPLE CHOICE QUESTIONS

1. **All of the following are grievous injury except:**
 - A. Tooth fall
 - B. Avulsion of nail
 - C. Facial injury
 - D. Emasculation
2. **All are grievous hurts except:**
 - A. Dislocated shoulder
 - B. Incised wound on abdominal wall without peritoneal injury
 - C. Nasal bone fracture
 - D. Permanent disfigurement of face
3. **Which of the following is a grievous injury?**
 - A. Extensive lacerated wound on the forehead
 - B. 6 cm x 4 cm laceration present on the scalp
 - C. 6 cm x 2 cm bruise present on extensor aspect of forearm
 - D. Abrasion present at the back of trunk
4. **Which of the following is a Grievous injury?**
 - A. Simple fracture of radius
 - B. Sharp injury of abdomen without penetration of peritoneum
 - C. Bruises over arms and back due to lathi
 - D. Lacerated wound over the thigh with exposure of muscles
5. **Which of the following is grievous injury?**
 - A. Fall of tooth
 - B. 7 day's absence from work
 - C. Clean incised wound on face
 - D. Lacerated wound scalp
6. **A person beaten up but having no fracture or dislocation and is hospitalised for 20 days is considered grievously injured:**
 - A. Even if there is no other injury
 - B. If during the twenty days he is in severe body pain or unable to follow his ordinary pursuits
 - C. If during the first ten days he is in severe body pain but later on could follow his ordinary pursuits
 - D. Even if has sharp injury over abdomen without penetration of peritoneum
7. **Which of the following indicates that the wound in a hospitalized woman typist hit by 'lathis' admitted for 20 days was grievous?**
 - A. She is not able to recognize
 - B. Wound does not heal completely
 - C. Incised wound over scalp
 - D. She is not able to do her daily routines
8. **Grievous injury is:**
 - A. Disfiguration of face due to injury
 - B. 14 days hospital stay
 - C. 20 days hospital stay
 - D. Permanent impairment of hearing
9. **Which of the following is a grievous injury under IPC section 320?**
 - A. Emasculation
 - B. Privation of joint
 - C. Fracture of bones
 - D. All of the above
10. **The exact age of the abrasion can be assessed by:**
 - A. Histological method
 - B. Histochemical method
 - C. Naked eye examination
 - D. None of the above
11. **Which of the following is not a feature of antemortem wounds?**
 - A. Copious haemorrhage
 - B. Marks of spouting of blood are present
 - C. Deep staining of edges & cellular tissue
 - D. Wound edges are closely approximated
 - E. Leucocytic infiltration along the margin of the wound with edema of wound margins
12. **One of the following is Not a delayed cause of death from injury:**
 - A. Tetanus
 - B. Fat Embolism
 - C. Thromboembolism
 - D. Air Embolism

1 B 2 B 3 A 4 A 5 A 6 B 7 D 8 A
9 D 10 B 11 D 12 A

Asphyxial Deaths

GORDON'S CLASSIFICATION OF DEATH

Gordon in 1944 postulated that vital functions of the body depend upon the availability and utilization of oxygen by the body tissues and tissue anoxia finally leads to cardiac failure and death. It is a classification that lays stress on the pathogenesis in different forms of death of medicolegal importance. It is brought about in following four ways:

1. *Anoxic Anoxia*: Defective oxygenation of the blood in the lungs
 - By obstruction to air passage—suffocation, smothering, overlaying
 - By obstruction to the passage of air down to the respiratory tract—drowning, choking, throttling, hanging and strangulation
 - Excessive compression of the chest and abdominal wall
 - Primary cessation of respiratory movements causing respiratory failure—in narcotic poisoning, electrical injury
 - Inhalation of carbon dioxide and carbon monoxide gases
2. *Anaemic anoxia*: Reduced oxygen carrying capacity of the blood—acute poisoning due to CO, Chlorates, nitrites
3. *Histotoxic Anoxia*: Depression in the oxidative process in the tissues—Hydrocyanic Acid poisoning
4. *Stagnant Anoxia*: Insufficient circulation of the blood in the tissue—Traumatic shock, heat stroke, acute Irritant and corrosive poisoning.

The term asphyxia is commonly applied to a variety of conditions in which interference with respiratory exchange occurs to a greater or lesser degree. Many of these conditions vary so greatly in their physiological mechanisms and in their pathological appearances, they present that the use of the term is best avoided, whenever possible. It is usual to divide the effects of asphyxia in to a number of stages:

1. Stage of inspiratory dyspnoea with deep and forceful respiration, cyanosis lasting for a minute or so.
2. Stage of expiratory dyspnoea with spasmodic efforts at expiration. During this stage, the consciousness is lost, pupils are dilated and blood Pressure is raised.
3. There is fall of blood pressure, pulse is increased and spontaneous defecation occurs. The erection of penis and ejaculation of semen may occur.
4. Respiratory movements cease except for terminal irregular occasional respiration. Heart continues to beat for 10-15 minutes.

MECHANICAL ASPHYXIA

Mechanical asphyxia is a term applied to circumstances in which mechanical interferences to the respiration occurs that either

- a. Impedes access of air to lungs, or
- b. Reduces blood supply to head and neck or
- c. Causes sudden cardiac arrest due to stimulation of carotid sinus resulting vagal reflex mechanism

It is doubtful if there are any constant post mortem changes produced by the direct effect of anoxia upon the tissues except the appearance of cyanosis. There are abnormal appearances of respiratory obstruction in the form of local effects of the constricting agent, raised intravascular pressure and terminal heart failure.

Classical Signs of Asphyxia

The classical signs of asphyxia are nonspecific and can occur in deaths from other causes.

1. *Cyanosis (Greek 'dark blue')*: Significance of cyanosis in a cadaver must be evaluated very critically. Cyanosis depends upon the absolute quantity of oxyhaemoglobin and reduced Hemoglobin in red cells. The normal pink colour of well-oxygenated blood may change to purple or blue when oxygen is lacking. Cutaneous cyanosis depends upon absolute amount of reduced haemoglobin, rather than amount of reduced oxy-hemoglobin. There must be at least 5 gm% of reduced hemoglobin before cyanosis becomes evident, irrespective of total amount of hemoglobin. In constriction of neck, cyanosis almost invariably follows congestion of face, as venous blood contains much reduced Hemoglobin. After perfusion to head and trunk, it is directed back to tissues and becomes bluer as blood accumulates. When the airway becomes blocked, then impaired oxygenation in the lungs leads to a diminution in the oxygen content of tissues, which leads to darkening of organs and tissues and accentuates the cyanosis of face.

This depends upon complete or substantial obstruction of the airway or restriction of respiratory excursions of the chest. If the body is examined within few hours of death, presence of intense cyanosis is of some significance. The loss of oxygen of cadaveric blood is very variable, but certainly, after 24 hours the appearance of cyanosis may be due entirely to postmortem changes. Absence of cyanosis within few hours of death does not necessarily signify that cyanosis was not present at death.

Terminal cyanosis is common in many forms of death and its presence is seldom of much use in the diagnosis of respiratory obstruction.

2. *Congestion*: Visceral congestion is due to capillo-venous congestion and is the result of susceptibility of capillaries to hypoxia. This results in dilatation of capillaries with stasis of blood in the dilated capillaries and the venules. Systemic and pulmonary congestion and dilatation of right side heart are signs of asphyxial death. Their significance will be treated with greater reserve than the presence of cyanosis. There is considerable redistribution of blood in the cadaver under the influence of gravity and to some extent by rigor mortis. The effect of these factors also depends upon the fluidity of blood after death.

Right ventricular dilatation and pulmonary congestion are common to many forms of death, by no means specific for death from interference with respiration. Conversely, when death is due to blockade of airway, one may fail to find this phenomenon. Furthermore, the size of heart chambers can alter with rigor mortis and so the amount of blood in the pulmonary circulation.

Congestion immediately above a mark on the neck may be of great significance in the diagnosis of the cause of the mark. The time and force of infliction of pressure on the neck but general observation on capillo-venous congestion is of little value

3. *Fluidity of blood*; At one time, it was thought that fluid blood in the cadaver was an indication of asphyxia. The presence of fibrinolytic enzymes in the cadaver has been established, so fluidity depends upon fibrinolysin and the amount of fibrinolysin depends upon the rapidity of death rather than the nature.

4. *Pulmonary edema*: Some degree of pulmonary edema is common but not invariable in deaths associated with anoxia. It is debatable whether it is due to heart failure or increased capillary permeability. It has little or no value in the diagnosis of death due to respiratory obstruction.

tion. It is common in many modes of death. In anoxial deaths edema of anything more than a minor degree indicates that death has not been very rapid. Lungs should be weighed properly to know the extent of edema.

5. **Pulmonary hemorrhages:** Large submucosal hemorrhages in pharynx especially over the dorsum of cricoid due to direct trauma of this part against anterior surface of spine. Submucous venous plexus at this site is made of very large thin walled vessels and rupture of these vessels due to severe venous congestion. These haemorrhages are also found in variety of deaths unconnected with trauma.
6. **Petechial hemorrhages:** In the year 1866, Tardieu a French police surgeon described it and found beneath the pleura and pericardium. He was of the opinion that these hemorrhages were the pathogenesis of mechanical asphyxia. Later on, these hemorrhages were known as Tardieu's spots. Injuries to the walls of the epithelium may result in the development of petechial haemorrhages (Fig. 23.1). Until 1944, Lime and Gordon questioned their significance as they had found it in many forms of death. In 1953 Gordon, Trevor and Trice also found it in secondary shock and natural cases. In 1955,



Figure 23.1: Petechial hemorrhages over the heart

Gordon and Mansfield questioned their ante mortem origin as they commonly developed after death and were found during autopsy. In 1955, Shapiro again opined that some of these are spontaneous post mortem artefacts and disappeared in a short period. This disappearance was confirmed and depicted in the year 1981 by Zaini and Knight who considered them to diminish when intravenous pressure is reduced in major pulmonary vessels at autopsy. They found that it was difficult to distinguish between true and false petechiae by naked eye examination. Only one third of these haemorrhages are true and two thirds are false. These false hemorrhages were due to intra pleural venous channels air blisters, thickened pleural plaques and dust pigments. They are mostly unrelated to mechanical asphyxia. Tardieu spots develop from:

- i. Increased venous stasis causing congestion leading to increased pressure causing rupture of the vessels.
 - ii. Increased permeability due to hypoxia.
- Tardieu spots are usually pinhead, but vary in size and shape. They may be scanty detectable under low magnification or may be numerous like measles rash involving the skin of the face and eyelids, beneath the conjunctiva etc. These are most prominent in the visceral pleura and epicardium. Examination for evidence of injury and severity should be done.

Stasis Haemorrhages: These hemorrhages may be seen beneath the mucosa of larynx in the subglottic space and above the level of constriction of the ligature as in homicidal strangulation. venous stasis occurs leading to rupture of the capillaries.

Differential Diagnosis of Petechial Haemorrhages

1. Coronary deaths
2. In terminal stages of asphyxia.
3. Diseases: The hemorrhages found in diseases are small with greater distribution and more in number. Blood may reveal the underlying cause but laboratory findings may be misleading.

Classification of Asphyxial Deaths

1. Hanging
 - i. Mode of hanging
 - Judicial
 - Sexual (Autoerotic)
 - Lynching (Homicidal hanging).
 - ii. Manner of hanging
 - Suicidal hanging
 - Homicidal hanging
 - Accidental hanging
 - iii. Position of noose
 -
 - Typical hanging
 - Atypical hanging
 - iv. Position of feet
 - Complete hanging
 - Partial hanging
 - Partial standing
 - Sitting
 - Reclining (Kneeling position).
2. Strangulation
 - i. Ligature strangulation
 - ii. Manual strangulation (throttling)
 - iii. Mugging
 - iv. Garroting
 - v. Bansdola
 - vi. Palmer strangulation
 - vii. Bend of knee/ elbow
 - viii. Foot strangulation.
3. Suffocation
 - i. Smothering
 - Overlaying
 - Burking
 - ii. Gagging
 - iii. Choking
 - iv. Café Coronary
 - v. Traumatic Asphyxia
4. Drowning
 - i. Dry drowning
 - ii. Wet drowning
 - iii. Secondary drowning
 - iv. Immersion syndrome.

Sudden Cardiac Arrest

There are cases where these findings like petechial haemorrhages, cyanosis, congestion etc. are present but death is rapid and diagnosed as sudden cardiac arrest like that in café coronary. In Knight's experience, 50% of his cases where death occurred from mechanical asphyxia, cause was sudden cardiac arrest, as the cyanosis, haemorrhages or congestions were absent. These cases were deaths resulting from karate blows to the neck, mugging and some cases of drowning. Sudden death might occur due to cardiac arrest, accurate history of the circumstances of the death is important. Sudden collapse and loss of consciousness is the outstanding feature. Heartbeat or breathing may continue for 15-30 sec and there might be atrial fibrillation. An important factor is evidence of surprise, emotion, apprehension or fright. Alcoholic intoxication is commonly there. If no injury or slight injury is present over the body, the diagnosis of acute cardiac arrest is made.

HANGING

Hanging is a form of asphyxial death, which is caused by the suspension of the body by a ligature that encircles the neck, the constricting force being the weight of the body. Asphyxia in hanging is secondary to compression or constriction of the neck structures by a noose or other constricting band tightened by the weight of the body. There may be either complete or incomplete (partial) suspension of the body

Types of Hanging

Complete hanging: This is the type of hanging when the constricting force is the weight of the body as the body is fully suspended in air.

Partial (Incomplete) hanging: In this type of hanging, the constricting force is the weight of the head (about 5-6 Kg) and the lower part of the body such as toes, feet, knees or buttocks touches the ground (Fig. 23.2).



Figure 23.2: Partial hanging

Virtually all hangings are suicidal and in India, it is quite popular method of suicide more common amongst males.

Ligature Mark

On the neck, it will be a furrow (ligature mark). This furrow, as a rule, does not completely encircle the neck, but slants upwards, towards the knot, fading out at the point of suspension that is the knot. If the knot is under the chin, the site of the knot may be indicated by an abrasion or indentation beneath the chin. The clarity and configuration of the ligature mark depends upon the material used. The ligature mark produces a groove or furrow, which may be pale, yellowish or yellow brown and look like parchment due to drying of the abraded skin. It is usually inverted V shaped with the apex of the V at the knot. Impression of the knot may be found, depending upon its position. Along the edges of the depression, a thin line of congestion or haemorrhage will be seen above and below the groove at some point, usually

the deepest. Ecchymosis and slight abrasion in the groove are rare. Ecchymosis alone has no significance but it is important to establish whether it is antemortem or postmortem. Abrasions with haemorrhage are strongly suggestive of suspension taking place during life.

Position of ligature mark: In 80% cases, ligature mark is present above the thyroid cartilage, in 15% at the level and in 5% it is present below the thyroid cartilage (common in partial hanging). The width of the groove is about or slightly less than the width of the ligature material. A definite pattern may be seen but when fresh, the ligature mark is less clear. After drying it becomes well marked (Figs 23.3 to 23.4). There may not be any visible mark due to beard, long hair, clothes intervening between the ligature material and the neck. Loop of soft material, towel, and scarf may



Figure 23.3: A case of hanging with ligature material *in situ*.



Figure 23.4: A case of hanging same as fig. 23.3 with ligature mark

not produce a ligature mark. In partial hanging there may be little mark or not at all. Decomposition obliterates ligature mark. It may disappear after several hours when the loop is removed.

On *microscopical examination*, in case of antemortem hanging signs of vital reaction are present in the neck structures. The absence of vital reaction however does not exclude an antemortem hanging.

The appearance of the furrow over the neck varies with the following factors:

1. *Type of Ligature Material*: In hanging, a simple slipknot type of noose is typically used. The noose is constructed from anything that is handy. Most common are ropes, electrical cords, wires, strings, belts, *dupatta*, sari, and any piece of cloth or any other material that is available at that moment. Even in jails or prisons, the convict may use his own clothes to hang. Occasionally to prevent change of mind, the individual may tie his hands. The usual point of suspension is the side of the neck, followed by the back or front. At the time of suspension, the noose typically slips above the larynx, catching under the chin. A rope will give a deep, well-demarcated, distinct furrow, often with a mirror image impression of the weave of the rope on the skin. This furrow initially has a pale yellow parchment appearance, with a congested rim. With time, the furrow dries out and becomes dark brown. If the ligature material is a soft material, the ligature mark may be poorly defined, pale and devoid of bruises and abrasions. In some cases, the lower margin of the groove (ligature mark) is pale with upper margin red due to postmortem congestion of vessels. A soft broad cloth such as a towel, *dhoti* or *lungi* may show no ligature mark over the neck. In majority of suicidal hangings, the ligature consists of a single loop and mark. If the noose is a belt, it will produce two parallel ligature marks, as the upper and lower edges of the belt will dig



Figure 23.5: A case of hanging showing V-shaped, brownish, parchmentised like ligature mark

into the skin. Less commonly, ligature consists of two loops (Fig. 23.5). This results in two furrows which may be parallel, overlap at points or follow two completely different paths, for example, one oblique and other horizontal. The two loops may pinch the skin between them, producing a haemorrhagic strip of skin. If a soft noose is used and the body is brought down by cutting the ligature shortly after death, no marks of ligature may be present on the skin. The groove will be narrow, deep and distinct when a thin and hard ligature is used. The mark becomes more prominent when it has been suspended for long. When the ligature is broad such as *dupatta*, strip of cloth, *sari*, the furrow will be broad and shallow.

2. *Point of suspension*: Usually, the ligature mark is deepest opposite the point of suspension. In addition, rarely there may be scratch abrasions above and below the ligature when the victim tries to undo the noose during terminal stages.
3. *Type of hanging*: When the hanging is complete, the ligature mark is more prominent while in partial hanging it is less prominent.
4. *Tightness of ligature*: When the ligature is tightly present around the neck, the mark is.

5. *Time of suspension*: The longer the body remains suspended the deeper is the ligature mark.
6. *Position of the knot*: The main force to the neck is applied, opposite to the point of suspension. When the knot is present over the occipital region (Typical hanging) the front of the neck shows a prominent ligature mark.
7. *Slipping of ligature* may cause a double impression of a single turn of ligature material.
8. *Knot*: Usually a simple slipknot is made to produce a running noose fixed by granny or reef knot. At times simple loop is used. It is placed either on sides or over occiput, but rarely under chin. Knot is higher than the ligature. Removal of the noose is done by cutting the noose away from the knot and ends are tied with string/wire.

In most hangings, the face is pale and tongue is protruding and is black due to drying except in partially suspended individuals where the noose is tightened only by the weight of the head or the torso. In these instances while venous drainage is completely occluded, the blood supply of the vertebral arteries continues, in part producing congestion of the face and petechiae. Blood will pool in the dependent areas of the body, usually the forearms, hands and legs secondary to gravity. With time punctate haemorrhages, Tardieu spots due to hydrostatic rupture of the vessels will be seen.

Prior to the removal of the noose from the neck, it should be described in detail as to the nature and composition, width, mode of application, location and type of knot. After removal of the noose, the ligature mark on the neck should be described in detail. One should describe the location of ligature mark over the neck in relation to anatomical landmark direction of the furrow as obliquely upward or horizontal, continuous or interrupted, its colour and dimensions and whether there is distinct imprint of the pattern of the ligature over the neck.

Causes of Death

Death in hanging is due to compression of the blood vessels of the neck such that insufficient amount of oxygenated blood reaches the brain. Obstruction of the airway can also occur and is due to either compression of the trachea or, when the noose is above the larynx, elevation and posterior displacement of the tongue and floor of the mouth. Blockage or compression of the air passages is not necessary to cause death in hanging. A number of individuals have hanged themselves with the noose above the larynx and a permanent tracheostomy opening below. . As only a small amount of pressure is sufficient to occlude carotid arteries, quite commonly one can hang oneself in sitting, kneeling or lying down position. The weight of the head (10-12 lb) against a noose is sufficient to occlude the carotid arteries and cause death. Fracture of the neck plays virtually no role in nonjudicial hangings. It is extremely rare and is usually seen only in individuals with advanced degenerative diseases of cervical spine such as osteoarthritis, accompanying complete suspension of the body, a sudden drop, and frequently obesity or very old age

Causes of Immediate Death

In hanging immediate death can be caused by the following mechanisms.

1. *Asphyxia*: Ligature forces the root of tongue against the post wall of pharynx, and folds the epiglottis over the entrance of Larynx to block the airway. A weight of 15 Kg (33 lb) is required to compress the trachea to cause asphyxia.
2. *Venous congestion*: Jugular venous cerebral circulation can be stopped with a pressure of 2 Kg (4.4 lb) on the neck
3. *Cerebral anemia*: Carotid arteries require a pressure of 4-5 Kg (11 lb) to stop the flow of blood in them while vertebral arteries require 30 Kg (66 lb) of pressure
4. *Reflex vagal inhibition*: Vagal sheath or carotid bodies when compressed can get stimulated

and cause vagal inhibition of the heart causing it to stop.

5. Fracture dislocation of cervical vertebral, leading to injury to the spinal cord, as a result there is ascending edema that affects the vital centers in the medulla.

Causes of Delayed Death

In hanging delayed death occur due to the following reasons:

1. Aspiration pneumonia
2. Infections
3. Hypoxic encephalopathy
4. Edema of lungs
5. Encephalitis
6. Cerebral abscess.

Autopsy Findings

External

General Findings: Typical asphyxial signs are present in about 50-60% of all hangings.

- 1 Neck may be stretched and elongated and rarely may be severed from the body if the weight of the body is too heavy and the fall is from a greater height.
- 2 Head is bent opposite to the knot.
- 3 Face is usually pale but at times it may be congested and swollen. Swelling disappears when the ligature is cut down.
- 4 Petechiae on the skin and conjunctiva are present.
- 5 Signs of Asphyxia are more marked when the noose is high up in the throat. If only veins are obstructed there is engorgement of head and neck. The eyes protruded and firm due to congestion or may be closed and the pupil is dilated. The tongue is swollen, coming out between the lips (Fig. 23.7). Tip may be black due to drying and may be clenched between the teeth. There is dribbling of saliva from the angle of mouth, which is an indicator of antemortem hanging. The salivary stain may dribble over to the face, neck and sometimes chest (Figs 23.7 and 23.8). It is caused due to the stimulation of salivary glands by the ligature.



Figure 23.7: A case of hanging showing tongue clenched between teeth and salivary stain over the chest



Figure 23.8: Salivary stains over the chest in hanging

6. Slight hemorrhage or bloody froth is sometimes seen caused by the congestion in the lungs and the pulmonary edema. In the head sometimes hemorrhages are present due to rupture of engorged vessels. In the middle ear hemorrhages may be seen due to rupture of the small blood vessels. The penis may show semen drops coming out of the urethral meatus.
- 7 Lower limbs show hypostasis (Figs 23.9 and 23.10) due to the prolonged hanging posture and are an indicator of the duration of hanging



Figure 23.9: Hypostasis in lower limb in a case of hanging



Figure 23.10: A case of hanging showing hypostasis over the forearm and lower limbs

and later on seen on the back when the body is laid down. In few cases the post mortem petechial hemorrhage may be seen in the lower limbs. These hemorrhages are differentiated from the tardieu's spots, being larger in size and bilaterally symmetrical. They are due to rupture of capillaries in lower limbs due to pressure of accumulated blood.

Local Findings

1. Ligature mark over the neck.
2. Usually, except for the ligature mark, no other injuries are present over the body. However, when other injuries are present they should be

carefully interpreted whether they are self-induced, occurred during convulsive phase preceding death, produced when body was brought down, during attempted resuscitation, when the swinging body contacted other objects or were produced by a second party in a homicide.

Differential Diagnosis of Ligature Mark

1. Skin folds on the neck of Infants simulate a ligature mark, especially after refrigeration of the body has caused coagulation of the subcutaneous fat.
2. Swelling of the neck due to decomposition and jewellery of the neck or clothing causing the ligature mark.

Internal Findings

The dissection of neck structures should be ruled by two considerations. (i) Need for an extensive exposure and (ii) Necessity of avoiding artefacts from seepage of blood from the neck veins. The dissection of neck structures should be done *in situ* in a relatively blood free field after the cranial cavity, chest and abdomen is opened and the organs removed. Schrader (1940) drew attention to artefacts and recommended that neck should be drained of blood by removing the brain and dissecting heart followed by the dissection of neck structures done *in situ*.

On examination of internal structures of the neck in 83 consecutive cases prospectively by DiMaio, no injuries (Fig. 23.11) were seen in more than 50% of the cases. Absence of petechiae in most hangings is because there is complete obstruction of the arterial system, so there is no pooling of blood in the head, no increased pressure and thus no petechiae. A dried rivulet of saliva often runs from a corner of the mouth down the chest. Besides this, blood tinged fluid may be present in the nostrils. The fractures of the hyoid bone and thyroid cartilage are considered antemortem when there is blood at the fracture site. Tissues under the mark are dry, white and glistening. Ecchymosis may be seen in the muscles. Neck muscles, the sterno-mastoid and



Figure 23.11: Neck structure

platysma are ruptured in about 10-15% cases if considerable violence is involved during hanging. Carotid Artery may show transverse tear in the intima. In long drop tears may be seen at different places. Vertebral artery may show rupture at place. Hyoid bone is fractured in 15-20% cases but it is rare below the age of 40 years due to elasticity of cartilage and the mobility of joints. It is more common above 40 years and usually found at the greater horn and junction of inner 2/3 and outer 1/3. Thyroid cartilage is fractured in about 40-45 % of the cases. Superior horn is fractured or the thyrohyoid ligament is torn. Visual inspection of the trachea reveals petechial haemorrhages over the epiglottis, trachea and larynx. Abdominal organs are congested and in the brain sub arachnoid effusions are commonly seen.

Diagnosis of Antemortem Hanging

1. Presence of antemortem ligature mark with vital reaction.
2. Saliva dribbling mark from the angle of mouth.
3. Ecchymosis of larynx or epiglottis.
4. Extent of fracture of thyroid cartilage and hyoid bone.
5. Rupture of intima of carotid vessels.

Medico Legal Aspects

Hanging is most commonly suicidal but can be at times be accidental like in cases of sexual

asphyxias. Homicidal hanging is a very rare and is seen mostly in cases where the victim has been incapacitated by earlier assault or due to intoxication with alcohol etc.

Judicial Hanging: Judicial hanging is the term used when convicted criminals are hanged. The person's face is covered with a dark mask and he is made to stand over trapdoors that open downwards. The noose is put around the neck with the knot placed on the left side of the jaw, close to chin. A trapdoor is sprung and the prisoner falls a specific distance determined by his weight. The platform is at a height allowing for a fall of about 5 meters. If he falls an insufficient distance, he strangles rather than break his neck and if he falls too far he is decapitated. As the person falls there is a sudden jerk to the neck causing fracture dislocation at the 2nd to 3rd cervical vertebral level or sometimes at the atlanto occipital or atlantoaxial joint with transaction of the cord. In properly performed judicial hangings, the victim abruptly stops at the end of his fall when his head is jerked suddenly and violently backward, fracturing his spine. The neck is considerably lengthened and may be severed in some cases and the neck vessels are torn transversely. There is immediate unconsciousness leading to death though the heart may continue to beat for 8-20 minutes. In addition, there are muscular contractions of the facial muscles, twitching and convulsions of the limb and trunk, and violent respiratory movement of the chest. X-rays show bilateral fractures of either the pedicles or laminae of the arch of the 2nd, 3rd, or 4th cervical vertebra with dislocation of the 2nd or 3rd or 3rd or 4th cervical vertebra.

Suicidal Hanging: Suicide is the most common mode of hanging. The person finds it as one of the easiest and surest methods of death. Hanging is known as painless mode of death. The person hangs himself with any long ligature material available that is tied to a ceiling fan, roof, window etc. There may be a fixed loop or a knot. Usually a suicide note may be found at the scene (Fig. 23.12). The individual usually hangs himself in an isolated area and when hanged in his house, the room in such cases is bolted from inside.



Figure 23.12: Suicidal note in a case of hanging

Homicidal Hanging: Homicidal hanging is extremely rare. It can be a possibility in cases where the victim is a child, an unconscious person or a very frail person unable to defend himself. It would virtually be impossible for one of two healthy males, equally matched physically, to hang the other unless the victim was beaten unconscious or rendered helpless by alcohol or drugs. In the first instance, one would suspect homicide by virtue of the injuries on the victim. In the second scenario, or if the victim was rapidly subdued by two or more stronger assailants, there might not be any other marks on the body but for the noose mark or possible contusions of the arm inflicted while being forcibly restrained by the assailants. If there were also a marked disproportion in strength between the assailant and the victim such as an adult and a child, it would also be possible to hang the victim without any marks of violence. In addition, an individual might be first strangled with ligature and then hanged. In this case, the ligature mark over the neck would not have classic inverted V configuration of hanging. Although there are cases on record of suicidal hangings involving incomplete suspension, where the noose mark overlaid the larynx and was horizontal, not sweeping upward to the point of suspension in an inverted V configuration. To rule out any violence that might not be visible and to make sure that the individual was not drugged, in all alleged suicidal hangings, a complete toxicological screen

should be performed. If there are signs of violence then it may be homicidal in nature. Circumstantial evidence is of value in these cases. When an unruly mob hangs a person in public, it is called *lynching*.

Accidental Hanging: Accidental hangings usually involve children who are playing while simulating judicial hanging get entangled in a rope. Boys playing in gardens while jumping from trees get their clothing caught in the branches and are accidentally hanged. Adults engaging in the practice of sexual asphyxia may be accidentally hanged. In addition, women may get their *dupatta* entangled in the cycle rickshaw and it may tighten around the neck sufficient to cause hanging. The chin may be suspended from the steering wheel of a vehicle. In cases of a fall from a staircase, a person may get his neck caught in the railing and hang. An intoxicated person may accidentally hang himself from the arm of a chair. Rarely an infant accidentally hang himself from a pacifier placed around his neck on a cord or are entangled in a toy placed above his crib.

Crime Scene Examination

Examination of the scene of crime is very important and has to be done carefully. The body should not be cut down so that proper photographic documentation can be made. The important points, which must be observed, include the posture of the body, any signs of violence in the area like disturbance of furniture etc. the noose should never be cut from the body but left intact for transport with the body to the morgue. The ligature material has to be examined carefully for the texture and the knot etc. When the noose is removed from the neck, the knot should not be untied. The noose should either be slipped off the head or cut opposite to the knot with the cut ends secure with the tape or string. The height from which the body is suspended should be measured and the platform that was possibly used by the victim must be noted. Sometimes when the ligature is broken, the body may be found lying on the ground. In such cases, the part of the noose around the neck should be properly secured and the other part that

is left attached to the ceiling fan, hook or rod of window or doorknob should be removed and compared with the part around the neck.

Critical Evaluation

1. At times when the person is attempting suicide and the first attempt fails he may fall down and sustain injury. The victim then tries a second attempt with another rope at another place and may be found with bleeding and injuries. The scene may also be disturbed in such cases with blood present at places. The injuries on the body of the person may be misinterpreted as struggle marks. Careful evaluation of the crime scene is required in such cases to arrive at a conclusion.
2. When a person hangs with a rope then it leaves its groove marks on the hook or beam, which is used to tie the rope. The groove mark has to be carefully observed for the pattern of the cord on it and must be matched with the width of the cord. If some clothing or hair comes in between, then the mark may not be visible.
3. Dead persons can show grooves on the neck which can be confused with hanging grooves. Such marks are caused by clothing like tight collar or a tight necklace etc. These marks disappear after decomposition while a ligature mark does not decompose as easily as there is little blood in the skin underneath leading to much reduced bacterial growth.
4. Murder by hanging is very rare and is usually possible in children, unconscious persons and those who are unable to defend themselves.
5. Persons who have committed suicide by hanging may show other injuries which in themselves could be fatal.
6. When the body is hanging free but there is no jumping point in the vicinity such as chair, table, step etc., there is every reason to suspect murder. In such cases scene must be examined for marks of climbing over trees and development of postmortem staining over the lower limbs. If staining is present on the back of a freely hanging body it signifies that person

has been killed first and then hanged. Wrinkles on the clothes may indicate that limbs were previously bent.

7. Presence of material evidence on the clothes such as leaves, twigs of plants, soil, dust that is not present at the scene of hanging must be noted. In addition the observations such as presence of blood, urine and saliva flowing in the wrong direction should arouse suspicion of crime. If the knots and noose present around the neck are such that it is not possible for the individual to make them should raise suspicion of crime.
8. In a right-handed person, knot is usually on the right side and any reversal should raise suspicion of homicide.
9. Autopsy rarely reveals whether the hanging is suicidal or homicidal. It is the duty of the Police Officer to correlate the course of events.

SEXUAL ASPHYXIA (AUTOEROTIC ASPHYXIA)

Sexual asphyxias are asphyxial deaths principally due to hanging in which the asphyxia is intentionally induced to enhance sexual arousal produced by masturbation. These individuals use transitory anoxia produced by the noose to intensify the sexual gratification produced by masturbation and the impaired circulation leads to impaired consciousness. The impaired consciousness causes hallucinations and feeling of erotic rapture. Degree of asphyxia is controlled by mechanical means and a failure of the mechanism accidentally may cause death. Such deaths are rare and the victim is virtually always male with a homosexual tendency in them. Abnormal sexual behaviour like masochism, transvestism may also be present.

For hanging, the victim commonly uses a pad between ligature and neck. The weight of the body is used as control. The noose can be tightened by extending arms or legs, and then can be relaxed by flexing the limbs. Death may occur accidentally by a failure of the mechanism of neck constriction. The scene is usually the victim's own house, and his bedroom or attic etc.

On examination of scene it is of incomplete hanging with his feet on the ground so that he can relieve pressure of the noose just by standing a little straighter. A stool or chair is found near hanging victim at the scene. the pattern of behaviour is repetitive and there are evidence of previous episodes such as old scars in the neck. The person is usually naked or partly naked. One of the partners to simulate a female body does padding of breasts. In addition, they like to wear female undergarments sometimes. At the scene, there may be erotic or pornographic photographs, literature, articles of female clothing, a mirror opposite the individual so he can observe his actions. can be found around the scene. Evidence of recent ejaculation must be looked for in the form of seminal stains. Sexual gratification may be obtained by electrical stimulation using a low voltage, to stimulate the genitalia.

Addicts usually use plastic bags to inhale anaesthetics or narcotic vapours as an aid to intoxication. However, in the autoerotic practices, the victims use plastic bags to cover the head that induces partial anoxia accentuating sexual sensations. As a result, sometimes the victims may lose consciousness and die. Some individuals in order to obtain sexual gratification do inhalation of carbon tetra chloride, bichloro-ethane and other petroleum products (glue sniffing). To summarize, the scene in such a case should be examined for:

1. Evidence of abnormal sexual behaviour
2. Evidence of previous attempt
3. Evidence of padding between the ligature and the neck
4. No evidence of suicidal act such as absence of suicidal note etc

STRANGULATION

It is a form of asphyxia, which is caused by constriction of neck by a ligature without suspending the body. It is mainly of two types (i) Ligature strangulation and (ii) Manual strangulation. Nevertheless, the following types may also be encountered.

Classification

1. Ligature Strangulation
2. Throttling or manual strangulation
3. Mugging
4. Garroting
5. Bansodala
6. Palmar Strangulation
7. Foot strangulation.

Medicolegal Aspects

1. Whether death is due to strangulation? This can be ascertained from the general features of asphyxia mainly on the head and neck that are strongly presumptive of strangulation deaths., the ligature mark around the neck, internal findings in the neck, circumstantial evidence. The manner of death can be:

Homicidal: It is a common form of murder and is seen in adult women usually combined with sexual acts. Single turn of ligature with one or more knots, at the front or sides of neck is usually found. If two or more knots are present with each turn of ligature then homicide is almost certain. Abrasions are usually seen due to movement of the ligature in the neck during struggle by the victim. Evidence of struggle may be absent in cases like that of weak and frail individuals especially children or when the victim is attacked suddenly or made unconscious earlier by trauma or drugs.

Strangulation should be taken as homicidal unless otherwise proved. The observations which can prove to be of value in establishing the nature of the injury include ownership of ligature and the possession of the ligature material with the suspected person. Unusual ligatures may indicate a particular occupation of the assailant. Looseness of the ligature or missing ligature is a near definite sign of homicidal strangulation. If the mark does not correspond with the ligature material recovered, it is also indicative of homicidal strangulation. Circumstantial evidence is of value in deciding doubtful cases.

Accidental: Children may accidentally get entangled in ropes during play. An infant may be strangled with a string attached to a log-tied to the crib. Intoxicated persons may get accidentally strangled by their necktie, scarf or collar etc. In industry, belts, ropes and parts of clothing may be caught in rollers or parts of moving machinery and cause strangulation. Umbilical cord inside the uterus may also accidentally strangulate the foetus.

Suicidal: various methods for tightening the ligature are employed by the victims. Number of knots, tightness, method of knotting should be considered. The ligature material can be used like a tourniquet by the victim.

A person can have one or two knots before consciousness is lost. Some arrangement is always made to keep the ligature tight. A cord may be tied around and twisted by stick working as a lever. When consciousness is lost, the grip is released, but ligature will not become loose, as it gets arrested against the shoulder or chin. When a running noose is applied to neck, a weight is attached to the free end of the rope at the end of the bed on which the patient is lying. Signs of venous congestion are very well marked above the ligature and very prominent at the root of the tongue due to slow tightening of ligature and no draining of postmortem blood.

Injuries are less marked on the body of the person as less force is used. No signs of struggle are seen. Ligature is found in situ. Application of ligature with several turns whether closed with half knot or complete knot is consistent with suicide. Single turn of a broad ligature of rough cloth, closed with a half knot also indicates suicide.

Opinion is given based on the ligature material recovered, the way it was tightened, no marks of violence on the body and the surrounding environment.

Pseudo Strangulation

In persons having short neck, depression mark similar to ligature mark is produced after death

due to the fold of skin. Even in infants and children with short neck, similar marks are produced after death called Pseudo Ligature marks.

LIGATURE STRANGULATION

In ligature strangulation, the pressure on the neck is applied by a constricting band that is tightened by a force other than the body weight. This is the type of strangulation in which a ligature material is used to compress the neck and the assailant applies the constricting force without suspending the body. Usually a U shaped ligature is pulled around the neck from behind though multiple turns of the ligature material are often used. Virtually all cases of ligature strangulation are homicides, suicides and accidents are rare. Females are predominantly involved.

Ligature Mark

The ligature used in ligature strangulation may range from electrical cords, ropes, telephone cords, sheets and neckties. The appearance of the ligature depends on the nature of ligature, amount of resistance offered by the victim and the amount of force used by the assailants. When the ligature used is soft material, the ligature mark may be faint, barely visible or even absent in children or incapacitated adults. When a very thin ligature is used, the mark appears as deep mark and encircling the neck.

Facial Findings

The face and neck above the ligature mark are markedly congested with confluent petechial haemorrhages in the sclera and conjunctivae. Fine petechial haemorrhages over the face especially in periorbital area are seen. The findings of congested face, congestion, petechiae and haemorrhages in the sclera (Figs 23.13 and 23.14) in ligature strangulation are due to the fact that unlike hanging there is no complete occlusion of the neck vessels. The blood continues to enter the head from vertical arteries. This results in increase intravascular pressure, congestion and rupture of vessel.



Figure 23.13: Congestion of sclera in a case of petechiae in a case of manual strangulation



Figure 23.14: Subconjunctival haemorrhage in a case of ligature strangulation

Causes of Death

Death in these cases is due to asphyxia, cerebral anoxia, vagal inhibition and fracture dislocation of cervical vertebrae. By far the most common cause is asphyxia caused due to elevation of the larynx and tongue, which closes the airway at the pharyngeal level. If extreme pressure is applied by the assailant then the trachea or the larynx itself may be blocked and cause asphyxia. Cerebral anoxia is caused by compression of the blood vessels in the neck and if the pressure is

moderate then it may cause only the compression of the veins leading to venous congestion. Vagal nerves can get stimulated and cause depression of the heart to cause death. Fracture dislocation of the cervical vertebrae is a very rare finding but may happen in case of thin and frail person who is attacked by a very strong assailant.

Postmortem Appearances

External findings: the ligature mark is usually a well-defined depressed mark present at the middle or lower part of the neck (Figs 23.15 and 23.16). The mark may encircle the neck fully but is most prominent at the front and the sides of the neck. It is usually transverse but may be oblique if the



Figure 23.15: Ligature mark over the neck in a case of strangulation



Figure 23.16: Ligature mark in a case of ligature strangulation

victim has been dragged by the assailant for some distance.

Internal findings: Hemorrhages are found in the muscles and soft tissues underneath the ligature mark. If severe force is used carotid artery tear may be present. Fracture of Hyoid bone and thyroid cartilage may be seen. Hyoid fracture is seen in inward compression fracture, which can be bilateral while outward compression such as in hanging gives rise to antero-posterior compression fracture of the greater horn at the junction with the body.

Avulsion fracture may be seen in due to muscular over activity without there being direct injury to the hyoid bone. Called traction 'Tug' fractures.

The differences in findings between hanging and ligature strangulation are enumerated in Table 23.1.

MANUAL STRANGULATION (THROTTLING)

It is asphyxia produced by compression of the neck. The mechanism of death is occlusion of

the blood vessels supplying blood to the brain. Occlusion of airway probably plays a minor role in causing death. Virtually all manual strangulations are homicide.

Suicidal Throttling: Suicide by throttling is impossible as when the pressure by the victim's own hands is sustained for a sufficient time to cause unconsciousness, the hands relax and the victim recovers.

Accidental Throttling: The application of one or both the hands to another person's throat can cause sudden death from vagal inhibition. This may occur in unintentional circumstances brought on by touching, grasping and demonstration of affection such as during intercourse, or during physiological experiments. This is theoretically possible but highly improbable and suspicious. The mechanism of death in this case would be an arrhythmia produced by stimulation of the carotid sinuses. The carotid sinus is a focal area of enlargement of the common carotid artery at the point it bifurcates in to the external and internal

Table Difference in findings in hanging and strangulation

Features	Hanging	Strangulation
Ligature mark:		
i. Shape	• Oblique	-Transverse
ii. complete or incomplete	-Incomplete	-Complete
iii. Level of thyroid	-Above or over thyroid	-Below
iv. Base	-Hard, pale and parchment like	-Soft and reddish
Abrasions and ecchymoses at the edge of ligature	Uncommon	Quite common
Bruising of neck muscles	Not very common	Very common
Subcutaneous tissue	White, hard, glistening	Ecchymosed under the mark
Hyoid bone fracture	Very common	Less common
Carotid Artery damage	May be seen	Rare
Thyroid cartilage fracture	Rare	Common
Fracture of larynx & Trachea	Rare	May be found
Facial congestion and petechiae	Pale	Marked with petechiae
Protrusion of tongue	Less common	More common
Asphyxial signs	Less marked	More marked
Bleeding from nose, mouth & ear	Less common	More common
Salivary stains	Present	Absent
Neck	Stretched or elongated	Not stretched or elongated
Lungs	No bulking	Congested and oedematous
Subpleural hemorrhage	Less common	More common
Semen discharge	Common	Not very common
Bullous lesions of lungs	Not present	Present

carotid arteries. The compression or stimulation of the carotid sinuses causes an increase in blood pressure in these sinuses with resultant bradycardia, vasodilation and hypotension. Pressure on the common carotid artery below the sinuses reduces blood pressure within the sinuses by reducing the amount of blood flowing into it. This mimics hypotension or decreased blood supply from haemorrhage and shock causing tachycardia, vasoconstriction and hypertension. This explains the fact that why in some cases of throttling if the hands are lower down over the neck there is tachycardia, vasoconstriction and rise in blood pressure

Homicidal Throttling: Death resulting from asphyxia consequent upon manual strangulation raises the suspicion of homicide. This method is employed when the victim is an infant, a child, or woman. Healthy adults can be throttled only when they are under the influence of drugs or other intoxicants, are stunned or the attack is sudden. Throttling is a common method of infanticide.

Postmortem Findings

External Findings

The situation and extent of bruise on the neck will depend upon the relative positions of the assailant and victim, manner of grasping the neck and degree of pressure. Bruises are produced by tips and pads of fingers, are oval or round and of the size of digits (1.5-2 cm). The size may be altered due to bleeding underneath the skin. The grip can be produced by right hand with thumb on right side of neck and fingers on left side and vice versa. Finger marks may be present obliquely, separately or clustered together (Fig. 23.17). Both hands are commonly used with the thumb of one hand and finger of other hand present over both sides. Strangulation is easier for assailants with big hands on victims with small necks. Signs of struggle or bruises may be found anywhere, not definite to say whether the attack was from behind or from the front. Bruises may go for the usual colour change. When fingertips are deeply pressed, nails produce crescentic marks on the skin. During grip



Figure 23.17: External findings on neck in manual strangulation

thumb exerts more pressure than the other fingers. Two types of abrasions may be seen. When nails skid down the skin parallel, linear lines for several centimeters may be seen. In other cases regularly curved, comma like, disk like or straight 1.5 cm in length and few mm width. Many scratches are produced by the victim while trying to pull away from the assailant's hand. As most victims are females, usually having long nails, scratches are more severe than those of assailants. Parallel lines in vertical direction are seen in such cases. The nail marks are more marked when the skin is dry.

Internal Findings

1. Hemorrhages are found in the muscles underneath the ligature mark (Fig. 23.18). Carotid artery tear may be present.
2. Fracture of Hyoid bone is very common especially in the elderly. Thyroid cartilage may also be fractured in many cases. Hyoid fracture is seen in inward compression fracture, which can be bilateral
3. If death was due to throttling then suicidal mode of death is not possible. Homicidal or accidental possibility has to be ruled out. Sudden application of one or both hands on the other person's throat out to affection can also cause death.



Figure 23.18: Internal findings in neck in manual strangulation

BANSODOLA

Strong bamboo or stick is put in front of neck and another one at the back of neck. Both ends are tied and squeezed, from front of chest and back to cause death. It interferes with respiration, and causes fracture of ribs etc.

GARROTING

In this the victim is attacked from behind without warning, the throat may either be grasped by hand or a ligature which is quickly tightened, by twisting it, with a lever causing sudden onset of consciousness and collapse leading to death. In one of the refined methods of garroting, a spike was used behind a clamp, which penetrated the spine and caused the death of the person. It was a mode of execution in Spain and Turkey.

MUGGING

Strangulation is caused by holding the neck of the victim in the bend of the elbow. Pressure is exerted either on the front of larynx or at the sides of neck by forearm or arm. Marks in the neck may be minimal in such cases.

Fractures of hyoid bone

Various types of fractures of hyoid bone may be seen in asphyxial deaths such as

1. Inward compression fracture in which the greater horns of hyoid are compressed inwards causing fracture of the bone with tear of its periosteum on the outer side and not on inner side displacing the posterior fragment inwards. It is seen in manual strangulation. This type of fracture can occur on both the sides.
2. Outward compression (antero-posterior compression) fracture in which hyoid bone is driven backwards, when divergence of greater horns is increased, causing fracture with outward displacement of posterior small fragment. As a result, the periosteum on inner side of fracture only is torn when the fragment can be easily moved outwards but inner movement is limited to normal position only. Either this type of fracture can occur in the greater horn at its junction with the body or it may be bilateral. This type of fracture is seen in hanging and ligature strangulation.
3. Avulsion fracture that occurs due to muscular over activity without there being direct injury to the hyoid bone. This type of fracture is also called traction 'tug' fracture. This occurs as a result of traction on thyro-hyoid ligament either by downward or lateral movement of the thyroid cartilage from antero-posterior or lateral compression or when direct pressure is exerted between the hyoid and thyroid by the pressing fingers.

SUFFOCATION

The term suffocation is usually regarded as synonymous with asphyxia. However, the term is restricted to a group of circumstances except drowning in which asphyxia produced by causes other than constriction of the neck by a ligature, hands, or a stranglehold. This includes closing the external respiratory orifices either by hand or by other means or blocking up the cavities of nose and mouth by introduction of a foreign substance, mud, paper clothes etc. The following types of suffocation is usually seen:

1. Smothering
2. Overlying

3. Burking
4. Choking
5. Gagging
6. Traumatic Asphyxia
7. Suffocation due to irrespirable gases.

In all these forms of suffocation, the asphyxial symptoms and signs are severe since death is usually the result of slow asphyxia, often taking 2-5 minutes to kill. Usually the external signs of asphyxia such as cyanosis of the face and petechial haemorrhages of the facial skin, conjunctiva is common. However, there may not be any external injury at all. Alternatively, these injuries may be slight and can escape the eyes of the most experienced examiner.

SMOTHERING

Smothering refers to the death resulting from mechanical occlusion of the mouth and nose that prevents breathing. The smothering agent is usually a fabric, an impervious sheet or a hand, though occasionally a mobile solid such as sand, mud, grain or flour may be responsible for blocking the air-passages. In smothering, death may occur either by the occluding substance pressing down upon the facial orifices, or by the passive weight of the head pressing the nose and mouth into the occlusion. Deliberate homicide is seen usually in the old, the debilitated and in infants. It is extremely difficult to prove homicide from the objective findings. The smothering of babies whether intentional or accidental is not so rare in India compared to western world. However, in these cases, the findings are not significant as the classical signs of asphyxia such as congestion, cyanosis and facial and conjunctival petechiae are not commonly present. Since intrathoracic petechial haemorrhages are common in sudden infant death syndrome, these cannot be accepted in isolation as evidence of suffocation.

External Findings

1. No external signs are there when the obstruction is by some soft material such as bed-clothing, pillow, cushion, mother's breast or

- even assailants hand that is gently applied.
2. There are scratch abrasions, finger nail marks, laceration of soft parts of victim's face (Fig. 23.19).
3. There may be bruising and laceration of lips, gums and tongue (Fig. 23.20).
4. Pressure marks on the face can rarely be distinguished from postmortem postural changes, where circumoral and circumnasal pallor is caused merely by passive pressure of the dependent head after death, preventing the gravitational hypostasis from entering these areas. Even when the head is found supine,



Figure 23.19: Contused abrasions in and around the lips in a new born in case of smothering



Figure 23.20: Contused laceration over the inner sides of lips in a case of smothering

variation in colour is still common on the face, with contrasting white and pink patches, which usually change as the postmortem interval lengthens.

5. Unless there are bruises or abrasions on the cheeks, around the mouth, lips or lesions within the lips or mouth, it is dangerous to over interpret mere colour variations from alterations for blood in the facial capillary bed, which is usually a postmortem phenomenon.

Medicolegal Aspects

Suicidal smothering: This can occur in mental patients or prisoners. Suicide is practicable merely by burying the face in a flock mattress or even it would appear by lying against the bed clothing to obstruct the nose and mouth, especially when drunk. The death by cutthroat may occasionally terminate by smothering. If the trachea is completely severed, the lower end is likely to be drawn down within the thorax by the weight of the heart and lungs; if the soft parts then close over the wound, they can obstruct the trachea and the victim is smothered.

Accidental smothering: The circumstances of accidental smothering vary according to the age of the victim. The bedclothes covering the nostrils and mouth of the infant infants during first month of life especially premature. Suffocation of the infant is also possible when it turns on its face in its cot and buries his face in a soft pillow or mattress. Accidental smothering in children and young adults may be predisposed to by an epileptic fit. Another variety of accidental smothering likely to involve children in an airtight place or one in which ventilation is negligible such as disused refrigerator. Accidental smothering of adults by precipitation into a quantity of finely divided, solid materials such as flour, sand, coal-dust, cinders, grain or feathers may occur in the course of their occupation. Besides, smothering may occur subject to epileptic or epileptiform fits. Accidental smothering may occur outdoors when the victim, in the course of an epileptic fit or under the influence of drinks or drugs, falls face down into mud, snow or thick grass.

Homicidal smothering: homicide of an adult by smothering is possible when there is a gross disparity between the assailant and the victim or else when the victim is helpless by virtue of his age, ill health or incapacity from drink or drugs or when stunned by a blow. It is a common mode of infanticide. Usually infant's mouth and nostrils are closed by means of hand, or even a plaster. Usually the assailant uses far more force than is necessary to kill and consequently finger-marks in the form of scratches and bruises are found over the victim's face.

CHOKING

It is a form of asphyxia caused by an obstruction within the air passage. It is almost always accidental and is usually due to inhalation of a foreign body, but it can be caused by the inhalation of the products of disease or by anatomical changes due to disease. The usual mechanism of asphyxia in choking is simple mechanical obstruction. However, occasionally the entry of foreign material may cause sudden death from vagal inhibition (Café-coronary).

Causes of Choking

1. Inhalation of foreign body into the glottis such as bolus of meat, potato, button, coins, marbles, corns, fish, screws, pins or artificial dentures.
2. Inhalation of vomited material in an unconscious, when intoxicated, during anaesthesia or epileptic fits.
3. Belching out of milk by infants and young children may be aspirated into the air passages
4. Inhalation of products of disease or violence such as blood or pus while haemoptysis in Tuberculosis, rupture of an aortic aneurysm in air passages, or due to flooding of air passages with pus and necrotic debris in a lung abscess.
5. Anatomical changes due to disease such as a bronchial growth, laryngeal oedema and tumours, pharyngeal abscess, angioneurotic oedema.

6. In conditions such as poliomyelitis or bulbar palsy, accidental inhalation of food occurs due to disturbance in swallowing mechanism.

Mechanism

The usual mechanism of asphyxia in choking is mechanical obstruction but the entry of foreign body may cause sudden death from vagal inhibition.

Causes of Death

1. Asphyxia is most common
2. Vagal inhibition: There are cases where the immediate result of choking is vagal inhibition and sudden death. The diagnosis is difficult due to absence of overt signs especially in an infant with small quantity of foreign material in the respiratory passages. The diagnosis is simple when a massive foreign body causes the obstruction but even when the foreign body is small, yet the cause of death seems to be vagal inhibition. Sudden death can occur when there is flooding of postnasal space with regurgitated food or even when minute particles are exposed to the vocal cords. The surgical procedures on larynx with light anaesthesia may cause sudden death from vagal inhibition.
3. Laryngeal spasm: This occurs due to entry of an irritating foreign material in the air passages. This is likely if acid gastric juice alone suddenly enters the air passages.

Medicolegal Aspects

Death by choking is commonly an accident but occasionally it may be homicidal.

Accidental Choking

It is very common and is seen in very, young, elderly, psychiatric patients when the ability to chew or swallow is impaired. It may commonly occur during a meal when food is accidentally inhaled while the person is laughing or crying or even when someone unexpectedly slaps the person on the back.

Suicidal Choking

Suicide by choking is quite rare but may be seen in mentally ill patients or prisoners who do not have access to other methods of committing suicide.

Homicidal Choking

Choking is an uncommon and rare mode of committing homicide. However, the literature suggests that cases are on record where murders have been committed by choking the victim. Choking due to regurgitation of food may occur during rape or violent sexual intercourse.

Autopsy Findings

1. Presence of impacted foreign body in the air passages.
2. Regurgitated food particles such as vomitus, milk etc may be found mixed with mucus in trachea, bronchi and lower bronchioles.
3. All organs are congested.

CAFÉ-CORONARY

A condition where a healthy and grossly intoxicated person, suddenly turns blue, coughs violently, then collapses and dies. Death appears to be due to sudden heart attack. However, at autopsy one may find some food material or piece of meat obstructing the larynx. Clinical signs of choking are absent, as there is a high blood alcohol content, which anaesthetizes the gag reflex.

As the person vomits, the vomited matter may be inhaled leading to choking. Infants often regurgitate clotted milk after a meal into larynx and children may often place coins, small stones in the larynx. Rubber balloons may also be accidentally ingested by children. And may get stuck in the pharynx or glottis. Suffocation may be caused by some diseases like diphtheria, Infectious mononucleosis, Haemoptysis in pulmonary tuberculosis.

Larynx and bronchial growths may cause hemorrhage into trachea

Insect bites and drug reaction Larynx edema leading to death

Blow to the front of the neck may cause swelling of the mucosa, edema, haemorrhage and death may occur due to reflex vagal inhibition.

TRAUMATIC (CRUSH) ASPHYXIA

This is the name given to those kinds of asphyxia, which are caused due to mechanical fixation of the chest so that normal movements of chest wall are prevented. This occurs when a large weight falls onto or presses down on an individual's chest or upper abdomen. The most common form of traumatic asphyxia encountered is while repairing a car the jack slips and the vehicles falls on top of the individual. Great force is necessary to compress the chest and so this type of asphyxia is seen in cases like multiple deaths during stampede (chest is compressed and respiratory movements prevented by stampeding people piling on the top of each other—Riot crush or 'human pile' deaths), hotel fire, and lane crowd in an enclosed place, building collapse and in coal-mines. Traumatic asphyxia is usually accidental in manner.

Postmortem Findings

There is deep red or purplish black discolouration of the skin of head, neck and upper trunk with numerous petechiae and ecchymoses in these areas, petechiae of the sclera, conjunctiva and periorbital skin and retinal haemorrhages. There is intense cyanosis of deep purple or purple-red colour cyanosis above the level of compression usually to the level of third rib. Haemorrhage is seen in the tissues around the site of compression whereas below the level of compression, skin is pale and mild cyanosis may be seen. Areas of pallor are seen at the level of collar of the shirt, folds or creases of garments. Internally, there is often no evidence of trauma in spite of the heavy weight on the chest. In addition, fracture of ribs may be present. If the patient recovers, the purple colour gradually disappears in 10-14 days without the usual colour changes. Individuals who survive

an episode of traumatic asphyxia usually make an uneventful recovery, though occasionally there is some permanent visual impairment due to retinal haemorrhage.

Due to compression of chest, retrograde displacement of blood from superior vena cava into subclavian veins and veins of head and neck occurs leading to the cyanosis and congestion. Valves in the subclavian veins prevent spread of hydrostatic force to the veins of the upper limb and displacement of blood in to the valveless veins of the head and neck causes the rupture of distal venules and capillaries. As a result there is formation of numerous arterial haemorrhages into eyelid and conjunctiva with oedema of conjunctiva and bleeding from nose and ears. Internal organs are congested and petechial haemorrhages may be found in the pleura and pericardium. The abnormal purple red colour may persist for several days. in addition there may be presence of fractures of ribs.

POSITIONAL ASPHYXIA

Positional asphyxia is virtually always an accident and is associated with alcohol or drug intoxication. An individual becomes intoxicated and falls into a restricted area, where because of his position of his body, he cannot move out of that area or position and there is restriction on his ability to breathe. There is usually marked congestion, cyanosis and petechiae. Positional asphyxia occurs if an individual falls down a well and is wedged between the walls. Every time he exhales, he slips farther and farther down the well preventing inhalation

OVERLAYING

This form of asphyxia is a combination of mechanical asphyxia and smothering. It is seen most commonly when an infant is put to bed with one or more adults. During the night, one of the adult inadvertently rolls on to or otherwise crushes and asphyxiates the infant by compressing the chest thus preventing respiratory movement and occluding the nose and mouth with bedding or

the body of the adult. Later on the adult rolls off the child. The next morning the child is found dead without any evidence of trauma. These deaths are often thought to be due to SIDS. The autopsy findings are nonspecific and are few fine petechiae over the face but not over the sclera or conjunctiva. The history of an adult sleeping with the child is important to differentiate such deaths from the 'cot deaths'. In England under the Children and Young Persons Act, 1933, it is a criminal offence to share bed by a person of the age of sixteen years or more with an infant in an intoxicated state and the child dies of suffocation.. When individuals are buried in loose earth or sand (cave-ins) or in grain silos, they die as a result of occlusion of the nose and mouth along with immobilization of the chest and abdomen by external pressure sufficient to prevent respiratory movement.

Autopsy Findings

- 1 Presence of petechial haemorrhages beneath the conjunctiva, epicardium and pleura.
- 2 Evidence of contact flattening of face and nose may or may not be there. This is suggestive of infant being laid on the face at the time of or after death.
- 3 Blood tinged froth from mouth and nostrils that may stain the bed clothing.
- 4 Absence of injury marks over the body.

BURKING

Burking is a form of mechanical asphyxia plus smothering that is homicidal in nature. Burke and Hare were murderers during the 1820s in Edinburgh who supplied dead bodies for anatomical dissection to the medical schools. They first started out as 'resurrectionists' but later on decided to eliminate the chore of digging up bodies. Instead, they would go straight to the source and would invite the individual to their house and intoxicate him. According to Hare, who turned King's evidence, Burke would kneel or sit on the chest, covering the mouth and nostrils with his hand thus producing a combination of traumatic asphyxia and smothering. External injury was

absent and for Hare, the precise mechanism of asphyxia might not have been discovered. There was some bleeding into the tissues around the cervical spine but it was concluded that the injury occurred shortly after death. Burke later confessed to sixteen of such murders, which were committed for gain.

SUFFOCATION FROM IRRESPIRABLE GASES

Deaths from suffocating gases are due not to the toxic nature of the gases, but rather to displacement of oxygen from the atmosphere. Carbon dioxide and methane are two most commonly encountered suffocating gases. Both are essentially nontoxic and odourless and are found in sewers and mines. Methane is the principal constituent of the natural gas (94-96%). Reduction of atmospheric oxygen to less than 25% of normal displacement of oxygen by inert gases such as carbon dioxide and methane produces unconsciousness in seconds and death in a matter of minutes. The cause of death can be determined from the circumstantial evidences as there are no specific autopsy findings. If death is prolonged, the individuals appear cyanotic with petechial haemorrhages of the epicardium and visceral pleura. Petechiae are frequently absent in rapid anoxic deaths. Toxicological analysis for methane can be done. When is detected in blood being nontoxic death cannot be attributed to it. however, it suggests that the individual was exposed to an atmosphere containing methane. As far as presence of carbon dioxide is concerned, it is a normal constituent of blood.

DROWNING

Drowning is a form of asphyxia due to aspiration of fluid in to air passages caused by submersion in water or other fluid. Complete submersion is not necessary for drowning to occur. Our knowledge of the events that take place in the drowning victim is by no means complete. It is primarily derived from animal experiments and to some extent from the observations on human

where drowning was witnessed and reported. Submersion is usually followed by a panicky struggle to reach the surface. When energy reserves are exhausted, the struggle subsides and actual drowning begins. When a person falls in to water and sinks it is partly due to specific gravity of the body and partly due to force of fall. On sudden immersion in to cold water, he may take a deep inhalation of water due to reflex from stimulation of the skin. The person may hold his breath for a varying period and then inhale carbon dioxide to stimulate the respiration center. As a result, inhalation of water may occur. He then cries to help that leads to inhalation of more water causing coughing and air is expelled out. If cerebral asphyxia will continue then death occurs in 3-10 minutes.

Several phases are recognized in the drowning process:

- Breath holding lasts for a variable length of time until carbon dioxide accumulation in blood and tissues reaches a level at which stimulation of the respiratory center in brain leads to inevitable inhalation of large volumes of water.
- Swallowing of water, coughing, vomiting and progressive loss of consciousness follow in rapid succession. Escape of air remaining in the lungs and replaced by water leads to next phase.
- Profound unconsciousness and convulsions associated with gasping precede respiratory standstill, which is followed by failure of heart, irreversible changes in the brain and death supervene within a very short time.

Types of Drowning

Drowning is of following four types:

1. *Wet drowning*: In this type of drowning, more water goes to lungs.
2. *Dry drowning*: In this type of drowning, no water enters into the lungs and death occurs by laryngeal spasm.
3. *Secondary drowning*: It is post immersion syndrome and is also called near drowning. Death

occurs after several episodes of resuscitation due to electrolytic disturbances such as metabolic acidosis. Lungs show haemorrhages and atelectasis. Myocardial anoxia may occur and may cause delayed heart failure.

4. *Immersion Syndrome (Hydrocution)*: Death in immersion syndrome is due to cardiac arrest as a result of vagal inhibition. It is produced by (i) cold water stimulation by water striking the epigastrium and (ii) by cold water entering the ear drums, mucosa of pharynx or Larynx. Alcohol increases such effects due to general vasodilatation.

Pathophysiology of Drowning

Pulmonary alveolar lining is semi permeable and if water enters the alveoli, exchange of water takes place through the alveoli. The extent and direction depends upon the gradient between the osmotic pressure of blood and the water.

Drowning in Fresh Water or Brackish Water

In fresh water (NaCl content is 0.6%), water passes rapidly from the lungs to the blood, haemolysis and dilution of blood and volume increases 2.5 liters or more. Because of low salt content of these waters (brackish water has approximately 0.5% salinity), precipitous absorption in to the circulation takes place. This results in hemolysis and dilution of blood constituents associated with an abrupt violent increase in blood volume. Fresh water alters or denatures the protective surfactant, which lines the alveolar wall. Loss of or inactivation of pulmonary surfactant and alveolar collapse decrease lung compliances causing severe ventilation perfusion mismatch up to 75% of the blood perfusion non-ventilated areas. When water is inhaled, vagal reflexes cause increased peripheral air way resistance with pulmonary vasoconstriction, pulmonary hypertension and increase in lung compliance and ventilation perfusion and increased Na^+ and K^+ concentration in serum leading to over burdening of heart causing pulmonary edema. The heart is rapidly overburdened by this increased load and pulmonary edema becomes manifest.

Arrhythmias of heart beat, which culminate in ventricular fibrillation leads to death within 3-5 minutes from the start of submersion. In heart, there is hypoxia with decreased Na^+ and K^+ causing cardiac arrhythmias in the form of ventricular fibrillation and tachycardia and haemodilution leads to haemolysis causing haemoglobinemia and haemoglobinuria.

Drowning in Sea Water

In sea water (NaCl content is 3%) due to high concentration, water is drawn from the blood in the lung tissue causing severe pulmonary edema, hypernatremia and haemoconcentration. In contrast to fresh water, seawater is strongly salty (usually over 3.0% NaCl conc.). Due to this high salinity, fluids are drawn from blood in to lung tissue causing severe pulmonary oedema. At the same time, there is an attempt to re establish osmotic balance, salts from sea water in the lungs pass to the blood stream with slow death from Asphyxia. Death occurs in 4-8 minutes. Overall, death may occur by asphyxia, ventricular fibrillation, cerebral anoxia, injuries caused during drowning, vagal inhibition and laryngeal spasm. Saltwater drowning usually lasts longer and hence is likely to be more amenable to resuscitation than drowning in fresh water. The different mechanisms in freshwater versus saltwater drowning has significant implications with respect to blood alcohol concentration of the victim. In freshwater drowning dilution of blood may be expected to cause a decrease in the alcohol level, whereas in saltwater drowning a slight increase occurs.

Diagnosis of Drowning

Drowning is mostly accidental. In a relatively small number of cases, it is suicidal. In some cases, death in the water is due to natural causes, such as heart attack or cerebral haemorrhage during bathing or swimming. Disposal of victim of a homicide to simulate drowning is rare yet because of such possibility, distinction between changes due to drowning and those resulting from post-

mortem immersion of the body is of prime importance.

There is no reliable test permitting an equivocal diagnosis of drowning that are available. Thus, the central question in the case of a body recovered from water is whether the individual was alive at the time he entered the water. The scene, the circumstances surrounding the incident with all available background information, the deceased's clothing all play a significant role for example the finding of an alcoholic floating in water 20ft below the cliff with his zip open suggests that he had been urinating before he fell. The rare instance of hand clutching weeds concludes that the deceased may have struggled to stay on surface. The presence of water in the lungs and stomach of a drowning victim is of no real significance. Water may well reach these organs after death. At autopsy, the lungs of a drowning victim commonly resemble those seen in cases of atherosclerotic heart disease.

Drowning in the bathtub occurs only if unconsciousness is brought down by disease (epilepsy, or heart disease) or after consumption of alcohol or drugs. Accidental drowning in the swimming pool sometimes results from jumping off the diving board. Impact of the forehead on the floor of pool may cause hyperextension of the head and loss of consciousness with subsequent inhalation of water. Common autopsy findings in these cases consist of hemorrhage in the deep neck muscles in the region of cervical 1 & 2 vertebra with or without vertebral fractures.

There are cases seen in which young healthy individuals while engaged in underwater swimming died by sudden drowning. A plausible explanation to that is that usually a diver hyperventilate before entering the water, the over breathing followed by exercise under water cause fall in arterial oxygen tension to a level at which consciousness is lost and water is inhaled. Abundant foam is usually noted extruding from mouth and nostrils of a drowning victim. This foam may not be apparent when the body is recovered from water, but it

appears when pressure is applied to the chest. The foam is produced by mixture of air, mucous and water in presence of respiratory movements. Hence, the presence of foam in the airway indicates beyond doubt that the victim was alive at the time of submersion frequently the foam is bloodstained, not as a result of injury to the chest, but due to the tears of the lung tissue by increased pressure within the lungs, which is part of drowning process. When decomposition sets in foam is rapidly changed to a foul smelling, brown somewhat bubbly fluid.

Drowning cases with relatively dry lungs are occasionally observed. Heart action continues in drowning after cessation of respiration, this is sufficient to absorb the water from the lungs in to the bloodstream and as no significant amount of water reaches the alveoli after inhalation is stopped and the same can be seen when resuscitative measures are started immediately.

Spot like hemorrhages in the lungs especially under the pleura are often noted in drowning. These hemorrhages result from over distension of the lungs by drowning liquid, hemolysis and dilution accounts for their pink appearance. An occasional small haemorrhage may be seen in the conjunctiva but multiple pinpoint hemorrhages as seen in strangulation or hanging do not occur in drowning.

Hemorrhage in middle ear and mastoid air cell are occasionally encountered in persons recovered from water. However, this is not specific for drowning, as it can also be seen due to pressure change as in underwater diving or from extreme congestion as seen in other types of asphyxia deaths or heart failure.

Wrinkling of skin of hands and feet seen in individuals recovered from water is frequently referred to as washerwoman's skin. It is not associated with the process of drowning but is due to prolonged immersion. It cannot be used to determine the length of time the body was in water. After a certain time depending on the temperature of water and the length of immersion, the superficial layers of skin peel like a glove, including the nails. In cold water, several days may elapse,

while in warm water several hours may suffice. The glove may be used for fingerprint for identification. In second-degree burns and in decomposition, peeling of skin from hands and feet occurs in similar fashion.

The microscopic findings in the lungs in drowning are not characteristic, yet they differ to some extent depending on whether drowning occurred in fresh water or in seawater.

Differences in Lungs in Fresh Water and Sea Water Drowning

In freshwater drowning, the lungs are over-distended, alveolar walls are torn and structures in the way of the inundating fluid are disrupted. This picture is referred to as 'aqueous emphysema' and the intraalveolar hemorrhage leading to pinkish or red staining of foam in the airway. In seawater drowning, the architecture of lungs is often better preserved and aqueous emphysema is less pronounced.

Laboratory Diagnosis of Drowning

- **Carrara in 1902** established disproportionate dilution of left heart blood in freshwater versus saltwater based on specific gravity, and freezing point. Also there is difference in electrical activity between the two sides but no definite findings were given.
- **In 1903, Placzela** emphasized the diagnostic value of the specific gravity method.
- **In 1921, Gettler** published a test for drowning from right and left heart chambers, a difference of 25mg/100ml between the chlorides concentrations of the two sides of the heart indicated that the death was due to drowning. In freshwater drowning the chloride level in the blood in the left side of the heart was lower than in the right side; the reverse situation was noted in saltwater drowning. Positive is seen in seawater drowning due to hemoconcentration and negative in fresh water drowning due to hemodilution.
- **In 1944, Mortiz** suggested magnesium as being reliable than chlorides particularly for

seawater drowning. proposed that plasma concentration of magnesium differed between the left and right side of heart due to absorption of the ion from the drowning water especially if it is seawater.

- **In 1955, Freimuth et al** using specific gravity of heart plasma, concluded that negative differences between the left and right side may be obtained either in drowning or non drowning cases, while positive values usually indicates that the death was caused by means other than drowning.

There are considerable doubts about the reliability of these chemical tests.

- **In 1941, Incze** published the findings of Diatoms in the lungs and systemic circulation of individuals recovered from water as a diagnostic test for drowning. The portal of entry of diatoms in to bloodstream was believed to be via lungs through microscopic tears of alveolar walls that occur in the process of forceful water inhalation.
- In 1963, the value of this method was questioned by recovery of diatoms in the liver and in other organs of individuals who had died of cause other than drowning. These diatoms may have been absorbed through GI tract when the water containing diatoms was drunk.
- Quantitative assays of peroxidase in animal lungs yielded value close to 800units in freshwater drowning and 6400units in saltwater drowning.

Postmortem Findings in Drowning Deaths

A body in water will sink unless air trapped among the clothes keeps it afloat. The body will resurface when gas is formed because of putrefaction. Putrefaction in water proceeds at a considerably slower rate than in the air but once the body is removed from water, postmortem changes progress far more rapidly than is usual under the same prevailing environmental conditions. Putrefaction is slower in seawater compared to freshwater because high salinity retards bacterial

growth. The time until reappearance of the body depends on the temperature of water. It may take two to three days in summer and weeks or months in winter. While the body is underwater, it is subject to injuries by wildlife or by drifting along the ground. These injuries be recognized and be distinguished from injuries sustained during life. A victim of drowning tends to bleed more readily and more blood oozes from a wound of such an individual. Postmortem lividity is difficult to recognize in a body recovered from water due to swelling and loss of translucency of the upper layer of the skin, the skin becomes waterlogged if immersion is prolonged. Algae grow on the exposed part giving prolonged green or black colour to those areas. Adipocere formation is common in victims of submersion.

Caisson disease: It occurs in underwater diving when the diver remains underwater for prolonged period at great depths due to change of ambient pressure cause formation of bubbles in his blood and tissues if the ascent is too fast. At autopsy, the finding consistent of foci of ischemic necrosis especially in brain, lungs show haemorrhage, edema, atelectasis and areas of emphysema.

Signs of Immersion

1. Maceration of skin begins with minutes in warm water, such as death in bath tub, but in cold water is visible after variable time; the minimum probably being 4-5hours. The first sign tOends to be on areas with an appreciable keratin layer such as fingertips, palms, back of hands and then soles where the surface becomes wrinkled, pale and sodden, the so called 'washer women's skin'. After some days in warmer water and up to several weeks in cold, the thick keratin of hands and feet becomes detached and eventually peels off in 'glove and stocking' fashion. The nails and hair becomes loosened at the same time.
2. Cutis anserina or goose flesh: It is a common finding in immersed bodies but is related to cold water than warm water. The erector pilae

muscles attached to each hair follicle can contract in any type of death and cause a generalized dimpling of skin.

3. The distribution of postmortem hypostasis: It is usual for most corpses to float or hang in water with uppermost and the head and limbs hanging down but water movement often roll the body constantly unless in a placid lake. Other corpses may float upon the back and gravitational staining of the skin may therefore be in any pattern if the body is subject to frequent change of posture.
4. *Estimation of duration of immersion*: This is another difficult problem and one that is often too dogmatically answered by doctor with insufficient experience to appreciate the potential error. The overriding variable factor is water temperature that has most common effect on decomposition. When a body falls in to water in average temperature climate the following is an approximate guide to timing:
 - i. If no wrinkling of finger pads is present, less than few hours.
 - ii. Wrinkled fingers, palm and feet progressively from half a day to three days.
 - iii. Early decomposition often first in the dependent head and neck, abdomen and thighs is 4-10 days.
 - iv. Bloating of face, abdomen with marbling of veins, peeling of epidermis on hands and feet is 2-4 weeks.
 - v. Gross skin shedding, muscle loss with skeletal exposure, partial liquefaction in 1-2 months.

These times may be reduced or exceeded by wide margin according to animal predation, climatic changes and bodily built.

Other Signs of Drowning

During the autopsy at times it poses problems as findings are minimal, obscure or completely absent. In such cases, the cause of death may be given as (i) Consistent with drowning or as (ii) Cause cannot be given.

External

1. Clothing is wet and skin is wet, cold and clammy and pale due to vasoconstriction.
2. Post mortem staining is pink due to oxygenation, may be dusky and cyanotic.
3. Head, neck and face, upper part of chest and upper arm show postmortem staining.
4. Cyanosis may or may not be there.
5. Conjunctiva is congested and petechial haemorrhages may be present.
6. Pupils may be dilated and the tongue is swollen and protruded.
7. Rigor mortis appears early, when violent struggle takes place before death.
8. The froth is fine, leathery, tenacious and white or mixed with blood over mouth and nostrils into pulmonary bleeding and is more on pressing the chest. Inhalation of water irritates the mucosa, membrane and stimulates the secretion of mucous. Air with mucous and water creates a churning effect and causes the froth. Fine bubbles don't easily collapse when touched with the points of a knife. It may project as a small balloon or mushroom like mass or a cured horn from the mouth and nostrils. The froth may not be there in dry drowning.
9. *Cutis anserina or goose skin*: Skin has granular and puckered appearance due to spasm of erection fibers and the muscles attached to hair follicles. It can occur in living in cold weather and it may occur in submersion of body in cold water of the death during molecular death. Retraction of scrotum and penis occurs.
10. *Cadaveric spasm*: Presence of weeds, ground and grass. It indicates the person was alive when drowned. Damaged nails and abraded fingers may be present due to struggle.
11. *Washerwoman's hand*: Soddening of the skin due to absorption of water into its outer layer. First in fingertips, then in 2-4 hours it spreads

to palm and back of the hand in 24 hours. Similar changes are seen in the foot. If shoes are there, it takes twice as long. Skin is bleached, wrinkled and sodden and then turns white in colour.

Internal

Internally lungs show the main features. Macroscopically they are voluminous, completely covering the pericardial sac and bulge out of the chest when the sternum is removed. Passive collapse, which usually occurs in death, does not occur due to the fact that edema fluid blocks the bronchi. Due to distension rib impression on surface of lungs is seen and when they are removed and kept on a table they don't collapse.

On cut section they may appear dry, but large amount of water, sometimes foamy, ooze from the surface on squeezing. If lungs are left alone, water will start to trickle from the surface and it will slowly collapse because of increased pressure during forced expirations, alveolar walls may have ruptured, producing hemorrhages, when located subpleurally called paultaff's hemorrhages. They are shining, pale bluish and may be minute or 3-5 cm in diameter.

1. *Paltauff's haemorrhages*: Paultaff's haemorrhages are usually present in the lower lobes of the lungs. They may be seen on the anterior surface and interlobar surfaces of lungs. They are red or grey patches and they may be seen on the surface due to paultaff's haemorrhages and interstitial emphysema. They may however be absent. This condition of the lung is called 'Emphysema aquosum' and 'trockenes oedema'. This drowning lung is fairly characteristic but not pathognomic. Active respiratory movements were a prerequisite for the drowning water to reach all the peripheral parts of lungs. After death, also water could not reach the lungs by hydrostatic pressure when the body is submerged but the amount of water would be small and it would gravitate only the lower parts of the lungs. Therefore, hydrostatic lung cannot simulate drowning lung. Muller, Reh and Eisele,

using high pressure chamber or lowering the body in normal water shows that hydrostatic lung may develop that cannot be distinguished from drowning lung. Thus drowning lung was developed by putting the body 3 meters deep for 65 hours (death is due to hanging) and even 2 meters depth for 20 hours may produce drowning lung.

Therefore, **Reh** was of the opinion that drowning lung is diagnostic for drowning only if the (i) Body has remained floating on or very near the surface (ii) Drowning lung with frothy fluid over mouth and nostrils should not leave any doubt about the diagnosis of drowning.

2. *Microscopic findings of drowning lung*: The microscopic findings after haematoxylin and eosin staining does not yield much about the diagnosis. Following stages are encountered: *Stage 1*: Thickness of the alveolar wall (normally 2-3 times the capillary width) is reduced to capillary width. Capillary appear round or oval like a chain. Partial fiber ruptures that may be observed.

Stage 2: Alveolar are more distended and capillaries lie separately. Fiber rupture is more marked.

Stage 3: Maximal distension of alveolar walls. Capillaries appear thread like only occasional lumina are observed. Distinct intraseptal fiber rupture is there.

Stage 4: This stage is seldom seen and may involve only scattered alveoli. There is complete rupture of alveolar walls and retraction of the fiber like a corkscrew.

Differential diagnoses of drowning lung:

- i. Aspiration of blood.
- ii. Capillary bronchitis.
- iii. Fatal Asthmatic attack.
- iv. Hypoxia for strangulation and throttling.
- v. Chronic emphysema.

In hydrostatic lung, reticular fibers show different picture. The fibers do not withstand hydrostatic pressure. Therefore, the fibers show coarse rupture and fragmentation. No stage of 2 and 3 of drowning lung.

3. *Water in Stomach and duodenum:* Water is present in the stomach and duodenum. Much amount of water may be swallowed which passes in to the duodenum. It does not enter into the stomach after death as a result of the hydrostatic pressure. If a body is kept at a depth of 15 meters for 65 hours then no water enters the stomach. So it is very vital sign that the person entered the water when he was alive and no significant sign of drowning. The fallacy is that the deceased might have drunk water prior to death or taken beverages before drowning in water. Therefore, absence of water proves nothing.
4. *Froth in air passages:* The positive signs of drowning as opposed to mere immersion are scanty and not specific. The most useful is which in fresh bodies, often exudes through the mouth and nostrils. The froth is oedema fluid from the lungs and consists of proteinaceous exudates and surfactant mixed with the water of the drowning medium. It is usually white but may be pink or red-tinged because of slight admixture with blood from intrapulmonary bleeding. The froth extends in to trachea, main bronchi and smaller air passages.
Differential diagnosis of Froth in drowning:
 - i. Strangulation.
 - ii. Acute Pulmonary edema.
 - iii. Electrical shock.
 - iv. Epilepsy.
 - v. Opium poisoning.
 - vi. Putrefaction.
5. *Over-inflation of lungs:* Apart from generalized water logging, the lungs may be markedly over inflated, filling the thoracic cavity when the sternum is removed. The texture is rather pale and crepitant, superficially resembling that in asthma. The older name for this condition is 'emphysema aquosum'. In drowning on the lateral surfaces of the lungs impression of the ribs, leaving visible and palpable grooves after removal of the organs from the thorax.
6. *Other organs:* There are no reliable autopsy changes in drowning. The heart and great veins

have often been said to be 'dilated and engorged' with fluid blood especially the right side but this is a nonspecific finding. The stomach may contain watery fluid or even foreign materials from the water such as slit, weed or sand but this cannot be accepted as positive aid in the diagnosis. Haemorrhage in to middle ear has been postulated as a positive sign of drowning, but this is non-specific and occurs due to change in hydrostatic pressure as depth increases or due to congestion.

Diatoms and Diagnosis of Drowning

In 1941 **Incze** demonstrated that, diatoms could enter the systemic circulation via the lungs during drowning. All waters, fresh or salt water contain microscopic plants, the algae. The algae do not grow or grow only in small numbers in heavily polluted waters. Such water may also contain particulate matter characteristic for local pollution but have the algae are considered.

Revenstrof in 1904 was the first to attempt to use diatom as a test for drowning. The basic premise is that when a living person is drowned in water containing diatoms, many diatoms will penetrate the alveolar walls and be carried to distant organs such as brain, kidneys, liver and bone marrow. After autopsy, samples of these organs can be digested with strong acid to dissolve soft tissue, thus leaving the highly resistant diatom to be identified under microscope. When a dead body is deposited in water or when death in the water is not due to drowning then although diatom may reach lungs by passive percolation, the absence beating heart prevents circulation of diatoms to distant organs. The great advantage of the diatom test, if it were reliable would be a positive diagnosis of drowning could be made even in the frequently putrefied bodies that are removed.

There are two main types—green algae and diatoms that are supplied with a delicate and beautiful heat and acid resistant siliceous shell, which is unicellular. Cell wall is usually strongly impregnated with silica and contain chlorophyll and diatoms a brown pigment. Diatom secretes

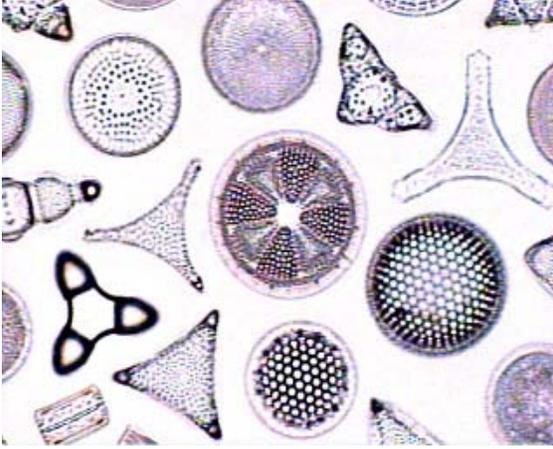


Figure 23.21: Diatoms

hard siliceous outer box like skeleton called frustule. They resist heat and acid and vary in size from 2 microns to 1 mm though most of them are 10-80 microns in size and 10 microns in width. Diatoms upto the size of 60 microns can enter the pulmonary circulation during drowning. They vary from place to place and there is seasonal variation in the same place. They occur in cultivated soils and surface of moist rocks. Large numbers of free-floating diatoms are found in seawater and fresh water. Their shape is circular, triangular, oval, linear or rectangular (Fig. 23.21). They can be demonstrated by the following:

Direct microscopic examination: This is only used in the examination of the lungs. Water is squeezed out of the lungs and centrifuged and the sediment is examined.

Microscopic examination of tissue sections: Optically empty sections are prepared and quantitative analysis is done.

Chemical digestion: The tissue is prepared with acids where by all organic matter is digested, but the siliceous shells of the diatoms are well preserved.

Incineration: Organic matter is destroyed with heat in an oven (Fig. 23.22).

Principle of diatoms: The drowning fluid and the particles in it i.e. diatoms and planktons pass from the ruptured alveolar wall into the lymph channels and pulmonary veins and left side of heart. Only in living body the diatoms are found in bone marrow, brain, liver and skeletal muscle etc. its also found in bile and urine etc. bone marrow is highly suitable and reliable after cutting the bone the marrow is curetted from the gutter.

There can be situations where the diatoms may be absent even though the person is known to have drowned or they are present without drowning.

- **Seasonal variation:** a person may have drowned when the water is almost devoid of diatoms so no diatoms are found.
- A person may eat raw fruits or vegetables, which have been in contact with soil diatoms and shellfish, which feed on diatoms and show diatoms in the body.
- Large amounts of diatoms are found in materials used for manufacturing of buildings like cement, paint, paper, insulators etc. the atmosphere near the plants providing such material is highly polluted and the person might have inhaled and may have such diatoms in his body.
- Certain dusting powders contain diatoms, which may have been inhaled by the person and are found in his body.
- Diatoms are ubiquitous, being present in soil, water supplies and in the air.
- They can also penetrate the intestinal lining and gain access to the bloodstream and hence any body tissues.
- Certain food, notably shellfish contains vast quantities of diatoms that may enter the circulation.

According to Henday, following two requirements to be fulfilled:

1. All the species recovered from the organs of the body must be present in the samples of water in the place of submersion.

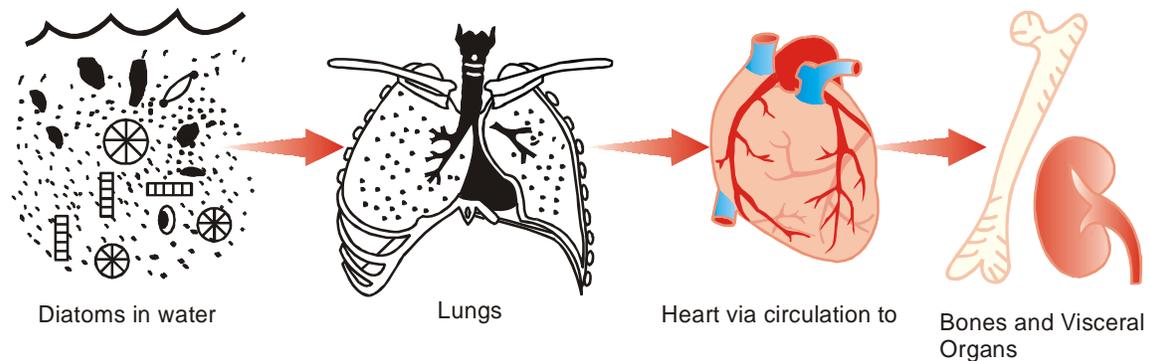


Figure 23.22: Transmission of Diatoms

2. They must be present in approximately the same proportion in the organ as in the sample from the place of submersion.

Histological Changes in Drowning

Much of the work has been done to confirm histologically the genuineness of 'emphysema aquosum' in both fresh and decayed bodies, but the dilation and rupture of the terminal air space can occur not only as a result of drowning but of passive immersion in water deeper than 4m. Dilatation of the alveoli, thinning of the walls and compression of the capillaries are easy to obscure but the significance is ambiguous. The number of macrophages in the alveoli adjusted to the size of the alveoli where distended was studied by Betz, who carried a diagnostic usefulness of the method as long as no autolytic changes are present. Haffner has used the weight of spleen as a diagnostic marker in drowning compared with other modes of asphyxia about an 18% reduction in weights and spleen-liver ratios were recorded. The skin changes in maceration are also well recorded.

Long Questions

1. Define asphyxia. Discuss the various types of hanging and the autopsy findings in a case of hanging.

2. Classify asphyxia. Discuss the types of homicidal asphyxia with the autopsy findings in manual strangulation.
3. Discuss the various types of drowning with the autopsy findings in fresh water drowning.

Short Questions

1. Hanging
2. Strangulation
3. Bansdola
4. Sexual asphyxia
5. Dry drowning
6. Diatoms
7. Judicial hanging
8. Cafe coronary
9. Gettler's test
10. Suffocation
11. Throttling
12. Mugging
13. Garroting
14. Burking
15. Paultauf's hemorrhages
16. Differences between fresh water and sea water drowning

MULTIPLE CHOICE QUESTIONS

1. **Hyoid fracture is common in:**
 A. Hanging
 B. Strangulation
 C. Throttling
 D. Choking
231. **At autopsy, a body is found to have copious fine leathery froth in mouth & nostrils which increased on pressure over chest. Death was likely due to:**
 A. Epilepsy
 B. Hanging
 C. Drowning
 D. Opium poisoning
232. **Gettler's test is done for death by:**
 A. Drowning
 B. Hanging
 C. Bums
 D. Phosphorus poisoning
233. **Feature indicative of ante mortem drowning is:**
 A. Cutis anserina
 B. Rigor mortis
 C. Washerwoman's feet
 D. Grass and weeds grasped in the hand
234. **Best indicator of ante mortem drowning is:**
 A. Froth in mouth and nostrils
 B. Cutis anserina
 C. Washerwoman's hand
 D. Water in nose
235. **Paltauff's haemorrhages may be seen in:**
 A. Hanging
 B. Drowning
 C. Strangulation
 D. Carbon Monoxide poisoning
236. **Goose skin or cutis anserina seen in:**
 A. Drowning
 B. Lightening
 C. Strangulation
 D. Fire arm injury
237. **Which of the following is not true regarding vascularity of lung:**
 A. Hypoxia causes vasodilatation
 B. Distended capillaries in lower lobe
 C. Increased perfusion of apical lobe
 D. Pulmonary resistance is half of the systemic vascular resistance
238. **Fine leathery froth that emanates from the nostrils on chest compression is diagnostic of death due to:**
 A. Drowning
 B. Hanging
 C. Morphine poisoning
 D. Strangulation
239. **In judicial hanging fracture of vertebral column is seen between:**
 A. C1 and C2
 B. C2 and C3
 C. C4 and C5
 D. C5 and C6
240. **Most important sign of death due to strangulation is:**
 A. Signs of asphyxia present
 B. Fracture of hyoid bone
 C. Horizontal ligature mark with ecchymoses round the edges
 D. Fracture dislocation of cervical vertebrae
241. **Which of the following is characteristic feature of strangulation:**
 A. Fracture of thyroid cartilage is present
 B. Fracture of cricoid cartilage is present
 C. Fracture dislocation of cervical vertebrae
 D. Extravasation of blood under the ligature mark and rupture of neck muscles
242. **Ante mortem hanging is best indicated by:**
 A. Dilated pupil
 B. Protruded tongue
 C. Patechial haemorrhages with congested face
 D. Oblique ligature mark which does not completely encircle the neck

1 B 2 B 3 A 4 A 5 A 6 B 7 D 8 A
 9 D 10 B 11 D 12 A

- 243. Which of the following is not true about fresh water drowning:**
- A. Hyperkalemia
 - B. Hypovolemia
 - C. Haemolysis
 - D. Ventricular fibrillation
- 244. In strangulation there is:**
- A. External signs of asphyxia are not well marked
 - B. Neck muscles rupture is common
 - C. Fracture dislocation of cervical vertebrae
 - D. All of the above
- 245. Surest sign of death from hanging is:**
- A. Signs of asphyxia well marked
 - B. Dribbling of saliva from angle of mouth
 - C. Bleeding from nose & mouth
 - D. Neck stretched & elongated
- 246. Diagnostic feature of ante mortem hanging is:**
- A. Dribbling of saliva
 - B. Ligature mark
 - C. Hyoid fracture
 - D. Incontinence of urine
- 247. Dry drowning is:**
- A. Due to sudden laryngeal spasm
 - B. Due to sudden immersion in cold causing cardiac arrest
 - C. Occurs in dry well
 - D. Same as wet drowning
- 248. Fracture of hyoid bone occurs in:**
- A. Drowning
 - B. Hanging
 - C. Strangulation
 - D. Traumatic asphyxia
- 249. Diatoms in bone marrow are seen in death due to:**
- A. Drowning
 - B. Electrocution
 - C. Asphyxia
 - D. Strangulation
- 250. Dry drowning is:**
- A. Water does not enter larynx due to laryngospasm
 - B. It is common than wet drowning
 - C. Common in bronchial asthma
 - D. If resuscitated patient does not have pleasant recollection
- 251. Most reliable evidence of death due to drowning is:**
- A. Froth in mouth and nostrils
 - B. Water in stomach
 - C. Voluminous oedematous lungs
 - D. Weeds and grass firmly grasped in hand
- 252. Ante mortem drowning is best demonstrated by:**
- A. Cyanosis
 - B. Diatoms in stomach and lungs
 - C. Froth in mouth and nostrils
 - D. Washerwoman's skin
- 253. Presence of fine white leathery froth in mouth and nostrils is seen in:**
- A. Hanging
 - B. Bums
 - C. Scalds
 - D. Drowning
- 254. Tardieu's spots are seen in death due to:**
- A. Haemorrhage
 - B. Vasovagal shock
 - C. Brain tumor
 - D. Obstruction of respiratory passages
- 255. Drowning is absent if there is no:**
- A. Froth in mouth and nostrils
 - B. Fracture hyoid bone
 - C. Cutis anserina
 - D. Water and diatoms in lungs and stomach
- 256. In strangulation, there is:**
- A. External signs of asphyxia are not well marked
 - B. Injury to neck muscles is rare
 - C. Subcutaneous tissue under the mark is white, hard and glistening
 - D. Abrasion and ecchymoses round about edges of ligature mark
- 257. 'Cutis anserina' or 'goose flesh' is seen in death due to:**
- A. Criminal abortion
 - B. hanging
 - C. Drowning
 - D. Strangulation
- 258. Burking is smothering and:**
- A. Throttling
 - B. Strangulation
 - C. Gagging
 - D. Traumatic asphyxia

- 259. Seen in drowning are following except:**
- Weeds in stomach and lung
 - Wet heavy, lungs
 - Frothing from mouth
 - Miosis
- 260. Diatom test is to determine death due to:**
- Drowning
 - Strangulation
 - Hanging
 - Bums
- 261. Hanging causes large amount of injury to:**
- Vertebral artery
 - Carotid artery
 - Trachea
 - Oesophagus
- 262. In child drowning, most common consistent feature after taking out from pond is:**
- Water in lungs alveoli
 - Patechial haemorrhages
 - Washer woman skin
 - Cyanosis
- 263. Which of the following is diagnostic of drowning:**
- Froth in the nostrils
 - Cutis anserina
 - Water in the stomach
 - Mud in the respiratory passages
- 264. The type of hanging with haemorrhages on the dependent parts of legs is seen in:**
- Partial hanging
 - continued postmortem hanging
 - Judicial hanging
 - Accidental hanging
- 265. Ligature mark is:**
- Laceration
 - Abrasion
 - Ecchymoses
 - Potential margin
- 266. Tardieu's spots are seen in:**
- Drowning
 - Electrocution
 - Mechanical asphyxia
 - Corrosive acid poisoning
- 267. In death due to hanging:**
- Trachea is obstructed by a weight of 5 kg
 - Usually the vertebra to get fractured is C3
 - Abduction fracture of hyoid may be seen
 - All are true
- 268. Ligature strangulation is also called:**
- Mugging
 - Gagging
 - Garroting
 - Throttling
- 269. Café-coronary refers to:**
- Myocardial infarction in cafeteria
 - Myocardial infarction due to intake of large amount of coffee
 - Choking
 - Strangulation
- 270. Hydrocution refers to:**
- Electrocution in water
 - Vasovagal arrest due to stimulation by cold water
 - Touching live with wet hands
 - All of the above
- 271. Emphysematous bullae are seen in lungs in case of:**
- Drowning
 - Electrocution
 - Strangulation
 - RTA
- 272. 'Intimal tears of carotid' are usually seen in:**
- Ligature strangulation
 - Mugging
 - Judicial hanging
 - Partial hanging
- 273. Which of the following postmortem findings suggest that the person was conscious and alive at the time of falling into water?**
- Emphysema aquosum
 - Edema aquosum
 - Washerwoman's hand
 - Postmortem staining on the face and shoulders
- 274. Beveling of inner table of skull suggests:**
- Stab wound
 - Wound of firearm entry
 - Wound of exit of firearm
 - Penetrating wound
- 275. Le facies sympathieae is seen:**
- At the side of knot in hanging
 - Usually in strangulation
 - Opposite the side of knot in hanging
 - Also in drowning
 - Any of the above

Infanticide

Infanticide is unlawful killing of a newborn child below the age of one year. It was a recognized practice in many primitive communities. This problem persists throughout the world and took place for a variety of reasons in the past. It does not include death during labour by craniotomy or decapitation.

Motives of Infanticide

Uncivilized societies: The motives in uncivilized societies could be

1. The basic motive is survival of the fittest with destruction of those suffering from malformation and females being of less potential value to the community are killed.
2. Some newborns are killed due to tribal superstitions due to the question of being unlucky such as twin births, foetus that have teeth at birth and those with leg presentations.
3. Due to the age long desire for strength and fertility some communities believe that destruction and eating the first newborn child added strength to those who devoured it.

Civilized societies: The motives in civilized societies are usually

1. To get rid of illegitimate children.
2. To get rid of child when mother is widow or unmarried.
3. When the parents belong to socially and economically weaker section of the society.

Laws in Relation to Infanticide

The infanticide law differs in different countries but its evolution in England is of particular interest. Until 1922, the unlawful killing of newborn was murder, no matter by whom it is done. This made no allowance for the fact that the effects of delivery may temporarily disturb the balance of the mother's mind and she would not then be responsible if, at that time, her act or omission led to the destruction of her child.

1. **The Infanticide Act (1922):** It was passed that made provision of the act of infanticide being committed by the mother with disturbed mental balance under the effects of delivery. This was applicable only to England (not applicable to Ireland or Scotland). This Act also did not take into account lactation and no age limitation was defined.

In 1926, this Act was re-enacted and defined the age limit that is the child under 12 months.

There are some points that should be stressed

- i. It is only the mother who can be charged with the offence.
- ii. The child must be borne alive (of course be viable)
- iii. It must have been killed.
- iv. There must be evidence to establish that the mother is suffering from the disease of the mind for the reason mentioned.

The Act clearly says that it is applicable only to the mother and anyone else including the

husband will be charged with *Murder or Manslaughter*.

2. **Infanticide Act 1938:** Section I enacts that “Where a woman by any willful act or omission causes the death of her child being a child under the age of 12 months, but at the time of the act or omission the balance of her mind was 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, then, notwithstanding that the circumstance were such that but for this Act the offence would have amounted to murder.” She is guilty of the felony of infanticide and may be dealt with and punished as if she had committed manslaughter. This Act talks of the ‘balance of mind was disturbed at the time of the act’. This being a broader term needs to be proved by the doctor who is conducting her obstetrical check-up and the psychiatrist.

In pursuance of Section 60 “Offence against the Persons Act, 1861” for which the child is deemed to have been recently born, if it has been born within 12 months before its death.

In every case of infanticide, legal presumption is that the child is born dead, and the onus of proving that the child was born alive rests on the crown. There is no need to produce the body and it is not necessary to prove that the child has been viable.

3. **Infant Life (Preservation) Act 1929:** The law was amended with regard to the destruction of children at or before labour. This Act defined the crime of child destruction “Any person who, with intent to destroy the life of a child capable of being born alive, by any willful act causes a child to die before it has an existence independent of its mother, shall be guilty of felony, to wit of child destruction and shall be liable on conviction thereof on indictment to imprisonment for life. Thus, this offence cannot be committed during the earlier stages of pregnancy. The criteria to be fulfilled or the purpose of this Act

- i. The woman must be pregnant for a period of 28th weeks or more.
- ii. There shall be prima facie proof that she was at that time pregnant of a child capable of being born alive.

This Act provides that if the person is not guilty of offence of murder, manslaughter or infanticide, he can be charged to be guilty of child destruction.

4. **Criminal law of England:** According to this law, a child is considered to be born alive when it is completely born external to the mother irrespective of the attachment or severance of the cord. Hence, according to the English Law destruction of an infant before its complete birth has taken place is not homicide. Nevertheless, the definition held by Indian law is more correct and appropriate. It constitutes live birth, even if any part of a living child has been brought forth external to the mother, though the child may not have breathed or completely born.
5. **Laws in India:** The law in India draws no distinction between the murder of the newborn and that of any other individual as the provisions of the law that apply to homicide also apply to infanticide. Hence, according to Indian law, killing of an infant before any part of it is born is not homicide, but destruction of an infant after any part of it is has been brought forth external to the mother, but before its complete birth constitutes homicide. Therefore, the definition of infanticide could be applied not only to the mother but also to the father. Legal bearing is same as culpable homicide except that the law presumes child is born dead. It is for the prosecution to prove that the child is born alive and died from criminal violence. The crime is usually committed at the time or within few minutes or hours after the birth of the child. Signs consistent with a precipitate or difficult labour are impossible to find if more than 3 weeks have passed after delivery. However, immediately after delivery extragenital and genital signs can be discerned. In such cases, the Medical Officer is required to examine the woman.

Examination of the Woman

It should include: (i) Time interval between delivery and examination (ii) Mental status that is general demeanor and emotional state (iii) Signs of recent delivery.

Points in Relation to Infanticide

1. Is the body of the child found that of a viable child?
2. Is the body of that child of the person accused?
3. Was the child still born or dead born?
4. Was the child born alive?
5. If born alive, how long survived?
6. What was the cause of death?

Is the Child Viable?

Viability means capability of having a separate existence after birth by virtue of certain degree of development. It should be explained that unless the child born has reached a stage of development, which is consistent with the possibility of a live birth, the charge of infanticide or child destruction would not be preferred against mother. In law, a foetus which has not attained a completion of 7th month of intra-uterine life is said to be incapable of maintaining a separate existence and therefore non-viable. Where it can be shown that the child was premature, there is a strong presumption of still-birth or death from prematurity shortly after live-birth.

Presumption of law: if the child shows evidence of less than 7th month failed to leave by reason of immaturity. On the other hand, if there is any evidence that the foetus has lived after birth; charge of infanticide will apply. Viability need not be proved, but in doubtful cases, it is improbable that

the child had a separate existence. The Court sometimes requires to know whether the infant had attained the 28th week of gestation at the date on which the pregnancy terminated. The opinion is based on not a single criterion but upon several parameters.

- i. General condition of the foetus: Its plumpness that is fullness or fatness of the body and absence of apparent disease or malformation
- ii. Weight of the foetus: In multiple births, the weight of each of the infants is appreciably less than that of a single birth at the same stage of gestation. The female foetus is 100gm less in weight than the male foetus.
- iii. Crown-heel length of the foetus: It has been established that there is a close relationship between age and height of the foetus. To calculate age from height, crown-heel length is the best criteria (Table 24.1)

Features of 6 months fetus: The height of foetus is 30 cms and weight is 2 lb. The skin is wrinkled and fat is absent. Eyelids are adherent and membrana pupillaris exists. The eyebrow and eyelashes start forming. The testicles are close to the kidney and the meconium is present in upper part of large intestine. In addition, ossification for all the four divisions of sternum is present.

Features of 6 months fetus : The height is 35 cm and weight is 2½-3 lb. the eyelids are separated and eyelashes are present. The membrana pupillaris disappears. The nails are thicker and they do not reach at the end of fingers. Meconium present in whole of the large intestine. The testicles present in external inguinal ring and there is appearance of center of ossification for talus.

Table 24.1: Weight and length of the foetus in relation to age

Age	Weight	Crown-heel length
28 weeks	2½-3lb (1.1-1.3 kg)	35cm
32 weeks	3½-4lb (1.6-1.8 kg)	40cm
36 weeks	About 5lb (2.2 kg)	45cm
40 weeks	6-7lb (2.7-3.2 kg)	50cm

Is the Body of the Child of the Person Accused?

Proof on this point is most usually established for the evidence of persons with whom the suspected woman has more or less intimately associated in work or social life. At times careful examination by the police of the wrappings, which envelop the body of a child, may throw some light on the identity of the mother.

Was the Child Still Born or Dead Born?

A distinction must be drawn between still born and dead born.

Stillborn fetus is defined as one, which has issued forth from the mother after 28th week of pregnancy and did not at anytime after being completely expelled breathe or show any other signs of life. This definition poses some problems in the English law. For example should the mother, whilst one foot of the child is still in the vagina, strangle it or kill it by some other means, even if it has cried since the delivery of the head, she is not guilty of offence, because the child by definition a still birth, not being completely expelled. Cases have occurred that have been acquitted.

From practical point of view it is highly unlikely that a woman who does not know the law would invent the story of incomplete expulsion, hence if she volunteers it in her original statement, it is difficult to repute.

In other aspect, in cases of unidentified children, it is slightly impossible for the pathologist even if he finds evidence of breathing to say that it had a separate existence.

Dead born child: The fetus had died in the uterus and may show one of the following signs after it is completely born:

1. Rigor mortis at delivery
2. **Maceration:** Maceration is a process of aseptic autolysis and is the usual change. If the foetus remains in the uterus for 3-4 days surrounded with liquor amnii, but exclusion of air. It is not seen if the foetus is born within 24 hours after death. If air enters, then putrefaction

will set in. Macerated baby is soft, flaccid and flattens out when placed on a surface level. It has a sweet, disagreeable odour; skin is red or purple.

- i. The earliest sign is slippage of skin that is seen within 12 hours of death of child inside the uterus.
- ii. If there is presence of gas in great vessels (Aorta), it indicates fetal death.
- iii. Large blebs filled with serous fluid are present.
- iv. Epidermis is easily peeled off leaving a moist and greasy area.
- v. The tissues are oedematous, abdomen is distended, and serous cavities are filled with serous turbid fluid.
- vi. Soft parts are readily detachable from the bone.
- vii. There is abnormal mobility of the joints and the small bones are separated.
- viii. All the viscera is softened except lungs and uterus which are unchanged for a long time.
- ix. **Spalding's sign:** Spalding sign is loss of alignment and overlapping of the bones of the cranial vault due to shrinkage of brain. It develops earlier in vertex presentation. It can be detached within a few days of death of the foetus but often takes much longer time like 2-3 weeks.
- x. **Mummification:** Mummification is dried up and shriveled appearance due to deficient blood supply and scanty liquor amnii; no air enters in the uterus.

Was the Child Born Alive?

Separate existence and live birth are the two terms that are not synonymous with each other. The principal requirement of legal birth is that the child had or was capable of having a separate existence independent of its mother.

In cases of child destruction, it is required to be proved that that the child was capable of being born alive. The pregnancy must have reached the 28th weeks or over and that the child was viable.

Further, it must be proved that a willful act caused its death before it had an existence independent of its mother. Viability is the crux of matters of child destruction. Infanticide requires proof that the child has had a separate existence; it had lived after complete extrusion of its body from that of his mother. The viability need not be proved.

However, when the viability is doubtful, it is improbable that the child had a separate existence.

External examination: A separate existence requires complete extrusion from the body of the mother but does not include the severance of the cord and detachment of the placenta. In accidental tear of the umbilical cord, the break is usually close to one of its attachments, either near the placenta or the navel of the infant. At the naval end of placenta little haemorrhage is unlikely to cause death. According to Morris and Hunt (1966), the cord can be easily broken by the hands, a force of 5 kg is required.

The ends of the cord should be examined at a low magnification by placing two portions in water or on a board and their ends are then gently spread out. Gross irregularities suggest tearing whereas a linear break with regular margin is indicative of separation by cutting. A blunt instrument can also produce a ragged line of separation. In addition, the tear can resemble superficially a clean-cut division of the cord. If the body has decomposed or there is severe drying, no opinion can be formed on the mode of severance of the cord.

Internal Examination: It may provide strong evidence of a separate existence. If extraneous material, which could enter only after complete extrusion of the infant, is present either in the air passage or digestive tract.

1. Extraneous material can enter the air passage for a limited distance after death but its entry in to intrapulmonary bronchi is resisted by air in the lungs. Therefore, for material to be demonstrated in secondary bronchi or beyond, it must have been inhaled. It excludes contamination and milking of the material, downwards from trachea and

bronchi by compression of the infant's body. The examination must avoid these artefacts and thoracic contents should be removed by 'no touch technique'. The thoracic viscera is placed on a clean slab or dish. The bronchial contents should be obtained by a clean pipette or syringe. Specimen obtained (material) must be compared with the source of extraneous material; soil or sand and proof obtained that the two samples are identical.

2. Extraneous material may enter in to the oesophagus, stomach and pass beyond even in to small intestine during life but unlikely to reach up to the stomach after death. A sample of extraneous material must be compared with the probable source. The demonstration of food e.g., milk in infant's stomach is suggestive of live birth.

Examination of Respiratory system

The Aeration of Lungs

This is based upon the expansion of the alveoli by postnatal breathing and is ascertained by inspection. However, it does not take into consideration the phenomenon of partial aeration or the fact that the child may cry in utero or breathe after expulsion of the head and before complete expulsion. Proof of breathing is not proof of live birth. There are illustrations of authentic cases in literature where breathing can occur before complete delivery. In addition, breathing is by no means rare while the head of the child is still in the vagina and in the uterus itself (Clouston 1933). About 130 cases have been recorded out of which 122 are authentic. Earliest cases of Lebarius (1596) alleges that the child's cry could be heard from a distance. Ryter (1943) reviewed the literature relating to vagitus uterinus. Vagitus uterinus and vagitus vaginalis is the hearing of the cry while the foetus is in the uterus or vagina respectively. This is due to rupture of the membranes resulting in entry of air in the lungs and the stimulation of the baby. Other signs like movements of the limbs, muscle twitching etc.

Gross examination of lungs: When the lungs are generally distended, soft and crepitant they have been aerated out. Osborn (1953) disproved the belief that size of lungs has any relation to aeration. He has disproved the former belief that small solid lungs are indicative of stillbirth. The lungs fill the thorax in at least three-quarters of all cases whether the child is born alive or not. Osborn also demonstrated that lungs may fail to expand due to bilateral pneumothorax with surgical emphysema, a result of artificial respiration. The presence of Tardieu's spots of asphyxia and evidence of bronchopneumonia are not evident of live birth as they may be seen in the still-born. Occasionally sub-pleural interstitial emphysema may be seen and may represent attempted resuscitation by mouth-to-mouth respiration after

birth. (Table 24.2)

Hydrostatic test: Scheyer first noted hydrostatic test in 1683. The basis of the test is that, even with compression, if the alveoli are compressed they will still contain some air if aerated at all. It is because on breathing the lung volume is increased. The specific gravity of lungs 1040 to 1050 diminishes to 940 after respiration. The test involves the determination of the buoyancy of lungs. The test depends upon the ratio of the specific gravity of the lungs to that of water as a lung containing air is lighter than water and therefore it floats. The lungs should be removed at autopsy with the bronchi, trachea and larynx intact. This 'pluck' is then placed in water, and if it floats, the test is positive. Finally, each lung is cut into 15-20 fragments which are again tested,

Table 24.2: Differences in findings in respiratory system before and after aeration

Features	Before respiration	After respiration
Shape of the chest	Flat, circumference is 1-2 cm less than the abdomen at the umbilical level.	Expands, arch shaped or drum shaped.
*Position of the diaphragm	At the level of 4th rib	At the level of 6th-7th rib.
**Changes in the lungs of the cases	Fills pleural cavities in 75% cases.	Fills pleural cavities in 75% of the volume
Margins	Sharp	Rounded
***Consistency	Dense, firm, non-crepitant and liver like.	Soft, spongy, elastic and crepitant
****Colour and expansion of air sacs	Uniformly reddish brown, bluish or deep violet according to the degree of anoxia. On pressing little froth-less blood comes out	Air sacs are distended with air and expanded raised slightly above the surface. Polygonal areas on the surface—mosaic appearance. On cut section, frothy blood comes out. As blood becomes aerated, mottled or marbled appearances
Blood in the lungs	The circulation is not increased	The circulation is twice after respiration.
*****Weight of the lungs		
i. Fodere's test or Static test: Lungs are ligated across hila and separated	30-40 gm	60-70gm
ii. Ploucquet's test	1/70th of the body weight	1/35th of the body weight

*Abdomen should be opened before the thorax.

Position of diaphragm is affected by decomposition.

**It's not true that the lungs are not expanded in the stillborn.

***The lungs are crepitant in artificial respiration and putrefaction also.

****In artificially inflated lungs, mottling is absent and on cut section little blood with no froth comes out.

*****Both the tests result due to increased circulation and the weight of the lungs is not constant.

and if they float the test is positive (this should exclude irregular and partial aeration). The pieces are then squeezed between thumb and index finger under the surface of water so that air bubbles comes out. If it still floats, then it is wrapped in piece of cloth and squeezed by putting a weight. If these pieces are still floating that means there is presence of residual air that indicates that respiration has been established. Before performing this test, a piece of liver that serves as control is allowed to dropped in water and if the liver also floats the test is of no use.

Fallacies of hydrostatic test:

1. Expanded lungs may sink due to:
 - i. Diseases such as acute pulmonary oedema, pneumonia and congenital syphilis
 - ii. Atelectasis (non expansion)
 - Air not reaching the alveoli due to feeble respiration.
 - Complete absorption of air from the lungs by blood if circulation continued after respiration has been stopped or a state of asphyxia.
 - More air being expelled from the lungs during expiration due to recoil of lung tissue.
 - Obstruction by alveolar duct membrane.
 - iii. Drowning: When birth occurs in a toilet bowl or in to a bucket containing water, the foetus may die due to drowning.
2. Unexpanded lungs may float:
 - i. *Putrefactive gases*: Foetal lungs are resistant to putrefaction due to less blood. Soft, greenish air bubbles of different sizes that shift by pressure sink in water.
 - ii. *Artificial inflation*: In conditions of mouth-to-mouth respiration and blowing air by a tube, the lungs are inflated partially and stomach contains air.
 - iii. Alcoholic fixation.
3. Hydrostatic test is not necessary, if
 - i. Foetus is <180 days.
 - ii. Foetus is a monster.
 - iii. Foetus is macerated or mummified.
 - iv. Umbilical cord is separated or cicatrized.

- v. Stomach contains milk.
- vi. Whole of the thoracic contents float.

Applications of hydrostatic test:

1. If the body is not putrefied and all portions of lung float after squeezing, it suggests complete respiration has taken place.
2. Some fragments of lung float it suggests that partial respiration has taken place.
3. All pieces of lungs sink, suggests that the lungs are unexpanded and the infant has not breathed. In putrefied bodies, the problem is that whether such floating lungs were expanded before decomposition. The sinking portion of lung does not exclude the possibilities of having been expanded because putrefactive changes might have destroyed lung tissue, so that the respired air is squeezed out along with putrefactive gases.

Microscopic examination of lungs:

1. *Preparation of the lungs*: Osborn (1953) stressed the importance of "No touch" technique, which aims at the elimination of artifacts produced by careless manipulation. Thoracic contents should be removed intact by cutting with a scalpel, the parts being controlled by holding the tongue or larynx with the forceps. After fixation for 48 hours, samples for microscopy are taken of whole lung in cross-section.
2. In the past, microscopical examination of the lungs was a secondary line of investigation and was confined almost exclusively to the condition of lining of the air sacs. If these retained a gland like appearance, it was concluded that the child has not breathed and therefore was still born. However, it is now known that the change in the kind of cell that lines the air sacs is not abrupt nor is it coincident with the onset of breathing. This gland like air sacs may be present in the lungs of live-born infants also. This development occurs entirely during intrauterine existence before term. This gland like interpretation is indicative only of prematurity or of some diseased state in the mature foetus e.g., hydrops foetalis that prevents

normal differentiation.

- a. *4-5 month foetus*: Lung has a gland like appearance with thick walled ductules, lined by cubicle or columnar epithelial cells. Part of lung did not float in water.
- b. In stillborn full term foetus, the alveolar spaces have developed and lungs did not float. In typical stillbirths, the sections will show
 - i. Alveoli and bronchi are collapsed.
 - ii. In the aerated lung, the alveoli and the bronchi are more or less expanded.
 - iii. Degree of dilatation of vessels
 - iv. Existence of pathological change.
- c. In live birth full term foetus, lungs float in water but microscopically findings are alike stillborn foetus.
- d. According to Ham, during 5th month, gland like structure is seen. Up to this stage alveoli appear as hollow round epithelial structure lined by cubical or columnar epithelial cells. After 5th month, alveoli separate from each other, capillaries in the alveolar walls bulge in to the spaces. This development occurs inside the uterus, independent of extra-uterine respiration. Alveoli undergoes expansion, fluid is displaced by air. Amniotic fluid is partly absorbed from the alveoli and partly drained through the upper respiratory tract.

Microscopical examination is nowadays the main line of investigation and only reliable means of solving many problems:

1. Some of the foetus are clearly stillborn, and show maceration on naked eye examination. These appearances are uncommon and unless seen soon after the delivery is difficult to interpret ones putrefaction has started.
2. Another 1/3rd children have lung appearances simulating to those who had breathed.
3. Some children showed signs of obstructive emphysema.
4. Rest showed signs of "struggle to breathe". Of these some may be still born and some die soon after delivery.

Signs of Struggle to Breathe

Sequence of changes in both the groups include:

1. Dark fluid blood due to raised carbon dioxide haemoconcentration—"pseudo-clots".
2. Cyanosed, expanded lungs, the expansion is due to: (i) Inhaled liquor amnii, vernix or vomitus (ii) Obstructive emphysema (iii) Oedema with or without air or liquor amnii.
3. Tardieu's spots on the pericardium, pleura, and thymus in both live born and stillborn fetus.
4. Liver swelling due to congestion of the whole organ.
5. Distention of the large bowel with meconium. This may be extruded in to liquor amnii and later inhaled, a point of importance in the recognition of stillbirth because phagocytosis of meconium may be demonstrated in the lungs.
6. Development of oedema of lungs: It is a late change and not found always.
 - Osborn found that the newborn are prone to oedema.
 - It can arise very rapidly i.e., within 1-2 minutes.
 - It is not a terminal event.
 - Acute neonatal oedema is a primary cause of death.

The recognition of oedema may be difficult, if the fluid had been drained out during preparation of sections and cellular contents are very few. In the **Osborn** series the oedema was present in the ratio of live birth (17): still birth (1). The absence of oedema in stillborn is to be expected since a rise of blood pressure after delivery favours production of oedema. The haemorrhagic oedema may be found in some cases since increased capillary permeability permits escape of red cells in fluid in the air sacs.

Other important findings

1. On examination of the sections specially stained to demonstrate fat, it may be seen that the alveolar ducts, the passage leading to air sacs are lined by a membrane which is made prominent by the stain. This membrane may

be a fatty material derived from the vernix (**Ahlstrom 1942**). This was called alveolar duct membrane by Osborn. Nowadays it is well known as hyaline membrane disease which is associated with Respiratory Distress Syndrome (**Davis 1981**).

2. *Desquamation of bronchial epithelium*: Specimen is obtained by 'No touch technique' and thoroughly fixed prior to sampling. Its occurrence is a probable indication of maceration of the lungs, uterine death and stillbirth.
3. *Bronchopneumonia*: It is not an indication of live birth as it is also present alike in stillbirths.
4. *Phagocytosis of meconium*: By the cells that line the air sacs provided artefacts be excluded, is important evidence of inhalation of liquor amnii and thus of still birth.
5. *Contusion of the lungs*: This is subpleural bleeding can be readily distinguished from Tardieu's spots as it more extensive and bears the pattern of rib markings. These result from vigorous attempts at artificial respiration. Osborn finds them in 1:4 live births but in none of the stillborns. These are taken to be good evidence of live birth.

Summary

1. Still birth is permissible when there is (i) Maceration of the infant (ii) Flooding of lungs with liquor amnii: (iii) There is evidence of phagocytosis of meconium by cells lining air sacs (iv) Desquamation of the bronchial epithelium (v) Lower bowel distended with meconium (vi) Small solid lungs of uniformly dark colour do not necessarily indicate stillbirth (vii) Gross oedema makes stillbirth doubtful and probably excludes it.
2. Live birth is probable when: (i) All the lobes of the lung are fully expanded with or without obstructive emphysema (ii) Gross pulmonary oedema (iii) Alveolar duct membrane is present and is widely distributed in the lungs (iv) Pulmonary atelectasis due to obstruction by an alveolar duct membrane is present (v) Pulmonary contusions (Haemorrhagic disease

of the newborn has been excluded).

3. A gland like appearance of alveoli does not exclude live birth but indicates pre-maturity.
4. Tardieu's spots although indicate the struggle to breath are present in both live and stillbirths.
5. Bronchopneumonia is also indicative of both live and stillbirths. History of the case is important to diagnose stillbirth or live birth.

Other Important Findings Suggesting Live Birth

1. **Saliva in the stomach** is suggestive of live birth even though the child has survived a few hours after birth.. If no saliva is found in stomach, indicates stillbirth.
2. **Air in the Gastro-intestinal tract (Breslau's second life test)**: If the air has reached the duodenum, it is a strong evidence of separate existence. In stillbirths, there is absence of air. In artificial respiration, air can enter the stomach. Hajkis (1943) believed the radiological demonstration of air in the stomach and intestine to be a confirmatory sign of respiration. If air has reached the duodenum, he considered this a strong evidence of a separate existence. air was not shown in the stomach and intestine of the still born child.
3. **Unequivocal Neonatal line in the enamel of unerupted teeth** is suggestive of separate existence (**Gustafson 1966**). This would be of considerable importance to establish whether the child really had been born alive and survived, provided off-course the child in this case had lived for longer then it would be possible to detect a neonatal incremental line in the enamel and dentine of all the deciduous teeth and perhaps the first permanent molar. According to **Stack 1960** assuming knowledge of the rate of growth of the hard tissues to know the length of time the child had lived after the birth, incremental lines have a uniform intensity and a regular periodicity. Developing teeth increase in tissue thickness, weight and volume. According to him, weight of dry mineralized tissue is the best criteria.

According to **Euler 1955** by 7 month, dentine of central incisor is 2.9mm in width and that of lateral incisor, canine and molars has 2.4 mm width.

4. **Changes in the middle ear (Wredin's test):** Before birth, middle ear contains gelatinous embryonic connective tissue. With respiration, sphincter at the pharyngeal end of Eustachian tube relaxes and air replaces the gelatinous substance within few hours to 5 weeks. To demonstrate air, middle ear should be opened under water and tegmen tympani to be removed, the test is positive if air comes out.
5. **Changes in kidney and bladder:** Brownish yellow crystals of uric acid in the pelvis of the kidney and bladder and absence of urine.

Other Signs of Live Birth Depending on the Time of Survival

1. **Blood:** By 24 hours, nucleated RBCs disappear and by 3rd month of life foetal haemoglobin (normal 70-80%) is reduced to 7-8% and disappears completely by 6th month.
2. **Meconium:** It is a green viscid substance consisting of inspissated bile and mucous. It is acid in reaction, stiffens and stains the cloth brownish green. It is completely expelled from the GIT within 24-48 hours. In anoxia and breech presentation, it is completely excreted before birth.
3. **Caput Succedaneum:** It is a swelling formed in the scalp over the presenting part on the head during delivery. It contains blood and serum beneath the pericranium. It is limited by attachment of pericranium to the bones of the vault. Caput disappears in 1-7 days.
4. **Cephalhaematoma** is a haemorrhage under the pericranium. It is limited by attachment of the pericranium to the fibrous tissue between the sutures.
5. **Skin changes:**
 - i. At first the skin is bright red and by 2-3 days it becomes darker then brick red then yellow and is normal by 7th day.
 - ii. Vernix caseosa covers the skin in the axilla, inguinal region and neck folds. It persists for 1-2 days. It may be absent at birth and can be removed by washing
 - iii. Skin of the abdomen exfoliates for the first three days.

6. **Findings in the Gastro-intestinal tract:** The air travels in the GIT at the same speed in full term infants as in premature infants. The air reaches the stomach after 15 minutes, small intestine after 1-2 hours, colon after 5-6 hours and rectum after 12 hours. The attempts at resuscitation and bacterial gas formation may cause an error.

7. **Changes in the umbilical cord:** If the cord is attached to placenta, it is suggestive of newborn. If the cord is clamped or tied and there is no evidence of reaction it is newborn. The site of severance and method of severance should both be noted, but whether cut or torn may not always be easy to decide. It is best to remove the cord and examine microscopically in the laboratory. As far as finding of mummification of cord is concerned, it can also occur after death if exposed to air.

The changes in umbilical cord with time are shown below:

Time	Changes in the umbilical cord
2 hours	Blood clots
12-24 hours	Shrinks and dries
36-48 hours	Inflammatory ring forms at the base
2-3 rd day	Shrivels and mummifies
5-6 th day	Cord falls off leaving a raw area
10-12 day	Heals and leaves the scar

8. **Changes in the umbilical vessels:**
 - i. Umbilical artery contraction starts in 10 hours and closes by 3rd day
 - ii. Umbilical vein contraction of left umbilical vein starts on 3rd day and obliterates by 4-5th day. Right umbilical vein closes by 6-7th month of intrauterine life.
 - iii. Ductus venosus (left umbilical vein & common hepatic vein) obliterates on the 5th day.

- iv. Ductus arteriosus obliterates on the 10th day.
- v. Foramen ovale closes by 3rd month, at times 2 years.

CAUSES OF DEATH

1. Natural (i) Causes in the mother (ii) Causes in the newborn
2. Unnatural (i) Accidental (ii) Criminal

Natural Causes

Causes in the Mother

1. Diseases in the mother: (i) Syphilis (ii) Smallpox (iii) Plague
2. Pre-eclamptic toxæmia: (i) Hypertension (ii) Proteinuria (iii) Fits
3. Placenta prævia
4. Abnormal gestation (Ectopic)

Causes in the Newborn

1. Immaturity
2. Debility (lack of genital development)
3. Congenital diseases of heart and lungs
4. Malformations
5. Hemorrhage from umbilical cord, genital organs
6. Post maturity.
7. Neonatal infections.
8. Cerebral trauma
9. Erythroblastosis foetalis: (i) Congenital hydrops foetalis (ii) Icterus gravis neonatorum (iii) Anaemia of the newborn

Unnatural Causes

Accidental Causes

1. During birth:

- *Prolonged labor*: Intracranial haemorrhage and death with or without linear fracture of parietal bones of skull may result when the head is severely compressed against the contracted or deformed pelvis. Subdural haemorrhages are present bilaterally. Rarely extradural haemorrhage may occur. The cause of haemorrhage is rupture of bridging

veins and internal or great cerebral veins and rarely tears of falx cerebri. There is finding of moulding and caput succedaneum are usually seen. In addition fracture dislocations of clavicles and limb bones may be present.

- *Prolapse of cord*: This usually occurs in breech presentations when the cord gets compressed by head of fetus. The fetus dies as a result of asphyxia and on autopsy blood, meconium, liquor amnii, vernix caseosa is found bronchial tubes.
 - *Twisting of the cord*: The twisting of the cord and knots in the cord can cause death by strangulating the fetus. The cord get compressed and there are no findings of abrasions or ecchymosis in the cord.
 - *Injury to the mother*: When the mother abdomen is hit by heavy blows or kicks or fall from height, there may be fracture of skull or rupture of blood vessels and signs of concussion of brain.
 - **Death of the mother**: The child may die with death of the mother and can only be saved when delivered within 5-10 minutes of her death.
2. **After birth:**
- *Suffocation*: The child may die when the fetus membranes cover the head during birth, face gets pressed accidentally on to the clothes or when the fetus is submerged in the blood, liquor amnii or meconium discharges. There are chances that the fetus may survive when covered by membranes only for 20-30 minutes.
 - *Precipitate labor*: Precipitate labor terminating in a disproportionately shorter time than that taken on an average either by a primipara or multipara is called precipitate labor. All the stages of labor are merged in to one and delivery occurs without the knowledge of the mother. Foetus is normal or premature. It usually occurs in multiparous woman with large, roomy pelvis. A woman may deliver unconsciously because the contractile power of uterus is independent

of volition. The child may die from

- Suffocation by falling in to the lavatory pan.
- Head injury and fracture of skull (usually a fall from 30" height and it can occur in a height of 18")
- Haemorrhage from the torn ends of the cord

If birth occurs in the toilet bowl, the infant may inhale the liquid, blood, meconium and vaginal mucus that may be found in the air passages. Microscopical examination of the lungs and examination of the cord on the slide under low magnification. The foreign particles can be seen in the drowning fluid.

In accidental fall usually bilateral subdural haemorrhage is present. The average length of the cord is 50cm that is sufficient to protect the child from falling on the ground. It can withstand the weight of the infant. In these cases, caput succedaneum is not formed. Fissured fracture of skull is present that is limited to the parietal bone and may extend to frontal or squamous part of the temporal (Table 24.3).

Medicolegal Aspects

- The mother or her relative may be accused of infanticide, whilst the death of the foetus may be due to injury, asphyxia etc.
- In cases of infanticide, death may be attributed to precipitate labor.

Criminal Causes

These may be (i) acts of commission (ii) acts of omission.

Due to acts of commission: These are the acts done intentionally to cause the death. The injuries may be found on the face head and neck of the fetus when there are attempts at self-delivery. On examination over the neck multiple circumscribed abrasions with scratch marks of finger nails may be found that may simulate homicide.

- Suffocation:** The usual method employed is closing the infant's nose with two fingers and pushing the jaw upwards with palm in order to occlude airway. Other method can be placing pillow or towel over child space or the face may be pushed down into bed clothing. Since the amount of force is less in these cases, at autopsy there may not be any evidence of trauma. Rarely overlaying, introducing mud, cotton wool, rags into the mouth to kill the child may also be used.
- Strangulation:** Manual or ligature strangulation are very commonly used. The umbilical may also be used as ligature at times.
- Drowning:** The body of fetus may be thrown into well, tank, drains.
- Burns:** This method is used for disposing the body but not used for infanticide purposes.
- Blunt force injury:** In cases of child battering, the head may be dashed against the wall or floor by holding feet. Findings of bruising over the ankles and feet at the site of gripping is seen. Besides this there are findings of subdural

Table 24.3: Differences in findings of head injury due to precipitate labour and blunt force

Features	Head injury due to precipitate labor	Head injury due to blunt force
Bruise	Present on the presenting part of scalp	Present anywhere on the scalp
Laceration fracture	Not present	Usually present
	Fissured fracture, involves the parietal bones, runs downwards to the right angle of sagittal suture	Extensive comminuted fracture, sometimes depressed, affects all the bones of the vault.
Brain	Usually not injured.	Contusions, lacerations and haemorrhages present.

and subarachnoid haemorrhages accompanied by fractures. Extradural haemorrhages in infants are usually limited to single bones since the dura adheres to the skull along to the suture lines.

5. *Fracture and dislocation of cervical vertebra by twisting the neck*: This is seen in cases of child battering when there is twisting of neck.
6. *Wounds*: Occasionally the child may be killed by stabbing or cut throat injuries.
7. Poisoning, rarely

Due to acts of omission or neglect: The woman will be guilty of criminal negligence if she does not take ordinary precautions necessary to save her child after birth. These are acts of unintentional neglect or care:

1. Failure to provide proper assistance during labour resulting in death by suffocation or head injury
2. Failure to tie the cord that results in death from haemorrhagic shock
3. Failure to protect the child from undue exposure to heat or cold.
4. Failure to supply proper food.
5. Failure to clear respiratory passages of mucous/amniotic fluid.

Concealment of Birth

It is an offence in England to conceal a birth and in Scotland to conceal pregnancy. The Offences against the Person Act 1861 (section.60), provides that "if any woman shall be delivered of a child, every person who shall, by any secret disposition of the dead body, whether such child died before, at, or after its birth, endeavour to conceal the birth thereof, shall be guilty of a misdemeanour."

This section makes it an offence for every person who has tried to conceal the birth of the child by any secret disposal of its dead body, at or after its birth. If the person is tried for the murder of the child and acquitted, he may be convicted under concealment of birth, depending upon the evidence. There is no need to prove live birth. It is sufficient that there has been a birth and that the child was dead at the time of concealment.

S. 318 IPC (Concealment of birth): Whoever by secretly burying or otherwise disposing the dead body of a child, whether dies before, during or after its birth, intentionally conceals the birth of such child shall be punished with imprisonment up to 2 years. This Act is similar to the Section 60 of 'Offence against Persons Act' 1861

Abandoning of infants (S. 317 I.P.C.)

If the mother or father of a child under the age of twelve years, or anyone having care of such child, leaves such child in any place with the intention of abandoning the child, shall be punished with imprisonment up to seven years

BATTERED CHILD SYNDROME

Definition

The term battered baby syndrome was coined by **Henry Kempe**. It is also known as the Cafey's syndrome, Child abuse syndrome or the maltreatment syndrome. The Brandel's University (Massachusetts) definition of child abuse is 'Non accidental physical attack or physical injury including minimal as well as fatal injury, inflicted upon children by persons caring for them'. Five classical features of the syndrome are

1. Obscure illness or unexplained injury in infants of up to four or five years-from six or eight weeks, due to.....
2. Repeated abuse, physical hurt over a period of weeks or months by.....
3. Either or both parents, guardian or baby sitter, who.....
4. Fail to report or delay reporting the incidents, and, who, when they do.....
5. Mislead, indeed deliberately deceive, the nurse, doctor over the cause.

Social History in Relation to Family and Parents

These factors appear to be consistent as all social classes may batter their children but it is more common in lower socioeconomic groups. The age of the parents is in early twenties have marital

discords and the family is usually isolated. Sometimes the father may not be the biological father and the child is unwanted usually as a result of failed contraception, failed abortion, and is undesired. The physical provision for the family is good, the standard of mothering is good and the nutrition and well being of the child is good. The intelligence of the parents is quite normal, usually a dominant aggressive father and a less intelligent dominated overstressed mother. The father may have criminal records

There is lack of stability of family in geographical sense. The battering parents were themselves battered children. The act of the child like crying, refusal to be quiet and persistent soiling of napkins precipitates the violence of the parents. In the family of battered children, if one child is battered other will be battered too.

The perpetrator is a low income or low education bracket with mothers of low intelligence with increasing family and domestic strain. The parents are aggressive with demand for discipline and may be schizophrenic

Incidence

Age incidence: Most of the children are below the age of three years; about 70% are less than two years of age, about 55% are less than one year and less than 20% are more than five years of age. The mean age is fourteen months

Sex incidence: The incidence is seen slightly more in boys that is in about 55-63% cases

Incidence of position in the family: Usually there is only one child in the family may be youngest or eldest and is often unwanted may be born before marriage, due to failure of contraception and is usually an illegitimate child.

Race incidence: The incidence of battered baby syndrome is not particularly related to a particular race.

Nature of Injuries

The assault are usually carried out by unaided hands of the adult that is the injuries are

'noninstrumental', but sometimes objects such as feeding bottles, pokers may be used for hitting. The injuries produced are of the following types

1. **Bruises, lacerations and abrasions:** Bruises, abrasions and lacerations are the commonly produced injuries and soft tissue injuries are almost universal with an incidence of 80-100%. The areas most commonly affected are head, face and neck. Bruising of scalp and forehead and underlying skull and brain injuries are produced that is visible on autopsy. Bruising of external ear, cheeks and the lips especially the upper lip is of diagnostic significance associated with laceration of frenulum. The detachment of inner surface of the lip from the gum margin suggests punching or slapping of the mouth. Bruising of the neck and sides of the chest may reveal finger tip pressure marks. The neck may be held on each side in turn to immobilize the face while it is assaulted with the other hand, leaving symmetrical bruises beneath the angle of the jaw. The child may be gripped forcibly on each side of the chest and shaken violently to stop him from crying thus leaving the bruises on the skin of the armpits and lower ribs with multiple fractures of the posterior parts of the ribs. Bruising of the abdomen is most common after head injuries. The rupture of abdominal viscera may occur and externally the classical 'Six penny piece bruises' extending from the breast bone to the pubis are seen. The limbs show bilateral multiple bruising of the forearms, arms and legs.
2. **Bites:** The bites are very commonly produced by the mother that is to be differentiated from the bites by other children.
3. **Eye injuries:** The injuries to the eye that are found in battered baby syndrome are retinal separation, lens displacement, subconjunctival, subhyaloid and retinal haemorrhages
4. **Head injuries:** Head injuries are common in a battered child. There may be presence of external scalp injuries. The fracture of skull along with the brain injuries are most frequent cause of death. The skull fractures are

commonly fissured sometimes a segmental break in the parietal area caused by impact of head against a solid object rather than direct blow against a fixed head. There are cerebral contusions and scattered patches of bleeding in the meninges.

4. **Visceral injuries:** Rupture of liver, intestine and mesentery account for most of the fatalities due to blows over front of abdomen. The visceral injuries are the most common cause of death. The lacerations of the undersurface of liver with gross intraregional bleeding due to tears in mesentery of small intestine are falsely attributed by the parents to an accident.
5. **Skeletal lesions and radiologic appearances:** In all the cases of battered baby syndrome, radiography is all important. A whole body X-ray must be performed before autopsy. It provides two kinds of useful information about general skeletal damage especially of different times of infliction and the characteristic bony lesions. The formation of callous is important in identifying and approximately dating old healing fractures. The characteristic radiographic lesions are the separation of epiphysis especially around the elbow and knee joint although any limb may be involved. There is shearing and elevation of periosteum which is not firmly attached in infants. The subperiosteal calcification in periosteal haemorrhages is a characteristic feature. There are multiple fractures of ribs that show a beading effect. The fracture of clavicle is also common. The chipping of the corners of epiphysis at large joints of limbs is again a characteristic feature due to shaking and rotating strains imposed upon limbs by forceful application of adult hands. The metaphyseal fragmentation is also common. The fractures of shaft of long bones from direct blows either from a fist or from the child hitting a fixed object.
6. **Burns:** Punctate burns often accompanied by older scars may indicate deliberate stubbing of cigarette ends upon child's skin. Child may be seated upon a stove or electric radiator and

may be dipped in hot fluids.

Diagnosis of Child Battering

1. **Discrepancy between history and findings:** Variation and inconsistencies in explanation given by the parents; the story changes even after hours or days and different versions are given to different investigators. This is a characteristic finding. There is inconsistency of parent's explanation with the extent and chronology of injury. The bruises are multiple and multiple fractures in different stages of healing. The explanations of parents may change several times on repetition. In addition, there is tendency of the parents to take the child to different doctors or to a different accident center at each episode of injury.
2. **Delay in seeking advice:** There is delay in seeking medical advice or treatment. This is a highly significant feature that is seeking medical advice and injuries have a long interval.
3. **Classic lesions** such as torn upper lip frenulum, multiple bruises on the face, thorax or abdomen and around joints of the limbs, bite marks, unusual burns, fractures in different stages of healing and multiple bruises with different colours are typically present.
4. There are evidence of repeated visits to different doctors or different medical centers.
5. A general medical history as mentioned before exists.

SUDDEN INFANT DEATH SYNDROME

Sudden death is known by the names of **Cot death** in Britain and **Crib death** in North America and is the commonest cause of sudden death in infancy.

Definition

International conference in Seattle in 1990 defined sudden infant death syndrome as "The sudden death of any infant/young child which is unexpected by history and in whom a thorough necropsy fails to demonstrate an adequate cause of death. It is not recognized cause of death in

India due to much higher level of infant mortality, the increased rate of death from causes like infections and malnourishment overshadows Cot death.

Historical Aspects

SIDS has been known for at least 3'000 years in Jewish Old Testament. It also occurs in animals such as pigs.

Incidence

UK—2/1000 live birth, USA—2.3/1000 live birth. The incidence decreased in Britain and other European countries as there were campaigns for sleeping babies on their backs and not on their face; avoiding overheating the baby and avoiding smoking near the baby.

In India, no studies are available signifying incidence of SIDS.

Age incidence:

Usually—1 month-1 year

Rare—after 1 year

Official age is 2 weeks-2 years

Most cases—2-8 months

Peak—4months due to lowest level of infant's antibodies and decline of maternal antibodies.

Sex incidence: Male to female ratio is 1.3:1.

Incidence of twins: There is an increased risk that five times more than in single babies. Prematurity and low birth weight increases the risk. Sometimes both the twins die on the same day.

Social class incidence: There is a marked increase in lower socio economic class in Western countries.

Seasonal Variation

In temperate regions, it is more common in colder seasons. In northern hemisphere it occurs in October to April and in Australasia it occurs in July to August.

Time of Death

Most of the deaths occur before mid morning, are usually found dead after night' sleep or after their

morning feeds. Minority of them dies in afternoon or in the evening.

Typical History

The typical history given is that the child was either quite well or had some upper respiratory tract infection or slight diarrhea the previous day and was found dead in the morning. Almost all deaths occur in the morning in sleeping place. In 85% of the cases no post mortem findings are found whereas in 15% of the cases some chest infection, congenital anomalies and old birth injuries are demonstrable. Petechial haemorrhages in lungs, epicardium and thymus are usually seen and due to these findings the death was attributed to suffocation, called overlaying when the child was smothered by mother in the bed but even when the children slept in bed this continued to occur.

Various Theories of Causation of SIDS

The various theories thought to cause SIDS are: (i) Cow's milk protein allergy (ii) House mite allergy (iii) Deliberate suffocation (iv) Botulism (v) Deficiency of selenium, vitamins E, C, D, thiamine, calcium, magnesium and biotin (vi) Staphylococcus aureus causes anaphylactic shock due to its growth in unabsorbed milk (vii) Hypoglycemia (viii) Hypothyroidism (ix) Carbon dioxide and carbon monoxide poisoning (x) Overlaying (xi) Pharyngeal hypotonia (xii) Nasal obstruction (xiii) Respiratory syncytial virus (xiv) Prolonged sleep apnea (xv) Deficient or abnormal pulmonary surfactant (xvi) Cardiac conduction anomalies (xvii) Narrow foramen magnum (xviii) Hypothermia or hyperthermia (xix) Hypogammaglobulinemia

Prolonged Sleep Apnea

In 1970's and early 1980s this theory gained favour. Some infants with poor respiratory drive suffered from exaggeration of normal periods of apnea that all infants have during sleep. Progressive hypoxia and failure to respond to hypercapnia and hypoxia in sleeping infant results

in hypoxia—Apnea—hypoxia—bradycardia—cardiac arrest. Respiratory infection, sleep and nasal obstruction contributed to acute on chronic state hypoxia. For this apnea alarms were built but the theory was deflated after prospective studies.

To summarize: SIDS is a final common pathway leading to death in infants compromised by a number of deleterious factors summing to final fatal outcome. It is multifactorial and the factors such as sleep depress the brainstem, virus infections of respiratory tract cause viremia, reduce oxygenation and narrow effective airway lumen by mucus and exudates. The constitutional factors like botulism, Prematurity and low birth weight are contributory. Mode of death is respiratory failure due to brain stem dysfunction.

Role of pathologist in SIDS

1. A meticulous autopsy examination should be undertaken.
2. Natural diseases and any injuries should be excluded.
3. Parents to be given sympathetic advice.

Autopsy Findings

External findings: Scene of death is rarely available and at autopsy following are the external findings but are not significant:

1. Gross findings are nil.
2. Hands are clenched around the fibers from bed clothes as if there were spasmodic agonal grasping movements.
3. Some children are found under bedclothes huddled upside down at the foot of the cot but this has no significance as some infants habitually sleep this way.
4. Body is moist with sweat with raised body temperature and there is preventive advice in Britain to avoid infants from over wrapping and overheating.
5. Length, weight and physical measurements should be recorded care fully.
6. A few will have significant pathological lesions in about 15% cases that may from mild congenital heart lesion or some

congenital anomaly such as Down's syndrome.

7. Face is pale, slightly cyanosed and congested.
8. If the infant has slept face down pallid area is seen around the mouth and nose where pressure prevented post mortem staining from settling.
9. There is inconstant froth at lips and nostrils.
10. Slight oedema fluid that is blood tinged is exuding out of the mouth and nostrils.
11. Stomach contents may be present on mouth, nostrils and face.

Internal findings: Internal findings are non-specific in a typical case of SIDS and for this no diagnosed criteria have been confirmed.

1. Petechial haemorrhages on visceral pleura, thymus glands and epicardial surface of heart especially posteriorly occur in 70% of true SIDS cases and are almost agonal in origin. The theory for this that is not substantiated is that they are the result of forced inspiratory efforts against a closed airway due to laryngeal spasm or collapsed pharynx in hypotonic infants.
2. The thymic haemorrhages and large ecchymoses are found in SIDS as opposite to mechanical suffocation. These haemorrhages are in thymic cortex rather than the medulla but few pathologists accept this criterion.
3. Gastric contents: The presence of milk curd in air passage, is either an agonal regurgitation or a postmortem phenomenon.
4. Respiratory infections in the form of inflamed laryngo-tracheal mucosa. If the infection is severe, it produces pus with obvious inflammatory changes in lung parenchyma.
5. Pulmonary oedema is moderate and the lung surface shows patchy sub lobular partial collapse with areas of blue lung alternating with better-aerated pink zones.
6. Other abnormalities such as Down's syndrome, congenital heart disease and chronic systemic disease are contributory causes of death to SIDS.

Histological findings in SIDS:

1. Changes in the lungs: Inflammatory changes and peribronchiolar cell infiltration usually sparse or small collections of leucocytes. In control infants, lymphocytes are found.
2. Chronic hypoxia occurs due to thickened pulmonary artery walls, gliosis in brain stem, retention of brown fats in adrenals and abnormalities in the carotid body.

MUNCHAUSEN SYNDROME BY PROXY

It is a peculiar and dangerous type of child abuse usually involving the mother, in which children are brought to doctors for induced signs and symptoms of illnesses with a fictitious history. The sex ratio is almost equal. It has been described in children of few weeks of age to 21 years. The child is admitted frequently in the hospital for medical evaluation for the non-existent conditions. These patients appear to be compulsively driven to make their complaints. The person is aware that he is acting an illness, but he cannot stop the act. There is continuity, ranging from exaggerated claims of infirmity to actual self induced illness. At the extreme end, life-threatening injuries are masqueraded as being legitimately contracted. Rosenberg (1989) gave four diagnostic criteria (i) Illness produced or alleged, or both by a parent. (ii) Repeated requests for medical care of a child, leading to multiple medical procedures (iii) Parenteral denial of knowledge of the cause of symptoms (iv) Regression of symptoms when the child is separated from the parents.

Methods of Production

(i) The mother pricks her finger and adds blood to the urine of the child and takes the sample to the doctor (ii) The child's nose is closed with two fingers and the lower jaw pushed up with the palm to block the airway. (iii) A pillow or towel is put over the face of the child and the face is pushed down into bed clothing (iv) The mother gives insulin to the child and takes to hospital with hypoglycaemia (v) Vomiting: allegation or by ipecacuanha (vi) Diarrhoea: laxatives, salt poisoning (vii) Convulsions: allegation or by theophylline, insulin, psychotropic drugs (viii) Bleeding: anticoagulants, phenolphthalein poisoning, exogenous blood (ix) CNS depression: barbiturates, benzodiazepines (x) Alleged fever (xi) Scratching or intoxication rash.

Long Question

1. Define infanticide. What are the signs of live birth?
2. Describe the causes of death in case of infanticide.

Short Questions

1. Munchausen syndrome by proxy
2. Battered baby syndrome
3. SIDS
4. Hydrostatic test
5. Differentiate between respired and unrespired lung
6. Differentiate between head injury due to blunt force and precipitate labour

Impotence and Sterility

IMPOTENCE

Impotence is inability to have sexual intercourse.

STERILITY

Sterility is the inability of the male to beget children and in the female to conceive children. A person can be sterile without being impotent and can be impotent without being sterile. Impotence is a ground for voidable marriages under S 12 Hindu marriage act. The question arises in both civil and criminal cases.

Civil Cases

- Adoption.
- Nullity of marriage.
- Divorce.
- Adultery.
- Disputed paternity and legitimacy.
- Claims for compensation when loss of sexual functions is a result of accident.

Criminal Cases

- Adultery.
- Rape.
- Unnatural offence. In these situations the accused pleads impotence as a defence.
- When an injured individual thinks that he has become impotent as a result of wounds, injuries when inflicted by others.

Causes in the Male

1. Age

Power of coitus starts earlier. Then after 1-2 years power of procreation starts i.e. at 14-15 years. A male is sexually potent at the age of puberty. As age advances, potency and power of procreation decreases. There is no definite age but it occurs at the extremes of ages.

2. Malformations and developmental anomalies
 - i. Absence or non-development of penis.
 - ii. Adhesion of penis to the scrotum.
 - iii. Hypospadias associated with marked deformity and the person is also sterile as sperms cannot be deposited.
 - iv. Epispadias is rare; associated with rudimentary and stunted penis.
 - v. Loss of both testicles or absence of them.
 - vi. Cryptorchids are not necessarily sterile or impotent, but sterility is common due to azoospermia.
3. Local diseases:
 - i. Diseases causing temporary impotence that is cured by surgery
 - Large hydrocoele
 - Scrotal hernia
 - Elephantiasis
 - Phimosis
 - Para-phimosis
 - ii. Diseases of penis or scrotum causing impotence or sterility
 - Syphilis
 - Cancer
 - Tuberculosis
 - iii. Gonococcal infection of the testis, epididymis, prostate etc.

- iv. Atrophy of testicles—Mumps
 - v. Lithotomy operation may result in the injury to ejaculatory duct and sterility.
4. General disease
- i. Endocrine disturbances like diabetes, pulmonary tuberculosis and chronic nephritis
 - ii. Injury to spinal cord resulting in paraplegia.
 - iii. Tumour or injury to cauda equina.
 - iv. General paralysis of insane
 - v. Blow on the head affecting brain or spinal cord.
 - vi. Exposure to X-ray radiation leading to temporary azoospermia.
5. Excessive and continued use of drugs; like Opium, Alcohol, Cannabis, Tobacco, Cocaine
6. Psychical influence: Fear, anxiety, excessive passion, and sense of guilt all lead to impotence

Quoad

Quoad is also called selective impotence as the male is impotent with a particular woman. The phenomenon is known as *impotence quoad hoc*.

Examination of the Male Person

The examination is carried out when asked by the Court or in causality in rape cases. When the accused is brought by police for examination and for opinion whether the person examined is not capable of having sexual intercourse.

Procedure that is to be followed in such cases

1. Written order of the Court/investigating officer of the case.
2. Informed consent of the accused, duly attested by the witness with time date and address.
3. Identification of the male to be done by a close relative and at least two identifying marks to be mentioned. A recent photograph should also be attached.

Medical Examination Proper

History of previous illness like mental illness or nervous system disorders and sexual history

should be recorded.

1. General examination should include complete examination of the nervous system.
2. Local examination include examination of
 - i. Condition of testes, epididymis, spinal cord, penis etc. They are to be tested for the presence of sensations.
 - ii. Prostate and seminal vesicle to be palpated per rectum. Pressure to be applied over seminal vesicle, giving pressure by finger along the urethra thus the semen is brought to meatus and examined for spermatozoa.
 - iii. Some tests can be done to produce erection of the penis which include injection of papaverine in the penis, showing pornographic videos and taking elaborate history for nocturnal tumescence.

If there is no disease or abnormality, there is no reason to believe that the man is not capable of performing the sexual act. Existence of potency is a matter of inference to be drawn from negative findings.

Casper states: that the possession of virility and the power of procreation neither requires to be, nor can be proved to exist by any physician but is rather like any other function to be supposed to exist within the usual limits of age.

Opinion

If there is no detectable finding or disease in a person, the opinion is given in a negative form. that is there is no finding to suggest that the person examined is not capable of perform the sexual act. However if there is any gross abnormality in the penis or scrotum, then the doctor can opine that person examined is not capable of doing the sexual act because of the disease.

Causes of Impotence and Sterility in Females

- 1 Age: Puberty starts at 13-14 years of age. **Catemenia** is the first menstrual flow. It lasts till menopause that is upto the age of 45-50 years. But sexual instinct lasts till old age.

There are cases in which 61-62 years old gave birth to a full term child and 63 years old gave birth to her 22nd child.

2. *Congenital malformations such as :*

- i. Total occlusion of vagina
- ii. Adhesion of labia
- iii. Imperforate hymen
- iv. Congenital absence of vagina
- v. Absence of uterus, ovaries and fallopian tubes
- vi. Turner's syndrome: has XO chromosomes; anatomical structure is female and nuclear sexing is male

3. *General diseases:* As women is the passive agent general diseases cannot cause impotence. The general disease and debility do not cause a barrier to sexual act or conception. A paraplegic can also become pregnant

4. *Local diseases:* As a passive agent local diseases usually do not produce impotence but can produce sterility provided per vaginum examination is normal.

Vaginismus is a definite cramp like spasm of the adductor muscle and hysterical hyperasthenia co-exists. The hyperasthenia is present all over the vulva, abdomen and thigh etc. Contraction is so severe that penile penetration is not possible. It occurs equally in virgin and in those who have children. The causes of vaginismus are as follows.

- i. Sexual frustration secondary to husbands impotence.
- ii. Psychosexual inhibiting influence due to social, religious and orthodox practices.
- iii. Prior sexual trauma as a virgin at low age

- iv. Dyspareunia—painful intercourse
- v. Personal repulsion to the idea of intercourse
Psychotherapy is essential to overcome this disorder.

STERILITY

Sterility is produced as a result of following conditions:

1. Gonorrhoeal infection of cervix, ovaries and fallopian tubes.
2. Removal of both ovaries due to disease
3. Diseases of uterus
4. Recto-vaginal fistula, leucorrhoea, menstrual disorders.
5. Psychological: Fear, passion and neurotic temperament leads to hysterical fit during an attempt of coitus and leads to temporary impotence

Opinion: The female examined is capable of taking part in sexual act (Aptavira) or she is not capable of taking part in sexual act (Nonaptavira).

Long Questions

1. Define impotence: What are the causes of impotence and sterility in male. How you proceed to examine such cases and give your opinion?
2. Define sterility: What are the causes of impotence and sterility in female. How you examine such cases and opine.

Short Questions

1. Vaginismus
2. Quoad.

MULTIPLE CHOICE QUESTIONS

1. **Impotence may be pleaded as a ground for defense in males in all except:**
 - A. Divorce
 - B. Rape
 - C. Adultery
 - D. Unnatural sexual offence
2. **'Impotency' is defined as:**
 - A. Inability to conceive
 - B. Inability to perform sexual act
 - C. Premature ejaculation
 - D. Inability to achieve an orgasm
3. **The Quoad is impotent to:**
 - A. All the ladies
 - B. To a particular woman
 - C. To wife
 - D. To elderly ladies

Virginity, Pregnancy and Delivery

VIRGINITY

Virgin (*Virgo intacta*) is a female who has not experienced sexual intercourse whereas loss of virginity is known as **defloration**.

Marriage is a contract between man and woman which implies the sexual union.

The question of virginity arises in cases of nullity of marriage, divorce, rape, defamation etc.

1. *Nullity of marriage* (S.12 Hindu Marriage Act): Marriage may be annulled that iss never existed in the following conditions.
 - i. Either party being under age at the time of marriage
 - ii. Either party already validly married.
 - iii. One party is of unsound mind.
 - iv. Marriage has not been consummated due to impotence or willful refusal.
 - v. When the woman was pregnant at the time of marriage.
2. *Divorce* (S.13 Hindu Marriage Act) The following are the grounds for divorce. Dissolution of previously valid marriage will be granted in the following circumstances:
 - i. Adultery (S.497 I.P.C.).
 - ii. Husband is found guilty of rape, sodomy or bestiality.
 - iii. Incurable insanity, leprosy, venereal disease.
 - iv. Cruelty
 - v. Deserted the petitioner for two years continuously.

- vi. Ceased to be Hindu*
 - vii. Renounced the world by entering into religious worship*
 - viii. Not heard for 7 years.*
 - ix. *Section 13B of Hindu Marriage Act:* Divorce by mutual consent on the ground that they are living separately for the period of 1 years or more.
3. Defamation
 4. Rape.

The doctor is called upon to give the opinion after examining the female whether she is virgin or not.



Figure 26.1: Anatomy of female external genitalia

* Only divorce can be given

Signs of Virginit

1. *Extragenital*

Breasts: In a young adult breasts are hemispherical, firm and rounded. Nipples are small and areola is pink or darker. Occasional act of coitus will not alter the character of breasts, hence little significance is given to findings of breasts as evidence of virginit. Breasts can be pendulous and large as a result of handling even though the woman had never been pregnant and still she is a virgin.

2. *Genital*

- **Labia majora:** Labia majora are two elongated folds of skin projecting downwards and backwards from Mons veneris. The point at which they meet centrally is known as anterior commissure and meet posteriorly in front of anus is known as posterior commissure. In virgin they are thick, firm, elastic and rounded; lie in apposition to close the vaginal orifice. This is not seen in woman habituated to sexual intercourse.
- **Labia minora:** These are soft, small, pink and sensitive. The labia minora meet anteriorly to form the clitoral hood. The clitoris is small. The triangular area between clitoris as apex and anterior margin of hymen as base is known as vestibule, which is very narrow. The lower part of labia minora fuses posteriorly in midline and forms a fold called posterior fourchette. Fossa navicularis is the concave area of vestibule bound anteriorly by vaginal opening, posteriorly by posterior fourchette and laterally by labia minora. The depression between fourchette and vaginal orifice remains closed in a virgin. The edges of labia minora project between labia majora.
- **Vagina:** Vaginal passage is a pocket, irregular in shape, about 7.5 cm long; 6 cm anterior wall, 9 cm posterior wall. It is collapsed to form a slit like opening crosswise of the body. It is pear (balloon) shaped when distended. Width of the upper end is 3-4 cm in nulliparous; 6-7 cm in multiparous. Vagina is narrow and tight, full of rugosity, reddish and sensitive to touch and its walls are

approximated. Rugosity is removed only after childbirth but in some cases it is absent in virgin with intact hymen. A single intercourse does not alter the anatomy of genitalia area apart from the rupture of hymen.

- **Hymen:** A fold of mucous membrane about 1 mm thick, situated at the vaginal outlet partially closing the vaginal orifice. The average adult hymen consists of folds of membrane having annular or crescentric shape. In children, it appears as a taut membrane when thighs are separated. During puberty, hymen enlarges and appears as a series of folds. Normal hymen may be rigid and fibrous. It is usually elastic and easily distensible. It is supplied by blood vessels that bleed during rupture. In virgins it admits tip of little finger.

Hymen is practically always present in a virgin though it may be congenitally absent according to some authors. However, latter view is erroneous.

Hymen may remain intact but the woman may not necessarily be a virgin. The hymen may remain intact even after repeated acts of coitus depending upon: (i) force of penetration (ii) structure (iii) consistency (iv) nature of opening.

In children, the hymen is usually not ruptured due to high up position during attempted sexual intercourse.

The term **Virgo intacta** is applied to such woman when the hymen is intact despite the fact that she had experienced sexual intercourse.

When the hymen is intact, the woman may not necessarily be virgin. However, if hymen is ruptured she may be still be virgin because the rupture had resulted due to causes other than the sexual act.

Types of Hymen (Fig. 26.2)

1. **Semilunar or crescentric:** The hymenal opening is placed anteriorly present at the lateral and posterior margins of vaginal opening.

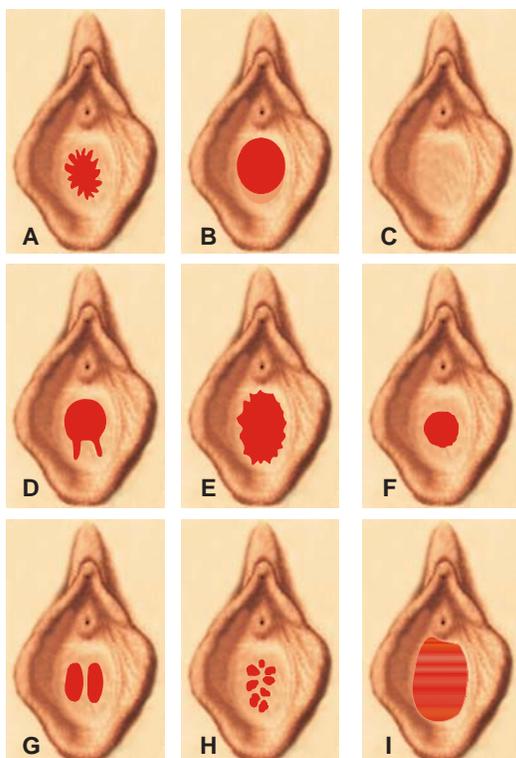


Figure 26.2: Different types of hymen (A) Fimbriated hymen (B) Semi lunar hymen (C) Imperforate hymen (D) Hymenal tears (E) Carunculæ myrtiformes (F) Annular hymen (G) Septate hymen (H) Cribriform hymen (I) Parous introitus

2. *Annular:* Hymenal opening is oval situated in the center and encircles the vaginal opening.
3. *Infantile:* There is small linear opening in the midline.
4. *Cribriform:* There are several hymenal openings.
5. *Vertical:* A vertical hymenal opening is seen.
6. *Septate:* There are two lateral openings side by side.
7. *Imperforate:* No hymenal opening is there.
8. *Fimbriated or notched:* Free margin of hymen sometimes presents natural notches. This is to be differentiated from tears as a result of sexual intercourse. This type of hymen is also called **frilly edged**. The differences between

fimbriated and ruptured hymen are as given below:

Features	Fimbriated hymen	Ruptured hymen
Situation of notches	Present anteriorly; doesn't extend to the base of hymen	Present posteriorly at one or both sides or median line; extend to the point of attachment of hymen to the edge of vagina.
Shape and size of notches	Bilaterally symmetrical	Asymmetrical
Mucous membrane over notches	Intact	Not intact
Cause	Occurs naturally; making the edges of hymen notchy	Caused by sexual intercourse or by introduction of foreign bodies.

Hymen in Relation to Sexual Intercourse

In majority of cases hymen is ruptured during the first act of coitus but certain types of hymen will not rupture even by repeated acts. These are:

- i. Annular hymen (fringe like).
- ii. Loose, folded and elastic type.
- iii. Thick, tough and fleshy type.

As a result of sexual intercourse, tears are present posteriorly usually at 5'O and 7'O clock position. At times these tears may be present in midline and usually more than two tears are not found.

Recent tears look raw, red, swollen and painful to touch. They (i) Bleed on touching; within 1-2 days (ii) Heal in 5-6 days (iii) Shrunken and look like granular tags of tissues within 8-10 days.

Carunculæ hymenalis or myritiformis: It is a condition which is found in those who have habitual sexual intercourse or given childbirth, the hymen is destroyed and small, round, red fleshy tags are found around the hymenal ring (Fig. 26.2).

Causes of Rupture of Hymen

1. *Accidental:* Accidental fall on projecting substance or slipping on furniture or fence. In

these cases, injuries to other parts of body will be there. Forcible separation of thighs in children will not rupture the hymen unless perineum is ruptured. Riding, dancing, jumping will not rupture the hymen as is commonly believed.

2. *Masturbation*: In cases where long objects are used for masturbation, clitoris is enlarged and vaginal orifice is dilated.
3. *Foreign body*: Introduction of *Sola pith* into the vagina, which soaks water and swells. This is done specially in prostitutes when the young girls are asked to sit in water with the *solapith* introduced in to the vagina. The vagina swells and rupture of hymen occurs and these girls are fit for taking part in sexual intercourse.
4. *Ulceration*: This could result from Diphtheria, fungal infection.
5. *Scratching*: Due to uncleanness' and poor personal hygiene of the part.
6. Surgical and gynecological operations.
7. *Sanitary tampons*: A plug of cotton wool that is used to absorb secretions and stop haemorrhage can result in rupture of hymen.

Diagnosis of Virginit

Intact hymen indicates virginit but sometimes married woman and even prostitutes have intact hymen. Thus an intact hymen does not automatically prove virginit. With an intact hymen there may be true virgin or false virgin. Hence due considerations should be given to accessory signs of virginit before giving the diagnosis of virginit.

If hymen is intact, labia majora is flabby and not in apposition and in addition, labia minora are not in apposition, vagina is roomy and admits two fingers then it is highly probable that the female is not a true virgin.

A virgin was once defined by judges as a *raraavis* and so far as the medical evidence is concerned, the definition is almost correct.

Signs of breasts and genitalia, particularly intactness of hymen was always held to signify the physical virginit. However, hymen has limited value in defining virginit as sometimes repeated acts of coitus may not rupture the hymen.

Intactness of hymen is not an absolute proof of virginit. With intact hymen there are true as well as false virgins.

Findings in True Virgins

1. Hymen is intact and is well stretched.
2. Edges are distinct and regular.
3. Vaginal orifice is of small dimension and allows admission of terminal phalanx of a finger.

When all these findings are present, presumptions are in favour of the virginit or non-penetration of penis in to the vagina.

Findings in False Virgins

1. Hymen is intact.
2. Hymenal orifice lets one, two or more fingers to pass easily.
3. Hymen is relaxed as to undulate and allow itself to be depressed in a virgin. However, the size of penis in erection can also pass without rupturing the hymen one or several times.

In such cases, accessory signs of virginit are to be reviewed and carefully weighed. These signs are: (i) labia majora are separated and flabby (ii) labia minora are thickened and separated (iii) fourchette is torn (iv) vagina is roomy and enlarged. Thus, with intact hymen there is little possibility of the female being a true virgin.

If the above accessory signs are not present, there is greater possibility of female being a true virgin. It is not possible to comment on the findings of repeated sexual intercourse.

Thus, it is quite clear that in certain cases diagnosis of virginit is a delicate matter and there are cases when one cannot say for absolute in favour for or against it. However, unfortunately defloration without rupture of hymen is not the rule and at first coitus, hymen is torn in majority of cases.

To conclude there are no definite findings suggestive of a true or a false virgin and one cannot be certain of either nor can one express such certainty.

Anyways, the diagnosis of virginit is a very delicate matter and undoubtedly difficult in some

of the cases.

Virginity in Relation to Rape

On medical examination, there may be all signs of virginity but rape has been committed. By analyzing section 375 I.P.C., It is said that even slight vulval penetration is sufficient to constitute rape.

1. The question arises when the victim is a virgin prior to rape. Laceration or tear of hymen is the principal evidence of defloration or loss of virginity. Other signs are not affected by a single act of coitus. Even at times repeated acts do not rupture; so rupture of hymen not to be considered as principle evidence of loss of virginity due to rape.
2. Age of rupture should be correlated with time of occurrence.
3. Other cases of rupture are to be taken in to account.

Hymenoplasty

It is an operation for the repair of ruptured hymen and is more popular in western countries when the unmarried girl gives gift to her husband. However, Indian women are also not lagging behind. The unmarried girls undergo hymenoplasty before marriage, so that their husbands will not suspect anything. Even cases have come to notice that a lady on her 25th wedding anniversary underwent hymenoplasty to give a gift to her husband. The findings are narrow hymenal orifice that is unusual for the age group. There is congestion and scars at the site of united tears.

Differences between Virginity and Defloration

The differences between virginity and defloration are:

Features	Virginity	Defloration
Hymen	Intact, rigid, inelastic, edges are regular with a narrow opening tip of little finger	Torn or intact; loose elastic; two fingers pass easily.

Labia majora	Fully developed, appose to each other; close the vaginal orifice completely.	Not apposed, loose; vaginal opening may be seen at lower end.
Labia minora	In contact and covered by labia majora	Exposed, separated from labia majora.
Fossa navicularis	Intact	Disappears
Vagina	Narrow, rugae more folded; vault more conical	Grows in length; rugae less obvious, dilated.

PREGNANCY

Pregnancy is a state that occurs in a female when an ovum is fertilized by spermatozoa. It occurs during the reproductive period of the female, the average being from 15-45 years. The question of pregnancy arises in the following cases.

Civil Cases

1. To avoid attendance in a Court of law as a witness. However, pregnancy is not a bar unless advanced state is reached as at that time delivery may be imminent and there may be risk to child.
2. Feigned pregnancy soon after death of her husband to claim succession to the estate.
3. To assess damage in a seduction or breach of promise of marriage.
4. When the woman blackmails a man and accuses him that she is pregnant by him.
5. In cases of libel or slander of an unmarried woman, widow or wife having depart of from her husband and she is pregnant.
6. To secure greater compensation when her husband dies through negligence of the other.
7. A surgeon may terminate pregnancy by carrying out surgery for fictitious tumour.
8. To get more relief in cases of divorce.

Criminal Cases

1. To reduce the capital punishment, when convicted of capital crime (S. 416 Cr.P.C.). The High

Court can postpone or commute it to life imprisonment.

2. Motive for suicide or murder of unmarried girls or widows.
3. In cases of alleged criminal abortion.
4. Alleged concealment of birth (S. 318 I.P.C.), pregnancy, infanticide.

To examine such cases a written order either from the police officer or court is obtained and informed written consent is taken. The lady must be examined by a female registered medical practitioner (S. 53 Cr.P.C.) or by a male doctor in the presence of a female attendant. In case the registered medical practitioner is encountering difficulty, the help of the specialist is taken.

Diagnosis of Pregnancy in the Living

1. *Presumptive signs:* (i) Amenorrhoea (ii) Morning sickness (iii) Breast changes (iv) Quickening (v) Pigmentation of the skin (vi) Changes in vagina (vii) Urinary disturbance (viii) Fatigue (ix) Sympathetic disturbances.
2. *Probable signs:* (i) Enlargement of abdomen (ii) Uterine changes (iii) Cervix changes (iv) Intermittent uterine contractions (Braxton Hick's sign) (v) Ballotment (vi) Foetal parts (vii) Uterine soufflé (viii) Biological tests.
3. *Positive signs:* (i) Foetal movements (ii) Foetal heart sounds (iii) X-ray Diagnosis (iv) Ultrasonography.

Presumptive Signs

1. *Amenorrhoea:* It is the earliest and the most important symptom of pregnancy. There may be occasional spotting but full menstruation is not possible. Besides pregnancy, amenorrhoea may occur due to anaemia, TB, disorders of thyroid and pituitary gland, mental stress, fear or nervousness of illicit relationship and when there is intense desire of pregnancy. Amenorrhoea may sometimes be feigned as a strong motive for deceit.
2. *Morning sickness:* It appears from the end of first month upto three months. Vomiting in the morning is not a reliable sign and may be due

to migraine or some hormonal imbalance.

3. *Breast changes:* These changes important in primigravida but are of less value in multipara. The breast have increased growth and become physiologically more active. These changes are also found in ovarian or uterine tumours and in spurious pregnancy. The various breast changes are:

- Tenderness
- Tingling sensation
- Size is increased due to hypertrophy of mammary gland
- Superficial veins are engorged
- Nipple becomes deeply pigmented and erectile
- Areola becomes deep darkly brown in 2nd month
- Sebaceous glands are enlarged and tubercles are formed around the nipple.
- Colostrum formation occurs during 3rd month, is thin yellowish fluid consisting of fat globules and large phagocytic cells filled with droplets of fat.
- After 6 months, silvery lines and striae are seen due to stretching of skin
- Rarely no appreciable breast changes occur and even the size may diminish.

4. *Quickening:* Quickening literally means "Coming to life". It is first perception of fetal movements by the mother that occurs during the **14-20th weeks**. In a non-pregnant woman flatulence and peristaltic movement can be mistaken for quickening. In spurious pregnancy commonly seen in hysterical woman and those who are anxious to have children, false quickening can be felt.

5. *Pigmentation of skin:* The skin of vulva, abdomen and axilla become darker. There is formation of dark circles around eyes. The formation of linea nigra starts from pubes, divides to encircle the umbilicus and continues upto Ensiform cartilage. These are dark, pigmented lines.

6. *Changes in vagina:* The mucous membrane of vagina changes from pink to violet and finally

blue as a result of venous obstruction. It is seen **after 4th week** and is known as **Jackquemier's sign** or **chadwick's sign**. The tissue becomes softer, secretions are increased and the pulsations of vaginal artery can be felt at an early period.

7. **Urinary disturbance:** The enlarging uterus presses upon bladder and results in increased frequency of micturition. This symptom gradually passes off as the uterus comes in to the abdomen and again reappears before term when head descends in to pelvis.
8. **Fatigue:** It is very frequently present
9. **Sympathetic disturbances:** These are salivation, perverted appetite and irritability.

Probable Signs

- i. **Enlargement of abdomen:** It occurs due to enlargement of the uterus as:

Months	Enlargement of uterus
End of 3rd	Fills the pelvis
3-4th	Over the pelvic brim
5th	Midway between symphysis pubis and umbilicus
6th	At the level of the umbilicus
7th	Midway between umbilicus and ensiform cartilage
8th	At the level of ensiform cartilage
9th-10th	The uterus sinks in to pelvis

Due to stretching of the abdominal wall, red coloured subcutaneous scars are seen on the abdomen

- ii. **Changes in the uterus:** These are:
 - At 3rd month, angle between body and cervix is accentuated. On bimanual examination, uterus has a doughy or elastic feeling.
 - **Hegars sign** is appreciable at **8-10 weeks** and can be elicited by putting one hand over the abdomen and two fingers inside the vagina. The cervix is felt as firm and hard whereas uterus is felt as elastic body. In between the two, the isthmus is felt as a soft compressible area.

- iii. **Changes in the cervix:** The cervix softens from below upwards from the 2nd month onwards. This is well marked in the **4th month** and is known as **Goodell's sign**. There is softening of the cervix towards the last month of pregnancy. The cervical orifice becomes circular instead of transverse, admitting the tip of finger more readily.
- iv. **Intermittent uterine contractions (Braxton—Hick's sign):** These are intermittent, painless uterine contractions, are usually felt **after 3rd or 4th month**. These contractions last for one minute with relaxation for 2-3 minutes. The sign is elicitable even in cases of dead foetus, disease like myomas, haemometra etc.
- v. **Ballottement:** Ballottement means 'to toss up like a ball'. It is positive during **4th and 5th month** because the foetus is small in relation to liquor amnii. It is of two types:
 - **Internal ballotment:** It can be elicited by inserting two fingers in to the anterior fornix and the uterus is tapped upwards suddenly. The foetus moves upwards and after a while drops back on the finger tip like a ball.
 - **External ballotment:** It can be obtained by sudden motion of the abdominal wall, in a few seconds the foetus rebounds back. These findings can be negative if there is less amniotic fluid.
- vi. **Foetal parts:** These can be felt by the **5th month** by palpating through the abdominal wall.
- vii. **Uterine soufflé:** It is a soft blowing murmur, heard by auscultation, which is synchronous with the mother's pulse. It is heard above the pubis at the end of 4th month due to passage of blood through the uterine vessels. The uterine soufflé is also heard in enlarged ovaries and myomatous tumour of uterus.
- viii. **Biological tests:** These tests depend upon the presence of chorionic gonadotropins in the urine of a pregnant woman. The early morning sample of urine is taken for the test.

Table 26.1: Biological tests

Tests	Animals	Procedure	Time	Observations
Rapid Rat test	Two immature albino female rats	2 ml of morning sample of urine or 1 ml of serum is injected intraperitoneally	4-24 hrs later killed with chloroform asphyxia.	Congested ovaries positive
Aschheim-Zondek test	Five immature white female mice; wt. 6-8 gm, 3-4 week old	0.2-0.3 ml of morning urine sample is injected subcutaneously BD for 3 days.	Killed on 5th day	Haemorrhagic follicles or corpus luteum positive of ovary
Friedman's test	Adult female Rabbit; 12-12 week old, wt. 2kg.	5-10 ml of urine through ear vein for 2 days.	24 hrs after last injection	Haemorrhagic follicle or graffian follicle
Hogben (Female Toad) test	Mature female African Toads	5 ml of morning urine in dorsal lymphatic sac.	Animal is isolated in a glass jar 1/3rd of water	12-18 hrs ovulation Takes place
Galli Mainini (Male frog) test	Rana tigrina	5-10 ml of morning urine in dorsal lymphatic sac of two male Toads	Cloacal urine is withdrawn ½-1hr for 3hrs	Presence of sperms in the urine in 2-3 hrs and latest within 24 hrs, if pregnancy is present

The tests depends on the hormones derived from the placenta. The diagnosis is made as early as **10-12 days** whereas other external signs of pregnancy prove not to be reliable. The test is false positive in hydatidiform mole, chorion epithelioma, ectopic gestation, pituitary tumours. **Galli Mainini test** has certain advantages as it is rapid there is easy availability of animals, has low cost and the accuracy is 94-96% (Table 26.1).

ix. *Immunological tests of pregnancy:*

Agglutination test: Human Chorionic Gonadotropins has antigenic properties and when it is injected in to animals, Anti Human Chorionic Gonadotrophic serum (Anti HCG) is produced. The urine (suspected case of pregnancy-HCG) is mixed with anti HCG and incubated for one hour. If the patient is pregnant, HCG in urine will neutralize Anti-HCG in serum. Then RBC or latex particles coated with HCG are added and incubated for 2 hours. The mixture is then centrifuged and if the mixture remains turbid without deposit (No agglutination), it is taken as positive for pregnancy, because Anti HCG has

neutralized HCG in the urine of pregnant woman. Absence of agglutination indicates neutralization of the antiserum by gonadotropins in the urine.

x. *Pharmacological tests for pregnancy:* The sudden withdrawal of exogenously administered progesterone may precipitate endometrial bleeding in an amenorrheic patient. Thus, if no withdrawal bleeding occurs, pregnancy may be the likely explanation.

xi. *Radio-immuno assay for chorionic gonadotropins:* This assay has been recently developed. The test detects minute amounts of HCG (0.003 international units per ml of serum) with accuracy concentration in 1 liter of plasma (mother)= 24 hrs of urine concentration.

HCG level starts rising after 2 weeks of missed period and the level is maintained upto 5th month. It gives 98% accuracy in the diagnosis of pregnancy. The test is false positive, due to luteinizing hormone (L.H.) of pituitary cross-reacting with antibody of HCG. It is false negative from more than 20

weeks to less than 10 days.

Positive Signs of Pregnancy

- i. **Foetal movements:** Fetal movements are felt at **4 months** and the parts can be felt at **5 months** by placing the hand on the abdomen.
- ii. **Foetal Heart sounds:** This is an important and definite sign of pregnancy. The foetus heart sounds become audible at **18-20 weeks** like tickling of a watch placed under a pillow. The heart rate is approximately 160/min at 5th month and 190/min at 9th month. The fetal heart sounds are not synchronous with mother's pulse and is not heard in a dead foetus or when there is excessive liquor amnii, fatty abdomen, or foetus < 18 weeks. **Funic soufflé** is a hissing sound synchronous with fetal heart rate due to movement of blood in the umbilical vessels.
- iii. **X-ray diagnosis:** On radiological examination foetal parts can be detected from 16 weeks onwards and occasionally can be detected as early as **10-12 weeks**. On X-ray the foetus is seen as:
 - a. Annular shadow for skull.
 - b. Small dots with linear arrangement for vertebral column.
 - c. Series of parallel lines for ribs.
 - d. Linear shadow of limbs.

At a later stage, X-ray is of value in the diagnosis of twin pregnancy, foetal abnormalities and death and hydatidiform mole.
- iv. **Ultrasonography (B-scan ultrasonography):** It is direct visualization of ultrasonic apparatus. Gestational sacs with uterus can be visualized at **5-6 weeks** of conception. At 5-6 weeks of gestation the sac is large enough to be visualised by ultrasonography. Heart is the first functional organ and cardiac activity can be seen at 5-6 weeks of gestation.
- v. **Foetal electrocardiogram:** It is more accurate at **after 17 week** of intrauterine life.

Determination of Period of Pregnancy

1. By counting the days since last menstrual period.
2. From quickening that occurs at 14-18 th week.
3. Height of uterus above symphysis pubis.
4. **Macdonald's Rule:**

$$\text{The no of lunar mths of pregnancy} = \frac{\text{Ht of fundus above the symphysis (cm)}}{3.5}$$

For example: If the height of uterus is 30 cm or 12 inches, then period of pregnancy = 30/3.5 = 8½ lunar months = 34 weeks.

Pseudocyesis (spurious pregnancy): It is usually seen in women nearing menopause, in young woman who intensely desire a child or those with psychic or hormonal disorder. Such patients may present with all the subjective symptoms of pregnancy like:

- i. Abdominal size is increased due to ascitis, fat etc.
- ii. Breast changes are sometimes present.
- iii. In many cases, women imagine fetal movements.
- iv. In some cases, pregnancy has gone to full term and even labour pains started when the patient was told that she is not pregnant. In these situations, the patient must be convinced about her condition and X-ray abdomen should be taken that will allay her doubts.

Diagnosis of Pregnancy in the Dead

An examination may help to retrieve or restore the reputation of a dead woman suspected of unchastity. The findings of pregnancy in the dead woman are:

1. **Presence of an ovum:** Presence of ovum for 7-10 days, presence of an embryo for 1-9 weeks or presence of foetus from 9 week onwards or presence of placental tissue and membranes or any other product of conception signifies that the woman is pregnant. A well formed corpus

luteum in one of the ovary is very important feature. Even in an exhumed body foetal bones may be found.

2. *Corpus Luteum*: Corpus luteum continues to develop during pregnancy and maximum size is reached at 5th month forming a firm projection on the surface of the ovary. It then passes through a retrogressive change. However, it is well marked at the time of delivery and may be evident up to 1-2 months thereafter.
3. *Uterine changes*: Marks of placental attachment are found up to 8-9 weeks after delivery. Period of gestation is the possible period of pregnancy; not the average period.
4. In doubtful cases, microscopic examination will reveal products of conception, condition of endometrium and corpus luteum.
5. *False positive pregnancy* can be due to fibroid tumour, other pathologies of uterus and ovaries. Pregnancy can also occur without the formation of corpus luteum.

Average Period of Pregnancy

The average period of pregnancy is 280 days from the 1st day of last menstrual period; so that actual period is 270 days or less. It is 40 weeks or 10 times the normal intermenstrual period i.e., 28 days.

Maximum period: Maximum period of pregnancy is 354 days that is the period required from coitus upto live birth of the foetus though a period of 349 days is accepted in English courts. The longer the gestation period the larger will be the infant.

Minimum period and viability: The minimum period of pregnancy is 180 days when the foetus may be viable and acceptable for medicolegal purpose. At 210 days also, the foetus is viable.

DELIVERY

The term delivery means expulsion or extraction of the child at birth. It means spontaneous delivery. The question of delivery may arise in situation arises such as:

1. Abortion before foetus is viable
2. Infanticide
3. Concealment of birth (S. 318 I.P.C.)
4. Feigned delivery
5. Contested legitimacy (S. 112 I.E.A.)
6. Black Mailing
7. Defamation of character
8. Nullity of marriage
9. Divorce.

Signs of Recent Delivery in the Living

Recent delivery means 10-14 days after the expected date of delivery and is applicable to full term deliveries, rather than premature ones.

In strong and vigorous woman especially multiparous, the signs of recent delivery disappear within a week or so and sometimes earlier. The signs of recent delivery are:

1. *General appearance or indisposition*:
 - The woman has languished look with shrunken eyes for the initial 2-3 days.
 - There is dark coloured pigmentation over the lower eyelids
 - The pulse and body temperature are slightly increased.

These signs may be found in some other illness also and not specifically produced after delivery. These may not be produced in multiparous woman who resume work immediately.

2. *Intermittent contractions of uterus*: Intermittent contractions of uterus known as **after pains** are vigorous and painful and remain up to 4-5 days.
3. *Findings in the breasts*: Breasts are full and prominent and have a knotty feeling are tender. Nipples are enlarged and surrounded by darkened areola. **Montgomery's tubercles** are present. Milk or colostrum corpuscles in milk are present within few days. Veins over breasts are prominent.
4. *Abdominal findings*: Abdomen is lax, flabby with wrinkled skin. Stria gravidarum and linea alba are suggestive of past delivery in

the absence of other possible causes. These are pink when recent and subsequently becomes white. These may not be present in previous prolonged distension of abdomen and if present, may disappear.

Linea nigra, a dark pigmented line in the midline of abdomen extending from symphysis pubis upwards is also seen.

5. **Uterine changes:** After delivery, uterus is contracted, retracted and feels like a hard cricket ball. Its height is three cm below the umbilicus. It diminishes in size at the rate of 1.5 cm/day. On 6th day, its height is midway between umbilicus and pubis. On 14th day, fundus is at the level of pubic symphysis and comes to normal position at about 9th week.
6. **Labia majora:** Labia majora are tender, swollen, bruised or lacerated.
7. **Vagina:** Vagina is smooth walled, relaxed and capacious. It shows recent tears, which heal by 7th day. The rugae start reappearing in 3rd week.
8. **Fourchette:** Fourchette is lacerated in primipara and perineum shows recent tears.
9. **Cervix:** Cervix is soft and patulous, edges are torn and lacerations are present transversally. By 24 hours the internal os closes, external os admits two fingers and is soft. At the end of 1st week, the external os admits 1 finger with difficulty and by 2nd week the external os closes.
10. **Lochia:** Lochia is the discharge for 2-3 weeks after delivery and has a peculiar sour, disagreeable odour.
 - For 4-5 days (**Lochia Rubra**)—bright red containing blood clots.
 - Next 4 days (**Lochia Serosa**)—serous and pale in colour.
 - After 9th day (**Lochia Alba**)—yellowish green or turbid.
 - It finally disappears.
11. **Biological tests:** They are positive up to 7-10 days after delivery and is strong corroborative evidence of recent delivery.

Signs of Recent Delivery in the Dead

1. **Uterine Changes:** All the local signs of delivery may be present. Size of uterus will vary according to the period of gestation and time of survival. Immediately after the delivery, uterine wall is 4-5 cm thick and uterine cavity is obliterated by apposition of anterior and posterior walls. Total length of the uterine wall is 20 cm and length of the cavity is 15 cm. The size of uterus varies according to the passage of time after the delivery as tabulated below:

Duration	Length of uterus	Breadth of uterus	Thickness of uterus
2-3 days	17.5 cm	10 cm	—
7 days	13-14 cm	5 cm	—
14 days	<12 cm	—	—
6 weeks	7-8 cm	5 cm	5 cm

2. **Uterine weight:** The changes in the weight of uterus is:
 - After delivery—1000 gm.
 - End of 1st week—500 gm.
 - End of 2nd week—350 gm.
 - By 6 weeks—100-120 gm.
3. **Changes in placental area:** Shortly after the delivery placental site appears as an irregular, nodular and elevated area 15 cm in diameter. By the end of 2nd week it is 3-4 cm in diameter and at 6th week it is 1-2 cm in diameter.
4. Ovaries and fallopian tubes are congested and normal within few days.
5. Corpus luteum is found in one of the ovaries for 1-2 months.
6. Peritoneum covering the lower part of the uterus is arranged in folds.
7. Bladder is hyperemic and submucosal haemorrhages present.

Signs of Remote Delivery in the Living

1. Abdomen is lax and flabby and linea alba is present.
2. Breasts are soft and pendulous with dark areola.
3. Labia are not in apposition, are more or less separated.
4. Vagina is capacious, dilated with relaxed walls.

5. Fourchette and posterior commissure are lacerated.
6. Hymen shows carunculae myritiformes.
7. Perineum will show old tears.
8. Cervix cleft is transverse with ragged, irregular margins.

These signs may disappear in a woman who had delivered only once several years ago. Passage of uterine fibroid tumor through the vagina may produce some of the above mentioned signs.

Signs of Remote Delivery in the Dead

1. Abdomen lax and flabby and linea albicantes present.
2. Breasts are soft and pendulous.
3. Various uterine changes are:

Features	Parous uterus	Multiparous uterus
Size	9 cm long; 6cm wide; larger and thicker	7cm long; 4.5cm wide; smaller and thinner
Weight	80-120 gm	40-60 gm
Length	Body is twice the length of cervix	Body is of same length as cervix
Fundus	Top of fundus is convex and at higher level than the line of broad ligament.	Not so
Cavity	Walls are concave from inside and wider with rounded cavity.	Walls are convex with smaller with triangular cavity.
Cervix	Irregular, external os transverse admits tip of finger; internal os not well defined.	Regular with no scars. External os small dimpled in the middle; internal os is well defined.

LEGITIMACY

Legitimacy is defined under *S. 16 of Hindu Marriage Act* of children of void and voidable marriages. According to this section

1. Any child of null and void marriage who would have been legitimate if the marriage had been valid, shall be legitimate whether such child is born before or after the commencement of the Marriage laws (Amendment) Act, 1976 and

whether or not a decree of nullity is granted in respect of that marriage under this Act and whether or not the marriage is held to the void otherwise than on a petition under this Act.

2. Where a decree of nullity is granted in respect of voidable marriage under section 12 of Hindu Marriage Act, any child begotten or conceived before the decree, who would have been the legitimate child if at the date of decree marriage had been dissolved instead of been annulled, shall be their legitimate child.

Illegitimate children are: (i) Born out of wedlock between parents (ii) within wedlock, if the husband is sterile, is under age or due to other causes (iii) born out within competent period after cessation of relationship between husband and wife (iv) after blood tests paternity is excluded.

S. 5 of Hindu Marriage Act 1955: It defines the conditions of a Hindu Marriage as:

- i. Neither party has a spouse living at the time of marriage.
- ii. At the time of marriage neither party:
 - a. Is incapable of giving valid consent at the result of unsoundness of mind.
 - b. Though capable of giving a valid consent has been suffering from mental disorders to such an extent to be unfit for marriage and procreation of children.
 - c. Suffering from recurrent attacks of insanity.
- iii. The groom has completed 21 years of age and the bride 18 years of age at the time of marriage.
- iv. The parties are not within the degrees of prohibited relationship unless the custom allows such a marriage.
- v. Both the parties are not *sapindas* of each other unless customs or usage permits such a marriage.

S. 11 of Hindu Marriage Act: It defines **void marriages** as any marriage solemnised after the commencement of this Act shall be null and void and may be so declared by a decree of nullity if it contravene any of the condition in clauses (i), (iv) and (v) of section 5 of Hindu Marriage Act.

S. 12 of Hindu Marriage Act: It defines voidable marriages as:

1. Any marriage solemnised, whether before or after the commencement of this Act shall be voidable and may be annulled by a decree of nullity on any of the following grounds: (a) the marriage has not been consummated due to impotence of the respondent or (b) marriage in contravention of condition specified in clause (ii) of section 5 or (c) or the consent of the petitioner/guardian was obtained by force/fraud (d) that the respondent was at the time of marriage pregnant by some person other than petitioner.
2. No petition for annulling a marriage (a) on the ground specified in clause (c) of sub section (1) shall be entertain if (i) the petition is presented more than one year after the force had ceased to operate or fraud has been discovered (ii) the petitioner with his/her full consent had lived with the other party after the force had ceased to operate or fraud has been discovered (b) of the ground specified of clause (d) of sub section (1) shall be entertain unless (i) the petitioner at the time of marriage was ignorant of the alleged facts (ii) the proceeding have been instituted within one year of commencement of this Act and in case of marriages solemnised after the commencement of this Act, within one year from the date of marriage (iii) the marital intercourse has not taken place with the consent of the petition since the discovery of the existence of impotence of respondent.

Until 1976, the children of legal marriage and voidable marriages were called legitimate and that of void marriage were called illegitimate. After 1976, Marriage Laws Amendment Act 1976 came into force and the children born of void or voidable marriage shall be legitimate as if the marriage between their parents were valid, irrespective of the fact whether or not the marriage has been declared null and void under this act or whether or not the marriage is held to be void otherwise than in a petition under this Act.

Thus, it is quite encouraging policy by the legislature conferring legitimacy on all children, born of void and voidable marriages.

The question is why the innocent children should suffer for the fault of their parents and why they should be deprived of their legitimacy as to their birth and be forced to carry the social stigma throughout their life, apart from being deprived of the rights in the properties and assets of their parents.

The only provision is that under provisions of the Hindu law of succession legitimate children have right to properties of their parents and also to the properties of relatives as reversionary. Children of void and voidable marriages are liable to the properties of parents only.

S. 112 Indian Evidence Act

A child is presumed to be legitimate if it was born during the continuance of a valid marriage between his mother by any man or within 280 days after its dissolution, the mother remaining unmarried. The presumption can be rebutted if it is shown by competent evidence that:

1. Parties to marriage had no access to either at any time when the child could have been begotten.
2. It does not necessarily mean that the child is bastard because he is born beyond 280 days, as there is a wide variation.

Differences from English law

Presumption of legitimacy can be rebutted in England by proof of impotency and sterility of the husband, but no special point is given in the Indian law. In English law, illegitimate children become legitimate after subsequent marriage between parents and the child inherits the property.

Posthumous child is the one who is born after the death of the father; begotten during the lifetime of his father, the mother being conceived by said father.

Medicolegal Aspects of Legitimacy

The question of legitimacy arises in connection to right to inherit property when disputed, affiliation cases etc. The following questions need to be discussed:

1. **Inheritance:** Legitimate inherits the property of the father; a monster not having the shape of the mankind is incapable of inheriting. No special points relating to this are mentioned in Indian law.

2. **Affiliation cases:** A woman may allege a putative man to be father of her illegitimate child, may file a case. By some suitable method like exclusion of blood group, DNA fingerprinting paternity is fixed.

S. 125 CrPC: Order for maintenance of wives, children and parents. If an person having sufficient means neglects or refuses to maintain his wife, his legitimate or illegitimate minor child, his legitimate or illegitimate child who has attained majority, his mother or father—all unable to maintain themselves; the magistrate of 1st class may order such a person to make a monthly allowances of Rs. 500.

In determining the amount of maintenance under the Hindu Maintenance Act 1956 the court has to consider following points:

- The position and status of the party
- Reasonable wants of the claimant.
- The value of the claimant's property and any income derived from them.
- The number of persons entitled for the maintenance

3. **Supposititious children:** Supposititious children are fictitious that is not real or imaginary. A woman may substitute a living male for dead child or a living female or may feign pregnancy and delivery and subsequently produce a living child as her own when she wants to extort or the right to property. It is to be decided whether the age of the child corresponds to the date of pretended delivery.

4. **Paternity:** The question of paternity arises in cases relating to legitimacy, posthumous child or supposititious children. It is decided from

- *Paternal likeness:* The child may resemble the father in features and mannerism etc, but this is only corroborative.
- *Atavism:* The child does not resemble the parents, but resembles its grandparents. Due to inheritance of characteristics from remote rather than immediate ancestors, due to recombination of genes. Mental, physical, characteristics, tendencies, disease, and diathesis peculiar to remote ancestor may be inherited.
- *Developmental defects:* Disease or deformity may sometimes be transmitted from parents to offspring.
- *Blood groups and mutations:* Change is seen between generations resulting in an altered character in the child in 1:30,000.

Medico-legal Points of Importance

1. *Average duration of pregnancy between conception and delivery:*

- a. Period between last menstrual period and delivery; cannot be relied upon because menstruation may cease from other causes, may continue after pregnancy has commenced and impregnation may occur at any period during menstrual period.
- b. Observation of the period between a single coitus and delivery.

It's not so precise. Single coitus does not fix the date of conception, but only the date of insemination. Sperms retain their activity for 2-3days in the vagina and 4-5 days in cervix, uterus or fallopian tube. So the exact time of conception is not known. It is known that ovulation occurs 14 days prior to next period and the ovum probably persists in a day or two after it is shed unless fertilised. So fertilization occurs if sperm retains its

power and unites with the egg cell in the fallopian tube. So exact period is not known but calculated from experience i.e., 280 days or 40 weeks or 10 lunar months which is equivalent to 10 times of normal intermenstrual period.

2. **Maximum period of pregnancy:** No fixed limit is laid in India, England or USA. English Courts have accepted up to 349 days as maximum period, the child being legitimate.

Mckown & Gibson in their extensive study stated that 354 days are not impossible.

3. **Minimum period and viability:** Legitimacy may be disputed when a child is born within a short time of the husband and wife living together after a long separation. So important question is whether it is possible for a fully developed child to be born before usual period of gestation. Age of viability is 210 days, even at 180 days it is viable and live separately.

4. **Superfecundation:** It is fertilization of two ova of same cycle of ovulation by two separate acts of coitus, committed at short intervals. Incidence of twin pregnancies is 1.5%. It occurs in 70% of bin ovular twins, resulting from separate fertilization of two ova at the same period. Development of twins in the uterus is parallel, not equal depending upon placental blood supply. The two placentas are developed. One foetus may be aborted, die or may remain till the end. The other may press the dead foetus, so that it becomes one mass called *foetus compressus*. Spermatozoa causing fertilization may be from different man having different blood group. Cases are on record where one of the twins belonged to the one father by blood group.

5. **Superfoetation:** It is the fertilization of second ovum in a woman, who is already pregnant. Subsequently the two foetuses are born either at the same time showing different stages of development or two fully developed foetuses are born at different periods varying from 1-3 months. There are cases where 2nd child is delivered after a period of the 1st one, twin is

expected. So after the delivery of 1st one 2nd got proper nourishment and delivered. But now a days, it is said that it is possible though rare because of:

- a. Ovulation may take place during 1st three months of gestation until decidua Vera and decidua reflexa come in contact and close the decidual cavity.
 - b. Double uterus
6. Lastly it is to be examined whether the woman has delivered a living child or not i.e., signs of recent or remote delivery to be found.

Short Questions

1. Types of hymen.
2. True and false virgin.
3. Vaginismus.
4. Frigidity.
5. Superfecundation.
6. Medicolegal importance of pregnancy.
7. Precipitate labour.

MULTIPLE CHOICE QUESTIONS

1. In Nulliparous, cervical opening is:
A. longitudinal B. Circular
C. Transverse D. Fimbriated
2. A 22 year-old-lady died. The postmortem appearance that will indicate that she has had delivered a child are all **except**:
A. Walls of uterus are convex from inside
B. Cervix is irregular and external os is patulous
C. Body of uterus is twice the length of cervix
D. Uterus is bulky, large and heavy than nullipara
3. The common site of rupture of hymen in a virgin is:
A. Anterior B. Posterior
C. Antero-lateral D. Postero-lateral
4. Atavism is resemblance of features to the:
A. Mother B. Father
C. Neighbor D. Grandfather
5. A normal full term placenta weighs about:
A. 200 gm B. 400 gm
C. 500 gm D. 600 gm
6. Genitals in virgin girl are, **except**:
A. Fourchette and posterior commissure are intact
B. Labia minora are covered by the labia majora
C. Hymen is a narrow opening
D. Vagina is rugose and separated apart.
7. Spot out the wrong statement about hymen:
A. It is a membranous structure about 1 mm thick
B. In children it looks like a taut membrane when the thighs are separated
C. It is unyielding or elastic and easily distensible
D. It is sufficiently distensible to always admit male adult penis without rupture on first occasion
8. True virgin includes all, **except**:
A. Woman has intact hymen
B. Its edges are distinct
C. Edges are regular
D. Admits only tip of the little finger
9. Accessory signs of false virginity includes all the following, **except**:
A. Labia minora are fully covered by labia majora
B. Clitoris is enlarged
C. Vestibule is gaping
D. Vagina is roomy
10. Spot out the wrong statement about nullity of marriage:
A. Either party is suffering from incurable impotency
B. There is willful refusal to consummate
C. When wife was pregnant by any person other than the husband
D. Unnatural sex practices

Abortion

Bleeding is one of the important abnormalities during early pregnancy that occurs due to spontaneous abortion (due to natural causes), ectopic gestation, tumors, cervical lesions and endometrial bleeding.

Medical Definition of Abortion

Abortion means spontaneous or induced expulsion of products of conception before viability (28 weeks) whereas premature labor is delivery of fetus after 28 weeks of pregnancy up to 40th week. In medical practice, the abortion occurs in 1st trimester, miscarriage in the 2nd trimester premature labor in the 3rd trimester. Legally all the above terms are synonymous.

Legal Definition

Abortion means expulsion of products of conception from the uterus at any period prior to full term. The products of conception may be ovum, embryo or fetus. Law makes no distinction between abortion, miscarriage and premature labor. The products of conception are termed by the terminologies as mentioned below as Ovum is the product of conception between 7-10 days of gestation. Embryo is after 10 days until the end of 9th week of gestation. Foetus is called when it is more than 9th weeks of intra uterine life until birth

Classification of Abortion

1. Natural due to natural causes before 28 weeks
 - i. Spontaneous

- ii. Accidental
2. Artificial (Induced)
 - i. Justifiable
 - ii. Criminal
3. Clinically
 - i. Inevitable
 - ii. Incomplete
 - iii. Threatened
 - iii. Complete
 - iv. Septic abortion may be due to incomplete abortion
 - v. Missed abortion in which there is formation of careous or blood mole with more than 8 weeks gestation
 - vi. Habitual or recurrent—When abortion occurs at three consecutive times
 - vii. Criminal abortion when the pregnancy is terminated unlawfully without any proper indication.

Threatened abortion: Threatened abortion usually occurs in the early weeks of pregnancy. The patient presents with moderate vaginal haemorrhage and mild pain. On examination, the opening of cervical os is closed.

Inevitable abortion: The patient presents with severe pain, increased uterine contractions dilated cervix.

Incomplete abortion: There is continuous bleeding from the placental site and retention of placenta is one of the commonest complications of incomplete abortion. The patient presents with severe anemia. These cases are to be evacuated by surgery and drug like ergometrine is to be given to induce uterine contraction so that the placenta

comes out.

Missed abortion: In this type of abortion, the fetus dies in utero and is retained for more than weeks in the uterus. The history is quite characteristic in such cases, as initially for 12 weeks, there are normal signs and symptoms of pregnancy. Thereafter slight bleeding occurs that stops suddenly and the patient develops amenorrhoea. The abdomen is not enlarged but brown discharge starts per vaginum after several weeks or months. In this type of abortion, the products of conception are converted to ovoid, fleshy mass due to recurrent or slow haemorrhage in to chorionic spaces.

Mechanism of Abortion

Abortion corresponds to the process of normal labor at term; body of uterus contracts and retracts and cervix dilates.

1. Expulsion of the whole ovum in one piece: During early weeks of pregnancy, the vera and basalis layer of Decidua separates, so that the entire decidua and ovum are expelled out together.
2. Expulsion of the whole ovum by inversion of the decidua vera or parietalis: This common method of typical abortion is due to detachment of ovum by means of retro-placental clot, the ovum is pushed in to the uterine cavity is expelled and then the inversion of deciduas vera and parietalis occurs.
3. Incomplete expulsion of ovum with the retention of the placenta and its membranes.
4. In missed abortion, ovum is separated from its attachment to the uterus to a degree sufficient to kill the embryo, but the detached embryo is retained.
Pregnancy test previously positive becomes negative.

NATURAL ABORTION

Natural abortion occurs before 28 weeks of pregnancy and the incidence being 5-20% of all pregnancies with an average of 10%.

Etiology

1. Abnormalities of the fetus (50% of all abortions):

- i. *Maldevelopment of the fetus:* The abortus in first two months shows errors of development of fetus like hydatidiform mole, degeneration of placenta and maldevelopment of the embryo.
- ii. *Intrauterine death:* The death of the fetus in utero can develop from the following causes
 - *Infections:* If mother contracts infections such as pneumonia, malaria, typhoid, dysentery, smallpox, syphilis etc, foetus is liable to die. The infecting organism in syphilis, smallpox and rubella does not pass through the placental barrier. As a result, patient usually dies from the pyrexia and not the ill effect of the causative organism.
 - *Poisons:* The most dangerous of the poisonous substances is lead and to some extent quinine. In such cases, the mother becomes gravely ill before there is any effect on the ovum.
 - *Radiological effects:* Usually, the patient is being treated for malignant disease without the knowledge of status of pregnancy. When the patient gets sufficient exposure in the 1st trimester, the foetus dies in the uterus or else in foetal abnormalities occur.

2. Abnormalities of the placenta and membranes:

- i. *Acute hydramnios:* In acute hydramnios, the amount of amniotic fluid is more than 2000 ml.
- ii. Hydatidiform degeneration of placenta
- iii. *Placenta praevia:* The placenta is attached in lower uterine segment resulting in severe bleeding after abortion. This usually occurs in second trimester.

3. General diseases of the mother:

- i. *Acute infections:* The generalized infections can result from the diseases namely meas-

- les, scarlet fever, cholera, typhoid, diphtheria, malaria and small pox. The localized infections resulting in abortion are pneumonia, pneumococcal peritonitis after evacuation, erysipelas and upper respiratory tract infections.
- ii. *Chronic infections*: The chronic infection in mother that can cause abortion are syphilis (Never causes an abortion before mid pregnancy), Tuberculosis (not a common cause but if the disease is widespread and rapidly progressive, abortion can occur), other chronic diseases such as hypertension and chronic nephritis. These diseases basically interferes with placental circulation and foetal blood supply.
4. *Local abnormalities of the mother*:
- i. Local abnormalities of the genitalia such as genital hypoplasia, hypoplasia of uterus associated with gross ovarian deficiency, partial duplication of upper part of uterus, bicornuate uterus, septate uterus and subseptate uterus.
 - ii. *Displacement of uterus*: The attempts at correcting the position of retroverted uterus in second and third trimester causes abortion. In fourth month, the uterus comes out of pelvis so there are no chances of abortion.
 - iii. Fibromyoma of submucous or polypoid type may also result in abortion. However, removal of tumour surgically resulted in normal pregnancy.
 - iv. *Cervical incompetence*: The cervix is patulous and abortion usually occurs in 2nd trimester due to premature rupture of membranes.
 - iv. *Surgical operations*: Any operation on pregnant woman however trivial and anaesthesia involves risk of abortion. The risk increases nearer to the genital tract the operation is performed. Myomectomy carries the greatest risk of abortion and nervous women are liable to abort even after tooth extraction.
 - v. *Drugs*: Drugs such as phosphorus, lead, Quinine, ergot, mercury in poisonous quantities may lead to abortion from their toxic effects upon the mother.
- vi. *Injuries*: The following injuries are liable to cause abortion
- The accidents and injuries to lower abdominal region and vulva results in formation of haematoma in the region of uterus and the placenta is dislodged
 - Trauma of coitus
 - Fatigue and jolting of a long car journey
 - Violent physical exercise, riding bicycle
- vii. *Immunologic causes*: The maternal lymphocytes invade the foetus and the women who reject skin graft of their husband are liable to abort more.
- viii. *Disturbance of Endocrine System*: Secretion of corpus luteum is essential for embedding of the fertilized ovum in the first few weeks of pregnancy. After 12-14 weeks, the functions of corpus luteum are replaced by placenta and even if both ovaries are removed, patient goes to term. In diabetes and thyrotoxicosis, there is tendency to abort because of the increase in Prostaglandins E₂ & F₂ in amniotic fluid.
- ix. *Psychiatric causes*: These are the frights, fear, acute mental tension and acute anxiety neurosis.
5. *Abnormalities in the male*: During investigations for infertility and repeated abortion the semen analysis is done and in large number of abnormal sperms are seen causing foetal abnormalities and abortion.

ARTIFICIAL ABORTION

Justifiable (Therauptic) Abortion

Abortion is justified when done in good faith to save the mother's life if it is materially endangered by continuation of pregnancy. It is not done just to save family honour or for ethical or economical reasons. Abortion is justifiable if mother's health is likely to be permanently damaged by continuation of pregnancy. WHO defines health as a state of complete physical, mental and social

well being and not merely the absence of illness and disease.

Precautions to be take by the doctor while performing therapeutic abortion

1. Another doctor to be consulted, preferably a specialist in the field of obstetrics and gynaecology
2. The written consent of woman and her husband to be taken
3. The procedure should be undertaken in a government hospital
4. The doctor should wait for conducting abortion till the child attains viability.
5. While performing abortion, the presence of a witness preferably a nurse is must

Indications for Performing Therapeutic Abortion

1. *Obstetrical indications:*
 - Severe eclampsia (hypertensive disorder; convulsion and coma).
 - Psychiatric conditions (relative depression, suicidal tendencies and schizophrenic states).
 - Unwanted pregnancy in a mentally sub-normal woman.
2. Malignant conditions such as invasive carcinoma of cervix, carcinoma of ovary and breast cancer with metastasis.
3. Cardio-vascular conditions such as valvular heart disease, cardiac failure, congenital heart disease, atrial fibrillation and hypertension.
4. Respiratory conditions such as respiratory insufficiency in lung diseases, chronic bronchitis and asthma.
5. Alimentary conditions such as peptic ulcer, ulcerative colitis with perforation and bleeding, pancreatitis and acute hepatitis.
6. Renal conditions such as nephrotic syndrome.
7. Endocrine and Metabolic conditions such as Diabetes mellitus, parathyroid tumour and osteomalacia.

8. Neurological conditions such as spinal and cerebral tumours, recurrent epilepsy, Hereditary spastic paraplegia, myasthenia gravis.
9. Psychological and emotional conditions when the child is unwanted and is the result of incest and rape. When the patient is suffering from grave neurosis and suicidal tendencies.
10. Conditions causing foetal abnormalities: These are:
 - Infective conditions (Rubella, Mumps).
 - Mother exposed to drugs (Thalidomide, androgens and oestrogens).
 - Rh incompatibility.
 - Down's syndrome (mongolism).

Methods of Termination of Pregnancy

1. Dilatation and curettage.
2. Cervical dilatation and oxytocin drip.
3. Vacuum suction at 6th-12th week
4. Intra amniotic injection of prostaglandin F₂ alpha: 25 mg in 2nd trimester.

Criminal Abortion

Criminal abortion is the induced destruction or expulsion of the foetus from the womb of the mother unlawfully that is where there is no therapeutic indication for operation. Criminal abortion is usually undertaken by the

1. Widows for remarriage.
2. Unmarried girls owing to rigid social customs and pregnancy resulting from illicit intercourse.
3. Married woman of educated middle class to avoid addition to the family. Now a days it is less due to widespread knowledge of contraceptives.
4. The abortion may be undertaken solely for the purpose of sex determination to avoid female child. It is done in 2nd and 3rd month but rarely during 4th or 5th month. It is investigated when the woman dies or some one informs the police.

Types of Abortionists

The abortionists can be divided in to three groups:

1. Expert or medically qualified professionals.

2. Semi skilled abortionists such as midwives, nurse, chemists.
3. Unskilled.

Methods to induce Criminal Abortion

1. Mechanical violence (general and local).
2. Abortifacient drugs.
3. Instruments.

General Mechanical Violence

These methods may act directly on the uterus or act indirectly by promoting congestion of pelvic organs and causing haemorrhage between uterus and pelvic membranes. These methods are:

1. Severe pressure over the abdomen as caused by the blows, kicks, tight lacing and jumping.
2. Violent exercise such as riding, cycling, jumping from height, jolting, driving on rough roads and lifting heavy weights.
3. Cupping: Cupping is placing a lighted wick over the hypogastric area and turning a brass mug over the wick. The traction is applied over the mug that results in separation of placenta. The method is usually employed in advanced months of pregnancy.
4. Application of Leaches to pudenda, perineum and inner surface of thigh.
5. Very hot and cold baths alternatively.
6. Massaging the uterus through abdominal wall.

Local Mechanical Violence

While correcting the retroverted uterus bimanually, the abortion may result.

Abortifacient Drugs

The abortifacient drugs are:

1. *Ecbolics*: They act directly on the uterus and increase the uterine contraction that causes abortion. These are Ergot (Ergometrine) that is used quite commonly, Quinine, Cotton root bark, posterior pituitary extract, potassium permanganate tablets (used locally in vagina and cause local ulceration and bleeding), Lead pills (lead oleate causes tonic contraction of uterus), Nitrobenzol and Strychnine

2. *Emmenagogues*: They produce an increase menstrual blood flow and act as abortifacient when given in large doses. These are Savin, Borax, Apiol (toxic effects are due to tricesyl phosphate) and Prostaglandins

3. *Irritants of genito-urinary tract*: They provide reflex uterine contraction and are Oil of tansy, Oil of turpentine, Cantharides (in large doses produce inflammation of the kidney and albuminuria), Potassium permanganate (120-300 ml through vaginal route) produces inflammation and hemorrhage due to erosion of the vessels.

4. *Irritants of the Gastrointestinal tract*: Such as Emetics (tartaric acid), Purgatives (Colocynth), Castor oil, Croton oil and magnesium sulphate. They will excite uterus to contract "in sympathy" with the violent contractions of stomach and colon and may produce hyperemia.

5. *Drugs having poisonous effect on the body*:
 - i. Inorganic irritants such as lead, copper, antimony, mercury and arsenic.
 - ii. Organic irritants such as unripe fruits of Papaya and Pineapple, bark of Plumago rosea and juice of Calotropis. Criminal abortion
 - iii. Abortion Pill F-6103 was developed in Sweden and has Diphenyl-Ephylene. It concentrates in the corpus luteum and acts by stimulating a woman's natural inclination to menstruate even after presence of fertilized ovum and causes incomplete abortion. The other forms of abortifacient pills are Dr. Reed's extra strong female pills and Widow Welch's pills.

Instruments

The instruments are used for the purpose of:

1. Rupture of membranes by introduction of penetrating instrument such as uterine sound, catheter, pencil, knitting needle, hairpin, stick and even fingers. The patient visits the medical practitioner and falsely tells that uterine displacement has occurred. The doctor passes a sound to correct the displacement with the

resulting abortion. Before passing sound, the doctor must be satisfied that the patient is not pregnant.

Dilatation of the cervix: The bark of *Slippery elm*, a tree found in central and North America is used by unskilled abortionists to dilate the cervix. The soft flat pieces of wood of different size with the usual thickness is about 3 mm is passed in to the cervical canal and left in situ. It absorbs moisture and vaginal secretions and swells up that dilates the cervical canal resulting in abortion. Even compressed sponge when left in situ in the cervical canal swells up to cause cervical dilatation and abortion. *Slippery elm* bark on each side bears a jelly like layer as thick as the bark itself. This property makes the bark self lubricating that swells on absorbing the vaginal secretions and causes cervical dilatation. The disadvantages are that the method is unhygienic and there are chances of infection. At times the stick may pass in to urethra and gets lodged in bladder as foreign body.

2. *Abortion stick:* Abortion stick is a thin wood or bamboo stick, 12 to 18 cm (5-8 in) long and its one end is wrapped with cotton wool or a piece of rag whose greater part is soaked with juice of Marking nut, Calotropis or a paste made of Arsenic oxide, sulphide or red lead etc. Instead of stick, a twig of irritant plant such as calotropis, Nerium odorum or Plumago rosea
3. *Air insufflation:* The air is instilled in to vagina by means of syringes or pumps
4. *Electricity:* Negative pole is applied over the cervix and positive pole over sacrum or lumber vessels and then the current is passed that results in uterine contractions and subsequent abortion.
5. *Paste:* The paste containing iodine, thymol or mercury (*utus pastis*) is injected from a collapsible tube with uterine applicator into the uterus
6. *Syringing:* Enema syringe with a hard bulb is commonly used to inject fluid in to the uterus and the hard nozzle is inserted in to cervix.

Sometimes Higginson's syringe is used. The suction valve is placed in a bowl of fluid and pressure is applied on the bulb. A mixture of air and fluid is forced in to uterine cavity at a higher pressure than uterine veins. The fluid detaches parts of amniotic sac and placenta from the uterine walls. The uterus contracts causing haemorrhage and thus abortion. The fluid comprises of soap water, water containing inorganic and organic acids, Lead, Arsenic, Lysol, cresol, formalin, $Kmno_4$ etc. the fluid can be administered by the patient or by an abortionist.

Major risks of syringing are: (i) Due to the injection of cold or hot water death may result from vagal inhibition when injected in to cervix (ii) Air embolism due to stripping of the edge of placenta and the air enters in the venous channels.

Complications of Procuring abortion

1. Toxaemic shock and death due to absorption of substances through uterine and vaginal mucosa.
2. Sepsis from peritonitis, endometritis and salpingitis.
3. Fatal haemorrhage resulting from retained products of conception perforation of cervix, vaginal vault and fundus of uterus.
4. Necrosis of cervix.
5. Delayed air embolism: The mechanism of delayed air embolism is yet to be explained. Air is held in the same fashion in the uterus till such time placental separation opens the vessels to allow it to pass in to circulation. The mucus plug would possibly prevent escape of air via cervix. The lethal dose to cause air embolism and death is minimum 100 ml whereas in animal experiments it is 10-480 ml. (Fig. 27.1, Table 27.1).

Fabricated Abortion

To have a false charge of abortion the females sometimes present animal foetus. In such cases, precipitin test is confirmatory as is a species

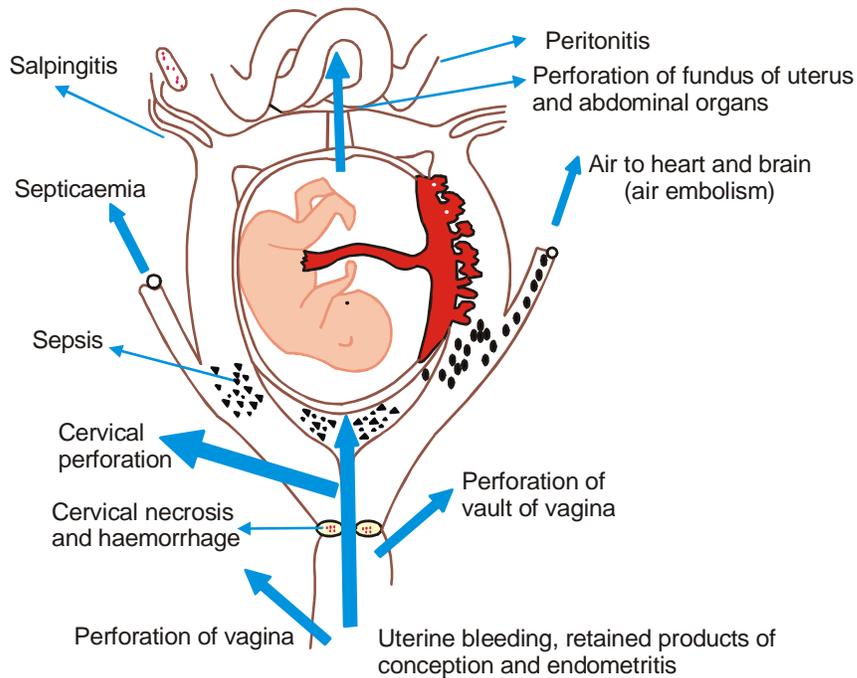


Figure 27.1: Complications of procuring abortion

specific. The symptoms and signs of extrauterine pregnancy give rise to the suspicion of criminal interference

Trauma and Abortion

To establish causal relationship between trauma and abortion, following factors should be considered:

1. The traumatic event was followed within 24 hours by a process that ultimately leads to abortion.

2. The foetus and placenta should be normal.
3. The appearance of foetus and placenta should be compatible with the state of gestation when the traumatic event occurred.
4. Factors known to cause abortion should be absent.

Rupture of membranes or separation of placenta may occur in some cases directly due to trauma. Following criteria suggests a causal relationship:

1. Normal progress of pregnancy.

Table 27.1: Differences between natural and criminal abortion

Features	Natural abortion	Criminal abortion
Cause	Any woman with predisposing disease.	Unmarried woman or widow
Infection	Rare	Frequent
Marks of violence on abdomen	Not present	May be present
Genital organs	No injuries	Injuries present
Toxic effect of drugs	Absent	Present
Foreign bodies in genital tract	Not present	Present
Foetal wounds	Absent	Present

2. Absence of predisposing factors.
3. Signs and symptoms start within 48 hours of placental detachment.
4. There is adherent clot or depression of the placental surface.

Medicolegal Questions that Should be Answered

1. Was the deceased pregnant recently?
2. Was there any evidence of abortion?
3. Was there any evidence of criminal abortion?
4. Was the cause of death related to abortion?

Causes of death in criminal abortion

1. *Immediate causes:*
 - i. *Shock and haemorrhage:* Due to injury as a result of criminal interference uterine perforation and death results from haemorrhage
 - ii. *Air embolism:* Entry of about 100 ml of air in to the veins of uterus causes air embolism. When a soapy solution that is mixed with air is injected with pressure, the patient collapses within 10 minutes.
 - iii. *Reflex vagal inhibition:* Vagal reflex starts by touching the cervix, lower uterine segment by an instrument or irritant fluid and during anaesthesia prior to abortion
 - iv. Fat embolism may result due to injection of soapy water and corrosives such as Lysol
2. *Delayed causes:*
 - i. *Septicemia and pyaemia:* Sepsis may result from the dirty surroundings, dirty instruments and injury to uterus while performing abortion. The organisms commonly involved are *Clostridium welchii* and *Clostridium tetani*.
 - ii. General peritonitis.
 - iii. Toxaemia.
 - iv. Tetanus.
3. *Remote causes:*
 - i. Jaundice and renal suppression
 - ii. Bacterial endocarditis
 - iii. Pulmonary embolism.
 - iv. Pneumonia.
 - v. Empyema and Meningitis.
 - vi. Poisonous effect of the drug itself.

Diagnosis of Abortion in the Living

The cases come to the doctor when:

1. Woman alleges abortion after a blow or quarrel in order to inflict penalty on assailant
2. Woman is charged with abortion but wishes to conceal it

The signs of recent abortion are essentially the signs of recent delivery. The findings depend upon the length of gestation and the time elapsed between abortion and examination. The signs of recent abortion are

At 2-3 months

- Haemorrhage
- Slight softening of external os and maternal passage.
- Slight enlargement of uterus.
- Breast changes.

4-5 months

- Increased haemorrhage
- Internal os – one finger.
- The os is not injured if abortion less than 6 weeks.
- Vagina may be lacerated.
- Os injured.
- May be sepsis due to *Cl. welchii*, *E. coli* or *Streptococcus pyogenes* infection

Material and liquid from the vagina, uterine cavity and blood should be collected for chemical and bacteriological examination. Urine, faeces and vomitus should also be preserved for chemical analysis.

Examination of alleged material

1. The material alleged to be the product of conception should be thoroughly washed and kept for sometime in water to dissolve the blood clot.
2. A portion should be examined microscopically and the blood clots submitted for grouping and precipitin test.
3. The mass suspected to be foetus is cut and mounted on a slide with glycerine or water and then seen under low power.
 - i. In early months, if embryo is not found there is formation of chorionic.

- ii. Up to 9th week; decidual cells have cells with eccentric nuclei, central core of blood vessels.
- iii. At 1st to 3rd month foetus is expelled with its membranes en mass.
- iv. After this period the foetus is born first then placenta is detached and expelled with a portion of it may remaining inside.
- v. If placenta is there, it should be examined whether in one piece or torn at any place or have any degenerative changes.
- vi. If it is foetus it should be assessed for intrauterine age, viability and presence or absence of injuries over the body.

Findings on microscopical examination: The decidual cells appear as fine, pale staining, round or polygonal cells, often with eccentric nuclei. The chorionic villus has a central core of connective tissue with blood vessels. The trophoblast consist of inner layer of cells of Langhans and the outer syncytial layer that is richly nucleated protoplasm without cell boundaries.

Postmortem Evidence of Criminal Abortion

The evidence of abortion should be obtained from:

1. Medical history
2. Clinical or autopsy examination.
3. Examination of aborted material.

Sudden death of a woman of childbearing age when the:

 - i. Deceased is pregnant and deeply cyanosed.
 - ii. Instruments or drugs found at the scene of death.
 - iii. Underclothing is apparently disturbed
 - iv. Fluid, soapy water or blood stained discharge is coming out of vagina should always arouse suspicion of criminal abortion.

To convict the abortionist it is important to ascertain that the:

1. Dead woman was pregnant
2. Accused was responsible for the act that resulted in the interruption of pregnancy.
3. Accused acted in for the purpose of producing an illegal abortion.

4. Death occurred as a result.

The autopsy findings depend upon the mode of abortion, time elapsed between death and examination. The pathologist should exclude causes of spontaneous abortion while conducting autopsy.

Special methods of dissection while performing autopsy

An incision is made just outside the labia majora that is carried backwards to include anus and forwards up to symphysis pubis that is then divided.

Parietal peritoneum is cut around the brim of pelvis and by careful dissection the whole of pelvic viscera including uterus, vagina, anus, rectum, and bladder is removed and examined carefully in detail.

Duties of the Doctor

Professional duties

1. Detailed history should be taken:
 - Record the condition and treat her with best available facilities.
 - The doctor should not try surgical methods on the patient as there may be some false charge. Another doctor should be consulted if some treatment is needed
 - For chemical analysis, the doctor should preserve every article and uterus with contents should be preserved for histopathological examination. The blood, urine etc should be preserved for bacteriological analysis.

Legal Duties

1. The doctor should maintain professional secrecy.
2. If the patient is seriously ill, it is imminent to inform the police.
3. When death is eminent, the doctor should arrange for recording the dying declaration.
4. In case of death, the doctor should not issue death certification but instead inform the police.

Legal Aspects of Abortion

The abortion should not be conducted for monetary gains or sympathy. It should only be performed as per the provisions of the MTP act.

MTP Act 1971

The Central Family Planning Board of Govt. of India- a policy making body, at a meeting held on 25th August 1964 expressed concern about problems of abortion and recommended to appoint a committee to examine the subject of abortion in all its legal, medical, moral, and social aspects and to make suitable suggestions to alter the existing law. On 19th September 1964 Ministry of health appointed, Mr. Shantilal Shah, Health Minister of Maharashtra as Chairman of the committee. In Dec 1966, the committee submitted its recommendations and the Bill was introduced in 1969. the Bill was passed in Rajya sabha and Lok sabha on 2nd August 1971. the Act was enforced in India except the state of Jammu and Kashmir on 1st April 1972. The act lays down the:

1. Conditions under which a pregnancy can be terminated
2. The person/persons who can perform such terminations.
3. The place where such terminations can be performed.

Conditions under which the pregnancy can be terminated: Section 3(2) provides the grounds on which MTP can be done.

1. *Therapeutic:* The continuance of pregnancy would involve a risk to the life of the pregnant woman or grave injury to her **physical or mental health**.
2. *Eugenic:* There is a substantial risk that if the child were born, it would suffer from such **physical/mental abnormalities** as to be seriously handicapped.

Explanation 1: When pregnancy of the alleged woman has been caused by rape, which shall be presumed to constitute a grave injury to mental health.

Explanation 2: Where the pregnancy has occurred as a result of failure of contraceptive, which may be presumed to constitute grave injury to the mental health of the woman.

The person/persons who can perform the terminations: The act provides safeguards to the mother by authorising only a registered medical practitioner having experience in obstetrics and gynaecology; up to 12 weeks to perform such terminations. Two registered medical practitioners are necessary for termination after 12 weeks up to 20th week.

Place where terminations can be performed: The Act stipulates that it should be done in the govt., semi govt., or private hospitals approved for this purpose.

Consent:

1. Written consent of the guardian is required where age is less than 18 years and in cases of lunatics.
2. In other cases consent of the pregnant woman is necessary and that of the husband is not required.
3. Profession secrecy should be maintained by the doctor.
4. Sterilisation is not a criterion but the patient should be advised to use contraceptive methods.

Section 8 provides safeguards to the doctors. No legal action will be taken against any registered medical practitioner for any damage caused or likely to be caused which has been done in good faith.

The medical termination of pregnancy rules 1975 stands repealed because medical termination of pregnancy rules 2003 has come into force. It lays down the experience and training for the registered medical practitioners.

1. Any medical practitioner having experience of 3 years in obstetrics and gynaecology or who has completed 6 months housemanship in obstetrics and gynaecology or worked in a hospital in a department of obstetrics and gynaecology for at least one year can perform the MTP.

2. Any registered medical practitioner who has assisted 25 cases out of which five cases he has done independently in a hospital approved by the government can perform the MTP. The registered medical practitioners who have assisted in 25 cases can do only first trimester terminations.
3. Any medical practitioner having diploma or degree in obstetrics and gynaecology can perform MTP.

MTP Rules 1975

MTP Rules were altered in October 1975 to eliminate time consuming procedure and to make services more readily available. These include the

1. Approval by Board: Under the new rule, Chief Medical Officer of the district is empowered to certify that a doctor has the necessary training in Obs & Gynae, to do the abortion.

The procedure of doctors applying to certification to Board was removed.

2. *Qualification required to do abortion:* The new rules allows for the Medical practitioners to qualify through on the spot training; if he has assisted in 25 cases of MTP in an approved institution.

The doctor also qualifies if he has done

- 6 months houseman ship in Obstetrics and Gynaecology.
- A postgraduate qualification in Obstetrics and Gynaecology.
- 3 years practice in Obstetrics & Gynaecology before 1971 Act.
- 1 year practice in Obstetrics & Gynaecology after 1971 Act.

3. Place where abortion can be conducted: Under new rules, non-governmental institutions may take up abortions provided they get a license from the Chief Medical Officer of the district, thus eliminating the requirement of private clinics obtaining a Board license.
4. Emergency provisions of the Act.

MTP Regulations 2003

These regulations define the conditions of

maintaining records in the centres in which MTP can be done.

All the forms filled for the procedure of conduction of the medical termination of pregnancy must be kept confidential and are not to be kept open to inspection. The consent form filled up by the patient together with the certified opinion of the doctor along with the intimation of termination of pregnancy should be kept in a sealed envelop and marked SECRET and then sent to the head of the hospital or the Chief Medical Officer of the state.

Other aspects of the Act: Failure of contraceptive is quite rare and difficult to prove and sometimes there are circumstances where unmarried girls undergo voluntary intercourse.

Provisions in Indian Law

Sections 312, 313, 314, 315, 316, I.P.C. refers to the offences of criminal miscarriage and punishments awarded for these offences.

S. 312 I.P.C. 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 the 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: The woman should be pregnant and miscarriage should be caused with her consent; both are liable to punishment unless done in good faith to save the life of mother. The punishment is extended to seven years if the woman is quick with the child i.e. above 16th week.

If the means used for causing miscarriage are not successful, they are liable to be punished under S.511 I.P.C. that half of the punishment under Section 312 will be awarded.

S.313 I.P.C 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 is not, shall be punished with imprisonment for life or with imprisonment of either description for a term which may extend to ten years and shall also be liable to fine.

S. 314 I.P.C. Death caused by act done with the intent to cause miscarriage: Whoever, with the intent to cause miscarriage of a woman with the child, does any act which causes the death of such woman, shall be punished with imprisonment of either description for a term that may extend to ten years and shall also be liable to fine. If the act is done without the consent of the woman, shall be punished either with imprisonment for life or with punished above-mentioned.

Explanation: It is not essential to this offence that the offender should know that the act is likely to cause death.

S. 315 I.P.C Act done with intend 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 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.

S. 316 I.P.C Causing Death of the Quick Unborn Child by Act Amounting to Culpable Homicide:

Whoever does any act under such circumstance, that if he thereby caused death would be guilty of culpable homicide, and does by such act cause 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.

In cases of offences against children in uterus, where pregnancy has advanced beyond stage of quickening and where death is caused after the quickening and before the birth of the child. The person would be guilty of culpable homicide if he caused the death of the woman by an act which he knows is likely to cause her death and if his act causes the death of unborn child and injured the woman will be held guilty under Section 316 I.P.C.

Long Question

1. What is abortion? Discuss various methods of medical termination of pregnancy and criminal abortion. What findings can be present in a case of death from criminal abortion?

Short Questions

1. Abortion stick.
2. Cupping.
3. MTP act 1971.
4. Precipitate labour.

MULTIPLE CHOICE QUESTIONS

1. **MTP Act was passed in the year:**
 - A. 1952
 - B. 1961
 - C. 1971
 - D. 1981
2. **Which is the incorrect statement about therapeutic grounds of abortion?**
 - A. Where pregnancy is a threat to the life of mother
 - B. Grave injury to her physical and mental health
 - C. Organic heart disease with failure
 - D. Anemia of the mother
3. **Indications of abortion on eugenic grounds includes all the following, except:**
 - A. Where there is substantial risk to the child of physical and mental abnormalities
 - B. If the pelvis has received exposure of 30 rads and definitely if over 200 rads
 - C. When woman has received cytotoxic drugs
 - D. When mother has suffered from German measles
4. **The doctor should take all the following precautions in a case of criminal abortion, except:**
 - A. The information regarding a woman having undergone abortion should be kept as professional secret
 - B. Doctor should gather all information about place and procedure of abortion being conducted
 - C. If death is imminent he should arrange for dying declaration
 - D. He can refuse to give death certificate but should inform the police
5. **Complications of criminal abortion includes, except:**
 - A. Shock
 - B. Hemorrhage
 - C. Air or fat embolism
 - D. Anemia
6. **One medical officer can allow MTP upto:**
 - A. 8th weeks of pregnancy
 - B. 10th week
 - C. 12th week
 - D. 20th week
5. **According to MTP Act 1971, medical termination of pregnancy can be done in pregnancy upto:**
 - A. 8 weeks
 - B. 12 weeks
 - C. 20 weeks
 - D. 24 weeks

Assisted Reproduction Technique and Cloning

ARTIFICIAL INSEMINATION

Artificial insemination is a process when the sperms are deposited in the female genital tract artificially when the female cannot conceive through the sexual intercourse with her husband. The sperms may be introduced in the cervical canal (intracervical) or inside the uterus (intrauterine). Less common techniques are intrafallopian and intraperitoneal insemination when the sperms are placed near the mouth of the fallopian tube and ovaries respectively. Rarely, a technique called intravaginal insemination is used, in which sperm are placed in the female partner's vagina.

Modern techniques for artificial insemination were first developed for the dairy cattle industry to allow many cows to be impregnated with the sperm of a bull with traits for improved milk production. Artificial insemination is used in animals to propagate desirable characteristics of one male to many females or overcome breeding problems, particularly in the cases of horses, cattle, swine, pedigree dogs, and honeybees. Artificial insemination of farm animals is not uncommon, especially for breeding dairy cattle. It provides an economical means for a livestock grower to breed their herds with males having very desirable traits.

History

Artificial insemination is commonly thought of as a modern technology but it has a long history. Thus, apparently artificial insemination was attempted on Juana, wife of King Henry IV of Castile. Efforts to develop practical methods for AI were started in Russia in 1899. Papers on

artificial insemination in horses had been published by 1922. By the mid 1940's artificial insemination had become an established industry. In 1949 improved methods of freezing and thawing sperm were developed. The idea for adding antibiotics to the sperm solution came in 1950 from Cornell. Improved methods of sperm collection were developed in the 1970's and 1980's. Research to improve methods of artificial insemination continues till today.

Indications for Artificial Insemination

Indications Related to Males

1. Incurable defects in the husband's semen rendering him incapable of procreation.
2. Premature or retrograde ejaculation.
3. Hereditary disease in the husband as to contraindicate paternity.
4. Rhesus incompatibility resulting in failure of children to survive because of erythroblastosis foetalis and husband being homozygous Rhesus positive.
5. When the husband is impotent making sexual intercourse impossible.
6. Abnormalities of penis such as epispadias or hypospadias etc.
7. When the husband is being treated for cancer with radiotherapy or chemotherapy.

Indications Related to Females

1. The cervical mucus may not be enough to allow sperm to move easily, or it may be too thick

- and sticky. Sometimes, cervical mucus is not compatible with the sperms.
2. Women with mild uterine endometriosis thus preventing the passage of ovum.
 3. Anatomical defects of the uterus or cervix that prevents sperm reaching the ovum.

Types of Artificial Insemination

Artificial Insemination Homologous -A.I.H.

In this type the semen of woman's husband is used. This is usually employed when the husband is oligospermic.

Artificial Insemination Heterologous (Donor)-A.I.D.

The semen from some unrelated donor is used. This is usually undertaken when the husband is impotent, azoospermic, sterile or suffering from some genetic or hereditary abnormality.

Pooled Donor -A.I.H.D.

In this to the husband's semen the semen of the donor is mixed.

Pre-requisites for Artificial Insemination

1. The consent of both the husband and wife as well as donor's wife should be taken.
2. The identity of the donor should not be revealed and the donor should not know the recipient parents' identity and the results of the technique.
3. The donor should be less than 40 years of age, unrelated to the couple and should have his own children.
4. The donor should be of sound physical, mental and emotional health as well as devoid of any hereditary or genetic disease.
5. Race and physical characteristics of donor should resemble as closely as possible to that of the husband.
6. The procedure should be properly explained to the donor and his wife.
7. The husband of the woman should be sterile or suffering from some genetic or hereditary disorder.
8. Pooled semen should be preferably used as in that case there is a technical possibility of that the husband being the father of the child.
9. The physician should select the donor after carrying out the semen analysis, chromosomal studies, testing for HIV status and other sexually transmitted diseases.
10. The physician should maintain relevant records of the donor avoiding the possibility of single fecund donor and consanguineous marriages in future.
11. While carrying out the procedure presence of a nurse as witness is must.

Procedure for Artificial Insemination

Artificial insemination is carried out close to ovulation as the time of maximum fertility coincides with it. About one mL of semen is injected into the top of vagina, cervix or uterus through a small plastic tube. The procedures for the two most common forms of Artificial insemination are:

1. **Intra-cervical Insemination:** The male partner produces a semen sample by means of masturbation. The patient is advised to avoid coitus three days before the sample is taken to obtain good quality sperms. The sample can be collected at the hospital, or should be delivered from home within one to two hours. The semen is injected into the top of the vagina of the female. A plastic cap, to be taken out about 6 hours later, can sometimes be placed in the vagina to keep the sperm near the cervix. Intra-cervical insemination is used for couples in whom sperms cannot reach the cervix.
2. **Intrauterine Insemination (IUI):** Intrauterine insemination has a higher success rate than other AI techniques, because in this sperms are placed near the Fallopian tubes. It may also be combined with hormone treatment to encourage ovulation. Sperm from the male partner or donor is inserted into the uterus using a thin plastic tube that is passed through the cervix. The treatment can be repeated for a further three menstrual cycles, although these

don't have to be consecutive for conception to occur.

Intrauterine insemination is often used when the cervical mucus is incompatible to sperms and many other unexplained infertility problems. Sperm washing prior to IUI removes most of the bacteria from the semen, but it is impossible to completely sterilize the sperm or the cervix. The sperm cells are washed to separate them from the plasma to avoid emergence of uterine infection and endometriosis (in less than 1% of the cases). The sperm are then incubated before being carefully placed in the uterus.

Success Rates with Artificial Insemination

Artificial insemination is successful in only 5%-30% of cases. The success rate is increased when fertility drugs are used before carrying out artificial insemination. Semen analysis is to be carried out to know whether the sperms are suitable for the technique. Total sperm count, their morphology, motility, survival period and presence of anti-sperm antibodies should be assessed. Success rate increases to 70-75% pregnancies within 3-4 months of starting the treatment. For 90% of couples, if treatment is successful, it occurs within the first six cycles of treatment.

Successful treatment depends upon the following factors:

1. The cause of infertility.
2. The morphology and sperm count
3. Conception is more likely to occur when the female has received hormonal treatment for ovulation
4. The condition of the fallopian tube, ovaries, uterus and the regularity of menstrual cycle
5. Age of the woman: The fertility decreases after the age of 30 years.

Ethical Issues Related to Artificial Insemination

Artificial Insemination-scenerio Around the World

Legal issues have arisen in cases where the gestational (and possibly genetic) mother decides

to keep the child. Likewise, there have been debates over the rights of sperm donors. When considering the ethics of sperm donation, several factors must be considered:

1. The rights of the sperm donor.
2. The rights of the clients (who are purchasing the sperm).
3. The criteria by which sperm are collected (i.e. choosing a donor who has certain traits).
4. The amount of sperm that a single man can donate.

These rights were developed through the principles of medical ethics and informed consent and are not mandated technically by law. However, sperm banks are approved by state health departments and national organizations; if certain criteria are not met, the banks are not accredited.

Sperm Donor Rights

The identity of the donor shall remain anonymous. The clients have no right to learn the identity of the donor or solicit donor identifying information from any other source. The donor shall also be free from any responsibility to the biological offspring produced by his sperm.

Client Rights

The clients have the right to be informed of the limitations and potential complications involved with sperm donation. Sperm donation is not always successful and multiple treatments might have to be performed. Also, the sperm bank cannot completely guarantee that the sperm they provide is disease free or free of genetic abnormalities. Although genetic testing and disease screening techniques are advanced and sensitive, they are not foolproof. The client also must understand that she/he is fully responsible for the offspring conceived by use of the specimens.

Sperm donor and client rights are usually established via an informed consent form that is signed by the client and verified by the client's doctor. This form ensures that the client understands his/her rights and the rights of the sperm donor. The principle of informed consent is based on the principles of scientific and medical ethics.

The Ethics of Choosing Sperm

Sperm banks differ in their selection of sperm donors. All are highly selective, but some are more selective than others. For example, California Cryobank only accepts donors who attend or have graduated from a “major four-year university.” Donors must also be tall, trim, heterosexual, and between 19 and 34 years old. One sperm bank, the Repository of Germinal Choice at one time only accepted sperm from Nobel Prize winners. Other sperm banks like CryoGam Colorado, Inc. is selective, but not excessively so. They carry a more “normal” gene pool and their philosophy reflects distaste for elitism. The disparity between ultra selective sperm banks and ones that cater to a more “normal” population raise ethical questions. Is only providing the most elite sperm a form of eugenics? As of yet, these ethical issues have not been resolved. Sperm banks can carry any type of sperm they wish as long as they adhere to proper laboratory standards and respect informed consent.

How Much Sperm Can Be Donated?

If a donor sires too many kids, the risk of producing children who will eventually copulate increases. Although it sounds unbelievable, half brothers and sisters (from the same donor father) have actually married not knowing they were related. Thus, sperm banks place a limitation on the number of children a donor can produce. A donor can produce a maximum of ten children with his sperm. This limit prevents the problem mentioned above and is adhered to by all sperm banks.

Modern methods of assisted conception have raised many legal, ethical and social questions. The legislatures have lagged behind in making laws, both to regulate the medical field as well as to tackle legal issues like inheritance, property rights etc. arising out of these reproductive techniques. As regards regulation of the medical profession, in United Kingdom (UK), members of medical community have themselves established a Voluntary Licensing Agency (VLA) to ensure that the assisted reproductive Technique is prac-

ticed only by persons with adequate qualification and facilities. As regards statutory laws, in UK the Surrogacy Arrangement Act of 1986 is the first legislation to be enacted, which deals with different aspects of surrogate motherhood. Subsequently other laws have been enacted dealing with different aspects of assisted conception viz, Human Fertilisation and Embryology Act (HFEA) 1990 of U.K and its subsequent amendments of 2000 and 2001.

Ethical Issues Related to Artificial Insemination-Indian Scenario

The Indian scenario in this field is quite bleak, the Delhi Artificial Insemination (Human) Act of 1995 being the only statutory act. It is unfortunate that not only the Indian Infertility Specialists have not set up any internal regulatory body, like the Voluntary Licensing Agency of UK, they have opposed steps towards regulation of practice in this field of medicine. The situation in India is grim and pathetic; hardly any protocols or parameters are followed. Many a times neither the consent of the wife and nor of her husband is taken. Sometimes the procedure is carried out with the consent of only one partner. Even the donors are sent to the laboratory on one pretext or the other for semen testing and his semen utilized for the purpose of artificial insemination. Also, the semen is collected of professional donors for the purpose of money. No record is maintained of the donor, recipient and of the procedure followed.

We hope that the Indian scenario will fast catch up with that of UK and USA in ethical and legal practice of management of infertility.

The Delhi Artificial Insemination (Human) Act 1995

This Act is applicable in the National Capital Territory of Delhi. The objectives of this Act are:

1. To allow issueless couple to have a child through artificial insemination and give it a legal status.
2. To control the spread of HIV 1 and 2 through artificial insemination.

3. To regulate donation, sale or supply of human semen/ovum for artificial insemination.
4. To make it obligatory on the part of the medical practitioner to:
 - i. Seek the written consent of the husband and the wife, seeking artificial insemination (AI).
 - ii. Seek the written consent of the donor and the recipient and their spouse, in case of requests of semen/ovum from specified donor/recipient.
 - iii. Test the recipient for HIV 1 and 2 and other sexually transmitted diseases before performing AI.
 - iv. Not segregate the XX/XY chromosomes for AI.
 - v. Not to disclose the identity of the donor/recipient to avoid disputes relating to succession as far as possible, likely to arise due to artificial insemination.
 - vi. The doctor should keep complete record of the bio-data including marks of identification of the donor and the recipient of the semen and ovum.
5. To prohibit carrying on semen bank without registration and yearly renewal that is punishable with fine up to 5,000 rupees and in case of second or subsequent offence, imprisonment for a term up to 3 months or with fine up to 5,000 rupees.
6. To provide for testing of donor before allowing to donate—The semen bank before accepting the semen for artificial insemination shall test the donor for the presence of Human Immuno Deficiency Virus Type 1 and 2 (HIV 1 and 2), antibodies by highly sensitive ELISA kit and screen for Hepatitis B surface antigen and if found negative, only then, the donor shall be allowed to donate.
7. The donated semen shall be stored either by cryo-preservation of liquid nitrogen freezing or any other safe method for a minimum period of three months in order to exclude window period of HIV 1 and 2 infections in the donor. Second ELISA test is performed on the donor after three months, and if negative, the semen shall then

be used. The doctor should seek the written consent of the recipient for using the semen on the basis of only one ELISA test, being negative, where facilities for cryo-preservation and liquid nitrogen for semen are not available.

Legal Aspects of Artificial Insemination in India

There is no statutory law for artificial insemination in India. Artificial insemination with the semen of the husband is justifiable and unobjectionable since the child is actually the biological product of both husband and wife but does not constitute evidence of proper consummation of marriage. The legal aspects in relation to A.I.D as applicable to India are:

1. *Adultery*: The donor and recipient cannot be held guilty of adultery in India as section 497 I.P.C. requires sexual intercourse necessary for adultery.
2. *Legitimacy*: The husband is not the actual father of the child thus the child is illegitimate and cannot inherit property.
3. *Nullity of marriage and divorce*: Mere artificial insemination is not a ground for nullity of marriage/divorce but if A.I. is due to impotence of the husband then it is a ground. If the procedure of artificial insemination has been carried without the husband's consent, he can sue his wife for divorce and doctor for damages.
4. *Natural birth*: The child will remain the product of natural birth if the parents do not disclose. The status of the child born by artificial means will be illegitimate unless adopted.
5. *Unmarried woman/widow/lesbian couple*: The status of such a child born by artificial insemination would be illegitimate.
6. *Incest*: Risk of incest exists between children born by artificial insemination and child of donor but the offence is not punishable in India.

Semen Banking

Semen banking is the preservation of human semen by adding glycerol and after slow cooling and rapid freezing it is stored below a temperature

of -79°C .

When the semen used was that of the husband after his death-Posthumous Artificial Insemination due to availability of semen banking, it may give rise to problems in connection with the inheritance rights of such a child born, which did not start its life when the genetic father was alive. Amendment to HFEA affected in 2000 has allowed posthumous artificial insemination using the semen of the deceased husband.

SURROGATE BIRTH

Surrogate birth means hiring of another woman who is the surrogate and is artificially inseminated with the barren woman's husband's sperms. The process is repeated till the surrogate conceives. When the child is born, he is handed over to the biological father and his wife. Usually it's the surrogate who is biological as well as gestational mother of the child but when the wife's ovum is fertilized with the husband's semen in the laboratory and then implanted in the surrogate, she remains only the gestational mother.

Full Surrogacy

It is a situation where the embryo is provided by the commissioning couple.

Partial Surrogacy

The carrying woman has her own egg fertilized. Reasons for surrogate birth/motherhood:

1. Inability of the couple to produce the child as the wife is unable to conceive or carry the child to term.
2. There are some eugenic reasons, genetic defects or inherited diseases.
3. Wife does not wish to take time to carry the foetus through.
4. Wife may suffer from anxiety or labour phobia.
5. The couple wish to adopt the child but since the facilities are scarce and it takes long for adoption.
6. Sometimes the adopted child is in emotional conflict when he discovers that they are not his biological parents.

Medicolegal Aspects of Surrogate Birth

The surrogate has to give custody of the child to the couple, as they remain his legal parents. The wife has to adopt the child. For the entire period of pregnancy the surrogate is to be given expenses for diet and medicines etc. and the surrogate should quit smoking, drinks and drugs for this entire period. In some countries like USA, there is a commercial transaction that includes fees for the surrogate mother and to those who manage the deal. Also adoption of the child is to be done when there is risk of some deformity to the child or when the couple separates or even when the wife conceives after entering in to contract.

1. The couple may refuse to accept the child as the surrogate is biological mother.
2. If the surrogate refuses to hand over the child, she can keep it since she is the biological mother of the child.
3. If both parties refuse to keep the child owing to its deformity, the child is given for adoption.
4. Issue of paternity of the child, it is for the genetic father who engaged the surrogate to prove the paternity.
5. The question of who is better suited to take the custody of the child arises between the surrogate and the father.
6. There is the risk of commercialization and trading in this procedure and even the quality of children may deteriorate.
7. There is always the risk of psychological trauma to the child when he discovers of the surrogate mother.
8. The surrogate may be emotionally disturbed on parting with the child.
9. If surrogate agrees without the consent of her husband, she may be charged with adultery and if forced to abort by her husband, she will be liable to pay damages. Even if the father agrees into contract without the wife's consent, she can refuse to accept the child. If the surrogate dies during childbirth or suffers serious or permanent injury or disability would the couple be liable for damages or would her heirs be entitled to claim damages for compensation.

IN-VITRO FERTILIZATION

Historical Perspectives

- World's first test tube baby was born in England on July 24, 1978.
- World's second test tube baby was born at Calcutta.
- The first test tube baby clinic started operating in Norfolk Virginia since Feb 1980.

In IVF, the eggs are surgically removed from the women, fertilized with available sperms in a dish and embryo replaced into the womb of the woman who completes the carriage till delivery. Semen samples of husband are screened two or three times for Hepatitis B surface antigen and HIV II and I.

Process of In-vitro Fertilization

Hormone like gonadotropin is injected into ovary, which induces super ovulation. Approximately 30 hours later, the oocytes are removed from the ovary by laproscope (a surgical procedure in which two thin glass tubes are injected into the ovary through a small incision in the abdomen. The removed oocytes are placed in a petridish or test tube containing a growth medium simulating the environment of the woman's body. The medium is composed of the woman's fluids from her reproductive tract. A sperm held in vitro then fertilizes the oocytes. The resulting conceptus is kept in an environment at room temperature where it divides and grows for a few days until it reaches the blastocyst stage. The conceptus is then picked up with a small hollow tube injected through the vagina and cervix in to the uterus where it is implanted at a proper time in the menstrual cycle. After successful transplantation the woman carries the blastocyst to term. The IVF may be performed in many contexts in which the presence/absence of the following four factors will emerge:

1. A genetic link between parents and child.
2. A gestational link between mother and child.
3. A marital relationship.

4. Necessity of the process to fulfill an individual procedure potential.

Possible hypothetical situations for in vitro fertilization

Sperm	Ovum	Uterus (Blastocyst implantation)
Husband	Wife is donor	Wife
Donor	Wife is donor	Wife
Husband	Third party is donor	Wife
Donor	Wife is donor	Wife
Husband	Wife	Surrogate
Husband	Donor woman	Donor woman

Case I: In this the process may be necessary because ovum donor may have a tubal occlusion which prevents her to conceive and in some cases voluntarily couple may desire to conceive through IVF to permit early screening of genetic defects, control the timing of pregnancy and select a blastocyst with certain characteristics. In this case the ovum of the wife is fertilized in vitro by sperm of the husband and then is transferred to wife's uterus.

Case II: In this case, the husband is sterile or he suffers from some genetic disorder that he does not want to transmit to the child or he is sterile. In this case the sperm of the donor are used for fertilizing the wife's ovum in vitro and then implanted in the uterus of the wife.

Case IV: In this case the wife is either sterile or has some genetic defect that she does not want to transmit to the child. The sperm of the husband fertilize the donor's ovum in vitro and then transferred to the wife's uterus.

Case IV: The couple employs this method when both the partners are sterile. The ovum from the donor is fertilized by the sperms of the donor in vitro and then transferred in the uterus of the wife.

Case V: This is employed when the wife is unable to carry the child to term then the uterus of the surrogate is used for the purpose.

Case VI: This might be used by a woman who wishes to have a child but cannot conceive naturally or through artificial insemination or does not wish to conceive by this. Then the husband's

sperms fertilize donor woman's ovum in vitro and then it is transferred in the uterus of the donor woman.

Various Techniques for IVF

1. *Gamete intrafallopian transfer* (GIFT): The egg and sperm are put together in the fallopian tube for fertilization
2. *Embryo transfer* (ET): The embryo that is created from the egg and sperm is transferred in to the carrier's womb, and the carrier may or may not be the originator of the egg.
3. *Zona drilling* (ZD): In this the prepared semen is introduced through a partial opening of zona pellucida.
4. *Partial zona pellucida* (PZD): This method is employed if due to any reason its not possible to penetrate zona pellucida. It is of two types:
 - *Sub zonal injection* (SUZI): The sperm is injected between the egg and the surrounding membranes with a sharp micropipette.
 - *Intracytoplasmic sperm injection* (ICSI): The sperm is directly injected into inner cellular structure of the egg.

ETHICAL AND LEGAL ASPECTS IN ASSISTED REPRODUCTION

The ethical and legal aspects involved with in vitro fertilization and allied procedure are many and most of them remain unresolved. The reason for this uncertainty is the rapid advancement of the technology thereby resulting in the lag in legal and ethical response. There are a number of ethical and religious issues regarding IVF of which some are unanswered and others remain the topic for debate.

1. Issues relating to experimentation on human beings as this is important to define when life begins in IVF.
2. The moral issues relating to non-coital reproduction.
3. What are the rights of inheritance in case of ovum or sperm donation?
4. Whether there should be any age limit?

5. Does surrogacy resemble baby selling as the ability to bear the child is being used commercially.
6. What should be the fate of orphan embryos and what are the legal rights of embryos?
7. The issues relating to embryo research and that should genetic engineering be totally banned.

Besides these others areas that have generated public enquiries and legal debate are many such as what is the fate of excess and stored embryos. Is selective breeding for male child being employed? Are there any practices where the use of drugs to trigger false positive pregnancy test are undertaken and the falsification of test reports indicating pregnancy. In relation to donation of genetic material it is important whether the secrecy is maintained or not and the donors are properly screened or not. The financial gains related to the procedure and regularization of sperm banks is important issues. The legitimacy of motherhood in relation to surrogacy is an important issue. There are chances of exploitation of surrogate mother ant the effects of this practice on the offspring.

Legislation in Relation to Assisted Reproduction

With more and more achievements in the filed of assisted reproduction technology more and more complexities and controversies regarding ethical and legal matters crop up. Many enquiry committees including Warnock committee in England were established. All of them suggested continuation of assisted technology with proper control. In most of the developed countries like USA, UK, Australia and Singapore there is strict control of regulatory bodies. Recommendations of these committees are amended from time to time and binding on all centers.

Warnock committee 1982: Political involvement started in U.K. and a commission of inquiry was appointed in 1982 with Mary Warnock as its chairman that gave the following recommendations

- a. A statutory licensing authority should be formed to regulate infertility services and research.
- b. Practicing in infertility services and research should be especially licensed.
- c. AID, IVF and egg and embryo donation is to be regulated by law.
- d. Cryopreservation was permitted.
- e. Research on spare embryos was permitted 14 days after fertilization.
- f. Gamete donors and recipients should be anonymous, limited to ten children and written consent was necessary from all parties.
- g. AID children legitimately belonged to the recipients if both have consented. Egg donation should also be treated in the similar lines.
- h. Frozen embryo and semen should be reviewed every five years and discarded after ten years.
- i. Surrogate recruiting agencies whether profit making or not were outlawed.
- j. Disobeying these recommendations would be grievous offence.

All these recommendations were accepted except research on human embryos.

Human fertilization and embryology Act (HEFA) 1990: This Act was enforced in England. It is related to various research and clinical procedures of assisted reproduction. HEFA has a corporate authority, which can issue licenses for treatment, storage and research. Other European countries also enacted code of ethics within their own clause. They formulated more or less similar regulation based on Warnock's report keeping in mind local social and political pressure.

Norwegian laws on assisted reproduction 1988:

- a. Activities of assisted reproduction can only be carried out in government approved centers.
- b. Facilities can only be offered to married couples.
- c. Incorporates secrecy to semen donors.

- d. Semen Cryopreservation is permitted.
- e. Oocyte Cryopreservation cannot be permitted
- f. Cryopreserved embryo cannot be stored for more than 12 months.
- g. Embryos cannot be donated.
- h. Research on human embryo is forbidden.
- i. Disobedience is punishable with fines or imprisonment for up to three months.

German Embryo Protection Act 1990: Misuse of reproductive technologies may result in imprisonment for three years and fine. The misuse clauses include:

1. Placing foreign or unfertilized egg into a woman.
2. Fertilizing an egg in vitro for other purpose than establishing pregnancy.
3. Transferring an embryo to a woman if she intends to surrender the child to some one
4. Replacing more than three embryos per cycle.
5. Fertilizing more egg than can be replaced in one cycle.
6. Purchase or acquiring a human embryo.
7. Growing an embryo for any other purpose than establishing a pregnancy.
8. Choosing X or Y sperms for IVF for gender selection.
9. Using spermatozoa from a diseased man for IVF.
10. Genetic manipulation on germ cells or cloning.
11. Cross species fertilization with human genome.

Assisted Reproduction in United States

American college of Obstetricians, gynaecologists, and American fertility society similar to British voluntary licensing authority formed a national advisory board on the ethics of reproduction. American society for reproductive medicine review publishes updated guidelines in the form of minimum standards for ART programme and guidelines for human embryology andrology laboratories. The latest revised minimum standards for IVF, GIFT and related procedures were

released in October 1998, the salient features are:

1. *Personnel*: Each programme should have a designated overall practice director, a medical director and laboratory director. One individual may fulfill more than one position. Each person involved in IVF should have performed a minimum of 20 procedures and a training of at least 6 months. And completed 60 assisted reproduction technology procedure.
2. *Embryology laboratory*: The lab should have quality assurance by mouse IVF, one cell or two cell mouse embryo in vitro or human sperm survival test. The lab facilities with periodic maintenance of records should be there. Facilities for embryo Cryopreservation and safety aspects according to strict guidelines issued and amended from time to time.
3. *Ethical experimental procedures*: American society for reproductive medicine's (ASRM) ethics committee has issued a report an ethical consideration of ART procedures and all centers are expected to practice in strict accordance of guidelines issued and amended from time to time.
4. *Record keeping*: All ART programme should confirm registry and federal trade commission guidelines relating to advertising and use of statistics.
5. *Informed consent*: All ART programme should confirm to the ASRM/SART guidelines concerning informed consent.

Rules of Good Clinical and Biological Practice in MAP (Medically Assisted Procreation)

These are the recent guidelines providing legal framework of MAP, the salient features are:

1. MAP to be carried out only for proven and standard indications, not for personal convenience.
2. Treatment to be provided only to a couple of reproductive age. They should be married or able to provide evidence that they have lived together for at least two years. Both members should be alive at the time of medically assisted procreation.

3. Gamete donation may only be practiced in compliance with the principle of non-remuneration.

4. Law prohibits any prior agreement between recipient couple and a designated donor.

FIGO recommendations on gamete and embryo research:

- Research is acceptable only for the purpose of human benefits in pre-embryonic stage only by an authorized body with prior permission.
- Pre-embryo should not be transferred to the uterus unless normality is guaranteed.
- It should not be subjected to commercial profit.
- Cloning beyond pre-embryo is banned.
- Interspecies fertilization is unethical.
- Implantation in other species is banned.
- Manipulation for the purpose other than treatment is unethical.

Indian Scenario

In India, the ethical fraternity has achieved a lot in the field of assisted reproduction but no licensing or regulatory authority exists yet. Ethics in the field of assisted reproduction techniques in India are self regulated and usually based on the guidelines of ASRM or other western bodies. In India IVF-ET costs around Rs 30,000 to Rs 1,50,000 per sitting varying according to center and city. Multiple trials multiply the cost with only 10-20% success. Assisted reproduction techniques are associated with risks such as abortion, multiple pregnancies, superovulation premature birth etc. there is no law in India that governs or controls insemination, donation of ovum or uterus etc. except in Delhi where such a bill is passed. In India, there is a need of some regulation or Act so that the public is protected against poor laboratory techniques, exaggerated claims of success, unethical practices, exploitation for monetary gains and selective termination of pregnancies.

CLONING

Cloning is the technique of producing a genetically identical duplicate of an organism

artificially.

Clone is an organism that has same genetic information as another organism or organisms. A clone is said to be all descendents derived asexually from a single individual, as by cutting, bulbs, by fissions by mitosis, or by parthenogenesis reproduction.

With the birth of cloned sheep, mice, pet and cloned cows soon to be borne in Japan, the art of carbon copying adult mammals is developing rapidly, but according to scientists, the prospect of humans is remote. Researchers predict that they will be able to rechannel the embryo's normal development to grow a new heart or any other organ of the body. But such technology would raise questions. Does embryo clone have rights? If so, does the medical benefit to the donor outweigh the right of the embryo to fulfill its human potential?

Introduction of Cloning

Nuclear transfer—This is transferring of nucleus from the cell of one organism to an enucleated egg cell. This will produce an organism that has the exact genetic material as that of the donor cell. Scientists are using current techniques exceedingly more and with variety of species.

In nature and even in the lives of humans, clones are present. Cloning occurs with all plants, some insects, algae, unicellular organisms that conducts mitosis or binary fissions, and occasionally by all multicellular organisms, including humans.

Monozygotic twins (identical twins) are clones of each other. They have exactly the same genetic information due to the division of an embryo early in development that produces two identical embryos. About eight million human clones inhabit the world. In unicellular organisms, a cell will produce two daughter cells that have the same genetic material.

Today, the only cloning research is occurring in scientific model organisms. The research scientists from around the globe have collected copious amounts of data to continue advancements in research more efficiently. The most

common scientific models are *E.coli*, mice, fruit flies and frogs.

The first organisms that were cloned using nuclear transfer were Frogs because they have large egg cells and scientists can obtain up to 2,000 of them from one ovulation.

Successful cloning has occurred with livestock. The drive towards success is not because livestock like Cows and Sheep are model organisms but farming industry has made and continues to make a big effort towards finding a way to implement the technique of nuclear transfer to livestock. Research in cloning is also occurring in Primates, due to their similarities with humans. This leads to the most talked about aspects of cloning, the use of the techniques with human cells eggs.

Dr. Seed, physicist who researched fertility sciences in 1980's and now specializing in embryology, states that he has set up a fertility clinic that can conduct nuclear transfer and thus accomplish the task of human cloning. He successfully transferred human embryo from one human to another in the 1970s.

Genetic screening is occurring in Britain. Fertility clinics aim this towards couples where mother/father has a genetic disorder. A fertility clinic will clone an embryo and test it for genetic disorder. If it is negative then the fertility clinic will implant a clone of that embryo, thus child will not have any genetic disorders. Dr. Seed is backed by private funding since US law has banned any federal funding for research in the area of human cloning.

History of Cloning

1. **Adolph Edward Driesch** could let the eggs of a sea Urchin develop in to the two blastomere stage, then he separated it by shaking it in a flask and allowing them to grow cells developed into dwarf sea urchins. Driesch could not explain his experiments and gave up embryology for philosophy.
2. The first implantation of a nucleus in to an egg cell occurred in 1952 by **Robert Briggs** and

- Thomas J King** in Philadelphia. They had transferred the nuclei of Leopard Frog's eggs; the egg cells did not develop.
3. **Dr John Gurdin** accomplished the successful nuclear transfer of embryo cells later in 1970's but frogs did not develop beyond tadpoles.
 4. In 1984, **Dr Steene Willadsen** announced that he had successfully transferred nuclei from embryo of sheep to produce clones. He was successful with Cows and even Monkeys. He began cloning embryos that were in 64-128 stages suggesting that perhaps nuclear transfer was possible with differentiated cells.
 5. In 1994, **Dr Neal** first produced Cows by nuclear transfer from more developed embryos. He produced four calves.
 6. In 1997, **Dr. Lan Wilmut** and **Dr Keith Campbell** of the Roslin Institute in Edinburgh, Scotland produced for the world Megan and Morag, the first cloned Sheep from embryo cells. Their new technique was starving the donor embryo of the nutrient serum they feed the cells. This would put the cell in the right moment in the cell cycle, thus allowing the genetic material to be accepted more easily by the egg cell. This was the integral step of nuclear transfer.

Dolly—First clone sheep

Dr Wilmut and Dr Campbell of the Roslin Institute in Edinburgh, Scotland produced the first cloned Sheep from embryo cells on Feb 22, 1997.

These scientists took a mammary cell from a Sheep and put it in to an egg. They let this egg to grow in to an embryo and then transplanted this fused embryo and put it in a recipient Ewe, acting as a surrogate mother. This occurred late in Jan 1996. This was the day of fusion date for Dolly. She weighed in at 14⁺ pounds and was healthy. The scientist who was given the credit was embryologist Dr Wilmut.

Methods used for Cloning

1. The method used is called nuclear transfer since nuclear information is transferred.

2. First step is to find something to clone; Dr Wilmut and Campbell grabbed some cells, which were growing in the lab from mammary cells of a six year old Finn Dorset Ewe.
3. Next step is to get an oocyte and take the DNA out so that the donor DNA can be added.
4. Donor cell and the oocyte without a nucleus are put together in a petridish and electric current is sent through it to fuse as a zygote.
5. This cell then grows and divides like a fertilized egg cell and then it becomes a blastocyst.
6. For each clone Roslin researchers combine material from the two sources—First, they extract immature oocytes from the ovaries of Ewes; Then remove the DNA from it to make enucleated egg cells and then take cells containing donor genetic material.

Cloning in Dolly

Dolly's DNA came from a Finn Dorset sheep, she was developed from an oocyte donated from a Poll Dorset and her surrogate mother is a Scottish Blackface Ewe. The variation in breeds was used to show that Dolly is not of the same breed as her surrogate mother or the egg cell she developed from Dr Wilmut and his colleagues fuse the empty oocyte with the donor cells by bringing them together and subjecting them to an electric current to fuse as one cell. After growing and dividing for a week or so in lab culture dish, the fused cell forms an early embryo called a blastocyst, which Wilmut's team implants in to a surrogate mother, if all goes to plan the surrogate mother gives birth five months later. Dolly was the only lamb born from 217 fusions of oocytes with udder cells.

Dr Wilmut says there were so many failures because it is difficult to ensure that the empty oocytes and donor cells are at the same stage of the cell division cycle.

Aims and Objectives of Cloning

1. *Producing animals of high genetic value:* Making copies of diseased to better the engineering of offspring in humans and animals.

2. Cloning could also directly offer means of curing diseases. As a technique that could extend means to acquiring a new data for the embryology and how organisms develop as a whole over time.
3. The agricultural industry demands nuclear transfer to produce better livestock with ideal characteristics for the agricultural industry and to be able to manufacture biological products such as proteins for humans since many human diseases are caused by defects in the production of proteins.
4. Cloning endangered species to increase their population.
5. *Xenotransplantation*: Cloning of Pigs that will produce organs which will not be rejected by humans after a transplant.
6. Livestock can produce biological proteins helping people having diseases like Parkinson's, Diabetes, Cystic fibrosis.
7. Cloning provides better research capabilities for finding cures to many diseases.
8. Provide benefit to those who would like to have children like in infertility, with genetic disorders could produce child.
9. Single women could have child using cloning instead of IVF/artificial insemination.
10. Also provide children who need organ transplants to have a clone born to donate organs.
11. Provide a copy of a child for a couple whose child had died.

Ethical Issues of the use of Cloning

1. Decline in genetic diversity, depriving a person of his uniqueness.
 2. If a population of organisms has the same genetic information then the disease would wipe out entire population.
 3. If we try to clone endangered species, we could possibly kill the last females integral to survival of species e.g. it took 227 tries to produce Dolly.
 4. If everyone has the same genetic material, ability to clone will be lost and we have to resort to natural reproduction, causing it to inbreed, which may pose problems.
 5. Taking nature in to our own hands by cloning animals and humans.
 6. Religious organizations consider nuclear transfer to cause men to be reproductively obsolete since cloning requires only oocyte, any cell and a woman to develop in.
 7. Rights to human beings will be defied since clones are not granted the birth of newness.
 8. Mental and emotional problems of a clone.
 9. Cloning would enable rich or powerful persons to clone themselves several times over and commercial entrepreneurs might hire women to bear clones of sports or entertainment celebrities.
 10. The current reproductive techniques can be abused.
- When people can have other methods of reproduction to have healthy children, why should they resort to cloning? The various applications for cloning can be:
1. Infertile couples that cannot conceive due to some genetic disorder can only seek cloning.
 2. Couples who are infertile as a result of gametic insufficiency might choose to clone one of the partners rather than the sperm, egg or embryo from some unknown donor.
 3. Couples, who are at the risk of having a child with some genetic disorder, may seek to clone one of them or some of their already present child.
 4. Obtaining tissues or organs for transplantation in case of a child lacking a suitable donor.
 5. Therapeutic cloning aims to develop medical therapies for which the cloned embryos grown only up to fourteen days can harvest the stem cells that will be useful in treating certain diseases.
- The potential uses of cloning are controversial because of the explicit copying of the genome. Somatic cell cloning raises issues of the individual, autonomy, objectification and kinship of the

resulting children. In other instances, such as the production of embryos to serve as tissue banks, the ethical issue is the sacrifice of embryos created solely for that purpose. Former US President Bill Clinton imposed a ban on human cloning and constituted a panel to study in details and give advisory guidelines. Recently, 20 European Countries have signed the bioethics pact in the first international convention to control research into human genetic engineering and cloning. It prohibits the trade of human bodies or parts and regulates organ transplants. The agreement is based on the general principle that the interest and rights of individual human must prevail over the interest of the science. Many countries including the U.S and the U.K have stopped funding of research activities related to human cloning. In the United Kingdom, government has endorsed the recommendations of chief medical officers' expert group on therapeutic cloning which include:

1. Approval should be given for the use of early embryos to investigate the potential of new medical treatments.
2. The Human fertilization and Human Embryology Authority should scrutinize all research proposal.
3. Permission should be granted only when it can be shown that there are no ways of meeting an experiment's objectives.
4. Embryos should not be retained in the growth cycle for more than 14 days.
5. No cloning technology should allow cell

materials of humans to be mixed with other animals.

6. Individuals whose eggs or sperms are used to create the embryos to be used in stem cell research should give specific consent.
7. Cloning for the purpose of creating a baby (reproductive cloning) should remain illegal.

Medicolegal Aspects of Cloning

1. *Identification*: Problems would arise in identification of the person.
2. *Paternity and maternity testing*: In matter involving paternity and maternity, it would be difficult who is the biological father or social father or genetic father.
3. *Property and inheritance*: In matters related to inheritance of property, it would cause great problems.
4. *Religious problems*: There is no sanction for religion.
5. *Ethical problem*: Believe only the natural method of reproduction, it is like playing with nature and taking nature into their own hands and men should not play God in a natural set up.

Short Questions

1. Artificial insemination.
2. Surrogate motherhood.
3. Cloning.
4. IVF.

MULTIPLE CHOICE QUESTIONS

1. **Legal problems of adultery in artificial insemination are dealt under:**
A. Sec. 497 IPC

- B. Sec. 420 IPC
- C. Sec. 32 IPC
- D. Sec. 175 IPC

Medicolegal Aspects of AIDS

Acquired immune deficiency syndrome is causing both health and legal problems. Much of the controversy about AIDS and its victims involve the legal rights of the public whose health should be protected; the risks and methods of transmission of AIDS; the right of those with the disease to receive full medical attention and to receive public services like schooling and the right of the victims to their privacy

This is further complicated by the need of the AIDS victim for protection from communicable diseases that are life threatening to the victim but not to the general public. Health personnel are concerned because of greater chance of contact with patients with the disease.

According to estimates from the Joint United Nations Programme on HIV/AIDS (UNAIDS) 37 million adults and 2.5 million children were living with HIV at the end of 2003. This is more than 50 percent higher than the figures projected by WHO in 1991 on the basis of the data then available. A UNDP sponsored study in 1995 found that a one percent increase in HIV/AIDS prevalence rates leads to 2.2 years lost in human development as measured by the Human Development Index. The worst affected are the countries of Sub-Saharan Africa and South and South East Asia, which are among nations with lowest human development indices, large pools of poverty, huge gender inequity, powerlessness, social instability etc. Out of the 40 million people living with HIV/AIDS in the world, more than 25 million are in Sub Saharan Africa followed by 9 million are in Asia. During

the period 2003, the total 5.3 million people were affected in India, which increased to 5.7 million during the period 2005 (Study 2005). During 2003, some 5 million people became infected with the Human Immunodeficiency Virus. The year also saw 3 million deaths from HIV/AIDS- a high number despite antiretroviral therapy which staved off AIDS and AIDS deaths in the richer countries

In 2003, an estimated 700,000 children aged 15 or younger became infected with HIV. Over 90 percent were babies born to HIV-positive women, who acquired the virus at birth or through their mother's breast milk. Of these, almost nine-tenths were in sub-Saharan Africa. Africa's lead in mother-to-child transmission of HIV was firmer than ever despite new evidence that HIV ultimately impairs women's fertility: once infected, a woman can be expected to bear 20 percent fewer children than she otherwise would

AIDS is a viral disease involving the breakdown of the body's immune system. The full blown syndrome is believed to be both incurable and almost inevitably fatal, striking most frequently among certain high-risk groups, such as male homosexuals, intravenous drug users, recipients of blood transfusions and hemophiliacs.

AIDS represents a form of secondary immunological disorder that results in profound depression of cell mediated immunity. It is caused by a reterovirus known as Human T-lymphotropic virus type III/ Lymphadenopathy-associated virus or HTLV III/ LAV. The virus inhibits body's ability to resist diseases by infecting white blood cells

called T-lymphocytes, which are an integral part of the human immune system. Specifically the disease selectively destroys and generates qualitative abnormalities in the patient's T-helper/inducer cells, which enable other components of the immune system to function and thus comprises T-lymphocyte subset primarily responsible for the generation of most specific human immune responses. As a result, the immune system of patients with AIDS is characterized by functional defects in virtually all limbs of the immune system. This suppression of the immune system exposes patients to a variety of 'opportunistic' infections and malignant conditions that generally do not afflict otherwise similarly situated individuals without AIDS. It also possibly infects B-cells and causes macrophage dysfunction and thus an acquired immune deficiency state results.

Included under 'diseases at least moderately predictive of cellular immune deficiency' are two neoplasia-Kaposi's sarcoma and primary lymphoma of the brain, and a wide spectrum of opportunistic infections such as:

1. Protozoal and Helminthic infections caused by *Pneumocystis carinii*, Toxoplasmosis, cryptosporidiosis and strongyloidosis
2. *Bacterial infections*: Atypical mycobacteriosis
3. *Viral infections*: Cytomegalovirus; herpes virus 1 or 2; and progressive multifocal leukoencephalopathy by papovavirus

The signs and symptoms in the patient may be non-specific and are often due to infections or neoplastic complications rather than to the disease itself. And there are no pathological lesions diagnostic of AIDS. The patients may present as:

1. Unexplained loss of weight.
2. Low grade fever.
3. Unexplained generalized lymphadenopathy.
4. Diarrhoea.

WHO Definition

The patient should present with at least one major and two minor signs to be diagnosed as a case of AIDS.

Major Signs

1. Weight loss >10% of body weight.
2. Chronic diarrhoea for more than one month.
3. Prolonged fever for more than one month.

Minor signs

1. Cough for more than one month.
2. General pruritic dermatitis.
3. Recurrent Herpes zoster infections.
4. Oropharyngeal candidiasis.
5. Chronic progressive disseminated H. simplex.

Pathogenesis

The incubation period for AIDS is from 4 months to 57 months. HIV attaches itself to the T₄ receptor molecule on T-helper lymphocytes in order to infect them. These T-helper lymphocytes are found in most fluids such as breast milk, semen, tears, and saliva. HTLV-III/LAV has been isolated in a wide range of body fluids including blood, semen, saliva, tears, breast milk and urine. How much exposure is needed for the syndrome to develop is unknown. Random exposure is not sufficient and in the absence of contamination with blood products by direct passage and/or sexual contact AIDS is unlikely. Persons are not considered to have AIDS merely because tests show them to be generating antibodies to the AIDS virus that is to be 'seropositive'. Indeed, a person is not considered to have AIDS even if he or she is seropositive, displays a number of elements in the definition of AIDS used for reporting purposes by the Centers for Disease Control (CDC), and is afflicted with one or more of the opportunistic diseases that take advantage of the patient's immune system.

The high risk groups in the community for acquiring AIDS are: (i) Recipients of blood transfusion (ii) Homosexuals (iii) Prostitutes and Eunuchs (iv) Drug addicts and drug abusers (v) Groups with sexual interaction with foreign travelers.

Routes of Transmission

AIDS may be transmitted in any of the following ways:

1. By way of unprotected (vaginal, anal or oral) sexual intercourse both homosexual or heterosexual.
2. By exchange of contaminated blood and blood products by way of sharing needles by drug abusers and through blood transfusions.
3. needle type exposure resulting from cuts by knife while carrying out surgeries in operation theatres and in the autopsy rooms.
4. Perinatal transmission—From HIV positive mother to her unborn child.

Precautions to be taken in Health care settings by the Health Care Personnel

1. Medical officer while examining a homosexual male especially one with history or the external stigmata of drug abuse, should take precautions to avoid contamination himself with blood or seminal fluid especially from infected syringes and needles.
2. In invasive procedures such as surgery, dental procedures in which bleeding might occur and vaginal or caesarean section deliveries gloves, masks, eye coverings and other barrier precautions should be routinely used.
3. Extraordinary care should be taken to prevent injuries to hands caused by needles, scalpels and other sharp instruments or devices during procedures; when cleaning used instruments; during disposal of used needles and when handling sharp instruments following procedures.

Precautions to be taken while Performing Autopsies

High risk postmortems are those wherein lies the danger of infection to the mortuary staff and doctor performing autopsies. Precautions to be taken for autopsy in cases of deaths due to AIDS include:

1. Double gloves, facemasks and goggles must be used by the doctor and the assistants.

2. A dead body should be well marked with a label as to 'HIV positive' before transfer from hospital to mortuary.
3. Bleaching powder should be used to cover areas wherever body fluids are spilled and instruments should be heat sterilized and then put in a solution of bleaching powder.
4. Re-capping of needles should be avoided.
5. All open wounds should be properly covered.
6. Body should be properly carried and cremated.

Medicolegal Problems Surrounding AIDS

Apart from the diagnostic and treatment problems, AIDS has thrown up many legal and social issues. It should be kept in mind that, AIDS patients, like all other patients are entitled to equality, confidentiality, right to information etc. At the same time the doctor has the social duty of making a privileged communication in the larger interest of the society. Recently Supreme Court of India has ruled that the sexual partner of an AIDS/HIV positive patient is to be informed of the fact. The treating doctor also has the duty to inform the paramedical staff involved in the treatment of such patients, the mortuary staff, pathologist and the staff of the crematorium so that due precautions can be taken by these people who are likely to come in direct contact with the infected biological material. The hospital administrators as well as the nursing officers' in-charge of safe custody of medical documents should ensure that HIV positivity is not revealed to unauthorized persons.

Admission to Hospital

Although people suffering from AIDS need medical care to prolog their lives, South African law does not guarantee such care. When there is no emergency, the Hospital Superintendent may decide on admission of patients taking various factors such as urgency and availability of beds into account. His unreasonable denial to admission may lead to the decision being contested on administrative law grounds such prejudice or gross irregularity. Also if the patient who is turned away dies and is proved in Court of Law that his life

would have been saved or prolonged by timely admission, a civil action for damages may be instituted against the hospital.

Medical Practitioners and their Freedom to Treat

A private medical practitioner is free to decide who he wants to accept as a patient. The relationship between patient and doctor is the result of an agreement which often comes about on an ad hoc and tacit basis. There are a number of exceptions to the patient's freedom of choice:

1. A doctor has to treat a patient in an emergency or when no treatment facility is available. If he refuses arbitrarily and reasonably to attend a seriously ill or injured person, he may be held liable if the patient cannot get another doctor to attend and suffers harm whether the doctor's failure was unreasonable, will be decided by taking various factors into account such as the seriousness of patient's condition; the professional ability of the doctor to do what is asked by him; the physical state of the doctor himself and availability of other medical care. If other medical care is available, it would not be unreasonable for the doctor to refuse treatment if he has open wounds and necessary with which to protect him and blood to blood contact is virtually certain. Danger to oneself may become the limit of responsibility to help others; and doctors cannot be expected to treat patients with a lethal transmissible disease, if they are virtually certain to contract the disease themselves.
2. Where the likelihood of infection is minimal, as in case of normal health care setting and where necessary precautions can be taken, it would be unreasonable for a doctor to refuse emergency treatment to an AIDS patient.
3. The doctor may not commence treatment and then stop when he finds the patient to be HIV positive. He can escape liability if he can show that the patient made it impossible for him to continue the treatment or if he transferred the patient, with the latter's consent, to another doctor.
4. A doctor may be under a statutory duty to treat. However, the regulations issued in terms of the Health Act only grant the power, but not impose duty on medical officers to examine patients in order to prevent the spread of AIDS. The medical officer will be obliged to exercise his discretion.
5. The doctors in government employment are obliged to treat all patients who consult them or referred to them in the course of their duties. Failure to treat could lead to disciplinary action and liability for damages vicariously for the hospital or personally for the doctor.
6. The ethical and professional guidelines of the South African Medical and Dental Council State emphatically that the ethical approach to the management of other infections and life threatening condition. The Council states that although the South African medical practitioner or dentist is under no legal obligation to accept a particular patient in his private practice (other than circumstances mentioned above) and may terminate the relationship between them if his diagnostic ability is compromised by the patient's refusal to undergo a simple investigation; such as a blood test, he should show compassion and make every effort to avoid such an impasse.
7. Article I of the Spanish Law of Spanish Law of special measures in matters of public health and to prevent its loss or deterioration, the health authorities or the various public administrators may within the scope of their powers, adopt the measures laid down in this law whenever medical reasons of emergency or need may so require. These measures cover a wide range of possibilities; examination, treatment, hospitalization, control. In the presence of reasonable evidence that the health of the population might be endangered (Article 2 of the aforementioned law). Therefore, the compulsory medical treatment of AIDS is only feasible in those situations in which reasons of emergency call for it; that are when the danger to public health runs its normal course and may be

checked or controlled by normal measures by the public administration, the above mentioned law may not be invoked.

8. In India it is considered unethical for a medical practitioner to deny treatment, investigation or operation to a person infected with HIV. An enquiry can be made by the State Medical Council or by the Secretary Medical Council of India and the practitioner may be held guilty of professional misconduct on this ground.

Informed Consent

Any medical treatment (including the taking of a blood sample) constitutes an invasion of the patient's bodily integrity, for which consent is essential, if intervention may constitute assault.

The United Nations HIV/AIDS and Human Rights International Guidelines express the requirement that apart from surveillance testing and other unlinked testing done for epidemiological purposes, public health legislation should ensure that HIV testing of individuals should only be performed with specific informed consent of that individual. Exceptions to voluntary testing would need specific judicial authorization, granted only after due evaluation of the important considerations involved in terms of privacy and liberty.

Although the content of the requirement for informed consent may vary according to the procedure in contemplation, the legal principles are basic in the case of HIV testing. The person administering the test has duty of care to avoid that risk of harm by ensuring that patient understands the risk before agreeing to be tested.

The 'golden standard' for HIV testing requires both pre-test and post-test counseling. A failure to perform adequate pretest counseling would ordinarily imply a lack of informed consent to any HIV test subsequently administered. To that end, it is a practice for a consent form to be signed by the patient once they have agreed to submit to an HIV test, and for that form to be retained for the protection of the doctor or counselor.

Post-test counseling is the process by which ideally, it is ensured that the test result and its

implications are understood, that the person being tested has a strategy for dealing with that information in relation both to her or himself and to others who might be affected such as a spouse, and has access to any resources needed. The golden standard for HIV testing requires that patients be informed face to face about the test result and not over the telephone or by a third party.

The United Nations HIV/AIDS and Human Rights International guidelines suggest 'In view of the serious nature of HIV testing and in order to maximize prevention and care, public health legislation should ensure, whenever possible that pre and post test counseling be provided in all cases. With the introduction of home-testing, States should ensure quality control, maximize counseling and referral services or those who use such tests and establish legal and support services for those who are the victims of misuse of such tests by others.'

Indian Contract Act 13 and 14 defines consent as:

Indian Contract Act 13—Two or more persons are said to consent when they agree upon the same thing in the same sense.

Indian Contract Act 14—Consent is said to be free, when it is not caused by (i) coercion, as defined in section 15, or (ii) undue influence, as defined in section 16, or (iii) fraud, as defined in section 17, or (iv) misrepresentation, as defined in section 18, or (v) mistake subject to the provisions of sections 20, 21 and 22.

The consent cannot be said to be implied when HIV testing is included in tests undertaken routinely in some medical or surgical treatment.

In **Australian State of Tasmania**, a person must not undertake an HIV test in respect of another person, except (i) with the consent of another person, or (ii) if that person is a child under the age of 12 years, with the written consent of a parent or legal guardian of that child, or (iii) if that person is a child between the age of 12 and 18 years and, in the opinion of the medical practitioner who wishes to undertake the HIV test, is incapable

of giving consent, with the written consent of a parent or legal guardian of that child, or (iv) if, in the opinion of the medical practitioner who wishes to undertake the HIV test, the other person has a disability by reason of which that person appears incapable of giving consent, with the consent, in order, of a legal guardian of that person, or a partner of that person, or a parent of that person, or an adult child of that person, or a prescribed independent authority; (v) if the other person is required to undergo such a test under this Act, or any other Act (vi) if an HIV test is required to be carried out on the blood of that other person under that or any other Act.

According to Tasmanian provision, does not stipulate the consent need be "informed". The patient should also be informed about the treatment options. If the patient is under the age of 18 years, his parents or guardian normally give consent to treatment.

The Tasmanian Act goes on to provide that a medical practitioner may undertake HIV test without the consent of a person, if (i) person is unconscious, unable to consent and (ii) the medical practitioner believes that such test is clinically necessary

The Child Care Act of South Africa provides that when parents refuse or are not able to give their consent, the minister may give his consent. In cases of urgency where the life of the child is endangered or lasting injuries may result, the Hospital Superintendent, where child is being treated or the principal of the institution can give consent.

According to **article 10.6 of Spanish General Law of health**, written informed consent is needed for any intervention.

The **American Medical Association** and the National Academy of Science Institute of Medicine also advise against mandatory testing and recommend voluntary informed consent for testing of patients in the high risk groups undergoing surgery or invasive procedures.

Marriage and Professional Secrecy

The relationship between a doctor and patient being sacred, the physician is expected to maintain professional secrecy, with regard to the information about the patient's condition in order to avoid discrimination. A World Health Organisation consultative committee has suggested that the treating doctor can override the rule to maintain professional secrecy and inform the sex partner of the HIV positive patient about his/her HIV status in the larger interest of the society.

In case of an HIV positive doctor who tested HIV positive in 1995 and was supposed to marry in December 1995, had to call off his marriage as the hospital where blood tests were performed had disclosed the result. He approached the Consumer Disputes Redressal Commission, claiming damages against the hospital for disclosing his blood tests reports and breaching confidentiality, the case was dismissed. He finally approached the Supreme Court.

It was argued by the appellant that 'duty of care' in the medical profession includes confidentiality and since this was violated by the hospital, it was liable to damages.

After going into the ethics of confidentiality base on the Hippocratic Oath, and the national and international codes of medical ethics, the Court ruled that the duty to maintain secrecy is not absolute. It said "The argument that the respondents were under a duty to maintain confidentiality formulated by the Indian Medical Council cannot be accepted as the proposed marriage carried with it the health risk to an identifiable person who had to be protected from being infected with the communicable disease from which the applicant suffered. The right to confidentiality, if any vested in the appellant was not enforceable in the present situation. The argument as regards to the right to privacy was also not accepted by the Court.

The Court observed that had the appellant married the lady, he could have committed offences under section 269 and 170 of the Indian Penal Code.

In an article in 'Physician's Digest' the Rajan T.D. suggests that 'preventing an AIDS patient from marrying violated the basic fundamental rights of a citizen. Besides, it prevents a lonely individual from getting help in caring for him and to raise a family. He suggested that the choice of marriage should be left to the couple and enough provisions should be incorporated into the marriage Act whereby both the parties are fully aware of what they are getting into.

In Spain, professional secrecy is protected by several legislative measures. The Spanish constitution (Article 18.1) states that 'the right to honour, personal and family privacy and one's own image is guaranteed. Contrary to this obligation to maintain professional secrecy, the judicial code stipulates the obligation or duty to disclose.

Article 262 of the Spanish Law of Criminal judgment imposed an obligation on a professional person to report any public offence made known to him or her by way of his or her profession or trade. This refers especially to medical, surgical and pharmaceutical personnel. The same obligation is reflected in article 57.1 of the Spanish criminal code.

But failure to disclose in the case of HIV infected patient does not constitute a criminal offence. If a doctor, who is treating a seropositive health care worker, considers that there is a risk of contagion to third parties, he or she may break the oath of professional secrecy, seeking judicial protection in article 8 of the Spanish Criminal Code.

The ethical guidelines of the South African Medical and Dental Council state that where there is a serious and identifiable risk to a specific individual who, if not informed will be exposed to HIV infection, the doctor shall consider it a duty to seek to ensure that such person is informed. As regards the legal position, AIDS and HIV infection are not notifiable diseases in South Africa. Justification for disclosure should lie in overriding public interest, such as protection of interest of third parties. If the patient who has donated blood is HIV positive, doctor should inform blood bank, as this would be in public interest. The doctor

would also have the right to inform the sexual partners of the HIV positive patient, if it is evident that the patient does not follow doctor's advice to inform them and take precautions.

The ethical guidelines of the South African Medical and Dental Council provide that doctors should inform other medical personnel in charge of the patient after first having tried to obtain the consent of the patient. If this is not successful, the patient should be told that the doctor is obliged to impart this information to such other personnel who is bound to secrecy. Failure to inform which leads to unnecessary exposure of other medical workers to HIV will be regarded in a serious light by the council. As far as a legal duty to inform medical personnel is concerned, the legal convictions of the community may again be influenced in the direction of a duty to inform colleagues 'who need to know' especially in view of the fact that medical profession has recognized this as an ethical duty in South Africa.

In United States, a doctor's ethical duty to keep medical information confidential is not absolute. He may have an overriding duty towards society, when the benefits of disclosure outweigh its harm. This utopian argument is even more convincing when an HIV patient is acting irresponsibly, engaging in risky behaviour without warning the partner. All persons who have a compelling interest, such as sexual partner, needle sharers and medical and nursing personnel must be provided with this information, including relevant mortuary personnel who handle the body when the patient dies. This duty to disclose confidential medical information in appropriate circumstances is not only an ethical duty, but a legal one under public health law in US. The partner of an HIV infected person may be considered to have a right to information that is directly relevant to his/her health.

Blood Transfusion

AIDS has an enormous impact on blood transfusion services because of 'window period' that is the period between contracting the HIV

virus and the HIV test becoming positive-this is generally taken to be about 2-4 weeks. During this period, the blood is infective and will transmit the disease to the recipient of the blood despite the test's being negative. The WHO in association with the League of Red Cross Societies and the Red Crescent Societies initiated a 'Global Blood Safety Initiative'. They published a consensus statement on the question of AIDS in blood transfusion in March 1989 as to "Accelerated plans to recruit safer donors should include to motivate and recruit donors from low risk community groups and components should be prepared only from blood of the safest donors." This is particularly important in areas of high HIV prevalence in which the possibility of false negative tests is increased.

There is universal agreement that screening of all blood, plasma, blood byproducts, donated tissues, organs and semen is necessary and required.

Laws and regulations Governing HIV Infected Persons

Strong laws have been enacted around the World to protect the confidentiality of people who undergo HIV testing. The United Nations HIV/AIDS and Human Rights International guidelines recommend "Public health legislation should ensure HIV and AIDS cases reported to public health authorities for epidemiological purposes are subject to strict rules of data protection and confidentiality.

Confidentiality

In the **Australian State of New South Wales**, the law state that

1. A medical practitioner must not state the name or address of the patient (i) in a certificate sent to the Director General under section 14 in relation to HIV/AIDS, or (ii) except as may be prescribed, in a written or oral communication made by medical practitioner for the purposes of arranging a test to find out whether the patient suffers from HIV/AIDS.
2. A person, who in the course of providing a service, acquires information that another person has (i) has been, or is required to be, or is to be tested for HIV, or (ii) is, or has been, infected with HIV must take all reasonable steps to prevent disclosure of information to another person.
3. Information about a person that is of a kind referred to in subsection (2) may be disclosed, (i) with the consent of the person, or (ii) in connection with the administration of this Act or another Act, or (iii) by order of Court or a person authorized by law to examine witness, or (iv) to a person who is involved in the provision of care to or treatment or counseling of the other person if the information is required in connection with providing such care, treatment or counseling, or (v) in such circumstances as may be prescribed.
4. A medical practitioner or other person who fails to comply with the requirement of this section is guilty of an offence. Maximum penalty is 50 penalty units.

Discrimination

Discrimination is defined as 'treatment of one person less favourably than the other in the same or comparable circumstances on the basis of a characteristic that is not immediately relevant to the situation'.

Article 14 and 16 of the Indian constitution guarantees equality and provide against discrimination in employment respectively. Article 16 prohibits discrimination in public employment on grounds of religion, caste, creed, sex, colour etc. however, these rights are available against the state and not against private employers. There is no specific employment law that provides protection from discrimination to people living with HIV/AIDS.

Laws have been enacted in every Australian State and territory and nationally to protect people from unwarranted discrimination on the grounds of Disability and perceived disability. To cover

asymptomatic HIV infection, "disability" is usually defined as including the presence in the body of organisms causing disease or illness. It is unlawful to discriminate even on the ground of a belief that a person has HIV simply because they had a test for the virus.

US Justice Department in 1986 concluded that S.504 of the Rehabilitation Act would inhibit discrimination based on the disabling effects of AIDS and related conditions but that an individual's real or perceived ability to pass on the disease is not a handicap within the meaning of the statute, and accordingly S.504 did not apply.

The U.S Department of Justice new AIDS policy states that a person infected with the HIV virus is only protected against discrimination if he or she is able to perform the duties of the job and does not constitute a direct threat to the health or safety of others.

In countries like UK, France, Germany, Spain and Netherlands, it is standard procedure for life and disability insurers to demand an HIV test when insuring a certain amount of money. Below this amount the insurers sometimes demand an HIV test on medical grounds.

At present, AIDS and HIV seropositive patients are uninsurable except in case of health insurance programme in Netherlands. In February 11, 1989, the Association of British Insurers issued a leaflet stating that 'having an AIDS blood test is no bar to getting life insurance. They said that anyone with HIV negative test result should be able to get the life cover they want unless there are other circumstances affecting the particular risk.

The joint declaration made by the WHO and the International Labour organization stated that a worker may not be dismissed because of HIV infection and that infected people must go on working as long as they are medically capable of performing their jobs satisfactorily.

The antidiscrimination laws in USA prohibit licensed facilities from requiring an individual to submit to any HIV test as a condition for admission, denying the patient any service, including services needed for major surgery, requiring

employees to have HIV testing unless such testing is based on a bonafide occupational qualification. There are also laws and regulations requiring health occupation board to take disciplinary action when licensed professional refuse to treat HIV positive individuals.

It is ethical and moral to treat patients regardless of disease. American Medical Association provides that a physician shall in the provision of appropriate patient care, except in emergencies, be free to choose whom to serve.

In many jurisdictions, police can require that a person submit to involuntary sample taking that they are compulsory examined or otherwise tested. These powers are enacted to assist police in crime investigation and collection of evidence to prosecute suspected offenders.

Many countries have policies that require visitors and migrants to be tested for HIV and to leave country or be refused entry if found positive. And in some countries, prisoners are also being tested for HIV sero-status. Armed forces establishment argue for mandatory testing of recruits and discharge of anyone found to be HIV positive irrespective of length of service, qualifications or duties.

Rights of AIDS Patients

1. Article 21 of the constitution of India guarantees right to life and personal liberty to every HIV positive individual and it is the fundamental right of every AIDS patient to have access to adequate treatment provided by the government.
2. The HIV infected person also has the right to privacy which is considered as part of the right of life with human dignity. The right to privacy of a person has been stated in Article 12 of the Universal Declaration of Human Rights, Article 17 of the International Covenant on civil and political Rights and Article 21 of the constitution of India.
3. The WHO guidelines state that there is no public health rationale to justify isolation, quarantine or discrimination based on a person's HIV status or sexual behaviour.

Legal Aspects in Relation to HIV/AIDS Patient

A person with HIV infection can be prosecuted under section 269 of I.P.C (Whoever unlawfully does any act which is, and which he knows or has reason to believe to be, likely to spread the infection of any disease dangerous to life, shall be punished with imprisonment for a term which may extend to six months, or with fine, or with both) if he performs a negligent act likely to spread the infection of the disease.

If the act is willful, he may be punished under section 270 of the I.P.C (Whoever negligently does an act which he knows or has reason to believe to be likely to spread the infection of any disease dangerous to life shall be punished with imprisonment of either description for a term which may extend to two years, or with fine or, with both).

A civil suit under the law of tort may be filed to claim compensation for violation of the fundamental rights to personal liberty.

Criminal liability for transmitting AIDS by sexual intercourse:

- I. Should a person knowing that he is suffering from the virus and knowing how it is spread; (i) intentionally have sexual intercourse in order to infect the other person from fatal disease (and the person dies), (ii) has sexual intercourse with another person/ persons not caring whether or they contract the disease and one or more of his sex partners contract disease and die, is held guilty of murder. (S 302 I.P.C).
- II. The criminal liability of person in high AIDS risk group such as male homosexuals, prostitutes who (i) knows AIDS and how it is spread (ii) is HIV positive or in fact has AIDS, but is genuinely unaware of the fact and (iii) has done nothing to ascertain whether or not he has AIDS, nor does he take any precaution in his sexual activity and who transmits AIDS to his sex partners who become infected and die, held guilty of manslaughter/culpable homicide (S.299 I.P.C).

III. If a person knows that he is suffering from AIDS and sexual intercourse spreads it which is fatal and incurable if such person indulges in sexual act with another person and as a result the later contracts the disease and subsequently dies will be charged of murder.

IV. An accused in layfayette, Indiana, US suffering from AIDS tried to commit suicide. Three risk workers stopped him, He beat and scratched the risk workers. He was found guilty of attempted murder (reference Cape Times 15 Jan 1988).

Civil liability for transmitting AIDS by sexual intercourse: The patient can be held liable for (i) negligence and/or (ii) battery and/or (iii) intentional infliction of physical harm or emotional distress and/or (iv) fraud.

Important Case Discussions in India and Abroad in Relation to AIDS

1. Earliest civil case for transmitting a venereal disease. In 1878 the Irish case *Hegarty vs Shine*, Hegraty sued her lover for battery for having infected her with venereal disease. She lost the case. Court held that *exturpicausa non iritur actio* (from a base or immoral action no legal cause will be upheld by the court). the fact that defendant concealed the fact that he suffered from venereal disease did not affect the judgement.
2. *R V Clarins 1888 22 QBD 23*: In the above case the man knowingly transmitted venereal disease to the wife but was not found guilty of assault. Court held that wife's ignorance for her husband's disease would not affect her consent for sexual intercourse. this is an old case and it is doubtful if the courts will still follow the same ruling.
3. *Crowel vs Crowel 1920*, A US court held that a wife whose husband has wrongfully infected her with a venereal disease may obtain damages from her husband.

First criminal law in the world on AIDS: According to Louisiana state legislation, "a person

who intentionally exposes another to the AIDS virus through sexual contact without the knowledge or lawful consent of the victim is guilty of an offence and liable of conviction to a fine of not more than 5000 dollar and / or imprisonment with or without hard labour for not more than 10 years. (Louisiana Act, 663 of 1987).

Relevant Clauses in Indian Penal Code

1. S. 54 I.P.C. injury.
2. S. 440 I.P.C.: The accused can be charged under various provisions of I.P.C. for fraud, deceit or misrepresentation.
3. S 320 I.P.C.: The accused can be prosecuted under the 8th clause of I.P.C.
4. S. 304-A I.P.C.: Causing death by rash and negligent act. Even if the accused did not know that he has AIDS or that AIDS is incurable and fatal may be charged under this section.
5. S. 302 I.P.C. Murder: This is the extreme for which an accused can be charged although the likelihood of this section being invoked for transmitting AIDS is not much.

What are the defences which the accused may raise: The following discussion is best on

actual defences raised by the accused in the recent cases.

1. Defence constitutional right to privacy barring any investigation by the court, many US courts have faced an objection by the defendants. The investigations into matters of marriage, family sex is an invasion of the constitutional right of privacy. But the courts held that:
 - i. No duty on the part of the defendant to disclose the fact that he is infected with the disease.
 - ii. No one acquires a right of action by his own wrong. This defence has not been sustained in the court.
2. *Volenti non fit injuria* (consent), in cases of AIDS a defence of consent may be sustained.
3. Contributory negligence.
4. Defence taken by the husband: Wife had given unconditional consent for sexual intercourse at the time of marriage.

Long Question

1. Discuss the Medicolegal as well as ethical issues that arises in case of AIDS.

Forensic Serology and Trace Evidence

In the time that has elapsed since the discovery of the ABO system by Landsteiner, knowledge in forensic serology has expanded tremendously excluding hormones and some temporary pathological factors, more than 160 antigens, 150 serum proteins and 250 cellular enzymes have been found in human blood. Three classes of blood constituents have been chosen by serologists for analysis of blood samples; Group specific antigens, cellular enzymes and proteins and serum enzymes and proteins.

EXAMINATION OF BLOOD STAINS

Collection of Blood Stains

A clean piece of white filter paper may be used, allowing blood to soak on to it, and then it is dried it at room temperature. A control filter paper should also be sent for examination.

1. If the object is porous, a portion of unstained area should also be taken.
2. If the object is non-porous and particularly, if it is metallic, stains can be recovered by scraping and placed in small glass containers. They should not be placed in envelop as they will be reduced to powder.
3. Stains on clothing may be scrapped of or a fragment of the material cut.
4. The solvents for bloodstains are the following:
 - 10% solution of potassium cyanide
 - 10% solution of glycerin in distilled water
 - A weak solution of ammonia

A coloured solution is obtained immediately with any of the above solvents. Otherwise the

material must be covered and left for 12-24 hours at room temperature.

Stains on Clothing

In the case of clothing, type of garment, its colour and consistency should be noted and if the garments are torn, position of the tears should be noted. Whether the clothes were dried, damp or wet when received should be noted. Examine both the outer and inner surfaces of the garments. The position of all the stains should be given correctly by a description of the stains in its relation to the manner in which a garment is actually worn i.e., a stain on the trouser should be described as being above, behind, or on the inner side of the knee. Stains may also be described in relation to the pocket, the button, or the seams of garments. The size and the shape of the stains should be noted. Bloodstains are extremely resistance to washing by water. Dried blood on a dead body or article is very resistance to water. The dried blood will remain intact for quite a long time, even though the body has been submerged.

Primary Blood Group Systems

The primary blood group systems are well known and defined blood group systems. These are ABO, MNS, P, PH, Diego, Donbrock, Duffy, Kell, Kidd, Lewis, Lutheran. XG and YT

Secondary Blood Group Systems

The secondary blood group systems are antigens that are either foreigners of new systems or

products of rare mutant genes in existing ones. They also include high incident antigens which have not been related to major systems. They include the following Auberger, August, Batly, Beaker, Bils, Bishop, Bga, Bgl, Box, Cavatiere and Chido.

Some of the red cell antigens such as A, B and I have been shown to be present on both leukocytes and platelets. Nevertheless, the antigens representing the majority of blood group systems such as Rh, Duffy, MN and so forth seem to be unique antigens characteristic to leukocytes and platelets.

According to the present state of knowledge, there are four established leukocyte group antigen systems namely HLA-A, HLA-B, HLA-C and HLA-D.

Red blood cells like any other cells shrink in solutions with an osmotic pressure greater than that of normal plasma. NaCl solution is isosmotic with plasma. In solution with lower osmotic pressure, the cell becomes spherical rather than disk shaped.

The hemolysed red cells release their contents in to the solution. More than 250 proteins and enzymes have been found in the red cells. The predominant erythrocyte protein is haemoglobin, whose more than 100 variants have been described. Many red blood cell enzymes show genetic polymorphism as do a significant number of proteins found in blood serum and other body secretions. Many of these persist in their biological activity even after blood has dried. Therefore, blood can potentially distinguish one person from the other.

Medicolegal Importance of Blood

Blood in itself is very important entity in medicolegal practices which alone or along with other trace evidences play key role to unfold different problems. Estimation of blood and bloodstains has importance in both civil and criminal fields of investigation concerning identification of an individual on one side and many other medicolegal

issues on the other side. No other body fluids or tissues have such divergent medicolegal importance as blood has.

Civil Importance

1. Paternity or maternity issues
2. Divorce and nullity of marriage cases, for example question of intersex and some forbidden diseases
3. Compensation cases related to Workmen's welfare e.g. industrial and commercial workers
4. Civil negligence cases arising in Hospital or medical practices e.g transfusion cases, chemotherapeutic practices etc.

Criminal Importance

1. Identification of victim or offenders of crime like homicide, sex offences or where death occurred due to rash or negligent acts in part of person who are expected to act with responsibility.
2. Cause of death e.g. detection of poison or some other pathology responsible for the death.
3. Time of death by use of different chemical or biochemical tests.
4. In cases of criminal abortion and investigation of sexual offence cases e.g. examination of vaginal or seminal fluids on the person or undergarments of victim and the accused.
5. To establish the relationship between the offence, offender, offended (victim) and offending agent. Locard principle is applied.
6. Cases of malingering.

Medicolegal Questions in Relation to Blood

1. *Whether the stain due to blood or other stain?*
Different stain that resembles blood stains are:
 - a. *Rust stain:* Instrument and weapons made of iron are rusted and the rust may resemble blood stain but on heating opposite side of the weapon they don't fall up in scales. They do not have a dark and a glazed appearance. Rust stain do not stiffen the cloth and are soluble in dilute hydrochloric acid. Moreover, they give positive test for iron.

- b. *Vegetable stain*: Fruit like mulberry, currants and *jamuns* produce tan that look like blood stain. In these cases vegetable debris and cells are found when visualized under microscope. On the other hand pan juice, tobacco, fruit and leaves some of the trees produce red stains resembling blood stain. In these cases by addition of a drop of ferric chloride they become black due to presence of tannin.
- c. *Synthetic dye*: In these cases, the colour changes to yellow by addition of nitric acid and the original colour is restored by again addition of strong solution of alkali.
- d. *Mineral stains*: Compounds of iron, red lead, and red sulfide of mercury give red colouration that looks like blood stain. These metal can easily be differentiated from blood stain by appropriate test.
- e. *Miscellaneous group*: Spots of resin, grease and coaltar on fabric looks like an old blood stain but gives negative test for the blood. The stain is due to blood can be found out by simple microscopic tests, different colour tests and crystal tests.
2. *Whether the blood stain is due to human origin are not?*
This question can be answered by performing the precipitin test that is an immunological test and is species specific.
3. When it is established that the stain is due to human blood, the following questions are to be answered:
- a. *The source of bleeding*:
- Menstrual blood*: Usually found in female undergarments during the reproductive period it is dark, fluid, has disagreeable smell and acid in reaction. On microscopy it shows endometrial cells, vaginal epithelium and number of bacilli and cocci. At times *Trichomonas vaginalis* or monilia-sis may be present. Menstrual blood contains fibrinolysins thus prevents clotting.
 - Haemoptysis*: It is bright red and is mixed with froth and alkaline in reaction.
- iii. *Vomitus*: The vomited blood is chocolate brown in colour and acid in reaction due to presence of gastric contents
- iv. *The blood in cases of rape*: This is usually mixed with semen and pubic hair may be present at times.
- v. *The blood stain due to boils and source*: A mixed appearance of blood and a pus is found and one can find pus cells and bacteria under microscope.
- b. *Age of the blood stain*: The age is usually determined by the colour change which is quite unreliable.
- Fresh stain—Bright red
 - 24 hours—Reddish brown
 - Within a few days—Brown
 - After a long time—Black.
- Fresh stains are moist, sticky and stiffen the cloth on drying due to presence of proteins a drop of blood dries of in one to two hours in normal conditions. However, in cases of pool of blood it takes 24 to 36 hours to dry depending upon the amount of blood, surface of collection and temperature etc. The solubility gradually decreases with the age. Fluorescence decreases as the stain becomes older. On the whole it can be ascertained that whether the stain is fresh, recent, a weeks duration or old.
- c. *The sex of the person*: The sex can be ascertained from the presence of sex chromatin.
- d. *The blood group of the person*: Grouping can be known by doing the different blood group test.
- e. *Whether it is arterial or venous in origin*: The bleeding from the arteries has a sprouting effect and is bright red in colour when fresh. On the other hand, the bleeding from a vein occurs passively and dark in colour even when it is fresh.
- f. *Whether it belongs to the accused or victim*: The blood stains on the inner side of the garment definitely belongs to the victims

whereas stains on the outer side may belong to the accused or the victim.

g. *Whether the blood stain is derived from a living person or dead one?*

The blood which has shed during life can be removed in scales on drying due to presence of fibrin but the postmortem blood breaks of into powder on drying.

Physical Tests for Blood

- **Naked eye examination:** On naked eye examination, the non coagulated blood is thick, viscid and bright red or bluish depending on the source or region. Antemortem bleeding causes coagulation that separate the serum. The clot can be taken out enmass from the spot but leaves an impression over there due to the fibrinous network that results in the clot formation. The postmortem clot cannot be taken enmass and it leaves no impression on the spot. Roughly three layers are detected in the mass; one by the platelets, second by the RBC and third by WBC.
- **Microscopic examination:** This can be established whether the sample is blood or not and can differentiate between the blood of various species. The human RBC is biconcave, spherical, nonnucleated and the average diameter is 7.3 micron, the maximum breadth is 2.5 microns and the minimum is 1 micron. The average volume of red cells is 90-95 cubic micrometers, whereas in other mammals it is biconvex, nucleated and the size varies with the species.
- **Spectroscopic examination:** This is one of the most definitive tests for establishing the presence of blood. It is based on the absorption spectrum of the various types of hemoglobin in the blood. Even less than 0.1 mg of blood sample is sufficient for doing this test. The blood stain is dissolved in water, salt solution, or dilute ammonia and is placed in a glass tube and examined through a spectroscope. The absorption spectrum is characteristic for each type of hemoglobin. This definitely establishes that

the sample is blood.

Colour Tests

Principle: The colour tests detect the presence of hemoglobin, the colouring matter of blood, a peroxidase which in the presence of hydrogen peroxide oxidises the colourless bases to coloured salt. It is also possible to utilise the same peroxidase reaction at a much higher sensitivity by using luminal reagent. It shows bright fluorescence in the presence of blood.

- **Benzidine test:** It uses 10% solution of benzidine in glacial acetic acid. When the stain extract is mixed with it, it produces an intense blue colour on addition of hydrogen peroxide if blood is present. The test is positive in dilution of one part of blood in 500,000 parts and was commonly used in medicolegal practice. Nevertheless, nowadays it is of limited use because of its carcinogenic properties.
- **Phenolphthalein test (Kastle Mayer tests):** The reagent for this test is obtained by mixing a 2% solution in 20% aqueous solution of potassium hydroxide till the solution becomes colourless. The phenolphthalein is reduced by zinc dust in a strong alkaline medium. If the reduced phenolphthalein is oxidised by nascent oxygen liberated by the action of peroxidase on hydrogen peroxide. This gives a pink or purple colour with blood stains.
- **Leuco malachite green test:** It uses a 1% solution in 40% aqueous solution of acetic acid and produces a bluish green colour with blood stains.
- **Orthotoulidine test (Kohn & Kelly test):** This utilises 1% solution in 40% of alcohol and gives a bluish green colour with blood stains
- **Luminal test:** This utilises a solution of 3-amino phthalic acid hydrazide, sodium carbonate and hydrogen peroxide and produces a bright luminescence in presence of blood.

Interpretation of Bloodstains

A negative result excludes the presence of blood. but a positive result may indicate that the

suspected stain could be blood as lots of other material gives false positive test. False positive coloured catalytic test reaction can be given by following substances:

1. **Chemical oxidants and catalysts:** This includes copper and nickel salts, rust, formalin, potassium permanganate, potassium dichromate, some bleaches, hypo chlorite, iodine and lead oxides. Phenolphthalein gives positive results with oxidizing compounds such as copper, potassium ferricyanide, nickel, cobalt nitrates and some sulfocyanides. Luminol reacts with cupric ions and some compounds of copper, cobalt and iron. Potassium permanganate and hydrated sodium hypochlorite also gives positive luminal reaction.
2. **Plant sources:** Vegetable peroxidases are the most important class of substances which show false positive reaction with chemical colour tests. The following plant tissues may be mistaken for blood such as Apple, Apricot, Bean, Blackberry, Jerusalem artichoke, horseradish, potato, turnip, cabbage, onion and dandelion root. Plant materials such as horseradish, beetle leaf, garlic, cabbage, tomato and cucumber react positively with tetra methyl benzidine. Higaki and pulp reported that plant peroxides does not contribute to false positive result in a three-stage phenolphthalein test.
3. **Animal origin:** The following substances of animal origin may give false positive reaction with benzidine reagent like pus, bone marrow, leukocytes, brain tissue, spinal fluid, intestine, lung, saliva and mucous.

Physicochemical test

Microchemical tests:

1. **Takayama reagent test:** This test is also known as the hemochromogen crystal test. Takayama reagent is used in this test which is prepared from sodium hydroxide, pyridine and glucose. The sample material is placed on a glass slide and takayama reagent is added to it and it is examined under microscope. Pink

feathery crystals of hemochromogen are formed which are arranged in clusters, sheaves etc. The test is considered negative if no crystals are formed in 30 minutes.

2. **Teichmens test:** This test is also known as haemin crystal test. In this a crystal of sodium chloride is added along with a few drops of glacial acetic acid and it is heated with a coverslip in place. After cooling faint yellowish-red to brownish black rhombic crystals are seen if blood is present in the sample. The test gives a negative reaction if the stains are old, washed or treated by chemicals.

Interpretation of crystal tests: The sensitivity and specificity of the Takayama test is essentially the same as that of the Teichmann test. The test is positive with as little as 0.001 ml of blood or 0.01 mg Haemoglobin. It is superior to the teichmann test in that it can often give positive results with blood removed from wood or leather surfaces. The drawback of the Takayama test is that a complete crystallization is difficult to obtain with old blood sample and there may be formation of different types of crystals. The failure to obtain a positive Takayama test does not necessarily indicate the absence of blood.

Immunoserological Tests

Principle: The serum of the blood stain contains protein. If antihuman serum is treated with blood stain from human source then there will be antigen antibody reaction which can be demonstrated by various serological tests mentioned above. It is to be remembered that many animal serum proteins may be agglutinated by or may be reacted upon by anti human sera. The serum proteins preferred for these tests are globulin and anti human globulin that is prepared by injecting human serum in rabbits. The antisera to be used should be first made free from reactivity with the animal blood proteins by treating the anti sera with the similarly available animal protein or sera.

1. **Ring test:** It is a precipitation test performed with antihuman serum and stain extract. 0.2

ml of stain extract of different dilutions are taken in round bottomed glass tubes of 5 cm length and 5.5 mm diameter. Equal volume of antihuman serum is added in the tube by running down along the wall of each tube. A precipitation ring is formed at the interface of the two fluids, with 1: 1000 dilution of the stain extract, if the stain was of human blood. Control tests are necessary with normal saline, human serum and sera of other animals.

- 2. Gel electrophoresis test:** In this test the extracts of different stains are placed in separate holes on the side of a gel strip and antihuman globulin and antisera for other common animals are placed in separate holes on the other side of the gel strip. The set is then subjected to electrophoresis in such a way that albumin and non gamma globulins from the stain extract side travels to the anti sera side and reacts with the gamma globulins there. A line of precipitate forms at the meeting point of proteins travelling from the stain extract side with the specific antisera, which denotes the species origin of the stain extract that underwent precipitation reaction with the antisera of a particular animal or human origin.
- 3. Mixed antiglobulin test:** In this test antihuman globulin rabbit serum is prepared and treated with A, B, O blood group cells and sera of other species of common animals to avoid error in record of study, due to possible reaction with antihuman globulin sera and these agents. The red cells chosen are used after washing, for at least ten times, in normal saline solution. The antihuman globulin thus treated and obtained is suitable for use in the test. O Rh+ red cells are used as the indicator cells for the positive reaction in the test. Sensitized red cells are prepared by mixing 1 ml of 2 % suspension of O group of Rh+ red cells with 1 ml of 10% in complete anti D sera and the mixture is incubated for 1 hour at 37°C. These cells are washed and made to 1% suspension with rabbit protein which then becomes agglutinable by antihuman globulin serum.

- 4. Passive haemagglutination test:** The RBCs, when treated with tannic acid becomes capable of binding proteins on their surface. If the extract contains protein due to the presence of human blood then, tanned human RBCs can bind the protein in the stain and hence the RBCs there after will be agglutinated when further treated with antihuman globulin. For the test a cell suspension is prepared and a drop of it is mixed with one drop of anti human globulin serum. Clumping indicates the presence of human protein in the extract. Control tests are to be performed alongwith the main test.

Points to be Remembered in Relation to Serological Tests for Extracting Stains

All reasonable control tests are to be performed along with the tests. Ageing of the stain, heat and washing with soap or detergents may give false negative results. However, if the stain is otherwise protected from decomposition or contamination from the dirt or chemical then ageing alone does not damage such the reacting capacity of the stain serologically.

Once it is established that the stain is due to blood and that it belongs to human being then the next series of serological tests is involved. For detection of group factors in the blood to help to relate the stain extract with a particular individual, further various blood groups and types of tests are available such as:

ABO GROUPING

The most well known and medically important blood types are in the ABO group. All humans can be typed for the ABO blood group. The ABO type of a person depends upon the presence or absence of two genes, A and B. These genes determine the configuration of the red blood cell surface. A person can be A, B, AB, or O. If a person has two A genes, their red blood cells are type A. If a person has two B genes, their red cells are type B. If the person has one A and one B gene, their red cells are type AB. If the person has neither the A nor B gene, they are type O.

The Table 1 shows the possible permutations of antigens and antibodies with the corresponding ABO type ("yes" indicates the presence of a component and "no" indicates its absence in the blood of an individual).

ABO Blood type	Antigen A	Antigen B	Antibody anti-A	Antibody Anti-B
A	Yes	No	No	Yes
B	No	Yes	Yes	No
O	No	No	Yes	Yes
AB	Yes	Yes	No	No

Genetic Inheritance Patterns

ABO blood types are inherited through genes on chromosomes, and they do not change as a result of environmental influences during life. An individual's ABO type is determined by the inheritance of the 3 alleles (A, B, or O) from each parent. The possible outcomes are shown below:

Parent Alleles	A	B	O
A	AA (A)	AB (AB)	AO (A)
B	AB (AB)	BB (B)	BO (B)
O	AO (A)	BO (B)	OO (O)

Both A and B alleles are dominant over O. As a result, individuals who have an AO genotype will have an A phenotype. People who are type O have OO genotypes. In other words, they inherited a recessive O allele from both parents. The A and B alleles are codominant. Therefore, if an A is inherited from one parent and a B from the other, the phenotype will be AB.

Tests for Determining A, B, O Blood Groups

1. *Tube method*: In this method a drop of washed suspension of red cells is added to equal

volumes of each of the antisera of A, B and O in separate tubes. When agglutination is seen by naked eye or microscope in the tubes the blood group can be identified.

2. *Tile method*: In this method a porcelain tile rather than a tube is used and the results are similar to tube test.
3. *Absorption-inhibition method*: In this technique the sample and anti sera are allowed to react with each other for a longer period. This test is useful in cases where the cell membrane is damaged and agglutination test cannot be done.
4. *Mixed agglutination tests*: In this test the sample piece of stained cloth is cut in to pieces of 2-3 mm and the fibers are separated. then they are added to tubes along with antisera of A,B, O types. clumps of red cells seen adherent to the fibres is a positive reaction and the group can be known by the antisera which clumped to the cloth fibres.
5. *Absorption-elution technique*: This test is similar to mixed agglutination test but the clumps are noticed on the walls of the test tube rather than the the sample cloth fibres. This technique can be used to find the blood group from body tissues also.

MN SYSTEM

The MNSs system was discovered by Landsteiner and Levin in 1928. Two antibodies called anti-M and anti-N were identified. These two antigens having shown to be derived from a single pair of genes which are allelomorphous and co-dominant. The frequency for these groups in human population is (i) Group M—20% (ii) Group N—22% (iii) Group MN—50%.

These two antigens can be paired as:

1. M pairing with M—genotype MM and phenotype or group M
2. N pairing with N—genotype NN phenotype or group N
3. M pairing with N producing genotype MN—phenotype or group MN.

Table 31.3

Groups of parents	Possible groups of children	Groups not possible
M × M	M	N, MN
M × N	MN	M, N
M × MN	M, MN	N
N × N	N	M, MN
N × MN	N, MN	M
MN × MN	M, N, MN	

Ss SYSTEM

This system was discovered in 1947 by Walsh and Montgomery. They discovered an antigen (S) and antibody (anti-S). The proportion of blood samples agglutinated by anti-S is about 55% and those not agglutinated are about 45% (in English and American white population). Of the M samples about 73% are agglutinated. The S occurs more often with M than with N suggesting an association between MN groups and S. Anti-S has been used for paternity testing but anti-s is too rare.

Rh SYSTEM

Landsteiner and Wiener in 1940 discovered human blood factor called 'Rhesus' or 'Rh'. The guinea pigs and rabbits were immunized with blood from *Macacus rhesus* monkey. They obtained an anti-serum that after suitable absorption agglutinated not only the red cells of rhesus monkey but also about 85% of blood samples from whites of Newyork. Those whose red cells were agglutinated by the new antibody were designated by Rh-positive and those whose red cell were not so agglutinated were designated Rh-negative. Anti-sera giving similar reaction to animal anti-Rh sera were found to occurring humans. The individual who lack Rh antigen may in certain circumstances from Rh antibodies. This could be due to receiving a blood transfusion of Rh-positive blood or carrying of an Rh-positive fetus.

KELL SYSTEM

Rarely there is presence of anti-K and this is sometimes helpful in paternity cases. The finding of a K-positive child with a K-negative mother and

putative father would constitute an exclusion of paternity.

DUFFY SYSTEM

This system employs the use of two sera anti-Fy^a and anti-Fy^b. Anti-Fy^a provides evidence of non-paternity when the putative father and mother are Fy^a—and the child Fy^{a+}. Anti-Fy^b is a rare serum but if obtainable in sufficient quantity and strength could be usefully used with anti-Fy^a in the testing of white races. In doubtful paternity cases involving Negroes it must be remembered that the phenotype Fy(a-b-) is common.

KIDD SYSTEM

This system is defined by anti-Jk^a and anti-Jk^b sera. Both at presnt are rare, particularly anti-Jk^b. At the moment insufficient family studies have been made for this system to be used except with caution and as additional evidence in special cases.

LUTHERAN SYSTEM

The finding of a Lu (a+) child with Lu (a-) mother and putative father can be regarded as evidence of for an exclusion based on another blood-group system. Anti-Lu^b is too rare for regular use and furthermore is of small value as it reacts with over 99% of samples.

OTHER ANTIGENIC FACTORS INHERITED IN SERUM

Haptoglobulins

Haptoglobulins are haemoglobin binding proteins that are found in human serum. They are observed as characteristics bands during electrophoresis. The differentiation of three different pattern of these proteins depends on superior resolving power of the starch-gel method. It is possible to classify most people into three consistently inherited heptoglobin (Hp)

Haptoglobulins 1-1	(Hp ¹ Hp ¹)
Haptoglobulins 2-2	(Hp ² Hp ²)
Haptoglobulins 2-1	(Hp ² Hp ¹)

Haptoglobulins are only occasionally present at birth and 97% of infants can be typed by six months of age.

GM factors in Blood Serum

GM factors in blood serum such as GM1 antigen

Red Cell Enzyme Systems

A large number of enzymes that catalysed various biochemical reactions are also present in blood plasma. Phosphoglucose mutase (PGM) and adenylate kinase (AK) have an established value in paternity testing. Besides this Red cell acid phosphatase (EAP), Adenosine deaminase (ADA), 6-phosphogluconate dehydrogenase (G-6PD), glutamate pyruvate transaminase (GPT) and esterase D (Es D) may also be used. These enzymes are demonstrated by starch gel electrophoresis technique and do not persist for more than one month in stain material.

Table 31.4: Paternity exclusion chances with different blood group systems

Group systems	Cumulative exclusion rate
ABO	18%
ABO + MNS	60%
ABO + MNS + red cell antigens and serum proteins	82%
ABO + MNS + red cell antigens and serum proteins + red cell enzymes	94%
ABO + MNS + red cell antigens and serum proteins + red cell enzymes + HLA	98.5%
DNA typing	100%

Abnormal Haemoglobins

There are three varieties of normal haemoglobin:

1. **Haemoglobin A**—Alpha + Beta chains.
2. **Haemoglobin A2**—2 Alpha polypeptide chain + Delta chain. It is present in 2% of total haemoglobin.
3. **Haemoglobin F**—Present in new born foetus up to extent of 80% maximum. Consists of

alpha type and gamma type. By two years of age blood will not have any foetal haemoglobin.

Criteria of abnormal haemoglobin:

- Haemoglobin H—All four chains are Beta chains
- Haemoglobin Barts—All four chains are gamma chains, the normal polypeptide chain are abnormally conjugated.
- Haemoglobin S
- Haemoglobin C
- Haemoglobin E
- Haemoglobin D Punjab.

There are substitutions of some amino acids at some points by some other which are not there in normal Haemoglobin. These abnormal Haemoglobins can be detected with paper electrophoresis. Fetal Hb can be differentiated by its property of alkali resistance and ultra violet absorption curve.

Medicolegal aspects of abnormal haemoglobin:

Haemoglobin A1 and A2 are normal varieties in adults and children above two years of age. Haemoglobin F is normal in the new born and decreases up to the age of 2 years by which time it is totally removed. This point may be helpful also to determine the age of infants and young children. Inheritance of abnormal Haemoglobin has importance in deciding the paternity and maternity disputes. Abnormal Haemoglobin has importance as being natural cause of death and for identification of a person.

Group Specific Substance

In the body tissues, agglutinogens of ABO system are present. In the tissues they appear in lipoidal form and in about 80% people they appear in water soluble form that can be demonstrated in all the body fluids except CSF. These substances are not found in nerve tissue, epithelium, appendages of skin, bone and cartilage. Non-secretors are persons who possess only lipoidal form whereas secretors are the one who possess a water soluble form. A pair of allelic genes S_e (dominant) and s_e (recessive) are responsible for capacity to

secrete these antigens in body fluids. Secretors possess genotype $Se Se$, and $Se se$ whereas non secretors possess genotype $se se$. Secretors are also possess H antigen on the red cells irrespective of the ABO system. The amount of H antigen is highest on RBC of persons with O blood group.

DNA Profiling of the Blood Sample

Procedure of Sample Collection and Transit to Lab

As red cells have no nuclear DNA, sufficient blood must be obtained to extract DNA from sparser leucocytes. At least 1 ml and preferably 5ml are taken in to an EDTA tube that extracts metallic ions and not only prevents clotting but inhibits enzymes in blood or microorganisms which may break down DNA during storage.

The blood sample in a plastic (not glass) tube should be frozen solid in a deep freeze or the ice making compartment of an ordinary refrigerator. As frozen blood is useless for some other forensic investigations, the DNA samples should be clearly marked as such. If a blood sample is to be taken directly to the laboratory for DNA profiling then freezing is not necessary. But whenever there is delay, either freezing or effective cooling should be carried out.

Blood stains should be sent intact on surfaces, kept as cool as possible before and during transit to laboratory or rubbed with cotton wool swab moistened with water. The swab is then dried without heat and frozen along with the control swab. Dried blood stains on hard surface can be scrapped off with the scalpel in to a small plastic container and sent as they are kept as cool as possible. Blood grouping in cases of doubtful paternity is only attempted by specialist serological laboratory.

Medicolegal Aspects

1. To identify the assailant by studying the nucleotide sequences of cells of the blood and hair bulb available at the scene of crime with

those of the cells of the accused.

2. Identification of the rapist by studying the sperm cells available on the body or the cloth of the victim and cells of an accused is possible by studying their nucleotide sequences.
3. Studying the nucleotide sequences of DNA of woman (mother), man (father) and baby for settling the disputed paternity.
4. Mixed babies in the hospital can be identified.
5. Missing persons can be identified, if the parents or children are available from the dead body remains, using the PCR technique, identification is possible, if his or her parents or children and wife are available as by the study of fingerprints (DNA fingerprinting).

Examination of Nails

From the nails, poisons (heavy metals like Arsenic) can be detected. Debris on the nail bed may show presence of foreign tissue and blood, which can give sex and blood group of the person.

Examination of Skin and Dandruff

The mixed agglutination principle is used for ABO grouping of skin and dandruff as described by Swine burne. Cell suspension of skin scrapings/ dandruff flakes is used in this method. Positive result is shown by firm adherence of indicator cells to the skin or dander cells under tests.

Differences from fibers: These mixed agglutination clumps will be circular in appearance rather than elongated as in the grouping of fibers.

Examination of skin may help to know the sex, blood group and detection of poisons, from dandruff blood group of the person can be known.

Examination of Vaginal Fluids

This is done for the purpose of:

- Detection of semen in vaginal fluid
- Detection of blood in seminal fluid
- Detection of blood group specific substances in vaginal fluid, if the person is a secretor

Examination of vaginal epithelial cells is a good way to know the actual sex in gonadal dysgenesis cases. Presence of vaginal epithelium on the glans

of penis of the accused supports sexual intercourse. The glans is rubbed with a moist blotter and subjected to the vapour of iodine. The blotter becomes partly brown if vaginal epithelial cells are present as they contain glycogen that turns brown due to iodine.

Vaginal stain extract should also be examined for poisons of a suspected case of criminal abortion.

Examination of Urinary Stains

The presence of urine is ascertained by the use of urease tablets, testing for creatinine and detection of an odour of urine in the concentrated extract. Blood group substances, anti A and anti B agglutinins are also detected. Species identification of urine can also be undertaken. A method of concentrating urinary proteins is based on a technique used by mejsbaum-Katzenellenbogen. The concentrated urine proteins are subjected to precipitin test by the agar diffusion method against the antiglobulin of various relevant species, including humans. It is suggested that the method might apply to urine stains both for species identification and for the detection of isoagglutinine.

Medicolegal Importance

Stain due to urine may be known by using urease test and also testing the stains of different constituents of urine. Examination of urine may also give blood group of the person if he is secretor. Many poisons are extracted through urine and can be detected in the urine.

Examination of Seminal Stains

Examination of seminal stains has both civil and criminal importance

Civil Importance

- Compensation on ground of acquired sterility
- Disputed paternity
- Legitimacy
- Artificial insemination
- Divorce cases –non ejaculation amounts to impotence

- Compensation on the ground of failure of vasectomy cases, leading to pregnancy of wife.

Criminal Importance

- Concerning commission of sexual offences
- Identification of offender of sexual offence cases.

Examination of Seminal Fluid

1. *Physical appearance:* Seminal fluid is pale or grayish white in colour, thick, viscid with and has a characteristic smell. The seminal stain fluoresces under ultraviolet light.

Seminal fluid is composed of spermatozoa, Lipoglycoprotein, hyaluronidase, deoxyribonucleoproteins and other enzymes. Seminal plasma is formed of choline and spermine. On florence test, choline is seen as dark brown rhombic crystals. In Barberio's test yellowish long needle shaped crystals of Spermine picrate are formed. Apart from this seminal fluids also contains citric acid, sorbitol, prostaglandin, inositol, phosphatases, proteases and other enzymes.

2. *Acid phosphatase test* The concentration of acid phosphatase in prostatic is 20 to 400 times more than any other body fluid. Undiluted semen has acid phosphatase activity of 340-360 bodansky units. The concentration of acid phosphatase gradually falls with time in vaginal secretions. Positive reactions are seen for 36 hours that disappear in 72 hours. The test is conducted using alpha naphthyl phosphatase and fast black K dye. Positive reaction is indicated by purple colour appearing very rapidly (within ½ minute) in comparison with acid phosphatase of other origin.

3. Creatinine phosphokinase test

4. Ammonium molybdate test

All these chemical tests are screening tests; many other body fluids also give positive reactions for these tests. If these tests are negative then presence of seminal fluids in the stain is excluded. Rapidly positive acid phosphatase test is a more dependable one.

5. *Gel electrophoresis test*: Gives almost diagnostic result for presence or absence of seminal fluids.
6. Microscopic demonstration of spermatozoa
Human spermatozoa is 55µm to 70µm in length and its microscopical examination by H and E stains using vaginal fluid or stain extract or swab extract or fresh semen and staining of stain extract using methylene blue or haematoxylin eosin
7. Test for blood group factors in the semen.

Examination of Salivary Stains

Saliva is a thin fluid secreted by salivary glands. It contains enzymes like ptyalin (alpha amylase, glucose 6 phosphate dehydrogenase, various proteins, lipids, chloride and thiocyanate ions etc.).

Stains of saliva are not found on the crime related articles as often as the stains of blood and semen. In very peculiar context, their significance is high. Unburnt portions of cigars, cigarettes or biddies are such example. Saliva stain grouping on such articles for the scene of crime is very helpful in the identification of the criminal. The presence of saliva can be confirmed by the presence of buccal squamous cells (microscopic). Detection of presence of amylase, if the stain was due to saliva, then there will not be any bluish discolouration because amylase digests starch partly to dextrose and then to maltose.

The sex of the person can be determined by the Barr bodied present in buccal mucosal cells present in the saliva.

Group of the person can be known by mixed agglutination test with the buccal squamous cells or the fibers from the stained parts of the clothes. Some of the poisons are secreted in the sputum. Sample preparation for DNA analysis.

The genetic procedure for DNA violation from evidence samples to digest the sample with proteins are K in a detergent followed by phenol/

chloroform extraction solvents and concentrated prior to amplification. This is conveniently done in several wash steps employing spin ultra filtration concentrators such as the centurion 100 micro dialysis units, especially for samples containing less than 500mg DNA.

In case of seminal stains, sperm head are resistant to digestion by proteins K in detergent. The intact sperm head are collected by centrifugation, worked and digested in a solution containing detergents.

Levels of DNA in biological materials

Type of sample	Amount of DNA
Blood stain (1cm ²)	20-40 µg/100 ml
Blood stain (1mm ²)	200 µg
Semen	2 µg
Vaginal swab	150-300 µg/ml
Hair plucked	1-750 µg/hair
Blood smeared hair	0-12 µg/hair
Saliva (buccal cells)	0.5-19 µg/ml
Urine (urinary tract cells)	1-20 µg/ml

Long Questions

1. Discuss the medicolegal impotence of blood.
2. What are the different blood groups. How they help in solving the disputed paternity cases?
3. Discuss the role of semen in sexual offence cases.

Short Question

1. Trace evidences
2. Locards principle
3. ABO group
4. Colour test
5. Haemin crystal test
6. Hemochromogen test
7. Atavism
8. Precipitin test
9. Acid phosphatase test
10. Composition of semen
11. Human sperm

MULTIPLE CHOICE QUESTIONS

1. All are tests done on blood *except*:
 - A. Acid Phosphatase test
 - B. Benzidine test
 - C. Hemochromogen test
 - D. Teichmann's test
2. A blood donor is not considered for safe transfusion, if he has:
 - A. Anti HBs Ab +ve
 - B. Anti-HBs Ab and HBc Ag +ve
 - C. Hbs Ag +ve, & IgM anti-HBc +ve
 - D. Anti HBe +ve
3. Investigation of choice for blood grouping in old blood stain on the cloth is:
 - A. Precipitin test
 - B. Benzidine test
 - C. Acid dilution test
 - D. Hemin crystal test
4. Most specific test to detect blood stains is:
 - A. Benzidine test
 - B. Teichmann's test
 - C. Orthotoluidine test
 - D. Spectroscopic test
5. In Teichmann's test microscopy shows-
 - A. Rhombic crystals
 - B. Due to Haemin crystals
 - C. Pink feathery structures
 - D. Due to haemochromogen:
 - a. A and B
 - b. A and D
 - c. C and B
 - d. C and D
6. The test used to differentiate blood of humans from those of lower animals is:
 - A. Benzidine test
 - B. Teichmann's test
 - C. Takayama test
 - D. Precipitin test
7. An old blood stain in a cotton fiber can be best identified by:
 - A. Haemin test
 - B. Precipitin test
 - C. Benzidine test
 - D. Leucomalachite test
8. If the father has blood group 'B', mother AB, the children are not likely to have
 - A. O
 - B. A
 - C. B
 - D. AB
9. Which of the following sign will indicate the menstrual blood was antemortem?
 - A. Alkaline
 - B. Light pink
 - C. Does not clot
 - D. Shows endometrial and vaginal cells

Forensic Psychiatry

Forensic psychiatry is the application of psychiatry in the administration of justice. The study of etiology, diagnosis and treatment of mental disorders is called psychiatry. The Indian Lunacy act, 1912 has become outdated because there has been rapid advancement in the field of medical science. The attitude of society towards persons afflicted with mental illness has changed considerably and it is now realised that no stigma should be attached to such illness. It had become necessary to have fresh legislation with provisions for treatment of mentally ill persons in accordance with the new approach accordingly, the mental health bill was introduced in the parliament.

Objects and reasons: The attitude of the society towards persons afflicted with mental illness has changed considerably and it is now realised that no stigma should be attached to such illness as it is curable, particularly, when diagnosed at an early stage. Thus the mentally ill persons are to be treated like any other sick persons and the environment around them should be made as normal as possible.

The experience of the working of the Indian Lunacy Act, 1912 has revealed that it has become outmoded. With the rapid advance of medical science and the understanding of the nature of the malady, it has become necessary to have fresh legislation with provisions for treatment of mentally ill persons in accordance with the new approach. The bill was passed in both the houses of parliament on 2nd May 1987 and came into force on 1st April 1993.

INSANITY

The word insanity has no technical meaning in law or in medicine. It does not connote any definite medical entity but is solely a legal and sociological concept used to designate those members of society who are unable, on account of mental disease to adapt themselves to ordinary social requirements, so that the community compulsorily segregates them and takes away their rights as citizens. Insanity is thus seen as a social inadequacy and medically it is a form of mental disease. Insanity is a degree of mental disturbance. The person may be considered from legal point of view to be immune from certain responsibilities. He may be disallowed certain privileges that may require a degree of competence for example to marry, to make contracts, to manage property etc.

The word **unsoundness of mind**, which finds a place in IPC and is used as a synonym with other terms like insanity, lunacy, madness or mental derangement or disordered state of mind, due to which the individual loses the power of regulating his actions and conduct according to the rules of the society to which it belongs.

Binet–Simon Test for IQ: A measure of intelligence, based on psychological tests, that is calculated as $IQ = \frac{\text{mental age}}{\text{chronological age}} \times 100$. When the mental age increases, the IQ provides an index of relative brightness that can be used to compare children of different ages. In adults, the concept of mental age becomes less relevant. There are separate age norms in

various age groups upto age 65. The IQ as measured by most intelligence tests, is an interpretation or classification of a total test score in relation to the norms. It can be misleading since it is an average of different abilities or levels of ability that themselves may show great variability. The IQ is a measure of present functioning ability, not necessarily of future potential.

Oligophrenia (Mental subnormalities): In the above condition there is retarded or incomplete and abnormal mental development. The I.Q. is below 70. It mostly results from environmental, genetic and endocrinal abnormalities. even at times birth trauma can be a cause. The following three categories are encountered:

1. *Idiot:* These are persons with an I.Q. between 0 to 20. There is defective mental development since birth or early childhood. It may be associated with physical abnormalities like microcephaly or mongolism and at times there is defective development of the other organs of the body.
2. *Imbecile:* These are persons with the I.Q. between 20 to 50. These persons also have the defective development of the brain from birth or early childhood and are not capable of being taught.
3. *Moron:* These persons have an I.Q. ranging from 50 to 75 and have the mental development of a child of the age of 6-11 years. They require constant care, and supervision for their protection.

Wechsler Adult intelligence Scale (WAIS): WAIS is a general test of intelligence, which Wechsler defined as, "the global capacity of the individual to act purposefully, to think rationally, and to deal effectively with his environment." In keeping with this definition of intelligence as an aggregate of mental aptitudes or abilities, the WAIS-R consists of 11 subtests divided into two parts, verbal and performance. It is further divided into six verbal subtests and five performance subtests. The *verbal tests* are: (i) Information (ii) Comprehension (iii) Arithmetic (iv) Digit span (v) Similarities (vi) Vocabulary. *The Performance*

subtests are: (i) Picture arrangement (ii) Picture completion (iii) Block design (iv) Object assembly (v) Digit symbol. The scores derived from this test are (a) Verbal IQ (VIQ) (b) Performance IQ (PIQ), and (c) Full Scale IQ (FSIQ). The FSIQ is a standard score with a mean of 100 and a standard deviation of approximately 15.

Causes of Insanity: (i) Hereditary conditions (a) Huntington's chorea (b) Amaurotic familial idiocy (ii) Environment factors (a) Faulty parental attitude (b) Lack of mental hygiene (iii) Psychogenic causes: Repression of mental conflicts (iv) Organic diseases (a) Head injury (b) Senile degeneration (c) Atherosclerosis (d) Myxedema (v) Insanity due to Drugs such as alcohol, cocaine, hashish and opium (vi) Insanity of pregnancy and childbirth.

Precipitating factors for Insanity: (i) Financial and business worries (ii) Frustration and disappointment in love affairs (iii) Death of near and dear ones.

Alcohol insanity:

1. *Delirium Tremens:* This is a type of delirium seen in chronic alcoholics. It occurs 1-2 days after sudden withdrawal of alcohol due to heavy bout of drink. Injuries, infections and shock may precipitate it. Patient becomes sleepless, restless, irritable and there are disorders of perception. There are coarse muscular tremors of face, tongue and hands. Also, there is disorientation and hallucination and the person may commit suicide, homicide etc.

Medicolegal Importance: It is considered as unsoundness of mind and the person is not responsible for the crimes.

2. *Korsakoff's disease:* In Korsakoff's Psychosis, there is loss of memory for recent events, extreme dissociation. The crimes related to sexual jealousy which are homicidal in nature are committed.

Other drugs: Addiction of drugs such as cocaine, hashish, opium may lead to insanity.

Insanity of pregnancy and childbirth: During pregnancy and child birth attacks of mania, melancholia, dementia etc. are precipitated.

Psychosis

A major mental disorder of organic or emotional origin characterised by extreme derangements of the personality often accompanied by severe depression, agitation, aggressive behaviour, illusions, hallucinations, delirium etc. In these the person loses touch with reality and shows psychotic symptoms.

Neurosis

This is a mental disorder or a disease of the nerves. Any fault of inefficient way of coping with anxiety or inner conflict, usually involving the use of an unconscious defence mechanism, that may ultimately lead to a neurotic disorder. Ability to function is markedly impaired but behaviour generally remains within acceptable social norms. Perception of reality can be affected. Usually no proof of organic cause can be found.

Delirium

A syndrome generally produced by a transient disturbance of brain tissue function. It is a disturbance of consciousness in which (i) orientation is impaired (ii) Critical faculty is blunted or lost (iii) Thought content is irrelevant or incoherent. In the early stage, the patient becomes restless, uneasy and sleepless, loses self-control, and becomes excited. Although the level of consciousness is altered, the patient is generally awake, and able to answer questions. However his thinking memory, perception and attention are disturbed. Delirium may be followed by complete recovery in an acute brain syndrome. It may also progress to an irreversible amniotic syndrome, which may be defined as chronic brain syndrome leading to coma and death in which case the matter of deliberation seems irrelevant. It is found in cases of: (i) Trauma (ii) Anoxia (iii) Vascular infarction (iv) Toxic metabolic disorders (v) Drug abuse (vi) Inflammatory conditions (vii) Psychological disturbances (viii) Mental stress (ix) Overload of work.

Medicolegal Importance: Delirium is less often associated with hallucinations and delusions and

if the person commits crime then he is not responsible for his act.

Delusion

It is a false belief that arises without appropriate external stimulation in some thing, which is not a fact, and which persists even after its falsity has been clearly demonstrated. The belief held is not ordinarily shared by other members of the patients sociocultural and educational group. Delusions are pathognomonic of the psychoses. They occur in schizophrenia. They can be observed in all psychotic states including that of organic origin. A normal person can have delusion but he corrects himself by his reasoning capacity.

Types of Delusion

1. *Of grandeur or exaltation (Megalomania):* A man imagines himself to be very rich, though really he is a pauper. He thinks himself to be a king, a great personality, but he is a common man.
2. *Of persecution:* He imagines that attempts have been made to kill him by or his nearest relatives.
3. *Of references:* He thinks that people, things and events etc. refer to him in a special way. People are talking about him. Newspapers are writing about him.
4. *Of influence or of control (in schizophrenia):* He complains that his actions, thoughts are being influenced and controlled by some outside agency. Like radio, hypnotism and telepathy etc. seen in schizophrenia.
5. *Of infidelity:* A man imagines that his wife is unfaithful to him while she is chaste.
6. *Of Self reproach (self accusation):* The person reproaches himself for the past failure and misdeeds which are often of a trivial in nature.
7. *Of Nihilistic:* He declares that he does not exist or that there is no world.
8. *Hypochondria:* A person has a firm belief in his mind that there is something wrong in his body, though he is quite healthy.
9. *Paranoid delusions:* Over suspiciousness leading to persecutory delusions.

Medicolegal Importance: Delusions often affect the conduct and actions of the sufferer. The tendency of committing suicide and homicide etc. It is usually taken as defence.

During the examination of the mental condition of the person, the medical man should make and carefully note to which law gives much importance. In the beginning of a disease delusion may not be evident. The person can conceal it. Hence the doctor should examine or watch the person for days together. The person cannot be fully responsible for the act.

Hallucination

False sense of perception without any external object or stimulus to produce it. Hallucination is the apparent perception of an external object when no corresponding real object exists. A dream is a simple example of a hallucination in normal experience. It occurs in a number of conditions: (i) Schizophrenia (ii) Under the influence of drugs—L.S.D., Mescaline (iii) Temporal lobe lesions (iv) Generalized organic brain diseases (v) Small doses of alcohol and cannabis (vi) Brain tumour, Subarachnoid haemorrhage, uraemia etc. (vii) High fever.

Types of hallucinations:

1. *Visual:* In which the person may see himself being followed by a lion though no lion exists.
2. *Auditory:* In this the person listens to the voice and imagines that someone is talking to him, when no body is there.
3. *Olfactory:* In this the person smells pleasant or unpleasant when no smell is there.
4. *Gustatory:* The person tastes sweet, sour or salt etc. when there is no food in mouth.
5. *Tactile:* The person imagines that mice or rats crawling over him when practically no mice or rats are present, for example cocaine bugs.
6. *Kinesthetic/Psychomotor:* Feeling of movement of some parts of the body when they are stationary.
7. *Lilliputation or microptic:* Small size objects may appear very large or vice versa.
8. *Hypnagogic:* False sensory perception occurring midway between falling asleep and being

awake, drowsy state after deep sleep and before awakening.

Medicolegal Importance: Hallucination may be pleasant or unpleasant. Delusions arising from unpleasant hallucinations may cause the person to commit suicide or homicides. He is not responsible for the deeds.

Illusion

False interpretation by the senses of external object which has a real existence. Some of the examples include Persons feels a dog as a lion, rope as a snake, tree as a ghost. A sane person also have illusion, but he is able to correct the illusion but an insane cannot correct. Illusions of sight and hearing are very common but those involving other senses also occur. Illusions occur in insanity and other toxic states and the person is not responsible for his acts.

Obsession

In this a single idea or emotion is constantly entertained by a person in spite of all efforts of the sufferer to drive it from his mind. It is a disorder of thought and attempts to resist makes them more strongly insistant.

Examples of Obsession: (i) Closing the door and checking for the locks repeatedly at bedtime (ii) Counting the money again and again before going to the market (iii) A wife may continuously believe her husband to be unfaithful to her or vice versa (iv) Washing hands again and again.

Medicolegal Importance: (i) It is borderline between sanity and insanity (ii) Sane person also have obsession but he can correct it (iii) It is usually seen in nervous people

Impulse

Sudden and irresistible force compelling a person to the conscious performances of some action without notice or fore thought. A sane person has sufficient faculties to control the impulse. An insane person can not control. Usually seen in imbecility, dementia, acute mania etc.

Types

1. *Kleptomania*: Tendency to steal small articles
2. *Pyromania*: To set fire to things.
3. *Multinomial*: Desire to maim animals.
4. *Dipsomania*: Excessive desire to drink alcohol.
5. *Sexual impulses*: It is a form of sexual perversions by abnormal means to get gratification.
6. Suicidal or homicidal impulse.

Lucid Interval

This is period occurring during insanity when all the symptoms completely disappear and the person becomes normal. It appears in melancholic and mania. The differences between lucid interval of head injury and insanity is given below:

Head injury	Insanity
1. History of injury	1. History of mental disease
2. Preceded by a phase of concussion	2. Preceded by phase of symptoms of insanity
3. May be followed by phase of cerebral irritation and compression of brain	3. May be followed by phase recurrence of symptoms of insanity
4. Only once	4. More than once

Medicolegal Importance: The person is not legally responsible for the crimes done during lucid interval but it is very difficult to prove the same.

MENTAL HEALTH ACT—1987

This Act defines the criteria for admission and restraint of the mentally ill persons. As per this Act the words lunacy, insanity and unsoundness of mind have been replaced by the word mentally ill that has been defined as a person who is in need of treatment by reason of mental disorder other than mental retardation.

1. It provides the guidelines for establishment and maintenance of psychiatric hospitals and nursing homes.
2. It describes the procedure for admission and detention of mentally ill persons, in psychiatric hospitals and nursing homes.
3. It describes the provisions for inspection, discharge, leave of absence and removal of mentally ill persons from psychiatric hospitals or nursing homes.

4. It provides for judicial inquisition regarding possession of property by alleged mentally ill person, custody of the person, and management of his property.
5. It provides for the liability to meet cost of maintenance of a mentally ill person detained in psychiatric hospital or psychiatric nursing home.
6. It provides for protection of human rights of mentally ill persons.
7. It provides for penalties and procedure for awarding the same, for violation of the provisions of the Act.
8. It also provides for certain other miscellaneous aspects regarding lunacy and the lunatics.

Admission of Lunatics or Insane Persons in to Mental Hospitals or Psychiatric Nursing Homes**Part I : Admission as voluntary patient:**

S.15: Request by major for admission as voluntary patient - He requests the medical officer in charge of a psychiatric hospital and can get admitted in the same as a voluntary patient.

S.16: Request by guardian for admission of a ward (minor less than 18 years as voluntary patient). He requests the medical officer in charge of the hospital who gets the patient admitted.

S.17: Procedure to be followed

1. On receipt of request under S15 and S16 the medical officer in charge shall make such inquiry as he may deem fit within a period not exceeding 24 hours and if he is satisfied that the applicant (major or minor) requires treatment as an patient, he may admit him as a voluntary patient.
2. They will abide by the rules and regulations made by the medial officer in change.

S.18: Discharge of such voluntary patients.

1. The medial officer after receiving the request for the discharge by the major or guardian of minor, shall discharge him within 24 hours.
2. When minor voluntary patient is admitted in the hospital, attains majority he should be informed by medical officer and unless a fresh request is made by him, with one month of such intimation he shall be discharged.

3. When the medical officer is satisfied that the discharge will not be in the interest of the patient, constitute a medial board consisting of two medical officers and seeks its opinion, whether the patient needs further treatment and if the board feels that the patients needs further treatment, the medical officer will not discharge but continue the treatment for the period not exceeding ninety days at a time.

Part II: Admission under special circumstances

S.19: Admission under special circumstances

1. Mentally ill persons who does not or are unable to express willingness for admission may be admitted by the application made by relative or friends. The medical officer can admit if he is satisfied for the benefit of the patient but will only be kept for 90 days.
2. For application, a prescribed medical form alongwith two medical certificates in the support of the mental illness of the person should be enclosed. If the application is not accompanied by medical certificates and the medical officer incharge of the hospital may arrange examination of the person by two medial officer of the mental hospital and if findings of the two medial officers are supportive of mental illness of the person then he may admit the person in the hospital.
3. For the discharge of such patient, the relative or friend may apply to the magistrate. The magistrate after making inquiry and after giving notice to the person as he deems fit, either allow or dismiss the application.
4. The provisions of the section shall be without prejudice to the persons exercisable by a magistrate.

Part III: Reception order

S.20 Application for reception and detention order

1. An application of reception order may be made by: (a) Medical officer in charge of psychiatric hospital (b) Husband, wife or any other relative.
2. Medical officer, incharge of the hospital where the ill person is undergoing treatment under a temporary order is satisfied that
 - a. the treatment of mentally ill person is required to be continued for more than six months or

- b. it is necessary in the interests and personal safety of the ill person or for the protection of others, such person shall be detailed. He may make an application to the magistrate with local unit of whose jurisdiction the hospital situated; for the detention of the ill person under a reception order.

3. Husband and wife can make an application.
4. Where the relatives make an application In the case of 3 and 4 the application must be accompanied by reason must be mentioned.
5. No persons (a) who is a minor or (b) who has not been within 14 days of the date of application shall make an application
6. The application must be made with the prescribed form along with two medical certificates out of which one should be of the Govt. Doctor.

S.21: The medical certificate shall contain:

- a. Each medical practitioners has examined the ill person independently and the opinion is given on the basis of own observation.
- b. The ill person is suffering from mental disorder of such a nature and degree that he requires detention in a hospital. It is needed in the interests of health and safety of the ill person and for the protection of others.

S 22: Procedure upon application for detention

1. On receipt of application the magistrate may make an order, if he is satisfied that: (a) The mentally ill person is suffering from such a nature and degree that he requires detention in a hospital. (b) It is in the interest of the ill person or for the protection of others and a temporary treatment order would not be adequate in the circumstances, and it is necessary to make a reception order.
2. On receipt of application, magistrate consider the statement of the certificate and form.
3. If the magistrate considers that there are sufficient to proceed further he shall personally examine the ill person unless for reasons to be recorded, he thinks that no necessary.
4. If he is not satisfied with the application he may fix a date for hearing of the application and may make injurries as he thinks fit.

5. The notice of the date fixed should be given to the applicant and any other person, as he thinks fit.
6. He may make an order for the proper care and custody of the alleged mentally ill person pending disposal of the application.
7. The magistrate shall consider the application in camera in the presence of the (a) Applicant (b) Mentally ill person (c) Person who may be appointed by the mentally ill person (d) Any other person as the magistrate thinks fit.
8. If magistrate is satisfied, he may pass a reception order and if not satisfied may dismiss the application with reasons and one copy is given to applicant.

S. 23: Powers and duties of police officer in respect of the mentally ill persons

1. Every officer in charge of police station may take: (a) Into protection any person found wandering within his jurisdiction, he has reason to believe that he is no mentally ill that he can not take care of himself and (b) Whom he has reason to believe to be dangerous by reason of mental illness.
2. The police officer must inform the grounds of the protection to the mentally ill person or relatives or friends if the mentally ill person can not understand otherwise he can not detain.
3. He must produce the mentally ill person within 24 hours before the nearest magistrate excluding the time necessary the journey he can not detain more without the authority of the magistrate.

S. 24: When produced before the magistrate by the police and in the opinion of the magistrate, there are sufficient grounds for proceeding further: He

1. Examines the person.
2. Cause him to be examined by a medical officer.
3. May make necessary inquires and after completion of proceeding, may pass a reception order (a) if the medical officer certifies that he is a mentally ill person (b) magistrate himself is satisfied that for the care of the ill person & protection of others such order is necessary.

- If the relative or friend wants to the send the mentally ill person to a particular licensed hospital and undertakes a bond to pay the expenses, with the consent of the doctor of that hospital admit there.
- If any relative or friend enters into bond that they will take care of the mentally ill person so that there is no danger to himself or to others so instead of making a reception order, hand over to the relatives.

S. 25: Order in case of mentally ill person cruelly treated or not under proper care and control

1. The police officer of the area reports the facts to the magistrate.
2. Any private person who has reason to believe the above fact may report to the magistrate.
3. Magistrate may cause the mentally ill person to be produced before him, and summons such relatives or other persons to ought to be in charge of that person.
4. If the person is legally bound to take the charge of that mentally ill person, pass an order to there care of, its cases of non-compliance of the order, fine up to Rs. 2000/- may be imposed
5. If no person legally bound there, he may proceed under S.24 pass an reception order.

S. 26: Admission as in patient after inquisition: Any district court holding an inquisition under chapter VI is of the opinion that in the interests of such person by order direct the admission into hospital as in patient. Every such order may vary from time to time or revoked by the district collector.

S.27: Admission and detection of mentally ill prisoners. They can be admitted into psychiatric hospitals or nursing homes after an order for reception.

S.28: This section provides for detention of the altered mentally ill person under proper medial custody pending receipt of medical report, the period of such detention should not exceed ten days at a time and 30 days in aggregates.

Discharge of Mentally ill Person

S.40: Order of discharge by medical officer in change: Not without-standing anything in chapter

IV the medical officer in charge, on the recommendations of two medical practitioners, one must be a psychiatrist discharge any person except voluntary patients and mentally ill prisoners.

S.41: Discharge of mentally ill persons on application: When any application is received to discharge, by the person on whose application the admission was made, the medical officer may discharge the patient except if he is satisfied that the person is dangerous and unfit to be at large.

S.42: Order of discharge on the undertaking of relatives or friends for due care. They will furnish a bond to take care and to prevent harm to others.

S.43. Discharge of person on his request Any ill person except prisoners will (i) Make an application to the magistrate (ii) It should be supported by a certificate from a psychiatrist. The magistrate after enquiry if satisfied then discharge the person.

S.44: Discharge of person subsequently found on inquisition to be sound mind: Medical officer in charge on production of a copy of such findings duly certified by district court discharge such person.

CRIMINAL RESPONSIBILITIES OF INSANE

The plea of insanity is taken:

1. In **bar of trial** when the accused is insane and cannot plead.
2. In **bar of conviction** of the accused was insane when the crime was committed.
3. In bar of **infliction of capital punishment** when a condemned prisoner is insane.

If insanity is established, the accused person is found "not guilty" and is kept in an asylum. The law presumes that every person is sane and responsible for his action, the proof that it is not so has to be produced by the person who set up a defence. The law also presumes that for every criminal act there must be a criminal intent or mind (*Mens = mind, rea = criminal*).

The doctor who examines, gives opinion that the person is sane or insane when it is taken as a defence in homicidal cases, the following points may be taken into consideration.

1. *Family history* of the disease, CNS disorders, chorea, epilepsy etc.
2. *Personal history:* Drugs, alcohol, sexual history, occupation, habits. Psychogenic factors like emotional conflict, repression and anxiety states should be noted.
3. *Physical examination:* dressing, walking, cleanliness, pulse, BP is raised in insane, tongue is furred and coated, skin is dry and wrinkled.
4. *Mental condition:* The general appearance has to be noted, the person appears dirty, improperly dressed and may be naked. Behaviour, co-operation, abnormal conduct, memory, power of recovery and judgment should be noted. Sleep disturbances, walking and gait, attitude and posture, altered sexual behaviour incoherent speech etc.
5. *Hand writings:* Shows mental confusion, misspellings omission of letters.

Following features should be taken into account if an act is the result of insanity (i) History whether melancholic, schizophrenic etc. (ii) Absence of motive (iii) Absence of secrecy (iv) Multiple murders (v) Want of pre-arrangement (vi) Want of accomplices.

McNaughten's Rule (Legal test or right or Wrong Test)

English courts dealing with responsibilities in mentally ill persons in criminal cases follow McNaughten's rule laid down in 1843. Daniel McNaughten a 29 year old Scotsman and a paranoid schizophrenic believed that the Prime minister of England Robert Peel was conspiring against him. He believed that the spies sent by the catholic priests with the support of the Tories Prime Minister were constantly following him, harassing him and hatching a conspiracy against him. He decided to kill the Prime Minister of England and made elaborate preparations for executing his plan. He heard voices accusing him of crimes which he claimed he never committed. But he shot Edward Drummond the Private secretary to Prime Minister,

Table 33.1: Differences between real insanity and feigned insanity

Features	Real insanity	Feigned insanity
1. Onset	Gradual	Sudden
2. Motive	Nil	Present of crime
3. Predisposing factor	Present	Absent
4. Signs and symptoms	Uniform	Present only when being observed and conscious
5. Facial expression	Peculiar vacant look	No peculiarity, frequently changing
6. Insomnia	Present	Absent
7. Exertion	Can stand exertion	Cannot stand exertion and fatigue
8. Habits	Dirty	Not so
9. Skin and lips	Dry	Not dry, normal
10. Frequent examination	Does not mind	Resists for fear of detection

by mistake. McNaughtan was acquitted as a result of medical evidence. Public reaction to this was most unfavourable. The debate took place in the House of Lords, and a number of questions were put to a bench of 15 judges but it was established that he was insane and acquitted. He was later sent to Bethlem Mental Hospital for the rest of his life.

To establish the 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 mind that he did not know the nature and quality of the act he was doing or he did not know that he was doing wrong.

In India the same law has been incorporated in **Sec. 84 IPC** which reads as: "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 the law".

Comments: It must be clearly proved that: (i) The offence is directly related to the insanity (ii) The offence could not have occurred if there was no mental abnormality (iii) Insanity subsequent to the act is not a defence.

Defect of reason: It must be clearly established that the reasoning powers of the accused were not functioning normally due to defect in intellectual and cognitive faculties. The nature and quality of act should be judged from the physical characters of the act but not from the moral aspects.

Wrong: It means punishable by law. It concerns with the defendant's ability to distinguish between right and wrong. If he had the capacity to know that his act was wrong he will be fully responsible even if he was mentally ill and unable to refrain himself from doing the act.

Examples:

1. If he thinks another man is going to kill him and he kills that man in self defence then it's not Punishable.
2. He thinks another to be a wild animal and kills him then there is no responsibility.
3. If he thinks of himself as a state executioner there is no responsibility.
4. Thinks himself as a king and his prerogative to execute other as a criminal, but he is not responsible.
5. If under insane delusion he thinks that another person has inflicted serious injury to his character and kills him, he is responsible because one cannot take revenge.

Defects: For deciding that a person is insane only intellectual factors (reason) are taken into consideration but the (i) emotional factors (ii) delusional beliefs (iii) ability of the individual to control the impulse (resistible impulse) are not considered.

Charles Freeman was the Heroin pusher and the Court found him guilty. The defense was that Freeman might have known that what he was doing was wrong. But had neither the capacity nor the will to be responsible for his acts and the court of appeal reversed the decision (Justice Irving Kannan).

Durham Rule 1954 (Monte Durham)

Monte Durham a 23-year-old who had been in and out of prison and mental institutions since he was 17 years old. He was convicted for housebreaking in 1953, and his attorney appealed. Although the

district court judge had ruled that Durham's attorneys had failed to prove that he didn't know the difference between right and wrong, the federal appellate judge chose to use the case to reform the *McNaughton* rule.

Citing leading psychiatrists and jurists of the day, the appellate judge stated that the *McNaughton* rule was based on "an entirely obsolete and misleading conception of the nature of insanity." He overturned Durham's conviction and established a new rule. The *Durham* rule states "that an accused is not criminally responsible if his unlawful act was the product of mental disease or mental defect."

The *Durham* rule was eventually rejected by the federal courts, because it cast too broad a net. Alcoholics, compulsive gamblers, and drug addicts had successfully used the defense to defeat a wide variety of crimes.

Advantages: (i) It was broader than Mc Naughten's rule (ii) Widely acknowledged by the psychiatrists.

Disadvantages: (i) Deciding whether the act was the product of mental disease or defect is very difficult, and almost impossible at times (ii) No definitions were given for these terms in the court (iii) It gave the psychiatric a blank cheque and hardly gives court any opportunity to render a judgment i.e. rendering an independent social and legal judgment (iv) It was abolished in 1972.

The Federal Rule

In 1984, Congress passed, and President Ronald Reagan signed, the Comprehensive Crime Control Act. The federal insanity defense now requires the defendant to prove, by "clear and convincing evidence," that "at the time of the commission of the acts constituting the offense, the defendant, as a result of a severe mental disease or defect, was unable to appreciate the nature and quality or the wrongfulness of his acts". This is generally viewed as a return to the "knowing right from wrong" standard. The Act also contained the Insanity Defense Reform Act of 1984, which sets out sentencing and other provisions for dealing with offenders who are or have been suffering from a

mental disease or defect.

Curren's Rule 1961

An accused person is not criminally responsible if at the time of committing the act he did not have the capacity to regulate his conduct to the requirements of law, as a result of mental disease or defect.

Irresistible Impulse Test

An accused person is criminally not responsible even if he knows the nature and quality of his act and is aware that it is wrong, if he is incapable of restraining himself from committing the act; because of the controlling agency or capacity has been destroyed by the disease. It is never used as a sole test for insanity.

American Law Institute Test 1970 (ALI)

Criminal responsibility as defined in ALI's model penal code 1970: "A man is not responsible for criminal conduct if at the time of such criminal conduct, as a result of mental disease or defect he lacks substantial capacity either to appreciate the wrongfulness of his conduct to the requirements of law." However repeated criminal conduct does not alone prove such abnormality (i) This accepts the theory of combined Mc Naughten and irresistible impulse test. It takes account of the impairment of volitional capacity and impairment of cognitions (ii) It is broader than both Mc Naughten and Irresistible impulse test (iii) It demands that impairment be complete (iv) Instead of knowing the difference between 'right and wrong' the defendant is subject to the subtler requirement of appreciating it (v) Instead of proving the act like "product of disease it has to be only shown that disease resulted in a loss of substantial capacity to obey the law.

Doctrine of Partial Responsibility

Certain types of mental disease like psychopathic personalities and paranoia in which criminal acts are committed. From the legal point of view they are responsible for the act. At the same time from medical or psychiatric angle, such a person is of unsound mind. So if those persons commit any

crime, they should be considered partially responsible. In England and some states of USA it is not accepted as defence. However, in Illinois (USA) psychopaths may be declared insane and they are treated in the hospital. In Scotland it is taken as a defence and the charge of murder is reduced to Manslaughter.

Homicide Act 1957 (England)

Which brings the conception of diminished responsibility and the mental health Act 1959 which defines the psychopathic personality as a persistent mental disorder or disability of mind which results in abnormality aggressive or seriously irresponsible conduct on the part of the patient, requires or susceptible to treatment, for which he may be compulsorily detained in the hospital. The defence took the plea of diminished responsibility in the ground of sexual psychopath, who was unable to control the desire. Appeal was allowed and the charge for murder was converted into Manslaughter. It may be considered for diminished responsibility.

Somnambulism

It means walking during sleep. A person leaves his bed and walks in the house or out of his house without any awareness of his actions but rarely injures himself. He is not asleep, but in a state of dissociated consciousness a hallucinatory state, unrelated to his immediate environment, similar to automatism. Such persons are well adjusted in life are socially well behaved and not aggressive. Immediate stress and strain, mental conflicts etc. are present prior to walk. Crimes are not willful or premeditated. Mental faculties are partially active and concentrated on a particular idea. So he may commit a crime and even solve a difficult problem but he has no recollection of the events. In some cases the events of one fit are remembered in a subsequent fit. Such persons when commit a crime are not criminally responsible.

Somnolentia

Often called sleep drunkenness and midway between sleep and walking. If such a person is

suddenly aroused from deep sleep, he may commit some crime, due to confusion of mind, if he is having a dream at that time. He is not criminally responsible for the crimes.

Drunkenness: The laws relating to drunkenness are laid down in S 85, 86 IPC. Drunkenness caused by voluntary use of alcohol or some other intoxicating drugs is no excuse for the commission of a crime but insanity produced by drunkenness, voluntary or otherwise, absolves one of criminal responsibility, if it can stand the usual legal tests applied in other forms of insanity. In cases where the intention of a person committing an ingredient of the crime itself is present then simply being drunk is of no defence. In *King Emperor Vs Bishan Singh*, the accused was charged with having murdered 3 persons by firing a gun in a state of intoxication. The court held that the accused was not in such an advanced state of intoxication as not to be fully aware of the act and held him responsible.

CIVIL RESPONSIBILITIES OF THE INSANE

Management of property: On application by advocate general or a relative or friend, court may direct to have a inquiry whether the person is of unsoundness of mind and incapable of managing himself and his property. The court has to know (i) nature of his property (ii) period of insanity (iii) name of relatives or friends (iv) any other relevant information. Then court fixes a date for medical examination and the psychiatrist certifies that: (i) The person is insane but the insanity is of such a degree to render him incapable in managing the property (ii) He is in sense but capable of managing the property (iii) In doubtful cases, opinion is given in favour of sanity.

When court is satisfied, that he is not capable it appoints a manager for the purpose the manager has to immaterial whether the vendor knew or not, but it is not responsible if the order is very much extravagant or if the vender has taken the advantages of his insanity. (i) give the account of the whole property (ii) submit the annual account within 3 months (iii) removal/fine upto 500/-.

The court grants certain powers to the manager. He will not sell mortgages or gift any immovable property exceeding 5 years without prior permission of the court.

The court may order to sell or raise money for the following purpose: (i) To pay lunatic's debts (ii) The discharge of encumbrance on his property (iii) Payment of debt or expenditure for the lunatics maintenance (iv) Provision for future maintenance and members who are dependent upon him (v) Payment of cost of judicial inquisition and other court expenditure.

If it is subsequently reported that the lunacy has ceased second inquiry is held and necessary orders are passed.

Contracts: Under S 12 of Indian contract act a contract is invalid, if one of the parties was insane at the time of making the contract, they were unable to understand the nature of the contract. Lunatic is however responsible to pay the things he has purchased in accordance with his social position and status. A sane person, who occasionally becomes insane can make a contract when he is sane not vice versa. When much became insane after making the contract the Court orders for the fulfillment. Court orders for the dissolution of partnership of firm

Marriage: Declared null and void if it existed at the time. But developed subsequently is not a ground for divorce. Mere weakness of mind or eccentricity are not sufficient to avoid marriage contract.

Evidence: According to section 118 I.E.A, a lunatic is not competent to give or depose evidence. During lucid interval he can give evidence. The judge decides whether if he is competent.

Consent: Section 90 I.P.C. provides that consent of an insane to a certain acts is not valid:

1. *Consent for sex:* Although consented to sex, would amount to charge of rape, abetment of suicide or grievous hurt.
2. Exception 5 to S 300 of I.P.C. provides that culpable homicide is not caused when the person whose death is case of being above 18

year suffers the death or take the risk of death with his own consent.

3. According to section 87 I.P.C, it is an offence when it is inflicted by the person with consent the age is above 18 years.
4. According to section 305 I.P.C, abetment of suicide of insane person is punishable with death or life imprisonment for 10 years and fine.

Testamentary Capacity: Testamentary capacity is the capacity to made a valid will. The following conditions to be fulfilled.

1. An understanding regarding nature of will.
2. Knowledge of the property to be disposed of.
3. Ability to recognize those who may have normal claims of the testator's bounty. A civil may court invalidate the will if the party was not of sound and disposing mind at the time of making the will. The medical officer should clearly examine the person and give opinion. If he thinks that he is under the influence of some body, he should be encouraged to tell the truth. It is not a valid will if under the influence. During lucid interval if he makes the 'will' it is valid. During fit of drunkenness it is valid if the linder stands the nature of will. In extreme ages, the mental condition is seldom normal and the person is most of the time suspicious. The will is valid if the person commits suicide after making it and no other abnormalities are present.

Long Question

1. Discuss the civil and criminal responsibilities of insane
2. Discuss the ways to restrain an insane person.

Short Questions

1. Delirium
2. Delusion
3. Illusion
4. Hallucination
5. Obsession
6. Lucid interval
7. Mental Health Act 1987
8. True and feigned insanity
9. McNaughten's rule
10. Impulse

MULTIPLE CHOICE QUESTIONS

1. 'Lucid Interval' may be seen in:
 - A. Intracerebral haemorrhage
 - B. Insanity
 - C. Subdural haemorrhage
 - D. Alcohol
2. McNaughten's rule is concerned with:
 - A. Civil responsibility in drunken person
 - B. Criminal responsibility in insane person
 - C. Professional misconduct by doctors
 - D. Capacity of a person to make a valid will
3. Under I.P.C., a person below years is not criminally responsible:
 - A. 5
 - B. 7
 - C. 12
 - D. 16
4. All are features of 'feigned insanity', except:
 - A. Sudden onset
 - B. predisposing factors present
 - C. Motive present
 - D. Insomnia cannot persist
5. Which of the following is not seen in 'feigned insanity':
 - A. Sudden onset
 - B. Uniform symptoms
 - C. Remission of symptoms when unobserved
 - D. Resents frequent examination
6. McNaughten's rule refers to:
 - A. Person is not responsible if he is not of a sound mind
 - B. Person is held responsible even if he is not of sound mind
 - C. Person is always held responsible
 - D. Any of the above
7. Lucid interval is seen in cases of:
 - A. Intracerebral haemorrhage
 - B. Insanity
 - C. Opium poisoning
 - D. Chronic alcohol poisoning
8. Mc Naughten's rule is for:
 - A. Civil responsibility
 - B. Testamentary capacity
 - C. Criminal responsibility
 - D. Capacity of the witness
8. Norwegian system deals with:
 - A. Blood grouping
 - B. Identification of blood stains
 - C. Identification of semen
 - D. Criminal responsibility
9. Kleptomania is a type of:
 - A. Obsession
 - B. Delusion
 - C. Illusion
 - D. Impulse
10. The irresistible desire to set fire to things is known as:
 - A. Kleptomania
 - B. Pyromania
 - C. Dipsomania
 - D. Mania
11. The Mental Health Act was enacted in the year:
 - A. 1971
 - B. 1976
 - C. 1980
 - D. 1987
12. According to law an insane person can>
 - A. Testify in court
 - B. Enter a contract of marriage
 - C. Manage his own property
 - D. None of the above
13. Feigned insanity is
 - A. Gradual in onset
 - B. Without any motive
 - C. Insomnia is absent
 - D. Has dry and cracked lips and skin
11. In real insanity all are present except
 - A. The person has dirty and filthy habits
 - B. Has a vacant expression
 - C. Signs and symptoms are uniformly present
 - D. Resents frequent medical examination
12. All the following are true about psychosis except:
 - A. It is a reaction to stressful circumstances
 - B. Empathy is absent
 - C. The person loses touch with reality
 - D. Insight is present

Starvation Deaths

Starvation is a state that occurs from withholding the food or administration of unbalanced food. Starvation can be acute or chronic. The acute starvation occurs from the sudden and complete stoppage of food but chronic starvation occurs from the gradual intake of deficient food. The starvation deaths may be intentional or accidental and even homicidal. The usual causes are famine, neglect on the part of the guardians or parents, willful refusal to take food like in case of hunger strike, the passengers during shipwreck or landslides if they are stranded and there is deficient supply of food.

Clinical Features

Acute starvation

The sign and symptoms usually appear after 12-16 hours of stoppage of food. There is a hungry feeling followed by epigastric pain, which is relieved by pressure. The absorption of subcutaneous fat starts after 5-6 days resulting in general emaciation. Therefore, the eyes are sunken, pupils are mildly dilated, the cheeks sink and the body prominences become more visible. The lips are dry and cracking is present, the tongue is coated and the person feels thirsty. There is salivation, voice becomes weak, the skin is dry becomes rough and pigmented at places. After a weeks time the muscles become weak. On physical examination the pulse becomes slow, the temperature is subnormal and constipation is the usual feature. The urine becomes scanty and evidences of acidosis are found. As time passes the body is

reduced to a skeleton, the thoracic cage becomes more prominent. The loss of 40-45% of body weight is fatal. In these cases the intellect remains clear. But in some cases the person experiences delusions, and at times hallucinations.

Chronic Starvation

The features of chronic starvation are:

1. The patient complains of hunger pains relieved by pressure, he becomes lethargic and there is disorientation. There is oliguria.
2. Evidence of pigmentation and the patient becomes anemic
3. Subnormal temperature leading to hypothermia
4. Edema of the feet and limbs
5. There is risk of infection like diarrhea, dysentery
6. The death occurs due to exertion, cardiac failure and chronic absorption.

Fatal Period

The fatal period if food is completely withdrawn then death occurs in 7-10 days. If the food alone is withdrawn death may prolong 8-10 weeks or even more. If there is complete starvation without intake of both food and water then death occurs in 10-12 days. If water is continued then death may occur in 6-8 weeks or more. 80-90% body fat should be lost along with 20-25% body weight should be lost for death to occur. However the following factors may modify the fatal period.

1. The young and the old die earlier than the adults
2. The females they tolerate starvation for a longer period than the males.

3. Fatty and healthy people tolerate better than the weak.
4. Persons with active physical exertion die earlier

Cause of Death

Death occurs from exhaustion, circulatory failure due to brown atrophy of the heart or intercurrent infection.

Postmortem Appearance

All organs and tissues show changes similar to premature senility. There is extreme emaciation and a reduction in the size and weight of all organs except brain. The brain can become soft and pale. Muscles get atrophied and become dark in colour as the muscle fibers lose the striations due to granular degenerations. The fat of orbits and female breast is the last to go. Fat is absent from the subcutaneous tissues and almost completely lost from the mesenteries. Subepicardial fat is replaced by a gelatinous material. In acute starvation subcutaneous body fat is not lost completely. In children, the skeleton shows spinal curve, rickets and dental defects. In adults progressive demineralization and osteomalacia are seen. Stress fractures may be present and the skin is lusterless and loses its elasticity. The face becomes pale. Ascites may be present in some cases. Hairs are also dry and lusterless and the nails are dry and brittle. The lungs are pale and collapsed and exude very little blood on cutting. The stomach and intestine are atrophied and are stained with bile. The spleen is shrunken and the gall bladder is distended with bile. The kidneys show atrophy of the nephron. Blood volume is reduced, and there is severe anemia. The urinary bladder is also

empty. In wasting diseases the gall bladder is empty in contrast to death due to starvation.

Medicolegal Aspects

The diagnosis of starvation is done on the basis of history and other general findings. But at the same time one has to exclude the wasting diseases like malignant diseases Addison's disease, diabetes mellitus, tuberculosis, even pernicious anemia and sometimes chronic diarrhea. So in the absence of above diseases diagnosis is usually made in favour of starvation.

Suicidal: Some persons have to voluntarily starve for the purpose of fulfillment of their grievances. At times the lunatics and hysterical women may refuse to take food. Rarely fasting is undertaken to attract the public attention. Right to life is guaranteed under article 21 of the constitution of India, which does not include the right to die. So forcible feeding of these persons are lawful.

Homicidal: The victim is usually an infant or an aged person. At times the lunatics or mentally deranged persons also deprived of their food resulting in chronic starvation and finally death.

Accidental: Accidental starvations may occur during famine or being trapped in landslides or shipwreck. There is failure to provide food due to ignorance of the people. Certain diseases produce stricture of the esophagus resulting in intake of insufficient food.

Long Question

1. Define starvation. What are the postmortem findings in a death due to starvation?

MULTIPLE CHOICE QUESTION

1. In acute starvation:
 - A. Feeling of hunger lasts around 10 hours
 - B. If both water and food are withheld death occurs by 6-8 weeks
 - C. Both are true
 - D. Both are false

1 D

General Toxicology

Historical Perspectives

The use of poisons dates back as far as spiritual and mythological beliefs have been recorded. Perhaps the first accounts deciphered are from the Sumerians of Mesopotamia, the modern day Iraq. Here there are associations of poisons with 'Gula' who was regarded as a spirit or 'the mistress of charms and spells'. This dates back to around 4500BC and tabulated accounts of her have been found from about 1400 BC. Even in Greek mythology there is reference to poison, although no apparent citation to specific poisons used exists. An example is that of Medea, one of the children of the sun. She became the wife of Aqueus, king of Athens. Aqueus' son Theseus, returned to Athens to claim his rights and Medea, according to myth, resented this and vainly attempted to poison Theseus with a poisoned goblet.

Records of Egyptian knowledge can be dated about 300BC. Menes, the earliest recorded Egyptian king studied the properties of poisonous plants. Detailed accounts at this time were not recorded as it was forbidden to reveal any secrets taught in the temples. Exposing these mysteries carried the penalty of death. There is however sufficient evidence from various papyri that the Egyptians were conversant with antimony, arsenic, copper, lead opium and mandrake etc. also the Egyptians were probably the first to master distillation and discovered how to extract a powerful poison from peach kernels. A translation by Duteuil on a papyrus in Louvre shows the earliest recording of a preparation of a substance for lethal purposes. Today this extract is known as prussic acid (cyanide). Also peach kernels contain 'cyanogenic glycosides' that release toxic substances

in the presence of water. The ancient Greeks knew of arsenic in the form of realgar and orpiment, metals such as lead, mercury, copper, silver and gold and some of their properties. Amongst vegetable poisons, the Greeks chiefly employed Hemlock, for suicidal purposes and as State poison as a form of capital punishment. Socrates was found guilty of corrupting the youth of Athens with his philosophical teachings and in 402BC was made to drink the State poison that is Hemlock.

The knowledge of poisons appears to have been more general among Eastern races. The Persians were very interested in the art of poisoning (405-359BC) Queen Parysatis supposedly poisoned her daughter-in-law, Statira by means of a poisoned knife. Poisoning at the dinner tables was certainly not uncommon, especially in ancient Roman times. The Chinese around 246BC adopted a custom, which still exists today is the Chou Ritual. They used 5 poisons of which four are known; cinnabar (mercury), realgar (arsenic), green vitriol (copper sulphate) and loadstone. They are burnt together and the fumes are used on a bunch of features that is to be used externally.

Now, the people started looking towards methods of prevention of fatal effects of the poisonous substances. Mithridates, the King of Pontus (Turkey) around 114-63 BC studied the subject of antidotes extensively. He tested various poisons on condemned criminals. During these ancient times, poisons were essentially viewed as 'mysterious' substances, and were poorly understood.

Even in the relatively unscientific world of medieval Europe, there was a remarkable amount of knowledge about the effects, uses and treat-

ments of poisonings. Poisoning was a popular subject of fiction along with the academic texts published on this subject. The monks often wrote these, as monasteries were the main seats of learning in a largely illiterate population. Magister Santes de Ardoynis wrote 'The Book of Venoms' in 1424 that had comprehensive account of poisons known at that time. Many of the poisons such as arsenic, atropine, opium, cyanide, strychnine and heavy metals were described in the treatises.

Poisons and their effects were being studied predominantly to create a more virulent effect by the 14th and 15th centuries. Italian alchemists realized that combinations of known poisons could create a more potent effect. By the 17th century, poisoning had become such an art that there were schools in both Venice and Rome. By 1589, even a publication was made on the art of poisoning. The most notorious poisoner of the 17th century, a woman named Toffana had a recipe named 'Aqua Toffana' that aided the murder of over 600 people, usually husbands. In Rome in 1659, a society of women was formed in secret; meeting regularly and the members were issued with the poison they required and the instructions for its use. In the latter part of the 16th century, the popularity of homicidal poisoning spread from Italy to France, where those practicing criminal poisoning became known as the 'French School of Poisoners'. In 1625, Louis XIV decided to limit the sale of poisons and passed a decree forbidding apothecaries to sell arsenic, sublimate or any other poisonous drugs except to persons known to them, and required purchasers to sign a register stating why they needed the substance. At this time Catherine Deshayes, a famous poisoner sold poison to many women wishing to rid themselves of their husbands. During the late 18th century, an important case of poisoning in Britain was that of Sir Theodosius Boughton, whose body was exhumed and found to be impregnated with what appeared to be arsenic. Mathieu Orfila is considered to be the father of toxicology, having given the subject its first formal treatment in 1813 in his *Trait des poisons*, also called *Toxicologie generate*. The late 19th century is often associated with poisoning epidemic. To try to improve the

way in which poisoners were caught and convicted the science of toxicology became increasingly important in the field of forensic medicine. However, as science progressed throughout the 19th century, so did poison detection (tests for arsenic—Marsh test in 1836 and the Riensch test in 1841) and the science of toxicology grew in importance and reliability.

During the 20th century, the source, preparation and administration of poisons were stripped down; each process became cleaner, more attenuated and intellectually mastered. Poisoning in the 20th century grew massively because the man was evolving and discovering new things. Industry, agriculture, petro-chemistry, chemical warfare and pharmacology are but a few areas where hugely toxic agents were being put to use, often without the knowledge of their possible effects on life. Due to toxicological studies, many formerly hazardous occupations are now controlled and present minimal risk. For example, exposure to substances such as lead, mercury and aniline is now strictly controlled. Pharmacological disasters such as the use of thalidomide as a sedative during pregnancy in the 1960s, led to the development of teratology and a greater understanding of potential in-utero poisons. Arsenic remained a favourite for homicides in early part of the century. Cyanide too was popular around the middle of the century. The poison found a niche during the Second World War, and was used in liquid form as a suicidal tool for tortured agents of the resistance. Homicidally, cyanide enjoyed a resurgence of popularity in the latter half of the 20th century, with most of the poison secreted in food, drink and pharmaceutical products. Ricin, called the political poison of the 20th century became famous in the UK as a result of the 'Umbrella assassination' of Georgi Markov in 1978, who died several days after he was stabbed by the point of the umbrella. At autopsy it was found that at the site of injury a device containing Ricin was implanted. Thallium, a true 20th century poison is found in pest control substances. It kills by disrupting the sodium potassium ATPase pump and thus causes cell depolarization.

In ancient India, women to get rid of their oppressive husbands used aconite, arsenic and

opium. When we have a look at the history of ancient India, the literature describes various instances of poisoning. These include the description in Mahabharata of Bhim Sen being poisoned by Duryodhana.

In India, poisonous substances are used to commit suicide, or homicide. It can also be accidental more commonly in the Children who may ingest the poison in lieu of the sweets. However, suicidal and homicidal poisoning is quite common in this country compared to the west.

These days the use of the chemicals for domestic and industrial purposes accounts for an increase in number of accidental poisonings. The use of quack remedies for a quick relief and the injudicious use of Ayurvedic and *desi* medicines also result in accidental poisoning very often. Poisoning is the fourth most common cause of accidents in children. Children less than 5 years of age and adolescent are prone to poisoning. Accidental ingestions are most common in children less than 5 years old. The pattern of misuse of substances is changing these days with increase in number of intake of pesticides and insecticides that is easily available. In India the most common agents for poisoning are the pesticides such as the organophosphates, carbamates and pyrethroids etc. Besides the pesticides, sedatives, corrosives and alcohol are also commonly used. Also plant toxins are rarely used in the rural India. In the north India, aluminium phosphide is most commonly used to commit suicide as it does not have any antidote and is highly fatal. Poisoning is the fourth most common cause of accidents in children. Children less than 5 years of age and adolescent are prone to poisoning. The problem of illnesses and deaths is getting worse with the emergence of new drugs and chemicals. The properties, mechanism of action, lethal dose, detection and management of various poisonings will be discussed in the next few chapters.

Forensic Toxicology

Forensic toxicology deals with medico-legal aspects of the harmful effects of the chemicals on the human beings. It combines the basics of toxicological principles with analytical chemistry.

Toxicology

Toxicology is the science dealing with the toxicity of substances. The word *toxicology* is derived from the Greek word 'Toxon' that refers to a bow used for shooting arrows. The word 'toxeuma' meant an arrow, and 'toxicos' is consigned to such a poison that in ancient times was often used on tips of arrows to produce a more lethal weapon. The substance inflicting toxic effect can be any drug, or any organic or chemical substance in the environment. Toxicology deals with the properties, actions, toxicity, lethal dose, symptoms, mechanisms, diagnosis, estimation and treatment of the poisonous substances.

Toxinology

Toxinology is the specialized area of toxicology that deals specifically with biological toxins, such as venoms or poisonous plants.

Poison

A poison is a solid, liquid or gaseous substance, which if introduced in to the living body, or brought in contact with any part produces ill effects or death by its local, systemic or both types of action. This definition is unsatisfactory as a substance in particular dose causes beneficial effects whereas in the same dose it can cause the toxic effects. Drugs that are used for the treatment of diseases in higher doses will produce toxicity. As **Paracelsus**, the father of modern toxicology said, "All things are poison and nothing is without poison. It is the dose that determines the poison". Thus every drug is potentially a poison when used erratically. **Paracelsus**, the 16th century toxicologist was the first person to explain the dose response relationship of toxic substances. In Indian law, there is no definition of 'poison' and the various expressions are for it. In biology, poisons are substances that cause injury, illness or death of organisms usually by a chemical reaction or other activity on the molecular scale. Some poisons are also toxins, usually referring to naturally produced substances that kill rapidly in small quantities such as bacterial proteins that cause botulism and tetanus. Animal toxins that

are delivered subcutaneously (e.g. by sting or bite) are called venom. A poisonous organism is one that is harmful to consume but a venomous organism uses poison to defend itself while still alive. A single organism can be both venomous and poisonous. Deliberate application of poison has throughout the ages been used as a method of homicide, suicide and execution. As a method of execution, the poison can be administered as ingested, as the ancient Athenians did, breathed, such as carbon monoxide or hydrogen cyanide or taken intravenously. Many substances are regarded as poisons are toxic only indirectly such as methyl alcohol, which is not poisonous itself, but is chemically converted to toxic compound formaldehyde in the liver. Many drugs are made toxic in the liver, and the genetic variability of certain liver enzymes makes the toxicity of certain compounds differ between one individual and the next. Exposure to radioactive substances can produce radiation poisoning.

LD50—It is the dose of a toxic substance that kills 50% of a test population (typically rats or other surrogates when the test concerns human toxicity)

Types of Poisoning

The poisoning can be acute, subacute or chronic.

Acute poisoning: In acute poisoning, whatever amount of poison is taken and the toxicity is manifested immediately

Chronic poisoning: Small amount of poison are repeatedly administered for a long period producing the toxic effects. The substances commonly causing chronic poisoning are metallic poisons, opium, antimony etc.

Subacute poisoning: In this the poisoning manifests between the acute and the subacute types of toxicity.

Mechanism of Action of Various Poisons

Some of the poisons on contact cause absorption and rapid death or impairment of function. The compounds acting on nervous system paralyze within seconds or less, include both biologically derived neurotoxins and so-called nerve gases, which may be synthesized for warfare or industry. Inhaled or ingested cyanide is used as method of

execution on US. Gas chambers almost instantly starves the body of energy by poisoning mitochondria and synthesis of ATP. Intravenous injection of an unnaturally high concentration of potassium chloride, such as in the execution of prisoners in parts of the United States, quickly stops the heart by eliminating the cell potential necessary for muscle contraction. Such rapid reactions are often called acute poisoning. A poison may also act slowly known as chronic poisoning and is most common for poisons that accumulate such as lead, mercury, copper etc.

Factors Modifying the Actions of Poisons

There are a number of factors that modify the actions of poisons:

Quantity: The toxic effects of a poison vary with its quantity. Every drug is a poison when taken in a large quantity. But sometimes when a drug is ingested in a large quantity, it may induce vomiting and is rapidly eliminated leading to reduced toxicity. This occurs in alcohol and copper sulphate poisoning.

Route of administration: The severity and rapidity of action depends on the route of administration or intake of the poison. Inhaled poisons are rapidly fatal compared to the parenteral poisons whereas ingested poisons are less severe and act slowly compared to that taken subcutaneously or intramuscularly. The poisons are more effective when taken on an empty stomach.

Age and state of the body: Children, old, weak and diseased are more susceptible to the effect of the poison compared to the adults, healthy and stout persons.

Idiosyncrasy: Idiosyncrasy is allergic or anaphylactic response to a substance in which even a drug that is otherwise safe produces toxic effect. This effect can be produced by a large number of substances. A substance that is being used therapeutically may produce toxic effects. This can be due to anaphylaxis, idiosyncrasy or from the over dosage of the drug.

Tolerance: When a person uses some substance continuously, he is liable to tolerate larger than

normal dose of that substance. This commonly occurs in an alcoholic who can tolerate larger than normal amount of morphine and related drugs.

Cumulative action: When a poison that is excreted may accumulate and produce toxicity when given in repeated doses for a long time.

Manner of Poisoning

- a. **Homicidal** poisoning is the administration of poisonous substance by a person to kill another.
- b. **Suicidal** poisoning is the intake of some poisonous substance by the individual himself for self-killing.
- c. **Accidental** poisoning generally takes place due to the storage of poisonous and non-poisonous substances at the same place. The injudicious use of the magic remedies and love-philters can also result in accidental poisoning. The characteristics of ideal suicidal and homicidal poison are described:

Characteristics	Suicidal	Homicidal
Availability	Easy and free	Not so
Cost	Low in cost	Not so
Colour, taste and odour	Tasteless or with pleasant taste	Colourless, tasteless and odourless
Solubility with food and drink	Present	Present
Signs and symptoms	Should not be there or very few	Resemble some natural disease
Onset of action	Quick onset	Slow onset
Antidote	Should not be available	Should not be available
Toxicity	Highly toxic	Highly toxic
Diagnosis from chemical tests	Difficult	Difficult
Death	Painless	Definite
Postmortem changes	May be produced	Nil
Common compounds used for the purpose	Aluminium phosphide, pesticides such as organophosphates and carbamates, barbiturates, potassium cyanide and copper sulphate and oxalic acid	Arsenic, antimony, aconite, thallium, organophosphorus compounds, strychnine, potassium cyanide.

CLASSIFICATION OF POISONS

I. Corrosives

- A. **Strong acids:**
 - Inorganic or mineral acids—Sulphuric acid, nitric acid and hydrochloric acid.
 - Organic acids—Carbolic acid, Oxalic acid, acetic acid and salicylic acid.
- B. **Strong alkalis:**
 - Hydrates
 - Carbonates of sodium and potassium.
- C. **Metallic salts:**
 - Zinc chloride
 - Potassium chloride
 - Ferric chloride.

II. Irritants

- A. **Inorganic:**
 - Metallic—Arsenic, lead, mercury, copper, zinc, thallium etc.
 - Non metallic—Phosphorus, chlorine, bromine, iodine.
- B. **Organic:**
 - Vegetable poisons—Castor oil seeds, croton oil, *madar*, *semicarpus anacardium*, *calotropis*, capsicum, aloes, *Abrus precatorius*
 - Animal—Cantharides, snake venom, insects, scorpion, spiders etc.
- C. **Mechanical:** Powdered glass, diamond dust, chopped hair etc.

III. Neurotoxic

- A. **Cerebral:**
 - Somniferous—Opium, barbiturates
 - Inebriants—Alcohol, Ether and chloroform
 - Deliriant—Datura, Belladonna, Cannabis, Hyocyamus, Cocaine.
- B. **Spinal**—Nux vomica and gelsemium.
- C. **Peripheral**—Curare, Conium.

IV. Cardiotoxic

Aconite, Quinine, Digitalis, oleander, nicotine and hydrocyanic acid.

V. Asphyxiants

These are poisonous irrespirable gases such as carbon dioxide, carbon monoxide, hydrogen

sulphide and coal gas. This category also includes the cyanides.

VI. Miscellaneous

A. *Food poisons*—Food borne botulism etc.

B. *Agaro-chemicals*:

- Pesticides and insecticides—Organophosphates, carbamates and organo chlorine compounds.
- Bipyridal herbicides—Paraquats and diquat
- Chlorophenoxy herbicides—Bromoxynil, ioxynil.
- Rodenticides—Zinc phosphide, Strychnine sulphate, Sodium monofluoroacetate, Floroacetamide.
- Fumigants—Aluminium phosphide, ethylene dibromide.

C. *Domestic and commercial poisons*:

- Petroleum distillates—Kerosene, Petrol mineral oil
- Disinfectants, detergents and cleansing agents.

D. *Drugs of abuse*—Alcohol, Tobacco, Cannabis, Cocaine and opium derivatives, sedatives and hypnotics

E. Chemical and biological warfare agents.

Common Household Poisons and Their Treatment

The common household poisons along with the symptoms and signs of their poisoning (Table 35.1).

Table 35.1

<i>Preparation</i>	<i>Poisonous substance</i>	<i>Symptoms and signs</i>	<i>Management</i>
Moth balls	Naphthalene	Nausea, vomiting, pain abdomen, albuminuria and hematuria, pulmonary oedema and coma	Stomach wash, soda bicarbonate, i.v. fluids. oxygen & artificial respiration
Baby powder	Boric acid	Nausea, vomiting, diarrhoea, convulsions, respiratory depression	Stomach wash, oxygen and artificial respiration
Insecticide spray	DDT/ organophosphorus compounds	Nausea, vomiting, tremors, convulsions, coma	Stomach wash, paraldehyde, oxygen and artificial respiration
Chalk	Arsenic, copper and lead in coloured chalks	Nausea, vomiting, pain abdomen, symptoms of metallic poisoning	Stomach wash and treatment of metallic poisoning
Fireworks	Arsenic, mercury, lead and phosphorus	Burns	Burns to be treated
Cosmetics (nail enamel, and thinners)	Potassium hydroxide, barium sulphate, acetone, methyl salicylates and denatured spirit	Nausea, vomiting, burning in stomach	Stomach wash
Baking powder	Tartaric acid	Nausea, vomiting, burning pain abdomen,	Immediate soda bicarb to neutralize the acid
Baking soda	Sodium carbonate	Nausea, vomiting, abdominal distension	Vinegar
Crockery washing substance	Sodium and sodium polyphosphates	Variable symptoms depending on the substance	Symptomatic treatment
Match box and sticks	Silicates, antimony, phosphorus and potassium chlorate	Antimony and phosphorus poisoning	Treat symptoms of poisoning
Rat poison	Zinc phosphide	Nausea, hematuria,	Stomach wash, Inj. vitamin K i.m.,
Grain preservative	Aluminium phosphide	bleeding	oxygen and artificial respiration

DUTIES OF MEDICAL PRACTITIONER IN A CASE OF POISONING

The duties of medical practitioner are both legal and professional.

Professional Duties

Legal Duties of the Doctor

1. In all cases of suspected or confirmed poisonings the attending doctor should record all the findings in a medicolegal case report and inform the nearest police.
2. If the homicidal poisoning is suspected by the doctor, the Police officer should be informed (S. 39 CrPC). If the doctor fails to report the matter to the police, he will be held culpable under section 176 I.P.C.
3. The attending physician should collect, preserve and seal the evidence related to the case of poisoning such as the gastric lavage fluid, vomitus, faeces and urine etc. for onward transmission to the Forensic Science laboratory for chemical analysis. If the doctor deliberately fails to do so he is liable to be punished under section 201 I.P.C.
4. In all the cases of poisoning whether suicidal or homicidal, it is the duty of the attending physician to divulge to the police whatever information he has. Withholding the information or providing wrong information makes the doctor liable to be punished under section 202 and 193 I.P.C.
5. In case of poisoned patient on the verge of dying it is the duty of the attending doctor to record dying declaration if the magistrate is not available at that time. Even when the magistrate is recording the statement of such a patient, the doctor should examine the patient in regards to his consciousness and whether there is clear faculty of thought and judgment or not (*compos mentis*).
6. In cases that are brought dead to the hospital or the patient has died during the course of treatment, the doctor should not issue death certificate but instead send the body for autopsy examination.

7. In cases of food poisoning, the doctor should collect the contaminated food and send it to the forensic science lab for chemical analysis. It is the duty of the doctor to report such cases to public health officials.

TREATMENT IN POISONING CASES

A. Stabilise the Patient

It is very important to stabilise the patient first of all and to treat the most imminent danger to life immediately.

B. Removal of the poisoning material from the system (Decontamination)

- In cases of inhaled poisons, the individual should be shifted to fresh air and artificial ventilation should be started.
- In cases of injected poisons from bite or an injection, a ligature should be tied to prevent it from further reaching the circulation. The ligature is loosened for one minute after every ten minutes to prevent gangrene formation.
- In contact poisons affecting the skin, mucous membrane, any wound or introduced in the uterus or vagina they should be irrigated with water and neutralized by application of suitable chemical.

The poisons are most commonly ingested orally. Depending on the time lapsed after ingestion; much of the poison lying unabsorbed in the stomach should be removed. In all ingested poisons the guidelines to treatment are:

1. The stomach should be emptied by gastric lavage or emesis but emetics are avoided in corrosive intake.
2. Appropriate antidote should be given to neutralize the poisonous compound even despite gastric lavage and emesis as some poisons are secreted again in stomach after having been absorbed.
3. Aid elimination of poison by the intestines and kidneys of what has been absorbed.
4. Symptomatic treatment.
5. Egg whites are useful in most poisonings and tannin is antidotal to all alkaloids.

In ingested poisons, the methods of decontamination are: (i) Gastric lavage (ii) Emesis (iii) Catharsis (iv) Administration of activated charcoal (v) Whole bowel irrigation.

Stomach Wash (Gastric Lavage)

Gastric lavage is indicated in patients who present within three hours of ingestion of poison. The stomach wash can also be carried in the presence of gastric secretions, delayed gastric emptying or in case of ingestion of sustained release medications. Gastric lavage beyond 6-12 hours is recommended in ingestion of salicylates, tricyclics, carbamazepine and barbiturates.

Gastric Lavage Tube: For gastric lavage, a soft rubber tube with a funnel at its one end known as Ewald or Boas tube is most commonly used. In adults an ordinary, soft, non-collapsible tube with 36-40 French size having one cm diameter and 1½ mts length is used. In children with 22-28 French size (Ryle's tube) diameters should be used. The tube should have an attached glass funnel at one end and the other end should be rounded with lateral openings. There is a mark at a distance of 50cm from its rounded end. A suction bulb is placed at the mid of tube to pump out stomach contents.

Procedure for Stomach Wash

Before performing stomach wash, the patient should be lying in a left lateral position or in a prone position with head hanging over the edge of the bed and the face is positioned downwards so the mouth is at a lower level than the larynx and chances of aspiration of fluid are eliminated.

The gastric lavage tube is gently passed in to the stomach through the mouth by lubricating with glycerin or Vaseline jelly up to a distance of 50cm in adults and 25cm in children. The position of the tube in stomach can be checked by stethoscope or putting the upper end in a cut of water, if the lower end in the trachea, air bubbles will come out. For carrying out stomach wash, initially 250-300mL of warm (35°C) saline or plain water is passed through the funnel held high up. In children, instead of water 10-15mL/kg body weight of warm

saline is used as in them there are chances of inducing hyponatremia and water intoxication. The stomach contents can be siphoned by the use of suction bulb. The first sample of stomach wash should be preserved for chemical analysis. Then stomach is carried out with chemical agents specific to the poisons. In certain specific poisonings, instead of plain water or normal saline, other solutions can be used that are specific for particular poisons. These solutions are:

1. Potassium permanganate (1:5000 or 1:10,000) is used as gastric lavage fluid in various oxidizable poisons e.g., alkaloids, salicylates.
2. Sodium bicarbonate (5%).
3. Tannic acid (4%).
4. Sodium thiosulfate (25%) can be used in cyanides and
5. Calcium gluconate is used for oxalates.
6. 1:2 Castor oil and warm water solution is to be used for carbolic acid and phenolic group of poisons.
7. Desferrioxamine (2 gm in 1 liter of water) is used for iron poisoning.

In cases where potassium permanganate (a powerful oxidising agent) is used as gastric lavage fluid, the gastric lavage is continued till the colour of lavage fluid is colourless, odourless and no particulate matter is visible. At this time, a small quantity of fluid containing specific antidote or 1 gm/kg body wt. of the suspension of activated charcoal and/or an ionic cathartic is left in the stomach.

Complications of Gastric Lavage

1. Laryngeal spasm.
2. Aspiration pneumonitis.
3. Perforation of stomach or oesophagus.
4. Sinus bradycardia and ST elevation on ECG.

Contraindication of Gastric Lavage

The contraindication for stomach wash are absolute and relative.

Absolute contraindication:

1. Corrosive poisoning except Carbolic acid as there is danger of perforation.
2. Convalescent poisons.

3. Comatose patient as there is risk of aspiration
4. Volatile poisons due to the risk of inhalation.
5. If the patient is hypothermic.

Relative contraindication:

1. If the patient is suffering from alimentary tract diseases like oesophageal varices.
2. Comatose patients.
3. Ingestion of alkali.
4. Advanced pregnancy.
5. Any haemorrhagic diathesis.
6. Any history of recent surgical operation.

Emesis

The emesis should be avoided as there is danger of aspiration of stomach contents in an unconscious patient. The easiest way to induce vomiting is by tickling the fauces. Also vomiting can be produced by the use of emetics of which ipecac is the most effective whereas mustard powder and warm saline can give rise to complications. Also Apomorphine and zinc sulphate are no longer used. These emetics are:

- i. Warm saline water comprising of 2tsf salt in 200mL of water.
- ii. Mustard powder 15 gms in 200mL of water
- iii. Zinc sulphate 1-2gms in 200mL of water
- iv. Apomorphine 3-6mg I.M. is the most potent and immediately acting emetic as it produces copious vomiting within 3-4 minutes. Apomorphine causes severe narcosis that is why it should not be used in comatose patients. If it is used, Naloxone hydrochloride, 5-10 mg i.m. should be administered that will counteract narcosis.
- v. Ipecacuanha powder 1-2gm or 30mL (15mL in children) of ipecac syrup causes vomiting with satisfactory results in a poisoned patient. It is derived from the root of *Cephalis ipecacuanha* and *C. acuminata*. The active principles are cephaline, emetine and traces of psychotropine and it causes activation of peripheral sensory receptors in the GIT and also stimulates the chemoreceptor trigger zone and vomiting center in the medulla thus causing vomiting. The contraindication of emesis are:

Absolute Contraindication

1. Pregnancy.
2. Heart disease.
3. Haemorrhagic diathesis.
4. Cardiotoxic poison ingestion.
5. In infants and old patients.

Relative Contraindication

1. During convulsions.
2. Ingestion of convalescent poison such as strychnine.
3. Ingestion of strong acids and alkalis due to the chances of perforation.
4. Ingestion of kerosene oil as there are chances of aspiration pneumonitis.
5. Comatose or unconscious patients.
6. Foreign body ingestion.
7. Impaired gag reflex.
8. Poison that causes emesis.
9. Ingestion of petroleum distillates or drugs causing altered mental status.

Complications of Emesis

1. Features of cardiotoxicity such as bradycardia, atrial fibrillation and myocarditis.
2. Aspiration pneumonitis.
3. Oesophageal tears may be caused due to protracted vomiting.

C. Administration of Antidotes

An antidote is defined as a remedy to counteract or neutralize the effects of a poison. In various poisonings antidotes are used to counteract the pathophysiology produced by a toxin.

According to the mode of action antidotes are classified as:

Mechanical or Physical Antidotes

The mechanical antidotes counteract the effect of poison mechanically preventing their absorption without inactivating the damaging action of the poisons. These are of the following types

Adsorbent like activated charcoal: Activated charcoal is a fine, black, odourless and tasteless type of amorphous carbon prepared by destructive

distillation of materials such as burning wood, coconut shell, bone, sucrose, or rice starch that have much higher surface area than charcoal followed by treatment with an activating agent such as steam, carbon dioxide etc. The large surface area of activated charcoal confers a great adsorptive capacity to this material. Each gram of activated charcoal works out to a surface area of 1000 m². Activated charcoal strongly adsorbs aromatic substances such as acetaminophen, salicylates, barbiturates and tricyclic antidepressants thus reducing their absorption from the gastrointestinal tract. Most inorganic substances are poorly absorbed by activated charcoal. The dose is 1gm/kg body weight (50-100 gm in adults and 10-30 gm in children) after making a suspension in 4-8 times the water. 4-8 gms of activated charcoal acts mechanically by absorbing and also retains in its pores organic poisons and mineral poisons to a less degree. The side effects are vomiting, diarrhoea, constipation, pulmonary aspiration and Intestinal obstruction. The contraindication of the use of activated charcoal are ileus, small bowel obstruction and when there is history of caustic or petroleum distillate ingestion.

Demulcents: Demulcents produce protective coating over the mucous membrane of stomach to protect it from the action of poison. These are milk, egg white, starch, milk of magnesia and aluminium hydroxide gel.

Bulky foods like Bananas, boiled rice or potatoes allow smaller amount of poison to be available to the stomach mucosa for absorption as they are admixed with the poison itself. The bulky foods are commonly used in ingestion of glass powder as these particles are embedded in them and prevent damage to the stomach mucosa.

Diluents such as water, milk or other similar drinks that dilute the poison and delay their absorption.

Chemical Antidotes

The chemical antidotes disintegrate and inactivate poisons by undergoing chemical reactions along with the poisons and forming harmless or insoluble compounds. These are:

- i. **Weak non-carbonate alkalis:** In corrosive acid poisonings, weak non-carbonate alkalis act as neutralizers. Strong alkalis are avoided as they can cause further damage the stomach. Non-carbonate alkalis are preferred as carbonate alkalis react with acids to produce carbon dioxide gas that inflates the stomach and may get ruptured.
- ii. **Weak vegetable acids:** In corrosive alkali poisoning, weak vegetable acids like citric acid, and acetic acid (vinegar, lemon juice) may be used. For arsenic poisoning, freshly prepared solution of ferric oxide can be used as it forms non-absorbable ferric arsenate.
- iii. **Albumen:** It is used for mercury poisoning as it precipitates Mercuric chloride.
- iv. Copper sulphate is antidote for phosphorus
- v. Potassium permanganate is an oxidizing agent use in poisoning with oxidizable substances like cyanides, phosphorus, atropine and other alkaloids. A dilute 1:5,000 or 1:10,000 solution of potassium permanganate is used in any poisonings such as aluminium phosphide, opium derivatives, insecticides, nicotine, cyanides, hydrocyanic acid atropine and strychnine etc.
- vi. Tincture iodine or Lugol's iodine in a solution of 15 drops in half a glass of water precipitates lead, mercury, silver, alkaloids and strychnine.
- vii. Tannic acid (4%) or strong tea or 1 tsf of tannic acid dissolved in water is used to precipitate metals like lead, mercury, nickel, zinc, copper, aluminium, cobalt and silver; strychnine, nicotine, cocaine etc.

Physiological Antidote

The physiological antidote acts on the tissues and various systems of the body and produce signs and symptoms opposite to the signs and symptoms produced by the poison. They are basically of use when some of the poison has already been absorbed in to the circulation. They antagonize the effects produced by the poisonous substance. These are like Atropine for organophosphorus compounds, physostigmine, and neostigmine for

Datura and barbiturate for strychnine. The different types of antidotes for various poisons are listed below:

Poisons	Antidotes
Acetaminophen	N-acetylcysteine
Anticholinergics (<i>Datura fastuosa</i>)	Physostigmine
Carbon monoxide	Oxygen
Anticholinesterases (Organophosphorus compounds)	Atropine sulphate, Pralidoxime chloride (2-PAM)
Cyanide	Amyl nitrite, sodium nitrite and thiosulphate
Ethylene glycol	Ethanol
Heavy metal poisoning	Chelators, calcium disodium edentate (EDTA), dimercap- rol (BAL), penicillamine and 2,3-dimercaptosuccinic acid (DMSA)
Iron	Desferroxamine
Methanol	Ethanol
Methaemoglobinemia	Methylene blue
Opioid	Naloxone hydrochloride
Arsenic compounds	Ferric oxide
Copper compounds	Potassium ferrocyanide

Universal Antidote

Universal antidote comprises of:

1. Activated powdered charcoal—2 parts by weight
2. Magnesium oxide—2 parts by weight
3. Tannic acid—1 part by weight
15 gms of this powdered mixture should be added to half a glass of warm water before consumption. The use of universal antidote is obsolete now-a-days but this can definitely used as a first-aid measure at homes.

Chelating Agents

Chelating agents inactivate a metallic ion with the formation of an inner ring structure in the molecule, when the metallic ion becomes the member of the ring. The chelating agents will form no-toxic stable soluble compounds with calcium and other heavy metals like arsenic, lead, mercury, copper, zinc, nickel, cobalt, manganese etc.

British Anti-lewisite (B.A.L)- Dimercaptopropanol

B.A.L. was originally used as an antidote for Lewisite, a vesicant containing arsenic that was

used as war gas. This compound is used in heavy metal poisoning especially arsenic, mercury, lead, antimony, gold and thallium and also to some extent against copper, bismuth etc.

Mechanism of action: Heavy metal ions have a great affinity for sulphhydryl (SH) radicals in the cells and tissue enzymes and they combine with them by displacing hydrogen, thereby depriving the body of certain tissue enzymes whose activity depend upon the SH groups. The thiol (SH) group of B.A.L. will combine with the heavy metals in the system and will dislodge them from their combination with the sulphhydryl radicals in the tissue enzymes and thus sparing the tissue from its toxic effects. Dimercaprol forms a rather stable compound with the heavy metal and gets excreted out of the body mainly in urine without causing any damage to the liver, kidneys etc.

Dose of B.A.L is 3-4mg/kg body weight that is to be administered deep intramuscularly within first 4 hours of poisoning. The ampoule of B.A.L (100mg/mL) contains 2ml of 10% compound in Arachis or peanut oil with 20% benzyl benzoate solution. The injection must be given 4 hourly for first 2 days and then thrice daily for 10 days.

Contraindications: Administration of Dimercaprol is absolutely contraindicated in cadmium poisoning (forms a nephrotoxic compound with cadmium) and any pre-existing liver disease. Also it is relatively contraindicated in any kidney disease.

Side effects: The side effects of administration of Dimercaprol appear when it is administered in a dose more than 3.5mg/kg body weight.

1. Anorexia, nausea and vomiting.
2. Excessive salivation or lachrymation.
3. Generalized aches and pains.
4. Hyperthermia.
5. Feeling of constriction of chest.
6. Burning of eyes and throat.
7. Fall of blood pressure.

E.D.T.A (Ethylene Diamine Tetra-acetic Acid)

E.D.T.A forms readily soluble, practically non-ionized and nontoxic compound with heavy

metals. It is a useful antidote for those heavy metals that have an affinity for calcium.

Mechanism of action: When lead, zinc, manganese, cadmium, iron and copper form compounds with edetate, calcium cannot displace them. Calcium disodium EDTA chelates extracellular lead in soft tissues as well as in bones. In practice, edentate is given in the form of calcium disodium versenate to prevent rapid removal of calcium from the body. In the presence of lead, this chelating agent readily exchanges calcium for lead and thus detoxification and excretion of lead is done. The chelating agent renders lead water soluble, non-toxic, non-ionized and non-metabolized to get excreted intact in the urine. Edetate does not get metabolized in the body like Dimercaprol and is poorly absorbed by the gut. Calcium disodium versenate is the best known chelating agent against inorganic lead intoxication but not so effective in tetraethyl lead poisoning. It is used principally against lead intoxication but can also be used in copper, zinc and nickel poisoning. It is less effective in manganese, iron, cadmium and radio-active elements poisoning. It is superior to both BAL in the treatment of arsenic and mercury.

Dose: 5 mL ampoule of 20% calcium edetate is dissolved in normal saline or 5% saline dextrose (250-500mL) solution. It is administered by slow I.V. drip. The concentration should not exceed 3% and the drip should not take less than two hours to complete. The usual dosage is 50-70mgm/kg per day and in adults 1gm can be given I.V. Twice daily for five days. This should be repeated again after a gap of three days. E.D.T.A. should not be given orally as lead will get chelated in the G.I.T. and more of it will be absorbed.

Side effects:

1. Thrombophlebitis from administration of strong solution.
2. Lower-nephron nephrosis.
3. Hypersensitivity to the agent.
4. Fever, headache, generalized malaise and fatigability.
5. Nausea, vertigo and vomiting.
6. Hypotension.

Contraindication: The only contraindication is raised intracranial pressure when fluids are to be restricted.

Penicillamine (Cuprimine)

Penicillamine is the product of hydrolysis of penicillin. It acts as a chelating agent because of possessing stable SH groups. It is advantageous to use this compound against poisoning due to lead, copper, mercury and zinc preparations, as it can be used orally and continuously for a long time without producing any major toxicity. It is also useful in treating Wilson's disease (hepatolenticular degeneration) resulting from disorders of copper metabolism and cystinuria.

Dose: It is given 0.5 gm orally half an hour before meals four times a day for a period of 8-10 days. Pyridoxine 25-50mgm/day can be given to counteract symptoms of pyridoxine deficiency.

Side effects: The side effects of Penicillamine therapy are rare and are more pronounced when the patient is suffering from copper storage disease, cystinuria or scleroderma for a long period.

1. Hypersensitivity reactions in the form of skin rashes and nephrotoxicity
2. Optic neuritis resulting from pyridoxine deficiency.
3. Leucopenia, thrombocytopenia and agranulocytosis.

Desferrioxamine Mesylate or Deferoxamine

Desferrioxamine is a water soluble compound that has a great affinity for ferric ion. This acts as chelating agent against iron intoxication especially in case of acute poisoning through its role in accelerating removal of iron from the body in case of haemochromatosis cannot be challenged. It removes iron from ferritin, hemosiderin, a little from transferring but not from haemoglobin and cytochromes.

Dose: It can be given orally, intramuscularly as well as intravenously:

Orally: 8-10gms dissolved in 80-100mL of distilled water

Intramuscularly: 1gm initially to be followed by 0.5gms twice or thrice daily

Intravenously: 1-2gm in 5% of 500 mL of dextrose saline solution; not more than 15mg/kg body wt per hour or 80mgm/kg in 24 hours should be administered.

D. Elimination of Poison by Excretion

When more than 6-8 hours have elapsed after ingestion of the poison, the absorbed poison should be eliminated by excretory channels that are kidneys. In barbiturate and salicylates poisoning, this is the only treatment. Diuretics like chlorothizide, mannitol or furosemide are commonly used drugs. Fluid balance is to be maintained by IV fluid infusion. Peritoneal dialysis and exchange transfusion is done in small children in poisonings due to barbiturates, salicylates and iron. Hemodialysis is employed for removing barbiturates, salicylates, bromides, boric acid and thio-cyanates. The methods employed for the purpose are:

Catharsis

Cathartics reduce the transit time of the poisonous substance in the G.I.T but their efficacy in reducing the mortality or morbidity is not established. The cathartics are:

Saline cathartics: These cathartics alter the physico-chemical forces within the intestinal lumen leading to osmotic retention of fluid which activates motility reflexes and enhances expulsion. The dose of recommended cathartics is:

- Magnesium citrate—4mL/kg.
- Magnesium sulphate—30gm (250mg/kg in children).
- Sodium sulphate—30.gm (250mg/kg in children) in copious amount of water.

The high dosage magnesium cathartics can result in hypermagnesemia.

Saccharide cathartics: Sorbitol (D-glucitol), 50 mL of 70% solution is the cathartic of choice in adults because of better efficacy than saline cathartics but in children there is risk of hypernatremia.

E. Symptomatic Treatment

The patient should be treated for the symptoms accompanied with good nursing care under

constant supervision of the physician. Respiratory tract infection is commonly encountered more common in older patients who have been unconscious for hours and in whom gastric lavage was done. In such cases, routine antibiotic prophylaxis should be given. Dehydration, anuria, convulsions, circulatory collapse hepatic and renal failure should be taken care of. Fluid balance to be maintained by I.V. fluid infusion and I.V. mannitol should be administered to combat renal failure. BP and pulse rate should be maintained continuously. The airways should be protected and artificial respiration with oxygen inhalation to be given.

Diagnosis of Poisoning in the Living

1. Symptoms appearing suddenly in a healthy person.
2. The symptoms appear uniformly and rapidly increase in severity.
3. When several persons are affected at the same time like food poisoning.
4. Detection of poison in the food.

In such cases the following material should be collected:

- i. Stomach wash fluid.
- ii. Blood with sodium fluoride as preservative.
- iii. Urine with sodium fluoride as preservative.
- iv. Faeces.

Diagnosis of Poisoning in the Dead

1. *Postmortem appearances:* Clothes and external surface of the body should be looked for stains of poison, vomitus and faeces etc. The dark brownish or black stains of lips and cheeks occurs in Sulphuric acid poisoning, yellowish brown stains appear in Nitric acid poisoning and grayish white stains in carbolic acid poisoning.
2. *Colour of postmortem staining:* The colour of postmortem staining depends on the poison ingested; Yellow coloured hypostasis develops in Phosphorus, Copper and Cherry red discolouration occurs in CO poisoning.
3. *Odour from the body cavity:* Many poisons have a characteristic smell that is peculiar to that particular poison.

Type of poison	Characteristic smell
Cyanides	Bitter almonds
Carbolic acid	Hospital disinfectant (soap like)
Alcohol	Acetone-apple like
Chloral hydrate & paraldehyde	Acrid-pear like
Aluminium phosphide & zinc phosphides	Fishy, phosphene like
Carbon monoxide	Coal gas
Organophosphates	Garlicky
Hydrogen sulphide & N-acetyl cysteine	Rotten eggs
Ethanol, Chloroform & Nitrites	Sweet or fruity

4. **Natural orifices:** The discharges from natural orifices are characteristic of some of the poisons.
5. Injection marks over the body should be noted in some injectable poisons.
6. Evidence of marks of violence may be seen in homicidal deaths. The bodies that are poisoned are not readily decomposed.

Postmortem Examination in a Case of Poisoning

Aims:

1. To identify the type of poison.
2. To find out time since death.
3. To find out cause of death.
4. To find out the manner of death whether suicide, accident or homicide.
5. In infants to find out whether it is a live birth and viability.

Diagnosis: The diagnosis in a case of poisoning should be made from postmortem appearances.

The autopsy surgeon should read the inquest papers and the information relating to the poisoning should be gathered from the relatives about: (i) Quantity and quality of poison consumed (ii) Time of onset of symptoms after taking the poison (iii) Duration of illness (iv) Treatment given.

Very often the information is inadequate so it is quite a difficult task when the bodies are decomposed or has ingested corrosive or irritant poisons. For this a thorough examination is to be carried out along with the histopathological examination and chemical analysis of viscera.

External Examination

1. **Postmortem staining:** Normal colour of postmortem staining is coppery red or purple. It is:
 - Pink in CO poisoning, hydro cyanic acid poisoning, and in burns.
 - Purple or violet in asphyxia deaths.
 - Coffee brown in Potassium chlorate poisoning.
 - Dark brown in phosphorus poisoning.
2. **Odour from the body:** No disinfectants should be used as they may mask the smell. On opening the body the characteristic smell of poisons like HCN, Carbolic acid, Alcohol, ether, chloroform and opium can be felt.
3. **Examination of the natural orifices:** In the natural orifices the evidence of corrosive changes over the mouth, lips and tongue can be seen. They may be discoloured yellow in HCN poisoning.
4. **Injection marks:** This is particularly important in injectable poisons.
5. **Colour of the skin:** The skin may be discoloured yellow or jaundiced in phosphorus or acute copper poisoning.

Internal Findings

1. All the organs must be examined and contents preserved for chemical analysis.
2. Any peculiar smell should be noted.
3. Mouth and throat should be examined for erosions or staining.
4. Upper respiratory tract should be particularly examined in cases of poisoning with volatile irritants or there may be presence of inhaled poisonous matter.
5. The mucous membrane of the stomach and gastro-intestinal tract should thoroughly be examined for the following signs of corrosive and irritant poisoning:
 - i. **Colour of the mucous membrane of stomach:** The normal mucous membrane of the stomach is pale and white. Irritant poisons cause patchy redness of the mucous membrane at the cardiac end and greater curvature of the stomach but rarely of the pyloric end. Due to the irritant action of the

poison, there may be small haemorrhagic areas along with mucous secretion. Redness of the mucosa of the posterior wall may also be found after death. In a case, gastric redness was detected after nineteen months of Arsenic poisoning and in another case twenty-one months after exhumation. The mucous membrane is velvety in appearance in Arsenic poisoning and is covered with viscid secretion that is blood stained.

Redness of mucous membrane of stomach encountered in asphyxial deaths is due to generalized venous congestion. The hyperemia of stomach occurring in some of the diseased states is uniform in distribution. The colour of stomach walls may be discoloured due to the bile, fruit juice or food ingested that is uniform in distribution with no signs of inflammation. The mucous membrane may be red or hyperemic:

- When the deceased had died during the process of digestion.
- Asphyxial deaths when there is generalized venous congestion.
- When the mucous membrane is exposed to oxygen.
- In diseased states like chronic gastritis or gastric ulcer, the redness is uniformly spread and the ridges are usually not involved.
- In deaths from poisoning, the redness is patchy; more marked at the cardiac end and greater curvature and also involves the mucosal ridges. The colour of stomach walls may be discoloured due to the bile, fruit juice or food ingested that is uniform in distribution with no signs of inflammation. In various poisonings, the colour of the walls of the stomach may be:

<i>Type of poisoning</i>	<i>Colour of stomach walls</i>
Ferrous sulphate	Green coloured
Amytal	Blue coloured
Soneryl	Pink coloured
Mercury	Slate coloured
Oxalic acid	Black coloured
Nitric acid	Yellow coloured
Copper sulphate	Bluish green coloured

- ii. *Softening*: Corrosive acids, alkalis and irritants cause softening of greater curvature and cardiac end of stomach as they damage the superficial epithelium. In diseased states such as peptic ulcer or malignancy this softening is uniform and limited to stomach whereas in putrefaction, the softening starts at the dependent parts and involves all the layers of stomach wall and inflammatory signs are absent. Carbolic acid causes hardening and shrinkage of mucous membrane.
- iii. *Ulcers*: Ulcers due to corrosives or irritant poisons are present on the greater curvature, have thin, friable margins and surrounded by signs of inflammation. The mucosa is soft and hyperemic. The ulcers due to disease or malignancy are seen on the lesser curvature with their well-defined punched out margins that are thickened and indurated. Gastric ulcer involves the lesser curvature, has sharply defined, thickened and indurated edges and redness in the neighbourhood of the ulcer. But the redness in the irritant poisoning is diffuse and extends to duodenum and small intestine. In chronic ulcer due to disease, aperture is oval or rounded and the margins are punched out. There is no charring and chronic adhesions to other organs are present. Rarely both the ulcers will look similar. After death, autodigestion by gastric juices occurs that appears near cardiac end, is large, irregular with rough edges and no inflammation or charring is there.
- iv. *Perforation*: The perforation of stomach may be found in strong acid such as sulphuric acid poisoning. The stomach is usually black in colour with extensively damaged mucosa. The aperture is large with irregular edge and the coats are lacerated through which acid escapes to the peritoneal cavity causing acute peritonitis. In a chronic gastric ulcer when perforation occurs, it looks either oval or round with a punched out appearance with indurated margin. In rare instances it may cause autolysis at the cardiac end of stomach without any peritonitis.

- v. *Stomach contents*: The contents of the stomach should be thoroughly examined for traces of poison and also for any distinguishable odour.

All the organs of the body should be thoroughly examined for the signs of diseases to rule out diseased states leading to the death of the patient. Sometimes abortifacient drugs are introduced through the vaginal route for which the vagina and whole of the female genital tract be examined.

Chemical Analysis in Poisoning

An important proof of poisoning is the detection of poisons in the excreta, blood and viscera. The finding of the poison in the food, medicines act as a corroborative but not a conclusive proof. The medical practitioner must preserve all the viscera and get it sealed in his presence for onward transmission to the police officer who will forward it to the Forensic science lab for chemical analysis.

Defence lawyer's argument: If the poison is present in the stomach, it might have been introduced after death. The argument is useless if the poison is found in the viscera and preserved in a clean container to avoid contamination. It is not necessary to lay stress on the amount of poison in the analysis, except that:

- Poison might have been taken as a medicine.
- Poison is used on a regular basis habitually.
- It is a natural constituent of the body.
- It is a normal constituent of food.
- It is produced during decomposition.

It is possible that a person may die from the effects of the poison but none is found after death. When the poison has not been detected, the judge should the symptoms, postmortem report and oral evidence.

The viscera along with certain body fluids should be collected, preserved and sent to the Forensic Science laboratory for chemical analysis by the forensic pathologist. The presence of poisons should be demonstrated by standardized analytical methods. The preservative for the viscera is rectified spirit or saturated saline solution. The blood can be preserved in potassium oxalate or sodium fluoride and urine should also be preserved

with sodium fluoride. The viscera that is preserved routinely should include the following:

Viscera	Quantity	Preservative
Stomach with its contents	Whole of the stomach with entire contents	Saturated saline or rectified spirit
Small intestine with contents	Up to 30 cm with 100 gms of contents.	–do–
Liver with gall bladder	500 gm	–do–
Kidney	One half of each or whole	–do–
Spleen	Whole	–do–
Blood	10-20 ml	Potassium oxalate or sodium fluoride
Urine	30-50 ml	Sodium fluoride

About 10-20 ml of blood should be taken in a glass bottle from the femoral vein and should not be taken from the abdominal or pleural cavity, with potassium fluoride or sodium fluoride as preservative in suspected alcohol poisoning. About 30-50 ml of urine should be kept with sodium fluoride as preservative.

Most of the poisons are ingested, so stomach and intestines are preserved. As they pass through the liver, it is also preserved and kidney being the organ of excretion and finally it is excreted in urine.

Points to be followed while preserving the viscera:

1. The viscera are preserved in saturated solution of common salt or rectified spirit. Rectified spirit is not to be used in poisonings due to Alcohol, Acetic acid, Phenol, Phosphorus and paraldehyde where saturated solution of sodium chloride is used. Formaldehyde should not be used as a preservative because it makes extraction of poisons difficult.
2. Stomach and intestine are kept together in a glass jar and in another jar pieces of liver, kidney and spleen are preserved. Blood and urine are preserved separately in two glass bottles. Rubber inserts should not be used as it extracts some poisons like chloroform and phenols.

3. Quantity of preservative should be equal to the bulk of the organ.
 4. Only two-thirds of the bottle should be filled allowing space for decomposition gases.
 5. Bottles should be properly sealed.
 6. Bottles should be well labeled bearing the name of the deceased, PM no., date and name of the police station.
 7. Sample of the preservative used that is 100ml of spirit or 25gm of sodium chloride should be sent to the lab to rule out contamination.
 8. A Copy of the postmortem report should also be sent along with the viscera.
 9. A sample of seal should be sent along with the viscera.
 10. Viscera along with the sample of preservative and sample of seal should be handed over to the police.
3. The chemical analysis may be faulty due to submission of wrong, insufficient or decomposed material for analysis whereas sometimes faulty techniques used in the lab or inexperienced technical staff resulting in improper evaluation of the death or the quantity of poison is below detection limits.
 4. Some of the alkaloids, toxins of *Clostridium* and *Salmonella* and chemicals like LSD-25 cannot be detected by chemical analysis.
 5. There may be uneven distribution of absorbed poison in various organs depending on the type of poison and mode of administration e.g. when a poison is injected intravenously the blood levels are higher than liver levels.

Poisonings where special viscera is to be preserved:

1. Heart is preserved in Strychnine poisoning.
2. Brain is preserved in Strychnine and Organophosphorus poisoning.
3. Spinal cord is preserved in Strychnine poisoning.
4. Lungs are preserved in gaseous poisoning, Alcohol and Chloroform poisonings.
5. Skin with subcutaneous tissue is preserved in deaths resulting from injectable poisons, drugs of abuse and deaths from snakebite. In all these types of poisonings, control site should also be preserved.

Failure to Detect Poison on Chemical Analysis of the Viscera

Failure to detect poison occurs many a times in forensic practice in an alleged case of poisoning, in spite of circumstantial evidences are pointing towards the poisoning. The poisonous substance may not be detectable due to some of the following reasons.

1. Some individuals may be unusually susceptible to the deleterious effect of the poisonous substance
2. The whole of the poison has already been eliminated from the stomach and intestines by

vomiting or other means such as purging and if absorption had occurred it got detoxified, neutralized, conjugated and finally eliminated by the kidneys etc.

3. The chemical analysis may be faulty due to submission of wrong, insufficient or decomposed material for analysis whereas sometimes faulty techniques used in the lab or inexperienced technical staff resulting in improper evaluation of the death or the quantity of poison is below detection limits.
4. Some of the alkaloids, toxins of *Clostridium* and *Salmonella* and chemicals like LSD-25 cannot be detected by chemical analysis.
5. There may be uneven distribution of absorbed poison in various organs depending on the type of poison and mode of administration e.g. when a poison is injected intravenously the blood levels are higher than liver levels.

Analytical Techniques in Medical Toxicology

The analytical techniques for the detection of poisons, drugs and various chemicals are more dependable and satisfactory than the usual chemical and biochemical methods. The common analytical techniques employed in toxicological analysis are:

- I. *Spectrophotometric*: It is based on the absorbance or transmission of light from a colour reaction at a specific wavelength. It include the following techniques: (i) Calorimetric (ii) Fluorimetric (iii) Automation.
- II. *Chromatographic*: It is based on the migration of compound on adsorbent (solid phase) by a mobile phase. It includes:
 - Thin layer chromatography (TLC).
 - Gas liquid chromatography (GLC).
 - High pressure liquid chromatography (HPLC).
 - Gas liquid mass spectrometry (GL-MS).
- III. *Competitive binding assay or immuno-reactive assay*:
 - Radioimmunoassay (RIA).
 - Enzyme immunoassay (EIA).
 - Fluorescent Polarization immunoassay (FPIA).
 - Immunotubidimetric assay.

Samples to be Collected for Various Analytical Procedures

- i. Blood for recently used drugs.
- ii. Serum or plasma sample for protein bound drugs.
- iii. 24 hours urine sample for free drug analysis.
- iv. Random urine sample for drugs with a longer half life.

Important Points to be Remembered

- The sensitivity and specificity of the technique determines its utility.
- The target of drug testing is either the parent drug or its metabolite.
- The urine and blood are analyzed usually but urine contains 1000 times more drug than the blood so it is the preferred sample.
- Blood is useful to identify recently used drugs
- The cut off value is also critical in determining if a drug test is to be reported as positive or negative.
- False negatives are far more common than false positives.

Comprehensive drug testing is very important mainly for psychiatrists in arriving at precise evaluation and appropriate treatment plan for a drug addict. Diagnosis of polydrug abuse is not possible of which the psychiatrists and forensic toxicologists are now more aware of and are therefore interested in laboratory testing of body fluids for various drugs of abuse.

Thin Layer Chromatography (TLC)

Thin layer chromatography is used as a broad spectrum screen for detection of drugs of abuse. It is most inexpensive technique and does not require any sophisticated instruments. The results of this technique are only qualitative and cannot be quantified. Its major drawback is that it has a low sensitivity (1000-2000 mg/mL) and low specificity. TLC relies on a reproducible migration pattern by the drug on a thin layer adsorbent such as silica gel coated glass plates. The characterization of a particular drug is achieved by colour reactions produced by spraying the plates with colour complexing reagents. The patient's sample

is spotted along with other known drugs on a TLC plate which is put in a solvent chamber. The solvent is dried and sprayed with various complexing reagents. The coloured spots of various known drugs are compared with unknown patient's sample. The location of spot is identified by an

$$\text{Rf number} = \frac{\text{Distance traveled by a drug}}{\text{Distance traveled by solvent from the origin, where sample was spotted}}$$

The spots in the TLC plates can be visualized by UV or fluorescent light. TLC method was designed to detect high dose of a drug, recent drug abuse and toxic levels of drugs. It is a good test for an emergency where drugs taken are not known and presence or absence of a drug is necessary to start the specific treatment. The TLC results are not generally admissible as forensic evidence. In this technique, it must be understood that whether the sample is positive or negative depends upon the concentration of drug in a sample or the sensitivity of cut-off value of the assay. Often the cut-off value is set high to avoid false positives especially for legal purposes. False positive results are quite unusual by TLC but they can be confirmed with a more specific test. The low level of substances is not easily determined by this method.

Gas Liquid Chromatography (GLC)

Gas liquid chromatography separates molecules by the use of a glass or metal tube that is packed with material of a particular polarity. The sample of drug is vaporized at the injection site and carried through the column by a steady flow of gas. The column terminates at detector that permits recording and quantification. The time from injection till a response is observed at the recorder or integrator, referred to as Retention time (RT). Identical compounds travel through column at the same speed because their interaction with packing material is same. The drug of abuse in patient's sample (urine/blood) is extracted and purified before it is injected on to the column. Retention time of patient's sample is compared with RT of known drugs of abuse, processed under similar conditions. If the two are identical, the sample is

confirmed with that particular drug of abuse. GLC is significantly more specific and sensitive than TLC and some drugs are detected better with GLC.

High Pressure Liquid Chromatography (HPLC)

High pressure liquid chromatography is similar to GLC but it uses liquid rather than gas to propel the sample through the column in HPLC. Some drugs are better chromatographed on HPLC e.g., tricyclic antidepressants and Benzodiazepines. HPLC is more specific and sensitive than TLC.

Gas Chromatography-mass Spectrometry (GC-MS)

Gas chromatography-mass spectrometry analyzes a drug according to its fragmentation pattern. Weaker bonds of molecules are broken under stress to produce a fragmentation pattern. A perfect match with a fragmentation pattern in a computer library is considered an absolute confirmation of the drug and is referred to as 'fingerprinting' of molecules. GC-MS is 100-1000 times more sensitive, specific, more reliable and most definitive forensic quality procedure than the TLC system. Being expensive, it is impractical for screening but vital for confirmation of drug presence and identity which is specific for medicolegal cases.

Radioimmunoassay and Enzyme Immunoassay (RIA and EIA)

They operate on the principle of antigen antibody interactions. The drugs of interest are compelled to large molecules and then injected into rabbits or sheep to produce antibodies against the drug. The immunologic methods used for detection employ antibodies against specific drug and competing drug molecules that are tagged enzymatically or that possess radioactivity. Immunoassay reliability depends on how specific and sensitive the antibodies are to a given compound. This is important because cross reacting compounds can result in false positives or falsely elevated results. That is why competitive binding assays are deemed to have much lower specificity than chromatographic techniques. Enzyme Multiplied Immunoassay Technique (EMIT) is the most widely used EIA methodology. It utilizes the

enzyme glucose-6 phosphate dehydrogenase (G_6 -PDH) as the active tag. The assay is based on the catalytic reduction of $NAD \rightarrow NADH$, which is measured spectrometrically at 340 nm. EMIT procedures are less sensitive and specific in cannabinoids than the RIA but EMIT is quite popular and commonly used because no extraction or centrifugation is required and system levels itself to easy automation. EIA screens yielding positives should always be confirmed by alternative methods GC-MS.

Prevention of Poisoning

The poisonings usually result due to lack of proper storage places in the house. Various accidental poisonings can be avoided when the following precautions are taken:

1. The medicines and chemicals should be stored out of reach and site of children and at a considerable height and preferably locked and separately from other household products.
2. The medicines, chemicals and household products should not be transferred and be kept in their original, labeled containers.
3. The medicines should be stored in child resistant packs and all the medicines and chemicals etc. should be properly labeled.
4. Before consuming the label with its directions for use should be properly read.
5. Empty containers of liquid medications, chemicals and household products should be rinsed with water before throwing.
6. Always protect skin and eyes while spraying insecticides with maintenance of adequate ventilation.

POISON INFORMATION CENTRES

The incidence of poisoning has increased markedly during the last decade but has received attention of public professionals and authorities quite recently. Poison information centre is a specialized unit that provides information on poisoning to the community. The main functions of such a centre are:

1. Provision of toxicological information.
2. Advice on the management of poisoning cases
3. Training to the professional and paraprofessionals on first-aid, management and prevention of poisoning.

4. It also provides toxicological laboratory services
5. In developing contingency plans for chemical disaster with other responsible bodies.
6. The center responds to chemical disasters.
7. The center carries out epidemiological and experimental studies to find out the toxic risks and mechanisms.

National Poison Information Centre, Delhi

In India, National poisons information centre was established in 1995 at department of pharmacology, All India Institute of Medical Sciences, New Delhi. The center provides information on poisoning management. It holds teaching and training program on poisoning prevention and management. It also plays an important role in developing contingency plans for chemical disasters and conducts research related to antidotes, environmental and biological monitoring of the chemicals and toxicokinetics. The centre provides information on management and treatment protocols of various poisonings all over the country. The centre provides round the clock service all 365 days of the year. Besides this, the center has the backup of latest literature on poisoning. The Poison Information Consultant after taking an exact history and the relevant details about the product consumed will provide the precise and concise information on the product involved, likely signs and symptoms, pre-hospital treatment, supportive and symptomatic care, antidotal therapy including dosage and availability. Also the entire information is recorded in a specific Performa for evaluation of data and further follow-up. The Poison Information Centre also conducts studies on the pattern of poisoning in a particular area and developing a prevention programme. National Poison Information Centre has readymade information cards covering the signs and symptoms, prehospital and hospital management protocols including therapeutic drugs and antidote dosing schedule on Organophosphates, Carbamates, Organochlorines, Aluminium phosphide, Ethylene dibromide, Pyrethroid, Rodenticides, Corrosives, Tricyclic antidepressants, Benzodiazepines, Phenytoin, Methyl alcohol, Carbon monoxide, Paracetamol, Opioids, Datura, Yellow Oleander, Scorpion sting,

Copper sulphate and Cyanide. Various poisoning manuals and books are available at the centre. The Centre is also in the process of setting up a National Antidote Bank that will stock all essential antidotes.

In India, besides this centre, there are Poison information centres at Ahmedabad, Cochin and Chennai.

Western Australian Poisons Information Centre

This centre serves more than half of the area of Australia including Western Australia, South Australia, the Northern Territory, the Australian Capital Territory and a small region of western New South Wales. The centre provides telephonic consultation to medical professionals and the general public in cases of acute and chronic poisonings. The centre provides toxicological advice on the management of exposures to prescription and nonprescription pharmaceuticals, household and industrial chemicals, plants, animals, pesticides and other agricultural products. The centre also conducts formal training in clinical toxicology and toxinology for all its staff members. The centre also undergoes many research activities.

The American Association of Poison Control Centres

It is a nationwide organization of poison centres and interested individuals with the objectives:

1. To provide a forum for poison centres and interested individuals to promote the reduction of morbidity and mortality from poisonings through public and professional education and scientific research.
2. To set voluntary standards for poison center operations.
The activities of this centre include:
 - i. Certification of regional poison centers and poison centre personnel.
 - ii. Interaction with private and governmental agencies whose activities influence poisoning and poison centers.
 - iii. Development of public and professional education programs and materials.
 - iv. Collection and analysis of national poisoning data.

The association also grants awards and research fellowships.

Victorian Poisons Information Centre

This centre is located at the Royal Children's Hospital, Melbourne. The centre provides timely and safe information in cases of poisonings and suspected poisonings. Telephonic advice is provided on first-aid treatment of poisonings or suspected poisonings with or without referral to the hospital. To the health professionals information is given about formulations of products and management of poisoned patients.

National Poisons Information Service, UK

This centre also responds to queries relating to toxic exposures to drugs and chemicals. The queries from doctors and other healthcare professional are answered. The NPIS on-line database, Toxbase is available for consultation.

LAWS IN RELATION TO THE POISON

Indian Statutes on Drugs

Many legal acts controlling and regulating the manufacture, sale, distribution and possession of drugs and poisons are passed in India. These include the following:

The Opium Act 1857

This Act was the earliest enactment concerned with the issue of licenses for cultivation of poppy, the delivery of produce to the officers of the central government at the established rates, fixing the limits of issuing licenses from time to time within which licenses can be issued and fixation from time to time of prices to be paid for the opium produced in respect of which it ensured.

The Opium Act 1858

This Act was enacted to regulate the possession, transport, export, import, and sale of opium. The primary aim was protection of public welfare by preserving health, eliminating undesirable moral and social effects, commonly associated with the indiscriminate use of this drug.

The Poisons Act, 1919

The Poisons act was passed in 1919 and it prohibits the import of any specified poison except when it is according to the conditions of license and regulates the grant of licenses. It also regulates the possession for sale and the sale of any specified poison. The Act was amended in 1958. The Act followed the Opium Act 1878 as an important drug legislation touching dangerous drugs.

The Dangerous Drugs Act 1930

This Act was enacted in pursuance of the obligation undertaken by the Government of India to ratify the Geneva Dangerous Drugs Convention 1925, for the suppression of illegal traffic in and abuse of dangerous drugs, especially cocaine and morphine. With that object it provided for the exercise of control by the Central government over the cultivation of dangerous drugs; production and supply of opium and opium derivatives; manufacture of or manufactured drugs like cocaine, methadone, morphine and other narcotic substances, medicinal opium; export, import and transshipment of dangerous drug. It deals mainly with opium, Indian hemp and coca leaves.

The Drugs and Cosmetics Act, 1940

The drugs and cosmetics Act deals with the import, manufacture, sell, stock or exhibit for sale, and distribution of proprietary medicine. Every patent or proprietary medicine should display on the label of the container either the true formula or list of ingredients contained. The Act was amended by the Drugs Act 1964 to include Ayurvedic, Siddha and Unani Drugs.

The Drugs Act 1940

The Drugs Act 1940 regulates the import, manufacture, distribution and sale of the following drugs in India:

- Patent or proprietary medicines.
- Vaccines and sera, toxins, toxoids, antitoxins, antigens and other biological products of such nature.

- Vitamins, hormones and analogous products
- The drugs meant for use of humans and animals and those used in the treatment, mitigation or prevention of a disease.

The Drugs Act 1940 was amended by the Drugs Act 1955.

The Drugs (control) Act 1950

This Act seeks to control the sale, supply and distribution of drugs. It provides for the fixation of maximum prices chargeable, the maximum quantity which may at any time be possessed by the dealer or producer, the maximum quantity which may in any one transaction be sold to any person. It also includes the issue of cash memo for sale, markings of prices and exhibiting list of prices and stocks, etc.

The Drugs and Magic Remedies Act 1954

This Act prohibits the advertisements of magic remedies for:

1. The procurement of miscarriage or prevention of conception in women
2. Improvement and maintenance of the capacity of sexual pleasure
3. To correct menstrual disorders
4. For the diagnosis, cure, mitigation, treatment or prevention of any disease, disorder or condition specified in the Schedule to the Act or the rules

The Act also specifies the exceptions to these advertisements.

The Narcotic Drugs and Psychotropic Substances Act 1985

This Act repeals the Opium Act 1857, The Opium Act 1878 and the Dangerous Drugs Act 1930. The Act shall extend to whole of India. The object of the Act is to consolidate and amend the law relating to narcotic drugs, make stringent provisions for the control and regulation of operations relating to narcotic drugs and psychotropic substances and make provisions for matters connected therewith. The Act defines Narcotic drug, psychotropic substance, medicinal cannabis, opium, opium derivatives, opium poppy, poppy straw, poppy straw concentrate, cannabis (hemp), canna-

bis plant, coca derivative, coca leaf, manufacture, production, preparation and addict etc.

Narcotic drug: It is defined as meaning coca leaf, cannabis (hemp), opium, poppy straw and includes all manufactured drugs:

Manufactured drug: It means:

1. All coca derivatives, medicinal cannabis, opium derivatives and poppy straw concentrate.
2. Any other narcotic substance or preparation that the Central government declares to be a manufactured drug.

Psychotropic substance: It means any substance, natural or synthetic, or any natural material or any salt or preparation of such substance or material included in the list of psychotropic substances specified in the schedule.

Addict: It means a person addicted to any narcotic drug or psychotropic substance.

Small quantity: It means such quantity as may be specified by the Central government in the official Gazette. The burden of proving that it was intended for personal consumption shall lie on the person found in possession of the drug or psychotropic substance. The small quantities as specified in the central govt. in the official gazette given in the table below:

Drug	Quantity
Heroin	5 grams
Cocaine	2 grams
Hashish/Charas	100 grams
Opium	25 grams
Ganja	1 Kg

Offences and penalties: Chapter IV of the Act defines offences and penalties under the Act. The Act provides punishment with rigorous imprisonment which shall not be less than 10 years but which may extend to 20 years with fine which shall not be less than one lakh rupees but which may extend to two lakh rupees for the following contravention. The Court may for the reasons recorded in the judgment impose a fine exceeding two lakh rupees. The contraventions for which such punishment is contemplated, may be of any rule or order made or condition of a licence granted thereunder relating to poppy straw, coca plant and coca leaves, prepared opium, opium poppy and

opium, cannabis plant and cannabis, manufacture dugs and preparations, psychotropic substances, embezzlement of opium by cultivator, illegal import or export or transshipment of narcotic drugs and psychotropic substances, external dealings in narcotic drugs and psychotropic substances in contravention of S.12 (S.24), allowing premises etc. to be used for the commission of an offence punishable under the Act (S.26).

LAWS IN RELATION TO POISONING IN THE INDIAN PENAL CODE

Section 284 I.P.C: 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 any 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.

Section 299 I.P.C: 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 an act to cause death, commits the offence of culpable homicide.

Hence if A administers a substance to B with such intent or knowledge and thereby causes B's death, A may be convicted under this section of the offence of committing culpable homicide, irrespective of whether the substance administered may or may not strictly be called a poison. For it is the intent which suffices to constitute a crime, irrespective of the dose or even the nature of substance.

Section 304A I.P.C: 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.

Section 324 I.P.C: Voluntarily causing hurt by dangerous weapon or means: Whoever, except in the case provided 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 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.

Section 326 I.P.C: Voluntarily causing grievous hurt by dangerous weapons or means: Whoever, except in the case provided for by the 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 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 ten years and shall also be liable to fine.

Section 328 I.P.C: 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.

Corrosive Poisons

Corrosives are the poison that fixes, destroys and causes erosion of the surface coming in its contact. Some organic acids like oxalic and carbolic acid act as corrosives in concentrated form as do carbonates of sodium and potassium. Certain metallic salts e.g. sodium chloride, potassium cyanide, ferric chloride, chromates and bichromates of alkalis also act as corrosives.

Classification

1. *Strong acids*:
 - A. Inorganic or Mineral: (i) Sulphuric acid (ii) Hydrochloric acid (iii) Nitric acid.
 - B. Organic (i) Carbolic acid (ii) Oxalic acid (iii) Acetyl salicylic acid
2. *Acid like corrosives*: (i) Dimethyl sulphate (ii) Diethyl sulphate (iii) Ozone, atmospheric oxidants (iv) Sulphur dioxide
3. *Alkalis*: (i) Sodium hydroxide (ii) Potassium hydroxide (iii) Sodium carbonate (iv) Potassium carbonate (v) Ammonia
4. Phosphates such as polyphosphates.

Mode of Action

The mode of action of inorganic or mineral acids like sulphuric acid, hydrochloric acid and the nitric acid are:

1. Causes only local action but no remote effects on the system.
2. In concentrated form, they cause corrosion and destruction of the tissue
3. Extracts the water from the tissues.
4. Fixes, destroys and erodes the tissues.
5. Converts Haemoglobin in to Haematin.
6. Causes coagulation necrosis by precipitation of proteins.

7. The mucosa of the Oesophagus is relatively resistant to the acids.
8. Mucous membrane of stomach especially pyloric region is very susceptible and in this region necrosis commonly occurs.
9. Complications develops from 3 weeks to 3 months of ingestion of the substance..
10. Corrosives act as irritant and when well diluted act as stimulant.

General Principles of Treatment

1. The stomach wash should not be given as there are chances of perforation of stomach. However Levin tube can be used for stomach wash within half an hour of ingestion of poison. The tube should be passed softly and with due care avoiding the risk of perforation of stomach and oesophagus.
2. Emesis is to be avoided as there is risk of perforation of already thinned out stomach and oesophagus
3. Acids should be immediately diluted and neutralized by drinking plenty of water containing a tablespoonful of calcium oxide, magnesium oxide or aluminium hydroxide gel. If these are not available then Demulcents like vegetable oil, soap solution, milk, limewater or white of an egg should be followed by Barley water and olive oil.
4. Bismuth subcarbonate 30gm should be given.
5. Morphine 15 mg i.m or i.v or Meperidine HCL 50-150mg orally or i.v should be given for pain.
6. 10mL of 10% Calcium gluconate should be given intravenously

7. Blood transfusion can be given if needed.
8. Tracheostomy can be performed if oedema of glottis is present.
9. Oxygen inhalation and artificial respiration can be given, if necessary.
10. Corticosteroids should be administered to prevent oesophageal strictures.
11. To prevent formation of strictures later on, ½ inch mercury filled bougie should be passed daily.
12. In case of skin burns, wash with large quantities of water or apply paste of sodium bicarbonate.
13. Eye burns are treated symptomatically after irrigating with water for 10-15 minutes.
14. Use of strong alkalis such as carbonates and bicarbonates of sodium and potassium should be avoided as they produce CO₂ and can cause distension and perforation.

Causes of Death

- I. Death occurring within few hours can be due to shock or spasm or oedema of glottis
- II. Within 24hrs, death results from perforation of stomach leading to peritonitis and shock.
- III. Within first week the death may result from septic absorption
- IV. After months or years due to exhaustion and malnutrition due to oesophageal or pyloric stricture or incurable dyspepsia due to destruction of coats of mucous membrane of the stomach.

SULPHURIC ACID (OIL OF VITRIOL)

Properties

- Pure Sulphuric acid is colourless with no fumes and it chars, and blackens the skin.
- Commercial Sulphuric acid is dark or brown in colour due to the addition of impurities like lead sulphate, arsenic or nitric acid.
- Nordhausen acid or H₂S₂O₇ (pyrosulphuric acid) is stronger; brown, oily and fuming liquid used in the manufacture of indigo.
- Sulphate of indigo is 1: 10 (H₂S₂O₇)
- Sulphuric acid is chiefly used for preparing chlorine, for dissolving metal, for cleansing

drains, for medicinal purposes, and to remove fur from kettles

Signs and Symptoms of Sulphuric Acid Poisoning

Immediately after swallowing are:

1. Intense burning pain in mouth, throat, oesophagus and up to the stomach.
2. Dribbling of acid mixed with saliva on the angles of mouth and chin causing corrosion and brownish discolouration of skin over angles of mouth, chin and over the chest
3. Frothy eructation, retching and vomiting; vomited matter is acid, contains blood, mucous and shreds of mucous membrane
4. *Oral cavity:* Mucosa of mouth and lips are softened, excoriated, corrugated and covered with dirty white necrotic membrane that assumes brownish black discolouration. The oral cavity is full of secretions consisting of saliva, blood, mucous making speech and swallowing difficult and may even result in death from asphyxia.
5. The tongue is swollen, excoriated and discoloured with white coating that becomes dark later on. It may get disorganized into a shapeless, pulpy mass
6. The teeth are dead chalky white deprived of polish
7. Lips are swollen, excoriated, brown or black streaks resulting from the action of acid running from the angle of mouth to the chin and chest
8. Intense thirst and an attempt to drink water brings vomiting that leads to dehydration. Along with vomiting, there is extreme dysphagia
9. Voice is hoarse and husky and speech is painful and difficult due to the inflammation of epiglottis, uvula etc.
10. Breathing is difficult and noisy due to swollen and inflamed fauces and tender abdomen
11. Asphyxia may result from glottic oedema or aspiration of regurgitated acid contents
12. Bowels are constipated with marked tenesmus and straining during defecation.

13. Urine is scanty or suppressed with pain during micturition.
14. General condition of collapse like cold, clammy skin, decreased Blood Pressure and increased pulse may be there.
15. Mind remains calm until the end.
16. Cold clammy skin, feeble pulse and difficult respiration, convulsions and death in 12-24 hours
17. Complications in the form of mediastinitis or peritonitis from perforation of oesophagus or stomach and stricture of oesophagus may develop within a few weeks

Acid burns: These are full thickness burns that have well defined edges and tend to heal slowly with scar formation.

Autopsy Findings

Postmortem findings depend upon the quantity and strength of acid and the time of survival.

A. *External findings:* Evidence of acid corrosion and chemical burns with brownish black parchment like and corroded spots are seen over chin, cheek, neck and chest resulting from trickling of the acid. Excoriation of lips, corrosion of mucosa of mouth and tongue, chalky-white teeth are characteristic. Mucous membrane of mouth, tongue and lips shows brown or brownish black corroded spots with necrosis that are grayish white initially but changes to brownish black and leathery. When taken by spoon, lips and mouth escape injury.

B. Internal findings

1. The changes produced from corrosive poisoning are limited to the upper digestive tract and respiratory system.
2. The mucous membrane of oesophagus is inflamed, swollen by oedema and severe interstitial haemorrhage is present even when corrosion is absent. The perforation is rare
3. The mucous membrane of stomach is inflamed, oedematous blackened with peppery feel.
4. Peritoneal cavity is filled with black grumous acidic liquid containing mostly altered blood



Figure: 36.1: Mucosa of stomach as seen in sulphuric acid poisoning

from escape of the acidic stomach contents into peritoneal cavity following perforation of stomach (Fig. 36.1).

5. The greater part of the stomach is converted in to soft, boggy, black mass that readily disintegrates on touching. The mucosal ridges are more damaged than intervening furrows. The mucosa on stripping shows intense congestion and inflammation underneath. The perforated stomach has softened walls and edges of rupture looking black and irregular.
6. The small intestine especially duodenum shows evidence of irritation.
7. As a result of perforation of oesophagus or stomach, the thoracic and abdominal viscera is blackened and corroded by acid with features of chemical peritonitis.
8. Corrosion and severe inflammation of larynx and trachea may be found.
9. Oesophageal strictures in delayed deaths.
10. If patient survives longer fatty degeneration and toxic swelling of liver and kidney may be seen.

Diagnosis

1. Haemoconcentration in red blood cells is noticed.
2. Diffuse mottling of lung fields on X-ray when there is inhalation of acid vapour.

3. *Chemical tests*

- i. When sulphuric acid is mixed with barium nitrate or chloride, white precipitate of barium sulphate is produced.
- ii. Strong sulphuric acid chars organic matter.

Medicolegal Aspects

(i) Accidental as it is mistaken for glycerine or castor oil (ii) Mostly it is employed for suicidal purposes due to its easy availability (iii) Not used for homicidal purposes (iv) Acts as an abortifacient when injected in to vagina (v) Employed for the purposes of vitriolage causing grievous hurt (vi) Cases are there when acid is thrown on the private parts, out of jealousy in extramarital affairs.

Vitriolage or Vitriol Throwing

Vitriolage is throwing of strong sulphuric acid, concentrated mineral acid, corrosive alkalis, carbolic acid, or the juice of marking nut over the face or body of the victim for the purpose of disfiguring the face, destroying the vision for causing injury on the body or even destroying the clothing of the victim. Malicious persons do this when they have a hatred or enmity to the victim. Some people use old electric bulb filled with acid to carry out their motive.

Local effects: (i) There is severe burning pain and corrosion of the tissues with formation of brownish black burned areas that later on leave a permanent scar. (ii) The stains on skin and cloth are yellowish when nitric acid is used but blackish when sulphuric acid is used. (iii) Blindness may result if eyes are involved. (iv) Extensive disfiguring injuries are caused on the face. (v) Death may occur from shock, toxæmia or infection from severe chemical burns inflicted on the skin. The burns are penetrating, painless and the acid devitalizes the tissues and predisposes to infection. Slow repair and formation of scar tissue with contracture occurs.

Treatment

1. Wash with plenty of water or soap solution.
2. Apply magnesium oxide or carbonate as thick paste.

3. Cover the raw surface with tannic acid jelly or soframycin or penicillin tulle gauze dressing.
4. Wash the eyes with plenty of water and irrigate with 5 gm of sodium bicarbonate and one ounce of water. Subsequently few drops of castor or olive oil are to be dropped in to eye. The corneal ulcers are treated with atropine ointment, hydrocortisone and antibiotics.

Medicolegal Aspects

(i) Blindness amounts to grievous injury (ii) Scar tissue formation also amounts to grievous injury (iii) Vitriolage amounts to dangerous injury.

NITRIC ACID— HNO₃ (AQUA FORTIS)

Pure nitric acid is colourless, clear liquid gives fumes when exposed to air. It has a peculiar chocking odour. A powerful oxidizing agent dissolves all metals except gold and platinum. It destroys organic matters with formation of xanthoproteic acid that imparts deep yellow colour to the affected tissues.

Signs and Symptoms

The signs and symptoms produced by ingestion of concentrated nitric acid are similar to those of sulphuric acid but since it has tendency to char the tissues, perforation of stomach does not occur.

1. Lips, tongue and mucous membrane of mouth are soft. Initially it is white then yellow due to formation of xanthoproteic acid.
2. The clothing is also stained yellow due to xanthoproteic reaction
3. Teeth and clothes are yellow stained.
4. Abdomen is more distended and tender due to formation of large quantities of gas by direct action of acids on the organic matter of stomach.
5. Violent vomiting occurs and the vomitus is yellowish brown containing altered blood.
6. Gaseous eructations are more frequent and distressing.
7. Oliguria and anuria occurs and albumin and casts are seen in the urine.
8. As nitric acid like hydrochloric acid but unlike sulphuric acid is volatile and gives fumes at

room temperatures, the effects produced by the inhalation of fumes are: (i) Irritation of the eyes with lachrymation and sneezing (ii) Photophobia (iii) Burning in the throat (iv) Cough (v) Feeling of constriction in the chest. (vi) Dyspnea—The death may occur immediately from suffocation due to oedema of glottis or later on from pulmonary oedema or bronchopneumonia.

Fatal period→12-24 hrs

Fatal dose→10-15 ml.

Tests for Nitric Acid

- i. When Ammonia water is added to the yellow stains, it turns to yellow.
- ii. When iodide and ammonia is added to the yellow stains, the stains disappear.
- iii. When bile and ammonia is added to the yellow stains, there is no change
- iv. If strong solution of ferrous sulphate and Sulphuric acid is added to a solution containing nitric acid, a brown ring is formed at the junction between the two. The test is for the presence of nitrates.

Autopsy findings

1. Postmortem appearances are similar to those of sulphuric acid but tissues are stained yellow.
2. Oesophagus and stomach are not stained yellow but is brown or brownish black due to the formation of acid haematin.
3. Perforation is not so common.
4. If the death is from inhalation of fumes then the respiratory passages are congested; lungs are congested and oedematous and the lining membrane of right auricle may show inflammatory changes.

Medicolegal Aspects

(i) Mostly accidental or suicidal (ii) rarely homicidal.

HYDROCHLORIC ACID (HCL)

Pure Hydrochloric acid is colourless and fuming liquid with strong pungent irritating odour. The strong solution gives fumes even at ordinary air and temperatures. When the solution is ingested, its liberated gas is also inhaled. It is a natural

constituent of the fluids of stomach and bowel. It is used for preparing chlorine, for dissolving metal, for cleaning drains, to remove fur from kettles and as medicine.

Muriatic acid is the solution of hydrochloric acid gas in water having yellow colour and fumes strongly in damp air. It is used commercially and is less destructive than sulphuric and nitric acids. HCL does not stain the skin and mucous membrane but stains dark clothes reddish brown. Since it is volatile, it readily affects the Respiratory tract mucosa.

Signs and Symptoms

1. Hydrochloric acid is less active than the other two acids hence symptoms are much milder.
2. It does not corrode or damage the skin.
3. It readily destroys the mucous membrane.
4. Mucous membrane is gray or gray white changing to brown or black.
5. Inhalation of fumes causes: (i) Irritation of the air passages (ii) Spasm of glottis (iii) Symptoms of suffocation (iv) Cough, Dyspnea and cyanosis.

Chronic Poisoning

Constant exposure to fumes produces chronic poisoning. The symptoms and signs of chronic poisoning are: (i) Coryza (ii) Conjunctivitis (iii) Corneal ulcer (iv) Pharyngitis (v) Bronchitis (vi) Inflammation of gums (vii) Loosening of teeth.

Fatal dose→15-20 ml

Fatal period→18-24 hours.

Autopsy Findings

1. The findings are the same as that of sulphuric acid poisoning but there is less tendency to charring and tissue destruction.
2. Oral cavity and oesophageal mucous membrane is ashy grey in colour but may get blackened, swollen and hardened if the acid is concentrated.
3. The stomach contains brownish acidic fluid and mucosa is corroded and grayish white in colour
4. Perforation is rare.

Chemical tests: On addition of silver nitrate to a solution of hydrochloric acid, a white precipitate of silver chloride is formed.

Medicolegal Aspects

(i) Mostly suicidal poisoning is common (ii) There are a few cases of accidental poisoning (iii) It is very rarely homicidal (iv) It is rarely injected in to the vagina to produce abortion and it causes atresia of the vagina (vi) It is used for erasing writing; attempts for forgery may be there.

CARBOLIC ACID

This is hydroxy benzene obtained from coal tar oil by fractional distillation. Pure carbolic acid has colourless, prismatic needle shaped crystals with burning sweetish taste. It turns pink and liquefies when exposed to air. It has carbolic smell, is slightly soluble in water and freely soluble in glycerine, ether and alcohol. It is used as an antiseptic and disinfectant agent.

Commercial carbolic acid is dark brown liquid containing several impurities chiefly cresol. Carbolic acid and its derivatives are commonly used as disinfectants, antiseptics, germicides, caustics and preservatives kept in cupboards and almirahs. Lysol is as dangerous as phenol, which is used in the manufacture of explosives, fertilizers, textile, rubber, plastics, dyes, drugs and in preservations of vaccines etc.

Significance of change in colour: It serves as a warning of toxic properties of acid when use as an antiseptic on the wounds

Poisoning by carbolic acid is known as **Carbolism**.

Absorption: It is readily absorbed from gastrointestinal tract, respiratory tract, rectum, vagina, serous cavities, wounds and skin.

Elimination: Phenol is converted in to hydroquinone and pyrocatechol in the body before it is eliminated in the urine. Only traces are excreted in lungs and it is partly detoxified in the liver. Complete elimination occurs by 36-48 hours

Fatal dose→10-15 gm

Fatal period→3-4 hours.

Mode of Action

1. Carbolic acid acts locally as a corrosive and systemically as narcotic, induces sleep and drowsiness.

2. Carbolic acid has a great penetrating power as it coagulates protein but does not occur in chemical combination with them.
3. When applied to the skin, it produces burning sensation, numbness, and anaesthesia due to its action on the nerve endings.
4. It produces a white opaque eschar, which falls off in a few days and leaves a brown stain that may persist for several weeks.
5. It causes irritation and necrosis of the mucous membrane that may slough and get inflamed.
6. In small doses, it stimulates respiratory centre and methemoglobinemia can occur following poisoning with hydroquinone.

Signs and Symptoms

The signs and symptoms arise immediately after swallowing the acid (i) Burning sensation in the mouth and throat. (ii) Mucous membrane of the mouth and lips is hard and white (iii) Rapid absorption of acid in to the blood stream is soon followed by giddiness insensibility and coma (iv) Face is pale and cyanosed (v) Pupils are constricted (vi) Temperature is decreased (vii) Pulse is increased, feeble and thready (viii) Respiration is slow and laboured (ix) Smell of carbolic acid is present in the breath (x) Convulsions and lockjaw may be present.

(xi) **Carboluria:** Urine is scanty and suppressed, when passed there is normal or greenish hue that becomes dark or olive green on exposure to air due to oxidation of pyrocatechol and hydroquinone in air that are the oxidation products of carbolic acid. Phenol is partly oxidized; partly unchanged that is excreted in the urine; partly free and partly unstable combination with Sulphuric acid and glucuronic acid

(xii) **Oochronosis:** The hydroquinone and pyrocatechol may cause pigmentation in the cornea and venous complexes. and various cartilages, it is seen in chronic poisoning. It is normal associated with alkaptonuria and gets deposited in cartilage.

Causes of death: (i) Syncope (ii) Asphyxia due to respiratory failure, oedema of glottis and complications such as pneumonia.

Treatment

1. Emetics at times fail due to anaesthetic effect
2. Stomach wash: The tube is passed carefully and the wash is done with plenty of warm water along with charcoal, olive oil, castor oil, magnesium or sodium sulphate. The combination with phenol of these substances forms a harmless compound. The soap solution or 10% glycerine can also be used, until the washing are clear and odourless. Then 2 ounce of castor oil or liquid paraffin is left in the stomach.
3. Demulcents such as milk and white of an egg are given.
4. To combat circulatory collapse, i.v. Saline containing 7gm/litre of sodium bicarbonate is administered. Sodium bicarbonate also helps to dilute carbolic acid content of blood and it promotes excretion by promoting the flow of urine.
5. Oxygen inhalation and artificial respiration when needed.

Autopsy Findings

External findings

1. Dark brown excoriations may be seen at the angle of mouth and chin.
2. Signs of splashing may be present on the other body parts.
3. Tongue is white or swollen.
4. Smell of phenol comes from the mouth.
5. Mucous membrane of mouth is corrugated, sodden and white or ash gray small submucosal haemorrhages may be seen.

Internal findings

1. Oesophageal mucosa is tough, white gray, corrugated and arranged in longitudinal folds.
2. Stomach mucosal folds are swollen and covered with opaque gray or brown mucous membrane with a leathery feel (Fig. 36.2). The intervening furrows are less damaged and the mucosa is often dark red, partly separated and necrotic with severe congestion of underlying mucosa. The stomach may contain reddish fluid



Figure: 36.2: Mucosa of stomach as seen in carbolic acid

- mixed with mucus and shreds of epithelium emitting odour of phenol.
3. Duodenum and upper part of small intestine shows changes similar to the stomach.
 4. Liver and spleen are in the form of white, hardened patch when in contact with stomach.
 5. Kidney is inflamed and shows haemorrhagic spots.
 6. Lungs are congested and oedematous.
 7. Brain is congested and at times oedematous.

Medicolegal Aspects

(i) Used for suicidal purposes (ii) Accidental poisoning results from its medicinal use and when it is introduced in to vagina for abortion. Application of phenol preparations on wounds, ulcers, abraded skin, injections into abscess cavity, rectum, vagina or uterus can also result in accidental poisoning (iii) Due to its peculiar phenolic odour and taste, it is rarely is it used for homicidal purposes.

OXALIC ACID (ACID OF SUGAR AND SALT OF SORREL)

Oxalic acid is colourless, transparent and prismatic crystals resembling magnesium sulphate. It is an organic corrosive acid. It is prepared from sugar by oxidation with nitric acid or by heating sodium or potassium formate. The differences

between oxalic acid and magnesium sulphate are summarized in the table below:

Difference in oxalic acid and magnesium sulphate		
Features	Oxalic acid	Magnesium sulphate
Taste	Sour and acid	Nauseating and bitter
Reaction	Strongly acid, pH<7	Neutral, pH 7
On heating	Sublimes	Fixed
With sodium carbonate	No precipitate	Precipitates
Stains of ink or iron-moulds	Disappears	No action

Commercial use of oxalic acid: (i) Calico-printing (ii) Manufacture of straw hats (iii) Cleaning brass and copper articles, wooden surface and leather (iv) Photography (vi) Used by book binders.

Domestic use of oxalic acid: Oxalic acid is a common household remedy used for removing stains of ink and marks from clothing.

Illegal uses of oxalic acid: It is used for removing writing and signature from the paper.

Mode of Action

Oxalic acid has local as well as remote or systemic action:

Local action: (i) In solid or concentrated form or in large quantities, it acts as a corrosive (ii) In weaker solutions, it acts as an irritant and this is more evident in the nervous system (iii) When applied to the wound, it acts as a poison.

Remote action: (i) It precipitates ionized calcium and produces symptoms of hypocalcaemia (ii) Large doses cause rapid death from shock (iii) Causes tubular necrosis, uremia and death within 2-14 days.

Fatal dose→15-20 gm

Fatal period→1-1½ hours.

Signs and Symptoms of Oxalic Acid Poisoning

The signs and symptoms produced vary with the amount taken and the concentration of the solution.

Fulminating Poisoning

Large doses about 15gm or more produces immediate symptoms and death within minutes. The following symptoms are produced: (i) Burning, sour and bitter taste in the mouth (ii) Throat constriction (iii) Burning pain in the mouth and up to the stomach; pain starts in the epigastrium and radiates to the whole abdomen (iv) Tenderness (v) Nausea, vomiting and eructation; vomitus contains blood and mucous—coffee ground colour (vi) Thirst may be there (vii) Death usually occurs before the bowel is affected but if the life is prolonged, diarrhoea may occur.

Acute Poisoning

(i) In a large dose when patient survives for hours, the symptoms are more of hypocalcaemia and less of digestive upset (ii) Muscle irritability and tenderness (iii) Convulsions (iv) Numbness and tingling of fingertips and legs (v) Cardiovascular collapse (vi) Stupor and coma at times.

Delayed Poisoning

Symptoms of ureamia such as scanty or suppressed urine containing traces of blood, aluminium or calcium oxalate crystals are found.

Treatment

1. Stomach wash by calcium lactate to test in each lavage or gluconate
2. Antidote is calcium lactate or gluconate, it converts oxalate to calcium oxalate that is insoluble; 10ml of 10% calcium gluconate is administered intravenously at regular intervals.
3. Parathyroid extract 100units i.m in severe cases.
4. Demulcent drinks
5. Bowels may be relieved by enema.
6. Symptomatic treatment.
7. dialysis or exchange transfusion may be done.

Autopsy Findings

1. Skin is rarely burned.
2. The strong solution may cause white as if bleached appearance of mucous membrane of mouth, tongue and lips. It may be red due to irritation.

3. Outer coat of stomach is inflamed and reddened with patchy softening, perforation is rare. The contents of the stomach are brownish due to acid Haematin. At times the whole stomach is corroded and perforated. If death is immediate, stomach is pale and not corroded.
4. Kidneys are swollen and congested, tubules show oxalate crystals.
5. All other organs are congested.

Medicolegal Aspects

(i) Accidental commonly as it is mistaken for magnesium sulphate (ii) Rarely it is found as suicidal poison (iii) Very rarely it is used for homicidal poisoning owing to its bitter taste (iv) It is used as an abortifacient rarely at times.

CORROSIVE ALKALIES

The strong solutions of alkalis act as corrosives but in dilute form, they act as irritants. Concentrated corrosive alkalis are more dangerous than acids. The chief alkaline corrosives with their fatal doses are the following:

1. Potassium hydroxide (caustic potash)—5 gm
2. Sodium hydroxide—5 gm
3. Potassium carbonate (caustic soda)—18 gm
4. Sodium carbonate (Washing soda)—30 gm
5. Ammonia—30 gm; gaseous ammonia dissolved in water forms strong solution of ammonia that is strongly alkaline, pungent liquid.
6. Ammonium carbonate.

Lye is a mixture of caustic soda and sodium bicarbonate that is used for cleaning purposes.

Uses of corrosive alkalis: (i) Caustic potash and caustic soda is extensively used for commercial purposes as cleansing agents in washing powders, pipe-cleaners (ii) Used as liniments in medicines.

Action Produced

1. The caustic action results from the absorption of water from the tissues.
2. They precipitates proteins.

3. They combined with proteins to form proteinaes and with fats to form soaps, thus produces soft, necrotic deeply penetrating areas on contact with tissues.

Signs and Symptoms

Signs and symptoms produced are similar to that of corrosives: (i) Soft, oedematous, translucent, swollen, red brownish scar formation is there (ii) Charring is not present (iii) Vomited matter is alkaline (iv) Purging is frequent; mucous and blood is present in the stools (v) When a strong solution of alkali is ingested, the mucosa of mouth and lips is discoloured and blister formation is there.

Inhalation of Ammonia Vapours

The following symptom are produced, when the ammonia vapours are inhaled: (i) Congestion and watering of the eyes (ii) Sneezing, coughing and choking (iii) Sudden collapse and death may occur (iv) Death is due to suffocation and oedema of glottis.

Treatment

1. Neutralize the poison by giving vegetable acids and water.
2. Demulcents are given.
3. Stomach can be washed carefully.
4. Oxygen inhalation, when ammonia vapour is inhaled.
5. Symptomatic treatment.

Autopsy Findings

The findings are similar to corrosives.

Medicolegal Aspects

(i) Accidental poisoning is common as it is mistaken for medicines (ii) Homicidal poisoning is rare (iii) Poisoning by inhalation of ammonia vapour is common (iv) Sometimes caustic soda is thrown over the face and body producing chemical burns.

Metallic Poisons

LEAD

Lead is one of the truly toxic elements present in the human body. It is a poison though in evidence in criminal history than arsenic or antimony is of infinitely greater importance owing to its industrial incidence. The compounds of lead are more dangerous than the lead itself, except when lead is in volatile state. Lead when inhaled acts 10 times more dangerously than when ingested.

Uses of Lead

(i) Lead is a major constituent of the Lead-acid battery used extensively in car batteries. (ii) Lead was used as a white pigment in Lead paint. (iii) Lead is used as a coloring element in ceramic glazes. (iv) Lead was used for plumbing in Ancient Rome. (v) Lead sticks were used as pencils but has been replaced by graphite for the last 450 years (vi) Lead is used as projectiles for firearms and fishing sinkers because of its density, low cost versus alternative products and ease of use due to relatively low melting point (vii) Lead is used in some candles to treat the wick to ensure a longer, more even burn. However, because of the dangers, European and North American manufacturers use more expensive alternatives such as zinc (viii) Lead is used as shielding from radiation (ix) Lead glass is comprised of 12-28% lead. It changes the optical characteristics of the glass and reduces the transmission of radiation (x) Tetraethyl lead has been used in leaded fuels to reduce engine knocking. However, this is no longer a common practice in the Western World due to health concerns (xi) Lead is used as electrodes in the process of electrolysis.

Mechanism of Action

Most lead poisoning symptoms are thought to occur by interfering with an essential enzyme Delta-aminolevulinic acid dehydrase (ALA). ALA is a zinc-binding protein which is important in the biosynthesis of heme, the cofactor found in hemoglobin. It combines with sulphhydryl groups and inhibits tissue enzyme systems. Lead also directly causes haemolysis of mature red cells. It affects erythropoiesis, and myelopoiesis by depressing the bone marrow, resulting in anemia, basophilic stippling of RBCs, anisopoikilocytosis and thrombocytopenia.

Absorption of Lead

Lead is absorbed into the system by: (i) Inhalation (ii) Gastro-intestinal tract by ingestion (iii) From abraded skin.

Absorption of lead occurs at the rate of 1-2mg/hour for a prolonged period. 0.01-0.1 μ diameters are readily absorbed. Plumbism is caused ten times more by inhalation as fine dust or fumes. Lead is deposited in bones as tribasic lead phosphate that is an insoluble form. The lead is eliminated mainly in faeces and to a less extent in urine, bile, saliva, milk and sweat. Absorption of lead occurs within 3-6 minutes. Lead has a blastophoric effect on the spermatozoa.

Idiosyncrasy: Some persons are not affected by the effects of lead compounds whereas persons addicted to alcohol are more prone to the effects. In addition, individuals with gout are affected soon.

Daily Intake of Lead

- Daily intake of lead is 0.45 mg.
- 0.2-0.4 mg is taken along with the food.
- 0.02 mg is ingested along with water.
- 0.03 mg is taken by inhalation.
- 0.04 mg is inhaled while smoking.

Salts of Lead that are Poisonous

1. *Lead acetate* or sugar of lead; tastes sweet.
2. *Lead sub-acetate*.
3. *Lead carbonate* or white lead; used in oil painting. Commonly responsible for lead poisoning in children who suck and bite toys.
4. *Lead nitrate*; used in calico printing.
5. *Lead sulphate*; is supposed to be non-toxic.
6. *Lead chromate* (yellow chrome); poisoning occurs from ingestion of sweets coloured with the salt.
7. *Lead chloride*; pigment, yellow oxychloride.
8. *Lead iodide*; bright yellow powder.
9. *Lead sulphide* (galena); in powder form as *soorma* (in place of Antimony sulphide) used to colour the eyes.
10. *Lead monoxide* (Litharge, *Mudrasang*, Massicot); is pale brick red, used by the painters. Hair dyes also contain it.
11. *Lead tetraoxide* (Red lead or *sindur*) is used as a pigment.
12. *Lead tetra-ethyl*: It is an organo-metal compound, highly toxic, heavy, oily liquid and volatile at ordinary temperatures. It is added to the petrol to prevent it from knocking. This mixture is known as ethyl petrol or ethyl gasoline, used as a fuel for motorcars. Lead tetra-ethyl is absorbed by inhalation or through the intact skin. Poisoning occurs in persons engaged in the manufacturing of lead tetra-ethyl.

The main compounds that produce poisonous symptoms are acetate, carbonate, chromate and oxides of lead.

ACUTE LEAD POISONING

The symptoms of lead poisoning include: (i) Neurological problems, such as reduced IQ (ii)

Nausea and abdominal pain (iii) Irritability, insomnia and excess lethargy (iv) Hyperactivity and headache (v) In extreme cases, seizure and coma (vi) There are also associated gastrointestinal problems, such as constipation, diarrhea, abdominal pain, vomiting, poor appetite, weight loss (vii) Other associated affects are anemia, kidney problems, and reproductive problems (viii) In humans, lead toxicity often causes the formation of bluish line along the gums, which is known as the **Burtonians's line** (ix) A direct link between early lead exposure and extreme learning disability has been confirmed by multiple researchers and child advocacy groups.

Acute poisoning occurs mainly due to lead acetate. The symptoms start immediately: (i) Metallic taste (ii) Burning sensation (iii) Dryness of throat (iv) Intense thirst (v) Vomiting within ½ hour; The vomitus is mixed with blood (vi) Colicky pain coming at intervals but relieved by pressure (vii) Constipation is a constant feature (viii) Urine is scanty (ix) Tongue is coated (x) Foul offensive breath (xi) Nervous symptoms like drowsiness, insomnia, headache, vertigo, muscle cramps, convulsions and paralysis of lower limbs (xii) Wasting follows and death occurs due to exhaustion.

Acute poisoning is very rare and usually terminates in recovery hence it has little toxicological significance.

Acute Lead Encephalopathy

It is rare in the adults but many cases are reported in children. The clinical features include: (i) Anaemia (ii) Mild colic (iii) Vomiting (iv) Apathy (v) Drowsiness (vi) Stupor (vii) Ataxia and (viii) Hyperactivity.

Normally, blood contains 0.03 mg/100 ml of lead. In poisoning, the concentration of lead is usually between 0.1-0.6 mg/100 ml. In acute poisoning by lead tetra-ethyl, central nervous system is usually affected resulting in the signs and symptoms like irritability, convulsions, headache, delirium, low blood pressure, bradycardia and hypothermia. Sometimes nausea, vomiting, and loss of appetite occur.

Subacute Poisoning

Intake of repeated small doses of a soluble salt like lead acetate results in sub-acute poisoning. The presentation of subacute poisoning: (i) A blue line on the gums (ii) Gastro-intestinal symptoms such as nausea and vomiting (iii) Face is livid and shrunken and bears an anxious look (iv) CNS symptoms are more prominent (v) Death is due to coma and convulsions within 3 days.

Fatal Dose

Lead acetate→20 gm.

Lead acetate→1.8-2.0 gm; usually uncertain.

Lead carbonate→30 gm.

Fatal period

Uncertain, usually 2-3 days.

Management

1. Stomach wash by 1% Magnesium sulphate or Sodium sulphate or warm water and simple emetics should be administered.
2. One ounce of Magnesium sulphate to remove lead from the gastro-intestinal tract.
3. Demulcents such as milk, egg white etc. can be given.
4. Calcium gluconate 15 grain (1 grain= 0.0648 gm) i.v. for the abdominal colic.
5. Injections of morphine or atropine should be given for severe pain.

De-leading

Body lead is stored in bones as triple phosphates and poisoning does not occur so long as the metal is inhibited and blood content of lead remains low. This method is replaced by chelating agents. The de-leading is carried by

Correction of acidosis: They become soluble, flow in to blood stream and excreted in normal manner but an attack of plumbism may occur.

Correction of alkalosis: They tend to fix the lead in the bones so formerly the principle was to correct acidosis, thus lead is immobilized.

Therefore in first two days:

1. High calorie diet including 3 pints of milk daily and calcium gluconate are adminis-

tered. All these lead to alkalosis that is fixed in the bones and the symptoms are relieved then de-leading is done.

2. Low calorie diet like meat, potato, rice and bread (no milk, egg or vegetables are added).
3. Mild acidosis is induced with (i) Ammonium chloride 8-12 gm/day and (ii) Dilute phosphoric acid 4 ml, 6 times/day.

4. Chelating agents:

i. **The derivatives of EDTA:** that is Ethylene-diamine-tetra-acetic acid (Edetic Acid), Calcium or sodium edetate are used. For therapeutic use, calcium disodium versenate is used. Lead is one of the metal for which calcium disodium edetate has a great affinity. Calcium disodium versenate acts as an ion exchanger in which the calcium is exchanged for the heavy metal ion and a soluble, stable and unionized chelate of lead is formed that is excreted in the urine as lead ethylene-diamine-tetra-acetate. The complex is stable and is a less toxic molecule. The rate of excretion of lead as the EDTA complex in the urine increases fifty times above the normal untreated excretion rate. Therefore, chelation therapy serves to detoxify lead, withdraws it from effector sites and promotes excretion.

Dose: EDTA 5ml of 20% solution (15 grains) is diluted with 250-500 ml of normal saline or 500ml of 5% glucose; the concentration should not exceed 3% and is given by I.V. drip over a period of 2 hours twice daily for 5 days. Then after a gap of 3-5 days, repeat the course for 5 days or calcium EDTA can be given 50 mg/kg/24 hours i.m. or i.v. for 5 days.

Side effects: The daily dose should not exceed 220 mg/kg in 12 hours and 75mg/kg every 6-12 hours. The side effects that are produced are lachrymation, nasal congestion, sneezing, muscle pain, hypotension and chronic renal damage.

- ii. **B.A.L.:** 4 mg/kg body weight is given every 4 hours. It chelates lead both extracellularly and intracellularly. Two molecules of B.A.L. combine with one

atom of lead to form a complex that is excreted in the bile and urine. B.A.L. is the chelator of choice in conditions of impaired renal function. B.A.L. should be given at least 4 hours before EDTA as the latter mobilizes lead from tissue stores and aggravates symptoms of lead poisoning.

- iii. **Penicillamine:** It is another chelating agent that is effective in the excretion of lead by oral administration and is useful in children. It is a degradation product excreted in the urine when patient's with liver damage receive injections of penicillin. Commercially it is obtained by controlled decomposition of penicillin. It is at present very expensive. It is given orally in the doses of 0.6 to 1.5G daily in two equal doses for 7 days. It can be administered intravenously as 1-3gm solutions in normal saline drip for 2-4days. This increases the urinary excretion of lead.
5. Necessary measures are taken to prevent deposition of lead in the skeleton. This includes administration of diets such as plenty of milk or Calcium chloride. Besides this, alkalosis is to be induced.

Autopsy Findings

(i) Findings of Gastrointestinal tract irritation are usually found. The stomach and intestines are contracted and thickened (ii) Stomach mucous membrane is thickened, softened, eroded at places (iii) A blue line may be visible on the gums (iv) The liver and kidneys are also contracted (v) Brain is pale and swollen (vi) Heart is hypertrophied with atheroma of aorta (vii) In the bone marrow, hyperplasia of leucoblasts and erythroblasts are seen.

CHRONIC LEAD POISONING

Chronic poisoning occurs with a daily intake of 1-2 mg of lead.

Sources of Chronic Lead Poisoning

1. Inhalation of lead dust and fumes among persons employed in factories and industries

such as painters, compositors, plumbers, enamel workers, glass blowers, electric light workers and lead smelters.

2. Drinking water stored in laden cisterns.
3. Eating tinned food contaminated with lead.
4. From use of ghee stored in brown or copper vessel lined inside with tin. Ghee becomes impregnated with lead derived from tin that contains it as an impurity and forms a poisonous salt (Oleate of lead).
5. Taking food cooked in lead vessels.
6. Absorption of lead through raw and intact skin: Hindu married females apply *Vermillion* on the parting of scalp hair. *Vermillion* contains red lead mixed with red synthetic dye that is absorbed through the scalp. The use of hair oil helps to hold the lead in contact with scalp by forming lead soap with fatty acids of oil that favours absorption.

Signs and Symptoms

1. *Facial pallor:* It is usually present around the mouth. It is one of the earliest and consistent signs and is independent of the degree of anaemia, seems due to vasospasm.
2. *Anaemia:* Anaemia occurs at the lead level of 70-80 $\mu\text{g}/100\text{ml}$ of blood. In the early stages, polycythemia, polychromatophilia occurs and in late stages, microcytic or normocytic anaemia but not the macrocytic anaemia results. There is reticulocytosis, poikilocytosis, anisocytosis and nucleated red cells are seen. The number of polymorphs and platelets is also decreased. Punctate basophilia or basophilic stippling of red cells due to clustered ribosomes at blood levels of 60-80 $\mu\text{g}/100\text{ml}$ are pathognomonic of plumbism. Punctate basophilia is the number of discrete dark blue particles of pinhead size in the cytoplasm. Under UV-light, the number of fluorescent red cells increase to 75-100% due to increased protoporphyrin. There is toxic action of lead on porphyrin metabolism, which is excreted in the urine at the rate of about 500 $\mu\text{g}/\text{day}$. In chronic lead poisoning anaemia occurs due to: (i) Decreased life span of red cells as they are

mechanically more fragile (ii) Synthesis of Haemoglobin is inhibited that further inhibits the action of Delta-amino-levulinic-acid dehydrogenase.

3. **Lead line (Burtonian line):** In 50-70% of the cases, the blue or the lead line is seen on the gums due to sub-epithelial deposits of granules at the junction with the teeth. It is deposited in the dirty or carious teeth within a week of exposure usually in the upper jaw. This line is due to the formation of lead sulphide by the action of H₂S, which is formed by the decomposed food in the mouth. There is sweetish, metallic taste in the mouth.
4. **Colic and constipation (Dry belly-ache):** It is usually a late symptom. Colic of intestine, ureter, uterus etc., occurs in 85% of the cases. It is very severe usually occurring at the night. During colic, the abdominal wall is rigid and contracted and heavy pressure results in some relief. The attack of colic lasts for few minutes. Constipation is usually present but diarrhoea and vomiting may also occur.
5. **Lead palsy:** Lead palsy is a late feature, seen usually in less than 10% cases. Tremors, hyperesthesia, numbness, cramps and muscle weakness occurs. It is more common in adults than children and in men than women. The muscle group that is more prone to fatigue is commonly affected. These are the extensor muscles of wrist except supinator longus (**wrist drop**) but deltoid and biceps can also be affected. All the extensor muscles of foot are affected (**Foot drop**). It is a purely motor type of paralysis due to (i) Interference with phosphocreatine metabolism and (ii) Peripheral neuropathy—axonal degeneration due to toxic action of lead in the anterior horn cells.
6. **Lead Encephalopathy:** Encephalopathy is quite common in the children. It is present in almost every case in one form or the other. It depends on the (i) Intensity of the disease (ii) Duration of exposure (iii) Age of the patient.

The symptoms of lead encephalopathy are insomnia, headache, vomiting, restlessness, delirium, hallucinations, convulsions and coma develops after the nervous symptoms and finally the death.

7. **Cardiovascular and renal symptoms:** Lead causes vascular constriction that results in hypertension, arteriolar degeneration, chronic arteriosclerotic nephritis, interstitial nephritis.
8. **Coproporphyrin III:** In non-exposed individuals coporphyrin is less than 150 mg/L in urine. It is detected as reddish fluorescence when seen under U-V lamp; when a mixture of small quantity of urine, few drops of glacial acetic acid and equal quantities of ether are taken.
9. **Reproductive system:** Menstrual derangements, sterility, abortion at 3-6 months and foetal abnormalities may occur.
10. **Other symptoms:** Other symptoms like anorexia, emaciation, alopecia, vertigo etc. may develop.

Diagnosis

1. Careful and detailed history regarding the nature of work to be taken.
2. **Blood levels:** One measure of lead in the body is the blood lead level (BLL), measured in micrograms of lead per deciliter of blood (µg/dL). Nearly everyone has a measurable BLL. Normal lead levels are 0.03mg/100gm in blood. The Centers for Disease Control and Prevention (CDC) states that a BLL of 10 µg/dL or above is a cause for concern. However, lead can impair development even at BLLs below 10 µg/dL. The average person has less than 10 micrograms per deciliter, or 100 parts per billion (ppb) of lead in their blood serum. People who have been exposed to an unusual amount of lead will have lead serum levels higher than 200 ppb. Most of the clinical symptoms of lead poisoning begin at around 100 ppb.

Estimation of lead levels in blood:

- >0.8mg/litre is suggestive of chronic lead poisoning.

- 5pg/dL—indicates exposure.
- >30pg/dL—there is a need to find the source.
- >60pg/dL—de-leading agents should be given.
- >80pg/dL—there is a risk of Encephalopathy
- >100pg/dL—should be treated as emergency.

Urine Levels

1. In a 24-hour urine sample, an excretion of more than 0.08 mg/litre is significant. On examination of urine:
 - Coproporphyrin level more than 0.15 mg/24hrs are suggestive of lead exposure.
 - δ - Amino-levulenic acid >6 mg/L has some lead effects.
 - δ -Amino-levulenic acid >19 mg/L leads to lead poisoning.
2. Glycosuria, haematuria, proteinuria occurs.
3. The urinary excretion of more than 1 mg of lead/mg of calcium edetate at 25 mg/kg but not exceeding a total of one gram.

Spinal fluid: Spinal fluid analysis should be avoided unless it is quite essential. The pressure is raised with increased proteins and pleocytosis.

X-Ray findings: Radiological examination shows transverse bands of increased density at the growing i.e., metaphyseal ends of the bones in the children 2-5 years old mainly. Multiple bands suggest repeated episodes of exposure. Abdominal X-ray shows opaque particles at the recto-sigmoid area.

Autopsy Findings

(i) Blue line on the gums at their junction with the teeth (ii) Fatty degeneration of paralysed limbs (iii) Stomach and intestine show ulceration and haemorrhages and the walls are contracted (iv) Liver and kidneys are contracted and show granular degeneration (v) Heart is hypertrophied and shows atheromatous changes.

Treatment

1. Remove the individual from the source of exposure.
2. Potassium or sodium iodide 1-2 gm thrice daily for 5-7days to assist in the elimination of lead by the kidneys.

3. Sodium bicarbonate 20-30 G/day for 4-5 doses leads to transformation of tri-basic lead phosphate to soluble dibasic phosphate and carbonic acid is liberated.
4. De-leading may be carried out.
5. EDTA, Penicillamine or B.A.L. is given i.m. for 7-10 days; 18 mg/kg/24 hrs for 2 days then 12 mg/kg/24 hrs for third day and 6 mg/kg/24 hrs for next 2 days.
6. Saline purgatives can be administered
7. In lead encephalopathy, EDTA is given.

Prophylactic Treatment in Chronic Poisoning

(i) Adequate exhaust ventilation in factories (ii) Personal cleaners (iii) The worker should take a diet rich in calories and plenty of milk (iv) Water containing minute quantities of H_2SO_4 (v) Saline purgatives should be given (vi) Periodical medical examination of the staff working in lead factories.

Medicolegal Aspects

Metallic forms not poisonous, but when acted upon by secretions of the intestine and may act as a poison after being absorbed in to the system as a salt:

1. Chronic lead poisoning is regarded as one of the industrial diseases.
2. Compounds of lead are poisonous provided they are in a condition fit for absorption. In the absence of air, pure water has no action on lead, but in the presence of air, it is slightly soluble and lead hydroxide is formed.
3. Acute lead poisoning is very rare, when it occurs, recovery usually occurs.
4. In chronic poisoning, workers are referred to medical practitioners under 'Workman's compensation Act 1923. Children get compensation, if death occurs.
5. Homicide by lead is rare as very few cases are reported when lead acetate is mixed with beer.
6. Accidental poisoning occurs by use of *Litharge (Mudrasang)*—lead monoxide as a remedy for syphilis by quacks.
7. Used criminally as abortifacient, the paste used on abortion stick contains red lead or lead tetra oxide.

8. Cattle poison: Red lead alone or when mixed with Arsenic is used as a cattle poison.
9. It is used as to adulterate *snuff* (powdered tobacco or medicine sniffed through the nostrils) to improve its colour.
10. Lead is normally present in all human tissue, deposited in the bones, nail, tooth and hair and very poorly in lead. No lead is found in ovary but is present in the testes. It has got no affinity for fetal tissues.

Accidental poisoning occurs in the children by eating pica, old paint, plaster; feeding bottles made of glass contain lead, lead nipple shields, toys, and street dirt. Also the cosmetics like face powders, hair dyes used by the mothers secreted in the milk can cause accidental poisoning in the children.

ARSENIC

Arsenic is a metallic poison known since ancient times. The Greeks knew it as early as 500BC and Hippocrates used it as an external remedy for ulcers, although there is no record of it being used as a poison this early. An eight-century Arab alchemist Jabir ibn Hayyen was the first to obtain white arsenic by heating realgar. It is thought that it was around this time when criminals first employed arsenic as a poison. Before white arsenic was known, most of the poisons used had a distinguishing taste, smell or colour but arsenic has very little smell or taste so it is undetectable by the victim. It is the common environmental toxicant and is found in soil, water and air. It ranks 20th in elemental abundance in earth's crust and 12th in the human body.

Properties of Arsenic

Arsenic is element 33 in the periodic table and is classically considered a heavy metal. It exists in 3 allotropic forms and in several ionic forms. Environmental arsenic exists mainly as sulphide complexes realgar (As_2S_2), Orpiment (As_2S_3) and iron pyrites. On heating of these ores, arsenic sublimes and oxidizes to form arsenic trioxide (As_2O_3) a fine granular white powder also known as white arsenic. Arsine gas (AsH_3) is another

arsenic compound, formed by the hydrolysis of metal arsenides and by the reduction of metals of arsenic compounds in acidic solutions. The main source of Arsenic is Arsenopyrites, realgar (Arsenic di-sulphide, As_2S_2) orpiment (Arsenic trisulphide, As_2S_3) and Arsenolite (Arsenic trioxide, As_2O_3). It is usually obtained as a by-product from smelting of copper, lead, zinc and ether.

Natural Sources of Arsenic

The average daily intake of arsenic in adults is 0.5-0.1mg. Arsenic is a widely distributed element and is found in: (i) Rocks and soil in concentrations of less than 1ppm (ii) Hot spring mineral waters (iii) Drinking water supply (iv) Sea-water contains 2-5mg/L (v) Plant and plant products (vi) Vegetables, fruits and grains (vii) Seafood (Fish, Shellfish, crabs and Dover-sole) and meats. Marine organisms particularly shellfish may contain large amounts of well-absorbed trimethylated organo-arsenic, arsenobetaine that exerts no known toxic effects when ingested by mammals and is excreted unchanged in the urine (viii) Poultry and meat products: Arsenilic acid has been used as a food additive for poultry and other livestock to promote growth and weight gain. The poisoning may result from regular consumption of such products but at times accidental poisoning of livestock has occurred. There are instances of Beef or cattle being poisoned by ingestion of field grass that has been previously sprayed by herbicides.

The interaction of Arsenicals with bacteria is thought to account for the changes in the oxidation state and chemical form of arsenic.

Industrial Sources of Arsenic

Arsenic is used primarily in the production of glass and semiconductors, in wood and hide preservation, as an additive to metal alloys to increase hardening and heat resistance. In the past Arsenic was used as weed killer, rodenticide, in chemical warfare and in the production of pigments and enamels.

Arsenic may be introduced in to the environment through (i) Industrial process such as smelting of other metals (ii) Application of arsenical

pesticides and herbicides (iii) Generation of power from coal (iv) Geothermal sources (v) Coal and combustion of coal.

In the metallic form Arsenic is not poisonous as it is insoluble in water and cannot be absorbed from alimentary tract, but when it is oxidized by exposure to air becomes poisonous. But some workers believe that the metallic form may undergo oxidation in the GIT producing symptoms.

Compounds of Arsenic

The compounds of arsenic are organic as well as inorganic. The inorganic compounds are Arsenate (AS^{5+}) and Arsenite (AS^{3+})

1. *Arsenious oxide or Arsenious tri-oxide* (AS_2O_3): It is a heavy metal with a specific gravity 3.669. Its curious property is that it floats in water as a white film and is soluble in spirit. It is found as impurity in iron ores, mineral acids. Also found in soils, mineral water and coal smoke. It is widely used in: (i) Calico-printing (ii) Taxidermy (preparing, mounting skin of animals) (iii) Preparation of artificial flowers (iv) Wall papers preparation (v) Preservation of timber and skin against Ants (vi) Used by *Hakims* and *Vaidhs* to treat Rheumatism, skin diseases, syphilis and impotence.
2. Copper Arsenite (Scheel's green, Paris green)
3. Red Arsenic (Arsenic disulphide)
4. Yellow Arsenic (Arsenic trisulphide)
5. Arsine gas (AsH_3)—is hemolytic and nephrotoxic.

Organo-chemicals

Many organo-chemicals ranging in toxicity from the war gas Lewisite to lead additives such as Arsenilic acid. In general their toxicity is similar to that of inorganic ones. These are: (i) Cacodylic acid (ii) Sodium cacodylate (iii) Atoxyl (iv) Salvarsan (v) Neo salvarsan.

Pharmacokinetics and General Toxicity of Arsenicals

Arsenic can be inhaled and absorbed through the skin or through GI tract after ingestion. After a very small dose of arsenic (like that experienced

daily by most people) most of the absorbed inorganic arsenic undergoes methylation, mainly in liver to monomethylarsonic acid and dimethylarsinic acid, which are excreted along with residual inorganic arsenic in the urine. However, if the dose of arsenic is very large, the elimination half-life is prolonged. Once absorbed, arsenic rapidly combines with the globin portion of haemoglobin and therefore localizes in the blood. There is minimal penetration of the blood-brain barrier and within 24 hours arsenic redistributes itself to the liver, kidney, spleen, lung and GI tract, with lesser accumulation in muscle and nervous tissue. Once in the tissues, arsenic exerts its toxic effect through several mechanisms, the most significant of which is the reversible combination with sulphhydryl groups. Arsenic blocks the Krebs cycle and interrupt oxidative phosphorylation thus resulting in a marked depletion of cellular ATP and eventually death of the metabolizing cell. Arsenic also inhibits the transformation of thiamine in to acetyl CoA and succinyl-CoA resulting in the features of thiamine deficiency. Arsenolysis is a major form of toxicity in which the arsenic anions can substitute for phosphate in many reactions. Arsenic is capable of inhibiting oxidative phosphorylation through two different mechanisms 2-4 weeks after ingestion, arsenic is incorporated into hair, nails and skin as it binds to the sulphhydryl groups of keratin. Four weeks after ingestion, arsenic begins to localize in the bone where it substitutes for phosphate.

Organic Arsenicals are more rapidly excreted than the inorganic form. Pentavalent forms are cleared faster than trivalent form. The toxicity is related to:

- i. How fast it is excreted from the system? The inorganic forms are more toxic than the organic forms and the trivalent forms are more toxic than the pentavalent forms depending on their rapidity of excretion.
- ii. To what degree it may accumulate in the tissues? Pentavalent forms are accumulated more rapidly than the trivalent forms which is approximately the reverse order of excretion.

Arsine gas (AsH_3) causes rapid haemolysis and renal damage may persist for years following single acute poisoning. Arsenite is absorbed from GIT and retained in high levels in the tissues.

The poisoning due to Arsenic compounds can be acute, subacute and chronic.

Acute Arsenic Poisoning

The acute arsenic poisoning results from the: (i) Oral ingestion (ii) Inhalation of arsenic gas (iii) Rubbing in to the skin (iv) Introduction in to rectum or vagina.

Arsenic poisoning is usually the result of accidental or suicidal ingestion of insecticides; (Paris green) that is copper arsenite or calcium or lead arsenate. Pesticide containing arsenic is freshest source of poisoning

The toxic symptoms of acute arsenic poisoning depends upon individual susceptibility. Orchardists can ingest 6.8 mg /day without any signs and symptoms but arsenic trioxide as low as 30 mg has been found fatal. This extraordinary degree of tolerance to ingestion of arsenic could be due to blockage of absorption, alteration in its distribution to tissues, enhanced or accelerated transformation of arsenite to less toxic arsenate, and decreased reduction of arsenate to arsenite by peripheral tissues.

Fatal dose→The fatal dose of arsenic trioxide is 180 mg but the dose depends on the compound ingested, its physical form and the inherent tolerance of the patient to arsenic

Fatal period→Fatal period for arsenic poisoning is 12-48 hours but can be fatal even within 2-3 hours.

Mechanism or Action

The mechanism of toxicity of all arsenic compounds is the same. Once in the tissues, arsenic exerts its toxic effect through several mechanisms such as: (i) Reversible combination with sulphhydryl groups (ii) Reacts with SH group in tissue proteins (iii) Interferes with number of enzyme system essential for cellular metabolism (iv) Capillary poison, dilates capillary (v) Fatty degeneration of liver

(vi) Hyperemia and haemorrhages in the intestine (vii) Renal tubular necrosis (viii) Peripheral nerves show disintegration of axis cylinder (axonal neuropathy) with fragmentation and resorption of myelin.

Signs and Symptoms

Acute Poisoning

The symptoms of acute intoxication usually occur within 30 minutes of exposure:

1. Initially the patient experiences a metallic taste in the mouth and slight garlicky odour in the breath along with xerostomia (dry mouth) and dysphagia.
2. This is followed abruptly by severe nausea and vomiting, colicky abdominal pain and profuse diarrhoea (bloody in some cases) with rice water stools, due to vasodilation with transudation of fluid into the bowel lumen in addition to mucosal vesicle formation and sloughing leading to increased peristalsis.
3. Generalised vasodilation caused by capillary damage results in transudation of plasma and severe hypovolemia.
4. If the dose of arsenic ingested is large, cyanosis, cold clammy extremities, hypoxic encephalopathy, convulsions and acute tubular necrosis may occur following shock.
5. Hyperpyrexia and acute haemolysis may also occur.
6. If the ingested dose is smaller, the patient will have severe headache, vertigo, periorbital oedema, skeletal muscle cramping and evidence of renal damage manifested as oliguria, proteinuria, and haematuria.
7. If death does not occur in the first few hours from shock, the patient may die a few days later from acute hepatic or renal failure.
9. Cardiac manifestations include acute cardiomyopathy, subendocardial haemorrhages, and ECG changes (prolonged QT interval and non-specific ST-T changes). The cardiac symptoms are due to decreased cardiac output due to hypovolaemia and direct toxic effect on the cardiac muscle.

10. Arsenic has a predilection for keratin that is why the concentration is higher in hair and nails.
11. Death from acute arsenic poisoning is usually caused by irreversible circulatory insufficiency.

When the dose is not large enough to kill the patient, a number of secondary effects can be seen 2-4 weeks after ingestion of the poison. These include hair loss, **Mee's lines** (white transverse lines seen on the nail plate up to a year after arsenic intoxication), sensorimotor peripheral neuropathy (may develop within a few hours of ingestion but usually seen 2-8 weeks after exposure), skin changes (as in chronic poisoning) and possible chronic renal failure.

The signs and symptoms start within half an hour but may be delayed for several hours when it is given in rectum, vagina or skin. The dilatation of capillaries is responsible for most of the symptom: (i) Burning pain and throat constriction (ii) Stomach pain which may increase on pressure (iii) Increased salivation (iv) Intense thirst (v) Vomiting that is projectile, dark brown or yellow in colour and contains stomach contents, blood and mucus (vi) Purging, tenesmus, pain and irritation about anus. The stools are dark coloured and bloody and later on are colourless, odourless and watery resembling rice water (vii) Due to capillary transudation of plasma following rupture of vesicles under intestinal mucosa (viii) Cramps in the calf muscles (ix) Restlessness (x) The patient is dehydrated (xi) Skin is cold and clammy (xii) Face is pale and anxious (xiii) Eyes are shrunken (xiv) Pulse is irregular and quick (xv) Respiration is laboured (xvi) Hypoxia, convulsions and coma precede death (xvii) Cirrhosis and ascites results from the therapeutic doses of Flower's solution (1% Arsenious trioxide) (xviii) Due to direct action on the myocardium the ECG shows T-wave inversion and persistent prolongation of QT interval (xix) *Arsine dust*: When arsine dust is inhaled, it causes cough with frothy sputum, pulmonary oedema, cyanosis, breathlessness, corneal ulcer and conjunctivitis.

Table 37.1: Differential diagnosis of acute arsenic poisoning

Findings	Arsenic poisoning	Cholera
Pain in throat	Present before vomiting	Present after vomiting
Purging Vomitus	Follows vomiting Consists of mucous bile and blood	Precedes vomiting Watery
Stools	High coloured, discharged with straining and tenesmus	Rice water and involuntary
Voice conjunctivitis	Not affected Inflamed	Peculiar and rough Not inflamed

Narcotic form produces mainly CNS symptoms such as: (i) Giddiness (ii) Precordial distress (iii) Tenderness of muscles (iv) Delirium (v) Dilated pupils and complete paralysis of muscles at times. *Unusual symptoms* such as (i) Convulsions (ii) Lockjaw (iii) Raised temperature (iv) Loss of speech and memory (v) Joint pains can also be produced.

Arsine Gas

Arsine gas acts as a poison to haemoglobin of red blood cells and causes haemolysis. It most commonly produces haemoglobinuria and anaemia. The other symptoms produced as a result of poisoning due to arsine gas Jaundice, dark red urine, heart failure, delirium and coma.

Subacute Poisoning

Subacute poisoning results when the compounds of arsenic are given in small doses at repeated intervals. The signs and symptoms that are produced are dysphagia, cough, foul smelling tongue that is dry and congested, the motions are bloody.

Chronic Arsenic Poisoning

This is much more insidious and the diagnosis is often difficult to establish. The earliest cutaneous manifestation is persistent erythematous flushing caused by cutaneous capillary dilation. Hyperkeratosis then occurs with desquamation of the palms and soles, brittle nails with Mee's lines, patchy or diffuse alopecia (hair loss) and oedema

of the face, periorbital region, or ankles from localised transudation of intravascular fluid. Bowen's disease is a long-term complication of chronic arsenic intake and often indicates that systemic neoplastic processes are occurring. Haematologic abnormalities include normochromic and normocytic anaemia caused partly by haemolysis, leukopenia and thrombocytopenia and mild eosinophilia. Karyorrhexis is normally seen and manifested by bizarre nuclear forms seen on bone marrow examination. Interference with folate metabolism may lead to a megaloblastic picture.

Neuropathy is the hallmark of arsenic poisoning. It is usually a symmetrical sensorimotor polyneuropathy, often resembling Guillain-Barre syndrome. The predominant clinical features of neuropathy are paresthesia, numbness, and pain, particularly of the soles of the feet. Eventually, muscular atrophy (possibly even paralysis) and ataxia are seen. Encephalopathy may be seen in some cases, presenting as severe headache, personality disturbance, convulsions or coma. Other features of chronic arsenic poisoning are nausea, vomiting, diarrhoea and irritability.

Patients who recover from the acute form of poisoning develop (i) Skin and mucosal changes (ii) Peripheral neuropathy (iii) Linear pigmentation in the fingers (iv) Cutaneous manifestations appear within 1-4 weeks and consist of dry, diffuse and scaly desquamation with hyperpigmentation over the trunk and extremities and hyperkeratosis of palm and soles (v) Mucosal surfaces also show signs of irritation like conjunctivitis, photophobia and pharyngitis (vi) After 5 weeks, transverse white streaks 1-2mm width appears above base of each fingernail called **Mees line**. Those who have more than one exposure may show double line, several millimeters apart (vii) After 1-3 weeks, features of peripheral neuropathy like decreased sensations of touch, pain and temperature appear in a symmetrical fashion. There is 'glove and stocking' distribution. There is distal weakness resulting in wrist drop, unable to walk, distal reflexes are decreased and atrophy of muscles.

Laboratory Findings

Diagnosis

Definitive diagnosis of arsenic poisoning is difficult

because of the natural presence of trace amounts of arsenic in the body and because, due to the multi systemic toxicity of arsenic, the clinical manifestations vary widely so both acute and chronic poisoning simulate many other diseases and be overlooked in the differential diagnosis. Diagnosis is often particularly difficult in homicidal poisoning attempts because the patient will not usually know that he/she has ingested arsenic.

Arsenic poisoning is usually diagnosed with a urine test for arsenic since monomethylarsine and dimethylarsine are present in the urine 24 hours after ingestion and arsenic ions are present very shortly after ingestion (useful for diagnosis of acute poisoning). A urinary excretion of arsenic >200 mg/24 hr is regarded as indicative of exposure to a potentially toxic amount of arsenic. The gastric contents can also be screened for arsenic and, if it is very soon after ingestion a blood test will also reveal arsenic. Analysis of body tissues, nails and hair is important in diagnosis of chronic arsenic ingestion. Colorimetry, polarography, atomic absorption spectroscopy and neutron activation analysis are effective methods for arsenic detection. Hair and nail samples containing >3ppm or 100mg or arsenic per 100g of specimen are diagnostic of arsenic poisoning. Analysis of hair is often used many years after death to determine the cause of death. This is how the true cause of death (chronic arsenic poisoning) of Napoleon Bonaparte was determined 140 years after he died (Weider, 1999).

- i. *Urine*: Normal value of arsenic is 0.01-0.06mg/litre of urine and it may be even upto 0.2 mg/litre. Most patients with exposure having more than 0.1 mg/litre, upto 1mg/litre of urine. Proteinuria, casts and albuminuria are there.
- ii. *Blood*: The normal blood level is 100mg/litre. There is moderate anaemia and leucopenia 2000-5000 mm³ except Arsine poisoning causes leukocytosis and mild eosinophilia.
- iii. *Liver function tests*: There is failure to excrete bilirubin, decreased metabolism of protein, serum alkaline phosphatase and bilirubin is raised, urobilinogen in the urine
- iv. CSF is normal.

- v. *Hair*: In normal persons, average concentration is 0.05/100 gm of hair but more than 1mg/100 gm indicates poisoning.

As arsenic is widely distributed in nature, their presence may not always indicate of the poisoning.

Treatment

1. Empty stomach by emetics, tartar emetics should not be used; copper sulphate also should not be used as it leads to the formation of copper arsenite.
2. Stomach wash should be done with freshly prepared solution of ferric oxide in table spoonful doses suspended in water; with arsenious acid it forms ferric arsenite that is a harmless and insoluble salt. Ferric chloride (45 ml) and sodium carbonate or magnesium oxide (15 gm) in half glass of water, strained through muslin is used for the lavage. The following reaction occurs $\text{FeCl}_3 + \text{MgO} \cdot \text{Fe}_2\text{O}_3 + \text{MgCl}_2$
3. For gastric lavage, 1% sodium thiosulphate in water is helpful.
4. Dialysed iron may be used as a substitute.
5. 10% solution of sodium thiosulphate as 7½ gm I.V. injection but it is of doubtful value.
6. British Anti Lewsite (B.A.L.) is administered 4 hrly for 2 days, 6 hrly for 1 day and total dose 12 hrly afterwards; total dose is 2.5-3mg/kg body wt. The duration of B.A.L. depends upon (i) Clinical state and (ii) Response of the patient. B.A.L. is to be stopped if side effects like nausea, vomiting, burning sensation in the mouth, tachycardia, hypotension, constriction in the throat appear.
7. To know when the chelation is to be stopped, 24 hr urine sample is to be collected and if Arsenic levels fall below 50 mg/24 hrs, it should be stopped for 5 days and then started again so that the tissue arsenic is mobilized and ready for chelation.
8. Oral Penicillamine 100 mg/kg body wt in four divided doses is given in 24 hrs for 4-8 days after initial 12-48 hrs of B.A.L. therapy.
9. Demulcents such as ghee and barley water are given.

10. Castor oil or magnesium sulphate is administered to diminish intestinal absorption of arsenic.
11. Morphine is given to relieve pain.
12. Saline is administered intravenously for dehydration and enuresis.
13. To relieve cramps, massage is employed.
14. Body temperature is to be maintained.

Treatment of Poisoning by Arsine Gas

(i) Patient to be taken to fresh air (ii) Oxygen inhalation to be started (iii) Exchange transfusion should be undertaken (iv) Haemodialysis (v) Alkaline drinks. All of the above methods help to eliminate arsine from the blood. B.A.L. is not effective in arsenic gas poisoning.

Autopsy Findings

1. Rigor mortis lasts longer.
2. General shrunken appearance due to dehydration.
3. Eyeballs are shrunken.
4. Cyanosis of hands and feet is there.
5. Jaundice is also present.
6. Mucous membrane of mouth and pharynx is inflamed and ulcerated.
7. Stomach is the main organ to be inflamed; food particles are mixed with arsenic embedded in the mucous. The inner wall of the stomach is swollen, congested and tinged with streaks of blood and arsenic. On scraping the mucous membrane is congested and inflamed and is brownish red or scarlet in colour. There is petechial haemorrhages. The inflammation is more marked in the greater curvature and the cardiac end. Ulceration, gangrene and perforation have been rarely reported.
8. Small intestine is flabby and contains large flakes of mucous with little faecal matter, submucosal haemorrhages are present and the epithelium is flabby and oedematous.
9. Large intestine contains seromucous and the intestinal glands are swollen and enlarged.
10. Lungs are congested, oedematous and show subpleural ecchymosis.

11. Caecum and rectum are inflamed and the mucous membrane is flabby.
12. Liver, spleen and kidney is enlarged and congested and shows fatty infiltration and degeneration.
13. Heart contains coagulated blood and ecchymosis of the endocardium and muscles of the left ventricle is there.
14. Brain is congested and ventricles are full of serum.

Autopsy Findings in Arsine Gas Poisoning

(i) Skin is dirty yellow in colour (ii) Mucous membrane of stomach and intestine is yellow and inflamed (iii) Liver is small or enlarged and shows fatty degeneration (iv) Spleen shows evidence of destruction of blood and deposition of blood pigment throughout the organ (vi) Kidney is enlarged, congested and shows tubular necrosis (vii) Lungs are congested and oedematous.

Chronic Arsenic Poisoning

Chronic arsenic poisoning results from: (i) Chronic repetitive inhalation of arsine gas (ii) Chronic repetitive ingestion. Chronic poisoning usually results from the:

1. Persons engaged in the occupation of: (i) Smelting that is extraction of metals from ore by melting (ii) Refining of ores (iii) Manufacture of weed killers, insecticides, paints, dyes and cosmetics.
2. Persons who ingest it as medicine for long periods.
3. May follow the acute poisoning after recovery.

Chronic poisoning consists of the following stages:

First stage: The following symptoms are produced in the first stage: (i) Loss of weight (ii) Loss of appetite and salivation (iii) Colicky pain and constipation (iv) Vomiting and diarrhoea (v) Gums are red and soft (vi) Tongue is coated, is thin white silvery and furred (vii) Oedema of eyelids and ankles (viii) Temperature and pulse is raised.

Second stage: In the second stage of chronic poisoning following symptoms are produced: (i) Cutaneous eruptions (ii) Catarrh of larynx and

bronchial tube (iii) Dryness and itching of skin (iv) Voice is hoarse and husky (v) Photophobia and conjunctivitis (vi) Running nose and coryza (vii) Cough with bloody expectoration (viii) Liver is enlarged and cirrhotic (ix) Kidneys are damaged (x) Prominent changes are produced in the skin:

- Generalized or localized, **rain drop** type of pigmentation of the skin involving the covered parts of the body such as flexors, nipples, lower abdomen, temples and eyelids. This can be differentiated from Addison's disease as mucous membrane of the mouth is spared.
- Epithelial hyperplasia, discrete multiple wart like keratosis on the palm, soles, head and trunk (Basal cell carcinoma—Bowen's disease)
- Epitheliomata are present in 20% of the cases.
- Nails are brittle with linear pigmentation.
- Hairs are dry and fall off.
- Painless perforation of the nasal septum.

Third stage: (i) Sensory symptoms are predominantly present such as headache, tingling, numbness and hyperaesthesia of the skin (ii) Tenderness and muscle cramps (iii) Circumscribed oedema of eyelids and ankles (iv) Knee jerk is usually lost (v) Impotence is commonly present (vi) There is evidence of bone marrow depression and aplastic oedema.

Fourth stage: (i) Peripheral neuritis with glove and stocking type of anaesthesia; there is decreased pain, temperature and touch sensation (ii) Muscular atrophy of extensor muscles resulting in wrist drop and foot drop (iii) Muscular weakness (vi) Ataxia (v) Cramps (vi) Tremors and general emaciation (vii) Anaemia and dysuria (viii) Death is due to failure of heart muscles.

Differential diagnosis: Chronic arsenic poisoning is to be differentiated from alcoholic neuritis. In the former the symptoms and signs are produced more rapidly. Are widespread and there is no glycosuria whereas in alcoholic neuritis glycosuria is positive.

Treatment

1. Remove the patient from source of poison.
2. B.A.L. is to be given 6 hourly for 2-3 days and then once daily.

3. Vitamin B₁ injection for peripheral neuritis.
4. Improve general health.

Medicolegal Aspects

1. It is an ideal homicidal poisoning, used for frequently in India; can be mixed with sweets, bread, cooked vegetable, drinks, milk, pan and cigarettes. Mass homicidal poisoning simulates cholera. It is tasteless, odourless, easily available and small doses are required for homicide. In western countries, small doses are given for a prolonged period that simulates gastroenteritis to conceal the crime. The disadvantages of being used as a homicidal poison are: (i) Can be detected even in the charred bodies (ii) It delays putrefaction (iii) Can be detected in decomposed bodies (iv) Can be found in bones, hair even years after the poisoning.
2. It is used on abortion sticks as paste or ointment.
3. It is used as a cattle poison, is mixed with water in the well.
4. Is used for suicidal purposes.
5. The poisoning can result accidentally due to its improper medicinal use when it is applied locally as a cure for impotence, poisoning can result from its application on the abraded skin, when it is accidentally mixed with food or drinks, when used as vaginal pessaries, and when it is mistaken for baking powder or soda. Beer drinkers in Lancashire country in 1900 developed chronic arsenic poisoning and peripheral neuritis was produced.
Arsenophagists: Arsenophagists use the drug as a habit for impotency
6. By dividing hair in small successive lengths from the root upwards and analyzing them separately, one may get the important information regarding the time that has elapsed since the administration of arsenic. Arsenic starts depositing in the hair after 15 days. If the length of hair is 1 cm that is equal to 25 days. By neutron activation analysis, the relation between ingested arsenic and arsenic content of the hair can be known.
7. Arsenic is excreted in the stomach and intestine after absorption, even when administered by routes other than mouth.

8. It has the power of retarding decomposition and it does not disappear with decomposition.

Postmortem imbibitions of arsenic: When there is a criminal charge of arsenic poisoning, the defence may take the plea that Arsenic was introduced after death and postmortem imbibition occurred in the tissues. The doctor can defend it by replying that it takes an anatomical course, more on the left side. Signs of inflammation and ulceration of stomach will be absent in case of postmortem imbibition of arsenic.

Exhumed body: Arsenic cannot percolate in the cadaver from the soil, as it is an insoluble form of salt. The nails and hair will have a higher concentration from the soil. The soil is to be kept for chemical analysis.

MERCURY

Mercury is known as quick silver, a liquid metal with bright silvery luster that is volatile at room temperature. We ingest some amount of mercury along with our diet; 5-20 mg is taken along with the foodstuffs daily.

The tolerable weekly intake of mercury per person is 300 mg out of which not more than 200 mg should be methyl mercury. It is not a normal constituent of the human body. Normal levels in unexposed individuals:

- 0-8mg/100ml of blood.
- 0.6-1.6µg per 100ml of urine.
- 5-150µg per 100gm may be found in liver and kidney.

Deadening of mercury: Mercury is converted in to a dull gray powder (contains 33% of mercury): (i) When it is shaken with oil (ii) Triturated with sugar, chalk (iii) Used for ointment purposes.

Uses of Compounds of Mercury

Have a wide range of use:

1. **Inorganic salts:** (i) Calomel cathartic—Mercurous chloride, used as purgative (ii) Mercurial ointment (iii) Vaginal creams (iv) Fingerprint powders (v) Amalgam (dental filling) (vi) Vermilion (Mercury sulphate).

2. *Organic salts* (Methyl and ethyl mercury: compounds) are an industrial waste and are used in manufacture of: (i) Thermometer (ii) Barometer (iii) Electrical industry (iv) Drug manufacturing (v) Persons working in mercury mining, hatters and furriers (vi) In agriculture as fungicide.

Medicolegal Points of Importance

- Pure metallic mercury is not considered poisonous, as it is not absorbed by the gastric juice. 1-2 pounds of liquid metal is taken to cure constipation but has no harmful effects. In exceptional cases, mercury may undergo chemical change and acts as a poison.
- Few cases of mercury embolism are reported while the blood is collected from arteritis under the mercury seal.
- When injected subcutaneously, local abscess formation occurs.
- The mercurial vapours cause poisoning; 0.5mg/m³ causes poisoning.
- When it is rubbed on to the skin, is absorbed and poisoning occurs. There is a reported incidence of the death of three persons when they applied mercury ointment for itching.
- Amalgams used for curing carious tooth sometimes cause poisoning.
- Mercuric salts are more poisonous than Mercurous salts. Trivalent salts are more poisonous than the bivalent salts.
- Mercuric chloride causes accidental poisoning when it is widely used as: (i) Disinfectant and antiseptic causes accidental poisoning (ii) Contraceptive vaginal tablets (iii) For anti-syphilitic treatment.
- Phenyl mercuric acetate jelly leads to poisoning when used as a contraceptive.
- Mercuric chloride (*Calomel*) is poorly absorbed and has a low solubility; used in teething powders, causes pink disease in infants
- Mercuric sulphide (*vermillion*) is not absorbed through the skin, is nonpoisonous.
- It is eliminated through the saliva, urine, faeces, milk and perspiration
- By neutron activation analysis, it is found that the maximum amount of mercury in the human tissues is in the kidney, nails, scalp hair and skin.
- Mercury may be detected in the bone in acute poisoning.
- Mercury is often used as a medicine; hence detection in small quantity in the viscera does not contraindicate the death from other causes.
- Mercury is not a constituent of human body; hence its presence indicates that it must have been introduced in to the system. 5-150 mg/100 gm is normally present in the tissues like liver and kidney.

Compounds of Mercury

- Mercuric oxide, HgO (Sipichand).
- Mercuric chloride (corrosive sublimate); has (i) Antiseptic properties (ii) Largely used as medicine, taxidermy (iii) Violent poison.
- Mercuric iodide (Red iodide of mercury).
- Mercuric cyanide, is poisonous.
- Mercuric nitrate.
- Mercuric sulphide (Vermillion), is not poisonous
- Mercuric sulphate.
- Mercurochrome: contains 25-28% of mercury, discolours readily in water and the toxic effects develop after prolonged use.
- Organic compounds are: (i) Ethyl and methyl mercury (ii) Thiomerin sodium (iii) Neptal.

Poisoning results from both organic and inorganic salts; the organic compounds produce irreversible effects and both the compounds affect CNS. Methyl mercury is more poisonous than the inorganic salts.

Acute Mercury Poisoning

This poisoning is mostly due to corrosive sublimate, mercuric chloride. The signs and symptoms start immediately after swallowing the poison, and rarely they are delayed for half an hour (i) Acrid metallic taste in the mouth (ii) Feeling of constriction or choking sensation (iii) Hoarseness of voice (iv) There is difficulty in breathing (v) Mouth and tongue are corroded and swollen with gray-white coating (vi) Hot, burning pain in the mouth, stomach and abdomen (vii) Stools are blood stained (viii) Urine is suppressed and scanty,

contains blood and albumin; is accompanied by necrosis of renal tubules and damage to glomeruli (ix) Pulse is quick, small and irregular; there is circulatory collapse (x) Thrombocytopenia and Bone marrow depression (xi) When vapours are inhaled, the following signs and symptoms are produced: salivation, gingivitis and loosening of teeth (xii) When injected intravenously, it produces: dyspnea, cyanosis, hypotension and convulsions (xiii) Death may be due to anaphylactic shock or ventricular fibrillation.

Diagnosis

Acute mercury poisoning is to be differentiated from arsenic poisoning from the following features: (i) The symptoms are produced sooner (ii) Acrid taste in the mouth (iii) Constriction of the throat is more marked (iv) Vomitus more often contains blood and mucous (v) Kidneys are more affected (vi) Urine contains >500µg.

Fatal dose→15gm; 2gm is the smallest recorded dose

Fatal period→3-5days

Treatment

1. Stomach wash with warm water and magnesium carbonate.
2. With albumen such as egg albumin, skimmed milk or vegetable gluten, albuminate of mercury is formed (is insoluble in water but soluble in excess of albumin); is digested and absorbed in the stomach.
3. Demulcent drinks are given to protect the stomach wall.
4. Activated charcoal and water absorb mercury salts. Add magnesium sulphate that has increased absorptive power and hastens the removal.
5. B.A.L. is to be given in the dose of:
 - 3.5 mg/kg 4 hourly for 2 days
 - 2.5 mg/kg 6 hourly for 1 day
 - 2.5 mg/kg twice daily according to the severity.
6. Penicillamine is given 250mg 4 times a day orally.
7. Sodium formaldehyde sulfoxylate is the chemical antidote that reduces the prechloride

to metallic mercury. 5-10% sulfoxylate and 5% sodium bicarbonate is used for stomach wash of which 200ml should be left in the stomach.. This is beneficial if given in first half an hour. If colitis has developed, high colonic lavage as 1:1000 solution is to be given.

8. For diuresis 5-10% glucose is used in normal saline.

Chronic Mercury Poisoning

1. Workers may get poisoned due to the vapours or dust.
2. When small doses are taken for prolonged period.
3. When used for external application as an ointment.

The signs and symptoms of chronic mercury poisoning start at a blood level of 100mg/ml. The daily urinary excretion is more than 300mg. The following signs and symptoms are produced: (i) Nausea, vomiting and diarrhoea (ii) Indigestion, dyspepsia and colicky abdominal pain (iii) Excessive salivation with swollen and painful salivary glands (iv) Foul smelling breathing (v) Inflamed and ulcerated gums with brownish blue line (vi) Loosening of teeth (vii) Necrosis of jaw; should be differentiated from phosphorus poisoning.

Mercuria lentis: A brownish reflex from the anterior lens capsule of both the eyes is seen when observed in slit lamp in persons exposed to mercury vapours for some years. It is bilateral and has no effect on the visual acuity. Erythematous, eczematous (watery and weeping) papular type of skin lesions mostly in the hands and feet accompanied with thickening of skin are produced.

Mercurial Tremors (Hatter's shake or glass blower's shake): These are coarse tremors that are detected early by writing and they involves the fingers, muscles of tongue, face, arms and legs; are excited by voluntary movements and are absent during sleep. There is incoordination of movements, increased deep reflexes, paresis of limbs and peripheral neuritis. The tremors should be differentiated from thyrotoxicosis in which there is presence of fine tremors, exophthalmos, raised pulse and goiter

Mental symptoms (Erethism): Erethism develops in the mirror-manufacturing firms, these include shyness, timidity, irritability, loss of confidence, mental depression, loss of memory, insomnia, hallucinations and delusions that are features of insanity

Treatment

1. Removal of the individual from the source of exposure.
2. Plenty of milk should be given.
3. Mouth wash by borax.
4. Saline purgative to be given.
5. Sodium thiosulphate i.v. to be used.
6. B.A.L.—one course of B.A.L. to be administered.
7. Symptomatic treatment for paralysis.
8. Prophylactic treatment including proper ventilation and periodic medical check up.

Autopsy Findings

1. If taken in concentrated form, appearances similar to corrosive poisoning will be present.
2. Signs of irritant poisoning will be there.
3. Mucous membrane of lips, mouth, pharynx and Oesophagus will be grayish white.
4. Stomach contents are coagulated, albuminous with mucous and blood.
5. Mucous membrane is eroded, inflamed and covered with grayish deposit of mercury or black deposits of mercury sulphide.
6. There is great softening of the walls so great care must be taken during post mortem examination to remove it; perforation is rare.
7. Caecum, colon and rectum are inflamed, ulcerated and gangrenous, if alive for sometime.
8. Liver is congested with cloudy swelling.
9. Spleen is congested.
10. Kidneys are congested, there is necrosis of tubules and deposition of lime.
11. Heart shows fatty degeneration.
12. Postmortem lesions are found in the alimentary canal even when applied on the skin or absorbed from the vagina or uterus.

COPPER

Copper is also known as 'Tamba'. Metallic copper is not poisonous but some of its salts have a medicolegal importance. All salts of copper are poisonous. Copper alloyed with other metals when given in fine powdered state acts as a poison.

1. Copper is a normal constituent of the body and is found in traces in urine, faeces, blood, tissue fluids and liver. It is also detectable in minute amount in various food materials such as potatoes, spinach, beans, spinach, fruits and mineral water. The normal dietary intake of copper is about 3mg/day. In Wilson's' disease there is increased absorption of copper in the system due to congenital defect in ceruloplasmin formation. All copper salts are powerful inhibitors of various enzyme systems.
2. *Elimination of copper from the body:* Copper is eliminated more through faeces and bile than through urine. It is also excreted in saliva, milk *in utero* from the mother to the foetus.

Copper Sulphate (*Blue vitriol, CuSO₄, 5H₂O, Nila tutiya*)

It occurs as large blue crystals, freely soluble in water having metallic taste. On heating upto 100°C, it loses its water of crystallization and becomes white with a blue tint and at 220°C it becomes completely white. On standing, it draws moisture and again becomes blue, as it is hygroscopic. Copper sulphate is used as astringent in small doses; as an emetic in slightly large doses and an irritant in larger doses.

Uses of copper sulphate: (i) It is used as an algacide (ii) It is also used by bookbinders and straw-hat makers (iii) Is a constituent of Fehling's solution I (iv) It was used for removal of excess granulation tissue in wounds (v) To impart rich green colour and for preservation of fruits and vegetables but their dose should not exceed 50mg/kg. But the toxic effects does not result from their use as copper salts are converted into albuminate of copper, which is a harmless substance (vi) In bakeries for fermentation of dough to make breads

in winters (vii) It is commonly used as an emetic in various poisonings except arsenic where it will form cupric arsenite or acetoarsenite of copper and produce toxicity (viii) It is used as an antidote in phosphorus poisoning where it forms cupric phosphide—A non-toxic substance.

Copper subacetate (Verdigris or Zangal):

Copper sulphate is a bluish green salt formed by the action of vegetable acids on improperly tinned copper cooking vessels. It is used as medicine for external use and is used in arts and industry. The signs and symptoms of verdigris poisoning are similar to that of copper sulphate poisoning.

Copper Sulphate Poisoning

Fatal dose→15-30 gms but small repeated doses are more fatal compared to single large dose

Fatal period→1-3 days

Acute Copper Sulphate Poisoning

Signs and symptoms

Signs and symptoms appear within 15-30 minutes of the ingestion of the poisonous substance: (i) Metallic taste in mouth and sense of constriction in throat (ii) Burning pain in the mouth, throat, oesophagus and up to the stomach (iii) Repeated, violent and profuse vomiting; the vomitus is bluish or greenish blue in colour that becomes bluer on addition of ammonium hydroxide. The colour of vomitus will not change if it is bilious (iv) Intense thirst, salivation, nausea and gaseous eructations (v) The gums and tongue are stained bluish (vi) Repeated diarrhoea with liquid, brown motions that is accompanied with tenesmus and colicky abdominal pain (vii) Oliguria, anuria and albuminuria leads to uraemia (viii) Hemolytic tendencies due to increased concentration of copper in the erythrocytes, resulting in hematemesis and malena (ix) Jaundice may develop (x) Muscular cramps, paralysis of limbs, convulsions, dyspnoea, quick, weak and feeble pulse, fall of blood pressure, cold clammy skin and coma and death occurs in 1-3 days (xi) Liver biopsy shows centrilobular necrosis and biliary stasis.

Chemical test: Ammonium hydroxide gives a greenish blue precipitate with substances containing copper salts; this precipitate is soluble in excess of ammonium hydroxide solution giving a deep blue colouration

Treatment

1. No emetics as copper sulphate causes emesis
2. Gastric lavage using 1% freshly prepared potassium ferrocyanide that acts as an antidote. It changes copper sulphate in to insoluble cupric ferrocyanide.
3. Demulcents like milk and egg albumen convert copper sulphate in to insoluble cupric albuminate.
4. To maintain fluid and electrolyte imbalance infuse 5% intravenous dextrose saline with vitamin C and amino acids.
5. Morphine is given to allay pain.
6. As chelating agent, Penicillamine 2 gm in divided doses is given orally for 5-7 days or parenterally 30 mg/kg body wt. or B.A.L. as 300mg i.m stat and then 150 mg 4hourly for next 6-7 days or until recovery.
7. Calcium E.D.T.A. is also given as an antidote

Autopsy Findings

(i) The skin is yellow due to jaundice (ii) Greenish blue froth comes out of the mouth and nostrils (iii) The mucous membrane of mouth, oesophagus, stomach and intestines will be inflamed, congested, swollen and excoriated and bluish green or bluish stained (iv) Stomach contents are stained greenish blue or blue (v) There is rectal perforation sometimes (vi) Liver is enlarged, congested, soft and greasy showing features of fatty infiltration degeneration (vii) Kidneys are congested and features of tubular degeneration with deposition of copper.

Chronic Copper Sulphate Poisoning

Chronic copper sulphate poisoning results from:

- (i) Preservation and cooking of food in unclean copper utensils may result in poisoning as it is

contaminated with verdigris (ii) The chronic consumption of fruits and vegetables preserved and coloured with copper sulphate (iii) Inhalation of copper dust or absorption through skin by workers in copper industry (iv) **Brass-founder's ague** (Metal fumes fever)—Inhalation of fumes of copper and zinc in workers engaged in smelting brass—an alloy of copper and zinc results in this condition.

Signs and Symptoms

(i) Styptic metallic copper taste in tongue and mouth with a bluish-green or purple line on the gums at its junction with the teeth (ii) Lassitude, giddiness and headache (iii) Nausea, vomiting, indigestion and dyspepsia (iv) Occasional diarrhoea with colicky abdominal pain (v) Laryngitis and bronchitis (vi) Peripheral neuritis (vii) Anemia and slight jaundice (viii) Muscular weakness with atrophy may lead to paralysis (ix) Hair may be brownish golden in colour (x) Features of renal and hepatic damage are prominent (xi) Conjunctivitis and corneal ulceration occur due to contact with copper dust (xii) Dermatitis due to skin contamination with copper salts.

Treatment

1. Patient should be removed to fresh air.
2. Correction of dyspepsia.
3. Copper utensils used for cooking should be very clean and well-tinned.
4. E.D.T.A, Penicillamine should be given.
5. Nutritious diet with multivitamins and intravenous fluids to be given.

Autopsy Findings

(i) Fatty degeneration, necrosis of liver cells and haematochromatosis (ii) Degeneration and cloudy swelling of kidney.

Medicolegal Aspects

(i) Copper salts especially copper sulphate are not commonly used for suicidal purposes due to its blue or bluish-green colour, being self-emetic, strong metallic taste and big lethal dose. However,

the compound is used for homicidal purposes (ii) Rarely used for cattle poisoning (iii) Accidental poisoning can occur from contamination of food stored or cooked in copper utensils due to formation of verdigris and eating fruits and vegetables preserved in copper sulphate (iv) Used on the abortion sticks to procure abortion.

IRON

Iron is a normal constituent of the body and is eliminated in faeces and urine. It is present in the food we take and in various multivitamin syrups, tablets, tonics and capsules. The salts of ferrous such as ferrous sulphate and ferrous carbonate are easily absorbed whereas ferric salts are not absorbed. The poisoning may result from ingestion of iron preparation available in the market by children in lieu of sweets. Teenagers and young females can also suffer from iron poisoning after a long period of treatment for anemia, in pregnancy or during postpartum period with these preparations. All iron preparations are dangerous except for ferrous gluconate.

Ferrous sulphate: It occurs as greenish crystals soluble in water and is used in industry for making blue black ink and dyes.

Ferric chloride: It is water soluble and a powerful irritant, forms triferriperchlor that produces sloughing endometritis and even peritonitis when applied locally in vagina and uterus.

Mechanism of action: In dilute forms, the iron preparations act as astringent and corrosive in concentrated form leading to corrosion or perforation of gastric mucosa. It causes increased capillary permeability and inhibition of mitochondrial function. They may cause stricture formation.

Fatal dose→20 -30 gm, Variable (24-48 hours).

Signs and Symptoms

The signs and symptoms are described in three stages:

Stage I: (i) Metallic taste in mouth (ii) Nausea and vomiting and epigastric pain (iii) Hematemesis, diarrhoea with black stools (iv) Severe gastro-

intestinal haemorrhage results in shock followed by convulsions and coma, peripheral circulatory failure and death (v) Iron released from the tablet or solution will exert its necrotizing effects on gastrointestinal tract mucosa and the unbound iron will soon get absorbed to produce systemic manifestations

Stage II: The phase may last for days and with improvement of signs and symptoms, the patient may recover in mild cases of poisoning. In severe cases: (i) Frequent black, offensive stools are passed (ii) Severe headache, confusion, delirium occurs (iii) Convulsions, photophobia, dimness of vision and loss of consciousness develops (iv) Respiration is deep and circulatory collapse can occur.

Stage III: (i) Recurrence of shock and development of hepatic failure (ii) Features of acute hepatic necrosis with jaundice progresses to hepatic coma and death (This results from release of iron from reticuloendothelial system). If patient survives for 3-4 days, recovery is the rule (iii) Pyloric obstruction is a late complication.

Diagnosis: (i) Iron levels in blood and gastric aspirate (ii) Urine for bilirubin, bile salts and urobilinogen (iii) Liver function tests and tests for rise in aminotransferases and prothrombin time should be carried out.

Chemical test: Ammonium sulphide when mixed with ferric or ferrous salts gives black precipitate that is soluble in dilute hydrochloric acid.

Treatment

1. Immediately wash stomach with 1% sodium bicarbonate solution or 5-15% disodium phosphate solution.
2. Desferrioxamine (1gm of Desferrioxamine chelates 85 mg of iron) is the most active antidote against iron poisoning. It is introduced in stomach in the dose of 5 gm dissolved in 100 mL of distilled water through stomach tube and be left out. Desferrioxamine 1 gm in 5 mL of distilled water can be given i.m and further 500 mg in 12-24 hours depending upon the serum iron estimation. Intravenously maximum of 60-80 mg/kg in 24 hours can be infused.
3. Cathartics can be given to evacuate bowel in the absence of perforation.
4. Shock and acidosis to be treated.
5. Symptomatic and supportive treatment.

Autopsy Findings

(i) Evidence of congestion, inflammation and corrosion of gastrointestinal tract (ii) Liver will show hepatic necrotic changes (iii) Kidney will show renal tubular necrosis.

Medicolegal Aspects

1. Ferric chloride is rarely given to intoxicated persons for homicidal purposes
2. Accidental poisoning occurs in children commonly who ingest it in lieu of sweets
3. Locally in the genital tract to procure abortion.

Irritant Poisons

ORGANIC IRRITANT POISONS

ABRUS PRECATORIUS (INDIAN LIQUORICE OR RATTI)

The plant grows throughout India and all parts of the plant are poisonous but the seeds are particularly used for the purpose of poisoning. The seeds of Ratti are 1/3 inches long and ¼ inches broad and are of the size of small pea. Usually the seeds are red coloured, egg shaped having a black spot on one end but it may be of white, black, or yellow colour (Fig. 38.1). Ratti seeds are used as decorative ornaments. The seeds are tasteless and odourless. The active principle is Abrin that is a toxalbumin similar to ricin and its action resembles the viperine snakebite. Besides this, it contains some poisonous proteins such as: (i) Abrussic acid (fat splitting enzyme) (ii) Abrin (nitrogenous compound) (iii) Ureases (iv) Haemagglutinin (v) Abraline (glucoside) (vi) Glycyrrhizin present in root, stems and leaves



Figure 38.1:
Ratti seeds

Preparation of 'Suis' or 'Sutari'

The seeds are first soaked in water, decorticated and made into a paste mixed with opium, onion, *datura*, little oil or water. The paste is then shaped in the form of small, sharp needles with pointed

ends about 2.5cm long and 90-120mg by weight. The needles are then hardened by drying in the sun and then fitted to the holes of a bamboo pole. These needles are stabbed on the buttock of the animal, implanting the needles but detaching the handle. At the site of inoculation of 'sui', there will be inflammation, oedema or necrosis with oozing of haemorrhagic fluid from the puncture site similar to the viper bite. The animal stops eating food for 3-4days, will be unable to move or stand. It will become apathetic and will develop convulsions before it gets comatosed. The collapse soon sets in and the animal dies by 48-96 hours later. The signs and symptoms simulate snakebite.

When the 'suis' are stabbed in the human body, there will be painful swelling with ecchymosis at or about the site of injection. The inflammation sets up and necrosis begins soon. The patient suffers from headache, nausea, vomiting, vertigo, drowsiness and apathy. There is increasing prostration, dyspnoea, quick, weak, irregular and feeble pulse, and cold clammy skin. The blood pressure falls, oliguria, haemorrhagic manifestations and lately uremia may set in. Death occurs from 3-5 days from cardiac failure.

Signs and Symptoms

The ingestion of intact 'Ratti' seeds does not result in poisoning. Powdered seeds are boiled with milk and are used as a nerve toxin but when taken uncooked it acts as severe gastrointestinal irritant causing vomiting and diarrhoea. Abrin is slightly inactivated by gastric juice when crushed seeds are swallowed and it loses all its poisonous properties when boiled:

1. Extracts of seeds when injected under the skin of an animal cause inflammation, oedema and necrosis.
2. In human beings, ingestion of crushed seeds results in: (i) Nausea and vomiting (ii) Vertigo, tinnitus and giddiness (iii) Cold clammy skin and fall of blood pressure (iv) Irregular pulse and laboured breathing (v) Convulsions, haemolysis, oliguria (vi) Death occurs from cardiac failure.

Fatal dose→90-120mg (½ to 1 crushed seed)

Fatal period→3-5 days rarely even 24 hours.

Treatment

1. Gastric lavage and emesis is carried out.
2. Locally the part containing 'sui' should be excised in both humans and animals.
3. Sodium bicarbonate 10 gm orally per day helps in maintaining alkalinity of urine and prevents agglutination of red cells and blocking of renal tubules with haemoglobin G.
4. Antiabrin, an antiserum that is prepared by injecting abrin in rabbits, is to be administered.
5. A mixture of dilute hydrochloric acid and pepsin is to be give orally.
6. Symptomatic treatment.

Autopsy Findings

(i) Inflammatory oedema and necrosis at the site of injection (ii) Sometimes fragments of 'suis' are found at the site of injection (iii) Gastrointestinal tract is congested and haemorrhagic (iv) Petechial haemorrhages are seen in the skin, pleura, pericardium as well as peritoneum (v) The visceral organs are congested.

Medicolegal Aspects

(i) It is a mainly a cattle poison (ii) 'Suis' are made by mixing with *Datura*, Opium, Onion and Spirit and used for poisoning the cattle and also for homicidal purposes (iii) The seeds act as an abortifacient preventing conception (iv) The application of powdered seeds on to the eye by malingerers to produce conjunctivitis (v) The paste made from the powder of Ratti seeds is used as an arrow poison and also as an aphrodisiac.

SEMECARPUS ANACARDIUM (MARKING NUT, BHILAWA)

The plant of marking nut is found all over India and the fruit of this plant is called marking nut (Bhilawan). The fruit is black, heart-shaped, having a rough projection at the base. It has a hard black rind, which contains a thick pericarp. This pericarp or fleshy pulp contains brownish oily acrid juice turns black when mixed with the lime and exposed to air used by washerman for marking cotton clothing and linen. That is why, it is known as marking nut. The juice mixed with the melted butter is used as a remedy for syphilis. The active principles are: (i) Semicarpol—0.1% (ii) Bhilawanol—15-17% (iii) Non-volatile corrosive residue—18% (iv) Fatty oils and tannic acids.

Symptoms and Signs

On external application: (i) When applied externally, it causes irritation and itching. (ii) When it is applied to the scrotum, it causes fever, haematuria, and painful micturition. (iii) Blisters are painful, black and raised containing acrid serum (iv) The acrid serum causes eczematous eruption of the contact area of skin (v) Blisters resemble bruises and may get infected and ulcerate (vi) The blisters are also associated with fever, Dysuria, haematuria and painful defecation. (vii) It is possible to detect poison from the serum from the blisters.

Table 38.1: Differences between true and false bruise

Features	True bruise	False bruise
Swelling and redness	Present	Not present
Ecchymosis	Present	Not present
Contents	Blood	Serum
Itching	Not present	Present
Fingers	No marks over the fingers	Fingers may show marks due to the juice
Chemical analysis	Negative	Positive for the vegetable poison

When ingested

(i) Intense, painful irritation with blister formation locally in the mouth, tongue and throat (ii) Gastroenteritis with nausea and vomiting (iii) Tachycardia

(iv) Quick weak pulse and hypotension (v) Pupils are dilated (vi) Cyanosis and dyspnoea (vii) Delirium and areflexia (viii) Collapse, coma and death occurs within 12-24 hours.

Fatal dose→5 gm

Fatal period→ Uncertain, usually 12-24 hours

Treatment

1. *External application:* Washing with lukewarm water with some antiseptic and application of antibiotic cream or ointment, if infected
2. *When ingested:* Stomach wash with plain water; Demulcent drinks; analgesics for pain and symptomatic measures to be employed.

Autopsy Findings

- (i) Fatty degeneration of the liver (ii) Blisters in the mouth, throat and even stomach

Medicolegal Aspects

(i) Accidental poisoning occurs when *Vaid*s administer the juice of *Semecarpus anacardium* for the treatment of pain or for treating paralysis (ii) Homicidal poisoning is rare (iii) It is criminally introduced in to the vagina as a punishment for infidelity (iv) The juice of marking nut is applied on the skin for the false charge of bruise (v) It is applied on to the uterus for criminal abortion (vi) It is sometimes applied on to the eyes by malingerers to produce ophthalmia.

RICINUS COMMUNIS (CASTOR OIL PLANT OR ARANDI)

History and Background

Ricinus communis or the Castor oil plant is cultivated in southern United States and also grows as a weed and ornamental plant (Fig. 38.2). It is native to tropical Africa but has naturalised sub-tropical and temperate areas as well. The whole plant is poisonous, containing the heterodimeric ribosome inhibiting protein ricin that reaches the highest level in the seeds. It consists of a water-soluble glycoprotein, Toxalbumin that causes agglutination, cell destruction and haemolysis. Its residue contains the toxin, ricin. The seeds also contain purgative oil, the triglyceride of ricinoleic acid. In the tropics, eating a single



Figure 38.2:
Plant of *Ricinus communis*

seed for the purgative effect is a widespread custom. The seeds have been used in folk medicine against many diseases for centuries. They appeared in classical Greek medicine and are described in the Sanskrit work on medicine, *Sushruta Ayurveda* from the 6th century BC. The oil is used for lubrication in the motor industry and is still being used as a laxative.

The dust of this plant causes irritation of the eyes, nose and throat that causes asthma, allergy and dermatitis. Each fruit of *Ricinus communis* contains three mottled seeds, 5-15mm long, that are red grey or polished brown in colour. The poison is more effective when injected than when taken by mouth as it is destroyed by digestive enzymes. The plant also contains compounds which may elicit anaphylactic reactions. Because of this, ingestion of any dose of *Ricinus communis* can be fatal within minutes. Ricin has been known as a poison for years, usually through livestock deaths. The cattle may eat fruit or in our country, malicious neighbours are said to have administered extract of castor bean to their neighbour's cattle using a metal nail or 'Suis'. In Nigeria the beans are eaten as a food after careful preparation as heat inactivates the toxin, but in East Africa, unwanted children in the past have been murdered by adding castor beans to their food. The seeds of castor are commonly mistaken for that of croton but can be clearly distinguished on close inspection (Table 38.1)

Fatal dose→The fatal dose is 1mg/kg or 8-10 crushed seeds in adults and 2-3 seeds in children. As lots of ricin is destroyed in the gastrointestinal tract, it is much more potent when administered parenterally. A dose of 2 millionths of the body weight may prove fatal.

Fatal period→2-24 hours

Table 38.1: Differences between castor and croton seeds (Figs 38.3 and 38.4)

Features	Castor seeds	Croton seeds
Appearance	Grayish with brown patches	Dark brown or brownish gray shell which on scrapping becomes black
Size	1.25 cm long and 0.85 cm broad	1.2 cm long and 0.82 cm broad
Cross section	Lumen is circular	Lumen is slit like with radiating creases
Transverse striae	Cells show ribbed appearance due to fine, transverse striae	No transverse striae and no ribbed appearance



Figure 38.3: Castor seeds



Figure 38.4: Croton seeds

Signs and Symptoms

Toxic effects of Ricin have a latent period and take 2-24 hours to develop.

After ingestion: The symptoms are: (i) Burning pain in the abdomen (ii) Vomiting and diarrhea often with blood (iii) Haemorrhages occur in intestines, mesenteries and omentum (iv) A diffuse nephritis and multiple necroses occur in liver and kidneys (v) Within several days there is severe dehydration, oliguria, thirst, burning throat, headache and patient may die of hypovolaemic shock (vi) The body temperature decreases before death and the patient often undergo a characteristic shivering (vii) Consciousness remains till the end (viii) Death occurs due to exhaustion or cramps.

When administered parenterally: Ricin is twice as toxic as cobra venom and is probably the most toxic parenteral substance in the plant kingdom. After parenteral administration, the patient may present with fever, leucocytosis and then hypotension and hypothermia. It primarily targets the liver and the kidneys.

Treatment

Measures should be undertaken to prevent absorption after ingestion of the poison as there

is no specific antidote for Ricin: (i) Stomach wash using potassium permanganate should be carried out (ii) Syrup of ipecac and activated charcoal should be administered (iii) Stimulants should be given (iv) Intravenous glucose saline to be administered for dehydration (v) Sodium bicarbonate (vi) Supportive care including blood transfusion to combat blood loss (vii) The patient should be constantly monitored for hypoglycemia, haemolysis and hypovolaemia.

Medicolegal Aspects

(i) The poisoning is usually accidental when is taken by children by mistake (ii) It is also given mixed in food for criminal intent (iii) The powder when instilled in to the eyes causes conjunctivitis (iv) The castor oil is non poisonous and is used as purgative (v) The triglyceride of ricinoleic acid may act as an irritant poison (vi) It is used to make 'Suis' and injected in to the buttock of the animal.

Autopsy Findings

(i) There is congestion of stomach, intestine and other visceral organs (ii) Inflammation and hemorrhages in the stomach, intestines, mesenteries and omentum are identifiable (iii) There is softening of the organs (iv) Fragments of seeds may be recovered from the stomach (v) There are findings of hepatic and renal necrosis.

CROTON TIGLIUM (JAMALGOTA OR NAEPALA)

The plant grows all over India and the seeds as well as oil expressed from the seed and the root of the plant are highly poisonous. The seeds are 0.65 × 0.85 cm in size, grayish white in colour with a white oily kernel having some longitudinal

lines over the surface. Croton oil is vesicating agent, has brown colour and disagreeable odour and contains *crotonoleic acid*, *methyl crotonic acid*, *crotonol* and other fatty acid. The oil does not contain *crotonin* that is left out in the croton seed cake on expression of the oil. When applied on the skin, it causes burning, redness, vesication and suppuration. When the seeds are swallowed, they cause irritation of the gastro-intestinal tract. Croton seeds are poisonous to fish and a case is reported where croton oil was used to poison fishes at Midnapur, West Bengal.

Active Principles

The active principles of seeds of *Croton tiglium* is:

- (i) Croton a toxalbumin is less poisonous than Ricin
- (ii) Crotonaside is a glycoside.

Signs and Symptoms

It is a gastrointestinal irritant and produces symptoms similar to Ricin poisoning (i) Severe hot burning pain in mouth, throat and abdomen (ii) Salivation, nausea, copious vomiting (iii) Frequent watery stools mixed with blood and mucus accompanied with gripping pain abdomen, anal irritation and tenesmus (iv) Features of dehydration are prominent (v) Quick weak pulse, shallow respiration and vertigo (vi) Circulatory collapse and death (vii) Being toxalbumin, it produces agglutination of red cells causing hemolysis, jaundice, pain in renal angles, haemoglobinuria (viii) The blocking of renal tubules leads to oliguria, anuria and uremia and resulting renal failure accounts for delayed deaths (ix) On application to skin, it produces burning, redness and vesication. The vesicles may suppurate resulting in scarring on healing. On contact with eyes will produce conjunctivitis and corneal ulcer.

Fatal period→4-6hours to days.

Autopsy Findings

(i) Gastrointestinal tract mucosa shows erosion, inflammation and congestion (ii) Liver, and spleen are congested (iii) Kidneys may show cloudy swelling and tubular necrosis (iv) In rare cases, the findings are negative.

Medicolegal Aspects

(i) Poisoning results from eating seeds or inhaling dusts (ii) Accidental poisoning results from over-dosage of croton oil that is used as purgative or administered after mixing with food (iii) It is rarely suicidal (iv) Used for homicidal purposes by boiling the root and adding to food (v) Root of the plant is commonly used as abortifacient (vi) It is used as an arrow poison

CALOTROPIS PROCERA AND GIGANTEA (MADAR OR AKDO) (Fig. 38.5)

The *Calotropis* plant is found all over India in two varieties; *Calotropis gigantea* and *Calotropis procera*. The dried root freed from its outer cortical layer is called *Madar*. Its constituents are Madaralbum, a crystalline colourless substance; Madarfluavil, an amber coloured viscid substance and Mudarine, gelatinizes on heating.



Figure 38.5:
Calotropis procera

Active principles: (i) Uscharin (ii) Calotoxin (iii) Calotropin (cardiac glycoside) (iv) Calactin.

Uses: (i) The flowers are dried and powdered and used as digestive, carminative and tonic (ii) The dried leaves are used as purgative in small doses; applied locally it relieves headache and in large doses act as an emetic (iii) The tincture prepared from bark or root may be used in dysentery (iv) The acrid milky juice may be used as an emetic, purgative, vesicant and also for treatment of chronic skin diseases (v) The dried bark is used by vaidas as an expectorant, emetic and purgative; bark may also be utilized for making madar or ushar fibres (vi) Madar juice or the powdered bark or root of the plant may be used by tanners as a depilatory; this imparts yellow colouration to the skin and destroys offensive odour of fresh leather (vii) The

plant may also be used for treatment of impotence, asthma, syphilis, gonorrhoea and elephantiasis.

Medicolegal Aspects

(i) Madar juice is commonly used as an abortifacient either taken by the mouth or applied locally into the uterus by abortion stick, either alone or in the form of paste with lead oxide or arsenic preparation (ii) Madar juice may be used for the purpose of homicide, suicide or infanticide; for infanticide, the madar juice may often be given by mouth mixing it with milk and water (iii) Madar juice is commonly used as a cattle poison, when a cloth or rag smeared with the juice is either given with fodder or introduced into anal or vaginal canal of the animal (iv) The root of calotropis procera is reputed to be strongly poisonous to cobras and other poisonous snakes, who cannot stand its smell. Hence, snake charmers may carry it to control freshly caught cobras (v) When used with snuff, powdered madar root may cause death (vi) The juice can often be used to produce artificial bruises (vii) It may also be used as arrow poison.

CAPSICUM ANNUM (LAL MIRCH, CHILLIES)

Capsicum fruits or seeds are extensively used in India as a condiment, in preparation of curries, *chutneys* etc. They have pungent acrid odour and taste, which lasts for a long time; may be used as stomachic and carminative.

Chilli seed is approximately 0.30 cm (1/8") long and 0.20 cm (1/10") wide; they resemble very much *datura* seeds (Figs 38.6 and 38.7).



Figure 38.6: Red chillis

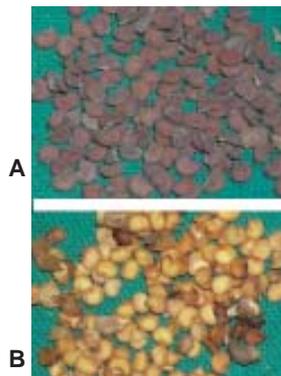


Figure 38.6: (A) Datura and (B) chillis Seeds

Differences between chilli and datura seeds

Features	Chilli Seeds	Datura Seeds
Size	Smaller in size	Larger in size
Shape	Round	Kidney
Surface	Smooth	Finely pitted or reticulated
Colour	Yellowish	Brownish
Smell	Pungent	Odourless
Taste	Pungent	Bitter
Convex Border	Single-edged	Double-edged
On section	The embryo lies embedded in the fleshy albumen and curved inwards	The embryo lies embedded in the white oily albumen and curved outwards

Active principles: (i) Capsaicin (ii) Capsaicin

Signs and symptoms: (i) Burning sensation in mouth (ii) Profuse salivation, thirst and difficulty in swallowing (iii) Pain in abdomen and constipation (iv) Burning sensation around the anus during passing stool (v) Nausea and vomiting (vi) Irritation and vesication on the skin, when applied locally (vii) Burning, lacrymation and chemosis, when applied to eyes (viii) Burning, lacrymation, coryza and cough, due to fumes of burned chillis.

Fatal Dose→Low toxicity

Fatal Period→Fatality is unknown.

Treatment

(i) No specific treatment (ii) Stomach wash with plain water (iii) Cathartics should be given (iv) Irrigation of eyes with plenty of cold water or sterile normal saline and application of eye drops, in case of eye contamination (v) Skin contamination should be treated with plenty of water and an emollient cream locally.

Medicolegal Aspects

1. Chilly powder may be thrown into the eyes to rob or steal valuables, money etc. from the persons by burglars and robbers specially from the bank counters
2. It can also be used to stupefy and disable a person to facilitate commission of crime
3. Chilli powder may be used for the purpose of torture to extract money or to extort confession of guilt, when it is introduced into the nostrils,

eyes, vagina or urethra or rubbed over the breasts of females, burnt under the nose or when head is covered by nose-bags containing chilly powder.

4. Burning of chilly seeds may at times be done by superstitious people to scare away devils and ghosts.

PLUMBAGO ROSEA, PLUMBAGO ZEYLANICA OR LAL CHITRA OR CHITRA

They grow as two types, *Plumbago rosea* (red flowers) and *Plumbago zeylanica* (white flowers) all over India.

Active principles: The active principles is plumbagin contained in the roots and other parts of the plants. It is a highly acrid crystalline glycoside, the non-alkaloidal active principle. Plumbagin is powerful irritant and in small doses it stimulates contractions of heart, intestine and uterus. In large doses, it produces respiratory failure.

Fatal dose and Fatal period→Uncertain.

Signs and Symptoms

On external application of crushed roots and twigs of plants in the form of paste causes swelling, redness and inflammation and blister formation.

Ingestion of crushed roots in large doses causes: (i) Burning pain in mouth, throat and stomach with vomiting (ii) Intense thirst and diarrhea (iii) Convulsion (iv) Uterine contractions leading to abortion (v) Collapse and death from cardio respiratory failure.

Treatment

1. In case of local application of paste or abortion stick, it should be removed and vaginal douching with warm water and an antiseptic solution.
2. In case of ingestion, stomach wash with plain warm water, demulcents, stimulants, analeptics to combat shock and collapse.

Autopsy Findings

(i) Inflammation and injury in vagina, cervix and uterus when used as an abortifacient (ii) Gastro-intestinal irritation with congestion of all internal viscera (Fig. 38.7).



Figure 38.7: Congestion and haemorrhagic gastritis in irritant poisoning

Medicolegal Aspects

(i) The crushed roots of plumbago are used to procure abortion. The quacks used crushed roots as birth control pills (ii) It is less commonly used for homicidal purposes (iii) The paste or milky juice of crushed stems or leaves is applied to the skin to form reddish brown artificial bruise (iv) The powdered leaves and roots may be used in the treatment of leprosy, scabies and influenza etc.

ERGOT

Ergot is dried sclerotium of the parasitic fungus *claviceps purpurea* that grows on cereals like wheat, rye, oat, barley and *bazra* etc. in wet seasons and ill-drained soils. The spores of ergot contaminate the grains through insects or wind. The spores then replace the grain and form dark purplish bodies larger than the original grain. These diseased grains when collected, dried and powdered constitutes ergot for the market.

Alkaloids of Ergot

These are grouped into:

1. Amino-acid alkaloids that comprises of: (a) Ergotoxine (mixture of ergocryptine, ergocornine and ergocryptine) (b) Ergotamine (c) Ergosine.
2. Lysergic acid and amino-alkaloids that comprises of: (a) Lysergic acid (b) Lysergic and diethyl-amide (c) Ergonovine (ergometrine) (d) Methyl ergonovine (e) Methysergide.

Ergotoxine: It promotes contraction of pregnant uterus in last months of pregnancy but no or little effects are there on the uterus in other periods. It also produces gangrene by causing vasoconstriction.

Ergometrine: It is an active ecbolic and it causes prolonged uterine contraction especially

on post-parturial uterus. It does not usually produce gangrene.

Lysergic acid and amino-alkaloids: They cause stimulation of central nervous system.

Ergot acts on smooth muscles of intestine, uterus and arterioles and it is commonly used in obstetrics for its ecbolic action. It is also used in the treatment of migraine, as abortifacient and in veterinary practice.

Fatal Dose→1-2 grams

Fatal Period→The death does not occur from single large dose but when small or medicinal doses are given for a long time. The fatal period is usually one day but death may be delayed for several days.

Signs and Symptoms

Acute poisoning: The signs and symptoms of acute poisoning are: (i) Dryness and irritation of throat with intense thirst (ii) Nausea and vomiting (iii) Pain abdomen and diarrhoea (iv) Vertigo, giddiness and dizziness (v) Tightness and sense of constriction in chest (vi) Disturbance of speech and vision (vii) Tingling of hands and feet with feeling of numbness and coldness (viii) Muscular weakness and cramps in muscles (ix) Body temperature falls with hypoglycemia and suppressed urine (x) There may be abortion or bleeding from uterus (xi) Epistaxis, haemetemesis and haematuria may occur (xii) Convulsions and confusion leading to stupor and coma and finally death results from cardio-respiratory failure (xiii) In survivors, the extremities may develop gangrene.

Chronic Poisoning (Ergotism): Ergotism develops in those taking medicinal preparation of ergot or consumption of flour contaminated with ergot for a long time. The patients with focal sepsis and hepatic damage are more sensitive to ergot alkaloids. Signs and symptoms may present in convulsive or gangrenous form.

Convulsive form: This form is presumed to occur with consumption of flour containing about 10% of ergot and is associated with vitamin A deficiency. The patient presents with (i) Tingling and itching (ii) Feeling of numbness in hands and feet and pain on walking (iii) Sensation of insects

crawling under the skin (iv) Drowsiness and giddiness (v) Dimness of vision and fixed dilated pupil (vi) Loss of hearing and ataxia (vii) Loss of arterial pulsation and finally tonic or clonic convulsive seizures (viii) Paralysis of sensory nerves and maniac depressive psychosis (ix) Death results from asphyxia due to spasm of respiratory muscles.

Gangrenous form: This form occurs with consumption of flour containing more than 10% of ergot. The following signs and symptoms are produced (i) Pain in the limbs with feeling of hot and cold sensation (ii) Tingling and numbness in fingers and toes resulting from vasospasm of arterioles (iii) Swelling of limbs and feet with red patches and blisters (iv) Loss of sensation of the part followed by gangrene formation from vasospasm and vasoconstriction with obliteration of the lumen of small blood vessels (v) The gangrene is of dry affecting fingers, toes and limbs that become cold to touch and dark in colour. The gangrene may extend upto elbow or knee and may affect the nose, ears or even intestines with formation of ulcers

Treatment

In case of acute poisoning: (i) Stomach wash with plain warm water, tannic acid or activated charcoal (ii) Purgatives or enema can be given (iii) Vasodilators such as amyl nitrite can be given orally or parenterally (iv) The convulsion should be treated (v) Heparin should be given i.m for hypercoagulability of blood

In case of chronic poisoning: (i) The patient should be removed from the source of poisoning (ii) Sodium nicotinate 140 mg i.v. to counteract the vasoconstrictive affect of ergotamine (iii) Gangrene to be treated by amputation (v) Nitrites and papaverine 100-150 mg orally or parenterally to restore circulation in incipient gangrene.

Autopsy Findings

(i) Gastrointestinal tract will be congested with inflammatory changes (ii) All the visceral organs will be congested (iii) Gangrene of toes and fingers is evident (iv) Small blood vessels will show

evidence of intimal degeneration

Medicolegal Aspects

1. Acute ergot poisoning may result from over dose of ergot preparations to cause abortion.
2. The use of ergot preparations for a long time can result in ergotism

CANTHARIDES (SPANISH FLY)

The dried beetle *Cantharis vesicatoria*, known as 'Spanish fly' contains a vesicant, cantharidin. Contact of either the live or the dried beetle with the skin results in immediate burning with vesication. Besides this, other beetles causing similar pictures include *Epicauta cinerea* (blister beetle) and members of genus *Paederus*. The compound is used as an aphrodisiac in both humans and cattle.

Signs and Symptoms

- A. *The ingestion results in:* (i) Burning sensation and vesication in mouth (ii) Nausea, vomiting and bloody diarrhoea (iii) Circulatory collapse (iv) Coma, convulsions and death.
- B. *Local application on genitals results in:* (i) Intense haemorrhagic cystitis and urethritis with resulting priapism (ii) An intense pelvic congestion leading to abortion and priapism (iii) The onset of oliguria, haematuria and finally anuria heralds the appearance of several renal involvements

Fatal dose→10-120 mg.

Treatment

1. Gastric lavage to be carried
2. Supportive and symptomatic treatment.

Autopsy Findings

(i) The forewings of the beetle may be found in the gastrointestinal tract (ii) Vesication of mouth and oesophagus may or may not be present (iii) Intense congestion and haemorrhage of the gastrointestinal tract (iv) Urinary bladder may be haemorrhagic (v) Haemorrhagic toxic nephritis in patient who survive longer

Snakes belong to the class Reptila, order squamata, and sub-order Serpentes. The snakes are found all over the world except New Zealand, Ireland and in Arctic lands. Of the 2500-3000 species of snakes distributed world-wide, about 500 are poisonous to Humans. There are 238 species of snakes in India out of which only 52 are poisonous. Of these 52 poisonous species majority of bites and consequent mortality is attributable to 5 species viz. *Ophiophagus hannah* (king cobra), *Naja Naja* (common cobra), *Daboia russellii* (Russell's viper), *Bungarus caeruleus* (krait) and *Echis carinatae* (saw-scaled viper).

In India, deaths as a result snake-biting are mainly accidental in nature. It has been reported that in most developing countries, up to 80% of individuals bitten by snakes first consult traditional practitioners before visiting a medical centre. It is reported that there were about 200,000 bites and 15,000 deaths in India due to snake bite poisoning as far back as 1954. In Sri Lanka, the overall annual mortality from a single venomous species ranges from 5.6 per 100,000 to as high as 18 per 100,000 in some areas. Myanmar seems to have the highest mortality in Asia and 70% snakebites are by Russell's viper. However, this may only reflect a better reporting system prevalent in that country. Maharashtra, one of the states of India with the highest incidence, reported 70 bites per 100,000 population and mortality of 2.4 per 100,000 per year. The other states with a large number of snakebite cases include West Bengal, Tamil Nadu, Uttar Pradesh and Kerala.

General Characteristics of Snakes

A snake is a reptile without limbs and has a short tail. Vent is the opening in the rear part of the body for intestinal and genitourinary system. The part behind the vent is the tail that is flat in sea snakes and round in land snakes. The body of snakes is covered with scales. On the head, there are two eyes, two nostrils but no external ear. The eyes are covered with transparent scale bearing no eyelids and the pupils are round or vertical. All snakes are cold-blooded creatures with carnivorous habits. They use sharp teeth and strong

SNAKES

muscles to catch the prey. The lower jaw comprises of two bones, in front joined by an elastic ligament and it does not properly articulate with the upper jaw. The mouth of the snake is easily distensible allowing it to swallow even large animals as a whole. Their pointed and backwardly teeth are found on both jaws and palate. The upper marginal teeth get modified to form fangs. A new fang will appear in place of a broken one within 4-6 weeks. The fangs are solid in nonpoisonous snakes but are grooved or channeled in poisonous ones for transport of venom from the poison glands to which they are connected through ducts. In poisonous snakes, parotid glands situated behind and below the eyes, one on each side secreting toxic saliva, and act as poison glands. The fangs of elapid snakes and sea snakes are short, fixed and grooved while those of vipers are long, movable and canalized. The tongue of the snake is forked at the tip and it acts as a sense organ for its search for food, other sex or enemy. Because of the shape of the body, the paired organs are laid one behind another. Poisonous snakes have only one right lung. The snake moults its skin several times a year throughout its life to keep oneself active. The snakes move by gliding motion.

Some snakes lay eggs, others give birth to young ones. Several species of egg layers such as pythons, cobras and water snakes have been found to stay with the eggs until hatching. The King cobra is the only snake in the world that

builds nest. In India the snakes range from the giant reticulated python (*Python reticulates*) that grows to 10 meters, to the tiny worm snake (*Typhina bramina*) which grows just 10 cm, and often mistaken for a worm. The King Cobra (*Ophiophagus Hannah*), the largest venomous snake in the world, which grows to 5 meters in India, is 'ophiphagus' that is they eat only snakes, mostly the ubiquitous rat snake and sea snakes eat fish.

Differences between poisonous and non-poisonous snakes: There are certain physical characteristics that help to distinguish between the venomous and non-venomous snakes. Of these there are the belly scales that can be seen after turning the snake with belly upwards. The other distinguishable features are the size of head scales, fangs, appearance of tail, the teeth bite mark over the body of the victim and the habits of the snake whether they are nocturnal or diurnal (Table 38.2).

VENOMOUS SNAKES

There are more than 52 species of venomous snakes in India of which there are four important types of snakes. These are King cobra, Krait (*Bungarus Russell*), Russell's viper (*Vipera Russell*) and the saw-scaled viper (*Echis carinatus*). Each of these snakes is easily distinguishable, though it is easy to confuse them with other harmless species. The venomous snakes are

Table 38.1: Differences between venomous and non-venomous snakes (Figs 38.8 to 38.10)

Features	Venomous snake	Non-venomous snake
Belly scales		
• Size	Large	Small
• Distribution	They cover the entire breadth of the belly. Some non poisonous snakes may also possess such scales similar to those on the back or may be moderately large.	Never cover the entire breadth of the belly
• Size of head scales	Usually small as in vipers except in pit vipers, Cobras or coral snakes and Kraits where they are large	Usually large
• Fangs	Are hollow like hypodermic needles	Short and solid
• Tail	Compressed	Not markedly compressed
• Habits	Usually nocturnal	May be nocturnal or diurnal
• Teeth-bite marks	Two fang marks are left at the site of bite usually with or without marks of other teeth. Usually they have two long teeth or fangs	Two fang marks are left at the site of bite and a number of several small teeth marks

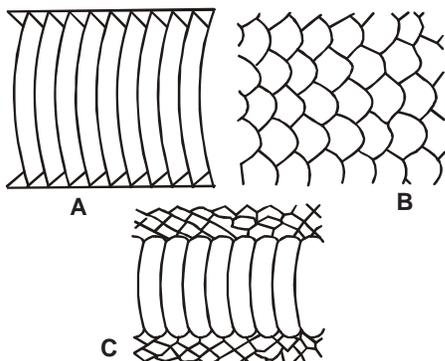


Figure 38.8: Large belly scales of venomous snakes with various patterns such as in (A) Belly scales small like those on the back as in (B) or moderately large but not covering the entire breadth of belly as in (C)

classified into three families based on the type of poison secreted.

I. Elapidae or Colubridae that secrete Neurotoxic poisons

Colubridae (Colubrid Snakes)

The colubrine snakes lay eggs. Their oval heads covered by large scales are nearly of the same girth as of their neck that is indistinct and the pupils are circular. Their anteriorly situated fangs are short, fixed and grooved, thus they cannot effectively bite through the clothing and inject a complete lethal dose. The Colubrid snakes are sometimes referred to as “typical snakes”. They comprise the largest family by far with over 2000 species worldwide. Most are medium sized snakes, and all lack a pelvic girdle and have no vestigial hind limbs and whose left lung is either absent or greatly reduced. Most species are considered members of two large subfamilies, Colubrine and Natricinae which are distinguished by the presence (Natricinae) or absence (Colubrinae) of spines on the lumbar vertebrae. The colubrines are subdivided into Elapidae or land snakes, viperidae and hydrophidae or sea snakes.

Elapidae (Cobras and Coral snakes)

The cobra family is thought to have evolved from Colubrid snakes and many appear very similar in appearance with long, slender bodies and large

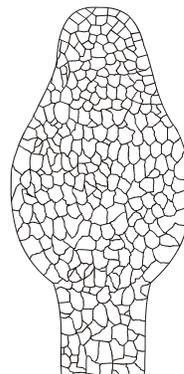


Figure 38.9: Small head scales of venomous snakes e.g. Russell viper

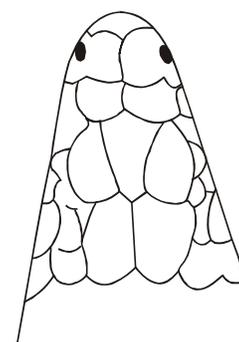


Figure 38.10: Large head scales of non-venomous snakes

scales (plates) on the head. They differ in having more advanced venom delivery systems than the venomous Colubrids. Elapids have fangs that are effectively tubular in that the fangs contain grooves that are enclosed by an infolding of the edges. The fangs are in the front of the mouth rather than the rear as is seen in venomous Colubrids. The Elapidae contains some of the world’s most dangerous snakes including: (i) Common cobras (*Naja naja*) (ii) King cobra (*Naja hanna*) (iii) *Kraits*: Kraits are bluish black in colour, shiny with transverse thin white cross bands, which may be very faint or sometimes even absent. They are subdivided into (a) Common Krait (*Chitti*) and (b) Banded Krait (*Sankhini*) (iv) Mambas (*Dendroaspis*) (v) Sea snakes (*Hydrophinae* and *Laticaudinae*) (vi) Coral snakes

In cobras and the coral snakes the third labial head scale touches the eye and nasal shields.

II. Viperidae that secrete Vasculotoxic poisons

The viperines are viviparous that is they give birth to young ones. The vipers are generally considered to be the most advanced family of snakes since they possess a very sophisticated venom delivery system. Large movable and tubular fangs are placed in the front of the mouth and they are hinged, allowing them to be folded back when not in use. The vipers can bite through the clothes

and inject the venom. Their triangular heads are covered with numerous small scales and their eyes have vertically elliptical pupils. All the viperine species are poisonous. The viperine snakes have following subfamilies: (i) Saw scaled viper (ii) Pit viper (iii) Russell's vipers.

III. Hydrophidae or Sea-Snakes that secrete Myotoxic poisons

There are about twenty types of sea snakes found in India and all are venomous. They have small heads, small eyes and a flat tail that helps in swimming. Their valved nostrils are situated on the top of the snout for free breathing. The belly plates are not broad and have dull trabeculated scales on their back. The poison glands are delicate with short fangs that are situated posteriorly. Their bodies are greenish black, dark black or bluish black in colour.

Common Cobra (*Naja naja*, Nag, Gokhurra)

Common cobra is seen throughout India, Sri Lanka and Burma. These are 1.5-2 meters long. The cobra varies in colour from cream to black, though generally it is brown. The hood may have the typical spectacle or monocle marking on its underside, which are two dark round spots and at times only one spot surrounded by an ellipse. The characteristic hood of the cobra is visible only when it raises its head while in danger, enraged or about to strike. The underside of the hood also bears three dark bands on the central part and a white band in an area where hood touches the body. The divided tail shield helps in identifying a cobra when hood is not visible. The maxillary bone extends beyond the palate and the poison fangs are followed by one or two small teeth.

King Cobra (*Naja bangarus*, Raj Nag or Daras)

The King cobra is found the hills and forests of South India, Orissa, Assam and the Himalayas. It is bigger in size compared to the common cobra and its length is usually 2.5-4.5 meters. The head is flat with a hood but no spectacle mark on it. It

is usually jet black but can also be brownish to greenish and sometimes yellow. The tail scales are entirely present in their proximal ends but divided in the distal ends (Fig. 38.11 to 38.13).

Common Krait (*Bangarus caeruleus*)



Figure 38.11: Cobra and its belly scales



Figure 38.12: Hood of cobra showing spectacle mark

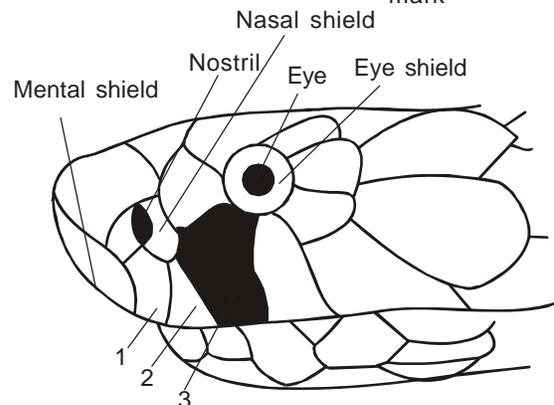


Figure 38.13: In Cobra head scales are large and third labial touches the eye and nasal shields with round pupil

The common krait is found throughout India and is known by the names of *Chitti* (Calcutta), *Kawriya* (Punjab), *Maniyar* (Maharashtra). They are nocturnal and their habitat is near dwelling houses. The size may be 1.5 meters long and are usually steel black in colour. They bear single or double narrow bands across their back that extends from below the head up to the tip of tail. The belly is creamy white in colour. Their head are oval with one enlarged central row of hexagonal scales on the back. The undersurface of the mouth has only four infra-labials-the fourth is the largest and there are bands or half rings across the back. The third labial scale does not touch the nose and eye. The tail is round

with tail scales that are entirely placed and not divided.

Banded Krait

These snakes vary in length from 2.5 to 3.5 mts, longer, bigger and stouter than common krait. It bears alternate jet black and yellow bands across its back, hence the name. It has a black mark on its neck spreading up to the eyes. It is deadly poisonous snake (Figs 38.14 and 38.16).

Rat Snake (*Zamenis mucosus*, Dhamana

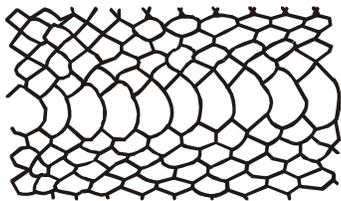


Figure 38.14: Central hexagonal scales on the middle of the back of krait

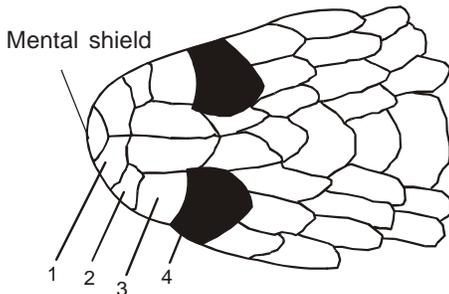


Figure 38.15: Fourth infralabial in krait is the largest (View is from below)



Figure 38.16: Banded krait

Samp)

They may look like a cobra but do not have a hood, spectacle mark or monocle mark on its head. They are 5-6ft in length with large nostrils.

Coral Snakes

The coral snakes are relatively small snakes that spend most of their time underground and are found in hilly areas. Their size may be up to 1.5 mts (3ft) and their primary food is other snakes. The bellies are pinkcoral coloured and the tail is round. Despite their small size and small fangs, their venom is extremely toxic.

Pit vipers (*Crotalidae*)

Pit vipers have a pair of heat sensing pits located on the head between each eye and nostril. The pit helps in detecting warm blooded prey in the dark. They are usually 2-4 feet (30-100 cm) in length and are found in the hills. These snakes are seldom fatal to humans. Their body is flat and broad and triangular head. They are found in hilly areas with their distinct green or sometimes yellow color (Fig. 38.17).

Russel's viper (*Chandra Bora* or *Daboia*)

Russel's vipers are heavy bodied snakes with narrow necks, triangular heads and three longitudinal regular chain-like pattern or rows on the back. The scales are quite rough and the overall colour is yellowish and brown. It is found throughout India in the plains. It is 1-1.5 m long and is stouter than any other poisonous snake. Head scales are small and the nostrils are bigger than any other Indian snake. The tail is short with divided shields and the belly scales are broad. It makes a loud hissing sound before striking the enemy (Fig. 38.18).

Saw-scaled Viper



Figure 38.17: Pit viper



Figure 38.18: Russels viper

(Phoorsa or Echis carinata)

The saw-scaled viper is the smallest of these four types of snake, growing to just a foot, it is brownish with white marking and has a triangular head typical of the vipers

The scales on the head are small and that on the body are broad having serrated edges. It makes a peculiar rustling sound while moving because of its saw-like scales on the body (Fig. 38.19).

Differences between Cobras and Vipers



Figure 38.19: Saw scaled viper

Features	Cobras	Vipers
Body	Long and cylindrical	Short and stout
Head	Small & seldom broader than body, same width as neck, covered with large scales	Larger & broader than body, wider than neck, triangular and covered with small scales
Upper jaw	Has 2 poison fangs and other teeth	Only poison fangs
Eyes	Round pupil	Vertical pupil
Fangs	Grooved, short & fixed	Canalized, long & mobile
Tail	Round	Tapered
Venom	Neurotoxic	Haemotoxic
Parity	Oviparous	Viviparous

SNAKE VENOM

The snake venom is the secretion of the racemose salivary glands that is a modification of the parotid salivary gland of other vertebrates, and is usually situated on each side of the head below and behind the eye, invested in a muscular sheath. It is provided with large alveoli in which the venom is stored before being conveyed by a duct to the base of the channelled or tubular fang through which it is ejected. Snake venom is poisonous only when it

is injected but is not absorbed by the stomach mucosa when ingested. The blood of animal bitten by snakes will be poisonous and lethal when injected into the human body.

The Vipers furnish examples of the most highly developed poison apparatus, although inferior to some in its toxic effects, the poison gland is very large and in intimate relation with the masseter or temporal muscle, consisting of two bands, the superior arising from behind the eye, the inferior extending from the gland to the mandible. When the snake bites, the jaws close up, causing the gland to be powerfully wrung, and the poison pressed out into the duct. From the anterior extremity of the gland the duct passes, below the eye and above the maxillary bone, where it makes a bend, to the basal orifice of the poison fang, which is ensheathed in a thick fold of mucous membrane, the vagina dentis. By means of the movable maxillary bone hinged to the prefrontal, and connected with the transverse bone which is pushed forward by muscles set in action by the opening of the mouth, the tubular fang is erected and the poison discharged through the distal orifice in which it terminates.

The Proteroglyphous Colubrids: The poison fangs are not tubular, but only channelled and open along the anterior surface. As the maxillary bone in these snakes is more or less elongate, and not or but slightly movable vertically, the poison duct runs above the latter, making a bend only at its anterior extremity, and the transverse bone has not the same action on the erection of the fangs. Otherwise the mechanism is the same.

The Opisthoglyphous Colubrids: The grooved teeth are situated at the posterior extremity of the maxilla and a small posterior portion of the upper labial or salivary gland is converted into a poison-secreting organ. This is distinguished by a light yellow colour, provided with a duct larger than any of those of the labial gland, and proceeding inward and downward to the base of the grooved fang. The duct is not in direct connection with the groove, but the two communicate through the mediation of the cavity enclosed by the folds of mucous membrane surrounding the tooth, and united in front.

The reserve or successional teeth, which are always present just behind or on the side of the functional fang of all venomous snakes, are in no way connected with the duct until called upon to replace a fang that has been lost.

When biting, a Viperid snake merely strikes, discharging the venom the moment the fangs penetrate the skin, and then immediately leaves go. A Proteroglyph or Opisthoglyph Colubrid, on the contrary, closes its jaws like a dog on the part bitten, often holding on firmly for a considerable time.

The poison, which is mostly a clear limpid fluid of a pale straw or amber colour, more rarely greenish, sometimes with a certain amount of suspended matter, is exhausted after several bites, and the glands have to recuperate. The venom retains its poisonous properties for several years in dried state. The Cobra venom is slightly viscous and when exposed to sun it becomes a little turbid. The venom of Russel's viper is usually white or yellow.

It must be added that the poison can be ejected otherwise than by a bite, as in the so-called **Spitting Snakes** of when irritated are in the habit of shooting poison from the mouth, at a distance of 4 to 8 feet. In all probability, the poison escapes from the sheath of mucous membrane surrounding the base of the fangs, and is mixed with ordinary saliva, the membranes of the mouth perhaps acting as lips, in which case the term "spitting" would not be incorrect. The spitting, which may take place three or four times in succession, has been observed to be preceded by some chewing movements of the jaws. If reaching the eye, the poisonous fluid causes severe inflammation of the cornea and conjunctiva, but no more serious results if washed away at once.

Snakes emerging out from hibernation after winter usually give fatal bite. Nocturnal bites are more serious than those occurring during the day. Less venom will be injected if the fangs are broken or if the snake had bitten a prey previously. The vipers give a strong and complete bite whereas in cobra bite the transfer of venom is not complete. Bite on limbs and adipose tissue is less dangerous than those on the trunk, face or scalp.

It is noteworthy that the size of the poison fangs is in no relation to the virulence of the venom. The comparatively innocent Indo-Malay Lachesis alluded to above have enormous fangs, whilst the smallest fangs are found in the most justly dreaded of all snakes, the Hydrophids.

Venoms of different species of poisonous snakes varies in the toxicity, composition and antigenic structure. It is basically a mixture of one or more of the toxic substances, toxalbumins and enzymes in varying proportions such as:

1. *Proteolytic enzymes or proteolysins*: It causes liberation of histamine from the damaged muscular endothelium leading to dissolution of walls of blood vessels with extravasation of blood in the tissue space, also causes digestion of tissue proteins and peptides and produces marked tissue destruction.
2. *Fibrin ferments or fibrinolysin thromboplastin* enhancing the coagulation process.
3. *Neurotoxins* (mainly in elapidae venom) producing curare like effect causing paralysis especially of respiratory center
4. *Cholinestrase* (mainly in elapidae venom) causing hydrolysis of acetylcholine to choline and acetic acid thus causes impairment of neuromuscular transmission.
5. *Haemolysin* (mainly in viper venom) causing widespread hemolysis in presence of lecithinase.
6. *Cytolysin* (mainly in viprine venom) causing lysis of cell structures of blood and tissues.
7. *Agglutinins* causing agglutination of red blood cells.
8. *Phosphatidases* produce haemolysis and toxic effects on heart and circulation with haemorrhage from lungs.
9. *Proteinases* have trypsin like action, causing tissue damage. It produces anticoagulant effect from destruction of fibrinogen. It also catalyses the conversion of prothrombin to thrombin thus producing coagulant effect.
10. *Phospholipase A, B, C, D* acts as catalyst in hydrolysis of lipids. By destroying phospholipids in nervous tissues, it alters neuromuscular conduction. It also helps in

- penetration of neurotoxin in to nervous tissue.
11. *Hyaluronidase* helps in rapid spreading of the venom from the local site of bite and thereby quick absorption. It is present in all snake venoms.
 12. *Ribonuclease* and *deoxyribonuclease* helps in rapid spreading of the venom from the local site of bite and thereby quick absorption. It is present in all venoms.
 13. *Ophi-oxidase* helps in autolysis and early onset of venom kile hyaluronidase.
 14. *Protease* causes dissolution of vascular wall and help in spreading of red blood cells and serum in the tissues.
 15. *Lecithinases* acts on the lipid layer of the cells causing increasing fragility and permeability leading to cell destruction.
 16. It also contains enzymes such as: Acetylcholine, 5-Hydroxytryptamine, 5-nucleotidase, endonucleases, transaminases, NAD nucleosidase, phosphodiesterase, ATP, acid phosphatase, phosphomonoesterase.

The Colubrine Venom

The effect of the poison of Proteroglyphous Colubrids (Hydrophids, Cobras, Bungarus, Elaps, Pseudechis, Notechis, Acanthophis) is mainly on the nervous system, respiratory paralysis being quickly produced by bringing the poison into contact with the central nervous mechanism which controls respiration; the pain and local swelling which follow a bite are not usually severe.

The colubrine venom is predominantly neurotoxic and is primarily toxic for respiratory and cardiac centres. It also has some toxic effects on the cardiovascular system. The cobra venom produces muscular paralysis involving firstly the muscles of the mouth, throat and lastly muscles of respiration, seat the action of venom being upon the motor nerve cells, the action resembling curare. It consists of: (i) Neurotoxin (ii) Cholinesterase (iii) Proteases (iv) Phosphotidases (v) Hyaluronidase (vi) Ribonuclease (vii) Thromboplastin (viii) Fibrinolysin (ix) Proteolysin (x) Cardiotoxin (xi) Phospholipase A.

Cobra venom produces convulsions and para-

lysis, while krait venom produces only muscular paralysis.

The Viperine Venom

The viperine venom is predominantly hemolytic and hemotoxic. It produces cytolysis of cells of vascular endothelium; causes lysis of red cells and other tissue cells and coagulation disorders. It consists of: (i) Proteases (ii) Hyaluronidase (iii) Haemorrhagin (iv) Haemolysin (v) Leucolysin (vi) Lecithinase (vii) Cytolysin (viii) Thromboplastin (ix) Phospholipase A (x) Proteinases.

Viper poison (*Vipera*, *Echis*, *Lachesis*, *Crotalus*) acts more on the vascular system, bringing about coagulation of the blood and clotting of the pulmonary arteries; its action on the nervous system is not great, no individual group of nerve-cells appears to be picked out, and the effect upon respiration is not so direct; the influence upon the circulation explains the great depression which is a symptom of Viperine poisoning. The pain of the wound is severe, and is speedily followed by swelling and discoloration.

There will be haemorrhage from the site of bite, associated with the necrosis of renal tubules, convulsions from intra-cerebral haemorrhage. Locally, there will be severe swelling of the bitten part, with oozing of blood and spreading cellulitis. Blood from such patients fails to clot even on addition of thrombin, because of the very low level of fibrinogen. The oozing of blood and serum is very much pronounced in case of viper bite than in cobra bite. Absence of oozing will indicate that the venom has not been injected into the wound.

A Cobra can inject about 40 times the fatal dose in a single bite. The amount of dried venom produced in a single bite is:

- In Viperine bite—150-200 mg
- In Cobra bite—200-250 mg
- In Krait bite—20 mg
- In Saw-scaled viper—4.5-5 mg

Fatal doses of various bites are: (i) Dried cobra venom—12-15 mg (ii) Dried viper venom—15-20 mg (iii) Dried krait venom—5-6 mg (iv) Dried Saw-scaled viper venom—8 mg.

Fatal Period→The death may occur instantaneously from neurogenic shock resulting from fright otherwise the fatal period is: (i) In colubrine bite—1/2-24 hours (ii) In viperine bite—2-4 days.

Signs and Symptoms of (Ophitoxaemia)

1. **Psychological trauma:** The most common and earliest symptom following snake bite (poisonous or non poisonous) is fright, particularly of rapid and unpleasant death. Owing to fright, a victim attempts 'flight' which unfortunately results in enhanced systemic absorption of venom. These emotional manifestations develop extremely rapidly (almost instantaneous) and may produce psychological shock and even death. Fear may cause also transient pallor, sweating and vomiting.
2. **Local manifestations:** With the possible exception of the psychological trauma of being bitten, local changes are the earliest manifestations of snake bites. Features are noted within 6-8 minutes but may have onset up to 30 minutes (i) Local burning pain with radiation and tenderness and the development of a small reddish wheal are the first to occur (ii) This is followed by oedema, swelling and appearance of bullae all of which can progress quite rapidly and extensively even involving the trunk (iii) Tingling and numbness over the tongue, mouth and scalp and paraesthesias around the wound occur mostly in viper bites (iv) Local bleeding including petechial and/or purpuric rash is also seen most commonly with viperine bites (v) The local area of bite may become devascularized with features of necrosis predisposing to onset of gangrenous changes (vi) Secondary infection including tetanus and gas gangrene may also result.
3. **Systemic manifestations:** Regional lymphadenopathy has been reported as an early and reliable sign of systemic poisoning. The time onset of poisoning is similar in different species. Cobra produces symptoms as early as 5 minutes or as late as 10 hours after the bite. Vipers take slightly longer - the mean duration of onset being 20 minutes. However, symptoms may be delayed for several hours. Sea snake

bites almost always produce myotoxic features within 2 hours so that they are reliably excluded if no symptoms are evident within this period. Other systemic manifestations depend upon the pathophysiological changes induced by the venom of that particular species. As mentioned previously, based on the predominant constituents of venom of a particular species, snakes were loosely classified as neurotoxic (notably cobras and kraits), hemorrhagic (vipers) and myotoxic (sea snakes). However it is now well recognized that such a strict categorization is not valid as each species can result in any kind of manifestations.

Features of neurotoxicity: Neurotoxic features are a result of selective d-tubocurarine like neuro-muscular blockade which results in flaccid paralysis of muscles. Cobra venom is however 15-40 times more potent than tubocurarine (i) Ptosis is the earliest neuromuscular manifestation followed closely by ophthalmoplegia (ii) Paralysis then progresses to involve muscles of palate, jaw, tongue, larynx, neck and muscles of deglutition-but not strictly in that order (iii) Generally muscles innervated by cranial nerves are involved earlier. However, pupils are reactive to light till terminal stages (iv) Muscles of chest are involved relatively late with diaphragm being the most resistant. This accounts for the respiratory paralysis, which is often terminal (v) Reflex activity is generally not affected in ophitoxaemia and deep tendon jerks are preserved till late stages (vi) Onset of coma is variable, however several cases of cobra bite progress to coma within 2 hours of bite (vii) Symptoms that portend paralysis include repeated vomiting, blurred vision, paraesthesiae around the mouth, hyperacusis, headache, dizziness, vertigo and signs of autonomic hyperactivity.

Features of cardiotoxicity: Cardiotoxicity occurs in about 25% viperine bites (i) There are fluctuations in rate, rhythm and blood pressure in the form of tachycardia, hypotension and ECG changes (ii) In addition, sudden cardiac standstill may also occur owing to hyperkalemic arrest (iii) Non dyselectrolytemic

acute myocardial infarction has also been reported.

Tetanic contraction of heart following a large dose of cobra venom has been documented. There is a single case report of non-bacterial thrombotic endocarditis following viper bite. Myalgic features are the most common presentation of bites by sea snakes. Muscle necrosis may also result in myoglobinuria.

Hemostatic Defects: Snake venoms cause haemostatic defects by a number of different mechanisms. Some cause activation of intravascular coagulation and result in consumption coagulopathy. Notable in this group is *Daboia russelli* which has procoagulant activating factors V and X. Certain other venoms cause defibrinogenation by activating endogenous fibrinolytic system. Besides direct effects on the coagulation cascade, venoms also can cause qualitative and quantitative defects in platelet function. In India and Sri Lanka, Russell's viper envenomation is often associated with massive intravascular haemolysis. Haematological changes, both local as well as systemic are some of the most common features of snake bite poisoning (i) Bleeding may occur from multiple sites including gums, GIT (haematemesis and melaena), urinary tract, injection sites and even as multiple petechiae and purpurae (ii) Even subarachnoid haemorrhages, subdural haemor-

rhages and extradural hematoma have also been reported (iii) Almost every species of snake can cause renal failure. It is fairly common following Russell's viper bite and is a major cause of death. In a series of 40 viper bites, renal failure was documented in about a third. The extent of renal abnormality in them correlated well with the degree of coagulation defect; however in a majority renal defects persisted for several days after the coagulation abnormalities normalised: suggesting that multiple factors are involved in venom induced ARF.

Rarer Systemic Manifestations: These include (i) Hypopituitarism (ii) Bilateral thalamic haematoma (iii) Hysterical paralysis. (Table 38.3)

- 4. Delayed manifestations of snake bite:** The delayed onset of signs and symptoms of snake bite is rare but definitely occurs in the form of bleeding manifestations including brain haemorrhages up to one week after the bite. Some patients develop compartment syndromes several weeks after the bite. The possible explanation for delayed bleeding is that local blebs constitute a venom depot, which is suddenly released into the blood stream, especially when the wound is handled surgically.

Laboratory Diagnosis in Ophitoxaemia

The laboratory serves rather poorly in the diagnosis of snakebite but laboratory tests are useful for

Table 38.3: Comparative features of clinical manifestations of Colubrine and Viperine bites

Features	Colubrine bite	Viperine bite
Onset of signs and symptoms	Within 10-20 min	Within second to 10 min.
Development of gangrene	Early, wet type	Slower onset, dry type
Feeling of intoxication	Marked	Not marked
Gait	Staggering	Not so but features of general paralysis
Speech and power of deglutition	Lost	Not so
Involvement of tongue and larynx	Are paralysed with trickling of saliva from mouth	Not affected, salivation is absent
Pupils	Normal	Dilated, not reacting to light
Respiration	Slow, weak and laboured	Quick and laboured
Blood coagulability	Not affected	Completely deranged
Haemorrhagic manifestations	Not present	Most important feature
Death	From respiratory paralysis	When rapid, due to pulmonary thrombosis; when slow, from toxic action of venom on heart and blood

monitoring, prognosticating victims of Ophitoxaemia, as well as determining stages of intervention

- i. *Enzyme linked immunoabsorbent assay (ELISA)*: ELISA studies which are now available to identify the species involved, based on antigens in the venom. These tests are expensive and not freely available-hence of limited value; except for epidemiological study.
- ii. Blood changes include anaemia, leucocytosis and thrombocytopenia
- iii. Peripheral smear may show evidence of haemolysis, particularly in viperine bites.
- iv. Deranged coagulant activity manifested by prolonged clotting time and prothrombin time may also be evident. The quality of clot formed may be a better indicator of coagulation capability than the actual time required for formation, since clot lysis has been observed in several patients who had normal clotting time. Hypofibrinogenemia may also be evident.
- v. Among the metabolic changes, hyperkalaemia and hypoxemia with respiratory acidosis, especially with neuromyolysis may be present.
- vi. Urine examination could reveal haematuria, proteinuria, haemoglobinuria or myoglobinuria.
- vii. In cases of acute renal failure, all features of azotemia are also present.
- viii. CSF haemorrhage has been documented in a minority of victims.
- ix. ECG changes are generally non-specific and include alterations in rhythm (predominantly bradycardia) and atrioventricular block with ST segment elevation or depression. In addition, cases who develop hyperkalaemia manifest typical changes of this dyselectrolytaemia.
- x. Serum cholesterol levels correlates negatively with severity of envenomation.
- xi. Recently EEG changes have been noted in up to 96% of patients bitten by snakes; starting within hours of the bite. Interestingly none of them showed any clinical features suggestive of encephalopathy. These abnormal EEG patterns were picked up mainly in the temporal lobes.

Management of Ophitoxaemia

First aid for snakebite

1. The bite should not be washed as traces of venom that are left on the skin can be used to identify the snake, and therefore the type of antivenom that should be used if required.
2. *Incising and sucking*: Venom is injected deeply so there is no benefit in cutting or sucking the bite. It was formerly believed and therefore advocated that incision over the bite drains out venom. However, it has now been established from animal experiments that systemic venom absorption starts almost instantly; this form of 'therapy' is therefore being questioned. Some experts suggest that longitudinal incisions within fifteen minutes of the bite may be beneficial.
3. The most effective first aid for snakebite is the *pressure-immobilization technique*. The principle is to minimise the movement of the venom around the body until the victim is in a hospital by applying a firm bandage (or suitable alternative) to the bitten area and limb, and to immobilise the victim. When applied properly, this method can trap the venom in the bitten area for many hours. The victim might not suffer any effects of the venom until the compression is released, which is done in hospital where antivenom can be administered if required. The tourniquet should be tight enough to occlude the lymphatics, but not venous drainage; though some also prefer to occlude the veins. Enough space to allow one finger between the limb and bandage is most appropriate. Should the limb become edematous, the tourniquet should be advanced proximally. Tourniquets should never be left in place too long for fear of distal avascular necrosis.
4. In the case of Viper bites, tourniquets are also a risk. The Viper's venom contains pro-coagulant enzymes which cause the blood to clot. In the small space below the tourniquet the venom has a greater chance of causing a clot. When the tourniquet is released the clot will rapidly enter the body and can cause embolism and death.

5. The majority of the Indian venomous snakes have venom that contains toxins that do serious local damage at the bite site. This is true of all vipers and the Cobras. This toxin breaks down tissue and destroys it. Confining this toxin in a smaller area, by use of compression techniques creates a greater risk of serious local damage.
6. Most physicians are in disagreement with regard to nature, duration and even necessity of first aid. Russell advises minimal wastage of time with first-aid measures which often end up doing more harm than good. In a recent report from Brazil, two cases were reported to have increased local envenoming subsequent to a tourniquet.
7. *Cleansing the wound:* Reid has advised that the wound site be minimally handled. Most authors recommend saline cleaning and sterile dressing. Some however advise that the wound be left open.
8. There is disagreement over the use of drugs as part of first-aid care. It has been suggested that NSAIDs particularly aspirin may be beneficial to relieve local pain. Russell however dissuades use of analgesic and in particular aspirin for fear of precipitating bleeding. In Reid's experience, pain relief with placebo was as effective as NSAID. Codeine may be useful in some cases. Similarly, there are proponents as well as opponents for use of sedatives

Specific Therapy—Antivenins

Antivenins are prepared by immunizing horses with venom from poisonous snakes and extracting the serum and purifying it. Antivenoms or antivenins may be species specific (monovalent) or effective against several species (polyvalent). Monovalent antivenom is ideal, but the cost and non-availability, besides the difficulty of accurately identifying the offending species—makes its use less common.

Indications for use: There are specific indications for use of antivenom. Not every bite, even if by poisonous species merits its use. This caution against the empirical use of antivenom is due to the risk of hypersensitivity reactions. Therefore,

antivenin is indicated only if:

- I. Serious manifestations of envenomation are evident viz coma, neurotoxicity, hypotension, shock, bleeding, DIC, acute renal failure, rhabdomyolysis and ECG changes.
- II. In the absence of these systemic manifestations, swelling involving more than half the affected limb, extensive bruising or blistering and progression of the local lesions within 30-60 minutes are other indications.

Dose: Despite widespread use of antivenom, there are virtually no clinical trials to determine the ideal dose. Conventionally: (i) 50 ml is infused for mild manifestations like local swelling with or without lymphadenopathy, purpura or ecchymosis (ii) Moderate envenomation defined by presence of coagulation defects or bradycardia or mild systemic manifestations, merits the use of 100 ml (iii) 150 ml is infused in severe cases, which includes rapid progression of systemic features, Disseminated Intravascular Coagulation, encephalopathy and paralysis.

Administration: The freeze-dried powder is reconstituted with 10 ml of distilled water or saline or dextrose. A test dose is administered on one forearm with 0.02 ml of 1:10 solution intradermally. Similar volume of saline in the other forearm serves as control. Appearance of erythema or wheal greater than 10 mm within 30 min is taken as a positive test. In this event, desensitization is advised starting with 0.01 ml of 1:100 solution and increasing concentration gradually at intervals of 15 minutes till 1.0 ml s.c. can be given by 2 hours. Infusion is started at 20 ml/kg per hour initially and slowed down later.

Antivenom is administered by the intravenous route and not at local bitten area. Besides this, systemic administration of antivenom has been shown to be effective at the local site as well. Therefore, most experts do not advise local injection of antivenin. Efficacy of intramuscular administration of antivenom followed by standard hospital management has also been evaluated and a definite reduction in the number of patients with systemic envenomation, complications and mortality from Russell's viper toxemia has been noted.

Timing: There is no consensus as to the outer limit of time of administration of antivenom. Best effects are observed within four hours of bite. It has been noted to be effective in symptomatic patients even when administered up to 48 hours after bite. Reports suggest that antivenom is efficacious even 6-7 days after the bite. It is obvious that when indicated, antivenom must be administered as early as possible and data showing efficacy with delayed administration is based on use in settings where patients present late.

Response: Response to infusion of antivenom is often dramatic with comatose patients sitting up and talking coherently within minutes of administration. Normalization of blood pressure is another early response. Within 15 to 30 minutes, bleeding stops though coagulation disturbances may take up to 6 hours to normalize. Neurotoxicity improves from the first 30 minutes but may require 24 to 48 hours for full recovery. If response to antivenom is not satisfactory, use of additional doses is advocated. However, no studies establishing an upper limit are available. Infusion may be discontinued when satisfactory clinical improvement occurs even if recommended dose has not been completed. In experimental settings, normalization of clotting time has been taken as end-point for therapy.

Side effects: Hypersensitivity reactions including the full range of anaphylactic reactions may occur in 3-4% of cases, usually within 10 to 180 minutes after starting infusion. These usually respond to conventional management including adrenaline, anti-histamines and corticosteroids.

Availability: Several antivenom preparations are available internationally. In India, polyvalent antivenom prepared by C.R.I., Kasauli is effective against the 4 most common species. Antivenom produced at the Haffkine Corporation, Parel includes more species as well. This is about 10 times as expensive as the former. The WHO has designated the Liverpool School of Tropical Medicine as the international collaborating centre for antivenom production and/or testing.

Supportive Therapy

1. In cases of bleeding, replacement with fresh whole blood is ideal. Fresh frozen plasma and fibrinogen are not recommended.
2. Volume expanders including plasma and blood are recommended in shock, but not crystalloids.
3. Persistent shock may require inotrope support under Central Venous Pressure monitoring.
4. Early mechanical ventilation is advocated in respiratory failure though dramatic responses have also been observed with edrophonium followed by neostigmine.
5. Cases of acute renal failure generally respond to conservative management.
6. Occasionally peritoneal dialysis may be necessary.
7. In cases of Disseminated Intravascular Coagulation, use of heparin should be weighed against risk of bleeding hence caution is to be taken..
8. Routine antibiotic therapy is not necessary though most Indian authors recommend use of broad-spectrum antibiotics. Chloramphenicol has been claimed to be useful as a post bite antibiotic even when used orally since it is active against most of the aerobic and anaerobic bacteria present in the mouths of snakes. Alternatives include cotrimoxazole, fluoroquinolones with or without metronidazole or clindamycin for anaerobic cover. A study of the organisms isolated from the mouth of the Malayan pit vipers suggests that crystalline penicillin with gentamicin would also be appropriate antibiotic cover following snakebite.
9. Recent studies have reported the beneficial effects of intravenous immunoglobulin (IVIg) in ophitoxaemia. There are suggestions that its administration may improve coagulopathy, though its effect on neurotoxicity is questionable. A pilot study indicates that IVIg with antivenom eliminates the need to repeat antivenom for envenomations associated with coagulopathy.

10. A compound extracted from the Indian medicinal plant *Hemidesmus indicus* R (2-hydroxy-4 methoxy benzoic acid) has been noted to have potent anti-inflammatory, antipyretic and anti-oxidant properties, particularly against Russell's viper venom. These experiments suggest that chemical antagonists from herbs hold promise in the management of ophitoxaemia; particularly when used in the presence of antivenom.
11. Injection of tetanus toxoid is necessary.
12. Early surgical debridement is generally beneficial though fasciotomy is usually more harmful than useful.
13. There is no role for steroid therapy in acute snakebite. Although it delays the appearance of necrosis, it does not lessen the severity of outcome.

Autopsy Findings

Findings in a colubrine snakebite: (i) The puncture marks of fangs are usually 1.2cm (½") deep and are well visible by hand lens with oozing of serum, blisters and necrosis at the site of bite (ii) Blood remains fluid showing features of hemolysis (iii) Froth in mouth and nostrils (iv) Features of asphyxia are prominently seen (v) Microscopically, the nervous tissue will show changes in Nissl's granules, fragmentation of reticulum of the nerve cells, opacity, fragmentation and swelling of the nuclei. Cells of medulla show acute granular degeneration.

Findings in a viperine snakebite: (i) Puncture marks are usually 2.5cm (1") deep with swelling, extravasation of blood, inflammation, cellulitis and discoloration in and around it (ii) Blood-tinged serum oozes out of the punctured marks (iii) Blood is fluid with purplish tinge (iv) Evidence of haemorrhages from G.I.T, respiratory and urinary tract is there (v) Petechial haemorrhages in pleura, pericardium and substance of kidney, liver, lungs and brain with congestion of these organs (vi) Haemorrhage in the endocardium of left ventricle, interventricular septum and papillary muscles (vii) Regional lymphadenopathy is there.

Medicolegal Aspects

(i) Always occurs accidentally (ii) Suicide is unknown except by Cleopatra (iii) Homicides, rarely allegations of snake being thrown into the enemy's place but cannot be proved without circumstantial evidences (iv) Cattle poisoning does occur by placing snake and banana in an earthen pot and irritating snake by firing it. The cobra bites the banana injecting the venom into it. The fruit is taken out and smeared into a rag that will be injected into rectum of the animal by bamboo stick.

SCORPIONS

Scorpions are poisonous insects that have a fleshy segmented body with four pairs of legs, a pair of claws and a tail. The posterior segment is long and bulbous and contains the venom gland and a hollow sting at the end of the tail in its last joint. The sting communicates with the venom gland by means of a duct. The stings inject venom in the injury produced by it. The adult scorpion are 2-20 cm long and big, black coloured are more dangerous than the small, brown ones. The scorpion's bites are always accidental as they live in cracks and holes in the houses.

Scorpion Venom

Scorpion venom is a clear and colourless toxalbumin that is more toxic than the snake venom. The small quantity of venom is injected. The scorpion venom consists of a neurotoxin (acts on the preganglionic and postganglionic nerve terminals and muscle endplates), haemolysins, agglutinins, haemorrhagins, proteinases, phospholipase A, leucocytolysin, coagulins, cholesterolin and lecithin.

Signs and symptoms: (i) Site of bite is having a red wheal with a hole in the centre and severe burning pain radiates from the site. The area is swollen, red and oedematous (ii) Increased temperature with chills and headache (iii) There is feeling of giddiness and fainting (iv) Excessive sweating and salivation (v) Muscular cramps and convulsions followed by unconsciousness (vi) Paresis and

muscular weakness persist for a week (vii) Death occurs in children from pulmonary oedema and respiratory depression and it is rare in adults.

Treatment

1. Application of tourniquet or pressure bandage above the site of bite and incising and washing the bitten area with weak solution of potassium permanganate, borax or ammonia. The tourniquet should be loosened for 1-2 minutes after every 10-15 minutes.
2. Local infiltration of 5% cocaine, or lignocaine solution in and around the bite relieves pain
3. To treat shock, 5% of 500 ml of dextrose saline should be give I.V. along with glucocorticoids i.m.
4. Calcium gluconate 10ml of 10% solution should be given intravenously to combat muscular cramps.
5. To prevent pulmonary oedema, atropine sulphate should be administered.

BEES, WASPS AND ANTS

1. Produce a painful local reaction, which recedes with time.
2. An allergic phenomenon produced by them may kill the patient because of prior sensitization.
3. The number of stings may be so great or the patient is so young as to kill with primary toxicity.

Signs and symptoms: (i) Nausea, vomiting (ii) Thoracic oppression (iii) Cardiac irregularities (iv) Pulmonary oedema (v) Nephrosis (vi) Cyanosis, coma, convulsions and death.

The fatalities may result a short while after the infliction of stings

Fire Ants

This insect *Solenopsis saevissima richteri* causes an immediate and very painful local reaction in the form of vesicles and swelling. The insect inserts its stinger number of times and as a result, number of sensitization occurs to its bite. The systemic reaction can also be produced in sensitized persons that can be febrile and allergic.

INORGANIC IRRITANT POISONS

PHOSPHORUS

Phosphorus is a non-metallic, hepatotoxic and protoplasmic irritant type of poison. It exists in two forms; red phosphorus and white phosphorus. Of the two, red is not poisonous but white phosphorus is a deadly one.

White/yellow phosphorus: White phosphorus occurs in the form of white, waxy, translucent and pliable soft sticks. It is insoluble in water, somewhat soluble in alcohol and ether and readily soluble in carbon disulphide. On exposure to air, it slowly oxidizes or 'phosphorescences'—Phosphorescence is the condition of white phosphorus, when it emits white fumes of phosphorus trioxide, which is luminous in the dark giving strong garlic odour. At 34°C, it ignites in the air emitting greenish-white flame, hence it is to be preserved under water or kerosene oil. It should not be handled even by wet finger as even the body heat can cause ignition.

Red phosphorus: Red phosphorus is prepared by heating white phosphorus at 240°-250°C in an atmosphere with nitrogen or carbon dioxide gases. It occurs ordinarily as violet red solid mass that is odourless, tasteless, is insoluble in carbon disulphide, and does not luminescence in the dark (Table 38.5).

Uses of Yellow Phosphorus

1. It is used in the preparation of vermin pastes that contain arsenic, fluor, oil, sugar, some colouring agents mixed with 1-4% conc. of yellow phosphorus
2. In manufacture of gunpowder, fireworks, and incendiary ammunitions and for creating smoke screens during warfare.
3. In various chemical and fertilizer industries.

Medicolegal Aspects

1. Phosphorus is not commonly used for homicidal purposes due to its characteristic garlicky odour, taste, luminosity in the dark makes for its easy detection. But it acts as a god homicidal poison as phosphorus containing rat poison mixed with strong tea is not easily detectable.

Table 38.5: Differences between red and yellow phosphorus

Properties	Red phosphorus	Yellow phosphorus
Colour	Violet-red or reddish brown	Originally white, turns to yellow on exposure
Appearance	Amorphous, solid mass	Waxy, translucent and soft pliable sticks
Taste and odour	Tasteless, odourless	Garlicky taste and odour
Luminosity in dark	Non-luminous	Luminous
Solubility in carbon disulphide	Soluble	Insoluble
Exposure to air	Does not phosphorescences, non fuming	Phosphorescences
Toxicity	Non-toxic	Highly toxic
Ignition in air	Not so	Possible

Besides, there is delay in onset of clinical features and post mortem findings simulate that caused by hepatotoxic drugs and diseases.

2. Vermin pastes, rat killers containing phosphorus are commonly used for suicidal purposes.
3. Accidental poisoning can occur in children as they may ingest rat-poison. The poisoning can also occur from fragments of projectiles containing yellow phosphorus, inhaling hydrogen phosphide gas produced in ship holds from cargo and inhaling the gas evolved from ferro-silicon used in steel industry.
4. Arson using phosphorus is done in villages when moist cow-dung and yellow phosphorus are mixed together and thrown over thatched cottage-roof that when dries in sun catches fire from phosphorescent phosphorus

Fatal dose: Adults—60-120 mg

Children—10-25 mg

Fatal period: Upto 24 hours (4-10 hours)

Mechanism of Action

(i) Locally it acts as an irritant (ii) On absorption, it remains in the blood in elemental form for a day or two, then gets oxidized into hypophosphorus and phosphorus acid when it acts as hepatotoxic, protoplasmic poison disturbing the normal cell metabolism by affecting cellular oxidation (iii) It produces widespread fatty infiltration with degeneration of different organs especially the liver and cells of cerebral cortex (iv) It also causes tissue destruction by interfering with carbohydrate, fat and protein metabolism especially by deposition of fat in the liver at the cost of glycogen (v) Chronic absorption of phosphorus leads to bone formation in the epiphyseal cartilage and in the bone marrow

and Haversian canal thus impairing blood circulation in bones. This leads to necrosis and sequestration of bones with or without spontaneous fractures.

Acute Phosphorus Poisoning

Signs and Symptoms

The signs and symptoms of acute phosphorus poisoning usually appear within a few minutes and sometimes may be delayed for up to 6 hours.

First stage: (i) Garlic like taste in mouth and smell in breath that is luminous in the dark (ii) Sense of warmth with burning pain in mouth and throat radiating down to oesophagus, stomach and finally all over the abdomen (iii) Intense uncontrollable and non-querchable thirst with frequent gastric eructations and patient drinks lots of water trying to quench his thirst (iv) Copious vomiting occurs that is frequent, profuse and persisting; has garlicky odour, luminous in the dark, bile stained and even blood tinged in later stages (v) Painful throat as a result of repeated retching and vomiting (vi) Diarrhoea though uncommon is in the form of dark, offensive, and phosphorescent motions usually preceded by colicky pain (vii) Abdomen is tender and distended with epigastric discomfort (viii) Hypoglycemia may result (ix) Cardiac and respiratory depression, cold clammy skin, hypothermia occurs (x) Delirium is followed by convulsion, collapse, coma and death occurring within 12-24 hours of ingestion of poison (xi) Patient usually does not die in 1st stage but he enters 2nd stage by 36-48 hours when signs and symptoms reduce in intensity and vitals improve

Second stage: This is a stage of apparent improvement of signs and symptoms that may last for 2-4 days. There may be little pain in the abdomen with occasional vomiting and purging, malaise and headache.

Third stage: (i) Vomiting and diarrhoea reappear and is more intense and distressing and contains mucous and blood (ii) Abdomen distended with severe abdominal pain (iii) Jaundice sets in and deepens quickly (iv) Liver is enlarged, soft and tender (v) Skin becomes cold, respiration laboured and pulse is feeble (vi) Haemorrhages in the form of epistaxis, haematemesis, haematuria, melena and menorrhagia are commonly seen (vii) Petechial haemorrhages over the mucous membranes (viii) Blood urea is usually raised (ix) Urine is scanty, high coloured, contains blood, ammonia, casts, albumen, free fat globules, excess of lactic acid, bile and some amino acids like leucine, tyrosine and cysteine (x) Headache, restlessness, insomnia may be seen (xi) Tinnitus and vertigo are usually present (xii) Impaired vision, cramps, muscle twitchings and even paralysis, delirium and frequent priapism may also develop (xiii) Features of increasing hepatic and renal insufficiency followed by hypoglycemia, quick weak and irregular pulse, fall of BP, pulmonary oedema, dyspnoea, cyanosis, oliguria, even anuria, subnormal temperature progressing to death (xiv) Yellow phosphorus produces usually second or third degree burns that are slowly healing and are surrounded by blisters on the skin.

Treatment

1. Stomach wash using 0.5% potassium permanganate solution that oxidizes phosphorus into harmless phosphoric acid and phosphates itself changing into manganese dioxide.
2. Stomach wash using 0.1% copper sulphate solution or 250mg of copper sulphate in a glass of water may be given by mouth every 5 minutes until free emesis starts. Copper sulphate also acts as an antidote as it is reduced by phosphorus and is precipitated as copper phosphide forming a coating over

the phosphorus particles thus rendering them inert.

3. Hydrogen peroxide 2% solution can also be used as gastric lavage fluid.
4. Activated charcoal also helps in adsorbing the poison.
5. Oils, fats, milk etc. should not be given to the patient as they help in dissolving phosphorus and promotes its absorption.
6. Liquid paraffin retards absorption of phosphorus and hastens its elimination but castor oil should not be used.
7. Bowels should be thoroughly washed using potassium permanganates and purgatives especially magnesium and sodium sulphate.
8. Glucose and alkaline drinks by mouth in plenty to protect liver.
9. High carbohydrate and low protein and fat diet should be given.
10. To combat shock and dehydration plenty of glucose saline (500 ml of 5%) fluids and 10 ml of 10% calcium gluconate should be administered intravenously.
11. Symptomatic treatment such as non-narcotic analgesics for pain, multivitamins especially injections of vitamin K should be given.
12. Locally, skin can be irrigated with 1% copper sulphate solution in water for at least 10 minutes.

Autopsy Findings

The post mortem findings will depend on the period of survival:

Death within first 24 hours: (i) Slight icteric tinge of the skin (ii) Mucous membranes of pharynx, oesophagus, stomach and intestines shows signs of inflammation, redness, ulceration and corrosion (iii) Stomach is contracted and mucosa is inflamed, softened, yellowish green with garlicky odour that luminesce in dark

Death after 24-48 hours: (i) Skin will look jaundiced (ii) Petechial haemorrhages under the skin and over serous and submucous surfaces of the lungs, brain, leptomeninges, uterus and kidney (iii) Garlicky odour from the body (iv) Mucosa of stomach and intestine is yellow or yellowish green

with evidence of inflammation, softening, corrosion and haemorrhagic. This is also luminous in the dark (v) Liver is usually enlarged though it may be normal or contracted also. It is lemon yellow with doughy, soft and greasy consistency. A moderate pressure will allow the pressing finger to sink deep down in to the liver substance, which will be easily friable. Small haemorrhagic spots are seen on the glisson's capsule and in liver substance. Evidence of fatty degeneration, cellular necrosis, fibrosis, cellular infiltration is there. Evidence of accumulation of fat in Kupffer's cell is the earliest manifestation of necrobiosis the liver undergoes in phosphorus poisoning (Table 38.6) (vi) Heart is soft, flabby and dilated with evidence of fatty degeneration and subendocardial haemorrhages (vii) Kidneys may be filled with cellular debris, fatty casts, albumen etc. and are enlarged, soft, greasy, yellow with petechial haemorrhages and fatty degeneration.

Although phosphorus is readily oxidized in air, it may be detected in unoxidised form in dead body several days after death, even when the body is in an advanced state of decomposition. This may be explained by the fact that reducing gases that are formed during decomposition protects phosphorus from oxidation. Phosphorus occurs in combination with food articles, in tissue and body fluids mainly as phosphates. Hence, detection of phosphate has little medicolegal importance; but when detected in elementary form in the body, it is taken to be sufficient to have produced phosphorus poisoning as it does not occur in free form in nature.

Chronic Phosphorus Poisoning

Chronic phosphorus poisoning usually occurs due to inhalation of fumes of white phosphorus

amongst those employed in manufacture of fire works and some ammunitions where phosphorus is being used, inhalation of phosphorated hydrogen in preparation of acetylene gas from carbide and also from escape of the gas from ferrosilicon results in poisoning. It is also seen in workers in matchbox and sticks factory.

Signs and symptoms: The symptoms and signs usually develop after working for weeks, months or even years in these factories: (i) Nausea, vomiting, garlicky smelling eructation's and purging (ii) Generalized wasting, weakness, lassitude, emaciation and joint pain (iii) Abdominal discomfort and pain (iv) Anaemia, jaundice, shallow complexion (v) Tracheitis and bronchitis (vi)

Phossy jaw—It is osteomyelitis and periostitis of jaw due to chronic phosphorus poisoning associated with necrosis of the alveolar part of jawbone along with the sloughing of gums and loosening or falling of teeth. At first, there is history of tooth-ache, swelling of jaw, loosening of teeth, sloughing of gums, necrosis and sequestration of mandible with multiple foul-smelling pus discharging sinuses. Phosphorus mainly attacks the lower jaw through carious or decayed teeth or through interspaced between the missing teeth adjoining the raw surface.

Treatment

1. Prophylactic measures such as cleanliness and ventilation of factories; oral hygiene of workers regularly that is washing mouths with soda bicarb solution and extraction or filling up of carious tooth should be followed.
2. If necrosis of mandible occurs surgical intervention is undertaken.

Table 38.6: Difference in liver in yellow phosphorus poisoning and yellow atrophy of liver

Morphology of liver	Yellow phosphorus poisoning	Yellow atrophy of liver
Size	Usually enlarged, normal or even contracted	Contracted with wrinkling of glisson's capsule
Consistency	Doughy and soft	Leathery
Colour	Uniformly yellow	Dirty yellow
Greasiness	Greasy to touch & fingers dips easily	Soft & greasy to touch
Microscopic features of liver substance	Extensive fatty infiltration with degeneration, cellular necrosis and haemorrhages	Extensive cellular necrosis

IODINE

Iodine occurs in the form of bluish black, soft, scaly crystals that have a metallic sheen and unpleasant taste. It is freely soluble in water, the solubility increasing in presence of iodide. Idiosyncrasy is more common than bromide. It is very commonly used household antiseptic and potassium iodide is largely used as medicine.

Mechanism of Action

It acts as corrosive and coagulates cellular protein causing necrosis. The iodine vapours are irritant to respiratory passages. Iodine is converted to iodide in the body. The normal iodide content of the blood is 2-5 mg/100 ml. The prognosis is good if the patient survives for 48 hours after ingestion of the poison.

Fatal dose→2-4 gm (in solid form) and Two drachms of tincture iodine.

Signs and symptoms: (i) Burning pain in mouth radiates to throat, oesophagus, stomach and abdomen (ii) Increased thirst, nausea, vomiting, salivation and purging (iii) The lips and angles of mouth are stained brown; the vomitus and faeces are stained dark brown or blue mixed with blood smelling of iodine (iv) Urine is scanty or suppressed and reddish brown containing albumen; painful micturition (v) Headache, muscle cramps, giddiness (vi) Cold clammy skin and fall of BP (vii) Respiratory depression, quick weak pulse (viii) Delirium and collapse (ix) Consciousness is retained till the end (x) Iodides may cause enlargement of salivary glands and lymph nodes (xi) Application of iodine to skin causes weeping eruptions, with raised temperature in sensitive individuals (xii) Injection of compound of iodine may cause sudden fatal collapse due to idiosyncrasy (xiii) Inhalation of iodine vapours results in glottic oedema and death from asphyxia.

Chronic Iodine Poisoning (Iodism)

It results from chronic use of potassium iodide in large doses as medicine. The clinical features are in the form of frontal headache, running nose, sneezing, watering from eyes, conjunctivitis, bronchitis, parotitis, excessive salivation, nausea, vom-

iting and diarrhoea associated with oedema of face and eyelids that clears up once the medication is discontinued.

Treatment

(i) Gastric lavage using 5% sodium thiosulphate solution or water containing soluble starch and albumen (ii) Emetics should be given (iii) Demulcents as barley water, egg albumen, milk, alkaline drinks and castor oils should be given (iv) Shock and dehydration to be treated with 5% dextrose saline drip i.v with mephentin and other analeptics to maintain BP (v) Glucocorticoids and antihistaminics in repeated doses help in allaying oedema. Tracheostomy to be performed to relieve glottic oedema (vi) 5% Sodium thiosulphate 100-150mL orally helps to reduce free iodine to non-toxic iodides (vii) fluid and electrolyte balance should be maintained (viii) in chronic poisoning discontinue the drug and give large doses of Sodium bicarbonate or sodium chloride with fluids to hasten recovery.

Autopsy Findings

(i) The face and eyes are swollen and there is evidence of glottic oedema (ii) Mucosa of gastrointestinal tract is stained yellow or brown, is congested, inflamed and excoriated at places. Also the stomach contents are turned blue due to inter-reaction of iodine with starchy food (iii) Lungs are congested and oedematous (iv) Heart and liver shows fatty change (v) Kidneys may show glomerular and tubular necrosis (vi) Brain is usually congested and oedematous

Medicolegal Aspects

1. Accidental poisoning is common in children from accidental drinking of tincture iodine, betadaine etc. or when an alcoholic solution when excessively used for external application.
2. Suicidal and homicidal poisoning is rare.

CHLORINE

Chlorine is a greenish yellow gas having an unpleasant irritating odour even when diluted. It is largely used in industry as bleaching agent and disinfectant

Fatal dose→Exposure to air containing 1/1000 chlorine for 5 minutes is fatal by causing acute pulmonary oedema.

Fatal period→Inhalation of pure chlorine gas results in death within 12-24 hours.

Signs and Symptoms

(i) Inhalation causes intense irritation of respiratory passages, throat and eyes causing dry harsh painful violent cough with yellowish expectoration, intense watering eyes and coryza (ii) Headache, nausea and vomiting, abdominal pain occur (iii) Tachypnoea, extreme dyspnoea, palpitation and pyrexia results (iv) In extreme cases, glottic oedema, asphyxia, intense cyanosis, tachycardia occurs (v) Unconsciousness soon supervene and death results from acute respiratory or cardiac failure (vi) Chronic exposure to chlorine vapours shows features of anemia, cachexia, dyspepsia, chronic bronchitis and emphysema.

Treatment

1. Patient to be removed to fresh air immediately
2. Oxygen inhalation, artificial respiration and suction of frothy fluid from air passages
3. 5% dextrose saline drip with sodium bicarbonate to combat dehydration and shock
4. Treatment of pulmonary oedema, acidosis etc. should be ensued
5. As a prophylactic measure the respirator masks soaked in sodium bicarbonate solution and sodium hyposulphite and goggles for eyes should be used

Autopsy Findings

(i) The respiratory tract mucosa shows intense congestion (ii) Lungs are congested, oedematous exuding frothy, tenacious blood-stained fluid and showing areas of patchy haemorrhages and collapse (iii) Stomach and intestines are congested (iv) Heart is also enlarged (v) Odour of chlorine from the ventricles of brain that is congested (vi) All organs are congested.

Medicolegal Aspects

Poisoning is usually accidental by chlorine and its compounds especially bleaching powder in

industry. Use of chlorine gas in 1st World war caused numerous casualties

BROMINE

Bromine is a dark reddish brown liquid that volatilises at ordinary temperatures giving out irritating fumes of unpleasant odour. Compounds of bromine displace chlorides from the plasma and cells; thereby will cause depression of central nervous system hence are not commonly used in medicine as sedatives. They are also used as anticonvulsant agents. Children may get more readily affected from bromide poisoning than adults. The poison gets rapidly eliminated in the urine, saliva, sweat and milk.

Fatal dose→Uncertain; 20-30gms of sodium or potassium bromide can cause alarming symptoms causes alarming symptoms.

Fatal period→One ounce of undiluted bromine causes death in about 7 days.

Signs and Symptoms

(i) Intense burning sensation in mouth, throat, oesophagus and stomach (ii) Intense thirst and excessive salivation (iii) Nausea and vomiting, gaseous eructations, dysphagia and diarrhoea (iv) Inhalation of Bromine fumes causes acute bronchial catarrh, irritating cough, running nose and watering from eyes and intense chest constriction (v) Oedema of glottis, pulmonary oedema develops with death from suffocation

Chronic Poisoning (Bromism)

Long continued use of bromides in large doses as medicines result in bromism, especially when the blood level reaches >50 mg%. persons with low sodium level are more prone to bromism apart from those who are sensitive to these preparations

Signs and Symptoms

(i) Bromide acne vulgaris' with papular or pustular skin eruptions on the face, shoulders and upper part of chest (ii) Headache, coryza, watering eyes (iii) Swelling of face and eyelids (iv) Indigestion with loss of appetite, constipation, furred tongue, foul breath (v) Tremors, muscular weakness,

staggering gait, loss of memory, slurred speech (vi) Features of bromide psychosis such as confusion of ideas, delirium, delusions or hallucinations, loss of sexual ability with mental depression and drowsiness (vii) Stupor and coma may result

Treatment

(i) Patient to be removed to fresh air (ii) Stomach wash with plain water containing starch or albumen (iii) Emesis to be induced (iv) Artificial respiration and oxygen inhalation (v) Tracheostomy to relieve oedema of glottis (vi) Sodium chloride 1-2gm flavoured with fruit juice is given every 6 hours orally until bromide level falls below 50mg% (vii) 5% Dextrose saline drip to be given i.v. that helps in excretion of bromides (viii) Diuretics like frusemide or chlorthiazide help in excretion (ix) Analeptics to treat shock and collapse.

Autopsy Findings

(i) Mucous membrane and skin bears dark brown parchment like stains (ii) Gastrointestinal tract is congested with inflammation (iii) There are features of pneumonia and pulmonary oedema (iv) All organs are congested.

Medicolegal Aspects

1. Accidental poisoning occur in those sensitive to bromides preparations. It was used in 1st World War when bromide vapours were used as asphyxiating and lacrymating agents.
2. Used commonly for homicidal purposes.

MECHANICAL IRRITANT POISONS

The irritant mechanical poisons act mechanically to cause local irritation of the gastro-intestinal tract when ingested. These are powdered glass, pins, needles, nails, diamond powder, and chopped human and animal hairs.

POWDERED GLASS

The powdered glass and diamond dust does not act as poison as they are not absorbed in the blood but they do act mechanically causing irritation of the gastrointestinal tract. Their fatal periods as well as fatal doses are uncertain. The faint shadow of glass particles on X ray due to presence of radio opaque silica helps in diagnosis

Signs and Symptoms

(i) Burning pain in mouth, throat, stomach and intestines (ii) Nausea, vomiting and constipation (iii) Rarely diarrhoea with tenesmus and bloody stools occur (iv) Death may result from shock following perforation of stomach or intestines by sharp glass fragments.

Treatment

(i) Bulky foods such as rice, porridge should be given (ii) Purgatives and cathartics should be given (iii) Big pieces of banana when swallowed as a whole envelops the glass fragments within itself (iv) Symptomatic treatment.

Neurotic Poisons

DELIRIANT POISONS

The Deliriant poisons are *Dhatura*, Cannabis and Belladonna.

DATURA FASTUOSA

The flowers and fruits are supposed to have been blessed by Lord Shiva and are known as *Shivashekhara*. It exists in two varieties: (i) *Datura Alba*—‘Safed’ or white flowers (ii) *Datura Niger*—*Kala datun* with black or deep purple flowers.

Both the varieties grow in waste places all over India. The fruits are spherical with sharp spines—**Thorn apple** (Fig. 38) and contains yellowish brown seeds. *Dhatura stramonium* grows at high altitudes in the Himalayas. Two other varieties that are commonly met with are: (i) *Dhatura atrox* and (ii) *Dhatura metal*. All parts of the plant are poisonous but the fruit and seeds are most poisonous.



Figure 39.1:
Dhatura plant
with its fruit

Active Principles

The active principles are: (i) Hyoscine (ii) Hyoscyamine (iii) Traces of atropine. It has been suggested that atropine does not exist as such but is a racemic form of Hyoscyamine that is converted to atropine during the process of extraction. The

seeds of *Dhatura* seeds may be mistaken for capsicum seeds but the differences do exist.

Signs and Symptoms

(i) Contact with the leaves and flowers cause dermatitis in sensitive patients (ii) When the seeds are ingested, the symptoms appear within half an hour (iii) At times vomiting occurs immediately after taking the seeds when crushed due to gastric irritation (iv) Bitter taste and dryness of mouth and throat (v) Burning pain in the stomach (vi) Dysphagia and difficulty in talking (vii) Headache followed by giddiness (viii) Staggering gait and incoordination of muscles (ix) Flushing of face (x) Dry and hot skin with rise in body temperature to 107-108 °F (xi) Pupils are dilated; there is photophobia (xii) There is loss of accommodation for near vision (xiii) The conjunctiva is red and injected (xiv) There is inability to pass urine (xv) Drowsiness (xvi) At times scarletiform rashes or exfoliation of skin are seen over most of the body (xvii) Pulse is full and bounding, later on it is weak, irregular and intermittent (xviii) Respiration is increased (xix) Muscle tone and deep reflexes are increased; muscular spasm is present at times (xx) Delirium is restless and purposeless. He is silent or mutters indistinct or inaudible words, but usually he is noisy, tries to run up away from his bed, picks bed clothes, tries to pull imaginary threads from the tips of his fingers, threads imaginary needles—pin rolling movements (xxi) Dreadful hallucination of sight and hearing is present (xxii) Delusions are present.

The signs and symptoms of acute *Dhatura* poisoning can be summarized by **10 D's**: (i)

Dysphagia (ii) Delusions (iii) Dermatitis (iv) Delirium (v) Drowsiness (vi) Dry skin, mucous membrane (vii) Dilated pupil (viii) Dreadful hallucinations (ix) Dysuria (x) Dysarthria.

The signs and symptoms can also be remembered as: (i) Hot as a hare—increased temperature (ii) Blind as a bat—disturbed vision (iii) Dry as a bone—intense thirst (iv) Mad as a hen—mental symptoms (Delirium, hallucination, delusion, restlessness) (v) Red as a beet—flushing of the face

As intoxication advances, this excitement phase passes off in 1-2 hours, deep coma or sleep ensues that may end in death from respiratory paralysis. Patient may stay ill for 2-3 days but definite improvement occurs in 24 hours. The cases that improve develop secondary delirium as stupor passes away. They remember nothing about the illness and are surprised to find themselves in the hospital.

Fatal dose→4 fruits; 100 seeds or 0.5-1 gm

Fatal period→24hours.

Treatment

(i) Emetics can be used (ii) Stomach wash using 5% tannic acid or a weak solution of potassium permanganate (iii) Enema should be given using sodium sulphate as purgative (iv) Prostigmine 0.5-1mg subcutaneously as single dose is more effective and less toxic (v) Physostigmine 0.5mg i.v or i.m 1-2 hourly to relieve both cerebral as well as peripheral manifestations, it crosses the blood brain barrier (vi) Pilocarpine nitrate 5 mg is given subcutaneously as a single dose and repeated after two hours. It has no central action (vii) Morphine is contraindicated, though it is physiological antidote (viii) Short acting Barbiturates can be given for convulsions (ix) Sponging to relieve high temp. (x) Artificial respiration and oxygen inhalation.

Autopsy Findings

(i) The postmortem findings are not characteristic but signs of asphyxia are there (ii) Seeds or their fragments may be found in the stomach or intestine (iii) Stomach may show slight inflammation (iv) Lungs are congested and oedematous (v) All the organs are congested.

Medicolegal Aspects

1. Datura is used mostly for criminal purposes. It is used to stupefy travelers for the purpose of robbery, rape and theft by: (i) Seeds that are mixed with rice, *dal*, coffee and sweets (ii) Seeds and leaves are mixed with tobacco or *ganja* and smoked in *Chillum* (pipe) (iii) A decoction (boiling to extract the essence) of seeds is added to liquor to enhance its intoxicating property.
2. Accidental poisoning can result: (i) When children and adults eat raw Datura fruits (ii) The seeds may be mistaken for capsicum seeds. (iii) Seeds have an aphrodisiac property. (iv) Juice of Datura leaves is used to treat rheumatism and applied for pain and inflammation. (v) *Vaids* and *Hakims* use Datura as medicine. (vi) Medicated ghee is prepared with ghee for local application.
3. Active principle is excreted unchanged in urine immediately after administration for 10-12hours. Urine should be preserved for analysis.
4. Datura seeds resist putrefaction for a long time but mydriatic principle contained in them is destroyed by putrefaction. Can be chemically detected even after a long time.
5. Datura is sometimes used for homicidal purposes. In 1921, the wife gave Datura seeds to her husband in the food.
6. Is used as an abortifacient, in a case a man administered *Datura* powder to a woman to produce abortion but she died 3 hours later.
7. **Medicinal use of Datura:** (i) Dried leaves mixed with Cannabis in the form of Cigarettes are used for the treatment of Asthma; the smoke is antispasmodic (ii) Tinctures have been used for application on the wounds—belladonna tinctures (iii) Datura leaves are used to treat some types of fever.

Differential Diagnosis of Datura Poisoning

(i) Poisoning by Hashish (ii) Opium poisoning; differentiated by pinpoint pupil and slow respiration (iii) Cerebral haemorrhage is differentiated by the presence of hypertension and CSF is diagnostic.

CANNABIS INDICA OR SATIVA

The plant *Cannabis indica* or *sativa* grows all over India but its cultivation is restricted due to the monopoly of the state government. Female plant is tall about 16-18 feet and has more darker and luxuriant leaf foliage than male plant (Fig. 39.2). It yields sticky amorphous resin, cannabinone, which consists of cannabinoids ($C_{12}H_{26}O_2$), a colourless oily liquid, cannabidiol, cannabinolic acid and Tetrahydrocannabinol.



Figure 39.2:
Cannabis sativa plant

Medical Applications of the Drug

(i) THC appears to control nausea, vomiting, and lack of appetite in cancer patients receiving chemotherapy when other drugs such as antiemetics fail (ii) Used as antidepressants for cancer patients (iii) Antidepressants for cancer patients (iv) Marijuana dilates bronchioles but it is usually not given in asthma (v) THC in small doses acts as anticonvulsant; in large doses, it causes convulsions (vi) Workers have found that the topical application on the skin and mucous membrane has antibiotic activity (vii) It dilates the vessels of iris that leads to increased intraocular pressure and glaucoma.

Effects of Cannabis

The effects of cannabis depends upon: (i) Amount of Cannabis (ii) Route of intake, whether smoked or eaten (iii) Body size, weight and health of the person (iv) Tolerance and dependence (v) Individual's mood (vi) Circumstances under which it is taken whether to reduce stress or with others in a social setting.

Effects Produced by the Small Doses

(i) Sense of well being (Euphoria) (ii) Loss of inhibitions (iii) Tendency to talk and laugh (iv) Impairment of coordination and balance (v) Loss of concentration (vi) Increased appetite (vii) Increased pulse and heart rate (viii) Reddened eyes (ix) Then the person passes to calm state of mind and sleepiness that lasts for 2-3hours after smoking (x) There can be hangover, drowsiness and poor coordination for several hours.

Effects produced by Large Doses

(i) Confusion (ii) Restlessness (iii) Excitement (iv) Hallucination (v) Anxiety or panic. Sometimes inexperienced Cannabis user can hallucinate in small doses. Cannabis also affects and impairs short-term memory, logical thinking, motor skills and ability to perform driving.

Tetrahydrocannabinol (THC)

Tetrahydrocannabinol is the active principle of *Cannabis indica* (Marijuana). For time immemorial it has been the most popular recreational and ritualistic intoxicant All parts of the male and female plant contain active principles. Tetrahydrocannabinol (THC) is absorbed in to the blood stream through the walls of the lungs, when smoked and through the walls of stomach or intestine when ingested. The blood stream carries THC in to the brain that produces the 'high' effects of the drug. It exerts quicker action through inhalation. The Tetra hydrocannabinols, are labile compounds that may change when exposed to U-V light or acid. The typical psychological effects are due to them. The following are the forms in which it is used:

Bhang (*Siddhi, Patti* or *Sabji*) is formed of the dried leaves and flowering or fruiting shoots. It is used as an infusion in the form of beverage. It is prepared by rubbing on a stone and black pepper, dried leaves and sugar is added to form pill or bolus. The water is added and it is drunk after it is strained through the muslin cloth. People of Northern India consume this drink at festivals like Holi especially at some of the places like Varanasi.

It is the mildest form containing about 15% of the active principle. Bhang is usually intoxicated to produce cheerful mood for dancing and singing. It acts as an appetizer so the individual consumes more food and he also seeks sexual enjoyment. The intoxication lasts for 3 days resulting in sleep. Fresh bhang is highly intoxicating and narcotic. Bhang that is kept for 2-3 years is mildly intoxicating and pleasure giving.

Majun: It is sweet meat of confection prepared from bhang after treating with sugar flow, milk and butter. It has an agreeable odour and sweet taste and sold in the market in small lozenge shaped pieces. It increases appetite and sexual desire. The person feels happy and quite contented as if he belongs to Raja's family and he has all what he wants. Sometimes Dhatura is mixed with Majun.

Ganja: It is prepared from the flowering tops of female plant. Has rusty green colour and characteristic odour and its active principle is 15-25%. About 20-30 grains are rubbed with little water on palm till it becomes sticky and then mixed with tobacco and smoked in a pipe (chillum). The person on smoking feels heavy and lazy. Also conjunctiva gets suffused and tachycardia and hypotension is produced. The person indulges in pleasant reveries though he is able to discharge ordinary duties. *Sadhus*, *faqirs* and poor labourers, cut leaves and flowering tops of American hemp plant *Cannabis Americana* or *Marijuana* and rolled them in to cigarettes with their hands and then smoke them. These cigarettes are called 'reefers' or 'pot'. A good quality cigarette contains 500 mg of marijuana that is effective after 15 minutes of smoking and produces benign ergosyntonic experience.

Charas or Hashish: Is of dark green or brown colour and commonly known as Hashish. It is a concentrated resin exuding from leaves and stem of the plant that grows in hilly areas, 6-8 thousand feet above the sea level. It has 25-40% active principle and is smoked with tobacco in a pipe or *hookah*. It is most potent of all the forms.

Hashish oil: It is an extract of cannabis and is thick, oily liquid spread on a tip of paper of cigarette. It is smoked and is strongest of all the prepara-

tions of Cannabis. Unlike alcohol it is always taken in a company. It produces the effects such as fleeting, brief and pass rapidly.

Signs and Symptoms

They appear soon after smoking ganja or Charas and after half an hour after swallowing bhang. When taken in small doses, effects are slight and then patient becomes pleased with himself. He becomes talkative, cheerful though rarely violent or aggressive. There are two stages:

1. **Stage of inebriation:** Person becomes dreamy or semiconscious. There are realistic visions of erotic nature. He sees nude beautiful females dancing before him, playing music, and singing amorphous songs. The higher faculties are depressed, he may laugh, sing or make purposeless movements. Judgment in relation to time and space is lost and sensation of pain is lessened. It hampers the psyche motor activity (physical coordination, quick reaction time and visual perception) headed for driving and operating machinery. Interferes with immediate memory, arithmetic problem solving and thinking in general. Frightful hallucinations are there at 300-480 µg/kg oral and 200-250 µg/kg of THC. He is delirious, has homicidal tendency or a fear of death. Symptoms vary with the dose and personality of the user.
2. **Stage of narcosis:** 0.5-1 mg/kg as single oral dose produces giddiness and ataxia. Speech may be confused and rambling. Tingling and numbness of skin occurs and in severe cases general anaesthesia and muscular weakness develops. The victim becomes drowsy and passes in to deep sleep and wakes with lassitude and impaired mental function. Recovery occurs in about 6 hours. In extreme rare cases death is due to respiratory failure.

Fatal dose → 1000-2000 mg or 30 mg/kg
Charas 2 gm
Ganja 8 gm
Bhang 10 gm/kg

Fatal period → Cannabis is very rarely fatal and the period of fatality varies from 12 hrs to several days.

Treatment

(i) Stomach wash to be done (ii) Purgatives should be administered (iii) Oxygen inhalation and artificial respiration to be given (iv) Symptomatic treatment.

Autopsy Findings

(i) The postmortem findings are not characteristic (ii) Only signs of asphyxia are present (iii) Its necessary to preserve urine (iv) *Microscopic examination of the ingested material*: The suspected material is placed on a slide and one drop of oil potassium hydroxide is added, covered with cover slip and examined under the microscope. Morphological features of hair of the THC derivatives should be studied distinctly; ganja hair are smooth and Charas hair are wavy or warty.

Chronic Poisoning

Regular users develop features of intoxication and may experience some physical, mental or social problems. There is an: (i) Increased risk of bronchitis, lung cancer and respiratory diseases associated with smoking (ii) There is a change of motivation: The person feels that they begin to loose energy after the drink. Their performance at the place of work suffers. When cannabis is stopped, the symptoms disappear (iii) Decreased concentration, memory and learning abilities—Toxic psychosis (iv) Interference with sexual and hormone production—Impotency, gynecomastia, low sperm count and irregular menstrual cycle (v) THC is soluble in fatty tissue, can be deposited in fatty tissue including brain then gradually can be realized in to blood stream (vi) Insanity, hallucination, delusion of persecution.

Medicolegal Aspects

1. Poisoning by Bhang is mostly accidental. Majun and Charas are used as stupefying poisons. Cannabis can be used for homicidal purposes; there are cases where it has been used to kill the other person.
2. To steady nerves before committing crime.
3. **Run amok**: After its intake, the person first kills a person or persons against whom they have entertained fancied or real enmity then

kills anybody who comes in their way until homicidal tendency lasts. Then usually he may commit suicide or surrender before the police.

4. **Cannabis and other drugs**: Marijuana and alcohol when used together are more effective and dangerous than when it is used alone. There is no evidence as to the use of Cannabis automatically leading to the use of other drugs.
5. **Cannabis and pregnancy**: There is not much information about the effect of THC in an unborn child but experiments on animals do show some abnormal developments and behaviour in the offspring.
6. **Cannabis and the Law**: Cannabis is a banned drug and the punishment for cultivation of plant and ganja illegally is imprisonment of 5 years and fine of Rs 50,000. For other products the punishment can be vary from 10-20 years with fine of 1-2 lakhs. The small quantity for personal use of Hashish and *Charas* is 5gms and for Ganja it is 500 gms. Exceeding this quantity will be liable to punishment of six months and fine or both.
7. **Cannabis and driving**: While driving intake of Cannabis affects motor and coordination skills along with the vision, perception of time and space. There are increased chances of vehicular accidents.

COCAINE

Cocaine is also commonly known by the names of Coke, Snow, White lady and Cadillac etc. It is an alkaloid derived from the leaves of the tropical shrub coca-tree, (*Erythroxylon coca* and *Enovogranatense*) These trees grow well in Central and South America, Mexico, Indonesia East Indies and India. The leaves are traditionally chewed in many countries such as South America to allay fatigue. Purified cocaine first became commercially available in 1884. Cocaine acts as a good surface anaesthetic and it was introduced as first local anaesthetic for use as ocular anaesthetic.

Cocaine along with heroin and its associated drugs forms the core of the hard-drug problem from which fatalities may result. In areas where cocaine use is common, a significant proportion of fetal deaths are associated with this narcotic. In a

series reported in New York by Morild and Stajic, toxicology revealed cocaine in 64 out of 103 fetal deaths. The study also suggested that cocaine causes an increased incidence of fetal deaths, abortion and abruptio placentae. Many a times, the smugglers have died due to bursting of packets of cocaine in their gastrointestinal tract resulting in a massive overdose.

The leaves of coca plant (*Erythroxylon coca*) contain about 0.5-1% cocaine. Cocaine, methylbenzoyl ecgonine may be synthetically prepared.

Routes of Administration

(i) Orally, it is rapidly destroyed (ii) Injection (iii) Snuffing via nasal and oral mucous membranes when it is rapidly absorbed and causes cerebral stimulation followed by depression (iv) Inhalation of freebase cocaine produces almost immediate absorption and a rapid onset of effects.

Various forms Used for Abuse

1. *Cocaine hydrochloride*: It is water soluble and is typically used for injection or snorting but is not well suited for smoking as it is largely destroyed by heat of burning. The hydrochloride salt can be converted to the freebase form by treatment with alkali and extraction with organic solvents.
2. *Cocaine sulfate* (coca paste, pasta basica, basuca) is a crude intermediate product, usually contaminated with solvents is used for smoking.
3. *Crack*: Crude form of freebase cocaine may be obtained by heating the cocaine with sodium bicarbonate to yield 'crack', a hard white mass that is freebase plus impurities but without hydrochloride moiety. When smoked, this material gives off a cracking sound, hence the name.
4. *Speedball*: Sometimes cocaine abusers simultaneously inject intravenously a mixture of an opiate, such as heroin called speedball. This mixture is especially euphorogenic. .

Mode of Action

Cocaine acts as a CNS stimulant and local anaesthetic with potent vasoconstrictor properties.

It stimulates the vagal centre, vasomotor centre, vomiting centre and temperature regulating centre. It blocks uptake of noradrenaline and adrenaline in to adrenergic nerve endings producing sympathomimetic effects. The reinforcing effects of cocaine are related to effects on dopaminergic neurons in the mesolimbic system.

Metabolism

The half life of a single dose of cocaine in the blood is only about 30-90 minutes. It is hydrolyzed by butyrylcholinesterase (plasma pseudocholinesterase) and the liver esterase into inactive metabolites, mostly benzoylecgonine and ecgonine methylester. With repeated high dosages, cocaine or its metabolites may accumulate in the body compartments such as fat and central nervous system from where it is then slowly released. Consequently, using sensitive techniques, cocaine may be detectable in the urine of heavy users for a couple of weeks. Metabolites can also be detected in blood, saliva, sweat and hair. Blood and saliva provide a better index of current concentrations, whereas urine provides a longer window of opportunity for detecting use over the previous few days.

Use in Conjunction with Other Drugs

Drugs such as cannabis, ethanol, sedatives and hypnotics are frequently taken with cocaine to reduce its unpleasant effects.

Use with alcohol: Alcohol is most commonly used along with cocaine as it modulates both the 'cocaine high' and 'dysphoria' associated with abrupt disappearance of cocaine's effects. The concurrent use of cocaine and alcohol may result in the accumulation of a distinct metabolite, cocaethylene.

Cocaethylene; Cocaethylene is an active metabolite and produces changes in the cardiovascular functions as that produced by cocaine alone. It produces long lasting effects than cocaine itself and also accounts for the enhancement of subjective effects as well as toxic symptoms when the two are used simultaneously.

Fatal Dose

As little as 20-50 mg when applied to nasal mucosa has caused deaths but even a gram when taken orally does not result in death. Chronic users of cocaine develop tolerance and habituation that is why it is difficult to estimate dangerous dose levels. Intravenous doses are of 100 mg and a lethal dose is ten times this dose, though there are chances that far larger doses can be tolerated by habitual users. Absorption through the nasal mucosa is less effective and larger doses are to be used to obtain the same effect as that produced by the intravenous route.

Acute Intoxication

Cocaine users develop the signs and symptoms of intoxication during the course of single binge and a hyperadrenergic state is produced.

Stage of euphoria: This stage is accompanied with (i) Increased suspiciousness and hypervigilance (ii) Anxiety, hyperactivity and talkativeness (iii) Grandiosity and excitement.

Signs and symptoms resulting from central stimulation: (i) Increased sense of well-being with increased power of endurance (ii) Tachycardia and ventricular arrhythmias (iii) Changes in blood pressure (iv) Pupillary dilatation (v) Perspiration and chills (vi) Hallucinations especially tactile (vii) Judgment is impaired but insight into drug induced nature of the hallucination is usually retained (viii) Dyspnea.

Stage of depression: (i) Loss of reflexes (ii) Paralysis of muscles (iii) Coma and tonic-clonic seizures (iv) Circulatory and respiratory failure (v) Sudden death may follow i.v injection, smoking and snorting due to cardiac arrhythmias and cardiopulmonary arrest (vi) Complications such as pulmonary oedema after intravenous injection of freebase cocaine and pneumomediastinum and pneumothorax may occur after sniffing.

Causes of death: (i) Cocaine overdose or hypersensitivity (ii) Sudden cardiac arrest in first time users.

Chronic Intoxication

It is also known as Cocainism/cocainophagia/cocainomania. Cocaine is usually abused by upper classes of society to enhance self-image or improve professional performance. There is a tendency for compulsive use of the drug though classical physical dependence is uncommon. Psychic dependence is common with chronic use of the drug and the euphoria following injection is short-lived, perhaps only for 15-20 minutes. Immediately upon intake there is:

1. Euphoria (*rush*) followed about an hour or so later by rebound depression (*crash*). To avoid unpleasant effects of rebound depression, the person feels compelled to take the drug again
2. It can also induce a toxic delirium and a more persistent toxic psychotic disorder characterized by: (i) Suspiciousness (ii) Paranoia (iii) Visual and tactile hallucinations such as hallucinations of bugs (**cocaine bugs**) and worms crawling under the skin (**formication/magnan's symptoms**) (iv) Loss of insight.
3. Chronic intake of cocaine in males causes impotence and gynaecomastia and in females menstrual disorders including amenorrhoea, galactorrhoea and infertility.
4. In pregnant woman with cocaine abuse there is incidence of abortion, abruption placentae and cerebrovascular diseases in mother. In the foetus also incidence of congenital malformations. The smoking of 'crack' results in production of 'crack babies'.
5. Rarely, ulceration and perforation of the nasal septum occurs in chronic 'snorters'.

Treatment

(i) To control seizures, intravenous diazepam 0.5mg/kg may be given (ii) Propranolol 0.5-1 mg I.V. for ventricular arrhythmias (iii) To treat psychosis, haloperidol should be given (iv) Treatment of chronic cocaine abuse requires combined efforts of primary care physicians, psychiatrists and psycho-social workers.

Autopsy Findings

1. There are no specific autopsy features.

2. Findings of hypoxia are evident.
3. Oral ingestion results in findings of gastric ischemia.
4. The diluents such as quinine, mannitol and other bitter flavouring agents used to 'cut' the drug for illegal sale may be found at injection sites, the regional lymph nodes, in the lungs and in other organs.
5. Particles of cocaine can be seen in the form of microemboli.
6. Adulterants used with cocaine are same as that in heroin such as talc, starch, quinine, lactose and dextrose. Quinine was used as diluent to treat falciparum as malaria transmission was quite common among amongst needle and syringe sharers.
7. Formation of foreign body granulomas in the lungs when cocaine mixed with talc or starch is used intravenously. This is due to filtering out of the undissolved components in the pulmonary capillary bed.
8. Evidence of cerebral haemorrhage is found at autopsy. These haemorrhages are the result of hypertension when blood pressure rises to over 300 mm Hg.
9. *Complication of the use of septic methods of injections:* (i) infections HIV and Hepatitis (ii) endocarditis affecting the right heart valves (iii) phlebitis and distant embolic abscesses.
10. Nasal swabs, one from each nostril should be taken and sent for analysis along with an unused cotton wool swab as control.
11. *Samples to be preserved:* (i) swab from each nostril using a plain cotton swab along with another swab used as control (ii) Routine viscera and blood sample for cocaine levels (iii) Sample of urine as cocaine metabolites can be detected for varying lengths of time in urine depending on the dose of cocaine and sensitivity of the assay. The metabolite is generally detectable in the urine for 24-72 hours after brief periods of use. (iv) Injection prick site along with control sites.
12. According to **Baselt**, blood levels in fatal cases range from 1-21 mg/L with a mean of 5.2mg/L.

Medicolegal Aspects

1. Accidental cases occur from the addiction, hypodermic, urethral, vaginal or rectal injections
2. The drug is locally applied on penis as it is believed to be an aphrodisiac. Prostitutes sometimes inject cocaine solution into vagina to produce local constriction. Cocaine is also instilled in the urethra
3. Chronic users may develop mental derangements who may get involved in crimes, of which there are three categories: (i) Psychopharmacological effects (ii) Economic compulsion (violent crimes committed to obtain money for drugs) (iii) Systemic violence (associated with business methods and lifestyle of drug dealers).
4. Cocaine induces states of paranoid and aggressive behaviour.
5. Protracted cocaine abuse may cause paranoid ideation and visual and auditory hallucinations, a state resembling alcoholic hallucinations.

CNS Depressants

The term 'alcohol' always means ethyl alcohol. Alcohols are organic substances bearing 'OH' groups. Only human beings in the form of various alcoholic drinks consume ethyl alcohol. Methyl alcohol is not fit for human consumption and is found as a impurity in a number of cheap alcoholic drinks. The chemical formula for ethyl alcohol (ethanol) is C_2H_5OH . Its specific gravity is 0.79 that is 1 ml of alcohol weighs 0.79 gm.

Type of alcohol	Percentage of alcohol
Absolute alcohol	99.95%
Rectified spirit	90%
Denatured alcohol or industrial spirit	95% alcohol + 5% wood naphtha.

Proof spirit is one that at 10.5°C weighs exactly 12/13 part of an equal measure of distilled water. The weaker spirits are known as 'Under proof' and stronger spirits are called 'Over proof'.

Alcoholic drinks contain alcohol with water and congeners (organic acids and esters). Wine and brandy have methyl alcohol as congeners. The percentage of alcohol in various drinks is given below:

Products of alcohol	Alcohol by volume
Rum	50-60%
Whisky, Gin, Brandy	40-45%
Port, Sherry	16-20%
Wine	10-15%
Beers	4-8%
Arrack (Liquor distilled from palm rice, sugar or jaggery)	40-50%

Absorption

Alcohol is absorbed 20% through the empty stomach and 80% through the small intestine to

the blood. From the blood, the alcohol goes to liver where it is damaged by a specific enzyme at a constant rate. Blood alcohol reaches the lungs through the venous circulation and through the arterial side of the heart in to all the organs and tissues of the body mainly the brain. In brain, it interferes with the normal brain functions such as judgment and coordination of muscle movements. The blood concentration is influenced by many factors.

Only the concentration of alcohol in the blood influences the behaviour. The amount in stomach and intestine indicates only the ingestion and has no effects as such. After death, alcohol in the stomach may diffuse in to the blood and tissues. This results in higher blood level than actually existed during life, if the blood sample is taken from the part in to which post mortem diffusion has taken place. In reference to absorption of alcohol, certain points need to be elaborated:

1. Absorption is slower in the stomach than intestine.
2. Absorption is faster in empty stomach than when it contains food.
3. Food affects the absorption by delaying the emptying time of the stomach: (i) Fatty foods, proteins and starch delay the absorption (ii) Milk has similar effect i.e., it delays absorption (iii) Food prevents 10-20% of the ingested alcohol from being absorbed.
4. Diluted form of alcohol is absorbed slowly than the concentrated form
5. The type of beverage also affects the rate of absorption. Whisky is absorbed rapidly and its complete absorption occurs within 1-3

hours. Then there is equilibrium between blood and tissue concentrations of alcohol. After 15 minutes of drink, the level of alcohol in arterial blood may be 40-60% higher than peripheral venous blood and may persist during the first hour. However, after absorption is complete alcohol levels in venous blood is slightly higher than the arterial blood. Therefore, the blood samples should be taken after equilibrium is reached.

6. The absorption of alcohol depends on habituation and tolerance of the individual as habituated heavy drinkers tend to absorb alcohol more rapidly.
7. Emotional state of the person affects the contraction of the stomach resulting in rapid emptying time and the rate of absorption.
8. Drugs like Bensedrine and atropine slows the absorption and retards the emptying time.
9. The age of the person also has an effect on the absorption as it occurs less rapidly in younger (<35years) individuals.
10. Individuals with gastrectomy have an increased rate of absorption.
11. *Inhalation*: Concentration in the blood is proportional to the concentration in the inspired air
12. *Weight of the subject*: The same amount when taken by subjects of different weight has different blood levels depending upon the amount of bone, fat and muscles.

Excretion of the Alcohol

Alcohol is excreted 5% in urine, 5% in breath and very little through the sweat, faeces and saliva. In hot climates, significant amount is lost through the sweat.

Metabolism

About 90% of the alcohol absorbed is oxidized in liver and rest 10% excreted. Alcohol is oxidized to acetaldehyde by alcohol dehydrogenase (ADH) and coenzyme nicotinamide-adenine dinucleotide (NAD) in the liver. This acetaldehyde is converted into free acetic acid (or acetyl coenzyme A). The acetate undergoes oxidation to carbon dioxide and

water in the citric acid (Krebs) cycle. This acetate can form glycogen, proteins and also fats and cholesterol. In case of a diabetic who is ketogenic will produce fat from alcohol as he cannot use sugar. Alcohol from blood disappear at a rate of 10-15 ml/hour (15 mg%/hour) and is not stored in the tissues. Due to an increase in liver enzymes, chronic alcoholic can metabolise alcohol faster at the rate of 40-50 mg%/hour. Of the metabolise alcohol 10% get deposited in the tissues in the form of cholesterol and neutral fats.

Pharmacological Actions of Alcohol

Alcohol being a central nervous system depressant causes an irregularly descending type of depression. Higher centres are depressed first followed by the midbrain and thalamus, spinal cord and finally the medulla. The recovery occurs in a reverse order.

Clinical Consequences of Alcohol

The disturbance of behaviour is due to the influence of alcohol on the central nervous system depending to what extent the brain function is affected. The initial symptoms such as feeling of well being, self confidence, garrulousness, exhilaration are due to the depression of the higher inhibitory control over the lower centres thereby causing a release phenomenon.

Signs and Symptoms

The signs and symptoms will be more marked as the alcohol concentration in the tissues increases. This depends on the (i) Amount (ii) Time lapsed and (iii) Tolerance. It is claimed that a certain degree of intoxication for the same level of alcohol in the blood is more marked during the phase of getting drunk than in the phase of sobering up. The departure from the normal is usually tested by a clinical examination of the subject to and the way in which the central nervous system is affected in response to acute mental and motor deterioration.

The normal discipline and controlled conduct is due to the control of higher centers of brain. When sufficient alcohol is taken the higher centers,

the normal inhibitions and restraints are removed. This is one of the earliest effects of alcoholic intoxication and may show in a variety of ways such as garrulity, impairment of judgment and recklessness. As the degree of intoxication advances, other CNS signs and symptoms manifest. There are three phases of intoxication:

1. **State of excitement:** This occurs at the blood alcohol level of 50-150 mg%, there is feeling of well-being, slight excitement, confidence is increased and a lack of self-control is there. The sexual desire is heightened but the performance is reduced. The visual acuity is also reduced in the blood alcohol concentration of 20 mg% in a non-drinker; 25-35 mg% in a moderate drinker and 40-70 mg% in a heavy drinker. The time and space orientation is altered with feeling of sleepiness. There is loss of critical judgment and the mental concentration is retarded. The memory is impaired.
2. **State of in-coordination:** The symptoms of in-coordination appear when the blood alcohol level is 150-250mg/100ml. The sense perception and skilled movements are affected. Inhibition of higher centers is lost resulting in change in the conduct of the individual. The person becomes irritable, gay, quarrelsome and sleepy. There is in-coordination and clumsiness of fine and skilled movements. The speech is also altered. There are fine movements of fingers and tremors. Nausea and vomiting is common. Pulse is raised and face is flushed. The sense of touch, smell and hearing is diminished. Pupils are dilated and if light reflex is positive, it is a hopeful sign.

AGN (Alcoholic gaze nystagmus): Undue importance has been given to this sign and it is by no means a constant or common sign. It is present only in about 10% cases. The nystagmus on lateral gaze at the normal position of head with open eyes may occur at blood alcohol of 0.08% (0.04-0.1%). It may even be absent at 0.06%. This sign by no means is indicative of alcoholic intoxication.

Physiological nystagmus also occurs in 20% cases. In addition, nystagmus can be brought about by emotion, fatigue and postural hypo-

ension. Nystagmus is also found in head injuries, liver diseases

Positional alcohol nystagmus (PAN): It is seen in two phases PAN 1 and 2. The subject rests in supine position with eyes closed and head remaining in the right or left lateral position. Specialized instruments record the eyes movements.

PAN 1—It appears after half an hour after taking a single dose of alcohol and lasts for 3-4 hours. Duration is independent of the dose. When the head is on the right lateral side, slow component is upwards (Antigravity) and the rapid component is downwards (to the right)

PAN 2—It starts after two hours of PAN 1. It beats in the reverse direction of PAN 1 that is the fast component beats to the left in the right lateral position of head and to the right in the left lateral position of head. The duration of PAN 2 depends on the dose of alcohol ingested.

3. **Stage of coma:** Motor and sensory cells are deeply affected. Speech is slurred and the coordination is markedly affected. Gait is staggered and the individual may fall. The patient is in a state of coma and there is stertorous breathing. The pulse is rapid, temperature is subnormal and the pupils are constricted. On painful stimuli the pupils are dilated which then return slowly to the contracted stage called **McEwan's sign**. The patient may die or may recover in 8-10 hours with acute depression, nausea and headache. If coma is more than 5 hours, the death may occur resulting from asphyxia due to respiratory paralysis. In acute alcoholic intoxication even <400mg% of blood alcohol concentration can cause death if the patient is suffering from heart or lung diseases.

Differential Diagnosis of Alcohol Poisoning

- (i) Severe head injury
- (ii) Metabolic disorders such as diabetic or hypoglycemic coma
- (iii) Intracranial tumours
- (iv) Epilepsy
- (v) Parkinsonism
- (vi) Intake of drugs such as Insulin, barbiturates, atropine, antihistaminics, hallucinogens or morphine
- (vii) Psychological disorders such as general paralysis of insane and hypomania
- (viii) High fever
- (ix) CO poisoning.

DRUNKENNESS

In 1927, the British Medical Council defined the word **Drunk** "The person concerned was so much under the influence of alcohol to have lost control of his faculties to such an extent as to render him unable to execute safely the occupation in which he is engaged at the material time".

In order to establish the diagnosis of drunkenness, the person should be carefully examined along with the collection of blood and urine samples for chemical analysis. Following points should be considered while examining a person for drunkenness:

1. The quantity of alcohol ingested is not related to the state of intoxication at the time of examination.
2. The signs and symptoms, none of which is peculiar to this condition except the odour of the breath
3. An individual can react differently to different situations. Mentally unstable, epileptics and those with cerebral trauma may react even with small amount of alcohol.
4. Even the smell of alcohol on the breath does not necessarily prove the person to be intoxicated because smell can persist for a longer time after the alcohol has been excreted from the body as it is due to the non-alcoholic constituents (congeners).
5. In many clinical conditions except the smell of alcohol in breath, all or most of the symptoms and signs of alcoholic intoxication are found. These are (i) head injury (ii) diabetic or hypoglycemic coma (iii) Uraemic coma (iv) neurological conditions such as intracranial tumours, parkinson's disease, epilepsy, disseminated sclerosis that show ataxia, tremors and drowsiness simulating drunkenness (v) cerebrovascular accidents and (vi) drugs as listed above. Smell of alcohol in breath is an important feature. However, any of the above clinical conditions can co-exist with drunkenness or alcohol may modify the clinical features of these conditions specially the effects of drugs like barbiturates and tranquilisers are markedly potentiated by alcohol and many alcoholics

develop the habit of taking alcohol and these drugs to experience 'kick'.

6. A quite characteristic and aggressive odour of alcohol in the breath, loss of clearness of intellect and control of himself, unsteady gait, vacant look, dry and sticky lips, congested eyes, sluggish and dilated pupils, increased pulse rate, unsteady and thick voice, random talks and want of perception of the passage of time are the usual signs of drunkenness.

Model Proforma for Examination of An Individual with Drunkenness

Special committee of British Medical Association has suggested the scheme of examination of an alleged case of drunkenness. In 1965 this committee has revised the original report of 1954 with a new title, 'The drinking driver'. The committee has defined alcoholic intoxication as a condition produced in a person who has taken alcohol in a quantity sufficient to cause him to lose control of his faculties to such an extent that he is unable to execute the occupation on which he is engaged at the material time. The committee has proposed this proforma and protocol for such an examination:

1. Date, time, place of examination.
2. Preliminary details of the person being examined such as name of person with father's name, age, residential address etc.
3. Consent and referring person that is investigating police officer.
4. Exclude injuries and pathological conditions, if any.
5. A brief history is to be recorded of (i) Amount of liquor taken (ii) Time of ingestion (iii) History of any disease (iv) Any treatment taken.
6. General behaviour (i) General appearance (ii) *State of clothing*: Whether soiled by vomiting or incontinence and the colour of the vomitus (iii) *Character of speech*: Whether it is thick, slurred with slight blurring of letters or over-precise. Memory of the recent things with mental alertness (iv) *Writing*: The following points should be noted: (a) Time taken to write (b) Repetition or omission of words or letters (c) Signatures can be compared (d) Ability to read his own handwriting.

7. General examination should include:
- (i) *Pulse*: Resting pulse at the beginning and at the end of the examination should be recorded; the pulse may be rapid
 - (ii) *Temperature* should be recorded; may be raised
 - (iii) *Skin*: Is dry or moist; flushed or pale
 - (iv) *Mouth*—note the smell and whether the tongue is dry
 - (v) *Eyes*: Whether the lids are swollen? (a) Is conjunctiva congested? (b) Colour of the eyes
 - (c) Visual acuity (d) Size of pupil (is dilated in early stages and constricted in late stages)
 - (e) Reaction of eyes to light (d) Nystagmus (fine lateral nystagmus suggest alcoholic intoxication)
 - (vi) Smell of breath and any hiccup
 - (vii) *Ears*: Impairment of hearing
 - (viii) *Gait*: Manner of walking and turning (note any lurching or reeling) by asking the person to walk on a straight line and suddenly asked to turn back. He is then asked to stand with eyes closed and open and his ability to stand is noted
 - (ix) *Muscular coordination*: This is assessed by finger nose test (asking him to touch nose with his index finger), picking up a pencil or coin from floor, lighting a cigarette, buttoning and unbuttoning clothes
 - (x) Reflexes knee and ankle.
8. Systemic examination including that of chest, CVS and abdomen should be conducted.

9. **Opinion**: The opinion may be given as: I am of the opinion that the person examined (i) did not consume alcohol; or (ii) consumed alcohol but not under the influence; or (iii) consumed alcohol and under the influence (intoxicated).

CONSUMPTION OF ALCOHOL

One unit = 10 grams of absolute alcohol
 60 grams = 66 ml of absolute alcohol
In males: (i) the amount of absolute alcohol should not exceed 4 units/day (ii) 4-6 units/day is Hazardous (iii) more than 6 units/day is Harmful.
In females: (i) the amount of absolute alcohol should not exceed 2 units/day (ii) 2-4 units is Hazardous (iii) more than 4 units is Harmful.

Indications of Hazardous and Harmful Alcohol Consumption

Hazardous alcohol Intake: (i) Regular intake of more than 60 gm/day in males and 40 gm/day in females (ii) Occurrence of episodes of intoxication twice monthly or more (iii) Drinking behaviour such as (a) Gulping the first drink (b) Difficulty in stopping drinking once started (c) Most of the leisure activities involve drinking (d) Most friends are heavy drinkers (e) Eating lightly or skipping meals while drinking (f) Attempts to cut down on drinking with limited success.

Table 40.1: Various medical terminologies and the effects of alcohol

Medical terminology	Blood alcohol concentration (mg%)	Effects	Symptoms and signs
Sober	<10	No effect	No features
Drinking	20-70	Impairment of cognitive function, motor coordination and sensory perception. Mild euphoria is produced	
Under the influence	80-100	Decreased inhibitions, increased self-confidence, alteration of judgment, euphoria,	Flushing of face, dilatation of pupil, loss of inhibitions, speech is heavy, staggering gait, careless and in-coordinated
Drunk	150-300	Muscular incoordination, staggering gait, drowsiness, dizziness, disorientation, heavyness of speech and marked mental conclusion	Face flushed, dilated and inactive pupil, rapid eyeball movement, loss of restraints, heaviness of speech, staggering gait and clouding of intellect
Coma and death	≥400	Respiratory failure, deep coma and death	The pupils are contracted, pulse is rapid, breathing stertorous and deep unconsciousness

Harmful alcohol consumption: (i) Psychological manifestations in the form of: (a) Amnesic episodes while intoxicated and blackouts (b) Insomnia and nightmares (c) Accidents where alcohol is involved (d) Charges of driving under influence (e) Being late or absent from work (f) Concern or worry about driving by self or family (ii) Physical symptoms such as (i) dyspepsia (ii) morning nausea and vomiting (iii) recurrent diarrhoea (iii) General appearance as (a) bloated facies (b) conjunctival injection (c) coating of tongue and (d) presence of scars and bruises especially of varying ages (iv) Demeanor: Anxious and agitated (v) Cardiovascular system: Tachycardia and hypertension (vi) Abdominal signs: Soft hepatomegaly is there (vii) Trauma; Rib fractures may be there.

Other Effects of Alcohol

Delirium tremens: It is the unsoundness of mind resulting from long continued action of alcohol on the brain. It occurs in chronic alcoholics due to: (i) Temporary excess (ii) Sudden withdrawal of alcohol (iii) Shock after receiving injuries such as fracture of bone (iv) Acute infections. Delirium tremens is a form of acute insanity in which the following signs and symptoms are present: (i) Coarse muscular tremors of the face, tongue and hands (ii) Restlessness (iii) Loss of memory (iv) Uncontrollable tears (v) Tendency to commit suicide, homicide, violent assault or to cause damage to the property (vi) Disorientation for time and place (vii) Hallucination of hearing, sight and tactile like the insects are crawling under the skin.

Alcoholic polyneuritis: The signs and symptoms of alcoholic polyneuritis are: (i) Generalized weakness (ii) Pain in the extremities (iii) Wrist drop and foot drop (iv) Unsteady gait (v) Loss of deep reflexes (vi) Tenderness of muscles of arms and legs.

Wernicke's Encephalopathy and Korsakoff's Psychosis: Deficiency of vitamin B₁ occurs in heavy drinkers resulting in lesions of brain or spinal cord. This condition is rapidly fatal with death resulting within 24 hours. The patient becomes disoriented and forgetful of the recent past events.

There is drowsiness, peripheral neuropathy, ophthalmoplegia, nystagmus, ataxia, delirium and stupor. This condition when untreated can progress to Korsakoff's psychosis in which there is impairment of short term memory, inability to learn new information and confabulation is there.

TREATMENT APPROACHES

The following treatment approaches are undertaken:

Detoxification

Detoxification is a process by which alcohol dependent persons recover from intoxication in a supervised manner so that the withdrawal symptoms are minimised. In general, symptoms of withdrawal (delirium tremens) are opposite to those of an intoxicated state and develop within 6 hours such as (i) Hyperactivity of central nervous system (ii) Tremors (iii) Sweating (iv) Agitation and anxiety (v) Disorientation (vi) Hallucination and (vii) Convulsions. Delirium tremens is a medical emergency and its treatment is of two types, namely.

I. Non-medicated detoxification

Home detoxification: (i) No history of severe withdrawal, fits or acute organic brain syndrome (ii) No current signs of severe withdrawal (iii) No medical illness is there (iv) No evidence of psychiatric illness exists. In this treatment regimen, friends or relatives should stay with the patient and they should call the doctor if needed.

In Patient Detoxification: This treatment regimen is followed when the conditions at home atmosphere are not favourable. Here the withdrawal symptoms can be relieved by the provision of supportive counseling in a non-stimulating and non-threatening environment. There are special techniques to relieve the anxiety and agitation.

II. Medicated Detoxification

In essence, one is substituting controlled drug intoxication for an uncontrolled one. Drugs that are used for medicated detoxification: (i) Chloromethazole: It is a short acting sedative and anti-

convulsant (ii) Diazepam: It is a long acting drug given orally every two hours until sedation is achieved; maximum 120mg can be given (iii) Clonidine: It is a major tranquilizer and antipsychotic drug that is given when the above drug fails (iv) Thiamine is administered intravenously (v) Electrolyte and acid base balance is corrected.

Pharmacological treatment: (i) *Disulfiram (Antabuse)*: It was introduced about 40 years ago. It acts by inhibiting hepatic enzyme aldehyde dehydrogenase whenever alcohol is ingested. It produces symptoms such as flushing of face, upper trunk, causes, vomiting, dizziness and headache. (Temposil 50mg/day) (ii) *Serotonin uptake inhibitors*: They depend upon the neurochemical basis of alcohol uptake. These are Zimelidine, Viaqualine; they inhibit the uptake of Serotonin at nerve synapses (iii) Alcohol antagonists is Imidazodiazepine: It antagonizes the effect of alcohol.

Behavioural treatment: Behavioural treatment is based on the theory that many important aspects of human learning takes place by imitating the behaviour of others by a much greater attention to cognitive process in specifically human learning. The behavioural therapy includes: (i) **Aversion therapy**: The principal of aversion therapy is simply to attempt to create an aversive conditioned response to alcohol by pairing stimuli as associated with alcohol taking. They produce effects like nausea or electric shock on taking alcohol e.g. Emetine (ii) **Cue exposure**: It is the latest method; the alcoholics are exposed to alcohol related cues such as Pubs or friends (iii) Psychotherapy and counselling (iv) Alcoholic anonymous

MEDICO-LEGAL ASPECTS

S.85 I.P.C: 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.

Scope of S.85 I.P.C: S.85 gives the same protection as S.84 does to a person of sound mind

who 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 the thing was given against the will and without the knowledge.

S.86 I.P.C: In cases where an act is 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 had if he had not been intoxicated, unless the thing which intoxicated him was administered to him without his knowledge or against his will. Under S.86 a person who gets in to a state of intoxication voluntarily presumed to have the same knowledge as he would have had he not been intoxicated. When the state of intoxication is such.

Principle: Since the criminal intention is the foundation of the criminal responsibility, it follows that a person who is drunk is in the same predicament as a person temporarily insane. However, since no man can be permitted to hear the clock of immunity by getting drunk, the rule justly accepts cases of voluntary drunkenness.

When drunkenness is a defence: Voluntary drunkenness as such is never a defence, still when habitual drunkenness produces frenzy that fact becomes material for showing how it was produced and when its existence is established, it will excuse a crime.

When drunkenness is relevant in considering intention: (i) Drunkenness caused without one's knowledge or against one's will excuses the crime (ii) Voluntary drunkenness is an excuse only as regards 'intention' so that it is a complete excuse in crimes requiring the presence of an intention to complete a crime (iii) However, voluntary drunkenness is no excuse for a crime, which requires the presence merely of knowledge as distinct from an intention (iv) In any case, though voluntary drunkenness is no excuse for knowledge, it does not imply actual knowledge giving rise to the inference or presumed intention.

Scope of S.86 I.P.C: The law places insanity and involuntary drunkenness on the same footing by

using the same criteria, that is incapability of knowing the nature of the act or that he is doing what is wrong or contrary to law.

A person who gets in to a state of intoxication voluntarily is presumed to have the same knowledge as he would have if he had not been intoxicated. When the state of intoxication is such as to make him incapable of knowing the nature of the act or that he is doing wrong or contrary to law, he can only be punished based on knowledge and not of any particular intention.

Principle: Voluntary drunkenness does not afford the defence that the knowledge required to complete an offence was wanting, though it may be used to show that any intent if required was absent.

The section draws a distinction between presumption as to knowledge and presumption as to intention and though intention is to be inferred from knowledge.

Presumption of knowledge and intention: 'Knowledge' means knowing what is known, there is personal range of information whereas 'intention' means ultimate aim or purpose.

Even a drunken man is presumed to intend the natural consequences of his act, unless he proves incapacity in himself to form the intent necessary to constitute the crime. A distinction must be made between motive, intention and knowledge.

'Motive' is something, which prompts a man to form an intention and knowledge, is an awareness of the consequences of the act. In many cases, intention and knowledge merge in to each other and mean the same thing more or less and intention can be presumed from knowledge. The demarcating line between knowledge and intention is no doubt thin but not difficult to perceive that they connote different things.

Alcohol and Traffic Accidents

There is deterioration in driving ability as blood alcohol level rises to 30-50% as it: (i) Increases reaction time (ii) creates false confidence (iii) impairs concentration (iv) produces muscular in-coordination (v) Decreases visual and auditory acuity.

At the blood alcohol concentration of 50 mg%, the drivers are able to carry out road safety rules properly. At the levels of 60 mg%, the chances of being involved in an accident increase twofold. There is markedly increased risk at levels above 80 mg%; increases 12 times at 100 mg% and 25 times at over 150 mg%. at this level there is a distinct impairment of driving whereas at 200 mg%, all drivers are intoxicated and again there is an increased risk of accidents. Drivers of motor vehicles often fail to realize that although after drinking, they may experience feeling of well being, their critical faculties are blunted and they are liable to take risks and make mistakes while driving. In some countries, driving with certain blood alcohol levels is an offence:

- Czechoslovakia—30 mg%
- Sweden and Poland—20 mg%
- Austria—50 mg%
- France and U.K.—80 mg%
- USA—100-150 mg%

In India, the statutory limit is 30 mg% (section 185, Motor vehicle Act 1988) and for contravention the punishment involves fine up to Rs 5,000 or 6 months imprisonment or both for the first offence. For subsequent offence, the fine is up to Rs 3000 or imprisonment up to 2 years or both.

LABORATORY ANALYSIS OF ALCOHOL

The concentration of alcohol in blood, urine, breath and saliva helps in estimating the degree of intoxication. Vitreous humour and urine are good for analysis as they are not effected by putrefaction for a longer period and also they do not contain much glucose. Blood is the best sample and gives the direct evidence of the concentration of alcohol in brain but it is difficult to collect the sample in an uncooperative patient. In addition the consent of the patient is necessary and sometimes substances such as acetone, ether and paraldehyde may be estimated as alcohol.

1. **Breath analysis of alcohol:** 60-100ml of breath is received in to a dry balloon and analyzed by Drunkometer, Intoximeter, Alcometer or Breath analyser. Breath analyser is more reliable

Principle of breath analyser: The concentration of alcohol in deep lung air is dependent on arterial blood. 2100 ml of alveolar air = 1ml of blood. The converted breath tests are in close agreement with those obtained by direct blood analysis. The person is asked to blow in to a plastic balloon through a glass tube containing a crystalline bichromate-sulphuric acid mixture. Blood alcohol >80mg% is positive (Green colour).

Causes of false positive results: (i) Alcohol may remain in the mouth up to 20-30minutes after a small peg. Therefore, the test is to be repeated after 20 minutes (ii) Hyperventilation (iii) Physical exercise (iv) Emesis (v) Regurgitation of stomach contents. So the plastic, aluminium and other flexible bags are made available so that the breath samples may be preserved for several hours.

2. **Blood analysis:** To clean the skin before withdrawing blood, spirit should not be used and syringe should not contain even a trace of alcohol in any form. The skin can be cleaned with a solution of 1:1000 mercuric chloride and can be wash with soap and water 10mL of blood is collected in a glass vial containing 10mg of sodium fluoride and 30mg of potassium oxalate as preservative. The bottle is tightly capped with screw and kept in a refrigerator but not frozen. The analysis should be performed within a week. Whole blood alcohol concentration is 12-20% lesser than the serum or plasma concentration.
3. **Saliva:** Before collecting saliva samples, mouth should be thoroughly rinsed with water. About 5mL of sample should be collected in a test tube containing 10mg of sodium fluoride.
4. **Urine analysis:** The concentration of alcohol in urine is 20% higher than blood when collected at the same time as urine has 25% more water compare to an equal volume of blood. A ratio of 1.3:1 is acceptable when urine and blood are in equilibrium. The concentration of alcohol in the second sample shows the blood level during the inter-specimen interval. When there is difference in alcohol concentration of the two urine samples, it indicates whether the patient

was in absorptive phase, at peak or in elimination phase. An approximate value of blood alcohol level at the time when this sample was being secreted can be obtained by multiplying the alcohol concentration in second urine sample by 0.75. This calculation is based on a blood urine alcohol concentration ratio of 1:1.35. The extent of intoxication at the time of incident can be detected by extrapolation from this blood level back to the time of incident. For urine analysis 2 samples should be obtained; first as soon as following the incident, completely emptying the bladder and second after a period of 25-30 minutes. The disadvantages of urine examination are (i) maximum concentration in urine reaches 20-25 minutes later than the blood as a time lag before equilibrium is reached between blood and urine (ii) after the maximum concentration of alcohol in blood has been reached, the urine alcohol concentration will be higher by 20-30% than in the blood at any given time as the sample of urine examined will have been secreted at some earlier period from the blood.

Widmark formula: This formula was devised taking into consideration the weight and sex of the person and type of alcoholic liquor consumed. For blood alcohol concentration, the formula is: $a = prc$, where

a = weight of alcohol (gm) in the body,

p = body weight (kg)

c = concentration of alcohol in blood (mg/kg)

r = constant (0.6 for males and 0.5 for females)

For urine analysis, widmark formula is: $a = 3/4 prq$, where

a = weight of alcohol (gm) in the body,

p = body weight (kg)

q = urine alcohol concentration (mg/kg)

r = constant

Postmortem Alcohol

After death, alcohol diffuses through the stomach wall in to surrounding blood, cavities (pleural, pericardial and peritoneal) and tissues. Therefore, it is important that for chemical analysis for alcohol, the blood sample should not be drawn from the areas in vicinity of the stomach. This

would result false high results due to gastric alcohol diffusion after death. If death has resulted from head injury due to trauma, subdural hematoma should be preserved for analysis, as it will contain the same concentration of alcohol as that in the blood at the time of when incidence has occurred. After death, no appreciable loss of alcohol takes place by evaporation from body or blood. In conditions of advanced putrefaction as the entire contents of vascular compartment including the alcohol are destroyed, the brain should be preserved for analysis. Alcohols including ethyl alcohol can be produced due to the action of enzymes, bacteria such as *E.coli*, yeast or fungi on the carbohydrates and proteins in putrefied bodies. This production increases with the longer time interval after death and when the temperature of surroundings is raised. The concentration of alcohol in stages of advanced putrefaction could be as high as 0.2%. When postmortem levels of alcohol are less than 0.2%, it could be attributed to the process of putrefaction. There are circumstances when alcohol is found in blood and visceral organs but not in urine, this could be due to putrefaction. At autopsy, the blood samples should be collected from femoral vein, cubital vein or tibial vein. Postmortem blood alcohol concentrations are valid for a period of 36 hours after death. On analysis, if the person has consumed alcohol, concentration will be same in all these samples. The changes in concentration of alcohol cannot occur at exactly the same level in all the samples if it is due to bacterial and enzymatic putrefaction. The results could be erroneous due to hemolysis, diffusion from body fluids or tissues, collection of a wrong sample from the wrong site and also due to effect of putrefaction. Postmortem examination of urine sample is quite reliable qualitative index of antemortem intoxication than blood. If the urine alcohol concentration after death exceeds by more than 25% than that of blood indicates a higher blood alcohol level during life. When the post-mortem urine alcohol concentration is equal to or less than the blood alcohol level after death, it can be suggested that the individual was in absorptive face at the time of death and about two hours could have passed after consumption of his last drink.

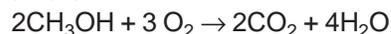
METHYL ALCOHOL

Methyl alcohol is prepared by destructive distillation of wood or molasses. When pure it is colourless, tasteless and odourless. It burns with pale blue non-luminous flame and its vapour produces explosive mixture in presence of oxygen or air.

Methanol, also known as methyl alcohol or wood alcohol, is a chemical compound with chemical formula H_3OH . It is the simplest alcohol, and is a light, volatile, colourless, tasteless, flammable, poisonous liquid with a very faint odor. It is used as an antifreeze, solvent, solvent, fuel and as a denaturant for ethyl alcohol.

Methanol is produced naturally in the anaerobic metabolism of many varieties of bacteria. As a result, there is a small fraction of methanol vapor in the atmosphere. Over the course of several days, atmospheric methanol is oxidized by oxygen by the help of sunlight to carbon dioxide and water.

Methanol burns in air forming carbon dioxide and water:



A methanol flame is almost colorless. Care should be exercised around burning methanol to avoid burning oneself on the almost invisible fire.

Because of its poisonous properties, methanol is also used as a denaturant for ethanol. Methanol is often called wood alcohol because it was once produced chiefly as a byproduct of the destructive distillation of wood. It is now produced synthetically by the direct combination of hydrogen and carbon monoxide gases, heated under pressure in the presence of a catalyst.

History: In their embalming process, the ancient Egyptians used a mixture of substances, including methanol, which they obtained from the pyrolysis of wood. Pure methanol, however, was first isolated in 1661 by Robert Boyle, who called it *spirit of box*, because he produced it via the distillation of boxwood. It later became known as *pyroxylic spirit*

Uses of Methyl Alcohol: (i) It is used as a solvent for gums, fats and varnishes for cinematograph films and as an antifreeze (ii) Methanol is used on a limited basis to fuel internal combustion engines, mainly by virtue of the fact that it is not nearly as

flammable as gasoline (iii) It is used for denaturation of ethyl alcohol (iv) Industrial methylated spirit contains 95% by volume of ethyl alcohol and 5% by volume of wood naphtha, 0.5% of crude pyridine. Methyl violet is added for colouring. It has disagreeable odour that is used for fouling rectified spirit, rendering it unfit for drinking. Nevertheless it is commonly consumed owing to its low cost and easy availability (v) Surgical spirit contains industrial methylated spirit with methyl salicylate or mineral naphtha and ethyl phthalate (vi) Methanol is also used as a solvent and as an antifreeze in pipelines.

Toxicity: Methanol is toxic, as its metabolites formic acid and formaldehyde cause blindness and death. The toxicity results by: (i) Ingestion (ii) Inhalation (iii) Absorption through the skin. Fetal tissue will not tolerate methanol. Dangerous doses will build up if a person is regularly exposed to fumes or handles liquid without skin protection.

Mode of action: Methyl alcohol is rapidly absorbed from the gastrointestinal tract and also through lungs and skin. It gets oxidised into formic acid and formaldehyde along with the production of large amount of lactic acid and other organic acids, producing severe degree of metabolic acidosis and widespread degenerative changes in the internal organs and retina. It is slowly metabolised in the body but its rate of elimination is very slow that is why it tends to accumulate in the blood. It concentrates in the vitreous body and optic nerve maximally. Ethyl alcohol counteracts the action of methyl alcohol from its intracellular attachments thus checking formation to formic acid.

Fatal dose→100–125mL (4 fl oz); is quite variable as recovery has even occurred with large doses

Fatal period→24-36 hours.

Signs and Symptoms

The effect of methyl alcohol on central nervous system is more intense and persistent than ethyl alcohol. The onset of clinical symptoms may be prolonged for 24-36 hours: (i) Headache, dizziness and vertigo (ii) Nausea, vomiting and severe epigastric pain (iii) Rise of serum amylase may result in acute abdominal pain due to pancreatitis

(iv) Cardiac functions are depressed (v) Muscular weakness, dyspnoea and cyanosis (vi) Visual disturbances: (a) Concentric diminution of visual field for colour (b) Photophobia (c) Blurred vision (d) Optic neuritis resulting in partial blindness: These are the most dangerous effect of methyl alcohol and are due to specific toxic effect of formaldehyde on retinal cells. The primary retinal damage is in the ganglion cells and optic atrophy is secondary (vii) Cold sweating and convulsions (viii) Subnormal temperature (ix) Delirium and coma may last for 2-3 days (x) Bradycardia and Kussmaul breathing (xi) Urine is strongly acid and contains albumen, acetone and formic acid (xii) Coma follows unconsciousness: coma induced is longer in onset and lasts longer compared to ethyl alcohol drunkenness (xii) Death from cardiac failure

Treatment

Correction of acidosis is the mainstay of treatment of methyl alcohol poisoning: (i) Gastric lavage using 4% sodabarbonate solution in warm water and 500mL of this solution is left in the stomach (ii) 5% dextrose saline i.v and molar sodium lactate solution are helpful in causing diuresis (iii) To control acidosis, sodium bicarbonate solution 1.2 gms orally every 15 minutes or through stomach tube if patient is unconscious. this can be repeated till the plasma bicarbonate level does not exceed 20 meq / l or hypokalemia is not induced (iv) Renal dialysis or haemodialysis (v) Stimulants and steroids (vi) Oxygen inhalation and artificial respiration (vii) Ethyl alcohol 50% 60mL initially and 10-15 mL per hour subsequently orally for 4-6 days because ethyl alcohol helps to reduce metabolism of methyl alcohol to formaldehyde and formic acid (viii) Barbiturates to treat convulsions.

Autopsy Findings

(i) Stomach and intestines are hyperaemic, congested and inflamed with patchy submucous haemorrhages (ii) Fat necrosis in the peritoneum (iii) Brain and meninges are congested and oedematous (iv) Lungs and Kidneys are congested and oedematous (v) Pancreas may show necrosis and haemorrhage (vi) The eyes will show degene-

rative changes in the corneal epithelium, retina will show oedema of the optic disc and there may be optic nerve atrophy.

Medicolegal Aspects

(i) The poisoning is always accidental (ii) Mass poisoning may result from consumption of liquor adulterated with methyl alcohol (iii) It is also used inadvertently, when ethyl alcohol is not available (vi) Consumption of *solox* (containing shellac, a paint solvent with ethyl alcohol and methyl alcohol) or *Khopadi*, a cheap liquor containing methyl alcohol and French polish or similar intoxicating products have given rise to methyl alcohol intoxication in various parts of the country.

OPIUM (PAPAVER SOMNIFERUM)

In Greek, Opium is the name for juice; the opium poppy originated in the eastern Mediterranean and its use has been well documented by ancient civilisations. In particular, many famous Greeks espoused opium's therapeutic properties to elevated mood and work as a general tonic. Opium was originally used in its raw state; it was first prepared in the 15th century by Paracelsus, who mixed it with alcohol to produce laudanum tincture. Later in the 18th century, **Le Mort** combined opium with camphor to produce paregoric, which was used to treat diarrhoea. It wasn't until 1803 that the alkaloid morphine was isolated from opium by a German apothecary called **Serturmer** who named the alkaloid morphine after Morpheus, the god of dreams. By the 19th century, various derivatives of opium, such as codeine came into use. In 1874 diacetylmorphine, commonly known as heroin was discovered. Heroin a semi-synthetic derivative of morphine was first made in London and was initially used to treat morphine addiction.

Papaver Somniferum grows throughout India but its cultivation is banned except on license obtained from the central government, for growing the plant strictly for the purpose of pharmaceutical industry. Government Opium factory is situated at Gazipur.

(i) Opium is the dried juice of Papaver somniferum, and dry capsule is used as a sedative fomentation and poultice (Fig. 40.1) (ii) Opium



Figure 40.1:
Opium plant with capsule

when fresh is plastic and internally moist, coarsely granular or smooth. It is reddish brown to dark brown, hard and brittle (iii) *Post ka doda* contains 0.1-0.3% morphine. The morphine content of the opium varies from 9-14 %, but when the morphine content is 10 % it is known as standard opium (iv) *Poppy seeds (Khus khus)*: These are white, harmless demulcent, nutritive and used as food (v) *Poppy seeds oil*: It is used for lightening purposes and culinary.

Opioid Antagonists

Opiates may be classified as pure agonists and those with mixed agonistic and antagonistic actions. Opiates undergo oxidative metabolism, catalysed by cytochromes P-450 of which there are several hundred isoforms. Cytochromes P-450 requires the presence of molecular oxygen, NADPH cytochrome. The concept of antagonism of the actions of Opioid is very complex. There are probably several subspecies of Opioid receptor, each with its own self of affinities for exogenous drugs and endogenous ligands and each apparently mediating different effect, when activated. There is no theoretical reason to expect that any substance would have identical activity at those various binding sites. Naloxone comes closest being a relatively pure competitive antagonist.

Martin and Gilbert in 1976, 77 have postulated three subspecies of Opioid receptors:

- I. μ receptors \rightarrow They are responsible for supraspinal analgesia and euphoric effects of opiates. They also cause respiratory depression, gastrointestinal dysmotility and miosis as well as physical dependence.

- II. κ - receptors: These are responsible for supra spinal analgesia, miosis and dysphoria
- III. α - receptors: These are responsible of CNS stimulation.

Opium alkaloids: There are two groups of opium alkaloids; Phenanthrene and Isoquinoline. The phenanthrene group has following derivatives (i) Morphine—9-14% (ii) Codeine—2-4% (iii) Thebaine—0.5%. The derivatives of isoquinoline group are (i) Papaverine—1% (ii) Narcotine—6%.

Fatal dose of various opiates

Crude opium	2g
Morphine	200mg
Codeine	800mg
Heroin	50mg
Pethidine	1gm
Methadone	100mg
Pentazocine	300mg
Propoxyphen	1gm
Diphenoxylate	200mg

Actions of Morphine

Morphine—The word is derived from the Greek word *Morphens* meaning 'Greek God of dreams'. (i) Initially, morphine's analgesic effects were considered that they were the result of their ability to affect emotional responses to pain rather than to act directly on the transmission of pain (ii) More recently, there is a belief that there are naturally occurring specific opiate receptors in CNS (iii) The uptake of morphine in the spinal cord occurs only in substantia gelatinosa in the brainstem. This is a specific site of uptake, with opiate receptors found in solitary nuclei that receive afferent vagal fibres and in the area postrema, which contains chemoreceptor trigger zone (CTZ), the structure that is responsible for the mediation of respiration, cough, postural hypotension and nausea (iv) It is the morphine's actions at CTZ that perhaps explains the common side effects of opiates, and indicates the main site where toxic levels of the drug will lead to death (v) In the cortex, opiate receptors are most densely populated within the medial thalamic nuclei, the periaqueductal grey matter and most importantly the amygdaloid nuclei. It is at these sites within the cortex that addiction is thought to be mediated (vi) Morphine

is an agonist that: (a) Depresses the cortex, respiratory and cough center is stimulated (b) Relieves the pain by causing depression of cortex (c) Exerts euphoric effect (d) The vagal reflex and vomiting is stimulated (e) Spinal reflexes are also stimulated. Codeine is slightly less depressant to the cortex and medullary centers but stimulates the spinal cord.

Signs and Symptoms of Acute Morphine Poisoning

Stage of excitement: (i) There is sense of well being, restlessness, and hallucinations (ii) There is flushing of face, raised pulse when taken for a short period (iii) When large doses are taken, these symptoms may be absent (iv) In children, convulsions are seen and the adults are widely excited.

Stage of stupor: Nerve centers are depressed at this stage and the following symptoms are produced: (i) Headache, nausea and vomiting (ii) Giddiness and uncontrollable desire to sleep (iii) Patient cannot be aroused by external stimuli (iv) Pupils are contracted and conjunctiva are congested (v) Cyanosis (vi) Itching sensation (vii) Pulse and respiration are normal.

Stage of narcosis: (i) The patient is in deep coma (ii) The muscles are relaxed and reflexes lost (iii) Secretions are suspected except skin (iv) The face is pale and lips are livid (v) Lower jaw drops (vi) The pupils are contracted and then dilated in terminal asphyxia (vii) Blood Pressure is low (viii) Respiratory rate is 2-4/min leading to chyne stokes breathing.

Unusual symptoms: (i) Vomiting (ii) Purging (iii) Convulsions (vi) Temperature is raised (v) Pupils are dilated (vi) Syncope occurs after subcutaneous injection.

Differential diagnosis: (i) *Apoplexy:* in apoplexy, temperature is 103°F-104°F (ii) *Uraemic coma:* In Kidney diseases, presence of casts and proteins in the urine (iii) *Epileptic coma:* The pupils are dilated and convulsions are seen (iv) *Hysterical coma:* Is seen in the females, the tongue is not bitten and the reflexes remain unchanged (v) *Acute alcoholic poisoning:* The face is congested and

the pupils are dilated, and the smell of alcohol is there. The paralysis of muscles does not occur (vi) *Carbolic acid poisoning*: White patches are seen on the angles of mouth. Carboluria—Urine becomes dark or olive green on exposure to air due to the reduction of hydroquinone and pyrocatechol.

Treatment

(i) Gastric lavage using plain water or potassium permanganate (ii) Tannic acid and charcoal should be administered (iii) Maintenance of patent airways with assisted ventilation (iv) Magnesium sulphate 15gm for enema (v) Physostigmine salicylate can be given 0.04mg/kg intravenously as it reverts symptoms of respiratory depression but it has an adverse effect of affecting the reticular system of the brain (vi) Atropine is not to be given (vii) Amphetamine, caffeine and coramine are to be administered (viii) Nalorphine hydrochloride 10 mg i.v to be given, a total of 40 mg should be given in 4 hours (ix) Naloxone hydrochloride is antidote for opiate poisoning administered in the dose of 0.01-0.02 mg/kg i.v; maximum up to 300 mg (x) Intravenous fluids to maintain fluid and electrolyte imbalance.

Autopsy Findings

(i) Evidence of drug abuse in the form of injection prick marks, abscesses and old scars can be there (ii) Signs of asphyxia are present (iii) Froth comes out from the mouth and nostrils (iv) Blood is dark and fluid (v) In the stomach contents, lumps of opium may be present (vi) Lungs are congested (vii) Bladder is full of urine.

Medicolegal Aspects

(i) Opium is the commonest drug used for suicidal purposes. The absorptive power increases when it is mixed with mustard oil (ii) Due to its colour and smell, it is rarely used for homicidal poisoning (iii) To poison the cattle it is mixed with *Gur*. (iv) It withstands putrefaction and can be detected after a long time (v) Accidental poisoning can occur at times when the labourers give opium to their children while going for work. At times there can be an overdose leading to accidental poisoning.

Prolonged Use of the Opium

The prolonged use of the drug produces the following effects: (i) Emaciation (ii) Dry skin and hair (iii) Pigmentation around the mouth and eyelids (iv) Dry and furred tongue (v) Anorexia (vi) Nausea and marked constipation (vii) Impotence (viii) Neurasthenia (ix) Dementia or mania

Withdrawal Symptoms

The following are the withdrawal symptoms when opium ingestion is stopped:

1st few hrs	: Psychological effects due to fear, anxiety
8-16 hrs	: Nervousness, restlessness, anxiety
14 hrs	: Yawning, sweating and running of eyes and nose
24 hrs	: Dilated pupils, goose skin and shivering
36 hrs	: Severe twitching of muscles, painful cramps in legs and abdomen, vomiting, diarrhoea, insomnia
3-4 days	: The symptoms gradually sub sides

Treatment of Withdrawal Symptoms

1. Opiates should be gradually withdrawn
2. Cyclazocin 4mg to be given daily
3. Methadone 100mg daily orally (1 mg Methadone = 4mg morphine = 20mg pethidine).
 - i. **Methadone maintenance**: It is usually used for intravenous heroin users. Methadone maintenance units differ in their selection procedures, dose, duration of treatment, their philosophy and rules as well as ancillary methods of treatment
It is the treatment of first choice and among other methods it enjoys a high retention rate of clients due to its effectiveness.
Methadone was synthesized by a German scientist and came to be brought to clinical use at the end of World War II. Methadone hydrochloride is a bitter white powder, that is soluble in water and ethanol. Under the trade names as 'Dolophine' and 'Westadone' It is available as 5mg and 10mg tablets for oral use and for 10mg/ml injections can be given intravenously
After subcutaneous injection, methadone can be found in the plasma within 10 minutes and after oral ingestion, is found after 30

minutes. The peak concentration occurs within 4 hours whereas in the brain, the peak is in 1-2 hours. Methadone is an antidote of morphine but due to its own depressive action on respiration and other side effects has made it unsuitable for the purpose. It is a synthetic opiate and is a narcotic agent. After use of methadone, patient should take bed rest. Movement precipitates dangerous side effects.

Side effects of methadone are excessive sweating, Lymphocytosis and increased prolactin, albumin and globulin in the plasma. In urine, the metabolites of methadone are excreted are Pyrolidine and Pyroline.

- ii. **Clonidine:** A centrally acting α -Adrenergic agonist can suppress the Opioid withdrawal from low to moderate doses of methadone. It is a potent antihypertensive.
- iii. **Propranolol:** 80 mg effectively relieves anxiety and craving associated with opiate addiction
- iv. **Nalorphine (Nalline/N-Allyl-normorphine):** It is a semisynthetic opiate. It causes analgesia, respiratory depression, dysphoria and hallucination. It was popularly used as antidote of morphine. But its own depressive action on respiration and other side effects has made it unsuitable for the purpose. Nalorphine hydrochloride 5mg i.v is given for 15-30 minutes, then 10mg i.v to a total dose of 40 mg I.V.
- v. **Naloxone Hydrochloride:** 0.4-0.8mg i.m/ I.V. is given in adults and 0.01mg/kg is given in children.
- vi. Psychological counselling of the patient has an important role.

HEROIN

Heroin is diacetylmorphine and is a semisynthetic preparation of morphine. It was first prepared at St. Mary's Hospital, London by acetylation of morphine with acetic anhydride in 1898. Morphine was first used for the treatment of cough but soon it became the most potent drug used by drug addicts due to its habit-forming properties. Manufacture of Heroin and its use in Medicine is

banned now a days but it is being illegally manufactured and smuggled, as its cost is very high in international market (1Kg of morphine costs about one crore). This is quite profitable to India as it lies in the 'golden triangle' on eastern side and 'golden crescent' on the Western side. India is one of the countries for transit of morphine and other drugs. Cultivation of opium and production of Heroin is done in 'golden quadrangle' namely Varanasi, Lucknow, Bareilly and Badain district of U.P, and adjoining states of Rajasthan and Madhya Pradesh. About 10kg of crude opium give 1kg of standard opium. This can be converted in to Chinese Heroin (called No.4 Heroin) and Brown sugar (called No.3 Heroin—60% pure) that is available in India. Brown sugar is also called smack and its colour is brown due to presence of sugar of milk, starch, powdered coffee, tea, and coco or brick powder. The smack of 'golden crescent' is grayish and that used of 'golden quadrangle' is blackish-brown in colour. Brown sugar is sold in small cellophane packets of 116gms each. The amount of Heroin in each packet may be 1/16th, 1/18th, or 1/2 of a gram. These cellophane bags are kept in a matchbox to avoid suspicion.

Routes of Consumption

(i) Sniffing in the form of snuff (ii) Smoking in cigarettes or *bidis* after removal of some tobacco (iii) Chasing (a cigarette foil or RS. 5 or 10 note is folded and instead of tobacco, the drug is put inside, this is then lighted using match stick, when thick smoke come out it is inhaled—called 'Dragon chasing') (iv) Injections by s.c (*skin poppers*) and I.V. route (*main-liners*)

Signs and Symptoms

(i) Anorexia (ii) Nausea and vomiting (iii) Constipation (iv) Emaciation (v) pigmentation around cheek (vi) Impotency.

Complication of Consumption

(i) Abscess formation along the routes of injection (ii) Formation of pigmented scars in cubital fossa described as 'rail-road track' (iii) Thromosed veins

(iv) Septicemia (v) AIDS and Hepatitis.

PETHIDINE (MEPERIDINE, DEMEROL)

It is a synthetic opiate and is a narcotic. Some of its actions are in variation with morphine. It is a good analgesic and sedative. In contrast with the action of morphine it is mydriatic. It causes dryness of the skin. It has direct action on the heart musculature which is inhibits. It liberates histamine from mast cells. Pethidine causes early loss of corneal reflex, due to its anaesthetic effect on the corneas. It is highly addictive agent and doctors and paramedical workers are the common victims. Pethidine is therapeutically contra-indicated in cases of high intracranial pressure. It is also not given in toxemia or pregnancy.

BARBITURATES

Since the synthesis of diethylbarbituric acid and its introduction into medicine as a sedative and hypnotic agent many other structural analogues have been prepared and investigated. Fisher & Von Mering introduced Diethyl Barbituric acid in to medicine in 1904 under the name of *Venoral* (vera means true) that proved to be an effective hypnotic without serious side reactions.

The barbiturates were used as sleeping tablets and sedatives that led to their widespread abuse till 1960s when they were replaced by nonbarbiturate hypnotics, such as the benzodiazepines. Unfortunately, barbiturates are still available in the market either alone or in combination with other substances such as amphetamines.

Classification of Barbiturates

The classification of barbiturates into short, intermediate and long acting is arbitrary and may be misleading. The onset of action is about $\frac{1}{4}$ – $\frac{1}{2}$ hour but the duration varies up to 8 hours or so for Phenobarbitone (Table 40.2).

Fatal period→1-2 days.

Pharmacological Action of Barbiturates

Pharmacological action of barbiturates of all types is the same that is depression of central nervous system. However, the structural variations result

Table 40.2: Classification of Barbiturates

Barbiturates	Duration of action	Fatal dose
<i>Long acting</i>	8-16 hrs	3-4 gm
• Barbitone (<i>venoral</i>), white tab		
• Barbitone sodium (<i>medinal</i>), white tab		
• Phenobarbitone (<i>gardenal</i> , <i>luminal</i>), white tab		
• Methyl Phenobarbitone (<i>prominal</i>)		
<i>Intermediate acting</i>	4-8 hrs	2-3 gm
• Allobarbitone		
• Amylobarbitone		
• Butobarbitone (<i>Soneryl</i>), pink tab		
• Pentobarbitone (<i>Nembutal</i>), yellow tab		
<i>Short acting</i>	3-6 hrs	1-2 gm
• Cyclobarbitone (<i>phanoderm</i>), white tab		
• Hexobarbitone (<i>evipan</i>), white tab		
• Quinalbarbitone (<i>secoral</i>), red tab		
<i>Ultra short acting</i>	1-few min.	1.5-2 gm
• Thiopentone (<i>pentothal</i>), white powders or ampoules		
• Metho- hexo- barbitone (<i>brevital</i>)		

in differences in rate of absorption and distribution. By altering the dose, the degree of depression can be altered. Phenobarbitone has a specific depressant action on cerebral motor cortex that makes it a valuable drug for epilepsy. In hypnotic doses, it has no analgesic action and if prescribed alone in painful conditions, there may be excitement, restlessness, mental confusion and delirium. Barbiturates act as synergists with analgesics and potentiate the action of alcohol.

As barbiturates are cumulative drugs, they are contraindicated in hepatic and renal disorders. After the oral ingestion, the peak concentration in blood and brain of

1. Medium and short acting barbiturates is after →1-2hours
2. Long acting is after→4-8hrs after ingestion
3. Ultra short acting is after→30seconds to a few minutes

They are rapidly absorbed from the gastrointestinal tract including rectum and from the subcutaneous tissues. They are concentrated in the liver for a short time and evenly distributed in the body fluids. Lipid solubility is the primary determinant of binding. Ultra short acting is 80% more bound to plasma proteins or stored in the body fat from which they are subsequently cleared and degraded in the liver. Long acting is only 5% bound to plasma proteins and mainly cleared by urinary excretion. Medium acting are bound more than the long acting and they are detoxified in liver and tissues and finally excreted quickly. Short acting barbiturates are also excreted quickly.

Highly lipid soluble barbiturates such as thiopental, methohexal undergo rapidly to the vascular areas of brain and first to the gray matter. Maximum uptake occurs within 30 seconds and sleep may be induced within few minutes. Within 30 minutes, there is redistribution to the less vascular areas of the brain and other tissues. Other factors that affect the binding capacity are the dose absorbed and the patient's habit

Excretion of barbiturates: The Barbiturates are removed from the body by two different mechanisms: (i) Long acting Barbiturates are mainly excreted by the kidneys. As much as 85% of these compounds may be recovered from the urine. Excretion is slow and takes place in several days (ii) The liver mainly metabolizes short acting Barbiturates. Other tissues may also participate in this process. These compounds are not recovered from the urine if taken in sedative doses.

Mode of action: Barbiturates are cellular, histotoxic agents. They (i) Produce histotoxic or tissue anoxia (ii) Partially inhibit the cytochrome enzyme system (iii) Toxic action may occur following a large single dose or repeated medication in slow excreting Barbiturates.

Signs and Symptoms

The clinical staging of barbiturates has been classified in to five stages by 'Sunshine and Hackett': (i) Awake, competent and normally sedated (ii) Sedated: (a) reflexes are positive (b) prefers

sleep (c) answers question when aroused (d) does not cerebrate properly (iii) Comatose with positive reflexes (iv) Comatose and areflexia (v) Comatose; difficulty in respiration and circulation and death is from respiratory failure.

Central nervous system: (i) Drowsiness (ii) Transient period of confusion, excitement, delirium and hallucinations (iii) Ataxia, vertigo and slurring sleep (iv) Headache (v) Consciousness is depressed in a variable degree; can be assessed by rubbing the clenched fist on patient's sternum. The response of the pupil and peripheral nerve reflexes are so erratic that it has a little help in assessing. (Pupils are constricted than dilated in terminal hypoxia) (vi) "Rising up" sign (vii) Babinski sign is positive (viii) The degree of consciousness can be expressed by the following classification

Grade I: Response to vocal commands

Grade II: Maximum response to minimal painful stimuli

Grade III: Minimal pain to maximum stimuli

Grade IV: Total unresponsiveness to maximal painful stimuli

Respiratory system: They directly depress the medullary centers. The rate and depth of respiration is reduced and towards the end Cheyne-Stoke's type of breathing and then death. If coma continues then infection, pneumonia and pulmonary oedema may develop

Cardiovascular system: Barbiturates exert direct toxic effect on the myocardium and interfere with the myogenic tone of peripheral arterioles (i) Fall in cardiac output (ii) Permeability of arterioles is increased leading to transudation and increase in extracellular fluid volume (iii) Cyanosis and hypotension (iv) Weak and rapid pulse (v) Cold and clammy skin.

Pupils: Pupils are little contracted and reacting to light; may be dilated and unequal in terminal asphyxia.

Body temperature: Barbiturates interfere with the control of body temperature; hypothermia is produced requiring the use of symptomatic measures. During recovery the patient may

become febrile.

Gastro-intestinal tract: Bowel sounds may be absent during severe poisoning and this is a bad sign. When bowel functions return further drug absorption may take place leading to fluctuating levels of consciousness. Incontinence of faeces may occur

Renal symptoms: Renal functions may fail especially with hypotension and hypothermia. Incontinence of urine may occur and sugar and albumin may be present in the urine.

Dermatological symptoms: The skin lesions develop in 6% of the cases; are diagnostic and very commonly present in poisoning with medium acting barbiturates. Bullous lesions occur where the skin surface rubs the other part of the skin such as inner aspects of thigh and pressure bearing areas like hands and feet. Initially there are slightly raised areas of erythema and later on bullous eruptions are formed. The blister contains serous fluid, the rupture of which leaves a red raw surface that may be mistaken for burns. It is suggested that bullous formation is either due to toxic effects of the drug or patient is unduly sensitive to the drug itself.

Differential diagnosis of bullous eruptions in Barbiturate poisoning

1. *CO poisoning:* The eruptions are present under the pressure areas, sacrum, spine, inner aspect of knee and ankle. They are produced due to impairment of circulation.
2. *Thermal heat:* The blisters due to burns will show the effect of heat and the hair will be seinged
3. *Pemphigus:* Pemphigus blisters are non-tense, larger and the bullous spreading test is positive
4. *Methaqualone (Mandrax) overdose:* The following symptoms are present; hypoproteinemia, gastric bleeding and cardiac arrhythmias.
5. *Glutathimide, meprobamate and tricyclics antidepressants overdoses*
6. Prolonged contact with petrol and paraffin

Clinical Diagnosis of Barbiturate Poisoning

1. In an unconscious patient, one has to rule out other causes of coma such as: (i) *Acute alcoholic poisoning* (a) odour of alcohol is present in the breathing (b) eyes are congested and pupils dilated (ii) *Carbolic acid poisoning* (a) the odour is characteristic (b) white patches can be seen on lips and mouth (c) carboloria is diagnostic (iii) *Co poisoning* (a) history of exposure to the carbon monoxide gas is there (b) intermittent convulsions (c) cherry red colour of the skin (d) carboxyhaemoglobin is present in the blood (iv) *Epileptic coma* (a) there is history of fits (b) pupils are fixed and dilated: (a) Froth at mouth (b) cyanosis (v) *Diabetic coma:* (a) gradual onset (b) odour of acetone is present (v) Sugar and acetone is present in the urine (vi) *Brain trauma:* (a) history is characteristic (b) injuries and bleeding from the nose (c) pulse is rapid (d) paralysis may be present.
2. Clear history of ingestion of barbiturates
3. Findings of general anaesthesia with low respiration and decreased respiratory function
4. Presence or absence of bowel sounds
5. Urine or first stomach wash is to be tested for barbiturates
6. Blood levels by gas chromatography, calorimetric methods and spectrophotometry are: (a) For long acting—8-10mg% (b) For medium acting—4-7mg% (c) For short acting—2-4mg% (d) For ultra short acting—0.8-1mg%.
7. ECG findings show inverted and flattened 'T' wave and depressed ST segment
8. **EEG Findings in Barbiturate poisoning**
Mild intoxication: Normal activity is replaced by fast activity in the range of 20-30Hz appearing first in the frontal regions and spreading to the parietal and occipital regions as intoxication worsens.
More severe intoxication: The fast waves become less regular and interspersed with 3-4Hz slow activity.
Still more advanced cases : There are short periods of suppression of all activity, separated by bursts of slow (delta) waves of variable

frequency

Extreme overdoses: All electrical activity ceases in extreme of overdosage of the drug. This is one instance in which a flat EEG cannot be equated with brain death and the effects are fully reversible unless anoxic damage has supervened.

Treatment

Mild cases need no treatment but should be kept under observation. Before treatment in comatose patients, other causes of coma should be excluded. The treatment includes the general and specific measures.

General measures:

First part: Clear the airway by tracheo-bronchial suction, oxygen inhalation, physiotherapy of thorax. X-ray of lungs shows evidence of collapse. No prophylactic antibiotics to be given unless infection is present

Second part: Gastric lavage and suction: (i) It is more useful if it is done within 4 hours of the ingestion of the poison (ii) Done with warm water mixed with potassium permanganate and suspension of animal charcoal and tannic acid (iii) First sample is to be obtained in plain water (iv) Magnesium sulphate is used for purgation as it minimizes absorption.

Third part: Regular charting of pulse and blood pressure along: (i) With correction of dehydration (ii) Nor adrenaline 2mg with 500ml of 5% glucose (iii) Intravenous saline drip for correction of shock and hypotension (iv) Patient should be kept warm.

Specific measures: (i) No specific antidote is known (ii) In prolonged coma with retention of carbon dioxide, mechanical respirator and tracheostomy may be considered (iii) Treatment of pulmonary oedema by relieving heart failure by aminophylline, digoxin etc. may be considered. 500 ml of 10% Mannitol should be given I.V. Furosemide is used as diuretic (iv) **Analeptics:** Analeptics stimulate the central nervous system especially respiratory center so they are used in the treatment of narcotic poisoning. Their use is opposed in barbiturate poisoning due to the following measures: (a) Are generally ineffective

in severe poisoning (b) Awakening effect is transient and followed by greater depression (c) Leads to cardiac arrhythmias and convulsions; cerebral ischemia and depression and then irreversible brain damage (d) Overall results without their use show a much reduced mortality rate (e) 10 ml of 0.5% Bemigrade (50 mg) and 1 ml of 1.5% Amiphenazole (15 mg) are added to the 5% glucose saline drip for two hours at 5 minutes interval or till consciousness returns whichever is earlier or return of pharyngeal or laryngeal reflexes. If vomiting and muscular twitching is seen the treatment should be stopped (f) Some use Coramine (Nikethemide) 5ml i.v at 15 minutes interval and then 10ml at 30 minutes interval till reflexes return. If muscle twitching are present stop the treatment. (v) Picrotoxin can be given 2ml intravenously but if muscle twitching are present or corneal reflexes return, stop the treatment (xi) Dialysis and exchange transfusion are at times life saving.

Autopsy Findings

(i) External findings are not characteristic; signs of asphyxia are present and cyanosis of face and nails is seen (ii) Traces of tablets and capsules of barbiturates may be found in the mouth, oesophagus and stomach (iii) Mucous membrane of stomach is congested and eroded badly from the alkaline attack of drugs like sodium amytal which, being the sodium salt of a weak organic acid, hydrolyses in the stomach. The fundus may be thickened, granular and haemorrhagic. The cardia and lower oesophagus may be eroded from the reflux and if the victim regurgitates, then black, altered blood may appear at the nose and mouth (iv) Lungs are congested, oedematous and findings suggestive of pneumonia may be present. The congested lungs in acute barbiturate poisoning are more intense than in any other condition. The lungs are almost black and the whole venous system is engorged with dark, deoxygenated blood (v) Petechial haemorrhages are seen in the pleura, pericardium and meninges (vi) Kidneys are congested and degenerative changes of the tubules are present (vii) Brain is oedematous, softening of globus pallidus is seen and multiple

petechial haemorrhages are seen in the white matter (ix) All other organs are congested (x) **Barbiturates blisters:** These blisters are found on the dependent parts of the skin surface, especially buttocks, backs of thigh, calve and forearms (xi) For chemical analysis of viscera, besides the other organs brain is to be preserved as venoral is retained in the brain.

Medicolegal Aspects

(i) Commonly used for suicide more by young male than female, next only to organophosphorus compounds. It is freely prescribed and easily available (ii) Therapeutic uses: It is used for sleeplessness, anxiety states, epilepsy, strychnine, picrotoxin and cocaine poisoning (iii) Rarely used for homicidal purposes (iv) Repeated small doses cause addiction that leads to withdrawal symptoms, used for the relief of worries and anxiety of modern life (v) **Automatism:** The accidental or suicidal overdose may lead to the poisoning

In ordinary doses, it induces natural sleep but occasionally instead of sleep, there is mental

confusion. It is likely to happen in those cases where insomnia is due to pain and an analgesic is not taken for its relief. In these cases, the use of barbiturates may lead to mental confusion. As a result, to induce pain patient takes more of the drug automatically for getting that he has already taken a dose known as barbiturate automatism. In some cases, the patient continues to take the drug that leads to overdose that is fatal.

However, some workers have a different concept; they feel that confusion and forgetfulness cannot account for overwhelming overdose that is found in these cases. It is possible that there are cases of intentional suicide or alcoholics where confusion is more likely and the action of barbiturates is potentiated by alcohol. It is now over 40 years since 'automatism' was offered as a socially acceptable explanation of self-administered overdoses of barbiturates. The idea was often accepted without any real evidence that repeated therapeutic doses were taken by a patient who did not remember taking the drug and in some studies by Dorpat concluded that drug automatism is a myth.

Spinal Poison

This group of poisons mainly acts on the spinal cord, the cerebral symptoms being either slight or absent. The stimulation of spinal cord results in spasms and convulsions, while depression causes paralysis and loss of sensation. Nux vomica is a spinal stimulant whereas and Gelsemium is spinal depressant.

STRYCHNOUS NUX VOMICA

Strychnous nux vomica is a spinal poison. The plant grows wildy in all parts of India and its all parts are poisonous (Fig. 41.1). The fruits of nux vomica are round, hard, slightly rough and glossy orange in colour. They contain 3-5 seeds and jelly like white pulp. The seeds are hard, flat circular discs with 2 cm diameter and 0.6 cm thickness being slightly convex on one side and concave on other. The seeds are ashy grey or light brown in colour and their shining surfaces are covered with fine, short, silky, yellowish grey hairs. The seeds are very hard and difficult to pulverize. The pulverized strychnine powder is extremely bitter.

Alkaloids in the Seeds

The seeds contain two principal alkaloids: (i) Strychnine (1.3-1.5%) occurs as a powerful alka-



Figure 41.1: Nux vomica seeds

loid in the dried ripe seeds of the plant *Strychnos nux vomica* and *Strychnos ignatii*, (ii) Brucine (1.55%). The pulp of the fruit has very low strychnine content whereas the bark, leaves and wood of the plant contains only Brucine. Brucine is similar in action to strychnine both chemically and physiologically but strychnine is 10-20 times more potent than brucine in its toxicity.

Circumstances of Poisoning

(i) Accidental poisoning results from (a) ingestion of tablets by the children kept for use by the parents (b) wrongly prescribed for quinine (c) is prescribed as tonic (ii) Suicidal poisoning results when pharmacists or agricultural workers who have access to the drug may ingest it. In a case, a doctor injected himself of *Strychnos hydrochloride*, went upstairs, told his wife about it, and then died (iii) Homicidal poisoning occurs when the crushed seeds or tablets are mixed with the food to kill the person (iv) In Malaysia it is used as an arrow poison.

Absorption

The poison is absorbed from all the mucous membrane including the stomach and intestines and 20% of its gets eliminated slowly in the urine, some in bile, sweat, saliva and milk. Much of it is impounded either in liver or in the muscles. This release of the impounded strychnine in liver and muscles is either to be released again to the blood or to be destroyed. The release of strychnine results in producing convulsions on the second or third day.

Mechanism of Action

Strychnos nux vomica depresses the inhibitory postsynaptic potentials in the spinal cord and prevents the effects of glycine, the presumed inhibitory transmitter resulting in widespread inhibition with release excitation. This results in loss of normal inhibition of motor cell stimulation, so a slight stimulus causes all the muscles to contract simultaneously, producing convulsive seizures. It primarily affects anterior horn cells of the spinal cord with greatly increased reflex excitability. It has a little stimulant effect on the cerebral cortex as patient remains conscious till the end. Death occurs from asphyxia due to spasm of muscles of respiration or from collapse occurring in the interval between spasms.

Fatal dose→Strychnine: 30-120mg

One powdered nux vomica seed—2gms of alkaloid

Extract of nux vomica powder—200mg

Fatal period→1-2 hours.

Signs and Symptoms

Uncrushed seeds have no effect and they are passed intact with the faeces. The signs and symptoms appear within 5-10minutes of ingestion of powdered seeds and half an hour of ingestion of crushed seeds: (i) Intense bitter taste in the mouth when ingested in powdered form (ii) There is difficulty in swallowing (iii) Before convulsions, there is increased activity of perception, increased rigidity of muscles and twitching (iv) Convulsions are due to the action on reflex centers of spinal cord and affect all the muscles at a time. They are clonic in nature to start with but become tonic very soon with paroxysms getting longer and interparoxysmal intervals becoming shorter (v) Violent convulsive seizures occur involving all the muscles of the body all at a time. As a result, the antigravity muscles become markedly stiff and rigid, when the body is thrown into an arch, head bent backwards, arms being either flexed on the chest or stretched out extended and the hands tightly clenched; ultimately the body may assume the position of **Opisthotonus** (being arched on the back with the back of the head and heels touching the ground in a bow like position) the

head hyperextended, feet arched and inverted, legs extended and slightly thrown apart and abdominal muscles spasmodically fixed, resulting in serious interference with respiratory movements.

Emprosthotonus that is forward bending of the body due to violent spasmodic contractions of abdominal muscles. The word *emprosthotonus* has been derived from the word emperor whom the people used to salute by bending forward.

Pleurosthotonus that is lateral bending of the body (vi) Convulsions lasts for 1-2 minutes and occur after an interval of 10-15 minutes. Between convulsions, the muscles are completely relaxed and the patient looks well, though exhausted and looks cyanosed (vii) Face is cyanosed and bears an anxious look (viii) Eyeballs are prominent with dilated pupil (ix) **Risus sardonicus**—The corners of mouth draw back due to the contraction of jaws and facial muscles and the mouth is covered with froth that is frequently blood stained. Antigravity muscles are affected (x) Consciousness remains clear until the death (xi) Death usually occurs after 4-5 convulsions due to asphyxia and exhaustion. In nonfatal cases, convulsions become less severe and recovery occurs within 1-2 days.

Treatment

(i) To control the convulsions, the patient should be kept in bed in a dark room free from noise and disturbance (ii) Inhalation of anaesthetic is of little value during convulsions as it causes fixation of respiratory muscles so it should be given after convulsions (iii) Chloroform may be given until the patient is unconscious (iv) Short acting Barbiturates like sodium Amytal and pentobarbital, as an antidote in the dose of 300-600 mg should be given intravenously (v) Stomach wash should be done using KMnO_4 (vi) Animal charcoal adsorbs the poison and tannic acid can be given (vii) Mephenesin 3 mg/kg as slow i.v drip is given to control convulsions (viii) Urethane is given in the dose of 5-15 mg (ix) Chloral hydrate and bromides should be given (x) Symptomatic treatment.

Autopsy Findings

(i) Rigor mortis develops earlier and is prolonged (ii) The post mortem findings are not characteristic

(iii) Signs of asphyxia are present (iv) Extravasation of blood is present in the muscles (v) Haemorrhages are seen in the peritoneal coat of the stomach (vi) Internal organs are congested (vii) Resists putrefaction, the poison can be detected in the viscera long time after death.

Medicolegal Aspects

(i) Nux vomica is rarely used for homicidal purposes when the crushed seeds are given in the food (ii) Suicide is also rare as it causes painful death (iii) Accidental death is common resulting from the overdose of medicinal form and sometimes children eat the seeds accidentally.

Diagnosis of Nux Vomica Poisoning

1. *Physiological tests*: Injection of aqueous

solution of suspended material in to the dorsal lymph sac of the Frog results in convulsions within few minutes. Stimulation of Frog will produce convulsions.

2. *Analytical tests*: (i) Thin layer chromatography for qualitative analysis (ii) High pressure liquid chromatography for quantitative analysis.

Differential Diagnosis of Nux Vomica Poisoning

(i) *Tetanus* (ii) *Epilepsy*: This is differentiated by the history and the fits are clonic, there is loss of consciousness and the reflexes are normal (iii) *Meningitis*: Here fever and neck rigidity is present (iv) *Hysteria*: At times the two are difficult to distinguish so its better to treat for the poisoning (Table 41.1).

Table 41.1: Differences between the poisoning by Strychnos Nux vomica and tetanus

Features	Nux vomica poisoning	Tetanus
Onset	Sudden	Gradual
History	No history of injury	History of injury is there
Convulsions	All parts of the body involved at the same time	All parts of the body are not affected at the same time
Nature of paroxysms	Clonic	Tonic
Lower jaw	Does not start from the lower jaw	Usually starts from and specifically affects the lower jaw.
Muscular condition between convulsions	Relaxed	Rigid
Fatal period	1-2 hours	>24 hours
Lab. Analysis	The poison can be detected from the food	No poison is found in the food.
Microscopical and culture examination	Not helpful to diagnose the poison	Cl. tetani bacillus can be demonstrated

Cardiac Poisons

Cardiac poisons have mainly actions on the heart, either directly on the musculature or through its nerve supply. Oleander, nicotine, digitalis and aconite belong to this group

NICOTIANA TABACUM (TOBACCO)

Nicotine is a colourless, hygroscopic liquid alkaloid. It was first given to Columbus by Native Americans that subsequently spread to Europe. However, the use of cigarettes is a modern phenomenon throughout the world.

Uses: Preparations of nicotine are extensively used in agriculture and horticulture for fumigating and spraying, as insecticides, fumigants, vermin paste, worm-powder, sheep dip etc. The dried leaves of the plant Solanaceae comprise of the tobacco used for smoking. These leaves of tobacco (*tambaku*) contain 1-8% of nicotine and are used in the form of smoking, snuff and for chewing. They are used for the preparation of cigars, cigarettes, *bidis* and powdered tobacco is used as a snuff. The tobacco is also consumed by mixing with lime alone (*khaini*) or with lime and betel.

Active principles: All parts of the plant are poisonous except its ripe seeds. The leaves contain two active principles as toxic alkaloids; Nicotine and Nicotianine. Nicotine is very toxic and exists in all parts of the plant, in combination with malic acid and citric acid. It first stimulates the vagus nerve then depresses autonomic, central and spinal nervous system. In small doses, it contracts the pupil but in larger doses, it causes dilatation. The poisoning occurs in persons employed in tobacco extraction, processing or spraying of insecticides containing nicotine.

Absorption and excretion: Concentrated liquid nicotine is exceedingly dangerous. However, tobacco is much less poisonous than would be expected from its nicotine content when smoked. Most of the nicotine is burned and when ingested in the form of tobacco, it is poorly absorbed from the stomach. Nicotine poisoning can occur from ingestion or application over skin and by inhalation. Nicotine in the cigarettes is rapidly absorbed through the arterial circulation to reach the central nervous system directly and the effect occurs within a few minutes. About 85-90% is metabolized in the liver, but some amount is metabolized in the kidneys and lungs. It has a half-life of about 2 hours.

Mechanism of action:(i) Nicotine binds to nicotinic receptors, which are present mainly on the CNS, spinal cord and neuromuscular junctions (ii) It also binds to autonomic nervous system and adrenal medulla. It first stimulates the vagus nerve, and then depresses the autonomic, central and spinal nervous system (iii) In small doses, it contracts pupil but in larger doses, it causes dilatation.

Acute Poisoning: Following signs and symptoms are produced in acute poisoning: (i) Burning acid sensations in the stomach (ii) Salivation, nausea and vomiting and diarrhoea (iii) Headache, Giddiness, numbness and faintness (iv) Muscular weakness and tremors (v) Cold and clammy skin (vi) Hearing and vision may be affected (v) Cardiovascular manifestations include, slow pulse at first and then rapid, Blood Pressure is raised and arrhythmias also develop (viii) Pupils are first contracted then dilated (ix) Respiration is at first rapid then slow (x) Sometimes delirium and

convulsions may occur (xi) Death occurs due to respiratory failure.

Chronic poisoning: (i) Chronic poisoning is produced in heavy smokers and persons working in tobacco factories (ii) Respiratory system is mainly affected; chronic cough, bronchitis and laryngitis are produced due to irritation of pyridine bases (iii) Pulmonary emphysema is quite common in smokers and smoking may lead to lung cancer.

The incidence of following diseases are more in smokers: (i) Coronary heart disease (ii) Oral cancer is directly related to smoking (iii) Hyperacidity and peptic ulcer are produced (iv) Dermatitis may be produced from dust and juice of tobacco leaves (v) Optic atrophy is also commonly observed in the smokers (vi) Hypertension, extrasystoles and ventricular fibrillation are frequent in the smokers (vii) Thromboangiitis obliterans may be present.

Fatal dose→60-100 mg of nicotine

15-30 gm of crude tobacco

Fatal period→5-15 minutes.

Treatment

(i) Gastric lavage with weak solution of potassium permanganate or warm water containing finely powdered charcoal or weak tannic acid solution. (ii) Saline purgation to be given by mouth (iii) In mild to moderate cases, inj atropine sulphate 1-2 mg i.m. and hexamethonium chloride 25-50ng s.c to counteract peripheral autonomic disturbance and it also acts as a respiratory stimulant (iv) Mecylamine (inversine) is the specific antidote. As it is only antidote in tablet form, is of little value if patient is vomiting (v) Vasodilators like nitrites, methacholine can be given for amblyopia (vi) Oxygen inhalation and artificial respiration is recommended.

As nicotine is completely excreted from the body within 16 hours thereafter, there is no danger to life.

Autopsy Findings

(i) Odour of tobacco and its leaves from the stomach contents (ii) Mucous membrane of stomach and intestines is congested and haemorrhagic due to caustic action of nicotine (iii) All the organs are

congested and oedematous (iv) Nicotine resists putrefaction so it can be recovered from the viscera for a long time after death.

Medicolegal Aspects

1. Accidental poisoning is quite common due to its extensive use and usually occurs under the following circumstances: (i) Tobacco is used in different ways like smoking, chewing, swallowing or by decoction. It is also widely used in agricultural and horticultural work (ii) Accidental toxicity occurs when tobacco is applied as a poultice for ulcers or from mucous membranes when used as enema (iii) Oral tobacco use can lead to gum diseases and oral cancer (iv) Poisoning is usually accidental resulting from excessive smoking (v) Application of leaves or juice to the wound or abraded surface may lead to the absorption of the poison (vi) Drinking hookah water and Cheroots in water are applied over the axilla (vii) Malingering by soldiers to avoid duty, they soak the leaves in water and place in axilla with the bandage.
2. Suicidal and homicidal poisoning is rare.

DIGITALIS PURPUREA

The plant is cultivated in whole of Jammu and Kashmir of India and throughout the World. The entire plant of digitalis purpurea is toxic. Only leaves are used for medicinal purposes.

Active principles: The active principles in the form of glycosides are present in the leaves, roots and seeds of the plant. There are about more than thirty glycosides out of which only four are important. Of this digitalin, digitoxin and digitalein are cardiac stimulants whereas digitonin is cardiac depressant. The poisoning by digitalis is mostly accidental as it is used in different types of cardiac diseases and over doses do occur.

Mode of action: The main action of the poison is on the heart due to its glycosides content. In the heart: (i) It prolongs the period of systole in cardiac cycle (ii) It regulates the rhythm of the heart by depressing both excitability and conductivity (iii) It shortens the period of diastole (iv) It causes rise of blood pressure (v) In toxic doses, there is increa-

sed excitability of the heart with development of extra-systole, ultimately toxic arrhythmias and bradycardia develops leading to heart failure.

Signs and Symptoms

The poison first acts on the gastro intestinal tract then the cardiovascular system. The signs and symptoms produced are: (i) *Gastrointestinal tract*: Anorexia, thirst, nausea, vomiting, abdominal pain, watery diarrhoea, vertigo and fatigue are produced (ii) *Cardiovascular system*: Extrasystoles, pulsus bigeminus and heart block (iii) *Respiration* is slow and sighing, pupils are dilated and visual derangements are there (iv) *Central nervous system*: Headache, neuromuscular disorders, confusion, disorientation, drowsiness, delirium and hallucinations are present (v) Death is due to ventricular fibrillation.

Tests for the Glycosides

The glycosides can be extracted from the acidified organic matter. When these glycosides are treated with strong sulphuric acid, the reactions of all three are different: (i) Digitoxin will give green colour, which does not change with bromine (ii) Digitalin gives yellow colour that soon changes to brick red and to violet red on addition of bromine water (iii) Digitonin produces red colour and the colour intensifies with addition of bromine water

Fatal dose→ Digitalis—2-3 gm
Digoxin—5 mg
Digitalin—15-20 mg
Powdered leaves—2.5 gm

Fatal period→ 24 hours.

Treatment

(i) Stomach wash using tannic acid and activated charcoal to be given (ii) Bowels should be evacuated (iii) Atropine 0.6mg i.v. is given for bradycardia that can be repeated (iv) Quinidine sulphate 0.2gm is given 4-6hourly (v) Lignocaine 50mg i.v is given for ventricular tachycardia (vi) Digoxin specific antibody fragments (Fab) are given in the dose of 0.4-0.8 mg i.v. slowly (vii) Phenytoin 50 mg i.v / min, can be given upto 1gm followed by 300 mg daily for ventricular fibrillation

(viii) Lignocaine 100 mg i.v. to be given for arrhythmias (ix) Potassium salts are administered to reduce extrasystoles (x) Electrolyte imbalance should be corrected using IV fluids

Autopsy Findings

(i) Fragments of leaves of digitalis may be found in the stomach (ii) Mucous membrane of stomach is congested and inflamed (iii) All organs are congested.

Medicolegal Aspects

(i) Poisoning occurs due to intake of overdose of medicinal preparation accidentally (ii) It is also sometimes used for homicidal purposes (iii) Digitalis being a cumulative poison, accidental death may occur in susceptible persons.

ACONITE (Mitha bish, Mitha zahar, Bikh)

The term aconite is derived either from greek akonitus or from the greek city "acona". Few suggest that the word aconite has come from the hill of aconitus. The plant *Aconite* is grown in garden for its showy flowers in the Himalayan ranges in India. *Aconitum napellus* belongs to the family Ranunculaciae. All parts of nearly all the varieties of the plant are poisonous but the root is chiefly used as poison. There are several species of aconite out of which *aconitum ferox* and *aconitum napellus* are commonly used as poison. They are also known as **Monk's hood, Blue Rocket, Bear's foot Mitha zeher, Bish and Bikh etc.**

The dry root is conical and shows scars of broken rootlets. The upper end of it is usually about 2 cms thick and has a length of usually 8-10 cms. The root is dark brown in colour (Fig. 42.1) that on cut section is whitish and starchy, which, on exposure to air, turns pink. It has to be differen-



Figure 42.1:
Root of aconite

tiated from horseradish root that is long, cylindrical and yellowish white externally and whitish internally that does not change in colour on exposure to air and it has a pungent taste. It has no odour but is sweetish in taste hence the name *Mitha Bish*. On chewing tingling and numbness in the tongue, lips and mouth is felt.

Mode of action: The poison first stimulates the sensory nerve endings, producing tingling sensation then paralyzing them causing numbness. It produces similar effect on the motor and secretory nerve endings, the centres of the medulla and cord, while the higher centres are always left out. The alkaloids depresses the myocardium, smooth muscles and skeletal muscles. Its depressant effect on motor ganglia and cardiac muscles produces heart block. Temperature falls gradually due to its action on thermogenic centre.

Elimination: Aconite is mainly excreted in urine, but traces are excreted in bile, saliva and sweat

Active principles: The alkaloids isolated from the different varieties of the Aconite are: (i) From *Aconite napellus* (a) *Aconitine* (Acetyl-benzoyl-aconine) (b) *Picraconitine* (Benzoyl-aconine) (c) *Aconine* (ii) From *Aconite ferox* (a) *Pseudoaconitine* (Veratroyl-aconine) (iii) From *Aconite charmanthum* (a) *Indaconitine* (iv) From *Aconite spicatum* (a) *Bikhaconitine*

Of all these alkaloids obtained from the Indian varieties of the aconite plant, pseudoaconitine is the most toxic, Indaconitine and Bikhaconitine are less toxic than pseudo-aconitine but a bit more poisonous than Aconitine

Signs and Symptoms

The symptoms of aconite poisoning start immediately after its consumption: (i) Bitter taste in the mouth (ii) Burning pain in the abdomen (iii) Nausea, vomiting and salivation (iv) Diarrhoea is rare (v) Pupils alternately dilate and contract which is known as **hippus**, but at a later stage, they remain dilated. The diplopia is also present (vi) Pulse is slow and irregular (vii) Temperature is subnormal (viii) Consciousness usually remains clear till the end (ix) Death results from ventricular fibrillation.

Tests for diagnosis: Paper chromatography and ultraviolet spectrophotometry helps in analyzing the poison.

Fatal dose→1-2 gm of root; 2-5 mg of aconitine.

Fatal period→1-5 hours.

Treatment

(i) Stomach wash with tannic acid or milk (ii) Maintain body heat (iii) Atropine sulphate 1mg i.v is given for bradycardia (iv) Oxygen inhalation and artificial respiration to be given.

Autopsy Findings

(i) Bronchial tree contains froth and mucous (ii) Mucous membrane of stomach and small intestine may be congested (iii) All organs are congested (iv) It is extremely unstable and is destroyed by putrefaction.

Medicolegal Aspects

(i) As aconite is largely used as medicine, accidental poisoning is common (ii) It may be mistaken for Horse radish shoe (iii) It is given with betel leaf to mask its taste (iv) It is added with liquor to increase its intoxicating effect (v) It is used as arrow poison in hilly areas.

NERIUM ODORUM (White oleander, Sweet scented oleander or Kaner)

The plant grows wildly all over the country. It is also grown in gardens for its beautiful pink and white flowers (Fig. 42.2). The plant has lanceolate leaves and bears two follicled fruit that contains numerous seed. All parts of the plant are poisonous

Uses: (i) Powdered roots are used for the treatment of venereal diseases, epilepsy, malaria, skin diseases and menstrual disorders by the quacks (ii) The powdered roots are also applied as paste to treat ulcerations and cancerous growths (iii) Decoction of the leaves may be applied to reduce swelling of body.

Active principles: The active principles are several cardiac glycosides known as (i) *Nerin* (ii) *Oleandrin* and (iii) *Karabin*



Figure 42.2: Nerium odorum plant

Fatal Dose→ Powdered root—15G-20 G
Neriodorin and karabin—150 mg

Signs and Symptoms

1. *On ingestion:* (i) Peculiar taste in mouth (ii) Nausea, vomiting and profuse salivation (iii) Difficulty in deglutition and articulation (iv) Acute pain in epigastrium associated with diarrhoea (v) Fibrillary twitchings of muscles of upper limbs followed by tetanoid convulsion occurs. It affects nearly all muscles of the body, but may be unilateral. It is accompanied by lock-jaw (vi) Pulse rate is slow, weak and feeble in late stages (vii) Fall of blood pressure and cardiac arrhythmias development respiration is hurried and stertorous with dilated pupil (viii) Exhaustion and drowsiness leading to insensibility, collapse and coma (ix) Death occurs from heart failure.
2. *On inhalation of the smoke from burning plant:* (i) Cough, frothy salivation and dysphagia (ii) Fibrillary twitching of muscles (iii) Pulse is slow, weak and feeble (iv) Blood pressure is low and respiration is hurried and stertorous.

Treatment

- (i) Stomach wash using weak solution of potassium permanganate or tannic acid (ii) Vomiting and diarrhoea should be controlled (iii) Morphine or pethidine 5mg i.m for sedation that will control spasm (iv) Absolute bed rest is a must (v) To treat

cardiac arrhythmias, phenytoin 100-200mg orally or 100 mg i.v every 5 minutes until they are reverted (vi) Symptomatic treatment for maintenance of circulatory collapse.

Autopsy Findings

- (i) Stomach and intestinal mucosa is congested (ii) All the chambers of heart distended with blood (iii) All organs are congested (iv) Subendocardial haemorrhages are pathognomic (v) The poison resists heat so it can even be detected in burnt human remains.

Chemical Tests

Keller's test gives green colour appearing slowly in the acetic acid layer and crimson colour in the sulphuric acid layer when nerium extract obtained by Stass-Otto process is added.

Medicolegal Aspects

- (i) Accidental poisoning can occur from ingestion of powdered root or decoction for treatment or when used as a love-potion (ii) The powdered roots, decoction of leaves or paste of the fruit may be ingested for committing suicide (iii) Cases are on record when it has been used for homicidal purposes (iv) It is commonly used as cattle poison; the paste of powdered root is smeared on a rag that is pushed into anal canal of the animal. It is also mixed with the cattle fodder (v) The paste is commonly smeared on the abortion stick to be used as an abortifacient

CERBERA THEVETIA (Thevetia nerifolia or yellow oleander)

The plant is also known by the names of *Bastard oleander*, *Pila kaner* etc. it belongs to N.O. Apocynaceae, alike *Nerium odorum* and is widely cultivated in gardens in India. The plant has linear lanceolate leaves and bears yellow coloured bell-shaped flowers. The fruit is globular in shape, green and contains a single pale-brown nut that is triangular in shape with a deep groove along the edge, having two cells (kernels), each enclosing a pale yellow cotyledon. All parts of the plant are highly poisonous

Uses: The powdered bark of the plant may be used as an antipyretic like Cinchona in the dose of 125mg; in little bigger dose, it acts as an emetic and purgative and in further bigger dose, it is toxic.

Active principles: The milky juice from nearly all parts of the plant gives out the active principle the are cardiac glycosides. Of these thevetin and thevetoxin can be recovered from the kernels of seeds: (i) *Cerberine*—It is a neurotoxic acts like strychnine (ii) *Thevetin*—It is a powerful cardiac toxin (iii) *Thevetoxin*—Action is similar to digitalis (iv) *Nerifolin*—Action is similar to digitalis.

Fatal dose→Uncertain but death can occur in adults by 8-10 seeds and children by 1-2 seeds.

Fatal period→Uncertain but usually 2-3 hours.

Signs and Symptoms

(i) Burning sensation in the mouth with feeling of dryness (ii) Tingling and numbness of mouth, tongue and throat (iii) Headache, dizziness and faintness (iv) Dilated pupils with indistinct vision (v) Loss of muscular power and have fainting fits (vi) Nausea, vomiting and loose frequent motions due to the irritant action on bowel (vii) Depressant action on cardiac muscles causes slow and irregular pulse rate (viii) In late stages, pulse is weak, feeble, irregular with fall of blood pressure and heart block (ix) Respiration hurried and stertorous (x) Tetanic convulsions may occur (xi) Collapse, Coma and death from peripheral circulatory failure.

Treatment

(i) Stomach wash using weak solution of potassium permanganate or tannic acid followed by animal charcoal suspended in water (ii) Barbiturates to control convulsions (iii) Stimulants, analeptics, digitalis, I.V dextrose saline, noradrenaline and corticosteroids to counteracting cardiac failure (iv) To treat cardiac arrhythmias, phenytoin 100-200mg orally or 100 mg i.v every 5 minutes until they are reverted (vi) Symptomatic treatment for maintenance of peripheral circulatory collapse.

Autopsy Findings

(i) Stomach and upper intestinal mucosa is congested (ii) The fragments of seeds may be

recovered from the stomach contents (iii) All organs are congested (iv) Subendocardial haemorrhages are characteristic

Chemical Tests

As cerberine is easily destroyed by hydrolysis with dilute hydrochloric acid and gastrointestinal secretions, it is rarely detected in the viscera on chemical analysis. Thevetin is resistant to such destruction and is available for extraction in poisoning by yellow oleander. Thevetin contained in the acid ether extract obtained by subjecting the viscera of suspected yellow oleander poisoning to Stas-Otto procedure can be recognized by:

1. **Keller's test**—When the ether extract is dissolved in 1 mL of glacial acetic acid containing 5% ferric sulphate and this solution is put over a mixture of 100 parts of concentrated sulphuric acid and 1 part of ferric sulphate, blue colour appears on acetic acid layer and mauve colour on the sulphuric acid layer.
2. When portions of any part of yellow oleander is boiled with dilute hydrochloric acid, a blue colour develops and bluish colour also appears when alcoholic extract of the seed is warmed with dilute hydrochloric acid, this may disappear on adding potassium permanganate solution.

Medicolegal Aspects

(i) Accidental poisoning can occur from ingestion of powdered root or decoction for treatment and common in children (ii) The powdered roots, decoction of leaves or paste of the fruit may be ingested for committing suicide (iii) It is not used for homicidal purpose. It is commonly used as cattle poison that is given mixed with the cattle fodder (iv) The paste is commonly smeared on the abortion stick to be used as an abortifacient. The powdered root or seeds are taken after mixing with food or drink to cause abortion

CERBERA ODALLAM (Dabur or Dakur)

This plant closely resembles that of *Cerbera thevetia* that grows wild in the swams and the creeks of the seacoasts of India. The plant has a dark, fleshy lanceolate leaves similar to jasmine

and the fruit resembles unripe mango contains a single oval white seed that is flat and oval, has two cotyledons (kernels) that appears bluish when dry. Milky juice exudes out from all parts of the plant. The kernels yield a non-poisonous oil that is used for burning.

Active principles: The kernel contains a glycoside *cerberine* and alkaloid –*Cerebroside* that has digitalis like action.

Fatal dose→Kernel of one fruit.

Fatal period→About 1-2 days.

Signs and Symptoms

They appear within one hour of ingestion. Initially gastrointestinal symptoms appear followed by cardiac toxicity: (i) Violent vomiting and diarrhoea. (ii) Irregular respiration (iii) General paralysis and collapse (iv) Death results from heart failure (v) ECG changes—Sinus bradycardia, S.A. node block, atrial and ventricular fibrillation.

Treatment

(i) Mainly symptomatic treatment (ii) Stomach wash using weak solution of potassium permanganate or tannic acid followed by animal charcoal suspended in water (iii) Injection atropine sulphate 0.5mg i.v is to be given and repeated if needed (iv) Hyperkalemia should be corrected (v) Barbiturates to control convulsions (vi) Stimulants, analeptics, digitalis, I.V. dextrose saline, noradrenaline and corticosteroids to counteracting cardiac failure (vii) To treat cardiac arrhythmias, phenytoin 100-200 mg orally or 100 mg I.V every 5 minutes until they are reverted.

Autopsy Findings

The autopsy findings are non-specific.

Chemical test: The extract is treated with boiling dilute hydrochloric acid and cerebrine will form a bluish or bluish green colour.

HYDROCYANIC ACID (Cyanogen, Prussic acid)

Hydrocyanic acid is a vegetable acid that is widely distributed in nature in many fruits and leaves. Here it exists in the form of harmless glucoside

known as amygdalin, which, coexist with the enzyme emulsion in the kernels of various fruits such as peaches, plums, apricots, bitter almonds, Bamboo shoots, certain oil seeds and beans and leaves of cherry laurel and bitter almonds. The emulsion can readily hydrolyse amygdalin in presence of water to form hydrocyanic acid, glucose and benzaldehyde. It is present in the leaves of Cherry laurel in the strength of .08-0.1% and 10% in oil of bitter almonds. Several people suffer from the effects of hydrocyanic acid poisoning after eating handful of bitter almonds.

Liquid hydrocyanic acid when pure, is a highly volatile colourless liquid having a peculiar odour of bitter almonds or peach-kernels. The hydrocyanic acid is usually obtained by distilling potassium cyanide or potassium ferrocyanide with dilute sulphuric acid.

Hydrocyanic acid is decomposed in neutral or alkaline solution with formation of ammonia. It rapidly decomposes when exposed to air and light

The odour of hydrocyanic acid is that of bitter almonds and all persons cannot smell hydrocyanic acid gas as the ability to do so is a sex-linked trait and when a person is habituated to the smell for few minute, it disappears and again he has to come to fresh air, then only he can smell it.

Uses of Cyanide: (i) Potassium, sodium or silver cyanides are used in the industry for (i) Metallurgy (ii) Photography (iii) Electroplating (iv) As spray to destroy blight (v) Fumigation of ships (ii) Sodium cyanide is extensively used in: (i) Metallurgy (ii) Electroplating (iii) Case hardening of steel and iron (iv) Tanning (v) Manufacturing of dyes (iii) Calcium cyanide is used for (i) Mining industry (ii) As a source of hydrocyanic acid (iii) Fertilizer (iv) Preparation of basic chemicals for plastics (iv) Hydrocyanic acid is used for (i) Photography (ii) Electroplating (iii) Agriculture and horticulture for spraying citrus trees (iv) Fumigation of rooms, ship-holds to free them from bugs, lice and vermins (v) For metal cleaning of, refining of ores and production of synthetic rubber (v) *Flowering agent* (i) Crude oil of bitter almond—2-10% HCN (ii) Cherry laurel water 0.1 HCN that loses strength on storing. The hydrocyanic acid gas is capable

of being absorbed by the skin, when it is moist with perspiration as the poison is readily soluble in water. It is too dangerous to remain too long in a high concentration of the gas in the atmosphere even when wearing a respirator. The hydrocyanic gas is used for fumigating premises and ship-holds

Mode of action: (i) Inhibits the action of cytochrome oxidase, carbonic anhydrase and other enzyme systems as a result the cells are unable to utilize oxygen by dissociating it from the oxyhaemoglobin of the red cells. Tissue anoxia or histotoxic anoxia results even with normal oxygen content in blood that accounts for bright red colour of the venous blood. (ii) There is an interference with the intracellular oxidative process in the tissues (iii) Death is due to histotoxic or cytotoxic anoxia although the blood may contain normal oxygen.

Absorption and Excretion

Cyanide gas is absorbed rapidly from the respiratory system and the acid and salts from the stomach. The acid is also absorbed through the skin. Absorption is delayed when cyanide is taken on a full stomach or with much wine. Cyanide when ingested, in contact with hydrochloric and gastric juice leads to the formation of hydrocyanic acid and chloride. Therefore, it has been suggested that achlorhydric patients cannot be poisoned. It cannot be held not true as the water in the gastric juice and tissues can cause hydrolysis, and leads to liberation hydrocyanic acid.

Signs and Symptoms

Signs and symptoms develop most rapidly when gas is inhaled and the action is instantaneous. Massive doses produce sudden loss of consciousness and the death due to respiratory failure. When large doses are inhaled, symptoms start immediately that is within 1-2 minutes. Voluntary acts are possible like walking and throwing some articles. On ingestion by mouth, the symptoms produced from cyanide poisoning may be delayed up to 15-20 minutes until the liberation of hydro-

cyanic acid by the action of gastric juice on salts of cyanide.

Double cyanides—Potassium Ferro cyanide and ferricyanide are practically non toxic, but they give off hydrocyanic acid in certain conditions. They are toxic when they are taken with the acids. Potassium and sodium cyanide has a corrosive action on the mouth, throat and stomach: (i) Burning taste and feeling of constriction in the mouth and throat (ii) Nausea and vomiting (iii) Excessive salivation and blood stained froth from the mouth (iv) Anxiety and confusion (v) Headache, vertigo and giddiness (vi) Initially Hyperpnoea and Dyspnea is present caused by stimulation of the chemoreceptors and the respiratory center, then respiration becomes rapid, slow and irregular with short, inspiration and long expiration (vii) Smell of bitter almonds in breath and vomitus (viii) In early stages, blood pressure is increased and heart rate is decreased. Later on pulse is rapid, weak and irregular (ix) There is sense of constriction in the chest (x) Unconsciousness and violent convulsion; paralysis follows the convulsive state (xi) Opisthotonus and trismus may develop (xii) Involuntary micturition and defecation (xiii) Skin is covered with sweat and is brick red in colour (xiv) Eyes are glassy and prominent; pupils are dilated and non-reactive to light (xv) Inhalation of gases produces (a) Sense of constriction in throat and chest (b) Dizziness and vertigo (c) Insensibility and death from respiratory failure (d) Epigastric pain and vomiting (xvi) Death is due to respiratory failure

Fatal dose→HCN—50-60 mg.

KCN and NaCN—200-300 mg.

Air concentration 1:500 causes immediate death.

Fatal period→HCN—2-3 minutes.

KCN and NaCN—half an hour.

Treatment

Treatment should be started immediately without losing any time. To reverse cyanide cytochrome combination, haemoglobin is converted in to methaemoglobin by the action of nitrites:

- Cytochrome oxidase + NaCN@cytochrome oxidase cyanide

- Sodium Nitrate + Hb → Methhaemoglobin
- Methhemoglobin + NaCN® Cyanmethhemoglobin (non toxic).

(i) Break 0.2cc ampoule of Amyl nitrite in a handkerchief and hold over the patient's nose for 15-30 minutes until sodium nitrite is given (ii) 0.3gm of 3% sodium nitrite and 10ml of sterile water is given intravenously over 2-3 minutes (iii) Through some needle 25gm of sodium thiosulphate in 50% solution is given i.v. over 10 minutes. It converts cyanide to thiocyanate that is non-toxic. Repeat the nitrite thiocyanate injection after one hour if necessary (iv) Gastric lavage by 3% hydrogen peroxide, Sodium thiosulphate (5-10%) and Potassium permanganate (1:5000) of which, 200ml is left in the stomach (v) Methylene blue is an antidote; 50 ml of 1% aqueous solution is used. It converts haemoglobin to methaemoglobin that combines with free cyanide, which is non-toxic and is removed from the circulation (vi) Cobalt salts such as cobalt acetate is given 100 mg i.v. and cobalt edetate 600 mg (vii) Para amino propiophenon (PAPP): It converts haemoglobin to methaemoglobin (viii) Artificial respiration (ix) In delayed cases, a mixture of ferric and ferrous sulphate with potassium carbonate is the chemical antidote to form Prussian blue (x) Maintain clear airway (xi) Symptomatic treatment.

Therapy with amyl nitrate: Amyl nitrate converts a portion of the hemoglobin's iron from ferrous iron to ferric iron, converting the hemoglobin into methemoglobin. Cyanide is more strongly drawn to methemoglobin than to the cytochrome oxidase of the cells, effectively pulling the cyanide off the cells and onto the methemoglobin. Once bound with the cyanide, the methemoglobin becomes cyanmethemoglobin. Therapy with nitrites is not innocuous. The doses given to an adult can potentially cause a fatal methemoglobinemia in children or may cause profound hypotension. Treatment of children affected with cyanide intoxication must be individualized and is based upon their body weight and hemoglobin concentration.

Alternative management protocols in other countries:

1. *The United States standard cyanide antidote kit:* It uses a small inhaled dose of amyl nitrite followed by intravenous sodium nitrite.
2. In U.S.A, the next part of the cyanide antidote kit is sodium thiosulfate, which is administered intravenously. The sodium thiosulfate and cyanmethemoglobin become thiocyanate, releasing the hemoglobin, and the thiocyanate is excreted by the kidneys.
3. In France, hydroxycobalamin (a form of vitamin B₁₂) is used that combines with cyanides to form the harmless vitamin B_{12a} cyanocobalamin. Cyanocobalamin is eliminated through the urine. Hydroxycobalamin works both within the intravascular space and within the cells to combat cyanide intoxication. This contrasts with methemoglobin, which acts only within the vascular space as an antidote.
4. Administration of sodium thiosulfate improves the ability of the hydroxycobalamin to detoxify cyanide poisoning. This treatment is considered so effective and innocuous that it is administered routinely in Paris to victims of smoke inhalation to detoxify any associated cyanide intoxication. However it is relatively expensive and not universally available.
5. 4-Dimethylaminophenol (4-DMAP) has been proposed in Germany as a more rapid antidote than nitrites and with reportedly lower toxicity. It is used currently by the German military and by the civilian population. In humans, intravenous injection of 3 mg/kg of 4-DMAP will produce 35 percent methemoglobin levels within 1 minute.
6. Cobalt salt have also been demonstrated as effective in binding cyanide. One current cobalt-based antidote available in Europe is dicobalt-EDTA, sold as *Kelocyanor*. This agent chelates cyanide as the cobaltcyanide. This drug provides an antidote effect more quickly than formation of methemoglobin, but a clear superiority to methemoglobin formation has not been demonstrated. Cobalt complexes are quite

toxic, and there have been accidents reported in the UK where patients have been given dicobalt-EDTA by mistake based on a false diagnoses of cyanide poisoning.

Autopsy findings

(i) Rigor mortis starts early and lasts longer (ii) Eyes are bright, glistening, and prominent with dilated pupils (iii) Jaws are firmly closed (iv) Froth is coming from the mouth (v) Post mortem staining is of cherry red colour due to the formation of cyanmethaemoglobin, tissues cannot take up oxygen and oxygen remains in the cells as oxyhaemoglobin; asphyxia occurs in the presence of oxygen (vi) On opening the body, the odour of cyanide can be felt from the stomach contents and other organs (vii) Brain is to be opened first as the odour is well marked here (viii) Trachea contains blood stained froth (ix) All the visceral organs are congested (x) Lungs are oedematous (xi) CNS shows degenerative changes (xii) Potassium and sodium cyanide cause corrosion of the mouth (xiii) Gastric mucosa is crimson red in colour that changes to rusty brown due to the formation of acid Haematin (xiv) Blood is cherry red coloured due to formation of cyanmethaemoglobin.

Spectroscopic Examination of Blood

The spectroscopic examination of cyanides resembles that of reduced haemoglobin with a thick band between the lines D and E.

Chronic Cyanide Poisoning

Chronic poisoning may occur by continued inhalation of low concentration of cyanide gas for a long period; the symptoms are those of suboxia including cachexia, mental disturbance, visual defects, such as scotomata, optic atrophy and psychosis.

Judicial Execution

The condemned or prisoner person is strapped to a chain in a closed room and several cyanide eggs are dropped in to a pan of strong acid that leads to liberation of cyanide gas. Unconsciousness takes place very rapidly though heart continues to beat for 10-15 minutes then death occurs.

Medicolegal Aspects

(i) Commonly used for the purpose of suicide, as it is quick acting and only a small dose is fatal. (ii) Oil of bitter almonds (2-10% HCN) and cherry laurel is used as flavouring agent (iii) Causes of accidental poisoning: (a) Inhalation of cyanide vapours used as fumigating agent causes accidental poisoning (b) May be ingested in accidentally in lieu of some medicine (c) It may be accidental to the workers employed in the industry (iv) It is rarely used for homicidal purposes (v) Putrefaction produces 1/10th of the cyanide poison (vi) Cyanide is destroyed by embalming (vii) It is present in fire victims in very small amount (viii) Cattle poisoning results by eating *jawar*, *kadvi*, or *alsi* plants. Growing plants, flowers, and seeds contain maximum hydrogen cyanide. One pound of flower is fatal to a bullock—cyanogenic glucoside (ix) On keeping it for a long time harmless carbonate may be formed by the action of atmospheric carbon dioxide and moisture on potassium cyanide and potassium formate.

The nitrite forms methaemoglobin that reacts with hydrogen cyanide and allows the enzyme to function. The sodium thiosulphate helped by the enzyme Rhodenase inactivates the cyanide by converting it in to non-toxic thiocyanate. Methylene blue converts the haemoglobin of the blood in to methaemoglobin, which combines with free cyanide there by removing it forms, the reaction.

Drug Abuse

DRUG

In 1969, WHO Committee on Drug dependence defined drug as “A substance that when taken in to a living organism, may modify one aspect of its function”. As this definition included water and air also, certain modifications were made in the definition. These are: (i) Drug in the broadest sense is any chemical entity or mixture of entities other than those required for the maintenance of normal health, the administration of which alters the biological function and bodily structure (ii) Second level of definition could then specify the users to which these entities are put that is the drugs used for the treatment or alleviation of disease known as medications, therapeutic agents (iii) Third level of definition: One may differentiate chemical entities based on bodily system or function on which they exert their most marked or obvious affects e.g. Diuretics, CNS Stimulants etc. (iv) The main concern is about the possible adverse effects on health or social function e.g. psycho-active drugs, Anti histaminics.

Use, Abuse and Misuse: Abuse and misuse are unsatisfactory concepts because these terms involve value judgment, they are impossible to define in such a way that they are appropriate for different drugs in different situations.

Drug Abuse

1969 WHO Expert Committee defined drug abuse as “Persistent or sporadic excessive drug use inconsistent with or unrelated to acceptable medical practice”.

Further attempts to achieve a comprehensive definition of abuse have been considered depend-

ing on the medical need, individual and social harm and the degree of tolerance by the community.

Drug abuse refers to the use usually by self-administration of any drug in a manner that deviates from the approved medical or social pattern within a given culture. In some cultures, some drugs/substances are not considered against the society and so may not be against the legal system of the country. Use of medically prescribed drugs e.g. barbiturates to induce sleep is permissible but if it is used to induce euphoria (when not medically indicated); it is called drug abuse e.g. opiates can be taken when prescribed medically for the relief of pain but not solely for its euphoric effect. When people continue to take these drugs in the absence of disease, intensity of dependence varies from mild desire to the compulsion to use the drug is called drug abuse.

Clinicians and research workers can often manage without using the terms such as unsanctioned, hazardous, dysfunctional or harmful use as they are concerned more about the effects of drugs and their route of administration. Nevertheless, if such terms are to be used then following concepts may be useful (i) Unsanctioned use—Use of a drug, not approved by a society or a group within that society, it implies that we accept disapproval as a fact (ii) Hazardous use—Use of a drug that will probably lead to harmful consequences, for the users either to dysfunction or to harm e.g. smoking 10-15 cigarettes per day may not be accompanied by any harm, but we know it to be hazardous (iii) Dysfunctional use—Use of a drug that is heading to impair psychological or social functioning such as loss of job, marital problems etc. (iv) Harmful use—Use of a drug that

is known to have caused tissue damage on mental illness, in a particular person.

Drug addiction: Drug addiction is a state of periodic or chronic intoxication, produced by repeated consumption of a drug, which is harmful to the individual and to society such as Heroin, alcohol, cannabis, Hashish, barbiturates, opiates, cocaine, amphetamine, LSD, Mandrax and bromides. Of these, Heroin is the most dangerous.

Drug habituation: A condition resulting from the continued consumption of a drug, which does not cause much harm to the individual or to the society though there is psychological and emotional dependency on the drug. These are Caffeine, Nicotine etc.

Table 3.1: Differences between drug addiction and drug habituation

Features	Addiction	Habituation
1. Compulsion	Present	Desire
2. Dose	Increased tendency	No such
3. Dependence	Psychical & Psychological	Some degree of Psychological
4. Withdrawal symptoms	Characteristic	Mild or nil
5. Harm	Individual and society	Only to the Individual

Causes of Drug Abuse: The drug abusers start with a smaller dose but gradually increase the dose and frequency of intake until they develop tolerance. The abusers use the drug due to many reasons such as: (i) To get respite temporarily from various physical or mental ailments (ii) For the purpose of recreation and relaxation (iii) For better performance (iv) For controlling feelings of anger or distress and to get relief from chronic tension (v) For performing spiritual exercises e.g. LSD and Methaqualone (vi) For alleged aphrodisiac properties (vii) To get relief from frustration in life, love or sex (viii) Just out of curiosity.

Types of persons exposed: The persons likely to be exposed to drug abuse are: (i) Neurotic or mentally imbalanced subjects (ii) Persons in habit of taking risks and having rebellious nature (iii) Individuals with sexual promiscuity

Magnitude of problem of drug abuse: Drug abuse is a worldwide phenomenon because of its harmful effects on the health and morale of the individual and society at large. Misuse of drugs have become part of the modern life in order to obtain kicks, thrills, excitement and escaping from day to day problems of life. United States has described the threat of drug abuse as a menace to which no country feels immune. Of late, many cases of forgery of prescription, criminal activities following ingestion of drug, antisocial activities of teenagers, illicit trafficking of the drugs and their abuse is going on but only few are caught and reported. The trafficking of drugs is done in many countries such as Myanmar, China and Thailand (Golden triangle) that fall along the eastern side of the region and Pakistan, Afghanistan, Iran and India (Golden crescent) that fall on the western side. One third of the total Heroin seized is from India. Addiction to narcotics especially Heroin is important cause of committing crime. 50-80% of the street crimes committed in big cities of US are committed by addicts. These include the crimes of robbery, house breaking, borrowing and begging to keep heroin flowing in their veins (can even sell their property). The addicts call heroin by the names of 'our heroin' and 'white queen'. Besides heroin amphetamines, cocaine and barbiturates are also prevalent among the addicts. The drug addicts are involved in causing sexual offences, forgery, cheating, and vehicular accidents.

Demographic studies: The students both male as well as females are given Marijuana called 'Reefers' by their classmates free of cost. Later on when this person becomes habituated, these addicts start selling the drug at a higher price. Drug addicts indulge in polygamy and sexual intercourse in groups under the effect of marijuana or LSD. Devotees of marijuana call it social drug and are under the theory 'make love and not wars' 'Hippies' share their quota of drugs with the students. A survey conducted by Tata Institute of Social science revealed that most of the students taking drugs belong to the upper class and those of affluent stream are more inclined. The addicts belong usually to 10-20 years age group and males

are more prone. Drug abuse is more common in bright and brilliant students' particularly professional college students. In urban areas, narcotic drugs and psychotropic substances are commonly abused whereas in rural areas mainly Charas, Ganja and country liquor is consumed.

Classification of Drugs of Abuse

The abuse of narcotic as well as non-narcotic drugs is quite common and the non-narcotic drugs are commonly referred to as soft drugs to distinguish them from the narcotics or the hard drugs. The drugs of abuse are classified as: (i) Hypnotics such as Barbiturates (called 'sleepers' or 'goof balls' by drug abusers) (ii) Stimulants are commonly used by drug addicts and the tolerance to these drugs develops slowly but can be acquired to the massive doses. These are Amphetamines (called 'Purple Hurts', 'Black bombers', 'pep pills' or 'desk'), Benzedine, and Ephedrine (iii) Antidepressants—Tricyclic antidepressants and MAO inhibitors (iv) Hallucinogens drugs such as Cannabis, Mescaline, Cocaine, Lysergic, LSD, Phencyclidine, Methaqualone (Mandrax) and various hydrocarbon preparations (v) Sedatives as well as hypnotic and tranquilizers—Benzodiazepines (vi) Narcotics such as opium and its derivative (pethidine and heroin) (vii) Alcohol and alcohol containing beverages (viii) Deliriants.

The action of all these drugs overlap to a certain extent that is those substances acting as central nervous system stimulants can impair consciousness when taken in sufficient quantity whereas those that depress the CNS may arouse and cause loss of inhibitions before exerting their depressant action.

Routes of Administration

The route of administration depends predominantly on the nature of substance and the intensity of action desired. The possible routes are: (i) *Orally*—This route is commonly employed the absorption from the gastrointestinal tract is relatively slow and the impact of the drug on the brain is muted unless large quantities of the drug are taken (ii) *Inhalation*—The method is also quite commonly employed as there is increase in the speed of

absorption. This method is commonly employed for inhaling nicotine from tobacco and *charas* (iii) *Snorting*—This route is not commonly employed except in cocaine. Cocaine is commonly dissolved and the solution sniffed up in the nostrils where a part of it is absorbed (iv) *Injection*—This method is the fastest method of getting the desired effect as high concentration of the drug is absorbed through the brain. Two methods are commonly employed; *Skin-popping* is injection via subcutaneous route and *mainlining* is injection via intravenous route. Besides this injection is also taken intramuscularly. Rapid I.V. injection of an opioids produces warm flushing of skin and sensation in the lower abdomen described by addicts similar in intensity to sexual orgasm that lasts for 45-50 second and is described as 'rush', 'thrill' or 'kick'.

Fatal Results of Drug Abuse

The hazards of drug abuse are:

Overdosage of the drug: The overdosage of the drug commonly occurs accidentally, intravenous injection carries the highest risk of accidental overdosage, and when it occurs, the drug cannot be retrieved. When the supply of high quality drug is less, the drug pushers may *cut* (dilute) the drugs that are sold on the streets in order to experience the 'kick' or 'rush', they tend to increase the dose.

Toxicity due to contaminants: The substances such as talc are commonly used to *cut* the drugs in order to reduce the cost. These are used by the drug addicts while preparing the drug for injection. The abuser may develop pulmonary granulomas due to these contaminants and other toxicity e.g. quinine.

Infections: Infections such as Hepatitis B, HIV, bacterial abscesses, thrombophlebitis, endocarditis and moniliasis may result from the use of contaminated needles, sharing of needles following other unhygienic practices.

Gangrene: Gangrene may result from inadvertent intra-arterial injections.

Anaphylactic reaction: The drugs of abuse are likely to cause hypersensitivity reactions

Risks of body packing and body stuffing: For smuggling of the drugs, the individuals (*swallowers*) make small packets of the drugs and swallow them that can later on be recovered from the vomitus or faeces. The individuals (*stuffers*) may also insert the packets of drugs into their vagina or rectum. Drug preparations that are financially lucrative such as high quality cannabis, cocaine, heroin, and morphine are smuggled in this way. There exists a risk of overdose with these practices as the packets may burst and get absorbed through the gastrointestinal, vaginal or rectal mucosa. The body packer is at risk of acute intestinal obstruction as the drugs are commonly wrapped in aluminium foils, cellophane paper or condom etc. in such cases, abdominal radiographs help in revealing the physical characteristics of the wrapping material together with the nature of the drug contained therein. This helps the physician in deciding the protocol of management.

Drug Dependence

According to WHO recommendations of 1964, drug addiction and drug habituation should be substituted by the term 'drug dependence' meaning deviation from normal social behaviour of the patient under the influence of drugs. Addicts initially use the drug for some disease but gradually they develop the habit and become dependent on it as they find the drug an easy cure for the various diseases. They may also use the drug for their narcotic effect.

Types of dependence: WHO has recognized five types of dependence (i) *Morphine type*—In this type of dependence there is an overpowering craving for the drug, which tends to increase with time. There is tendency to increase the drug. Tolerance & cross-tolerance is seen in this type of dependence. The drugs are usually taken by intramuscular, intravenous route or by sniffing. Withdrawal symptoms develop if the drug is withdrawn over a period of hours. Death may result from arrhythmias (ii) *Barbiturate type*—There is a strong desire to continue taking the drug. Tolerance develops to high doses of the drug. Cross-tolerance is seen in this type also and postural

hypotension is also present in cases. There is development of withdrawal symptoms with maximum intensity within 2-3 days of withdrawal (iii) *Cocaine type*—In this type of dependence there is desire to continue taking the drug but there is no tolerance. Psychological dependence is present and there are no withdrawal symptoms (iv) *Cannabis type*—In this type also there is no tolerance and there are no withdrawal symptoms. There is no desire to increase the drug dose also (v) *Amphetamine type*—This type may be combined with barbiturates. There is increased desire to take the drug and there is a tendency to increase the dose. Tolerance and psychic dependence is present. No abstinence syndrome is seen and continuous use may lead to toxic psychosis.

Acute signs and symptoms: (i) Loss of weight and appetite (ii) Unsteady gait (iii) Tremors (iv) Reddening of eyes (v) Puffiness of eyes (vi) Slurring of speech (vii) Impairment of memory and concentration.

Withdrawal symptoms: These are the symptoms, which are seen due to sudden stoppage of the regular supply of drugs. Usually the symptoms start within 6-8 hours following stoppage of drug but may be delayed depending on the type of drug. The intensity of withdrawal symptoms depend upon the dose and type of drug, duration of addiction and whether the drug has been withdrawn suddenly or gradually

Early symptoms: (i) Chilliness (ii) Sensation of cold (iii) Uneasiness (iv) Rhinorrhoea (v) Yawning.

Late symptoms: (i) Respiration: rapid and laboured (ii) Loose skin (iii) Lachrymation (iv) Gross tremors (v) Dilated pupil (vi) Anorexia.

Third stage: Final stage of sleep lasting for 8-16 hours is seen and on awakening all the previous symptoms become intense. Other symptoms produced are: (i) Fever (ii) Tachypnoea (iii) Hypertension (iv) Pain and cramps in legs (v) Perspiration (vi) Diarrhoea & vomiting

In new borne: Newborns of addicted mothers develop withdrawal symptoms within 1-56 hours of birth in the form of hyperactivity, convulsions and twitching.

Causes of deaths in drug abuse

Most of the drug addicts die as result of complications of drug administration such as: (i) Infection and septicemia (ii) Endocarditis (iii) Hepatitis (iv) Tetanus (v) Pulmonary and cerebral abscess (vi) AIDS complications (vii) Suicidal tendency from overdose of drugs during withdrawal of certain drugs particularly amphetamine (viii) Person may die due to electrocution, fall resulting in head injury or fall in water (drowning) under the effect of drugs (ix) Accidental aspiration of food, vomitus or artificial dentures resulting in choking.

Autopsy Findings

External: (i) The person is emaciated with a shabby appearance with lots of tattoos and scars over the body. (ii) The fingertips may be stained with different types of drugs used (iii) IV drug addicts known as *main-liners* show typical linear needle prick and scar marks usually over veins of cubital fossa, forearms, dorsum of hands, lower extremities, neck, and dorsal vein of penis (iv) S.C heroin drug users known as *skin-poppers* develop typical depressed, circular, atrophic scars and ulcers (v) The superficial veins in hands and legs and rarely in scalp, penis and popliteal area may be thrombosed due to the injections (vi) Punctate areas of tattooing are seen along the needle tracks due to deposition of carbonaceous material along the needle track (vii) Various recent and old injection sites may show zones of inflammation and perivenous fibrosis (viii) Multiple recent as well as scars of old healed abscesses may be seen (ix) Habitual inhalation of cocaine or heroin by snorting & snuffing may lead to perforation of nasal septum.

Internal: (i) The gastro intestinal tract may still contain pills or capsules if they have been ingested recently. Active filler material like starch, talc or cellulose may be seen adherent to the gastric mucosa (ii) Microscopic examination of the various scars often shows foreign material like fibres of cloth, talc or dirt particles in the scar (iii) On repeated i.m and i.v injections, there is abscess formation in the muscle and at injections sites near the veins. The skin, subcutaneous tissue, muscle and vein surrounding the area of recent

injections along with the control from opposite side should be preserved in normal saline for chemical analysis (iv) Hepatic lymphadenopathy is a very common finding. The lymph nodes in the porta hepatis are enlarged and microscopically show non-specific hyperplasia (v) There is non-specific pulmonary edema, bronchopneumonia and aspiration of gastric contents. Froth is present in the upper respiratory tract and may be present over the nose and mouth (vi) Pulmonary hypertension with right ventricular hypertrophy may be present due to presence of extensive microcrystalline pulmonary emboli (vii) The lungs appear congested and edematous (viii) IgM and IgG concentration is increased in the spleen and lymph nodes indicating acute and chronic antigenic stimulation.

Treatment

(i) Chlorpromazine/promethazine is given in 5% dextrose 8 hourly along with vitamin B complex (ii) For the treatment of pain proxyvan is given (iii) To treat insomnia, alprazolam or nitrazepam can be given (iv) After 3 days, Methadone, Naloxone, Naltrexone can be given (v) By 2-3 week, the dose of Heroin is reduced gradually (vi) At the end of 3 week the drug is totally stopped and all other drugs tapered.

Treatment of addiction: (i) Steps to guide abuser to come back to a sober way of life (ii) Gradual withdrawal of drugs (iii) Use of others drugs (iv) Keep a careful watch to avoid obtaining secret supply of drugs (v) Person should be removed to an institution (vi) Administration of drugs like: Sedative, Benzedrine and hyoscine is beneficial (vii) Group and individual psychotherapy with counselling about bad affects of drugs on the patients and society (viii) Diverting the mind of the person by engaging physically/mentally in vocational courses (ix) Improving general health to the person with adequate nutrition and food (x) Symptomatic treatment.

HALLUCINOGENS (Psychedelics)

Hallucinogens are psychotomimetics, psychotogen, psychedelics or psychodysleptics. These are drugs that causes excitation of central nervous

system characterized by (i) Hallucination (ii) Mood changes (iii) Anxiety (iv) Sensory distortion (v) Delusion (vi) Depersonalization (vii) Increased pulse rate, temperature and Blood pressure (viii) Dilatation of pupil (ix) Psychic dependence and (x) Depressive or suicidal psychosis. Since prehistoric times man has used many natural products having hallucinogenic property. However, a number of synthetic compounds have also been formed. These are: (i) LSD (Lysergic Acid Diethyl amide) (ii) Mescaline (iii) Psilocybin and (iv) Phencyclidine. There is individual variation in response to these drugs but autonomic and perceptual reactions are prominent.

Signs and symptoms: (i) Hallucinogens produce a dream like state with disorientation, loss of contact with reality (ii) There is distortion of visual perception such as swaying of the field of vision, objects appear distorted similar to images in a curved mirror and faces may appear grotesque. There is an awareness of intense luminosity of colour and on closing eyes colourful images appear to surge (iii) There is alteration of time sense and music appears tangible (iv) Ability to concentrate is impaired (v) Ataxia is not a prominent feature (vi) The person feels relaxed and is extremely happy; may sometimes laugh uncontrollably or become sad or weep (vii) With higher doses, panic reactions and sinking sensations are common.

Tolerance and dependence: Tolerance is readily induced by hallucinogenic drugs and cross tolerance has also been demonstrated. Physical dependence does not occur but psychological dependence varying from mild (occasional trips) to marked (compulsive abuse) does occur.

Medicolegal aspects: Few of the hallucinogenic drugs are primary causes of death but some may lead to traumatic deaths because of the abnormal behaviour of the person who is under their influence.

Lysergide

It is psychomimetic, semisynthetic derivative of Ergot that acts at multiple sites in the central nervous system from the cortex to spinal cord.

Signs of symptoms: *Small doses* of 20-25µg produces (i) Pupillary dilatation (ii) Increased blood pressure (iii) Hyperreflexia (iv) Tremors (v) Muscle weakness.

Large doses produces (i) Dizziness (ii) Drowsiness (iii) Euphoria (iv) Colours may be heard (v) Sounds are visualized (vi) Time is passed to pass slowly (vii) Psychological dependence may develop (viii) Serious depression and (ix) paranoid behaviour.

Treatment: (i) Antianxiety drugs should be given (ii) Barbiturates to be given (iii) It is better to talk to the patient.

Lysergic Acid Diethylamide (LSD)

It is also known by the names of Acid, microdot, purple haze, white lightning etc. LSD has derived its name from the German '*lyserge saure diethylamid*'. It is the most powerful hallucinogen known to man. It is one of the indole alkaloid derivative; others being psilocybin and psilocin that is contained in the Mexican mushroom (*Psilocybe mexicana*). LSD was synthesized by Hofmann in 1938 who was working on the chemistry of ergot alkaloids and himself experienced its hallucinogenic effects. The properties of LSD were known in 1943. The discovery of psychedelic effects of LSD in 1947 led to an epidemic of LSD abuse during the 1960s. It is the most potent psychedelic and doses as low as 25-50 µg may induce profound psychological and physiological effects. The action of LSD may persist for 12-18 hours even though the half life of the drug is only 3 hours. Abrupt abstinence following continued use does not produce withdrawal signs and symptoms. The use of LSD for therapeutic purposes should be undertaken only with great caution.

Signs and symptoms: LSD causes alteration in consciousness; there is dream like state and sense of unreality. Easily induces laughter and crying. There are visual and tactile hallucinations (i) Within minutes of oral administration of LSD, following symptoms are produced (i) Tachycardia (ii) Hypertension (iii) Pupillary dilatation (iv) Tremors and (hyperreflexia) (ii) Within half an hour of oral

intake, following symptoms are produced (i) Bizarre and conflicting perceptual and mood changes (ii) Visual illusions (iii) Synthesias (iv) Extreme lability of mood (iii) Time seems to pass very slowly (iv) Behaviour becomes disturbed with paranoid delusions (v) Flashbacks in which effects of LSD may be reexperienced without further exposure to the drug for several years (vi) *Bad trip*: It is a medical emergency associated with use of LSD consisting of panic episode and that may persist for 24 hours.

Dose: (i) 1-3 µg/kg produces moderate effect and duration of action is 1-56 hours (ii) 20-100 µg produces the effect (iii) 14 mg is Fatal dose.

Treatment: (i) Supportive measures (ii) Anxiolytic drugs in small doses (iii) Chlorpromazine counteracts the effects of LSD.

Medicolegal aspects: Individual intoxicated with LSD rarely present for medical help. The drug was most popular with the hippies in the West in the 1960s. The deaths are commonly attributed to the bizarre behaviour induced by the drug that can result in accidental and suicidal deaths. Suicidal and homicidal tendency occurs. The persons explore their own feelings.

Mescaline

Mescaline is a phenylalkyl amine that was isolated in 1896 from Mexican 'Peyote cactus' *Lophophora Williamsi* growing in the deserts of Central America. Of the four alkaloids, alkaloids mescaline is most important. The extracts are used as an intoxicating drink called 'Mescal buttons'. 3-15mg/kg produces the moderate effects and duration of action is 1-56 hours 200mg of Mescaline Sulphate produces intoxicating effects. This is the oldest known drug with a primary hallucinogenic action.

Signs and symptoms: (i) Dilated pupil (ii) Static tremors (iii) Sweating (iv) Increased pulse rate (v) Visual hallucinations (vi) Disorientation of speech and time (vii) Other effects such as (i) Insomnia (ii) Vertigo and (iii) Headache are also produced (viii) Large doses produce symptoms of poisoning such as (i) nausea (ii) vomiting (iii) bloody diarrhoea

and shallow breathing (iv) BP is low and death is due to respiratory failure

Phencyclidine

Phencyclidine is also known by pseudonyms as Angel dust, Peace pill, Hog, Goon, Rocket fuel, Cadillac, Super grass etc. Phencyclidine (PCP) was developed for use as an anaesthetic in the late 1970s but was abandoned quickly because of an unacceptably high incidence of postoperative psychotic reactions. PCP (1-phenylcyclohexyl piperidine) is easily synthesized and several variants exist. It is usually smoked in combination with tobacco and less frequently with marijuana but it may also be ingested or injected. Phencyclidine binds to inotropic n-methyl-d-aspartate (NMDA) receptors in the nervous system, blocking ion current through these channels. The most common street preparation, angel dust, is a white granular powder that contains 50-100% of the drug.

Signs and symptoms:

1. *Pleasurable effects:* (i) Initial euphoria and a feeling of dissociation (ii) Numbness (iii) Perceptual distortion (iv) Visual hallucinations.
2. *Users may show:* (i) Horizontal or vertical nystagmus (ii) Flushing (iii) Diaphoresis (iv) Hyperacusis.
3. *Behavioural changes:* (i) Distortions of body images (ii) Disorganization of thinking (iv) Feelings of estrangement.
4. Higher doses of PCP (5-10mg) may be complicated by: (i) Hypersalivation (ii) Profuse sweating (iii) Generalized seizures (iv) Prolonged psychotic reactions (v) Dystonias (vi) Hypoglycemia (vii) Rhabdomyolysis and acute renal failure.

Diagnosis: (i) Determination of phencyclidine levels in the urine as PCP is excreted in the urine for a long time after ingestion and autopsy samples may also be positive for up to a week (ii) The levels of PCP in the serum are also diagnostic but CSF concentrations may be three to four times higher than those in the serum and may take much longer time to clear.

Treatment: (i) Gastric lavage is helpful in achieving excretion of PCP from the body and gastric suction may be of some value by removing PCP secre-

ted in gastric juice and preventing its re-absorption (ii) Oral diazepam is preferred but chlorpromazine can be given if hypertension is there (iii) Diphenhydramine i.m has been reported to abolish acute dystonic reactions (iv) Acidification of urine enhances renal elimination (v) Supportive measures including treatment of coma, convulsions and respiratory depression.

Medicolegal aspects: (i) It is commonly smoked by sprinkling on parsley or marijuana leaves and the intoxication can result from this as cases have been reported in children who are in the same room with adult smokers (ii) It is sometimes used as an adulterant in expensive drugs of abuse such as cocaine (iii) Addiction may lead to violent behaviour, psychosis, suicidal and homicidal behaviour.

TRICYCLIC ANTIDEPRESSANTS

Tricyclic antidepressants and benzodiazepines, the ubiquitous sedatives and hypnotics are the major causes of clinical problems with drugs which depress the central nervous system. Imipramine was found during clinical trial in 1958 to selectively benefit depressed but not agitated psychotics. Tricyclics include (i) Imipramine (ii) Amitriptyline (iii) Nortriptyline (iv) Trimipramine (v) Doxepin (vi) Dothiepin, and (vii) Clomipramine.

Mode of action: Tricyclic antidepressants have complex actions which account for the diverse nature of the features seen after overdose. They inhibit the active uptake of biogenic amines noradrenaline (produces antidepressant action), 5-hydroxytryptamine (produces antidepressant and sedative action) and dopamine (produces stimulant action) into their respective neurons. They however differ markedly in their selectivity and potency for the different amines. They are also potent anticholinergic and antiarrhythmic activity.

Signs and symptoms: Features of poisoning usually appear within an hour or so after an overdose and usually reaches maximum intensity in 4-12 hours. The features are: (i) *Anticholinergic symptoms:* (i) dry mouth (ii) constipation (iii) Epigastric distress (iv) Urinary retention (v) Dilated pupil and blurred vision and (vi) Palpitations are

produced Peculiar clumsy feeling (ii) Tiredness and light headedness (iii) Drowsiness and sleepiness (iv) Difficulty in concentrating (v) Unsteady gait (vi) Increased reflexes and extensor planter responses (vii) Tachycardia is due to anticholinergic and noradrenaline potentiating actions (viii) Postural hypotension is due to inhibition of cardiovascular reflexes (ix) *ECG changes:* T wave suppression or inversion is the most common consistent change (x) Arrhythmias occur due to interference with intraventricular conduction, combination of noradrenaline potentiating and acetylcholine blocking actions and direct myocardial depression that are major causes of death.

Tolerance and dependence: Tolerance to the anticholinergic and hypotensive effects develop gradually, though antidepressant action is sustained. Psychological dependence on these drugs is rare as their acute effects are not pleasant. There is some evidence of physical dependence occurring when high doses have been used for long periods.

Withdrawal symptoms: Withdrawal symptoms do occur on discontinuation of the drug that is why gradual withdrawal of these drugs should be done. The features such as malaise, chills, muscle pain may occur on discontinuation of the drug.

Treatment: (i) Gastric lavage with administration of activated charcoal (ii) Supportive measures such as adequate oxygen inhalation and control of convulsions (iii) Correction of acidosis by bicarbonate infusion intravenously (iv) Physostigmine 0.5-2mg i.v. should be given to reverse the anticholinergic effects (v) To treat arrhythmias, propranolol or phenytoin should be used (vi) Diazepam may be injected intravenously to control delirium with auditory and visual hallucinations as well as convulsion that occur during recovery phase.

AMPHETAMINES

Amphetamines were first synthesized in 1887 but began to be therapeutically used only since 1930s. Amphetamines are drugs of abuse and are capable of producing but little or no physical dependence. Because of its abuse potential, its therapeutic administration is greatly restricted today.

Ecstasy (methylenedioxy-meth-amphetamine), was developed in 1914 and was used in as a conscious altering agent in psychotherapy.

Mode of action: Amphetamines predominantly stimulate the central nervous system and maximum selectivity is exhibited by Dextroamphetamine and methamphetamine, which in the usual doses produce few peripheral effects. They also cause stimulation of respiratory system especially when it is depressed. They inhibit hypothalamic feeding centre and also have a weak anticonvulsant, analgesic and antiemetics actions.

Derivative of amphetamines: The derivatives of amphetamine are (i) Methamphetamine, (ii) Dextroamphetamine, (iii) Fenfluramine, (iv) Phentermine, (v) Mephentermine, (vi) Methyl phenidate, and (vii) Synthetic amphetamines ('Designer drugs')—Methylenedioxy-meth-amphetamine (MDA or 'Love drug') and Methylenedioxyethamphetamine (MDEA or 'Ecstasy').

Signs and symptoms: (i) Alertness and increased concentration (ii) Initial euphoria and more extrovert behaviour (iii) Increased talkativeness with rapid speech (iv) Increased work capacity with allaying of fatigue (v) Improvement of athletic performance initially followed by further deterioration (vi) Stimulation of reticular activating system causing wakefulness and postponement of sleep deprivation induced physical (vii) Hunger is suppressed (viii) Tremors (ix) Pupils are dilated (x) Tachycardia and hypertension are not so significant (xi) High doses produces euphoria, marked excitement, that may progress to mental confusion, delirium, hallucinations and an acute psychotic state (xii) Peripheral component of toxicity include vasomotor effects, palpitation, arrhythmias, vomiting, abdominal cramps and vascular collapse (xiii) Death is uncommon and is usually preceded by convulsions and coma.

Autopsy findings: (i) No specific findings (ii) Cerebral or subarachnoid haemorrhage from induced hypertension may be produced.

Treatment: (i) Chlorpromazine should be used for

sedation (ii) β -adrenergic blockers are used to antagonize the peripheral sympathomimetic actions of amphetamines (iii) Acidification of urine helps in increased renal elimination of methamphetamine (iv) Supportive measures.

Medicolegal aspects: (i) Acute overdosage is not so common (ii) Amphetamine abusers are generally teenagers seeking thrill or kick which is obtained on rapid i.v. injection and designer drugs are commonly abused by youngsters during parties (iii) Amphetamine abuse is also seen in athletes who use it to enhance their athletic ability and Amphetamine is one of the drugs included in the 'dope test' for athletes.

BENZODIAZEPINES

The benzodiazepines are one of the most important groups of psychotropic drugs. They are widely used as tranquilizers, sedatives and hypnotics. They include the following compounds: (i) Chlor-diazepoxide (ii) Diazepam (iii) Temazepam (iv) Lorazepam (v) Triazolam (vi) Bromazepam (vii) Clonazepam (viii) Desmethyldiazepam (ix) Medazepam (x) Prazepam.

Signs and symptoms: Benzodiazepines are one of the safe drugs when their overdosage occurs alone. Cases have been reported when as many as 70 tablets of any of them are unlikely to produce any thing more than mild effect in most adults. Many benzodiazepines have active metabolites that account for their prolonged sedative effects. Benzodiazepines potentiate the effects of other CNS depressants, particularly alcohol, tricyclic antidepressants and barbiturates. Flurazepam is most likely to produce significant CNS depression. The signs and symptoms that are produced are: (i) Drowsiness (ii) Dizziness (iii) Ataxia and slurred speech (iv) Respiratory depression (v) Hypotension and coma.

Treatment: (i) Supportive measures (ii) Gastric lavage is doubtful (iii) Flumazenil, specific benzodiazepine antagonist in the dose of 0.5 mg i.v over 30secs and further 0.5mg over next 30secs can be repeated, if necessary.

Asphyxiants

Classification of Asphyxiant Gases

Henderson and Haggard divided asphyxiant gases into five groups: (i) **Irritants:** The gases in this group infiltrates the air passages or lungs or both and produces inflammatory changes, such as smoke, ammonia, formaldehyde and chlorine (ii) **Chemical asphyxiants:** These are gases that by combining with haemoglobin or by acting on some tissue constituents prevent oxygen from reaching the tissues, or from using it. These are hydrocyanic acid and carbon monoxide; produce histotoxic anoxia (iii) **Simple asphyxiants:** These are inert gases which, when breathed in high concentration act mechanically by excluding oxygen. These are carbon dioxide and nitrous oxide (iv) **Volatile drugs:** Have little or no irritant effect after absorption; they act as an anaesthetic agent or toxic to the liver, kidney etc. (v) **Systemic poison:** These are insecticides, arsine gas etc.

CARBON MONOXIDE POISONING

Sources of carbon monoxide gas: (i) Carbon monoxide is prepared by the decomposition of organic substances such as oxalic acids and formic acids by means of sulphuric acid (ii) Where there is incomplete combustion of carbonaceous material on carbon, carbon monoxide is produced (iii) It is found in the gaseous products from charcoal fires, salamanders, refineries, blast furnaces, limekilns, gas engines, water heaters, gas refrigerators and burning houses (iv) It is also generated up to 8% in Gunpowder, Dynamite explosion and coalmine explosion (v) In coal gas it is present in 4-10% (vi) When blown in to red-hot coal, it is 30-40% (vii) In diesel engines, exhaust gas of motorcars and trucks, it is 7-13% that is represented

as 1% of carboxyhaemoglobin (viii) In city dwellers and smokers there is 3-5% of carboxyhaemoglobin (ix) The CO produced per minute by 20-horse power motorcar is approximately 1 cubic foot that is enough to render the atmosphere of small closed garage (10 × 20) feet poisonous in less than 10 minutes.

Properties: Carbon monoxide is colourless, tasteless and lighter than air with a garlicky odour. It is a highly poisonous gas that is absorbed in to the lungs and combines with haemoglobin of the RBC in the blood and forms a stable compound carboxyhaemoglobin. The affinity of haemoglobin for Carbon monoxide is 240 times more than that of oxygen.

Mechanism of action: Since Carbon monoxide has 200-300 times greater affinity for haemoglobin than oxygen, it displaces oxygen for haemoglobin and forms a stable compound known as carboxyhaemoglobin. It reduces oxygen content of blood and then of tissues. Since carboxyhaemoglobin does not take part in oxygen transport, symptoms of oxygen deprivation will follow inhalation of the carbon monoxide gas. Carbon monoxide acts as a chemical asphyxiant and it has a direct toxic effect on the myocardium. Since the main action of carbon monoxide is oxygen deprivation and not a toxic manifestation due to the carbon monoxide alone, the important factor is not the level of carboxyhaemoglobin in the blood but the percentage of the haemoglobin that is prevented from carrying oxygen. Normally after somatic death, cells near the capillaries continue to function by extracting oxygen from oxyhaemoglobin and cause blue staining. Nevertheless, in CO poisoning this cannot

occur because the cells cannot break carboxyhaemoglobin compound. In potent cellular poisons such as cyanide, fluoroacetate and freezing break the metabolism of cells so fast that they cannot extract oxygen from oxyhaemoglobin. In these cases, blood under the skin and tissues will remain cherry red due to oxyhaemoglobin. Some people are in a metabolic state in which they can withstand the oxygen lack better than the others are. This is the reason why some persons may recover and live with a level of carbon monoxide that would be lethal to others. It must be taken into consideration also that patients with anemia, coronary artery sclerosis and pulmonary disease are much less able to tolerate oxygen lack.

Medicolegal Aspects

(i) Carbon monoxide poisoning is mostly accidental. The diseased, drugged and drunk persons are involved more in the poisoning. The poisoning usually occurs in ill ventilated rooms where incomplete combustion of wood or coal occurring and the persons' sense of smell and hearing are impaired. It can also occur in dwellings with faulty fittings with leaky gas pipes and taps. In small garages people die from the gas from the motor exhausts. The poisoning can also result in narrow streets where there is dense traffic. Some sexual deviants may also be poisoned when they inhale coal gas. In suspected cases of poisoning air samples should be taken for analyses of Carbon monoxide. In deaths due to fire >50% concentration is of carboxyhaemoglobin. In some victims with blood carboxyhaemoglobin concentration of 15-20%, the symptoms similar to drunkenness are produced (ii) Sometimes Carbon monoxide poisoning may be confused with vehicular accidents (iii) The presence of occasional bullous lesions similar to the 3rd degree burns on the body is diagnostic of CO poisoning (iv) The tendency of the dying person to become wild may cause an impression of violent quarrel, can cause an erroneous suspicion of murder (v) Carbon monoxide poisoning is suicidal mostly in western countries when the room is closed after placing the fire; in the garage the exhaust fumes are inhaled through a pipe or sometimes the gas is inhaled through the gas taps. It is sometimes associate

with suicidal pacts, e.g., the mother ends life along with her children (vi) The poisoning is very rarely homicidal unless drink, drugs or disease incapacitates the person when a gas tap is opened when the person is sleeping in the bedroom (vii) Carbon monoxide with chlorine leads to the formation of carbonyl chloride—phosphene that is war gas (viii) Putrefaction has a little effect on the carboxyhaemoglobin as it is extremely stable, may be detected in the blood after several days even up to seven months after death.

Types of Poisoning

The Carbon monoxide poisoning can be:

1. *Acute poisoning*: The acute poisoning is further divided depending on the percentage of carboxyhemoglobin in the blood. The percentage of CoHb is 10-30 % (mild poisoning), 30-40% (moderate poisoning) and severe (>40%).
2. *Chronic poisoning*: The toxicity depends upon (i) Concentration (ii) Duration of exposure (iii) General health of the person.

Signs and Symptoms

The signs and symptoms run parallel to the percentage of saturation of CO in the blood. The effects produced are those of suboxia. The symptoms are not noticeable until 20% saturation of haemoglobin occurs. The central nervous system is the first to be affected. (1) **When the gas is inhaled in concentrated form:** (i) Sudden weakness and dizziness is followed by coma and death due to respiratory failure (ii) Pale or cyanosed appearance (iii) Colour of the skin is cherry red due to carboxyhaemoglobin; the colour is characteristic of the poisoning but its absence by no means excludes it (iv) Coma may last for 3-5 days, sometimes even longer so bronchopneumonia and oedema of the lungs occur. (2) **When the gas is inhaled in dilute form:** (i) Tightness of the forehead (ii) Dizziness (iii) Agitation (iv) Muscular weakness (v) Emotional instability and confusion (vi) Lethargy (vii) Slurred speech (viii) Dilated and fixed pupil (ix) Coma (x) Sudden apnea and death (xi) In some cases rapid and feeble pulse, low BP and tremors are seen (xii) Myocardial ischemia, infarction and arrhythmias have also been reported. ECG findings

are suggestive of Prolonged PR interval, A-V block, Bundle branch block, Prolonged QT interval and ST depression (xiii) Liver is enlarged (xiii) There is tendency to bleed resulting in retinal haemorrhages and papilloedema (xiv) increased sweating (xv) Glycosuria and albuminuria is there. (3) **When recovered after prolonged illness:** Due to hypoxic changes in the brain, nervous and mental symptoms are produced, that are (i) Cerebral haemorrhage (ii) Encephalitis (iii) Optic neuritis (iv) Retrograde amnesia (v) Mental confusion. (4) There is formation of bulla in CO poisoning, these are discrete and isolated, contains thick and cellular fluid with an inflammatory ring around them. The bullous eruptions can be localized by external pressure and are produced due to sudden hypoxia. (5) The presence of other diseases like respiratory diseases or severe anaemia with barbiturate etc reduces the lethal concentration of the Carbon monoxide. (6) Physical activity during the exposure increases the rate of saturation; children saturate their blood more rapidly due to increased rate of respiration. (7) Depending on the concentration of carboxyhaemoglobin in the blood, following signs and symptoms are produced (Table 44.1).

Diagnosis of Carbon monoxide poisoning: (i) Presence of smell of utility gas on the victim's clothing (ii) Exposure is fairly sudden (iii) Face is livid or sweaty (vi) Cherry red colouration of skin (v) Blood is highly saturated with CO (vi) No anaemia (vii) Cyanosis is present (viii) Cheyne-stroke breathing in comatose patients (ix) In mild cases, little involvement of the cardiovascular system with raised pulse and Blood Pressure (x) In severe cases, low BP; thready and irregular

pulse with extrasystoles (xi) Heart dilates rapidly after exposure and death is due to acute circulatory failure (xii) Pupils are dilated, loss of light reflex occurs late, and other reflexes are decreased (xiii) In deep coma extensor planter response is there, limb reflexes and muscle tone are lost (xiv) There is hyperpyrexia and neurological involvement (xv) In the urine glycosuria and albuminuria occurs (xvi) Bullous eruptions on the skin are seen on the pressure areas (xvii) On spectroscopic examination of blood, the line that is nearer to 'D' is half of the other and is unchanged after the addition of ammonium sulphate.

Concentration of carbon monoxide in the air: In the atmosphere: (i) 0.01%→safe limit (ii) 0.02-0.05%→produces toxic symptoms (iii) 0.25-3.0 or 3.33%→is fatal to an adult within 4 hours.

Chronic Carbon Monoxide Poisoning

This results from the intermittent exposure to the gas in the workers of automobile workshops, gas houses, and steel plant blast furnaces and in the persons staying in ill ventilated rooms for a long time. The following signs and symptoms are produced: (i) Dull frontal headache (ii) Nausea and vomiting (iii) Digestive disturbances (iv) Palpitations (v) Dyspnea (vi) Loss of memory (vii) Anaemia (viii) Visual disturbances (ix) Ataxia (x) Tachycardia (xi) Hepatomegaly (xii) Skin lesions (xiii) Angina (xiv) Mental symptoms (xv) Polycythemia.

Treatment

1. **Prophylactic:** The industry and domestic plants should be properly installed and well

Table 44.1: Signs and symptoms produced depending on % saturation of Carboxyhaemoglobin

CoHb level in % saturation	Signs and symptoms
<10%	No appreciable symptoms
10-20%	Breathlessness on moderate exertion, mild headache & tightness across forehead
20-30%	Throbbing headache, irritability, emotional instability, defective memory, rapid fatigue and lethargy
30-40%	Severe headache, nausea & vomiting, dizziness and confusion and dimness of vision
40-50%	Increasing confusion, hallucinations, severe ataxia like a drunk and rapid respiratory collapse
50-60%	Syncope and coma with convulsions, tachycardia, and tachypnoea
60-70%	Coma is increased with incontinence of urine and feces
70-80%	Prolonged coma, weak thready pulse, absent reflexes and convulsions
>80%	Rapid death from respiratory arrest

maintained. Adequate ventilation should be maintained and special masks should be used,

2. **Therapeutic:** (i) Removal of the patient to fresh air (ii) Clearing of the airway by suction (iii) Mouth to mouth respiration should be given (iv) 100% Oxygen inhalation can be employed through the mask for greater dissociation of carboxyhaemoglobin. Some advocate the use of 5%CO₂ and 95% O₂ (v) In serious cases, 0.5-1% adrenaline sulphate and 5ml of 25% solution Should be given intravenously for half to one hour and repeated (vi) Antibiotics should be given to combat infection (vii) Dehydration should be corrected (viii) In cases of acute heart failure, i.v digoxin to be given (ix) CSF pressure taken by lumbar puncture, if elevated then 500 ml of 10% mannitol i.v and furosemide to be given to relieve cerebral oedema (x) The body should be kept warm by hot water bottles and blankets

(xi) *In cases of severe collapse:* (a) Normal saline should be administered subcutaneously (b) Hypothermia or exchange transfusion may be helpful (c) If cerebral oedema is there, 500 mL of 20% mannitol and corticosteroids should be administered (d) Procaine hydrochloride 500mg in 500ml of 5% dextrose i.v once a day should be given for 7days (e) Cytochrome C should be administered intravenously (f) Patient should be kept in a chamber filled with **hyperbaric oxygen** at two atmospheric pressure; the oxygen dissolved in the plasma is raised from 0.25 to 3.8-vol% and rate of dissociation of CO for haemoglobin is greatly increased. The disadvantages of hyperbaric oxygen are oxygen poisoning, pulmonary oedema and haemorrhages (g) After the breathing is established, tea and coffee is given and antibiotics.

A man when brought to normal oxygen atmosphere, the carboxyhaemoglobin level reduces to half in 4 hours and with 100% oxygen inhalation, the carboxyhaemoglobin concentration reduces to half in just 40 minutes.

Prognosis: (i) A close watch should be kept on the complications like renal failure, gangrene,

pulmonary oedema and progressive paralysis due to hypoxia and local pressure (ii) The neurological sequel is well known but may leave residual disability after 20-30 days (iii) If the degree and duration of hypoxia has damaged vital centers, the period of coma may be prolonged but may be followed by complete or partial recovery (iv) Muscle rigidity and increased tone of limbs in severe poisoning (v) Persons more than 60 years of age and coma for 24 hours usually succumb to its effects (vi) Repeated exposure may lead to polycythemia.

Autopsy Findings

External findings: (i) Bright red colour of the lips, and fingertips (ii) Postmortem staining is cherry red in colour; to be differentiated from cold exposure and cyanide poisoning (iii) Fine froth is present at mouth and nostrils (iv) Muscle necrosis and skin blebs are present.

Internal findings: (i) Blood is cherry red and fluid (ii) Mucous membrane is red in colour (iii) Hyperemia is general and serous effusions are common (iv) Lungs are congested and oedematous, consolidations may be present (v) Heart shows haemorrhages and necrotic changes (vi) Brain has serous effusions in the ventricles, punctiform haemorrhages in the white matter and softening in the cerebral cortex, globus pallidus and corpus striatum. The bilateral symmetrical necrosis of ventricular nuclei is present. Brain is oedematous (vii) Renal failure is the pathological evidence.

CARBON DIOXIDE POISONING

The carbon dioxide intoxication usually results from the physiological disturbances. The gas may disperse slowly if ventilation is poor. The carbon dioxide gas may also be: (i) Formed during fermentation reactions (ii) Found also in grain bins, caves, ships' holds and in fires (iii) Released from water in wells by changes in atmospheric pressure (iv) Released from some fire extinguishers (v) Released from dry ice.

Features of hypoxia are prominent in carbon dioxide poisoning.

Biological and Chemical Warfare Agents

Chemical Warfare

Chemical warfare is warfare (and associated military operations) using the toxic properties of chemical substances to kill, injure or incapacitate an enemy. They are also used in civilian conditions to disperse unruly mobs. Chemical warfare is different from the use of conventional weapons or nuclear weapons because the destructive effects of chemical weapons are not primarily due to any explosive force.

The use of nonliving toxic products produced by living organisms (e.g., toxins such as botulinum toxin, ricin or saxitoxin) is considered chemical warfare under the provisions of the Chemical Weapons Convention. Under this Convention, any toxic chemical, regardless of its origin, is considered as a chemical weapon unless it is used for purposes that are not prohibited.

Biological warfare: The offensive use of living organisms (such as anthrax) is considered to be biological warfare rather than chemical warfare.

Chemical weapon agents: A chemical used in warfare is called a chemical weapon agent (CWA). About 70 different chemicals have been used or stockpiled as chemical weapon agents during the 20th century. These agents may be in liquid, gas or solid form. Liquid agents are generally designed to evaporate quickly; such liquids are said to be volatile or have a high vapor pressure. Many chemical agents are made volatile so they can be dispersed over a large region quickly.

Chemical weapons are classified as weapons of mass destruction by the United Nations, and their production and stockpiling was outlawed by

the Chemical Weapons Convention of 1993. Under the Convention, chemicals that are toxic enough to be used as chemical weapons, or may be used to manufacture such chemicals, are divided into three groups according to their purpose and treatment:

Schedule 1: Have few, if any, legitimate uses. These may only be produced or used for research, medical, pharmaceutical or protective purposes (mustard gas, lewisite).

Schedule 2: Have no large-scale industrial uses, but may have legitimate small-scale uses (dimethyl methylphosphonate, a precursor to sarin).

Schedule 3: Have legitimate industrial uses (phosgene, chloropicrin).

The earliest target of chemical weapon agent research was not toxicity, but development of agents that can affect a target through the skin and clothing, rendering protective gas masks useless. In July 1917, the Germans first employed mustard gas, the first agent that circumvented gas masks. Mustard gas easily penetrates leather and fabric to inflict painful burns on the skin.

Classes of chemical weapon agents: Chemical weapon agents are organized into several categories according to the manner in which they affect the human body. The names and number of categories varies slightly from source to source, but in general, types of chemical weapon agents are as follows.

There are other chemicals used militarily that are not technically considered to be chemical weapon agents, such as:

1. *Defoliants* that destroy vegetation, but are not immediately toxic to human beings. (Agent

Orange, for instance, used by the United States in Vietnam, contained dioxins and is known for its long-term cancer effects and for causing genetic damage leading to serious birth deformities.)

2. *Incendiary or explosive chemicals* (such as napalm, extensively used by the United States in Vietnam, or dynamite) because their destructive effects are primarily due to fire or explosive force, and not direct chemical action.
3. *Biological warfare agents* that includes the viruses, bacteria, or other organisms.

Chemical weapon agents are also divided into lethal and incapacitating categories. A substance is classified as incapacitating if less than 1/100 of the lethal dose causes incapacitation, e.g., through nausea or visual problems. The distinction between lethal and incapacitating substances is not fixed, but relies on a statistical average called the LD₅₀.

Although crude chemical warfare has been employed in many parts of the world for thousands of years, modern chemical warfare began during World War I. Initially, only well-known commercially available chemicals and their variants were used. These included chlorine and phosgene gas. The methods of dispersing these agents during battle were relatively unrefined and inefficient.

Use of Poison Gas in World War I

The French were the first to use chemical weapons during the First World War, using tear gas. The first full-scale deployment of chemical weapon agents was during World War I, originating in the Second Battle of Ypres, April 22, 1915, when the Germans attacked French, Canadian and Algerian troops with chlorine gas.

Chemical Warfare in World War II

During World War II, chemical warfare was revolutionized by Nazi Germany's accidental discovery of the nerve agents tabun, sarin and soman. The Japanese used mustard gas and the recently-developed blister agent Lewisite against Chinese troops. During these attacks, the Japanese also employed biological warfare by intentionally spreading cholera, dysentery, typhoid, bubonic plague, and anthrax.

Developments by the Western Governments

In 1952 the U.S. Army patented a process for the "Preparation of Toxic Ricin", publishing a method of producing this powerful toxin. Also in 1952, researchers in Porton Down, England, invented the VX nerve agent but soon abandoned the project. In 1958 the British government traded their VX technology with the United States in exchange for information on thermonuclear weapons; by 1961 the U.S. was producing large amounts of VX and performing its own nerve agent research. This research produced at least three more agents; the four agents (VE, VG, VM, VX) are collectively known as the "V-Series" class of nerve agents.

Classification of the Warfare Agents

Poisons used as war gases can be classified according to their primary physiological actions as:

Lung irritants (Asphyxiants or choking gases): (i) Chlorine (ii) Phosgene (iii) Chloropicrin (iv) Diphosgene.

Lacrimators (Tear gases): (i) Chloroacetophenon (C.A.P) (ii) Bromo-benzyl cyanide (B.B.C.) (iii) Ethyl-iodo-acetate (K.S.K).

Vesicants (Blister gases): (i) Mustard gas (ii) Lewisite.

Sternutators (Nasal irritants or vomiting gases): (i) Diphenyl-chlorarsine (D.A.) (ii) Diphenyl-amine-chlorarsine (D.M.) (iii) Diphenyl-cyan-arsine (D.C.).

Paralysants (Nerve poisons): (i) Carbon monoxide (ii) Hydrocyanic acid (iii) Hydrogen sulphide.

Nerve gases: These are toxic chemicals having acetylcholine like action.

Lung Irritants

These are irritant gases that exert their main action on the pulmonary alveoli through the upper respiratory passage. Of these, chlorine and phosgene are the gases that are kept liquid under pressure. Chlorine has bleaching powder like smell whereas phosgene has musty odour. These can be released from tanks, gas shells and canisters.

Chloropicrin is a yellowish oily liquid having fly-paper like odour and Diphosgene is also an oily liquid with smell of phosgene. Both phosgene

Agrochemical Poisons

Various pests, fungi, weeds and rodents cause much harm to the production and storage of food grains. A large number of pesticides including insecticides, rodenticides, herbicides and fungicides are available in the market. They enable the farmers to control their crops, from being destroyed by insects, diseases and weeds. As a result, though the yields of food crops have reached new high levels but their extensive use by the agriculturists has resulted in poisoning of human beings and domestic animals.

Fungicidal

Fungicides preserve the seeds for seedling purposes. These are mainly mercurial compounds

Herbicidal or Weed Killers

Since the growth of undesirable herbs and weeds in the field yields low production, the herbicides or weed killers are used to protect the field. These are: (i) Sulphuric acid (1%); is a corrosive acid (ii) Sodium chlorate; is irritant (iii) Potassium cyanide; causes tissue anoxia (iv) Paraquat.

Insecticides

Insecticides are the compounds that are employed for killing insects, are classified as: (1) **Organophosphorus compounds** (i) Alkyl phosphates (ii) Aryl phosphates

2. **Halogenated hydrocarbons** (i) Aldrine (ii) Endrine (iii) DDT
3. **Miscellaneous compounds**: (i) Mercurial salts (ii) Arsenic salts
4. **Rodenticidal** (i) Inorganic compounds (a) Aluminium phosphide (b) Zinc phosphide (c)

Thallium (d) Phosphorus (ii) Organic compounds (a) Fluoro-acetate (iii) Convulsants (a) Strychnine (iv) Anticoagulant (a) Warfarin.

Insecticides are further classified according to their toxicity as:

1. **Virtually Harmless** (i) Pheno-oxalic acid (ii) Copper oxide—fungicide (iii) Lime sulphur—orchard fungicide (iv) Petroleum washes—orchard insecticide
2. **Comparatively Harmless** (i) Sulphuric acid 20%—weed killer (ii) Sodium chlorate—mass herbicide
3. **Mildly toxic (25-60gm)** (i) Chlorinated hydrocarbons (ii) DDT (iii) Gammaxene (iv) Aldrin, dieldrin: used to control flies, louse, tick and as an agricultural insecticide.
4. **Moderately toxic (10-25 gm)** (i) Diazinon
5. **Highly toxic** (i) Sodium arsenite (ii) Lead and calcium arsenate (iii) Organic polyphosphates (iv) Organophosphorus compounds which include (a) HETP (Hexa Ethyl tetra phosphate) (b) TEPP (Tetra Ethyl pyro phosphate) (c) Parathion.

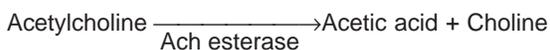
ORGANOPHOSPHORUS COMPOUNDS

Historical considerations: The first organophosphate insecticide was tetraethyl pyrophosphate (TEPP) and it was developed in Germany as a substitute for nicotine, which was in short supply in the country during World War II and in 1944, Parathion was synthesized. They are extensively used as agricultural insecticides being affective against a wide range of insects and pests. Nevertheless, a number of these compounds have proved to be more toxic to human beings, than to the insects and rodents.

Mode of Action

Organophosphates cause some degree of irritation to mucous membrane. They are primarily nerve poisons affecting both autonomic and central nervous system. On autonomic nervous system, it exerts parasympathomimetic action by inactivating cholinesterase. Therefore, both muscarinic and nicotinic action of acetylcholine is increased. It causes depression of central nervous system

Acetylcholine is recognized as mediator of impulses of autonomic ganglia. It is present in an inactive form that bounds loosely with a protein or lipoprotein. Acetylcholine as chemical transmitter plays a vital role to propagate nerve impulse across the synapses of peripheral and central nervous system. Traces of acetylcholine are produced at the myoneural junctions and synapses of the ganglions. On stimulation of the nerve, acetylcholine is set free and diffuses down the nerve fibers. These same nerve fibers contain an enzyme Choline acetylase that is capable of synthesizing acetylcholine with great rapidity. Free acetylcholine is unstable and gets spontaneously hydrolysed to Choline and acetic acid, the hydrolysis getting accelerated by the enzyme, acetylcholinesterase present in the plasma, cell membrane or cytoplasm of many cells of the body. In blood and nerve tissue, cholinesterase inactivates acetylcholine after its liberation as shown below:



Cholinesterases are of two types namely true cholinesterase that is present in blood cells and central nervous system whereas plasma and white matter of brain contains pseudocholinesterase. Usually true cholinesterase inactivates acetylcholine but when excess of acetylcholine escapes in the blood, plasma or pseudocholinesterase also plays a role. Cholinesterases are most effective in the presence of small amount of acetylcholine.

Organophosphorus compounds inactivate acetylcholinesterase by the process of phosphorylation to facilitate the action of acetylcholine. This occurs first at the plasma level then at the cellular level of brain. As a result, there is

accumulation of acetylcholine at the nerve endings resulting in interference at the passage of nerve impulses across the myoneural junctions and synapses of autonomic ganglions. As such, these compounds are known as Cholinesterase inhibitors. Hence, the action of organophosphorus will simulate actions of physostigmine, acetylcholine, Pilocarpine and muscarine. A drop in the activity of cholinesterase to 25-30% of the normal value will be characterized by signs and symptoms compounded as: (i) Muscarinic like effect on the parasympathetic system affecting the postganglionic cholinergic nerve fibres (ii) Nicotinic like actions followed by paralysis of preganglionic sympathetic fibres and somatic motor nerves (iii) Effect on central nervous system with stimulation followed by depression (iv) Porphyrinuria leading to shedding of red tears; **Chromadocryorrhoea** due to accumulation of porphyrin in the lachrymal gland.

The organophosphorus compounds may cause:

1. *Irreversible inhibition of cholinesterase* due to: (i) Direct action—TEPP (ii) Delayed action—Parathion and Malathion
2. *Reversible inhibition of cholinesterase*: (i) Carbamates (a) Carbaryl (b) Mactacil

Organophosphorus compounds are divided mainly in two groups:

- i. **Alkyl phosphate** (a) TEPP—Tetra ethyl pyrophosphate (b) HETP—Hexa ethyl pyrophosphate (c) Dimefox (d) Malathion (e) Sulfotepp (f) Systox
- ii. **Aryl phosphates** (a) Parathion (b) Paraoxon (c) Chlorothion (d) Methyl parathion (e) Diazion (f) Methyl umbeliferone

Routes of Absorption

(i) Absorption through intact skin (ii) Respiratory system by inhalation (iii) Gastrointestinal tract by ingestion (iv) Circulatory system by intra muscular and intravenous route.

Signs and Symptoms

1. *Muscarinic like effects*: Muscarinic receptors for acetylcholine are found primarily in the smooth muscles, the heart, and exocrine glands. The muscarinic like effects produced by the organophosphorus compounds are: (i)

Tightness in the chest (ii) Wheezing respiration (Expiration due to bronchoconstriction) (iii) Increased bronchial secretion (iv) Increased salivation (v) Increased sweating (vi) Tone and peristalsis is increased (vii) Nausea, vomiting and abdominal cramps (viii) Diarrhoea (ix) Tenesmus and involuntary defecation (x) Bradycardia and heart block (xi) Frequent and involuntary urination due to constriction of smooth muscles of bladder (xii) Constriction of pupil causing miosis

2. *Nicotinic like symptoms*: Nicotinic signs and symptoms result from the accumulation of acetylcholine at the endings of motor nerves to skeletal muscle and autonomic ganglia. The effects include: (i) Easy fatigability and mild weakness followed by involuntary twitching, fasciculation and cramps (ii) Muscles of respiration are affected leading to Dyspnea and cyanosis. Nicotinic action at autonomic ganglia masks the muscarinic effects, thus: (iii) Tachycardia is produced instead of bradycardia (iv) Pallor (v) Blood pressure is raised (vi) Hyperglycemia (vii) Cardiac arrhythmias (viii) Pulmonary oedema.
3. *Accumulation of acetylcholine in central nervous system* (i) Anxiety, tension and confusion (ii) Restlessness, insomnia and headache (iii) Irritability and neurosis (iv) Excessive dreaming and nightmares (v) Slurred speech and tremors (vi) Ataxia and convulsion (vii) Depression of respiratory and circulatory centers (viii) Coma leading to death.

Fatal Dose

TEPP is most toxic whereas HETP is the least toxic comparatively as 5 mg i.m or 25 mg taken orally of TEPP produces toxic symptoms. The fatal dose of various organophosphorus compounds are:

- TEPP→50mg i.m or 100mg oral
- Parathion→80mg i.m. or 100-175mg oral
- HETP→160mg i.m. or 350 mg oral
- OMPA→ 80 mg i.m. or 175 mg oral
- Malathion & Diazinon (Tik20)→1gm oral.

Fatal Period→In fatal doses symptoms start within half an hour and death occurs within three hours. In non-fatal cases, effects last for 36 hours and fade off in 48-72 hours to 3 weeks. Blood cholinesterase level drops to 22-28% that is up to 50% of the normal value. Normal value is 77-142 units in red blood cells that is more appropriate than the plasma levels of 41-140 units. About 3% of the population suffers from congenital cholinesterase deficiency and the level decreases in patients with severe liver disease.

Diagnosis

1. *Cholinesterase level in blood*: As organophosphates inhibit cholinesterase activity, level of cholinesterase should be estimated in the blood. The normal level of cholinesterase is about 40 - 142 in red cells and 90- 140 in the plasma. In organophosphorus poisoning, the cholinesterase activity of blood and plasma falls by 25- 85 % of its normal value. Periodic measurements of blood cholinesterase level helps in detection of poisoning because marked depression of cholinesterase activity of blood occurs, before the symptoms become evident. Normally the symptoms may not appear until the level of cholinesterase falls by 20% or less. Five ml of heparinised blood should be collected for estimating level of cholinesterase in blood.
2. *The cholinesterase level in the motor end plate*: The cholinesterase level can be demonstrated histochemically in muscles kept at room temperature for 1–2 days and up to several months when kept at 4°C. The cholinesterase activity at the myoneural junction is not affected by embalming the body or if the tissue is fixed by formalin or acetone up to 24 hours.
3. *Test by atropine*: When 2 mg of atropine is given intravenously, there are signs of atropinisation in a normal individual, while in a case of organophosphorus poisoning it will have no effect.

Treatment

1. Removal the patient from the source of exposure.

2. Strip off contaminated clothing
3. Wash all parts with soap and water under the shower bath.
4. Give life supporting measures such as oxygen inhalation
5. Gastric lavage with 2% potassium permanganate
6. Purgatives like sodium or magnesium 30gm orally
7. Dehydration and shock must be treated
8. Diazepam intravenously should be given to control convulsions
9. Morphine and aminophylline are not to be given
10. Exchange transfusion: It takes 2-3months for complete recovery after exchange transfusion. Blood cholesterol level should be checked every 15days
11. **Atropine sulphate** is the readily available best-known antidote against all organo-phosphorus compounds. It inactivates acetylcholine, arrests muscarinic and other effects but not the nicotinic action. Atropine sulphate, 2-4mg is given I.V or I.M in adults and 0.05 mg/kg in children. The dose is to be repeated as 2-4mg i.m. every 20-30 minutes until full atropinization is achieved. Then the dose has to be decreased and given less frequently. The repetition of dose of atropine depends upon severity of case:
 - In mild cases, 2-4mg i.m/i.v. initially that is to be repeated after 20 minutes as 2mg and then repeated again.
 - In moderate cases, 2-4mg i.m/i.v. that is to be repeated after 10 minutes interval.
 - In severe cases, 4-6 mg i.m/i.v. that is to be repeated after 3-10 minutes interval and then 50 mg is administered in 24 hours.
 - Usually 40-50 mg may be required during first 24hours and 12 mg may be given safely in the first 6 hours.

Signs of Atropinization (i) Flushing of face (ii) Dry mouth and dry, warm skin (iii) Blurring of vision (iv) Dilatation of pupil (v) Rapid and irregular pulse (vi) Loss of neuromuscular coordination

Precautions while Atropinization: (i) Adequate oxygen therapy should be given simultaneously to prevent myocardial infarction (ii) The drug should be withdrawn judiciously there may be rapid development of pulmonary oedema and respiratory (iii) Atropine is ineffective against autonomic ganglionic actions of acetylcholine and peripheral neuromuscular paralysis (iv) To combat overdose of atropine, physostigmine salicylate should be administered in the dose as given below:

 - In adults, 1-2 mg i.m/iv; to be repeated up to a total of 6 mg.
 - In children, 0.5 mg i.m/iv; to be repeated up to a total of 2 mg.

The dose of atropine should be reduced when oximes are given together.
12. **Specific Reactivator Oximes:** Relief of muscle weakness in particular respiratory paralysis can be achieved with certain Oximes, which can reactivate the cholinesterase by reversing the phosphate ester bond formed at the enzyme active site (Cholinesterases and organic phosphates are bound by phosphate bond). They should be used within 24 hours. They are superior to atropine but are not to be administered in Carbamate poisoning:
 - i. **PAM (Pralidoxime chloride):** Protopam chloride is to be given 1gm I.V. over a period of 2 minutes or in 250 mL of normal saline infusion for 20-30 minutes that is to be repeated after 8-12 hours. In children 25-50 mg i.m or i.v/kg, body weight is to be given within 2 minutes or as an infusion for 20-30 minutes
 - ii. **P₂AM (Pyridine-2-aldoxime methiodide)** can be given as 50 mg/kg in one litre of normal saline slowly to be followed by another litre of same infusion over a period of 12 hours.
 - iii. **DAM (Diacyl monoxime)** can be given as 2 gm in one litre of normal saline slowly

- iv. *P₂S (Pyridine-2-aldoxime methane sulfonate)* in the dose of 1-2 gm in adults and 25-50mg/kg in children. The dose is to be repeated every 12 hours.

Adverse Reactions of Specific Reactivator

Oximes: (i) When given intravenously, it produces tachycardia and muscular rigidity (ii) When given intramuscularly, conjunctival hyperemia is produced (iii) When given orally nausea, vomiting and diarrhoea may result.

Autopsy Findings

(i) Findings of asphyxia such as congestion of face and cyanosis of lips, nails and mucous membranes is present (ii) At times blood-tinged froth is found over the nostrils and mouth (iii) Smell of kerosene may be present on opening the oesophagus and stomach (iv) The mucosa of stomach is congested and submucosal petechial hemorrhages are present (v) Frothy fluid at times tinged with blood may be found in the trachea and bronchi (vi) The lungs show congestion and gross edema and submucosal hemorrhages (vii) The brain shows petechial hemorrhages, is congested and oedematous and the meninges are dilated.

Prophylaxis from Organophosphorus Poisoning

(i) The body should be properly protected while spraying in the fields with aprons, gloves and masks (ii) The agriculturist should not spray for more than 2-4hours a day and should not work for more than 6-7 successive days (iii) He should not eat, drink, chew or smoke while spraying (iv) After the operation, the face, hands, feet, eyes should be washed thoroughly with soap and water (v) Even the spraying machines, tanks or containers and hoses should be washed after each operation (vi) When the worker is suffering from upper respiratory tract infection, he should restrain from spraying

Medicolegal Aspects

(i) Accidental poisoning usually during manufacturing, spraying or dusting in the field and when mixed with other food stuffs. In the Kerala food poisoning tragedy in 1958, about 100 people died

due to inadvertent stocking of foodstuffs and parathion (ii) Less frequently used as homicidal poison (iii) Most commonly used for suicidal purposes next to barbiturates due to their easy availability, quick action, are cheap and can be easily purchased for domestic use.

PARAQUAT POISONING

Uses: Paraquat is an herbicide that is sprayed on unwanted weeds and other vegetation before planting crops. It is absorbed by the foliage and rapidly kills the plant, but is inactivated when in contact with the soil, so cannot harm the seeds or young plants that are placed in the same ground a short time later.

Properties: Paraquat and the Diquat are both dipyridyl compounds and paraquat is 1 : 1 dimethyl 4 : 4 bipyridium. It is produced commercially as a brownish concentrated liquid of the dichloride salt at 10-30% strength. Both are water soluble herbicides supplied in 20-25% concentration in bulk to the farmers and horticulturists. Its is marketed under the trade name gamaxone or weedol.

Toxicity: (i) Ingestion most commonly (ii) Inhalation through spraying of liquid stored in soft drink bottles (iii) Skin contamination.

Fatal dose→4 mg/kg

Fatal period→3-5 days.

Signs and Symptoms

(i) Paraquat is irritant to epithelial tissues in concentrated form and causes erosion of the mucous membrane of the lips, mouth, pharynx and oesophagus (ii) The lungs are usually affected because of aspiration of irritating liquid while regurgitation or swallowing (iii) Renal failure may develop within 2-3 days (iv) Death is due to failure of vital organs like liver, kidney. At times oesophageal perforation occurs resulting in mediastinitis.

Laboratory Findings

(i) Elevation of blood urea, serum alkaline phosphatases and bilirubin are characteristic of kidney and liver damage (ii) Serum trypsin inhibitor gets depressed indicating extent of damage to lungs.

Treatment

(i) Stomach wash using 4-7% bentonite-magma solution and repeated with caution and some of it is left in the stomach (ii) Activated charcoal should be given orally (iii) No known antidote (iv) Forced diuresis as early as possible to prevent renal damage (v) Hemodialysis to remove any poison left after forced diuresis (vi) Glucocorticoids for progressive lung proliferative lesions (vii) Supportive therapy (viii) Fullers earth or 7 % bentonite should be given.

Autopsy Findings

(i) Centrilobular necrosis of the liver with giant mitochondria and crystalline inclusion bodies are seen on electron microscopy (ii) Diffuse tubular damage of the kidneys (iii) Proliferative damage to the lungs is characteristic lesion of paraquat poisoning. When paraquat reaches the distal air spaces, diffuse pulmonary oedema and haemorrhages occur. Ulceration of mouth and lips may be present.

Medicolegal Aspects

(i) Suicidal by deliberate self ingestion of the liquid (ii) Accidental resulting from ingestion.

CHLORINATED HYDROCARBON INSECTICIDES

D.D.T (Chlorophenothane) is the most commonly used chlorinated hydrocarbon insecticide in agriculture and public health. The commonly used agents are Endrin, Aldrin, Chlordane, Dieldrin, Lindane (Gammexene), Methoxychlor, Toxaphene and D.D.D (Dichloro-diphenyl-dichloroethane). Even though some of the compounds individually exhibit differences in chemical configuration and physiological actions, from chemical standpoint, their effects on human beings are essentially the same and hence can be described in a group. Poisoning by Endrin is more common than D.D.T in rural India.

D.D.T (Dichloro-diphenyl-trichloroethane)

Properties: D.D.T is white crystalline powder with faint aromatic odour. It is insoluble in water but

soluble in kerosene, vegetable oil and other organic solvents. While dusting as powder, it is to be diluted to 10% with powdered chalk or kaolin. It can be used as spray in 5% solution diluted with suitable solvents.

Uses: D.D.T acts as both insecticide and parasiticide to kill mosquitoes, flies, bed-bugs, lice, arthropods and other insects. They usually become resistant to D.D.T later on.

Routes of Absorption: In pure solid form, it does not get absorbed through skin but when it is dissolved in kerosene oil or other solvents, it gets easily absorbed through the gastrointestinal tract, skin and lungs. A 2% solution of D.D.T acts as poison to human beings and its solution in fatty acids increase the toxicity. D.D.T powder is safe for dusting animals.

Mode of action: (i) It first stimulates then depresses the cerebellum and motor cortex (ii) It sensitizes the myocardium to the action of catecholamines, so that injection of epinephrine may induce ventricular fibrillation (iii) It depresses adrenal cortex causing irritation and sensitization of the skin (iv) Locally it acts as an irritant, when swallowed (v) Chronic exposure causes liver necrosis, degeneration of renal tubules, myocardium and voluntary muscles

Fatal Dose

- D.D.T. → 30 gm or 0.5gm/kg
- Lindane → 15 gm
- Chlordane → 30 gm
- Methoxychlor → 350 gm.

Acute Poisoning

The signs and symptoms develop within half an hour of ingestion of the poison: (i) Salivation, nausea, vomiting and abdominal pain (ii) Apprehension, hyperexcitability, restlessness, nervousness (iii) Tinnitus and vertigo (iv) Blurred vision with twitching of eyelids that is followed by muscular tremors, first affecting the head and neck, then involving the extremities (v) Tonic clonic convulsions are characteristic of D.D.T, Aldrin, dieldrin and lindane poisoning (vi) Generalised epileptiform convulsion occur in toxaphene poisoning (vii)

Rarely muscular weakness, ataxia, incoordination, numbness and paralysis may occur (viii) Pulmonary oedema, respiratory distress, Fall of B.P, delirium and unconsciousness, coma and collapse (ix) Death occurs from respiratory failure.

Chronic Poisoning

After absorption, these insecticides gets accumulated in body fat and concentration gradually falls over several months. The workers with history of many months of exposure having more than 648 ppm of D.D.T in their fat suffer from chronic poisoning. The signs and symptoms are: (i) Anorexia, nausea and vomiting (ii) Cachexia, loss of weight, anemia (iii) Anxiety, headache, hyperirritability (iv) Blurred vision (v) Tremors, convulsions and coma (vi) Liver moderately enlarged.

Treatment

(i) Immediate gastric lavage with 0.2% potassium permanganate (ii) Emetics should be given (iii) No known antidote of D.D.T (iv) Cathartics, fats or oils should be avoided as they promote absorption (v) Atropine sulphate should be administered (vi) Oxygen inhalation and artificial respiration (vii) To control muscular twitching, tremors and convulsion, Thiopentone sodium 100-250mg i.v followed by i.m injection of 100mg of Phenobarbital sodium (viii) Calcium gluconate 10mL of 10% with glucose i.v and calcium lactate orally are beneficial (ix) Adrenaline, epinephrine should not be used as they induce ventricular fibrillation. Morphine is also avoided (x) For skin contamination, thorough washing with soap and water (xi) In chronic poisonings, the patient should be removed from the site of exposure and given low fat, high protein & carbohydrate diet. The treatment is symptomatic.

Autopsy Findings

(i) Stomach contents may smell of kerosene and are blood-stained (ii) Mucosa of stomach and intestines is congested and haemorrhagic (iii) The liver is enlarged with fatty degeneration (iv) The lungs are congested and oedematous (v) Spleen, brain and kidneys are congested.

ENDRIN

Properties: Endrin is the most toxic of all the chlorinated hydrocarbons. It is 12-15 times more toxic than D.D.T and 3 times more toxic than Aldrin and dieldrin. It is insoluble in water, sparingly soluble in alcohol and freely soluble in aromatic hydrocarbons and fats. It remains stable for months or years.

Uses: Several preparations containing endrin are sold in the market in 20-25% concentration mixed with petroleum hydrocarbon. They all smell like kerosene. Endrin is also known as plant penicillin as it is a popular pesticide against insect pests of cotton, paddy, sugarcane and tobacco. It is used as spray in solutions, or as dust mixed with some other inert powders. It is marketed under the trade name Endox DR - 50 Trafdrine and Endrex.

Absorption: Endrin is not absorbed through intact skin in dry powder state and poorly absorbed from the gastrointestinal tract mucosa but when it is dissolved in oil it gets freely absorbed from skin as well as mucous membrane

Fatal dose→5-6gms

Fatal period→Usually 1-2 hrs (1-2 days rarely)

Mode of action: (i) Has no effect on cholinesterases (ii) It is neurotoxic primarily acting on central nervous system, causing respiratory failure.

Signs and Symptoms

(i) Salivation, nausea, vomiting and abdominal discomfort (ii) Apprehension, hyperexcitability, restlessness, nervousness, incoordination and ataxia followed by mental confusion and delirium (iii) Profuse frothing at mouth and nostrils (iv) Tonic clonic convulsions (v) Unconsciousness, coma and collapse and death from respiratory failure

Diagnosis: to diagnose, chemical examination of blood and subcutaneous fat is undertaken

Treatment

(i) Immediate gastric lavage with 0.2% potassium permanganate (ii) Emetics should be given (iii) There is no known antidote of endrin (iv) Cathartics, fats or oils should be avoided as they promote

absorption (v) Atropine sulphate should be administered (vi) Oxygen inhalation and artificial respiration (vii) To control convulsions, Thiopentone sodium 100-250mg i.v. followed by i.m injection of 100mg of Phenobarbital sodium should be given (viii) Calcium gluconate 10mL of 10% with glucose I.V. and calcium lactate orally are beneficial (ix) Adrenaline, epinephrine should not be used as they induce ventricular fibrillation. Morphine is also avoided (x) For skin contamination, thorough washing with soap and water (xi) In chronic poisonings, the patient should be removed from the site of exposure and given low fat, high protein and carbohydrate diet. The treatment is entirely symptomatic.

Autopsy Findings

(i) Congestion of face (ii) Cyanosis of lips and fingertips (iii) Conjunctiva is congested and pupils are dilated (iv) Blood stained frothy fluid discharge smelling of kerosene coming out of mouth and nostrils (v) Gastrointestinal tract is congested and stomach contents are bloodstained smell of kerosene is present (vi) Respiratory tract mucosa is congested, oedematous containing blood-stained frothy fluid mixed with mucus (vii) Lungs are congested and oedematous with subpleural ecchymotic patches (viii) Evidence of hepatic and renal tubular degeneration (ix) Blood remains fluid and dark for long (x) Subpericardial and subendocardial haemorrhages are seen (xi) Endrin resists putrefaction for a long time that is why it is be detected in the viscera for long after death.

Medicolegal Aspects

(i) For homicidal purposes, it may be given mixed with sweets, food and alcohol to mask its smell (ii) Suicidal use is quite common inspite of its unpleasant smell and painful death (iii) Accidental poisoning occurs commonly from skin contamination while spraying.

NAPHTHALENE (TAR, CAMPHOR, C₁₀H₈)

Properties: It occurs as large white crystalline plaques from coal tar distillation having a char-

acteristic odour. It is insoluble in water but freely soluble in ether, chloroform, alcohol and oils. It sublimates at ordinary temperatures.

Uses: (i) It is used commercially as moth repellent, wool preservative and as deodorant in lavatories (ii) It is also used in the manufacture of indigo and azo dyes (iii) Chlorinated naphthalene is used for giving insulated coating on electrical wires and other electrical equipments.

Mode of action: It produces haemolysis, causing blockade of renal tubules by precipitated haemoglobin and may cause hepatic necrosis. Only the patients with deficiency of glucose-6-phosphate-dehydrogenase in the red cells resulting in low level of reduced glutathione and increased susceptibility to hemolysis by the metabolites of naphthalene. In children below 6 years absorption occurs quickly.

Fatal dose→2 gm

Fatal period→few hours to 2-3 days.

Signs and Symptoms

On ingestion of naphthalene: (i) Nausea, vomiting, diarrhoea, abdominal pain and hepatomegaly (ii) Burning micturition, pain in loin and hypogastrium, oliguria and haematuria (iii) Urine is dark brown containing albumin, casts and haemoglobin (iv) Acute nephritis, haemolytic anemia, jaundice and optic atrophy are characteristic features (v) Profuse sweating (vi) Headache, excitement, delirium, muscular twitchings and staggering gait (vii) Cyanosis, tachycardia and drowsiness resulting in coma, collapse and death (viii) Anemia and leucocytosis on hematological examination.

On inhalation of naphthalene vapours: (i) Headache, malaise and tinnitus (ii) Photophobia, conjunctivitis, retina and choroid plexus are affected (iii) Nausea, vomiting (iv) Anemia and jaundice (v) Severe hyperthermia due to the formation of naphthylamine

Contamination of skin: (i) Dermatitis with scaling-weeping-crusting of skin and pruritis (ii) Conjunctivitis (iii) Headache (iv) Anemia, jaundice and hematuria

Treatment

(i) Stomach wash with warm water to be followed by one ounce of saturated solution of magnesium sulphate (ii) Oily and fatty preparations should be avoided as dissolve the poison (iii) Whole blood transfusion followed by sodium bicarbonate 15gm orally every 4 hours or exchange transfusion will be helpful in nervous complications (iv) 5% Dextrose saline i.v for anuria (v) Glucocorticoids should be administered as they prevent naphthalene hemolysis (vi) Diet rich in proteins, carbohydrates and vitamins but poor in fat to be given.

Autopsy Findings

(i) Skin is pale and yellow due to anemia and jaundice (ii) Gastrointestinal tract mucosa is inflamed, congested and tinged yellow (iii) Larynx and trachea contains blood stained froth (iv) Kidneys are congested (v) Liver is enlarged and congested and showing features of fatty degeneration.

Medicolegal Aspects

(i) Accidental poisoning is common in children as they ingest naphthalene balls and in adults resulting from inhalation of naphthalene vapours due to heavy dusting of bed clothes (ii) Very few cases of suicide using naphthalene balls have been reported (iii) Homicidal poisoning is also quite rare.

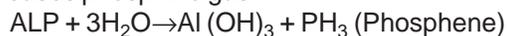
ALUMINIUM PHOSPHIDE

Aluminium phosphide tablets are marketed under the names of 'Celphos', 'Alphos' or 'Sulphas'. It is known as the agent of sure death. A single grayish green tablet weighs 3gm mixed with urea and ammonium carbonate. Each tablet releases 1 gm of phosphine gas (PH₃)

Fatal dose→4-5 gm (1-3 tablets)

Fatal period→12-36 hours.

Mechanism of action: On exposure to air and moisture, the aluminium phosphide tablets releases phosphine gas



Uses: Aluminium phosphide is widely used as fumigant to destroy grain insects and rodents. The tablets are removed from airtight containers and mixed with grains that on contact with moisture

and air, liberates phosphine and thus fumigates the grains.

Signs and Symptoms

Phosphine when inhaled by human beings results in severe pulmonary oedema. Due to the release of phosphine gas in contact with moisture, symptoms of severe gastrointestinal tract irritation are produced that cause cardiovascular collapse and death. The aluminium phosphide poisoning has a high mortality.

The signs and symptoms produced because of aluminium phosphide poisoning are: (i) Nausea, vomiting and burning pain in the epigastrium: (ii) Garlicy (fishy) odour from breath (iii) Intense thirst and diarrhoea (iii) Tightness in the chest and dyspnoea (iv) Excitement and agitation (v) ECG abnormalities—Sinus tachycardia, bradycardia and heart block (vi) Oliguria (vii) Arrhythmias (viii) Hypocalcaemia tetany, convulsions and coma (ix) Shock (xi) Hypotension (xii) Myocardial damage (xiii) Cold clammy skin with respiratory distress in terminal stages (xiv) Rarely muscular wasting, bleeding diathesis, cardiac complications like pericarditis, congestive cardiac failure (xv) Death occurs due to cardiac failure

Chemical tests: The patient is asked to breath on a piece of filter paper in the form of mass impregnated with 0.1 N silver nitrate solution for a period of 5-10 minutes. If the paper is blackened, it indicates presence of phosphine in breath as silver nitrate is reduced to silver on exposure to phosphine.

Management

(i) There is no specific antidote of aluminium phosphide (ii) Early stomach wash using 3-5% sodium bicarbonate, 1% copper sulphate, 1% potassium permanganate or mineral oil should be carried. All these compounds convert it to phosphate within half an hour. Recent studies advocate not to carry out stomach wash because in contact of moisture, the remaining aluminium phosphide in the stomach is converted in to phosphine (iii) *Magnesium sulphate* is given i.m, or I.V. to correct arrhythmias as 3gm bolus followed

by an infusion of 6gm for 24 hours for a period of 5-7 days (iv) Calcium salt is given intravenously (v) Steroids are administered to combat pulmonary oedema (vi) Low dose dopamine infusion to be given (vii) Antibiotics are given to treat infection (viii) To combat metabolic acidosis, sodium bicarbonate 50mEq is given ever 15 minutes until arterial bicarbonate level increases above 15mmol/L (ix) Oxygen inhalation and artificial respiration to maintain respiratory distress.

Distribution of magnesium in the body: In adults, 2000 meq of magnesium is present in the body, of which 60% is concentrated in the bones and 40% in the soft tissue. The Plasma concentration of magnesium is 1.5-2.2meq/lit and 4-8meq/day is excreted through the kidney in to urine, milk, saliva and gastrointestinal tract. In cardiovascular system high concentration of magnesium of about 10-15meq/litre produces symptoms similar to hyper alkalinity in where conduction time is increased and PR interval and QRS complex is lengthened. More than 15meq/litre of magnesium causes cardiac arrest. Hyper-magnesemia due to renal insufficiency results from the decreased excretion of magnesium. The manifestation starts at 12-15meq/litre level resulting in respiratory paralysis. The above concentrations can vary independently and deficit in one compartment may not be accompanied by a significant change in the other.

Plasma concentration and muscle biopsy is required to diagnose magnesium deficiency, but exchange of magnesium does not occur between bone, plasma and intracellular components. In patients with the renal failure the administration of magnesium is of little value as there is risk of hypermagnesemia.

Incidence of deaths from Aluminium Phosphide poisoning: There is a drastic increase in mortality from aluminium phosphide poisoning especially in Haryana, Punjab, Uttar Pradesh and Rajasthan since the last 20-25 years. In 1989, 14 cases are reported of which 4 are accidental deaths and 10 suicidal. In 1990, 9 cases were reported of which 6 are suicidal and 3 accidental. In 1991, 12 cases were reported of which 6 are accidental, 4, suicidal, and 2 homicidal deaths.

Autopsy Findings

(i) Cyanosis is present (ii) In few cases, froth over the mouth and nostrils are present (iii) On opening the body, fishy or garlicky smell of phosphine gas is present (iv) All the internal organs are congested and showing evidence of petechial haemorrhages (v) Lungs are congested and oedematous; In a study, pulmonary oedema is well established in 30 cases resulting from irritation from the phosphine gas.

Organic Solvents

Organic compounds used in industry or at home may result in death from their careless use for improper purposes. Environmental exposure occurs from improper disposal and leakage from toxic dump sites resulting in contamination of drinking water. The various compounds are: (i) Gasoline (ii) Lighter fluids (iii) Aerosol sprays (iv) Spot removers (v) Paint removers (vi) Floor/ tile cleaners.

The compounds such as anaesthetic agents, solvents and fluorohydrocarbons are used as propellants in aerosol products.

SOLVENT ABUSE

Solvent abuse is deliberately inhaling a variety of substances especially organic solvents that results in symptoms similar to alcoholic intoxication

The distortion of consciousness, euphoria (sometimes of erotic nature), distortion of perception and actual hallucinations are produced.

Predominantly males of 14-22 years to beyond the either end or solitary users are commonly involved. Most common substance inhaled was toluene-based adhesive that is why it is also called 'Glue sniffing'

Common Method of Solvent Abuse

1. A solvent or chemical is taken in a plastic bag; the open end is placed against the nose and mouth and air re-breathed. The warm air results in vaporization of solvent, enters tidal air stream and absorbed through pulmonary membranes into blood.
2. The inhalant is placed on the hanky and inhaled directly as pad over nose and mouth. Gaseous substances (butane or propane) are taken from

either a large cylinder used for camping, heating or cooking or from small ampoule cylinders sold for refilling gas cigarette lighters are introduced into mouth or nose.

3. They may be used directly from pressurized aerosol cans or pain relieving sprays. They can be derived from tins, jerry cans and petrol filters of motor vehicles. Fire extinguishers contain an organic bromine derivatives that when inhaled sensitizes the myocardium to catecholamines.

Various Compounds Used For Solvent Abuse

I. Aliphatic Hydrocarbons

C1-C4 Aliphatic hydrocarbons: Straight chain hydrocarbons with ≤ 4 carbon atoms are present in:

- *Natural gas (Methane and ethane):* Are simple Asphyxiants, act by high concentration in air and decreased oxygen concentration
- *Bottled gas (Propane and butane):* Are used directly from pressurized containers, are light hydrocarbons from upper fractions of oil distillation

C5-C8 Aliphatic hydrocarbons: Like most organic solvents it depresses CNS causing dizziness and incoordination:

- n-hexane is a widely used solvent contained in glue and polyneuropathy is primary toxic reaction.
- 2-hexane (methyl n-butyl ketone) is metabolized to 2,5 hexanenedione binds to amino groups of neurofilaments, aggregates and axonal swelling is produced.

Clinical symptoms: There is symmetrical sensory dysfunction of distal portion of extremities, muscle weakness in toes and fingers and loss of deep sensory reflexes.

Mechanism: Peripheral neuropathy associated with exposure to these solvents is due to cytochrome P450-mediated biotransformation of n-hexane and 2-hexane to 2,5 hexanedione.

II. Gasoline and Kerosene

Gasoline and kerosene are petroleum distillates prepared by fractionation of crude petroleum oil. They contain aromatic, aliphatic and variety of branched and unsaturated hydrocarbons. They are used as illuminating fuels, heating fuels, motor fuels, and vehicles for many pesticides and cleaning agents. Accidental poisoning occurs due to being preserved in containers for beverages and is common in children. Chronic exposure to gasoline (2% benzene) results in leukemia.

Signs and Symptoms

Signs and symptoms are similar to that of ethyl alcohol poisoning such as: (i) Incoordination (ii) Restlessness (iii) Excitement (iv) Confusion (v) Disorientation (vi) Ataxia (vii) Delirium and coma (viii) Severe irritation of skin with resulting chronic eczematoid dermatitis and causes second degree burns of skin if left in contact for some time (ix) Gasoline and kerosene may sometimes be inhaled or 'sniffed' resulting in elation and hypoxia (x) When inhaled, the toxic effects are produced on the tracheobronchial tree resulting in chemical tracheobronchitis and pneumonitis.

Inhalation of high concentration of gasoline vapours by workman cleaning storage tanks leads to immediate death (a) Sensitive myocardium such that small amounts of circulating epinephrine precipitate ventricular fibrillation (b) Rapid depression of Central nervous system and death from respiratory failure (c) Inhalation of high concentration for hours results in pneumonitis.

Cause of Death

(i) Sudden cardiac arrest following arrhythmia (ii) Persistent rebreathing in toxic environment results in hypoxia or hypercapnia (iii) Plastic bag asphyxia (iv) Aspiration of vomitus.

Poisoning results from inhalation of vapours and ingestion of liquid. Ingestion of liquid is more hazardous due to low surfaces tension of liquid. The liquid is aspirated into respiratory tract by vomiting or eructation with resulting chemical peritonitis, secondary bacterial pneumonia and pulmonary oedema. The last two are the most serious sequel to aspiration. Death occurs within 16-18 hours.

Autopsy findings

1. Lungs are heavy, oedematous, haemorrhagic and congested.
2. Alveoli are filled with an exudates that is rich in proteins, cells and fibrin
3. Alveolar walls are weakened thus rupture occurs with resulting emphysema and Pneumothorax
4. Inflammation and enlargement of pulmonary lymph nodes is present

Radiological findings

On X-ray examination many small areas of pneumonitis similar to virus pneumonitis, except that the areas of infiltration; nimbus-like clouds in the chest may give the impression of consolidation.

Treatment

Treatment mainly consists of symptomatic and supportive care.

Medicolegal Aspects

1. Accidental poisoning due to being preserved in containers for beverages.
2. Chronic exposure to gasoline (has 2% benzene) results in leukemia.
3. It is commonly used for glue sniffing.

Miscellaneous Poisons

ANALGESICS AND ANTIPYRETICS

ACETYLSALICYLIC ACID (ASPIRIN)

It is white powder commonly used as an antipyretic and analgesic.

Signs and symptoms

(i) burning pain (ii) vomiting, occasional diarrhoea (iii) sweating (iv) rise of temperature (v) dyspnea (vi) vertigo (vii) ringing in the ears and deafness (viii) impairment of vision (ix) headache (x) restlessness (xi) confusion (xii) delirium and hallucinations (xiii) pupils are dilated.

Blood level of 50 mg% are toxic whereas of 100 mg% are fatal to life.

Fatal dose→15-30 gm

Fatal period→few minutes to several hours

Death rate is 7-8%.

Causes of Death

(i) Uremia leading to circulatory failure (ii) Respiratory failure

Treatment

Stomach wash with sodium bicarbonate:

- Dialysis
- Exchange transfusion
- Symptomatic treatment.

Autopsy findings: (i) Pupils are dilated (ii) Skin rashes may be present (iii) Gastric mucosa is congested (iv) Subpleural haemorrhages (v) Lungs are congested and oedematous (vi) Hepatitis (vii) Myocardium, liver and kidney are soft and greasy to touch, if the survival rate is for few days.

Medico-legal aspects: (i) Almost always suicidal (ii) Not accidental due to bitter taste.

PARACETAMOL

Paracetamol is also known as acetaminophen, N-acetyl-p-aminophenol or 4'-hydroxyacetanilide. It is an analgesic and antipyretic, without the anti-inflammatory properties of aspirin, for which it is often used as an alternative because of the lack of gastric irritation.

In combination with other drugs such as codeine and dextropropoxyphen, Paracetamol is one of the most common agents used in self-induced poisoning by medicinal products. It is used alone in therapeutic doses of up to 500 mg. overdoses of 20 g or more are potentially lethal, but much less is needed in combination with other drugs, such as propoxyphen.

Paracetamol poisoning is more common in children who have low hepatic glucuronide conjugating ability. When a large dose (>150mg/kg or >10g in an adult) is taken, it causes serious toxicity. Fatality is common with doses >250mg/kg. Paracetamol is a potent liver poison, as a small portion is converted by the liver enzyme 'P450' (microsomal mixed function oxidase) into a toxic compound, probably N-acetyl-p-benzoquinone. Normally glutathione and other sulphhydryl compounds detoxify this substance, but in overdose, these are exhausted and the toxic agent causes a profound centrilobular necrosis. The plasma half-life is a guide to hepatotoxicity; it is dangerous to have a half-life of more than two hours at a level of 300mg/L at four hours after ingestion. Typical blood levels in overdoses when at least 10-15gm

has been taken are 100-400mg/L, with an average around 250 and the urine may contain 150-800mg/L. All the levels depend on dose and survival time.

Mechanism of Toxicity

N-acetyl-benzoquinone-imine is a highly reactive arylating minor metabolite of Paracetamol, which is detoxified by conjugation with glutathione. When a very large dose is taken, hepatic glutathione is depleted and this metabolite binds covalently to proteins in liver cells and renal tubules causing necrosis. Toxicity thus shows a threshold phenomenon, manifesting only when the glutathione is depleted to a critical point. In chronic alcoholics, even 5-6gm/day taken for a few days can cause hepatotoxicity. Paracetamol is not recommended in premature infants that are of less than 2 kg for fear of hepatotoxicity.

Signs and Symptoms

Early manifestations: (i) Nausea, vomiting and abdominal pain (ii) Tenderness of liver (iii) No impairment of consciousness

After 12-18 hours: (i) Centrilobular hepatic necrosis (ii) Renal tubular necrosis (iii) Hypoglycemia progressing to coma (iv) Fulminating hepatic failure and death may occur when plasma levels are above the level of 200µg/mL at 4 hours and 30µg/mL at 15 hours.

Treatment

Treatment should be started as early as possible as it is practically ineffective if started 16 hours or more after Paracetamol ingestion: (i) Gastric lavage and induction of vomiting (ii) Activated charcoal given orally or through the tube (iii) N-acetylcysteine 150 mg/kg should be infused intravenously over 15 minutes followed by the same dose over the next 20 hours. Alternatively, 75 mg/kg may be given orally every 4-6 hours for 2-3 days. It replenishes the glutathione stores of liver and prevents binding of the toxic metabolite to other cellular constituents.

Autopsy Findings

(i) Nothing specific is in the gastrointestinal tract (ii) In massive overdoses, death may result from depression of central nervous system (iii) Most deaths are delayed for 2-4 days when liver failure develops; the liver may be enlarged but is often < 1500 gms. It is pale yellow to tan coloured and centrilobular necrosis is visible microscopically (iv) Renal tubular necrosis may be seen (v) Myocardial fibril damage may be visible histologically.

INSULIN POISONING

Medicolegal Aspects

Death from parenteral administration of insulin is quite common. Fatal insulin toxicity can be accidental, suicidal and even homicidal. Accidental fatalities are mostly medical errors resulting from misreading the label on the box or ampoule. Suicide insulin is also quite common. Insulin is inactive orally and has to be given parenterally for its hypoglycemic action.

Autopsy Findings

1. Peripheral blood samples, skin, and underlying tissue from the injection as well as control site should be preserved. The needle marks are usually not evident at autopsy unless a small vessel has been damaged. The samples should be either frozen or kept in refrigerator.
2. Frozen serum separated from red cells should be sent for analysis.
3. Porcine or bovine insulin can be detected as such but immunoassay of the insulin itself, measuring C-peptide, produced on a one-to-one basis by the pancreas assists in distinguishing endogenous from exogenous insulin.
4. Attempting to prove insulin-induced hypoglycemia by measuring glucose levels in human post-mortem fluids is impracticable, due to the unreliability of such estimations after death.
5. Very low vitreous humour glucose levels suggest hypoglycemia but not well acceptable.

Food Poisoning

Food poisoning or the food borne illness includes all the illnesses resulting from ingestion of food containing bacterial products, toxins, viruses, prions or parasites or acute gastroenteritis due to bacterial infection of food or drink. The contamination may result from improper handling, preparation or storage of food. Food borne illness can also result by adding pesticides or medicines to food or by accidentally consuming naturally poisonous substances like poisonous mushrooms or fish. Contact between food and pests, especially rodents, flies and cockroaches is a further cause of contamination of food.

The **WHO** defines it as 'diseases, usually either infectious or toxic in nature, caused by agents that enter the body through the ingestion of food.'

Ptomaines: An early theory on the causes of food poisoning involved the ptomaines that are alkaloidal bodies are formed as a result of bacterial decomposition of protein in decaying animal or vegetable protein. These alkaloidal bodies when formed in the dead tissues are called 'cadaveric alkaloids'. These are not bacterial poisons and do not cause food poisoning but are formed only when food becomes too disagreeable to eat. The ptomaines are usually nonpoisonous except for neurine and mydalin that are produced in traces 5-7 days after death. The signs and symptoms produced resemble that of atropine poisoning.

Leucomaines: These are alkaloids that are secreted by living cells during metabolism that are toxic when injected but harmless when they are ingested. While some poisonous alkaloids are the cause of poisoning, the discovery of bacteria left the ptomaine theory obsolete

Causes and Incidence of Food Poisoning

(i) Poisoning due to bacteria and toxins (ii) Poisoning of vegetable origin (natural food poisons): (a) *Lathyrus sativus* (b) Poisonous mushrooms (c) Ryes, Oats, Barley etc. (d) Poisonous berry such as *Atropa belladonna* (e) *Argemone mexicana* (f) Cotton seeds (g) Groundnuts (h) Cabbage (i) Soyabeans (j) Sweet clover (iii) Poisons of animal origin (a) Poisonous fish (b) Mussel (iv) Chemicals that are added intentionally (a) Flavouring agents in processed form (b) Colouring agents (c) Preservatives (d) Extraction of fat by solvents like hydrocarbons (e) Accidentally added such as pesticides and insecticides (f) Products of food processing such as smoking of fleshy food (g) Radionuclides.

Incidence: (i) Chemical—28.4% (ii) Staphylococcal—21.4% (iii) *Salmonella*—17.4% (iv) *C. botulinum*—10.4% (v) Parasitic—8% (vi) *C. perfringens*—7.5% (vii) Other bacterial—4% (viii) Viral—3%.

Infectious dose: The infectious dose is the amount of agent that must be consumed to give rise to symptoms of food poisoning. The infective dose varies according to the causative agent and the victim's age and generalized health. In *Salmonella* food poisoning, as few as 15-20 cells may suffice.

BACTERIAL FOOD POISONING

Bacterial infection is the most common cause of food poisoning. The symptoms are delayed for 12-36 hours after consumption of contaminated food. Bacterial food poisoning is divided into two groups: (i) **Infection type:** The pathogenic orga-

Table 49.1: Difference between infection and toxin types of bacterial food poisoning

Features	Infection type	Toxin type
Causative organisms	1. <i>Salmonella group</i> (i) <i>S. enteritidis</i> (ii) <i>S. typhimurium</i> (iii) <i>S. choleraesuis</i> (iv) <i>S. paratyphi</i> 2. <i>Shigella sonnei</i> 3. <i>Proteus</i> , <i>E. coli</i> , <i>Streptococci</i> , <i>Sh flexneri</i> are also involved	1. <i>Staphylococcus toxin</i> 2. <i>Botulinum toxin</i> 3. <i>Bacillus cereus</i>
Type of food ingested	Ingestion of contaminated, uncooked or imperfectly cooked food	Ingestion of: (i) Preformed toxin in prepared (canned and preserved) food (ii) Imperfectly or undercooked food such as cheese, meat, fish, and eggs

nisms in food multiplies in the body (ii) **Toxin type:** Ingestion of poisonous substances that are formed in the food due to bacterial proliferation (Table 49.1).

The common bacterial foodborne pathogens are *Aeromonas*, *Bacillus*, *Brucella*, *Campylobacter*, *Corynebacterium*, *Coxiella burnetii*, *E. coli*, *Listeria*, *Salmonella*, *Shigella*, *Streptococcus*, *Vibrio cholera*, *Vibrio parahaemolyticus* and *Yersinia enterocolitica*. The incubation period of various food poisonings (Table 49.2).

Table 49.2: Incubation periods of food poisoning caused by various organisms

Causative organism	Incubation period
<i>C. botulinum</i>	2 hrs-several days (12-36 hrs)
<i>C. perfringens</i>	8-20 hrs
<i>Salmonella</i> except <i>S. typhi</i>	12-48 hrs
<i>S. typhi</i> causing typhoid fever	10 days or less to 14 days or more
<i>Staphylococcus aureus</i>	1-6 hrs (24-48 hrs)

Some foodborne illnesses are caused by exotoxins that are secreted by the cell as the bacterium grows. Exotoxins can produce illness even when the microbes that produced them have been killed. Symptoms typically appear after 1-6 hours depending on the amount of toxin ingested. These are caused by *Clostridium botulinum*, *Clostridium perfringens* and *Staphylococcus aureus*.

Clostridial food Poisoning

Clostridium is gram positive, anaerobic, spore forming bacilli that are responsible for three major diseases namely tetanus (*Cl. tetani*), gas gangrene (*Cl. perfringens*, *Cl. septicum*, *Cl. novyi*, *Cl. histolyticus* & *Cl. fallax*), food poisoning (*Cl. botulinum* & *Cl. perfringens* type A & C and acute colitis (*Cl.*

difficile). Infant botulism is produced by ingestion of contaminated honey.

***Clostridium perfringens* food poisoning:** The organism is a square ended gram positive, non-motile rod that is fastidious anaerobe. It is an important cause of gas gangrene. The incidence is more often reported at restaurants and public gatherings. The organism is found in soil and is a normal inhabitant of intestinal tract of man and animals. It has been isolated from meat, poultry and fish purchased from the market. The organism is heat resistant and if the meat is left unrefrigerated the level rises to hazardous concentrations. The organism grows in a food is improperly refrigerated after initial cooking.

***Clostridium botulinum* toxin:** *Cl. botulinum* elaborates powerful exotoxin that is produced intracellularly and released only on the death and autolysis of the organism. This is the most powerful toxin known to humans. The lethal dose for humans is just 1-2 mg or 1 picogram/kg. It is basically a neurotoxin that acts slowly taking several hours to kill. The food contaminated by the toxin can be rendered safe by pressure cooking or boiling for 20 minutes. The spores resist boiling at 100°C even for several hours.

Mode of action: Botulinum toxin enters preganglionic nerve terminals by endocytosis and binds rapidly and irreversibly to the cell membrane. It inhibits calcium dependent exocytosis preventing the release of Acetyl choline that results in pre-synaptic blockade and interference with cholinergic transmission at all acetylcholine dependent synapses in peripheral nervous system. The toxin has no effect on the central nervous system or on

axonal conduction system.

Food borne botulism: This type of botulism occurs when *Cl. botulinum* multiplies in food such as Sausages, tinned meat, fish and fruits and vegetables infected with soil contamination, as *Cl. botulinum* is normally present in the soil. The incidence of food borne botulism in: (i) Canned food is 2% (ii) Restaurant food is 4% (iii) Family home canning is more than 90%.

It produces a powerful exotoxin and is caused in humans by its distinct strains type A, B, E and rarely F. The food contaminated by type A, B & E strains looks and smells abnormal due to the action of proteolytic enzymes and that contaminated by type E strain is normal in appearance and odour. The initial phase is so subtle as to go unnoticed or misdiagnosed. The clinical features are: (i) Vomiting, thirst and abdominal pain (ii) There may be constipation (iii) Ptosis, difficulty in visual accommodation, mydriasis and diplopia (due to ocular paresis) (iv) Dysphonia and dysphagia (v) Bilaterally symmetrical descending motor paralysis beginning with abducens or oculomotor nerve palsy and progressing to respiratory insufficiency (vi) Urinary retention (vii) Mental status, sensorium, reflexes, body temperature and pulse are usually normal.

Wound botulism: It is a rare life threatening complication of trauma. It occurs after spores of *Cl. botulinum* have germinated in a wound and the produced botulinum toxin results in flaccid paralysis. It may also result from fatal intravenous drug abuse. Cocaine and Heroin when injected subcutaneously result in wound botulism. Deep wounds crush injury and compound fracture treated with open reduction predisposes to wound botulism. The incubation period is 4-18 hours and the clinical features are: (i) Fever associated with sinusitis, abscess and tissue infection acts as a focus of infection (ii) There is absence of gastrointestinal manifestations (iii) Cranial nerve palsies result in ptosis, diplopia, poor accommodation, ophthalmoplegia, dysphagia, Dysphonia and dysarthria (iv) Flaccid paralysis (v) Shortness of breath (vi) Respiratory failure.

Infant botulism: This type of botulism results from the ingestion of *Cl. botulinum* organism with subsequent in vivo production of the toxin followed by absorption from the gastrointestinal tract. Bacterial growth associated with breast-feeding may favour *Bifidobacterium* development instead of bacteria known to inhibit *Cl. botulinum* i.e. *Coliforms*, *Enterococcus* and *Bacteroides* species. Honey is the most common food source contaminated with *Cl. botulinum* spores. The clinical features produced are: (i) Feeding difficulties (ii) Constipation (iii) Feeble crying (iv) 'Floppy' baby with decreased muscle tone particularly of neck and limbs (v) Loss of facial grimacing (vi) Ophthalmoplegia (vii) Diminished gag reflex (viii) Dysphagia (ix) Poor anal sphincter tone (x) Respiratory failure.

Diagnosis: The diagnosis is made from: (i) History of ingestion of canned food, vegetables, fruits and honey is there (ii) Clinical features (iii) Laboratory analysis of sample of blood, stool, vomitus, gastric contents and suspected food item, which are tested for *Cl. botulinum* and botulinum toxin (iv) *Tensilon test*: Tensilon (edrophonium) is a rapid acting anticholinesterase used to differentiate botulism from myasthenia gravis. 10mg of the drug is injected slowly intravenously, 1-2 mg at first followed by rest of it by next 5 minutes. The muscle strength in myasthenia will improve with in ½-1 minute and last for about 5 minutes and there will be no or little improvement in botulism (v) *Electromyography*: In all forms of botulism, EMG pattern is characterized by brief, small, abundant motor unit action potential. The motor nerve conduction velocity is normal.

Treatment

1. All patients should be hospitalized.
2. Respiratory status should be monitored for vital capacity, peak expiratory flow rate, negative inspiratory force, pulse oximetry and gas reflex. The moment signs of bulbar palsy occur; intubation or tracheotomy should be done.
3. Evacuate GIT of spores and toxins with the help of activated charcoal, emetics, gastric lavage and catharsis-Sorbitol.

4. *Botulinum antitoxin*: Trivalent botulinum antitoxin is available in 10mL vial containing: (i) 7500 IU of type A (ii) 5500 IU of type B (iii) 8500 IU of type E produced by horses immunized against botulinum toxin and then defibrinated, digested and dialyzed and prepared as a 20% protein antitoxin. Equine globulin preparation is available in the west since 1960s but not yet produced in India. One vial is administered by slow i.v. as a 1:10 vol/vol dilution in 0.9% saline, and intramuscular dose of single vial and then after 2-4 hours again i.v. dose is administered. The antitoxin is not administered in infant botulism. It has got serious side effects such as anaphylaxis and serum sickness. It has a lack of effect on toxin producing organisms in the gut.
5. *Guanidine*: Its use is controversial as its efficacy is low and there is high incidence of adverse effects. If found appropriate, the dose is 15-40 mg/kg/day orally until EMG improvement occurs at least in ocular muscles.
6. *Human derived Botulism Immune Globulin (BIG)*: It is used to treat infant botulism. It is a pentavalent (types A, B, C, D & E) immune globulins harvested by plasmapheresis from donors who received multiple immunizations with pentavalent botulinum toxoid. It avoids the use of foreign equine protein, thereby eliminating the risk of hypersensitivity reactions.
7. *Supportive measures*: These include the nutritional supplementations as oral feeds are contraindicated unless there is intact gag reflex and respiratory supportive measures.

Prevention of Botulism

(i) Improperly preserved home canned food should be avoided especially the green beans, Asparagus and peppers (ii) The food should be pressure cooked at >100°C for 10 minutes that kills spores (iii) Jams and jellies can be consumed, as they do not contain Cl botulinum due to their high sugar content (iv) Cooked food should not be kept at temperatures of 4°-6°C for more than four hours (v) Boiling the food before eating destroys the toxin (vi) Food pH should be maintained to less than 0.5; NaCl should be more than 3.5% and nitrites

level should also be sufficiently high (vii) Acidifying agents like citric and phosphoric acids are used while canning low acid food like mushrooms, beets, beans, corns and olives etc.

Medicolegal Aspects

The poisoning is commonly accidental and in literature, there is no incidence of use of botulinum toxin as homicide. Its incidence is more common in western countries and recently in Iran, Russia and Japan. It is quite rare in developing countries like India due to non-popularity of preserved and canned food.

Autopsy Findings

(i) The visceral organs and meninges are congested (ii) The mucosa of gastrointestinal tract is congested, swollen and haemorrhagic (iii) Microscopically fatty degeneration of the liver can be seen.

Viruses

Viral infections make up perhaps one third of cases of food poisoning in developed countries. They are usually of intermediate (1-3 days) incubation period, cause illnesses which are self-limited in otherwise healthy individuals, and are similar to the bacterial forms described above. The viruses that commonly cause food poisoning are: (i) Norovirus (formerly Norwalk virus) (ii) Rotavirus (iii) Hepatitis A is distinguished from other viral causes by its prolonged (2-6 week) incubation period and its ability to spread beyond the stomach and intestines, into the liver. It often induces jaundice, or yellowing of the skin, and rarely leads to chronic liver dysfunction (iv) Hepatitis E.

Parasites

Most foodborne parasites are zoonoses and are caused by: (i) Platyhelminthes (*Taenia saginata* & *Taenia solium*) (ii) *Fasciola hepatica* (iii) Nematode (*Ascaris lumbricoides*, *Trichinella spiralis* and *Trichuris trichiura*) (iv) Protozoa (*Acanthamoeba* and other free-living amoebae, *Cryptosporidium parvum*, *Entamoeba histolytica*, *Giardia lamblia*, *Sarcocystis hominis*, *Sarcocystis suihominis*) (v)

Toxoplasma gondii.

Prevention of Food Poisoning

1. In a final product, it must be important to know the origin of the ingredients (originating farm, identification of the harvesting or of the animal) and where and when it was processed.
2. Respect of hygiene procedures maintenance of "cold chain"
3. There should be power of control and law enforcement of the veterinarians.
4. At home, the prevention mainly consists of: (i) Separating foods while preparing and storing to prevent cross contamination. (i.e. clean cutting boards, utensils, and hands after handling meat and before cutting vegetables, etc.) Wash hands and/or gloves before handling ready-to-eat foods (ii) The respect of the food storage (hot foods hot and cold foods cold) and food preservation methods (especially refrigeration), and checking the expiration date (iii) Washing the hands before preparing the meal and before eating (iv) Washing the fresh fruits and vegetables with clear water, especially when not cooked (e.g. fruits, salads) Scrub firm fruits and vegetables with a brush to clean (v) Washing the dishes after use (vi) Keeping the kitchen and cooking utensils clean. (vii) Bacteria need warmth, moisture, food and time to grow. The presence, or absence, of oxygen, salt, sugar and acidity are also important factors for growth. In the right conditions, one bacterium can multiply using binary fission to become four million in eight hours. Since bacteria can be neither smelled nor seen, the best way to ensure that food is safe is to follow principles of good food hygiene. This includes not allowing raw or partially cooked food to touch dishes, utensils, hands or work surfaces previously used to handle even properly cooked or ready to eat food (viii) High salt, high sugar or high acid levels keep bacteria from growing, which is why salted meats, jam, and pickled vegetables are traditional preserved foods (ix) The most frequent causes of bacterial foodborne illnesses are cross-contamination and inadequate temperature control. Therefore control of these two

matters is especially important (x) Thoroughly cooking food until it is piping hot, i.e. above 70 °C (158 °F) will quickly kill virtually all bacteria, parasites or viruses, except for *Clostridium botulinum* and *Clostridium perfringens*, which produces a heat-resistant spore that survives temperatures up to 100 °C (212 °F). Once cooked, hot foods should be kept at temperatures out of the danger zone. Temperatures above 63 °C (145 °F) stop microbial growth (xi) Cold foods should also be kept colder than the danger zone, below 5 °C (41 °F). However, *Listeria monocytogens* and *Yersinia enterocolitica* can both grow at refrigerator temperatures.

POISONOUS FOODS

POISONOUS MUSHROOMS

The *Amanita* genus of mushroom that is *Amanita phalloides*, *Amanita muscaria* and *Amanita pantherina* are responsible for most cases of mushroom poisoning. These are

Amanita Phylloides Poisoning

This specie of mushroom is very toxic even after cooking. The onset of symptoms is slow and the prognosis grave. The mushroom is responsible for majority of deaths in cases of mushroom poisoning. The toxic constituents are peptides.

Signs and symptoms: The signs and symptoms vary with the passage of time after ingestion of mushroom: (i) *After 8-12 hours of ingestion* (a) nausea (b) vomiting (c) diarrhoea and dysentery (d) intense thirst (ii) *Within 24 hours of onset:* (a) circulatory collapse (b) restlessness (c) twitching (d) delirium proceeding to coma (iii) *After 3-4 days* (a) hepatic damage (b) renal damage (c) death after 2-4 days.

Treatment: (i) Gastric lavage (ii) Saline cathartics (iii) Symptomatic

Medicolegal aspects: (i) Mainly accidental poisoning (ii) The mushrooms have been rarely used for homicidal purposes.

Autopsy findings: (i) Extensive fatty infiltration of the liver (ii) Renal tubular necrosis (iii) Haemor-

rhages in gastrointestinal tract (iv) Renal and hepatic haemorrhages (v) Basal ganglia necrosis.

Amanita Muscaria and Amanita Pantheria Poisoning

Both these mushrooms contain muscarine and give rise to a syndrome resembling that seen in pilocarpine poisoning

Signs and symptoms: They appear within a short time of ingestion of the mushroom: (i) Profuse salivation (ii) Perspiration (iii) Severe nausea, vomiting (iv) Watery diarrhoea (v) Pupil is miotic with no reaction to light (vi) Muscular twitching may be present (vii) Sensorium is usually clear (viii) Maniac excitement or hallucinations may be present occasionally more common with *A. pantheria* than *A. muscaria* (ix) In fatal cases death may occur within a few hours.

Autopsy findings: (i) Congestion of all visceral organs (ii) Findings suggestive of effects of hypoxia.

TOXIC HONEY

The Bees have little selection in the type of nectar, which they transport back to the hive to make honey, as it requires about 17,000 bee-loads to make 1kg of honey, the stand of poisonous plants must be rather great for enough of the toxic material to get in to the honey. The most common type of toxic honey is that from the laurels or rhododendrons. The following plants have been thought to be present in large enough plantings to cause production of toxic honey: (i) The laurels: Sheep laurel (*Kalmia angustifolia*), Mountain laurel (*K. latifolia*), Swamp laurel (*K. polifolia*), Great laurel (*Rhododendron maximum*), Black laurel (*Leucothoe davisiae*), Dog laurel (*L. catesbaei*) (ii) Rhododendron (*Rhododendron species*): California rose bay (*R. californicum*) and Rose bay (*R. catawbiense*) (iii) Bog rosemary (*Andromeda glaucophylla*) (iv) Jasmine (*Gelsemium sempervirens*) (v) Soapberry (*Sapindus drumondii*) (vi) Indian hemp (*Cannabis indica*) (vii) Oleander (*Nerium odorum*) (viii) Black locust (*A. androsaemifolium*) & Clammy locust (*R. viscosa*) (ix) Lily of the valley (*Convallaria majalis*) (x) Death campus (*Zygadenus*) (xi) California buckeye

(*Aesculus californican*) and Horse chestnut (*A. hippocastanum*) (xii) Dogbane (*Apocyanum cannabinum*).

Signs and symptoms: (i) Are of the toxic material in the honey? (ii) Tingling of fingers (iii) Vomiting and diarrhoea (iv) Inability to stand (v) Features of mania.

FISH POISONING

Poisoning by ingestion of fish may result from bacterial growth in partially decomposed fish or by the presence in certain fishes of a neurotoxin. The neurotoxin is present in the whole fish but is concentrated in the ovaries, testes and liver. This is particularly marked during spawning. The syndrome is called 'ichthyotoxicosis' or 'ciguatera'

Signs and symptoms: The signs and symptoms begin within 10-30 minutes following ingestion of the fish: (i) Burning of the throat (ii) Nausea, vomiting and pain abdomen (iii) Ascending motor and sensory paralysis (iv) Mydriasis (v) Hypotension and bradycardia (vi) Respiratory difficulties (vii) Conduction disturbances (viii) Syncope and death resulting from respiratory failure.

Treatment: (i) Gastric lavage (ii) Symptomatic treatment (iii) Cerebral depressants should be avoided.

Venomous Fish

1. Some fishes cause poisonous wounds with their spines either while trying to get the fish off a hook or out of net. These fishes are commonly *Plutosus* and *Paraplutosus*. They cause painful wound with swelling and redness. The patient is treated with morphine and prevention of secondary infection.
2. One genus of fish, the *Muraena* (eel) inflicts poisonous bite producing local symptoms of pain to general symptoms of coma, respiratory failure and death
3. The stingrays' fishes are most important, the tail has a series of barbs which close down for the thrust into the victim, flesh and then swing open to make the exit very painful. These fishes are fat and lie in the sand and mud at the bottom of water and attack with the poison tail or string

when disturbed or frightened. The tail is cut out to remove it from the victim's body. Intense pain is produced and paralysis and other systemic manifestations may develop. Marked abdominal pain with cramps is produced. Respiratory difficulty, delirium and circulatory collapse progress until death ensues. In cases that recover, the necrosis of sting site occur and sloughing and healing may be delayed.

Coelenterate Urtication

This group of invertebrates includes jellyfish, corals, Portuguese man of war and medusae and is present in waters in great numbers. Especially dangerous is the *Rhizostoma* sp. that causes severe fatigue and depression along with others symptoms. This group of fish's causes release of an urticating fluid that contains several toxic fractions; a neurotoxin, a primary irritant, a haemotoxin and an anaphylactic principle. It causes intense burning pain and irritation, severe abdominal pain, muscular cramps, convulsions and respiratory depression. The treatment is symptomatic.

Shellfish Poisoning

Both mussels and clams contain neurotoxins during certain months. These are heat stable and not destroyed by cooking. The shellfish contains poisonous plankton ingested as food. Intoxication has been reported with two species of *Gonyaulax catanella*, these dinoflagellates in larger numbers impart a reddish tinge to the water and luminosity at night.

Signs and symptoms: The signs and symptoms appear within 10-20 minutes after ingestion of shellfish: (i) Nausea and vomiting (ii) Burning paraesthesiae of the hands and feet (iii) Numbness of lips and tongue (iv) Ataxia (v) Extreme mydriasis (vi) Convulsive seizures (v) Death from respiratory failure 2-12 hours later.

Treatment: (i) Gastric lavage (ii) Symptomatic.

LATHYRUS SATIVUS (Keshari Dal)

Keshari dal grows under extreme conditions of drought in several parts of India. It is a staple diet

of people from low socio-economic strata. Continuous consumption of this pulse in quantities more than 30% of the total diet for more than six months results in a condition called Lathyrism. When mixed with three times its weight of wheat, the grain becomes apparently harmless but when cooked alone, it acts as a poison. The pulse loses 90% of its toxic amino acid when it is steeped in hot water or parboiled

Lathyrism

Lathyrism is a condition characterized by features of paralytic manifestations due to the presence of a neurotoxin 1% BOAA [B (N) Oxalyl amino-L-Alanine], a free water soluble amino acid in the seed cotyledons. This neurotoxin has predilection for pyramidal tract.

Signs and symptoms: (i) The patient suffers from severe agonizing pain in the calf muscles and limbs that are paralyzed (ii) There is weakness in the legs with difficulty in sitting and getting up (iii) The patient later on finds difficult to walk with spastic gait characterized by walk on tip-toes (iv) Complete spastics paraplegia of lower limbs occurs in late stages and patient can just crawl (v) The motor nerves of the muscles of trunk, upper limbs and sphincters are spared (vi) There will be no atrophy or loss of muscle tone and no reaction of degeneration (vii) The knee jerks will be increased, ankle clonus well marked and extensor planter reflex (viii) There are no sensory changes and no loss of consciousness (ix) There is usually no involvement of bladder and rectum unless the lumbar segment gets affected.

Treatment: (i) No specific treatment (ii) The pulse should be excluded from the diet and if it is necessary to consume, it should never exceed 25% of the total amount of cereals and pulses consumed per day (iii) The diet rich in vitamin A, carotene and other vitamins should be given (iv) Physiotherapy for muscular and neurological involvement.

ARGEMONE MEXICANA

The plant *Argemone mexicana* grows widely all over India in winters at the same time when mustard plants are growing. The seeds are commonly con-

fused with the mustard seeds and are deliberately or accidentally mixed with mustard seeds. Mustard oil is widely used for cooking purposes in Bengal, Assam, Maharashtra, Orissa and Bihar and is often adulterated with the oil of *Argemone mexicana*. The plant has thistle like, sessile and spiny leaves and yellow flowers. The seeds of argemone are dark brown, globular, smaller than the mustard seeds and are covered by regular minute projections and depressions. The seeds on pressing burst with a sound giving out argemone oil whereas mustard seeds on pressing burst without any sound and give out mustard oil.

All parts of this plant are poisonous and seeds being more poisonous. The plant contains two alkaloids—*berberine* and *protopine*, whereas oil extracted from the seeds contains—*sanguinarine* and *dihydrosanguinarine*, the former being more toxic. The adulteration of mustard oil with the oil of *Argemone mexicana* causes Epidemic dropsy and glaucoma. The symptoms usually break out in an epidemic form amongst the consumers of adulterated oil (Fig. 49.1).

Signs and symptoms: The symptoms appear gradually on consumption of the adulterated oil: (i) Loss of appetite, nausea, vomiting, dyspepsia and diarrhoea (ii) Oedema of legs and generalized anasarca (iii) Fall of blood pressure, quick weak pulse, dyspnoea with features of myocardial damage (iv) Breathlessness on slight exertion (v) Enlarged, tender liver (vi) Subcutaneous telangiectasis or hemangiomas due to peripheral vasodilatation (vii) Small fleshy dark red growths may be noticeable on various body parts and also



Figure 49.1:
Argemone mexicana

on the mucosa of cheek, gums, tongue and nose (viii) Tingling and hyperaesthesia with diminished or even absent jerks (ix) About 1/10th of the cases develop glaucoma with dimness of vision and raised intraocular pressure (x) The application of adulterated oil on scalp causes burning and fall of hairs (xi) Death results from heart failure.

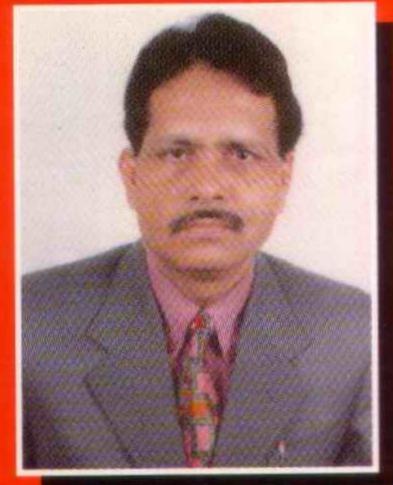
Tests for Argemone

To the edible oil mix equal volumes of nitric acid, the development of crimson colour indicates presence of argemone oil

Treatment: (i) The adulterated oil should be discarded and sent for analysis (ii) Mainly symptomatic treatment should be undertaken (iii) Supportive treatment to combat heart damage in the form of digitalis, diuretics and prednisolone (iv) Good nutritious diet with multiminerals and multivitamins especially B complex group should be administered.

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- ★ He has edited a textbook 'HWV Cox textbook on Medical Jurisprudence and Toxicology' and a book on MCQs.
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- ★ He is a member of the expert committee of University Grants Commission.



The book covers basic concepts of Forensic Medicine including Medical Jurisprudence, Forensic Pathology, Toxicology and Forensic Sciences in an easy to understand format. All the chapters are concise as well as comprehensive incorporating tables, diagrams, photographs and salient features for a better grasp of the subject. It is a reader friendly book primarily aimed for undergraduate and postgraduate students and Medical Officers posted in Health care settings and would be beneficial for judiciary and lawyers too. Moreover, there is inclusion of laws and Acts wherever relevant. The book also incorporates possible questions at the end of each chapter for preparation during the examination.

ISBN 81-88867-96-9



Code:969 Rs.295.00

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Publishers and Distributors (P) Ltd.

7/31, Ansari Road, Daryaganj, Post Box-7243

New Delhi-110002 (India)